Annu. Rev. Phytopathol. 1991. 29:279–303

BREEDING FOR DISEASE RESISTANCE IN PEANUT (*ARACHIS HYPOGAEA* L.)

J. C. Wynne and M. K. Beute

Departments of Crop Science and Plant Pathology, North Carolina State University, Raleigh, North Carolina 27695

S. N. Nigam

Groundnut Improvement Program, International Crops Research Institute for the Semi-Arid Tropics, Patancheru, A.P. 500324, India

KEY WORDS: groundnut, genetics of peanut, oilseed crop, pathogen variability, food legume

INTRODUCTION

The peanut (*Arachis hypogaea* L.) is an important oilseed crop and food legume grown on approximately 20 million ha in warm tropical or subtropical areas throughout the world. Diseases of the peanut reduce yield and quality and increase the cost of production wherever the crop is grown. Because of the economic impact of diseases, much effort has been given to developing both chemical and nonchemical disease management strategies. The use of crop protection chemicals in areas where the crop has high value such as the United States, not only adds to production costs but is becoming controversial because of environmental and food safety concerns. In many developing countries where chemicals are not readily available, diseases cause significant losses in spite of management strategies designed to manage or control the disease. Thus, one major objective of peanut breeding programs throughout the world is to develop disease-resistant or tolerant cultivars that can be used in managing or controlling peanut diseases.

0066-4286/91/0901-0279$02:00

Diseases of Peanut

During the early years of cultivation in the US, the peanut was regarded as relatively free from diseases (45). However, diseases of the peanut now occur throughout the growing season and into the postharvest period, and attack all parts of the plant. Diseases of the peanut are caused by fungi, bacteria, viruses, nematodes, and a mycoplasm (109). Only a few diseases are of worldwide importance. Most diseases and pathogens are of local or sporadic importance (4). Of the fungal diseases, early leafspot (CA) caused by *Cercospora arachidicola,* late leafspot (CP) caused by *Cercosporidium personatum,* and rust caused by *Puccinia arachidis* have widespread occurrence and are of great economic importance (24). The aflatoxin problem associated with the fungi *Aspergillus flavus* and *A. parasiticus* is also of great economic importance. Two virus diseases, rosette and tomato spotted wilt, can cause severe losses even though the peanut mottle virus is more widespread. Although several nematodes attack peanut, the two root knot nematodes, *Meloidogyne arenaria* and *M. hapla,* are the most important.

Breeding for Resistance

Progress in breeding for disease resistance in the peanut has lagged behind that for many major crops for several reasons including: (*a*) the late initiation of breeding programs for the crop; (*b*) the regional importance of the crop in the US; (*c*) the relatively few scientists assigned to peanuts; and (*d*) the lack of financial resources for peanut research (148). Most of the literature on peanut diseases prior to 1970 was published by American scientists (89). Because of the high value of the crop in the US and the availability of chemicals for disease control, breeding for disease resistance was not given a high priority until the late 1970s. There was also a prevalent idea that variability for resistance among cultivated peanuts was lacking (56). However, during the past 15 years, numerous germplasm accessions among cultivated peanuts have been identified as sources of disease resistance. Breeding for disease resistance has received high priority by scientists at the International Crops Research Institute for the Semi-Arid Tropics (ICRISAT), which adopted peanut as a mandate crop in 1976 (48) and received additional emphasis in the US when the United States Agency for International Development (USAID) funded the Peanut Collaborative Research Support Program (CRSP) in 1982. Breeders have tended to focus on the major diseases of peanut, although breeding for locally important diseases such as rosette virus in Africa or Cylindrocladium black rot (CBR) in North Carolina and Virginia has also received considerable attention.

HOST VARIABILITY

One of the first and most important phases of breeding for disease resistance is to identify a source of resistance. In peanut, as in other crops, it is most desirable to find resistance in closely related materials such as local or foreign cultivars or landraces. The less related the source of resistance to the germplasm being improved, the more difficult it is to transfer resistance without also transferring undesirable genes or gene complexes. Much of the variability found in the cultigen, including genes for disease resistance, arose in South America where the peanut is native (56). Six centers of genetic diversity (gene centers) for the cultivated peanut are recognized in South America: (*a*) Peru, (*b*) northeastern Brazil, (*c*) the Guarani region (Paraguay and southeast Brazil), (*d*) Rondonia and northwest Mato Grosso (Brazil), and (*e*) the eastern foothills of the Andes in Bolivia. The cultivated peanut has been classified into two subspecies, *A. hypogaea hypogaea* Krap. et Rig. and *A. hypogaea fastigiata* Waldron. These two subspecies have been subdivided into two botanical varieties, with three of the four varieties being grown commercially in the US (56). In addition to the cultivated peanut, considerable variation for disease resistance exists among the species of *Arachis* (68). All species of *Arachis* are native to South America and provide potential sources of disease resistance for use in genetic improvement. The genus probably originated in central Brazil and was first domesticated in northern Argentina and eastern Bolivia. Peanut was thought to have been taken from Brazil to Africa, India, and the Far East by the Portuguese; from the west coast of South America to the Western Pacific, Indonesia, and China by the Spaniards early in the 16th century. By the mid-16th century, peanuts were grown in North America and were distributed worldwide (24). Africa is an important secondary center of genetic variation despite evidence that introductions to Africa were from a single center in South America (60).

Since the initial introduction of peanut to the US, additional germplasm has been collected through expeditions sponsored by the US Department of Agriculture, with the cooperation of state experiment stations and several foreign countries. Approximately 6000 accessions have been assembled by the Southern Regional Plant Introduction Station at Experiment, Georgia. Nearly 12,000 collections are being maintained by ICRISAT at Hyderabad, India. Collections of cultivated and wild species of *Arachis* (maintained at ICRISAT, in Argentina and Brazil, and at several state experiment stations in the US) provide a major genetic resource for resistance to diseases, insect pests, and other desirable traits (148).

The first attempt to use these genetic resources to develop a disease-resistant cultivar was made in 1927 in East Java (Indonesia) by Dutch sci-

entists who developed Schwarz 21, a cultivar resistant to *Pseudomonas solanacearum* (23). The cultivar NC 2, released in 1953, was selected for partial resistance to *Sclerotium rolfsii* (30). Characterization of components of partial resistance of NC 2 indicated that reduced disease was related to plant phenology; however, NC 2 also suppressed inoculum production (117).

In spite of these early successes in exploiting host-plant variability, breeding for disease resistance did not receive high priority until the late 1970s. The increasing cost of production, the failure of chemical control methods to work effectively against diseases such as CBR caused by *Cylindrocladium crotalariae,* the availability of new accessions of cultivated peanuts, and the adoption of peanut as a mandate crop by ICRISAT in 1976 resulted in an increased priority for breeding for disease resistance.

With increased emphasis in screening peanut for disease resistance, numerous genotypes with resistance to several pathogens have now been identified.

Soil-Borne Pathogens

Screening for resistance to the CBR disease, identified in North Carolina and Virginia in 1970, was initiated in greenhouse tests in 1973 (149). Over 1200 genotypes were evaluated in preliminary screening. Although field screening was not extensive because of the necessity of measuring near-homozygous lines for percent visibly infected plants in replicated trials (53) (i.e. less than 150 genotypes), a few virginia types and several spanish types were confirmed as resistant. Subsequent heritability studies indicated that resistance was quantitative, with only additive genetic effects important in the inheritance of resistance (55, 58).

NC 3033, a line resistant to CBR and also found to be resistant to *Sclerotium rolfsii* (15), was used as a parent to develop breeding lines resistant to both CBR and southern stem rot (115). Genotypes resulting from crosses with commercial cultivars expressed partial resistance to southern stem rot in the field and greenhouse; combining phenological suppression (disease escape) and metabolic resistance was considered possible (117). However, because of limited resources, it was decided to emphasize breeding for CBR resistance and only evaluate genotypes that were resistant to CBR for resistance to southern stem rot.

Exploitation of PI 341885, Toalson, and TxAG-3 (a selection from PI 365553), which are resistant to southern stem rot and pythium pod rot caused by *Pythium myriotylum,* has been pursued in the Texas peanut breeding program (122). Breeding lines using these sources of resistance as parents varied in reaction to the two diseases, with some lines showing considerable resistance to both pathogens. Apparently the two mechanisms of resistance

differ and are independent. However, progeny was not as resistant as the resistant parents, and quality was reduced (122).

Resistance has also been found for sclerotinia blight caused by *Sclerotinia minor* (29). The cultivar Va 81B was selected for resistance to Sclerotinia blight in Virginia. Resistance in Va 81B is primarily phenological. Additional sources of resistance to Sclerotinia including Chico, germplasm from Texas (TX 498731, TX 798736, TX 804475) and Virginia (TRC 02056-1), and seven plant introductions from China (PIs 467829, 476831, 476834, 476835, 476842, 467843, 467844) are presently being evaluated as resistant sources in Virginia and North Carolina.

In the past, aflatoxin was considered predominantly a postharvest problem and as such, received little attention in crop improvement programs. In 1973, two germplasm lines, PI 337409 and PI 337394F, were found to be resistant to seed invasion and colonization by *A. flavus* (97). The screening was done on rehydrated sound mature seeds inoculated with conidia of *A. flavus* in an environment favorable to fungus development. Resistance to invasion and colonization by *A. flavus*, located in the seed coat, was suggested to be an effective means of preventing aflatoxin contamination. Differences have also been reported for the ability of peanut seeds to support the production of aflatoxins (11, 99, 111, 138). Several sources of resistance have been reported from Senegal (150), US (12, 84, 97), India (46, 90), China (137), and the Philippines (110). Sources resistant to preharvest field infection have also been reported from India (91), Senegal (144, 150), and the US (85). Three resistant genotypes—PI 337409, PI 337394F, and J 11—have been evaluated in more than one country. J 11 was found to be resistant to infection in the US and India. PI 337409 showed resistance in Senegal and India, but was susceptible in the US (85).

Percent colonization of seeds of F_1 and F_2 plants of reciprocal crosses between PI 337409 (resistant) and PI 331326 (susceptible) indicated a high broad-sense heritability (96). From the preliminary studies at ICRISAT on combining ability, using line x tester and diallel mating designs, Vasudeva Rao et al (142) reported UF 71513, Ah 7223, PI 337409, and PI 337394F as good combiners for seed coat resistance. Recent studies on heritability of all three types of resistances indicated that there was no correlation among mechanisms, suggesting that the three mechanisms are controlled by different genes (140).

Foliar Diseases

Considerable effort has been given to identifying sources of resistance to rust and early and late leafspot because of their worldwide importance. Sources of resistance to rust were identified more than two decades ago (22). In 1977, Hammons (59) reported resistant sources that consisted of three

lines: (*a*) Tarapoto (PIs 259747, 341879, 350680, 381622, 405132), (*b*) Israeli line 136 (PIs 298115 and 315608) and (*c*) DHT 200 (PI 314817). Tarapoto and DHT 200 both originated from Peru.

Resistance of PI 298115 to rust was controlled by two recessive genes (85). Resistance was also shown to be controlled by duplicate recessive genes in additional resistant sources, although some sources of resistance could not be explained by a two-gene system. Fourteen progeny from the cross of PI 298115 and an unknown pollen source were released as rust-resistant germplasm, FESR 1-14. ICRISAT has screened over 12,000 lines of *A. hypogaea* in the field using infector rows to develop disease pressure. One hundred fifty-three lines, including the 14 rust-resistant germplasm lines (FESR 1-14), have been identified as resistant (130). Subsequent studies indicated that many of the resistant lines also possess "rate-reducing" components, i.e. "slow rusting" epidemiological mechanisms. These genotypes had increased incubation period, decreased infection frequency, and reduced pustule size, spore production, and spore germinability (132, 133). Additive genetic effects and additive types of epistasis have been found for crosses of rust resistant by susceptible crosses (121).

Many wild *Arachis* species and their interspecific derivatives with the cultivated types have also been screened for resistance to rust under both field and laboratory conditions (135). Many species were found to be immune to rust. These include *A. batizocoi* (PI 298639, PI 338312), *A. duranensis* (PI 219823), *A. cardenasii* (PI 262141), *A. chacoense* (PI 276235), *A. pusilla* (PI 338449), *A. villosa* (PI 210554), and *A. correntina* (PI 331194), among others (131). Most of the interspecific derivatives showed a high degree of resistance to rust. They had small and slightly depressed uredosori that did not rupture to release the comparatively few urediospores produced. In some diploid wild *Arachis* species, rust resistance is partially dominant (121).

In recent years, germplasm for resistance to leafspots has been intensively screened in different parts of the world. Several sources of resistance to both CA and CP leafspots have been reported (40, 41, 52, 64, 92, 124). Screening for CP resistance has been most extensive at ICRISAT where genotypes screened for rust have also been screened for CP resistance. A total of 83 lines of *A. hypogaea* have been identified with some resistance and/or tolerance to CP while using infector rows for inoculation. Twenty-nine of these lines are also resistant to rust.

Among the many wild *Arachis* species screened for CP resistance, *A. chacoense* (PI 276325), *A. cardenasii* (PI 262141), and *A. stenosperma* (PI 338280) in section *Arachis* (cross-compatible with the cultivated *A. hypogaea*) showed either an immune or a highly resistant reaction to the pathogen. Highly resistant species in other sections included *A. repens, A. appressipila, A. paraguariensis, A. villosulicarpa, A. hagenbeckii,* and *A. glabrata* (134).

Only limited screening for CA resistance has been possible at ICRISAT Center. Germplasm already planted in the field is evaluated for CA whenever the disease incidence is high, which occurs every few years. Germplasm lines NC 3033, PI 270806, PI 259747, and PI 350680, which possess epidemiological components of rate-reducing resistance in the US (64, 124), did not maintain their resistance in India using infector row inoculation techniques (70). These lines were also found susceptible to the disease in Malawi using similar inoculation techniques (105). In 1987, screening on a limited scale was started in Pantnagar, India and, unexpectedly, disease at the ICRISAT Center was severe. This provided an opportunity to evaluate 3000 genotypes planted in the field. Several genotypes showed moderate levels of field resistance to early leafspot at Pantnagar and the ICRISAT Center. These include ICG 2711 (NC 5), ICG 6709 (NC Ac 16163), ICG 7291 (PI 262128), ICG 7406 (PI 262121), ICG 7630 and ICG 7892 (PI 393527-B), and ICG 9990 (145).

More than 1000 selected germplasm lines of cultivated types have been screened for CA resistance in Malawi using infector row inoculation. All failed to show any appreciable level of resistance to the disease. A "bulk" testing of germplasm was followed to include a large number of germplasm lines (S. N. Nigam, personal communication). One hundred and ten bulks, each with 500 seeds, were formed by compositing five seeds per germplasm line. This method allowed representation of 11,000 germplasm lines in the screening. Only two bulks had a few plants that warranted further testing. Component lines of these two bulks were planted separately and scored for the disease. Only three germplasm lines—ICG 50, ICG 84 and ICG 11282—have been retained for further testing (71). Other germplasm lines of *A. hypogaea* that retain a higher proportion of foliage in spite of heavy disease pressure are ICGM 189, ICGM 197, ICGM 281, ICGM 284, ICGM 285, ICGM 286, ICGM 292,ICGM 300, ICGM 473, ICGM 500, and ICGM 525. Thirty-five lines reported to have resistance to CA at the ICRISAT Center did not maintain their disease reaction in Malawi (71).

Many wild *Arachis* species and interspecific derivatives have also been screened for CA resistance in Malawi. Only *Arachis* species 30003 has consistently shown a high level of disease resistance using infector row inoculation. Other species, *A. chacoense* and *A.* sp. 30085, which showed high promise in the first year of screening, were subsequently rated as susceptible. *Arachis stenosperma,* which was rated as resistant in the US, was highly susceptible in Malawi (72). Several interspecific derivatives retained more foliage than the susceptible control.

Eight lines of *A. hypogaea* with moderate to high levels of resistance to the major foliage diseases—rust, CP, and CA—have been identified at the ICRISAT Center. These sources (with multiple foliar resistances) should

be used to produce new cultivars with resistance to the major foliar diseases. The rust and CP resistances of these genotypes are generally stable over a wide range of geographic locations. Only for NC Ac 17090 and PI 298115 has variation in rust scores been observed across locations.

Due to the observation that a high level of resistance to CA was not available for breeding, emphasis on developing CA resistance in the US has focused on incorporation of multiple components of rate-reducing resistance in commercial cultivars. Concerns about variability in virulence characteristics of both CA and CP were considered to be minimized by this strategy. Several sources of resistance to CA were identified in *A. hypogaea* (PI 109839, PI 270806, GP-NC 343, NC 3033, etc). Two diploid wild species, *A. chacoense* and *A. stenosperma,* were rated highly resistant (40). Studies on inheritance of resistance components for CA and CP were initiated (86). Substantial additive genetic effects have been found for both CA and CP among early generation progenies (9).

In studies conducted primarily in North Carolina, both additive and additive-by-additive epistasis have been found to be significant for progenies in late generation (54). Dominant genetic variance for CP in early generations was significant for several components of resistance (75). Estimates of narrow-sense heritabilities have ranged from low to high for components of resistance, with estimates varying considerably depending upon the component and cross. Estimates of broad-sense heritabilities have been higher indicating that nonadditive effects are also important.

Progenies in F_2 generation from crosses between two resistant and three susceptible cultivars were screened for components of resistance to CP in detached leaf tests at ICRISAT (104). A five-locus polygenic system assuming resistance to be completely recessive was proposed to explain the frequency of resistant plants in the F_2 generation. Nonadditive gene action was reported to be extremely important, but its nature could not be elucidated due to the absence of F_1 generation.

Except for rosette virus, evaluation of peanut for virus resistance has only recently occurred. Resistance to rosette virus was discovered in local land races in Burkina Faso in the 1950s (33). Resistance was reported to be due to the production of an antivirus substance by resistant plants (34). Resistant lines are not immune and individual plants can become infected with the disease under heavy inoculum pressure. This resistance apparently operates against both chlorotic (34) and green (61) rosette. Recent studies (98) have shown that resistance is directed against both the virus and its satellite RNA. Wild *Arachis* species are now being screened for rosette resistance by a southern African regional program of ICRISAT. Of seven species tested, two, *A.* sp. 30003 and *A.* sp. 30017, remained symptom-free throughout the season. Plant samples of these species were assayed and found free from both

the virus and the assistor virus. The apparent immunity of *A.* sp. 30003 is of great interest, particularly as this species is also highly resistant to early leafspot (19). Resistance to rosette in cultivated types is controlled by two independent recessive genes (33, 106). However, Misari et al (95) reported that it might not be simply inherited.

More than 7000 germplasm lines have been screened at ICRISAT for field resistance to tomato spotted wilt virus. Many lines have been identified that have shown consistently low disease incidence in the field. Some of these lines are C102, C121, C136, NC Ac 343, NC Ac 1741, NC Ac 2232, NC Ac 2242, NC Ac 17888, and Gujrat Narrow Leaf Mutant (70). Germplasm and breeding lines with resistance to thrips and low disease incidence in the field were also tested under laboratory conditions for resistance to tomato spotted wilt virus. Only two breeding lines, ICGV 86029 and ICGV 86031, showed tolerance to the virus (72). Forty-two wild species have been tested in the glasshouse by mechanical and thrips (*F. schultzei)* inoculation. Only *A. chacoense* remained free from virus infection in these tests, but became infected with the virus under graft inoculation (128). *Arachis chacoense* and three other species—*A. pusilla* (12922), *A. correntina* (9530), and *A. cardenasii* (10017)—when infected by mechanical and thrips inoculation, show no infection under field conditions.

Over 2500 germplasm lines belonging to the cultivated species *A. hypogaea* have been screened in the fields at ICRISAT for resistance to the peanut mottle virus (PMV). No line showed resistance to the virus. However, many germplasm lines had much lower yield loss than control cultivars. Two germplasm lines, NC Ac 2240 and NC Ac 2243, have shown insignificant yield loss due to disease over the years (70). A few breeding lines have also shown tolerance to the disease. Fifty wild *Arachis* species accessions have also been screened for virus resistance under glasshouse conditions using mechanical leaf rub and air brush inoculations. Of these, only two species, *A. chacoense* (10602) and *A. pusilla* (12911), remained free from infection even after repeated graft inoculations (128).

Seeds of PMV-infected plants of several germplasm lines were screened in the laboratory for virus presence using ELISA techniques. Two rust-resistant germplasm lines, EC 76446(292) and NC Ac 17133(RF), have failed to show any seed transmission in repeated tests over years on more than 13,000 seeds (72). Many breeding lines involving these rust-resistant parents in their parentage have also shown no seed transmission. An inheritance study on nonseed transmission is in progress at ICRISAT. Lines with low yield loss and no seed transmission have been crossed and advanced generation lines are in field tests for measuring yield loss due to the disease. Promising lines from these tests will be studied for nonseed transmission in the laboratory.

Over 7000 germplasm lines of the cultivated peanut *A. hypogaea* have been

screened by ICRISAT scientists for resistance to the peanut clump virus in farmers' fields in the Punjab and Andhra Pradesh, India. None of these lines showed resistance to the virus. A few lines showed tolerance to the disease, i.e. they did not suffer severely in growth and yield. Of 38 wild *Arachis* species and their 200 interspecific derivatives tested, only *Arachis* species 30036 did not become infected in the field.

PATHOGEN VARIABILITY

Breeding for disease resistance is generally considered more difficult than breeding for morphological or agronomic traits since efficacy of disease resistance is not static and is influenced simultaneously by host characteristics (magnitude of effectiveness, stability over environments, etc), and genetic variability of the pathogen (isolate aggressiveness, genetic selection for increased virulence, etc). The concept of "physiologic races," identified by pathogenic response on differential host genotypes, has assisted in understanding pathogenic variability in relation to breeding for major gene disease resistance. Major gene resistance has not been found, however, for most peanut pathogens. Effectiveness of minor gene resistance in peanut (oligogenic or polygenic; additive effects) is strongly influenced by exposure to inoculum density (16, 83) and conduciveness of physical environments that influence host-defensive responses and/or pathogen activity (17, 25, 119). In addition to pathogen aggressiveness, other "fitness" characteristics (geographic adaptation, inoculum survival, etc) are of concern to the breeder (13, 114, 123, 129). Variability of peanut pathogens generally is poorly understood and much additional research is needed to optimize breeding efforts. A brief summary of current knowledge of pathogenic variability for peanut pathogens follows.

Foliar Diseases

It is generally assumed that peanut rust (*P. arachidis)* has inherent capabilities for development of physiologic races when confronted by genotypes possessing major gene resistance. However, rust resistance presently used by peanut breeders appears to include factors for "slow rusting" (polygenic, minor genes) and no authenticated report of physiologic specialization is known (69, 129). Resistance to both early (CA) and late (CP) leafspot pathogens may also be based on additive genetic effects (9). Nevertheless, host specificity has been suggested for both CA and CP (47, 93). Inoculation techniques in testing, i.e. spreader rows, may mask levels of partial resistance, however. Indeed, putative resistance for both leafspot pathogens failed when genotypes were evaluated in diverse geographic sites (129, 131). Gibbons suggested that both host specificity factors and pathogen adaptation to local growing con-

ditions could occur (47). Although evidence for host specificity has not been conclusively demonstrated, pathogen adaptation to local environments has been reported (123). Additional information on differential sensitivity of partially resistant genotypes to temperature effects indicated that "location effects" on variability in severity or incidence of disease may be the result of alteration in host resistance metabolism rather than pathogen specificity (116). The possibility of environmental adaptation of local pathogen populations (penetration rate, survival of germinating spores, etc) should not, however, be discounted entirely (3, 13).

Although only minimal information is available on resistance to peanut virus diseases, extremely high variability obviously occurs in most plant and animal virus pathosystems. Peanut viruses are similar to many other viruses in this respect; several viruses of peanut are reported to have at least two to five "strains" based on symptomology and serology (35, 107, 109). Rapid development of virulent strains, specific to major gene resistance, is common in many crop species (43), and occurrence of new virulent strains of peanut viruses can be expected in the future.

Soil-Borne Pathogens

Peanut roots, pods, and stems are parasitized by a wide array of soil-borne fungi and nematodes. Resistance to soil-borne fungal pathogens is, to date, attributed to polygenic, additive gene effects. Physiologic specialization is generally not considered to develop under these circumstances (44, 141). However, increased virulence has been reported for *C. crotalariae* and other soil-borne pathogens in the presence of resistant genotypes (57). Effectiveness of partial resistance to soil-borne pathogens is influenced by inoculum density as well as conduciveness of environment (119). Aggressiveness characteristics of pathogen populations may, possibly, be of equal importance (31, 57, 122). Interactions between inoculum densities (ID) and pathogen aggressiveness characteristics often determine the effectiveness of resistance (16, 17, 25, 83). Knowledge of ID, range of aggressiveness for each major pathogen pest, as well as the potential for physiologic race development, are essential for long-term breeding efforts and control strategies (25, 30).

Peanut is host to several destructive ectoparasitic and endoparasitic nematode pests (109). At present no commercially available peanut cultivar in the US has any significant level of resistance to nematodes (102, 109). In other crops, however, development of resistant cultivars has quickly led to the recognition of new races or bio-types of the nematodes that place new cultivars in a pathologically vulnerable position (39). As Triantaphyllou (136) indicated, variability exists within each nematode species in both parasitism and aggressiveness, i.e. pathogenesis (136). Despite recurring problems in the development of hostspecific virulent races with most crops, breeding crop

plants for resistance to the most destructive pathogenic nematodes has been an effective and economical method of minimizing crop loss (136). Both hypersensitive necrosis and reproductive resistance to *Meloidogyne* sp. has recently been reported in complex hybrids (tetraploid) of *A. hypogaea,* which should be useful in peanut breeding while also preventing race development (66, 101).

Bacterial wilt is found wherever peanuts are grown but generally is considered to be a disease of minor importance except in certain areas of Asia and Africa (109). Strains of *P. solanacearum* have been differentiated by both host range and biochemical tests (65). Five biovars and three pathogenic races of the bacterium are recognized (80) with Race 1 having a comprehensive host range, including several plants in the Leguminosae (23, 80). However, strains of Race 1 can be cross-inoculated to tobacco and may differ in pathogenicity to both crops in some instances. Peanut represents the first crop in which resistance has been successfully employed against *P. solanacearium.* The high level of resistance in Schwarz 21 has held up for over 60 years, and the bacterium has not developed new virulent strains (21a, 23, 109). Two additional resistant cultivars have recently been released in China (21a). However, because of the incomplete (partial) nature of resistance, environmental conditions influence expression and effectiveness of resistance (21a, 32).

NATURE OF RESISTANCE

Although the biochemical nature of resistance to peanut pathogens has been postulated for several diseases, the physiological mechanisms for resistance are not well understood. It can be assumed, however, that metabolic resistance mechanisms in peanut are similar to those reported for other hosts (37, 38, 42, 49, 62). Current strategies in disease resistance breeding use polygenic, additive gene effects to provide varying levels of "partial," i.e. incomplete, resistance (49). Performance of partially resistant cultivars in the field is based both on physiological reduction in disease severity or incidence, and "escape" mechanisms (29, 36, 114, 117, 119). Plant anatomy, e.g. canopy density or branching habit, as well as root growth dynamics, may be important in avoidance or compensation for disease. With monocyclic root or stem pathogens, inhibition of initial infection and/or restriction of lesion development are considered primary epidemiological mechanisms of resistance (62, 63). With polycyclic leafspot pathogens, several components of "rate-reducing" resistance may function singly or in combination (26, 44, 77, 103).

Foliar Diseases

The principal phytoalexin of peanut leaves infected with any of four leafspot pathogens was identified as medicarpin (126). However, eleven other anti-

fungal compounds were also isolated from infected foliage (127). Although the role of phytoalexins in resistance to fungal pathogens is often debated, these antifungal compounds are assumed to inhibit pathogen ingress and/or reproduction in peanut tissues. Several anatomical and morphological characteristics of peanut tissue have also been associated with resistance to leafspot diseases. Resistance to both CA and CP was reported to be associated with formation of pectic substances and a thickening of cell walls (2). "Directed" growth of germ tubes toward stomata in susceptible cultivars has also been reported, whereas no directed growth was detected in resistant genotypes and less on moderately susceptible genotypes (2). Size of stomatal apertures has been correlated with resistance of field-grown *Arachis* sp.; however, Hassan et al and Cook did not find stomatal size to be a mechanism of resistance in later studies (30, 64). Ketring & Melouk (82) demonstrated that two peanut cultivars inoculated with *C. arachidicola* produced ethylene and had enhanced leaflet abscission, but an immune wild species produced only background levels of ethylene and retained its leaves (82). Recognition of epidemiological "components" of rate-reducing resistance in leafspot diseases of peanut has provided a major strategy for current breeding efforts (7, 26, 92). The "infection frequency" component of resistance has been used for both rust and leafspot diseases of peanut. Epidemics of peanut rust are apparently inhibited by a reduction in effectiveness of inoculum infecting resistant leaflets; thus, reproductive cycling of the pathogen is reduced even though only minor differences are detected in latent period (LP) or sporulation characteristics (7). Reduction in LP, lesion size, and duration of sporulation may also contribute, however, to inhibition of disease progress when infection occurs early in growing seasons.

A poor correlation is often reported between field performance of genotypes of *A. hypogaea* and number of CA and CP lesions developing in greenhouse tests when inoculated with a known range of inoculum densities (77, 103, 131). Resistance in other *Arachis* sp., however, does involve inefficiency of inoculum to induce lesions (40, 134). Reduction in size of lesions, and a corresponding reduction in number of conidia produced per lesion, is also a major resistance component for both CA and CP (7, 26, 77). Length of LP and percent of lesions that sporulate have both been reported to be important components of resistance in several studies (7, 92). Johnson et al concluded that the effects of several resistance components for CA were additive, and the higher magnitude of effectiveness of certain components could compensate for lesser effectiveness of others, resulting in a similar reduction in disease progress in field tests when interplot interference was minimal.

Biochemical and physiological mechanisms that determine host range and nonhost immunity to virus diseases are poorly understood (42), especially for peanut. However, most plants are nonhosts for most viruses and many viruses

are limited to a few species of plants as hosts. The limited host range of a particular virus might be the result of its specialized adaptation to several aspects of the biology of a plant that could be transferred by breeding or genetic transformation technology (14, 108). Field resistance to peanut virus diseases may result from characteristics of the plant that inhibit vector contact or virus transmission (42, 81, 112). Resistance to helper (assistor) virus, or vector specificity for either the primary or a helper virus, may also be possible mechanisms of field resistance to peanut viruses (10, 42, 43). Additional mechanisms of resistance to virus diseases include restricted virus replication or accumulation in host tissue, virus localizing mechanisms, and the hypersensitive reaction (10, 108). A direct-interference phenomenon, which inhibits virus infection, i.e. "cross protection," can be induced by closely related viruses (108). Virus coat-protein mediated resistance has been demonstrated for several plant pathogens but mechanism(s) providing protection are not totally understood (14). It has recently been demonstrated that transgenic plants that express coat-protein genes of one virus can interfere with disease development of other nonrelated viruses (6).

Soil-Borne Pathogens

The great diversity of pathogens attacking below-ground portions of roots, pods and pegs, makes it difficult to generalize about common mechanisms of resistance. Fungal pathogens—i.e. *Pythium* sp., *C. crotalariae, Sclerotium rolfsii, Fusarium* sp., *Aspergillus* sp., etc—each have unique characteristics for virulence. Similarly, peanut tissues have evolved physical, metabolic, and anatomical adaptations for escape, or resistance, to attack by these microbes. Resistance to parasitic nematodes is similar in some mechanisms of resistance to fungal pathogens but quite different in other aspects.

Constitutive anatomical traits in mature peanut shells have been associated with resistance to *P. myriotylum* and *Rhizoctonia solani* pod-rotting diseases in Texas (50). Induction of periderm formation in tap and fibrous roots has been associated with resistance to *C. crotalariae* (62, 63). In this disease, however, it was suggested that containment of the pathogen by periderm tissue occurred subsequent to partial inhibition of the fungus by unknown metabolic factors (62). As indicated for foliar pathogens of peanut, a number of antimicrobial compounds can be produced by roots and stems of *Arachis* sp. when challenged by microbe infection (87, 126, 127). Many workers have associated the seed coat resistance to *Aspergillus* sp. (*A. flavus*/*A. parasiticus*) with the presence of different chemicals—5,7-dimethoxyisoflavone (139), tannin (79, 87, 113), and total soluble amino compounds and arabinose (5). However, Jambunathan et al (73) did not find significant correlation between seed colonization and polyphenol content in the seed coat. Necrotrophic pathogens, i.e. *S. rolfsii* and *Sclerotinia minor,* use a combination of oxalic

acid and enzymatic degradation to parasitize limbs and stems. Mechanisms of field resistance for peanut and similar crops include escape due to canopy morphology, physical barriers to toxic compounds (waxy layers on stems, thick-walled cortical cells, cork cambium activity), and phytoalexin induction (1, 21, 29, 31). Physiological resistance to Sclerotinia blight of peanut appeared to be at least partially controlled by a cytoplasmic factor (29).

Extremely high levels of resistance or immunity to bacterial wilt have been reported for three peanut introductions (32, 109). Resistance in Schwarz 21 and two new Chinese cultivars is effective in field tests but can be overcome with high inoculum densities under conducive environmental conditions (21a, 32, 74). Although physiological mechanisms of bacterial wilt resistance in peanut have not been described, general mechanisms for resistance to bacterial wilt in other crops probably also function in peanut. It has been demonstrated in tobacco that postinfection host-responses limit multiplication (and subsequent distribution within host tissues) of virulent populations as well as incompatible races of *P. solanacearum* (23). Chemical alterations in host tissues during pathogenesis involved production or inhibition of several phytoalexins and related enzymatic pathways (23). Host respiration increased as water uptake and transport decreased. Mechanisms of resistance to plant parasitic nematodes of peanut are postulated to include both preinfection and postinfection phenomena (37, 94, 102, 143). Root leachates from healthy plants are reported to either enhance or suppress egg hatch, chemotaxis, and physiological behavior of some nematodes (67, 78). These mechanisms should be effective against both ectoparasitic and endoparasitic nematodes.

Several additional mechanisms of resistances are postulated to occur after penetration and feeding have been initiated by various nematode species (51, 67). A positive correlation between concentration of phenolics in plants and resistance to root-knot nematodes has been reported (120). Nutritional status of plants is thought to be important in resistance to nematodes in two ways: first, absence of certain nutrients may influence nematodes to move out of infected roots; and second, host nutrition may affect reproduction of ectoparasites or alter the sex ratio of root-knot nematodes within infected roots (67). Although production of phytoalexins has been investigated primarily in relation to fungal diseases, phytolexins also have been reported to be important in bacterial, viral, and nematode diseases (67, 78). Accumulation of phytoalexins in resistant plants has been found in root-knot infected soybean and cotton cultivars (67). Hypersensitive necrosis of peanut cells may localize invading pathogens and prevent further development of disease (101, 102). An incompatible host reaction to endoparasitic nematodes (hypersensitivity) is considered to be a defense mechanism by many pathologists (37). However, growth and development of juvenile nematodes in infected roots, as well as gall formation, can be inhibited without necrosis of host cells (101, 102).

Recognition by plant tissues of invading pathogens stimulates phytoalexin production, as well as hypersensitive necrosis. Breeders should be aware that dependence on hypersensitivity alone to provide resistance may result in the rapid development of new races of plant parasitic nematodes. Although it is desirable to provide high levels of protection in peanut to serious nematode pests, it may be more useful to incorporate levels of reproductive resistance in genotypes, whenever possible, to provide durable cultivar performance.

BREEDING STRATEGY

Although breeders are aware of the hazards of using sources of resistance with major genes, peanut breeders, like all breeders, use resistant sources that become available. However, only a few major genes for disease resistance have been identified in the peanut. Resistance to rosette virus, controlled by two independent recessive genes, is relatively easy to transfer to agronomically desirable types. Genotypes with resistance can be developed by backcrossing or by use of a breeding method that results in homozygous genotypes after hybridization. Some sources of rust resistance are also controlled by two recessive genes. These sources can be used in a manner similar to that for rosette resistance.

Most sources of resistance to soil-borne fungi in peanut show low levels of resistance or tolerance. Such partial resistance is presumably governed by polygenes and is assumed to be similar to horizontal resistance (44). There are practical difficulties in incorporating this type of resistance into germplasm with desired agronomic traits such as large fruit, high yield, a high oleic/linoleic ratio, and with superior organoleptic characteristics. Most resistant sources among cultivated genotypes originated from native landraces and generally have low yields and undesirable fruit and seed sizes, especially for the sophisticated market of the US. In the process of selecting plants for better agronomic traits in crosses involving these resistant sources, levels of resistance are often diluted. The persistent association of poor quality with southern stem rot and pythium pod rot resistance has concerned those breeding for resistance in peanut (122). A similar situation has been observed in breeding for CBR resistance. Resistant parents produce low yields and small irregular shaped fruit (149). Selection for larger fruit and higher yields reduces resistance (147). The highest yielding lines with large fruit (resulting from crosses of resistant and agronomically desirable types) are generally the least resistant of the progeny. The strategy has been to breed for a low level of host-pathogen coexistence that is stable, environmentally balanced, and economically useful; however, selections have been compromises between resistance and yield. Successful use of such cultivars requires excellent management

skills that simultaneously reduce disease severity and inoculum reproduction (18, 28, 119).

Resistance to late and early leafspots has most often been considered quantitative with a high heritability (9, 76); however, resistance to late leafspot has been reported to be governed by five loci (104). Regardless of inheritance, levels of resistance to the leafspots appear to be higher than for soil-borne fungi and are easier to manipulate genetically.

When multiple disease resistance is needed, it is difficult to accumulate enough polygenes to provide good levels of resistance to all diseases if the genes governing resistance are inherited independently. Attempts to incorporate polygenes for resistance to two diseases may result in the loss of resistance to one disease as selection occurs for the second disease. Exceptions to this will occur if the same genes confer resistance to more than one disease, as may be the case in peanuts. During the development of resistance to *Pythium* pod rot, variation in reaction to *S. rolfsii* was also found (122). Similar observations were made in screening for CBR resistance. Several genotypes resistant to CBR were also resistant to southern stem rot (115, 117).

There has also been interest in selecting simultaneously for both early and late leafspot resistance. Early leafspot is the primary leafspot in North Carolina, but evidence suggests that the development of cultivars resistant to CA may increase the incidence of CP. Two strategies are being used to attempt to combine resistance to both leafspots in a single genotype. One strategy is to select for CP resistance among germplasm already selected for CA resistance. NC 5 and GP-NC 343, originally identified as being resistant to CA, and progenies from crosses involving these parents that were first selected for CA, were also found to be resistant to CP (86, 146). The level of partial resistance to both pathogens was only moderate but may be sufficient to manage the disease if cultural methods to reduce inoculum density are used. A second approach is to combine individual sources of resistance to CA and CP into a single genotype. Genes for resistance to CA and CP are inherited independently and can be incorporated into a single genotype (8, 9, 86). Germplasm with resistance to both rust and CP is being used at ICRISAT in an attempt to develop high-yielding cultivars with resistance to both diseases (71). Since only one of the resistance is polygenic, this approach should be successful (71).

Principal component analysis, tree diagrams, and biplots have been used to assess the potential for selecting components of resistance to rust and both leafspots simultaneously (7). Selection of genotypes resistant to the three diseases can be made based on different levels of partial components of resistance, depending on the goals of the breeder.

PROGRESS IN BREEDING FOR DISEASE RESISTANCE

Considerable progress in breeding for disease-resistant peanuts has been made since the release of the bacterial wilt-resistant Schwarz 21 in 1927 and the release of NC 2, a cultivar with resistance to southern stem rot in 1953.

Since 1956, breeders have developed and released rosette-resistant cultivars RG1, KH 149A, KH 241D, 69-101, RMP 12, and RMP 91 in West or southern Africa (20, 118). Rosette resistance also has been successfully transferred by backcrossing to a released cultivar, 28-206(R) (88). The transfer of rosette resistance (which is governed by major genes) to newer cultivars, continues in several West African and southern African locations (100).

Although Va 81B (resistant to Sclerotinia blight) and NC 8C and NC 10C (resistant to CBR) have been released in recent years in the US, progress in breeding for resistance to the soil-borne fungi has been difficult and slow. All of these cultivars have partial resistance with field performance dependent on inoculum density. These cultivars also have compromised one or more agronomic traits that make the cultivars less competitive in absence of the disease. Considerable cooperative breeding and pathology research is needed if soil-borne pathogens of peanut are to be managed using the available sources of partial resistance.

Progress in breeding for resistance to early and late leafspots and rust has been accelerated in the past decade. Southern Runner, a high-yielding CP-resistant cultivar, was released for use in the US. Greater progress has been made at ICRISAT, where cultivar development can be targeted to a less sophisticated market. Breeding at ICRISAT has concentrated on developing high-yielding cultivars with resistance or tolerance to both rust and CP. From early generation material supplied by ICRISAT to cooperators in India, resistant cultivars such as Girnar-1, DOR 8-10, and ALR 1 have been developed. High-yielding lines, ICG(FDRS)4 and ICG(FDSR)10, resistant to rust and moderately resistant to CP, are being considered for release in India. Use of the diploid species, *A. cardenasii,* has resulted in several breeding lines with levels of CP resistance exceeding that found in cultivated peanuts. One line, 259-2, has excellent resistance to CP and has also shown resistance to CA (71).

Breeding for resistance to CA has not led to the release of a cultivar in the US to date; however, adequate levels of resistance were found among progenies from crosses of GP-NC 343 by NC 5. Inbred lines from this cross have been used as resistant parents and selection is now being practiced in several crosses. Selection also is being practiced in crosses with four other resistant lines—PI 109839, PI 270806, PI 269685, and Kanyoma.

Several laboratories have initiated breeding programs for aflatoxin resistance, tomato spotted wilt virus resistance, nematode resistance, and other

locally important diseases. Despite the intensified effort on screening for sources of resistance and the transfer of resistance genes to agronomically desirable genotypes, few cultivars with good agronomic traits and high levels of disease resistance have been developed. Perhaps the research effort has not been of suitable duration; however, the lack of qualitative sources of resistance for most diseases may partly be responsible for the slow progress in developing disease-resistant cultivars in peanut (4).

FUTURE EXPECTATIONS

Much progress in breeding for disease resistance in peanut can be expected during the next decade. Many sources of disease resistance among cultivated germplasm have been identified and are being used in breeding programs. Advanced breeding lines with disease resistance are currently being evaluated at numerous locations in the US and around the world; breeding for disease resistance has become a priority in most peanut breeding programs.

Considerable attention is also being devoted to using the wild species of *Arachis* for disease resistance. The high levels of resistance or immunity to early and late leafspots, rust, nematodes, peanut stunt virus, and tomato spotted wilt virus must be transferred to cultivated peanuts (125). Differences in ploidy level, and incompatibility of species outside section *Arachis* with the cultivated peanut, make it difficult to use the wild species. Stable 40-chromosome hybrid derivatives have been obtained for only a few interspecific hybrids. Because of the high levels of leafspot and rust resistance for these derivatives, the wild species will eventually contribute substantially to disease resistance in peanut.

Finally, several researchers are developing methodologies to use molecular techniques for improvement of the peanut. Perfection of a transformation and regeneration protocol for peanut, which is being evaluated at present, will allow researchers to incorporate genes from sources outside the genus (27). The first successes will probably involve cross-protection against virus diseases.

Literature Cited

1. Abawi, G. S., Provvidenti, R., Crosier, D. C., Hunter, J. E. 1978. Inheritance to white mold disease in *Phaseolus coccineus*. *J. Hered.* 69:200–2
2. Abdou, Y. A-M., Gregory, W. C., Cooper, W. E. 1974. Sources and nature of resistance to *Cercospora arachidicola* and *Cercosporidium personatum* in *Arachis* spp. *Peanut Sci.* 1:6–11
3. Alderman, S. C., Beute, M. K. 1987. Influence of temperature, lesion water potential, and cyclic wet-dry periods on sporulation of *Cercospora arachidicola* on peanut. *Phytopathology* 77:960–63
4. Allen, D. J. 1983. The pathology of tropical food legumes. In *Groundnut Diseases*, pp. 98–111. New York: Wiley
5. Amaya, F-J., Young, C. T., Norden, A. J., Mixon, A. C. 1980. Chemical screening for *Aspergillus flavus* resistance in peanut. *Oléagineux* 35:255–57
6. Anderson, E. J., Stark, D. M., Nelson, R. W., Powell, P. A., Tumer, N. E., Beachy, R. N. 1989. Transgenic plants

that express coat protein genes of tobacco mosaic virus or alfalfa mosaic virus interfere with disease development of some nonrelated viruses. *Phytopathology* 79:1284–90
7. Anderson, W. F., Beute, M. K., Wynne, J. C., Wongkaew, S. 1990. Statistical procedures for assessment of resistance in a multiple foliar disease complex of peanut. *Phytopathology* 80: 1451–59
8. Anderson, W. F., Wynne, J. C., Green, C. C. 1986. Potential for incorporation of early and late leafspot resistance in peanut. *Plant Breed.* 97:163–70
9. Anderson, W. F., Wynne, J. C., Green, C. C., Beute, M. K. 1986. Combining ability and heritability of resistance to early and late leafspot of peanut. *Peanut Sci.* 13:10–14
10. Ansa, O. A., Misari, S. M., Kuhn, C. W., Demski, J. W., Breyel, E., Casper, R. 1984. The current status of research on groundnut rosette virus disease in Nigeria. In *Proc. ICRISAT Reg. Groundnut Meet. W. Afr., 1st,* Patancheru, A. P. India: ICRISAT
11. Aujla, S. S., Chohan, J. S., Mehan, V. K. 1978. The screening of peanut varieties for the accumulation of aflatoxin and their relative reaction to the toxigenic isolate of *Aspergillus flavus* Link ex Fries. *J. Res. Punjab Agric. Univ.* 15: 400–3
12. Bartz, Z. A., Norden, A. J., LaPrade, J. C., Demuynk, T. J. 1978. Seed tolerance in peanut (*Arachis hypogaea* L.) to members of the *Aspergillus flavus* group of fungi. *Peanut Sci.* 5:53–56
13. Bashi, E., Rosen, J. 1974. Adaptation of four pathogens to semiarid habitats as conditioned by penetration rate and germinating spore survival. *Phytopathology* 64:1035–39
14. Beachy, R. N., Loesch-Fries, S., Turner, N. 1990. Coat protein-mediated resistance against virus infection. *Annu. Rev. Phytopathol.* 28:451–74
15. Beute, M. K., Wynne, J. C., Emery, D. A. 1976. Registration of NC 3033 peanut germplasm. *Crop Sci.* 16:887
16. Black, M. C., Beute, M. K. 1984. Relationship among inoculum density, microsclerotium size, and inoculum efficiency of *Cylindrocladium crotalariae* causing root rot on peanut. *Phytopathology* 74:1128–32
17. Black, M. C., Beute, M. K. 1985. Soil components that affect severity of Cylindrocladium black rot of peanut. *Plant Dis.* 69:36–39
18. Black, M. C., Pataky, J. K., Beute, M. K., Wynne, J. C. 1984. Management tactics that complement host resistance for control of Cylindrocladium blackrot of peanut. *Peanut Sci.* 11:70–73
19. Bock, K. R. 1989. ICRISAT Regional Groundnut Pathology Program: A review of research progress during 1985–87 with special reference to groundnut streak necrosis disease. In *Proc. Reg. Groundnut Workshop S. Afr., 13–18 March 1988, Lilongwe, Malawi, 3rd,* pp. 13–20. Patancheru, India: ICRISAT
20. Bockelee-Morvan, A. 1983. Les différentes variétés d'arachide: Répartition-géographique et climatique, disponibilité. *Oléagineux* 38:73–76. In French, summary in English
21. Boland, G. J., Hall, R. 1987. Evaluating soybean cultivars for resistance to *Sclerotinia sclerotiorum* under field conditions. *Plant Dis.* 71:934–36
21a. Boshou, L., Yuying, W., Xingming, X., Guiying, T., Yujun, T., Darong, S. 1990. Genetic and breeding aspects of resistance to bacterial wilt in groundnut. In *ACIAR Proc. No. 31,* pp. 39–47. Kingaroy, Aust.: Burnett Printing
22. Bromfield, K. R., Cevario, S. J. 1970. Greenhouse screening of peanut (*Arachis hypogaea*) for resistance to peanut rust (*Puccinia arachidis*). *Plant Dis. Rep.* 54:381–83
23. Buddenhagen, I. W., Kelman, A. 1964. Biological and physiological aspects of bacterial wilt caused by *Pseudomonas solanacearum. Annu. Rev. Phytopathol.* 2:203–30
24. Bunting, A. H., Gibbons, R. W., Wynne, J. C. 1985. Groundnut (*Arachis hypogaea* L.). In *Grain Legume Crops,* ed. R. J. Summerfield, E. H. Roberts, pp. 747–800. London: Collins
25. Carlin, D. E., Leiner, R. H. 1990. Effects of temperature on virulence of *Rhizoctonia solani* and other Rhizoctonia on potato. *Phytopathology* 80:930–34
26. Chiteka, Z. A., Gorbet, D. W., Knauft, D. A., Shokes, F. M., Kucharek, T. A. 1988. Components of resistance to late leafspot in peanut. II. Correlations among components and their significance in breeding for resistance. *Peanut Sci.* 15:76–81
27. Clemente, T. E., Weissinger, A. K., Moyer, J. M., Beute, M. K. 1990. Development of a transformation system for peanut. *Phytopathology* 80:1022
28. Cline, W. O., Beute, M. K. 1986. Effect of metam sodium, peanut genotype and inoculum density on incidence of Cylindrocladium black rot. *Peanut Sci.* 13:41–45
29. Coffelt, T. A., Porter, D. M. 1982.

Screening peanuts for resistance to Sclerotinia blight. *Plant Dis.* 66:385–87
30. Cook, M. 1981. Susceptibility of peanut leaves to *Cercosporidium personatum*. *Phytopathology* 71:787–91
31. Cooper, W. E. 1961. Strains of, resistance to, and antagonists of *Sclerotium-rolfsii*. *Phytopathology* 51:113–16
32. Darong, S., Chuenrung, C., Yuring, W. 1981. Resistance evaluation of bacterial wilt (*Pseudomonas solanacearum* E. F. Sm.) of peanut (*Arachis hypogaea* L.) in the Peoples' Republic of China. *Proc. Am. Peanut Res. Educ. Soc.* 13:21–28
33. de Berchoux, C. 1958. Etude sur la résistance de l'arachide en Haute Volta. Premiers résultats. *Oléagineux* 13:237–39
34. de Berchoux, C. 1960. La rosette de l'arachide en Haute Volta. Comportement des lignes résistantes. *Oléagineux* 15:237–39
35. Demski, J. W., Reddy, D. V. R., Wongkaew, S., Kaneya-Iwaki, M., Saleh, N., Xu, Z. Y. 1988. Naming of peanut stripe virus. *Phytopathology* 78:631–32
36. Dow, R. L., Powell, N. L., Porter, D. M. 1988. Effects of modification of the plant canopy environment on Sclerotinia blight of peanut. *Peanut Sci.* 15:1–5
37. Dropkin, V. H., Helgeson, J. P., Upper, C. D. 1969. The hypersensitivity reaction of tomatoes resistant to *Meloidogyne incognita:* Reversal by cytokinins. *J. Nematol.* 1:55–61
38. Ebel, J. 1986. Phytoalexin synthesis: The biochemical analysis of the induction process. *Annu. Rev. Phytopathol.* 24:235–64
39. Fassuliotis, G. 1987. Genetic basis of plant resistance to nematodes. In *Vistas on Nematology*, ed. J. A. Veech, D. W. Dickson, pp. 364–71. DeLeon Springs, FL: Painter Printing Co. 509 pp.
40. Foster, D. J., Stalker, H. T., Wynne, J. C., Beute, M. K. 1981. Resistance of *Arachis hypogaea* L. and wild relatives to *Cercospora arachidicola* Hori. *Oléagineux* 36:139–43
41. Foster, D. J., Wynne, J. C., Beute, M. K. 1980. Evaluation of detached leaf culture for screening peanuts for leafspot resistance. *Peanut Sci.* 7:98–100
42. Fraser, R. S. S. 1985. *Mechanisms of Resistance to Plant Diseases*. Boston, MA: Nijhoff/Junk. 462 pp.
43. Fraser, R. S. S. 1987. *Biochemistry of Virus-Infected Plants*. New York: Wiley. 259 pp.
44. Fry, W. E. 1982. *Principles of Plant Disease Management*. New York: Academic. 378 pp.
45. Garren, K. H., Wilson, C. 1951. Plant diseases. In *The Peanut, the Unpredictable Legume*, pp. 262–324. Washington, DC: Natl. Fertilizer Assoc.
46. Ghewande, M. P., Nagaraj, G., Reddy, P. S. 1989. Aflatoxin research at the Indian National Research Center for Groundnut. In *Aflatoxin Contamination of Groundnut: Proc. Int. Workshop, 6–9 Oct. 1987*, pp. 237–43. Patancheru, India: ICRISAT
47. Gibbons, R. W. 1966. Mycosphaerella leafspots of groundnuts. *FAO Plant Prot. Bull.* 15:25–30
48. Gibbons, R. W. 1980. The ICRISAT Groundnut Program. In *Proc. Int. Workshop Groundnuts, 13–17 Oct.*, pp. 12–16. Patancheru, India: ICRISAT
49. Giebel, J. 1982. Mechanism of resistance to plant nematodes. *Annu. Rev. Phytopathol.* 20:257–79
50. Godoy, R., Smith, O. D., Taber, R. A., Pettit, R. E. 1985. Anatomical traits associated with pod rot resistance in peanut. *Peanut Sci.* 12:77–82
51. Gommers, F. J., Bakker, J., Smits, L. 1980. Effects of singlet oxygen generated by the nematocidal compound α-terthienyl from Tagetes on the nematode *Aphelenchus avenae*. *Nematologica* 26: 369–75
52. Gorbet, D. W., Shokes, F. M., Jackson, L. J. 1982. Control of peanut leafspot with a combination of resistance and fungicide treatment. *Peanut Sci.* 9:87–90
53. Green, C. C., Beute, M. K., Wynne, J. C. 1983. A comparison of methods of evaluating resistance to *Cylindrocladium crotalariae* in peanut field tests. *Peanut Sci.* 10:66–69
54. Green, C. C., Wynne, J. C. 1987. Genetic variability and heritability for resistance to early leafspot in four crosses of Virginia-type peanut. *Crop Sci.* 27:18–21
55. Green, C. C., Wynne, J. C., Beute, M. K. 1983. Genetic variability and heritability estimates based on the F2 generation from crosses of large-seeded virginia-type peanuts with lines resistant to Cylindrocladium black rot. *Peanut Sci.* 10:47–51
56. Gregory, W. C., Gregory, M. P., Krapovickas, A., Smith, B. W., Yarbrough, J. A. 1974. Structure and genetic resources of peanuts. In *Peanuts—Culture and Uses*. Stillwater, OK: Am. Peanut Res. Educ. Assoc. Inc.
57. Hadley, B. A., Beute, M. K., Leonard, K. J. 1979. Variability of *Cylindrocladium crotalariae* response to resistant host plant selection pressure in peanut. *Phytopathology* 69:1112–14

58. Hadley, B. A., Beute, M. K., Wynne, J. C. 1979. Heritability of Cylindrocladium black rot resistance in peanut. *Peanut Sci.* 6:51–54
59. Hammons, R. O. 1977. Groundnut rust in the United States and the Caribbean. *PANS* 23:300–4
60. Hammons, R. O. 1982. Origin and early history of the peanut. In *Peanut Science and Technology*, ed. H. E. Pattee, C. T. Young, pp. 1–20. Yoakum, TX: Am. Peanut Res. Educ. Soc. Inc.
61. Harkness, C. 1977. The breeding and selection of groundnut varieties for resistance to rosette virus disease in Nigeria. *Inst. Agric. Res. Rep.* Ahmadu Bello Univ., Zaria, Nigeria
62. Harris, N. E., Beute, M. K. 1982. Histological responses of peanut germplasm resistant and susceptible to *Cylindrocladium crotalariae* in relationship to inoculum density. *Phytopathology* 72: 1250–56
63. Harris, N. E., Beute, M. K. 1982. *Cylindrocladium crotalariae*-induced periderm formation in tap root and fibrous roots of *Arachis hypogaea*. *Peanut Sci.* 9:82–86
64. Hassan, H. N., Beute, M. K. 1977. Evaluation of resistance to cercospora leaf spot in peanut germplasm potentially useful in a breeding program. *Peanut Sci.* 4:78–83
65. Hayward, A. C. 1991. Biology and epidemiology of bacterial wilt caused by *Pseudomonas solanacearum*. *Annu. Rev. Phytopathol.* 29:65–87
66. Holbrook, C. C., Noe, J. P. 1990. Resistance to *Meloidogyne arenaria* in *Arachis* spp. and its implication on development of resistant peanut cultivars. *Peanut Sci.* 17:35–38
67. Huang, J. C. 1985. Mechanisms of resistance to root-knot nematodes. In *An Advanced Treatise on Meloidogyne*, Vol. 1, ed. J. N. Sasser, C. C. Carter. Raleigh, NC: NC State Univ. Graphics. 422 pp.
68. International Board for Plant Genetic Resources (IBPGR). 1990. International crop network series. 2. Report of a workshop on the genetic resources of wild Arachis species including preliminary descriptors for Arachis(IBPGR/ICRISAT). Rome
69. International Crops Research Institute for the Semi-Arid Tropics. (ICRISAT) 1983. Rust disease of groundnut. *Info. Bull. No. 13*, Patancheru, India.15 pp.
70. International Crops Research Institute for the Semi-Arid Tropics. (ICRISAT) 1984.*Annual report 1983*. Patancheru, India: ICRISAT. 186 pp.
71. International Crops Research Institute for the Semi-Arid Tropics. (ICRISAT) 1986. *Annual report 1985*. Patancheru, India: ICRISAT. 250 pp.
72. International Crops Research Institute for the Semi-Arid Tropics. (ICRISAT) 1989. *Annual report 1988. pp. 119, 140*. Patancheru, India: ICRISAT
73. Jambunathan, R., Mehan, V. K., Gurtu, S. 1989. Aflatoxin contamination of groundnut. See Ref. 46, pp. 357–64
74. Jenkins, S. F., Hammons, R. O., Dukes, P. D. 1966. Disease reaction and symptom expression of seventeen peanut cultivars to bacterial wilt. *Plant Dis. Report.* 50:520–23
75. Jogloy, S. 1988. *Estimates of gene action on leafspot resistance and agronomic traits in peanut(* Arachis hypogaea *L.*). PhD thesis. NC State Univ., Raleigh. 121 pp.
76. Jogloy, S., Wynne, J. C., Beute, M. K. 1987. Inheritance of late leafspot resistance and agronomic traits in peanut. *Peanut Sci.* 14:86–90
77. Johnson, C. S., Beute, M. K., Ricker, M. D. 1986. Relationship between components of resistance and disease progress of early leaf spot on Virginia-type peanut. *Phytopathology* 76:495–99
78. Kaplan, D. T., Davis, E. L. 1987. Mechanisms of plant incompatibility with nematodes. See Ref. 39, pp. 267–76
79. Karchesy, J. J., Hemingway, R. W. 1986. Condensed tannins (4B– > 8; 2B– > O– > 7)-linked procyanidins in *Arachis hypogaea* L. *J. Agric. Food Chem.* 34:966–70
80. Kelman, A., Person, L. H. 1961. Strains of *Pseudomonas solanacearum* differing in pathogenicity to tobacco and peanut. *Phytopathology* 51:158–61
81. Kennedy, G. G. 1976. Host plant resistance and the spread of plant viruses. *Environ. Entomol.* 5:827–32
82. Ketring, D., Melouk, H. 1982. Ethylene production in leaflet abscission of three peanut genotypes infected with *Cercospora arachidicola*. *Plant Physiol.* 69: 789–92
83. Kinsbursky, R. S., Weinhold, A. R. 1988. Influence of soil inoculum density disease incidence relationships of *Rhizoctonia solani*. *Phytopathology* 78: 127–30
84. Kisyombe, C. T., Beute, M. K., Payne, G. A. 1985. Field evaluation of peanut genotypes for resistance to infection by *Aspergillus parasiticus*. *Peanut Sci.* 12:12–17
85. Knauft, D. A. 1987. Inheritance of rust resistance in groundnut. In *Groundnut*

Rust Disease: Proc. Discuss. Group Meet., 24–28 Sept. 1984, pp. 183–87. Patancheru, India: ICRISAT
86. Kornegay, J. L., Beute, M. K., Wynne, J. C. 1980. Inheritance of resistance to *Cercospora arachidicola* and *Cercosporidium personatum* in six virginia-type peanut lines. *Peanut Sci.* 7:4–9
87. Lansden, J. A. 1982. Aflatoxin inhibition and fungistatis by peanut tannins. *Peanut Sci.* 9:17–20
88. Mauboussin, J. C., Laurent, P., Delafond, G. 1970. Les variétés d'arachides recommandées au Sénégal et leur emploi. *Cah. Agric. Prat. Pays Chauds* 2:63–89
89. McDonald, D., Raheja, A. K. 1980. Pests, diseases, resistance, and crop protection in groundnuts. In *Advances in Legume Science*, ed. R. J. Summerfield, A. H. Bunting, pp. 501–14. London: HMSO
90. Mehan, V. K., McDonald, D., Ramakrishna, N. 1986. Varietal resistance in peanut to aflatoxin production. *Peanut Sci.* 13:7–10
91. Mehan, V. K., McDonald, D., Ramakrishna, N., Williams, J. H. 1986. Effects of genotype and date of harvest on infection of peanut seed by *Aspergillus flavus* and subsequent contamination with aflatoxin. *Peanut Sci.* 13:46–50
92. Melouk, H. A., Banks, D. J., Fanous, M. A. 1984. Assessment of resistance to *Cercospora arachidicola* in peanut genotypes in field plots. *Plant Dis.* 68:395–97
93. Miller, L. I. 1953. *Studies of the parasitism of* Cercospora arachidicola *Hori* and *Cercosporidium personatum* (Berk. & Curt.) Ell. and Ev. PhD thesis. Univ. Minn., St. Paul. 120 pp.
94. Minton, N. A., Hammons, R. O. 1975. Evaluation of peanut for resistance to the peanut root-knot nematode, *Meloidogyne arenaria*. *Plant Dis. Report.* 59:944–45
95. Misari, S. M., Ansa, O. A., Demski, J. W., Kuhn, C. W., Casper, O. F. R., Breyel, E. 1988. Groundnut rosette: Epidemiology and management in Nigeria. In *Coordinated Research on Groundnut Rosette Virus Disease: Summary Proc. Consult. Group Meet. Discuss. Collab. Res. Groundnut Rosette Virus Disease*, 8–10 March, Lilongwe, Malawi, pp. 15–16. Patancheru, India: ICRISAT
96. Mixon, A. C. 1979. Developing groundnut lines with resistance to seed colonization by toxin producing strains of *Aspergillus* species. *PANS* 25:394–400
97. Mixon, A. C., Rogers, K. M. 1973. Peanut accessions resistant to seed infection by *Aspergillus flavus*. *Agron. J.* 65:560–62
98. Murant, A. F., Rajeshwari, R., Robinson, D. J., Raschke, J. H. 1988. A satellite RNA of groundnut rosette virus that is largely responsible for symptoms of groundnut rosette disease. *J. Gen. Virol.* 69:1479–86
99. Nagrajan, V., Bhat, R. V. 1973. Aflatoxin production in peanut varieties by *Aspergillus flavus* Link and *Aspergillus parasiticus* Speare. *Appl. Microbiol.* 25:319–21
100. Ndunguru, B. J., Greenberg, D. C., Subrahmanyam, P. 1989. Groundnut improvement program at the ICRISAT Sahelian Center: Research problems, priorities, and strategies. See Ref. 19, pp. 105–13
101. Nelson, S. C., Simpson, C. E., Starr, J. L. 1989. Resistance to *Meloidogyne arenaria* in *Arachis* sp. germplasm. *J. Nematol.* 21:654–60
102. Nelson, S. C., Starr, J. L., Simpson, C. E. 1990. Expression of resistance to *Meloidogyne arenaria* in *Arachis batizocoi* and *A. cardenasii*. *J. Nematol.* 22: 423–25
103. Nevill, D. J. 1981. Components of resistance to *Cercospora arachidicola* and *Cercosporidium personatum* in groundnuts. *Annu. Appl. Biol.* 99:77–86
104. Nevill, D. J. 1982. Inheritance of resistance to Cercosporidium personatum in groundnuts: A genetic model and its implications for selection. *Oléagineux* 37:355–62
105. Nigam, S. N. 1987. A review of the present status of the genetic resources of the ICRISAT Regional Groundnut Improvement Program, of the Southern African Cooperative Regional Yield Trials, and of rosette virus resistance breeding. In *Proc. Reg. Groundnut Workshop S. Afr., 2nd, 10–14 Feb. 1986*, Harare, Zimbabwe, pp. 15–30. Patancheru, A. P., India: ICRISAT
106. Nigam, S. N., Bock, K. R. 1990. Inheritance of resistance to groundnut rosette virus in groundnut (*Arachis hypogaea*). *Ann. Appl. Biol.* 117:553–60
107. Paguio, O. R., Kuhn, C. 1974. Survey for peanut mottle virus in peanut in Georgia. *Plant Dis. Report.* 58:107–10
108. Ponz, F., Bruening, G. 1986. Mechanisms of resistance to plant viruses. *Annu. Rev. Phytopathol.* 24:355–81
109. Porter, D. M., Smith, D. H., Rodriguez-Kabana, R. 1982. Peanut diseases. In *Peanut Science and Technology*, ed. H. E. Pattee, C. T. Young, pp. 326–

410. Yoakum, TX: Am. Peanut Res. Educ. Soc.
110. Pua, A. R., Medalla, E. C. 1986. *Screening for resistance to* Aspergillus flavus *invasion in peanut.* Presented at *Anniv. Ann. Conv. Pest Control Counc. Philippines, 17th, 8–10 May,* Iloila City, Philippines
111. Rao, K. S., Tulpule, P. G. 1967. Varietal differences of groundnut in the production of aflatoxin. *Nature* 214:738–39
112. Rossell, H. W. 1977. Preliminary investigations on the identity and ecology of legume virus diseases in northern Nigeria. *Trop. Grain Legume Bull.* 8: 41–46
113. Sanders, T. H., Mixon, A. C. 1978. Effect of peanut tannins on percent seed colonization and in vitro growth by *Aspergillus parasiticus. Mycopathologia* 66:169–73
114. Shew, B. B., Beute, M. K. 1984. Effects of crop management on the epidemiology of southern stem rot of peanut. *Phytopathology* 74:530–35
115. Shew, B. B., Beute, M. K., Bailey, J. E. 1985. Potential for improved control of southern stem rot of peanut with resistance and fungicides. *Peanut Sci.* 12:4–7
116. Shew, B. B., Beute, M. K., Wynne, J. C. 1988. Effects of temperature and relative humidity on expression of resistance to *Cercosporidium personatum* in peanut. *Phytopathology* 78:493–98
117. Shew, B. B., Wynne, J. C., Beute, M. K. 1987. Field, microplot, and greenhouse evaluations of resistance to *Sclerotium rolfsii* in peanut. *Plant Dis.* 71: 188–91
118. Sibale, P. K., Kisyombe, C. T. 1980. Groundnut production, utilization, research problems, and further research needs in Malawi. See Ref. 89, pp. 249–53
119. Sidebottom, J. R., Beute, M. K. 1989. Control of Cylindrocladium black rot of peanut with cultural practices that modify soil temperature. *Plant Dis.* 73:672–76
120. Singh, B., Choudury, B. 1973. The chemical characteristics of tomato cultivars resistant to root-knot nematodes (*Meloidogyne* spp.). *Nematologica* 19: 443–48
121. Singh, A. K., Subrahmanyam, P., Moss, J. P. 1984. The dominant nature of resistance to *Puccinia arachidis* in certain wild *Arachis* species. *Oléagineux* 39:535–38
122. Smith, O. D., Boswell, T. E., Gricher, W. J., Simpson, C. E. 1989. Reaction of select peanut (*Arachis hypogaea* L.) lines to southern stem rot and Pythium pod rot under varied disease pressure. *Peanut Sci.* 16:9–13
123. Sommartya, T., Beute, M. K. 1986. Temperature effects on germination and comparative morphology of conidia for Thai and USA isolates of *Cercosporidium personatum. Peanut Sci.* 13:67–70
124. Sowell, G., Smith, D. H., Hammons, R. O. 1976. Resistance of peanut plant introductions to *Cercospora arachidicola. Plant Dis. Report.* 60:494–98
125. Stalker, H. T., Moss, J. P. 1987. Speciation, cytogenetics, and utilization of *Arachis* species. *Adv. Agron.* 41:1–40
126. Strange, R. N., Edwards, C., Richards, S. 1988. Phytoalexins of groundnuts and their role in resistance to fungal parasites. *Phytoparasitica* 16:202–3
127. Subba Rao, P. V., Geiger, J. P., Einhorn, J., Rao, B., Malosse, C., et al. 1988. Host defense mechanisms against groundnut rust. *Int. Arachis Newsl.* 4:16–18
128. Subrahmanyam, P., Ghanekar, A. M., Nolt, B. L., Reddy, D. V. R., McDonald, D. 1985. Resistance to groundnut diseases in wild Arachis species. In *Proc. Int. Workshop Cytogenet. Arachis,* 31 Oct.–Nov. 1983, pp. 49–55. Patancheru, India: ICRISAT
129. Subrahmanyam, P., Hammons, R. O., Nigam, S. N., McDonald, D., Gibbons, R. W., et al. 1983. International cooperative screening for resistance of peanut to rust and late leaf spot. *Plant Dis.* 67:1108–11
130. Subrahmanyam, P., McDonald, D. 1983. Rust disease of groundnut. International Crops Research Institute for the Semi-Arid Tropics (ICRISAT) *Info. Bull.* No. 13. Patancheru, India
131. Subrahmanyam, P., McDonald, D., Gibbons, R. W., Nigam, S. N., Nevill, D. J. 1982. Resistance to rust and late leafspot diseases in some genotypes of *Arachis hypogaea. Peanut Sci.* 9:6–10
132. Subrahmanyam, P., McDonald, D., Gibbons, R. W., Subba Rao, P. V. 1983. Components of resistance to *Puccinia arachidis* in peanuts. *Phytopathology* 73:253–56
133. Subrahmanyam, P., McDonald, D., Subba Rao, P. V. 1983. Influence of host genotype on uredospore production and germinability in *Puccinia arachidis. Phytopathology* 73:726–29
134. Subrahmanyam, P., Moss, J. P., McDonald, D., Subba Rao, P. V., Rao, V. R. 1985. Resistance to leafspot caused by *Cercosporidium personatum* in wild *Arachis* species. *Plant Dis.* 69:951–54

135. Subrahmanyam, P., Moss, J. P., Rao, V. R. 1983. Resistance to peanut rust in wild *Arachis* species. *Plant Dis.* 67: 209–12
136. Triantaphyllou, A. C. 1987. Genetics of nematode parasitism on plants. See Ref. 39, pp. 354–63
137. Tsai, A. H., Yeh, C. C. 1985. Studies on aflatoxin contamination and screening for disease resistance in groundnuts. *J. Agric. Res. China* 34:79–86
138. Tulpule, P. G., Bhat, R. V., Nagraj, V. 1977. Variations in aflatoxin in production due to fungal isolates and crop genotypes and their scope in prevention of aflatoxin production. *Arch. Inst. Pasteur Tunis* 54:487–93
139. Turner, R. B., Lindsey, D. L., Davis, D. D., Bishop, R. D. 1975. Isolation and identification of 5,7-dimethoxyisoflavone, an inhibitor of *Aspergillus flavus* from peanut. *Mycopathologia* 57:39–40
140. Utomo, S. D. 1990. *Inheritance of resistance to* Aspergillus parasiticus *Speare in peanut* (Arachis hypogaea *L.*). MS thesis, NC State Univ., Raleigh. 56 pp.
141. Van der Plank, J. E. 1968. *Disease Resistance in Plants*. New York: Academic
142. Vasudeva Rao, M. J., Nigam, S. N., Mehan, V. K., McDonald, D. 1989. *Aspergillus flavus* resistance breeding in groundnut: Progress made at ICRISAT Center. See Ref. 46, pp. 345–55
143. Veech, J. A. 1982. Phytoalexins and their role in resistance of plants to nematodes. *J. Nematol.* 14:2–9
144. Waliyar, F., Bockelee-Morvan, A. 1989. Resistance of groundnut varieties to *Aspergillus flavus* in Senegal. See Ref. 46, pp. 305–10
145. Waliyar, F., McDonald, D., Nigam, S. N., Subba Rao, P. V. 1989. Resistance to early leafspot of groundnut. See Ref. 19, pp. 49–54
146. Walls, S. B., Wynne, J. C., Beute, M. K. 1985. Resistance to late leafspot peanut of progenies selected for resistance to early leafspot. *Peanut Sci.* 12:17–22
147. Wynne, J. C., Beute, M. K., Bailey, J. E., Mozingo, R. W. 1990. Registration of 'NC 10C' peanut. *Crop Sci.* 30:In press
148. Wynne, J. C., Halward, T. 1989. Cytogenetics and genetics of *Arachis*. In *Critical Reviews in Plant Science*, ed. B. V. Conger, pp. 189–220. Boca Raton, FL: CRC Press
149. Wynne, J. C., Rowe, R. C., Beute, M. K. 1975. Resistance of peanut genotypes to *Cylindrocladium crotalariae*. *Peanut Sci.* 2:54–56
150. Zambettakis, C., Waliyar, F., Bockelee-Morvan, A., de Pins, O. 1981. Results of four years of research on resistance of groundnut varieties to *Aspergillus flavus*. *Oléagineux* 36:377–85