Chapter 10

PEANUT PLANT DISEASES

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Diseases of peanut (*Arachis hypogaea* L.) occur throughout the growing season and into the postharvest period. All parts of the peanut plant are subject to attack, and many diseases reduce the quantity or quality of pods, seed, and forage. Although crop losses attributable to some diseases are negligible, other diseases can be devastating. Certain diseases, such as Cercospora and Cercosporidium leafspots, are cosmopolitan, whereas other diseases, such as Sclerotinia blight and blackhull, have a restricted distribution. A disease may become epidemic in a field and be absent in an adjacent field. Also, in a given locality, a disease may be very destructive during a growing season and yet be difficult to find the next year in the same field.

During the early years of its cultivation the peanut was regarded as relatively free from disease (Garren and Wilson, 1951). However, peanut rust was reported in 1884 by Spegazzini (1884). In 1914 Cercospora leafspot was reported in Alabama (Wolf, 1914) and a bacterial wilt disease was reported in North Carolina (Fulton and Winston, 1914). Stem rot was reported in 1917 (McClintock, 1917). In 1922, a Sclerotinia wilt of peanut was reported in Argentina (Marchionatto, 1922). These diseases still contribute to significant crop losses in some areas.

Because of the increasing importance of the peanut as a food, feed, and oil crop, more attention has recently been given to improved crop management practices, especially in the areas of pest control (control of diseases, weeds, and insects), tillage, crop rotation, irrigation, and new cultivars. Some of these new crop-management practices have suppressed some diseases but increased others. Sclerotinia blight, a disease listed as minor in 1973 (Garren and Jackson, 1973), has recently become a major disease in Virginia, North Carolina, and Oklahoma. A shift from Cercospora leafspot to Cercosporidium leafspot has been reported in the southeastern United States (Smith and Littrell, 1980).

Diseases are usually classified as infectious or noninfectious. Infectious diseases of the peanut plant are caused by fungi, bacteria, viruses, nematodes, and a mycoplasma; only infectious diseases will be discussed in this chapter. The chapter is divided into 3 sections: foliar diseases, soilborne diseases, and nematode diseases. Mycotoxins are covered in Chapter 13.

FOLIAR DISEASES

Cercospora and Cercosporidium Leafspots

Early and late leafspots, caused by *Cercospora arachidicola* Hori and *Cercosporidium personatum* (Berk. & Curt.) Deighton, respectively, are widely distributed

throughout the world and are usually destructive if fungicides are not applied. Fungicides are routinely used for management of early and late leafspots in the United States. Because of the economic impact of these diseases, much attention has been given to developing chemical and nonchemical disease management strategies. Leafspot-resistant cultivars are urgently needed because of the rising cost of fungicides. A major international effort to develop leafspot resistant cultivars is under way.

The early and late leafspot pathogens are not restricted to the genus *Arachis*. Mercer (1977c) found that *Voandzeia subterranea* (groundbean) was a host of *C. arachidicola*. Pyzner (1980) observed natural infection of *Stylosanthes biflora* by *C. arachidicola* and reported infection and sporulation of *C. arachidicola* on detached leaves of various leguminous and nonleguminous plants.

Symptoms. Symptoms of early and late leafspot have been described by several authors (Woodroof, 1933; Jenkins, 1938; Jackson and Bell, 1969; Feakin, 1973; Garren and Jackson, 1973). Symptoms of early and late leafspots are small necrotic flecks that enlarge and become light-brown to black subcircular spots ranging from 1-10 mm or more in diameter (Figure 1A). Lesion frequency varies from 1 to many on peanut leaflets.

Early leafspot lesions usually consist of a light to dark-brown center surrounded by a conspicuous yellow halo. Late leafspot lesions are similar, but the halo is usually either less prominent or absent. The halo, however, is not a reliable characteristic for distinguishing between early and late leafspots, because it may be altered by genetics of the host, nutritional status of the host, or weather conditions. The early leafspot fungus sporulates primarily on the adaxial surface, and the late leafspot fungus usually sporulates on the abaxial surface of the leaflet. Conidial tufts of *C. personatum* are macroscopically visible as raised circles on the abaxial surface (Figure 1B). Early leafspot lesions with spores have a sooty appearance. Early and late leafspot lesions are usually brown and black, respectively. Positive identification of these fungi is made on the basis of conidial size, shape, and number of septa. Macroscopic diagnosis is difficult in some instances, because some pesticides produce phytotoxicity symptoms that resemble leafspot symptoms. *Cercospora arachidicola* and *C. personatum* also infect petioles, stems, stipules, and pegs.

Causal Organisms - *Cercospora arachidicola* Hori and *Cercosporidium personatum* (Berk. & Curt.) Deighton. The early and late leafspot pathogens are seen primarily in their imperfect states, i.e., as *C. arachidicola* and *C. personatum*. Perfect states of these fungi are *Mycosphaerella arachidis* Deighton and *M. berkeleyii* W. A. Jenkins (early and late leafspot pathogens, respectively). *Cercospora canescens* Ellis & Martin has been reported in Nigeria (McDonald, personal communication to Jackson, 1966), and Malawi (Mercer, 1977c). However, *C. canescens* is currently regarded as a minor pathogen of peanut foliage.

Jenkins (1938) described the imperfect state of the early leafspot fungus as follows: conidiophores mostly epiphyllous, sometimes amphigenous when old, arising from a stroma, fasciculate, geniculate, yellowish brown, continuous to 1 to several septate, 20-45 x 3-6 μ; conidia colorless to slightly olivaceous, obclavate, often curved, 35-108 x 2-5.4 μ, 4 to 12 septate, length and septation influenced by dry weather. Jenkins (1938) described the conidial state of the late leafspot fungus as follows: conidiophores mostly hypophyllous, sometimes amphigenous when old, arising in more or less distinctly concentric

tufts from heavy stromatic bases, fasciculate, geniculate, reddish-brown, with mostly hyaline tips, continuous or 1 to several septate, 24-54 x 2 8.2μ; conidia obclavate but more generally cylindrical with somewhat attenuated tips, pale brown to dilutely olivaceous, 18-40 x 5-11μ, 1 to 8 septate, length and septations influenced by dry weather. The ascigerous and spermagonial states were also described.

Disease Cycle and Epidemiology. Current evidence indicates since conidia of *C. arachidicola* and *C. personatum* are dispersed for only short distances; most of the initial inoculum is probably produced locally. Epidemics of early and late leafspot usually begin earlier and progress more rapidly in fields where peanuts follow peanuts. Conidia produced on peanut crop residue in the soil serve as the principal source of initial inoculum. Ascospores (Jenkins, 1938; Frezzi, 1960), chlamydospores (Miller, 1953) and mycelial fragments (Hemingway, 1957; Shanta, 1960) are also potential sources of initial inoculum.

Conidia of *C. arachidicola* germinate, forming 1 to several germ tubes which grow on the leaf surface and penetrate open stomata (Jenkins, 1938). Direct penetration of epidermal cells was also reported. *Cercospora arachidicola* kills cells in advance of its proliferating intercellular hyphae; *C. personatum* does not kill cells in advance of its intercellular hyphae but produces intercellular botryose haustoria (Woodroof, 1933; Jenkins, 1938). Chevaugeon (1952a) found that temperatures of 26 to 31 C with slight diurnal variations and long periods of high relative humidity were favorable for infection.

The aerobiology of *C. arachidicola* has been studied in India (Sreeramulu, 1970), the United States (Smith and Crosby, 1973), and Malawi (Mercer, 1977a). In all 3 studies a diurnal periodicity was observed, with peak catches of conidia at dew dry-off in the morning. Smith and Crosby (1973) also reported that numbers of conidia increased with the onset of rainfall. Sreeramulu (1970) found a forenoon pattern of dispersal for conidia of *C. personatum*. Smith and Crosby (1973) obtained evidence for the vertical dissemination of *C. arachidicola* conidia to heights of 2.7 m above the soil surface in a peanut field. Conidia can also be dispersed by insects and farm implements (Wolf, 1916; Higgins, 1956).

During a 5-year period in Georgia, Woodroof (1933) reported that early leafspot occurred annually and late leafspot was destructive in 2 of 5 years. In Virginia, late leafspot epidemics occurred about once every 4 years, and early leafspot epidemics occurred annually (Miller, 1953). Hemingway (1955) reported that late leafspot increased faster than early leafspot. Smith and Littrell (1980) reported that *C. arachidicola* was the predominant foliar pathogen in the southeastern United States from 1967 to 1976, and then there was a shift to *C. personatum* from 1976 to 1979.

Growth and sporulation of *C. arachidicola* have been studied. Landers (1964) developed a medium for growth and sporulation of *C. arachidicola*. The optimum pH for growth was 4.5. Miller (1953) found that the optimum temperature range for 3 races of *C. arachidicola* was 25 to 32 C and that the optimum range for *C. personatum* was 25 to 30 C. Das (1951) reported cardinal temperatures of 23, 27, and 32 C for growth of *C. personatum*. Abdou (1966) reported that light was required for sporulation of *C. personatum* but not for sporulation of *C. arachidicola*. Smith (1971) produced abundant quantities of *C. arachidicola* conidia on peanut oatmeal agar in cultures incubated at 28 C under continuous fluorescent light. Starkey (1980) reported that conidial production was greater on peanut hull extract agar than on peanut oatmeal agar.

Venkataramani (1967) isolated cercosporin, a biologically active red phytotoxin, from *C. personatum*. Alabi and Naqvi (1977a) reported that *C. arachidicola* produced cellulolytic and pectolytic enzymes. Alabi and Naqvi (1977b) also reported that infection of groundnut leaves by *C. arachidicola* altered the starch, sugar, and amino acid content of leaf tissue. Aulakh and Sandhu (1970a) reported that infection by *C. arachidicola* resulted in qualitative and quantitative changes in amino acids.

Shanta (1960) reported that application of P and N increased the incidence of late leafspot and application of K decreased it slightly. Bledsoe et al. (1949) reported that magnesium deficient peanut plants were more susceptible to early leafspot than were plants grown in a complete nutrient solution.

Boote et al. (1980) determined canopy photosynthesis rate and other characteristics of peanut foliage layers in response to leafspot, defoliation, and combinations of leafspot and defoliation. They reported that severe leafspot damage reduced the leaf area index by 80%, the $^{14}CO_2$ uptake by 85%, and the canopy carbon-exchange rate by 93%. Photosynthesis of diseased canopies was reduced not only by defoliation but also by the inefficient fixation of CO_2 by diseased attached leaves.

Control. Early and late leafspots can be partially controlled with crop management practices that reduce the initial inoculum (Smith and Littrell, 1980). Crop rotation delays the onset and seasonal progress of Cercospora and Cercosporidium leafspots (Mazzani and Allievi, 1971; Kucharek, 1975). Burial of crop residue reduces the initial inoculum and is especially useful when crop rotation is not a part of the management program. In some areas early planting can be used to either avoid or retard epidemics of early and late leafspot. Chevaugeon (1952b) found an inverse relationship between in-row spacing of plants and intensity of late leafspot, and Farrell et al. (1967) reported the same relationship with early and late leafspot.

Smith and Littrell (1980) indicated that methyl 1-(butylcarbamoyl)-2-benzimidazolecarbamate, cis-N-[(1,1,2,2-tetrachloroethyl)thio]-4-cyclohexene-1,2-dicarboximide, tetrachloroisophthalonitrile, copper ammonium carbonate, copper hydroxide, triphenyltin hydroxide, zinc ion and manganese ethylenebisdithiocarbamate 80%, a coordination product of manganese 16%, zinc 2%, ethylenebisdithiocarbamate 62%, manganese ethylenebisdithiocarbamate, and elemental sulfur have been registered for control of peanut leafspots in the United States. Benomyl tolerant strains of *C. arachidicola* and *C. personatum* have been reported (Clark et al., 1974; Littrell, 1974a). Fungicides are applied with tractor-propelled sprayers, fixed-wing aircraft, helicopters, and sprinkler irrigation systems. Application schedules are usually begun within 30 to 50 days after planting, with subsequent applications at 10-14-day intervals until 2 or 3 weeks before the anticipated harvest date. Some nontarget effects of foliar fungicides have been reported. Backman et al. (1975) observed a consistently higher level of southern blight, caused by *Sclerotium rolfsii*, in plots where Florunner plants were sprayed with benomyl than in unsprayed plots. Porter (1980a) found that Sclerotinia blight, caused by *Sclerotinia minor*, was more severe on peanuts sprayed with either chlorothalonil or captafol than on

unsprayed plants. Campbell (1978) reported that foliar sprays of either fentin hydroxide or copper ammonium carbonate suppressed populations of the two-spotted spider mite.

All currently grown peanut cultivars are susceptible to *C. arachidicola* and *C. personatum*. Sources of resistance in the wild species of *Arachis* have been reported (Abdou et al., 1974). Several immune and highly resistant collections of *Arachis* were found in the sections Erectoides, Rhizomatosae, and Extranervosae. *Arachis chacoense* (GKP 10602) in section Axonomorphae was highly resistant to *C. arachidicola* but susceptible to *C. personatum*. *Arachis cardenasii* (GKP 10017) was susceptible to *C. arachidicola* but immune to *C. personatum*. Sharief et al. (1978) reported heritable variation of leafspot resistance in F_2 populations of crosses made among resistant and susceptible wild species of *Arachis*. Melouk and Banks (1978) developed a detached-leaf technique for screening peanut genotypes for resistance to leafspot. Kornegay et al. (1980) reported that NC-GP 343 and NC 5 produced progeny resistant to both early and late leafspots. Subrahmanyam et al. (1980a, 1980b) also reported resistance to *C. personatum*.

Various characteristics have been associated with leafspot resistance. Miller (1953) reported a relationship between riboflavin content of the seed and leafspot resistance. Hemingway (1957) reported that a thick palisade layer, a darkgreen color, and small stomata were associated with disease resistance. Gibbons and Bailey (1967) found that resistance in 3 wild species of *Arachis* was associated with small stomatal apertures on the adaxial leaf surface. Foster et al. (1980) studied spore production on lesions of 4 genotypes with different levels of resistance and found that the smallest lesions and fewest conidia of *C. arachidicola* were produced on *A. batizocoi*, a susceptible diploid species.

Rust

Peanut rust, caused by *Puccinia arachidis* Speg., is 1 of the most destructive fungal diseases of peanut foliage. The first report of peanut rust was published by Spegazzini (1884) in Paraguay. Previously unimportant outside the Americas (Bromfield, 1971), rust is now present in almost all peanut growing areas of the world (Hammons, 1977; Subrahmanyam et al., 1979). In the United States, peanut rust has been reported in each of the principal peanut-producing states, but serious epidemics of rust have been limited to the peanut-producing areas of southern Texas. Bromfield (1971) reviewed the literature on peanut rust.

Symptoms. Peanut rust can be readily diagnosed by the appearance of uredial sori on the abaxial leaf surface (Figure 1C). The incubation period ranges from 8 to 18 days, and flecks are usually macroscopically visible 2 or 3 days before the appearance of erumpent sori. Generally, sori are restricted to the abaxial surface, but during extended periods of high relative humidity, uredial sori are sometimes formed on the adaxial surface. Uredial sori are usually circular, ranging in size from 0.3-1.0 mm. Sori appear on all aerial plant structures except the flowers and pegs. In contrast with the rapid defoliation associated with early and late leafspots, leaflets with rust become necrotic and remain attached to the plant for several days.

Causal Organism - *Puccinia arachidis* Speg. *Puccinia arachidis* is a prolific producer of uredospores, but teliospores have been only occasionally reported on *A. hypogaea* and on wild species of *Arachis* (Spegazzini, 1884; Hennen et al., 1976). Arthur (1934) described uredospores as: ellipsoid or obovoid, 16-11 x 23-29 μ, with a cinnamon brown wall and 2 nearly equatorial germ pores. Castellani (1959) reported slender conical ornamentations about 1 μ high on the outer wall. Arthur (1934) described teliospores as oblong, often with 3 or 4 cells, 14-16 x 38-42 μ, germinating at maturity, the wall chestnut brown, smooth, and with a colorless pedicel. Pycnidia and aecia have not been observed. Physiologic races of *P. arachidis* have not been reported.

Disease Cycle and Epidemiology. Cook (1980a) reported that *P. arachidis* uredospores germinated within 6 hours by the formation of an unbranched germ tube that emerged from 1 of the equatorial germ pores. Formation of an appressorium over the guard cells of a stoma was followed by the entrance of an infection hypha into the substomatal chamber, ramification of intercellular hyphae, and development of haustoria. Mallaiah and Rao (1979) found that the incubation period for rust was 7 days at 25 C and 14 days at 35 C.

Subrahmanyam et al. (1980b) found that neither size nor frequency of stomata were associated with rust resistance. The infection frequency was lower and the incubation period was longer for resistant genotypes than for susceptible genotypes. Uredospores germinated on the leaf surface, and germ tubes entered stomato of susceptible, resistant, and immune genotypes. In immune genotypes germ tube elongation quickly ceased. Differences in resistant genotypes were manifested by differences in the rate and degree of mycelial development in the substomatal cavities and in the subsequent invasion of leaf tissue.

Peanut rust does not survive from season to season in the United States; airborne uredospores are annually introduced from other peanut producing countries (Van Arsdel and Harrison, 1972). In 1971 a severe epidemic of peanut rust occurred in Texas, and rust was widely distributed in Georgia. In that year, uredial sori were first observed in southern Texas during the first week of July. This was the earliest documented appearance of rust in the United States (Harrison, 1973). Subrahmanyam et al. (1980a) reported that uredospores on crop residue were not viable after 4 weeks under postharvest field conditions at ICRISAT (Hyderabad, India). This demonstrates the brief longevity of *P. arachidis* uredospores.

Total soluble sugars, and amino N accumulated and total N and chlorophyll decreased with age in leaves infected with *P. arachidis* (Subrahmanyam et al., 1976). A small increase in ^{14}C incorporation and starch accumulation noted in the early stages of infection was followed by a decrease in the later stages of disease development.

Foudin and Macko (1974) identified the germination self inhibitor of *P. arachidis* as methyl cis-3,4-dimethoxycinnamate. They concluded that this compound probably inhibits 1 of the initial steps in germination, because the inhibitor lost its effectiveness at the onset of germination.

Cook (1980b) reported that the differential susceptibility of nonphysiologically resistant *A. hypogaea* cultivars was related to leaf wettability. Irrespective of leaf or plant age a similar positive regression of leaf susceptibility on wettability was detected for all nonphysiologically resistant cultivars in the study.

Control. Harrison (1973) controlled peanut rust with chlorothalonil and a tank mix of benomyl plus mancozeb. In southern Texas, the principle of dis-

ease avoidance can be used to reduce the impact of peanut rust; peanuts can be planted in early March, i.e., about 60 to 90 days before the arrival of airborne uredospores (Smith and Littrell, 1980). Two mycoparasites of peanut rust (*Darluca filum* and *Tuberculina costaricana*) have been reported (Bhama, 1972; Sharma et al., 1977). However, these organisms have not been used as biological control agents for *P. arachidis*. Agronomically acceptable rust-resistant peanut cultivars have not been developed. Sources of rust resistance have been reported by Bromfield and Cevario (1970), Hammons (1977), and Subrahmanyam et al. (1980b).

Web Blotch

In 1924 Woronichin (1924) reported *Ascochyta arachidis* on dead peanut leaves in Russia. Khokhryakov (1934) described a similar foliar pathogen on peanuts (*Mycosphaerella arachidicola*). Shoschiaschvili (1940) reported that *A. adzamethica* was the imperfect state of *M. arachidicola*. Cruz et al. (1962) described a foliar disease in Brazil and concluded that the causal agent was a species of *Ascochyta;* on the basis of foliar symptoms they named the disease muddy spot. Frezzi (1969) reported a similar disease in 1969, and he named the perfect state of the fungus *Mycosphaerella argentinensis*. Rothwell (1962a) reported the disease in Rhodesia, and Marasas et al. (1974) described the disease in South Africa. Alcorn et al. (1976) described a similar, if not identical, disease in Australia and named it net blotch. Pettit et al. (1973) found web blotch in Texas in 1972. This disease has also been observed in Oklahoma, New Mexico, Virginia, Georgia, and Florida. Web blotch causes defoliation and a subsequent reduction in pod yield. This disease is often present as part of a foliar disease complex that may include web blotch, early leafspot, and rust.

Symptoms. Marasas et al. (1974) reported that symptoms first appear on the adaxial surface of the lower leaves as scattered tan specks or streaks on the leaf surface, and that the discolored areas expand, forming purplish-brown to dark-brown blotches up to 20 mm in diameter. Margins are irregular and a paler brown color. Blotches often coalesce (Figure 1D). Old lesions are nearly black, with a roughened surface, and portions of the leaflet may disintegrate. Lesions on the abaxial surface develop only after extensive development of epiphyllous blotches. Abaxial blotches are usually pale brown, and their margins are more distinct than those of corresponding blotches on the adaxial surface.

Alcorn et al. (1976) described the symptoms of a similar, if not identical, disease and named it "net blotch." Lesions are initially small, consisting of a few linear anastomosing necroses in an irregular pattern. These lesions enlarge until large areas of the leaflet are covered by irregularly reticulate or netlike necrosis, with apparently healthy tissue in the interspaces. Lesions are confined to the adaxial surface until late in their development, and only then does necrosis appear on the abaxial surface. Eventually, the netlike pattern disappears, resulting in a large reddish-brown blotch.

Causal Organism - *Phoma arachidicola* Marasas, Pauer, & Boerema. Marasas et al. (1974) described the morphological features of the causal fungus on the host as follows: pycnidia globose, amphigenous, light brown, pseudoparenchymatous, ostiolate, 85-200 μ in diameter, immersed in brown necrotic tissue of the lesions. Pycnidia are not easily visible in cleared and stained leaf tissue. Conidia are hyaline, smooth walled, ellipsoidal with rounded ends, 1 celled or, usually, divided in the middle by a septum 7-17 x 3-6 μ.

Chlamydospores are produced singly or in chains, terminally and intercalarily and are thick walled, dark brown, subglobose to globose or ellipsoidal to irregular in shape, 1 celled or divided by 1 or more transverse septa (occasionally, longitudinal septa are also present), constricted at the septa, 9-12 x 8-17 μ, sometimes produced in strands of adjoining hyphae to form complex masses.

Alcorn et al. (1976) provided the following description of the ascigerous state (*Didymosphaeria arachidicola*) of the net blotch fungus *in vitro:*

"Pseudothecia are dark brown to blackish brown, globose, usually immersed, ostiolate, sometimes short beaked, single, separate, and 70-130 μ in diameter. Wall cells are rounded to mostly angular, isodiametric, protruding around the lower and middle parts of the ascocarp when viewed from above. Asci are hyaline, cylindrical to cylindric-clavate, bitunicate, usually constricted near the base to form a distinct foot, apex rounded, 8-spored, spores distichous, 37-58 x 11-15 μ. Pseudoparaphyses are thin-walled, hyaline, septate, and ca. 1.5 μ diameter. Ascospores are at first hyaline, eventually brown, smooth, ellipsoid clavate, 2-celled, constricted, the upper cell broader and more sharply tapered than the lower. The apex is bluntly conic. The basal cell is tapered gradually, and the base is rounded. Discharged ascospores, accumulated at the ostiole, are usually pale yellowish-brown to dark brown. Dimensions in lactofuchsin are 13-16 x 5-6.5 μ. Naturally discharged spores in water are broader, i.e., 6.5-7.5 μ."

Disease Cycle and Epidemiology. Blamey et al. (1977) compared the development of late leafspot and web blotch. They found that web blotch was more severe during rainy seasons with more total rainfall, more evenly distributed rainfall, and less evaporation. In addition, the optimum temperature for development of web blotch epidemics may be lower than the optimum temperature for the development of late leafspot epidemics. This observation is corroborated by the finding that the optimum temperature for *in vitro* growth of the web blotch fungus is ca 8 C lower than the optimum temperature for the early leafspot fungus.

Philley (1975) reported that conidia and ascospores germinated and penetrated peanut leaves in a similar way. Direct penetration was followed by subcuticular growth instead of immediate penetration of the epidermis. Epidermal penetration was intercellular. Stems and petioles were less susceptible than leaves. Adaxial leaf surfaces were more susceptible than abaxial surfaces. Symptoms appeared within 4 days after inoculation. *Arachis hypogaea* is the only known natural host of the web blotch fungus, but Philley (1975) successfully inoculated 6 leguminous genera. Sweet clover and hairy vetch were most susceptible.

Philley (1975) observed maximum mycelial growth on malt extract agar at 20 C, with no growth at 35 C. Pycnidial production on sterilized leaves was greatest at 20 C. Pseudothecia were produced at 15 and 20 C but not at 25 C. The web blotch fungus was homothallic and required light for reproduction. The fungus grew on malt extract agar, potato carrot agar, cherry agar, peanut leaf oatmeal agar, and potato dextrose agar.

Control. Blamey et al. (1977) reported that a tank mixture of benomyl, mancozeb, and a spreader-sticker adequately controlled web blotch. In addi-

tion, foliar sprays of chlorothalonil provide satisfactory control of web blotch. Smith et al. (1979) reported resistance to web blotch in some genotypes. Florunner, a widely grown cultivar in the United States, has an adequate level of resistance to web blotch. Alcorn et al. (1976) reported that valencia and spanish type cultivars were more susceptible than virginia types.

Pepper Spot and Leaf Scorch

Sechet (1955) reported peanut leaf scorch in Madagascar, and he reported that the causal agent was *Pleospora crassiasca*. Yen et al. (1956) reported leaf scorch in Taiwan, and they reported the causal fungus to be *Leptosphaerulina arachidicola*. Luttrell and Boyle (1960) reported pepper spot and leaf scorch in Georgia. Additional reports were subsequently published by Nayudu (1963) in India, Frezzi (1965a) in Argentina, and Pettit et al. (1968) in Texas. This disease is considered to be of minor economic importance throughout the world. Hosts of the fungus were apparently confined to the genus *Arachis*. Frezzi (1965a) reported that *A. burkartii* Handro, *A. glabrata* Benth., *A. hagenbeckii* Harms ex Kuntze, and *A. hypogaea* were susceptible. Graham and Luttrell (1961) found that *A. monticola* Krap. & Rigoni and *A. hypogaea* were susceptible.

Symptoms. This disease is characterized by 2 distinct symptoms. Pepper spot symptoms consist of dark brown to black lesions. Usually these lesions are less than 1 mm in diameter and irregular to circular in outline, and occasionally they are depressed. Discrete lesions usually appear on the adaxial surface and enlarge very slowly. Production of ascocarps is abundant in necrotic areas of abscinded leaflets. Leaf scorch symptoms frequently develop from the tips of leaflets; they consist of a wedge-shaped lesion with a bright yellow zone along the periphery of the advancing margin of the lesion (Figure 1E). Lesions caused by either *Cercospora arachidicola* or *Cercosporidium personatum* are commonly present in association with leaf scorch lesions, leading us to speculate that *L. crassiasca* is frequently a secondary invader of leaf tissue. Other organisms (*Macrophoma* sp., *Phomopsis* sp., and *Alternaria* sp.) frequently produce fruiting structures in the necrotic tissue of leaf scorch lesions.

Causal Organism - *Leptosphaerulina crassiasca* (Sechet) Jackson & Bell. The currently accepted binomial for the causal agent of pepper spot and leaf scorch (*Leptosphaerulina crassiasca*) was proposed by Jackson and Bell (1968). Jackson and Bell (1968) described the causal fungus as follows: perithecia at first submerged, becoming erumpent, amphigenous, yellowish-brown, spherical, thin walled, 60-120 μ in diameter, ostiole short, papillate; perithecia with 8 to 20 bitunicate asci; asci hyaline, ovoid to broadly clavate, with short stipes, 50-80 x 25-45 μ, paraphyses lacking; ascospores hyaline at first, becoming pale yellow to light brown at maturity, 8 ascospores per ascus arranged irregularly, oblong to ellipsoidal, muriform with 3 to 4 transverse septa and 1 to 2 longitudinal septa, commonly with constrictions at the septa, 23-40 x 11-17 μ. An imperfect state of *L. crassiasca* has not been reported.

Disease Cycle and Epidemiology. In a study of the aerobiology of *L. crassiasca*, catches of ascospores in a Hirst spore trap peaked when dew ceased in the morning and at the onset of rainfall (Smith and Crosby, 1973). In addition, the quantity of *L. crassiasca* ascospores in the air increased as defoliation increased, indicating abundant ascocarp production on abscinded leaflets. Mallaiah and Sreeramulu (1976) reported that the seasonal pattern of airborne *L. crassiasca* ascospores and the diurnal periodicity was in close agreement with the patterns observed by Smith and Crosby (1973). Mallaiah and Sreeramulu (1976) stated that ascospore catches peaked when the air temperature was 25-27 C and the relative humidity was 70-74%. Mallaiah and Rao (1976) reported an average of 2.77 germ tubes for each ascospore, formation of appressoria, and direct penetration of epidermal cells. Ascospores were ejected up to 7.5 cm when infected leaflets were placed in glass containers.

Yen et al. (1956) reported cardinal temperatures of ca 8, 28, and 35 C for *in vitro* growth of *L. crassiasca* on potato dextrose agar. Graham and Luttrell (1961) reported forcible ejection of ascospores. We have observed that *L. crassiasca* grows and fruits abundantly on V8-juice agar at 28 C under continuous fluorescent light.

Control. Since this is usually a minor foliar disease of peanuts, the efficacy of fungicides has not been determined under field conditions. Pepper spot and leaf scorch can probably be controlled with fungicides currently used for control of Cercospora and Cercosporidium leafspots. Porter et al. (1971) rated the severity of leaf scorch and pepper spot in variety tests and reported that genotypes susceptible to leaf scorch developed few pepper spot lesions and vice versa.

Anthracnose

Several *Colletotrichum* spp. have been reported as causal agents of anthracnose on peanut, but *Colletotrichum* is usually a minor pathogen. Small (1926) reported *Colletotrichum* sp. on peanut in Uganda. A *Colletotrichum* sp. was reported on peanut stems in Oklahoma (Larsh, 1944). Mehta (1951) reported *Colletotrichum* sp. on peanuts in India. Chevaugeon (1952b) reported *C. mangenoti* on peanuts in Africa, and Wallace (1955) reported a fungus similar to *C. capsici* (Syd.) But. & Bis. in Tanzania. Sawada (1959) reported *Colletotrichum* sp. in Taiwan, and this description was apparently made on the basis of specimens collected in 1909. Silvestre (1961) reported *C. mangenoti* in Senegal. Frezzi (1965a) described a species of *Colletotrichum* on *Arachis* sp. in Argentina. Saksena et al. (1967) reported *C. dematium* (Pers. ex Fr.) Grove as the causal agent of a peanut leaf spotting disease in India. Jackson and Bell (1969) reported that a species similar to *C. dematium* was often associated with leaf scorch symptoms in Georgia. Singh et al. (1975) reported *Colletotrichum* sp. on groundnut in India. Mukiibi (1975) observed anthracnose on stems and stipules in Uganda and concluded that the causal agent was probably *C. mangenoti*.

Symptoms. Chevaugeon (1952b) described *C. mangenoti* symptoms as brownish-gray marginal to elongate lesions on both leaflet surfaces, and rarely on petioles or stems. Silvestre (1961) reported that lesions induced by *C. mangenoti* covered up to half of the leaflet. Sawada (1959) described symptoms induced by *C. arachidis* as scattered lesions, 3-5 mm, circular to irregular, with grayish-white centers surrounded by brown borders. Saksena et al. (1967) described *C. dematium* disease symptoms as small water soaked yellow spots 1-3 mm in diameter. Older spots were dark brown. In some instances spots enlarged rapidly, became irregular, and covered the entire leaflet, sometimes ex-

tending into petioles and branches and resulting in death of the plant. Acervuli were abundant in necrotic tissue. Frezzi (1965a) noted that *Colletotrichum* sp. induced symptoms similar to Leptosphaerulina leaf scorch symptoms.

Causal Organism - *Colletotrichum* spp. Sawada (1959) gave a detailed description of *C. arachidis,* and Chevaugeon (1952b) described the morphology of *C. mangenoti.* Frezzi (1965a) described *Colletotrichum* sp. on *Arachis* sp. Saksena et al. (1967) described *C. dematium.*

Control. Singh et al. (1975) studied varietal susceptibility, fungicidal control, and crop losses associated with *Colletotrichum* sp. Plant mortality ranged from 16.6 to 30.5% among 8 varieties, and mancozeb gave good control. A pod weight loss of 47.7% was reported for 1 severely diseased variety. In an *in vitro* evaluation of fungicides for inhibition of *C. dematium* growth, Chauhan and Verma (1976) found that the percentage inhibition ranged from 26 to 100.

Scab

Bitancourt and Jenkins (1940) reported scab in Brazil. Ojeda (1966) reported scab in Corrientes, Argentina. Scab was also observed in Japan (Takahashi, personal communication).

Symptoms. Small spots, round to irregular, appear on adaxial peanut leaf surfaces (Bitancourt and Jenkins, 1940). Spots are tan with narrow, brown marginal lines, sunken centers and raised margins, and are spreading or frequently adjacent to one or both sides of the midvein. Lesions are often covered with continuous layers of grayish-olive conidiophores and conidia. Later, conidia fall and expose dark brown to black acervuli. On the abaxial surface spots are pinkish brown to red, at times with a brown margin. On petioles and branches lesions are more numerous, larger and oval, up to 3 mm long, sometimes coalescing and causing distortion of branches and petioles. Cruz et al. (1962) and Ojeda (1966) found that the scab fungus attacked all aerial and tender plant parts. Severe infection results in the development of sinuous stems.

Causal Organism - *Sphaceloma arachidis* Bitancourt & Jenkins. Bitancourt and Jenkins (1940) described the imperfect state of *Sphaceloma arachidis* as follows: Acervuli amphigenous, numerous, effuse, sometimes pulvinate, erumpent, 50-250 x 45 μ; conidiophores, caespitose in dense conical or palisade like aggregations, globose or pyriform, yellow, 8-12 x 3-5 μ; conidia, yellow, elongate cylindrical, bilaterally attenuate, 1 or 2 celled, catenulate, 12-20 x 3-4 μ; microconidia numerous, globose, 1 μ in diameter. *Sphaceloma arachidis* grew slowly on potato dextrose agar, forming a compact, superficially puberulent, convoluted, pinkish-tan colony.

Control. Ojeda (1966) reported a high level of resistance on the Guayuru and Overo cultivars, and some resistance in Prudente INTA. Manfredi 1, Manfredi 68, Manfredi Champaqui, Blanco Rio II, Blanco Santa Fe, and races of *A. hypogaea* subspecies *fastigiata* were susceptible. Soave et al. (1973) evaluated 639 entries for resistance to *S. arachidis;* scab was not observed on 15 entries.

Alternaria Leafspot

Kulkarni (1974) reported a leafspot caused by *Alternaria arachidis* Kulk. Balasubramanian (1979) reported that *A. alternata* (Fr.) Keissler caused a foliar disease of 3 cultivars (TMV2, 7, and 11). Orange-brown necrotic spots were observed in the interveinal areas and extending into the veins and veinlets. This disease is now important on the post-rainy season irrigated peanut crop in southern India (Subrahmanyam and McDonald, personal communication). In our experience *Alternaria* spp. are frequent colonizers of necrotic peanut leaf tissue.

Groundnut Leaf Blight

Subrahmanyam (1979) reported that *Myrothecium roridum* Tode ex Fr. caused a leaf blight of peanut in India in 1977. Leaf lesions were round to irregular, 5 to 10 mm in diameter, gray, and accompanied by a chlorotic halo. Lesions coalesced to produce a blighted appearance of leaves. Black fruiting bodies, often arranged in concentric rings, formed on both leaf surfaces. Currently this disease is not economically important in India.

Zonate Leafspot

A zonate leafspot caused by *Cristulariella pyramidalis* Waterman and Marshall in Georgia was reported by Smith (1972). *Cristulariella pyramidalis* was observed late in the growing season after most of the peanut crop was harvested. Pyramidal sporophores were observed on the adaxial and abaxial surface of infected leaflets but only on necrotic tissue. Sclerotia were produced on potato dextrose agar and V8-juice agar.

Rhizoctonia Foliar Blight

Littrell (1974b) reported a foliar blight of peanuts caused by *Rhizoctonia solani* (anastomosis group I). Infected foliage was observed on the lower third of the plant, and the sclerotia larger than 5 mm in diameter were observed on necrotic tissue.

Phomopsis Leaf and Stem Diseases

Atkinson (1944) reported the *Diaporthe sojae* Lehman was the cause of a peanut stem blight. Luttrell (1947) found *D. phaseolorum* var. *sojae* on dead stems and stipules of peanut plants in Georgia. The imperfect state of *D. phaseolorum* var. *sojae* was named *Phomopsis sojae. Phomopsis* sp. was associated with a leaf scorch of peanuts in Argentina (Frezzi, 1965a). Sharma (1974) reported a Phomopsis leafspot disease of *A. hypogaea* in India. He found partially immersed pycnidia with hyaline, 1-celled spores averaging 6.5 x 2.5 μ in necrotic leaf tissue. Luttrell (1947) observed that dense parallel rows of pycnidia on dead peanut stems gave the stems a blackened appearance. Garren and Jackson (1973) reported that *Phomopsis* sp. was commonly associated with *Leptosphaerulina crassiasca, Cercospora arachidicola, Cercosporidium personatum,* and *Colleto-*

trichum sp. in marginal necrotic lesions. Lesions were brown to black, frequently with a chlorotic zone between healthy and necrotic tissue, advancing from the leaflet apex to the petiole in a wedge-shaped pattern. Pycnidia of *Phomopsis* sp. were usually in rows paralleling the midvein or smaller veins. Lesions were circular to irregular, ranging in size from 1-10 mm. These lesions have centers that become paper like, white to light brown, with pycnidia in necrotic tissue, and a reddish-brown margin along the periphery of lesions.

Powdery Mildew

Powdery mildew has been reported in Israel (Chorin, 1961; Chorin and Frank, 1966). Hirata (1966) listed *Erysiphe communis* (Wallr.) Fr. and *Erysiphe pisi* (PC) SH-Amans on peanut in Mauritius, Portugal, and Tanganyika. Chorin (1961) named the powdery mildew fungus *Oidium arachidis* Chorin. *Oidium arachidis* occurs mainly on the adaxial leaf surface and only rarely on the abaxial surface on the mid-rib, and on the petiole. Mature oidia range from 31-44 μ in length and 13-15 μ in width. Conidiophores bear 1 to 2 oidia in a dry microclimate, but in moist, undisturbed air oidia are produced in chains of 3 or 4 or rarely even 6. Subspherical to pyriform haustoria are formed in epidermal cells. Disease development in Israel is rapid at 25 C.

Phyllosticta Leafspot

Phyllosticta leafspot, caused by *Phyllosticta* spp., is a geographically widely distributed but minor disease of peanuts. Moreover, there is still some uncertainty about the identity of the causal agent(s). Jackson and Bell (1969) reviewed the distribution and symptomatology of the disease as well as the taxonomy and morphology of the causal agent. Frezzi (1960) described the lesions as circular to oval, 1.5-5 mm in diameter with definite borders, halos absent, reddish-brown on the perimeters and becoming lighter or tawny in the centers. Shot-hole symptoms developed in some spots. Rao (1963) described the imperfect state of *Phyllosticta arachidis-hypogaea* in India, and Chevaugeon (1952b) described a similar leafspot fungus in Africa.

Melanosis

Frezzi (1960) reported melanosis of peanut leaves in Argentina. Although *Macrosporium* sp. and *Alternaria* spp. were isolated from the lesions, inoculation was successful only with *Macrosporium* sp. Jackson and Bell (1969) concluded that the *Macrosporium* sp. described by Frezzi was actually *Stemphylium botryosum*. Melanosis symptoms are characterized by small irregularly circular, oval or elongate dark brown solitary or confluent spots, 0.5-1.0 mm in diameter if circular and up to 1.5 mm long if elongate. Sometimes entire leaves are covered with flyspeck symptoms. Submerged juvenile lesions become crustlike with age, but defoliation does not ensue.

Pestalotiopsis Leafspot

Satya (1964) reported *Pestalotiopsis arachidis* Satya as the causal agent of dark brown circular leafspots on *A. hypogaea*. Diseased and healthy areas of the leaf were separated by a yellow halo, and were generally restricted to either side of the midrib. Black spherical acervuli were observed in the center of diseased tissue.

Choanephora Leafspot

Mukiibi (1975) observed *Choanephora* sp. on 1 leaflet. According to Mukiibi, Van Hall (1924) reported a similar leafspot in southeast Asia. The brown lesion apparently started at the margin and spread to cover almost the entire leaflet. Sporulation was observed on the adaxial and abaxial surfaces. The lesion was crossed by faint concentric circles with the center close to the leaflet margin.

Peanut Mottle

The first report of peanut mottle virus (PMV) was published by Kuhn (1965) in Georgia. PMV has been reported on peanuts in Africa, Australia, Europe, Japan, the Philippines, South America, and West Malaysia (Reddy et al., 1978). Kuhn reported that 16 species of Leguminosae were susceptible to infection by PMV. Although peanut mottle foliar symptoms are inconspicuous, yield losses have been reported (Paguio and Kuhn, 1974a).

Symptoms. Kuhn (1965) reported that a mold mottle on the youngest peanut was the most commonly observed symptom and that the light- and dark-green areas were readily visible with transmitted light. Curling of leaflets and depressions in the interveinal tissue were common in certain genotypes. Depressions in the interveinal tissue resulted in more prominent veins. Chlorotic rings and leaf patterns were observed infrequently. On *A. hypogaea,* PMV infection caused small irregular gray-to-brown patches on smaller pods, and a brown flecking was observed on the testa of infected seed (Kuhn 1965).

Causal Agent - Peanut Mottle Virus. The PMV is a flexuous rod, ranging from 704 to 812 nm and belongs to the potato virus Y group on the basis of its gross morphology and inclusion bodies (Herold and Munz, 1969; Paguio and Kuhn, 1973). Strains of the virus have been reported (Sun and Hebert, 1972; Paguio and Kuhn, 1973). The mild mottle strain reported by Paguio and Kuhn (1973) appears to be widely distributed (Bock, 1973; Reddy et al., 1978). Paguio and Kuhn (1973) identified 5 strains of PMV on the basis of particle morphology and serological reactions, host range and properties in crude juice and found that mild strains protected against severe strains.

Bock (1973) and Reddy et al. (1978) described the physical properties of PMV. Sap from infected cowpea or soybean was infectious at dilutions of 10^{-2}, rarely at 10^{-3}, and not at 10^{-4}. Infectivity was greatly reduced when soybean or cowpea sap was heated at 54 C for 10 minutes, and infectivity was completely destroyed at 56 C. Expressed sap was infectious for 2 days but not for 3 days at 20 C. Infectivity of sap was slightly reduced after leaves of systemically infected young cowpea were exposed to -12 C for 12 weeks. Virus infectivity was greatly diminished at pH values below 7.0.

Epidemiology. Kuhn and Demski (1975) studied the epidemiology of PMV in Georgia for 5 years and concluded that the initial inoculum was in the

peanut seed. The supporting evidence for this conclusion was as follows: PMV is transmitted through the peanut seed, but not through the seed of other natural hosts such as soybean, pea, cowpea, and *Cassia*. Natural reservoir hosts were not found, and soybeans with PMV were found only in areas close to where peanuts were grown. Moreover, the pattern of PMV spread indicated an internal source of inoculum. Kuhn and Demski (1975) collected *Aphis craccivora* and *Myzus persicae* in Georgia peanut fields and concluded the PMV was transmitted by these aphid vectors. Earlier Behncken (1970) indicated that *Aphis gossypii, Hyperomyzus lactucae* and *Rhopalosiphum padi* also transmitted PMV.

Control. There is no satisfactory control for this disease. Since the symptoms are inconspicuous, growers tend to be unconcerned about the presence of PMV-infected plants in their fields. Kuhn et al. (1968) found no immunity to PMV when they screened 37 peanut cultivars and 428 plant introductions in the greenhouse by mechanically inoculating peanut plants and sub-inoculating *Phaseolus vulgaris* 'Topcrop', a host that developed local lesions. In a later report Kuhn et al. (1978) indicated that PI 261945 and PI 261946 were tolerant to PMV because pod yield was not reduced.

The production of PMV-free seed is a potential control measure. Reddy (1980) reported that there was no transmission of PMV in large samples of seed from 2 genotypes PI 159747 and EC 76446(292). Demski et al. (1975) reported a low incidence of PMV in Texas and Oklahoma and suggested that these areas may be good locations for production of PMV-free seed, probably because of the absence or paucity of aphid vectors. Kuhn and Demski (1975) discussed the possibility of controlling PMV by control of aphid vectors with insecticides. However, they were doubtful about the practicality of this approach because PMV is transmitted in a stylet-borne manner, i.e., the virus is acquired by an aphid with one probe into an infected epidermal cell; hence, it can be transmitted immediately and for only a short time. Moreover, the first aphids probably come from outside the peanut field, and probably cannot be eliminated before they acquire the virus from infected peanut plants.

Groundnut Rosette

Groundnut rosette was first observed in Tanzania (Zimmerman, 1907), but it probably originated in Africa. It appears to be restricted to Africa, south of the Sahara. However, the conflicting reports on its occurrence and the causal virus or viruses indicate that groundnut rosette is, in some ways, still an enigma. In terms of crop losses, it is probably the most destructive of all groundnut viruses in Africa.

Symptoms. At least 2 types of symptoms, chlorotic rosette and green rosette, have been reported (Gibbons, 1977). Chlorotic rosette is prevalent in east and central Africa, and green rosette is predominant in west Africa. Chlorotic rosette is characterized by faint mottling of the young leaflets followed by the unfolding of pale yellow leaves with green veins. Leaves bear progressively smaller chlorotic, curled, and distorted leaflets. Shortened internodes and thickened stems develop. The rate of stunting depends on the time of infection. Although the number of flowers is reduced, flower parts are normal except for the shortening of the hypanthium. Mosaic symptoms and less stunted plants are more commonly seen in central and east Africa. In the case of

green rosette, plants become severely stunted if infection begins early, and leaves may have either isolated flecks or a normal green color.

Causal Agent(s) - Groundnut Rosette Virus/Groundnut Rosette Assistor Virus. There is still some uncertainty about the identity of the causal agent or agents. Sap transmission of groundnut rosette virus (GRV) has been reported (Okusanya and Watson, 1966; Hull and Adams, 1968; and Dubern, 1980). However, Rossel (1977) was unable to demonstrate sap transmission of GRV. Hull and Adams (1968) reported that rosette was caused by 2 viruses. They concluded that the sap-transmissible GRV is responsible for the green rosette symptoms that are usually found in diseased plants and a second aphid transmitted virus (groundnut rosette assistor virus-GRAV) is symptomless in groundnut plants. GRV was mechanically transmissible but required the presence of GRAV for aphid transmission. Okusanya and Watson (1966) reported that the virus consisted of spherical particles with a diameter of 25-28 nm. Buffered sap lost 1/3 of its infectivity at 40-45 C and was noninfectious at 50-55 C. Sap from an East African virus isolate was infectious at a dilution of 1/100, but not at 1/1,000. Virus particles were similar to particles of some persistent aphid-transmitted viruses, e.g., pea enation virus, barley yellow dwarf, and carrot motley dwarf. Local lesion hosts are *Chenopodium amaranticolor, C. album, C. quinoa,* and *Phaseolus mungo*. Systemic infections occur in several legumes and *Physalis floridana* (Dubern, 1980).

Epidemiology. Davies (1972) suggested that viruliferous alate aphids are probably carried to groundnut fields by moving rainfall fronts. The GRV is transmitted in a persistent or circulative manner by *Aphis craccivora* Koch. The matter of perennial plant reservoirs for the virus requires further study. *Stylosanthes* spp. and volunteer peanut plants may be possible GRV reservoirs in some areas of Africa. Although *A. craccivora* prefers the cowpea *(Vigna unguiculata),* this plant is not a host of GRV (Dubern, 1980). Winged aphids are responsible for most GRV transmission in Nigeria, but in Uganda and Malawi secondary spread by apterous aphids is more important (Gibbons, 1977). The optimum time for GRV acquisition access by aphids under experimental conditions is 24 hours. Over 92% transmission occurs within 10 minutes after the beginning of the inoculation access period (Dubern, 1980).

Control. Several cultural practices reduce disease incidence. Destruction of volunteer groundnut plants gives partial control. In some areas early planting and use of high plant populations will provide partial control of rosette because aphid vectors prefer not to colonize dense plant populations (Booker, 1963; A'Brook, 1964). Davies (1975) used S-(4,6-Diamino-s-triazin-2-ylmethyl)0-dimethylphosphorodithioate (menazon) to control *A. craccivora*. This insecticide decreased the prevalence of plants with rosette symptoms and improved the yield and quality of peanuts. Resistance to GRV is controlled by two independent recessive genes (Berchoux, 1960). Rosette resistant lines have been reported in Malawi (Gibbons, 1977), Nigeria (Harkness, 1977), and West Africa (Gillier, 1978). Some of the high yielding rosette resistant varieties are RMP 12, RMP 91, and KH-149A.

Tomato Spotted Wilt

A ringspot disease of *A. hypogaea* was described by Costa (1941) in Brazil,

and Costa (1950) concluded that it was caused by the tomato spotted wilt virus (TSWV). Dyer (1949) reported the disease in peanut in South Africa, and Morwood (1954) reported it on peanut in Australia. Halliwell and Philley (1974) reported spotted wilt in Texas, and one of us (Smith) has observed a few infected peanut plants in Texas each year since 1973. Ghanekar et al. (1979) reported that bud necrosis of *A. hypogaea* in India was caused by TSWV. According to Ghanekar et al. (1979), bunchy top and ring mottle disease of peanut (Sharma, 1966) may be caused by TSWV. The ring mosaic disease described by Narayanasamy et al. (1975) is probably caused by TSWV. Chohan (1972) and Nariani and Dhingra (1963) described groundnut virus diseases in India which are probably caused by TSWV (Ghanekar et al., 1979). TSWV has not caused significant peanut crop losses in Brazil, South Africa, and the United States, but Saint-Smith et al. (1972) reported yield losses of up to 90% in Australia. Ghanekar et al. (1979) reported TSWV infection rates of 5 to 80% in India.

Symptoms and Host Range. A wide variety of symptoms has been reported on *A. hypogaea*. Ringspots (Figure 1F) are the most commonly observed symptoms in Texas (Halliwell and Philley, 1974). However, terminal bud necrosis is very commonly observed in India (Ghanekar et al., 1979). Stunted plants result from shortened internodes. Stunting is especially severe when infection occurs in the seedling stage of development. Necrosis of leaves and stems develops in the advanced stages of the disease. Infection by the TSWV results in the reduction of the size and number of pods, and causes shrivelled seed with discolored seed coats. TSWV has a wide host range, with hosts in the Solanaceae, Compositae, and Bromeliaceae (K. M. Smith, 1972). Best (1968) listed 163 host species in 34 families, and 60 host species were solanaceous. Ghanekar et al. (1979) tested 28 plant species and found that all were susceptible to TSWV. Cowpea (*Vigna unguiculata* cv. 152) was a good assay host.

Causal Agent-Tomato Spotted Wilt Virus. Ghanekar et al. (1979) reported that virus particles in the cytoplasm were spherical, with particle sizes ranging from 70 - 90 nm in diameter. Some particles within infected cells were in the cisternae of the endoplasmic reticulum, and in some instances particles were enclosed in membranous bags. Best (1968) reported 6 TSWV strains on the basis of symptoms on various hosts.

The dilution end point for TSWV was between $10^{-2.5}$ to $10^{-3.0}$, and the thermal inactivation point was 45-50 C (Ghanekar et al., 1979). Leaf extracts retained infectivity for 4 hours but not for 5 hours at 30 C. K. M. Smith (1972) reviewed the pertinent information on serology, purification methods, and chemical composition of TSWV.

Epidemiology. Amin et al. (1981) reported that adults of *Frankliniella schultzei* Trybom and *Scirtothrips dorsalis* Hood could transmit TSWV from peanut to peanut and to several other hosts. Bald and Samuel (1931) reported that *Thrips tabaci* Lindeman, *F. schultzei, F. fusca* Hinds, and *F. occidentalis* Pergravde were vectors of TSWV. The virus is acquired by larvae and transmitted by adults. Although a latent period of a few days between acquisition and transmission is required, it is not known whether the virus multiplies in the vector (Amin et al., 1981). Seed transmission of TSWV has not been reported for peanut (Halliwell and Philley, 1974; Ghanekar et al., 1979).

Control. Ghanekar (1980) screened nearly 7,000 entries of *A. hypogaea* for resistance to bud necrosis under field conditions, and there was no satisfactory resistance. However, the average disease incidence for 2 entries (Robut 33-1 and NC Ac 2575) was less than the other entries. In preliminary field and laboratory tests, *A. chacoense, A. glabrata,* PI 262848, and *A. pusilla* did not become infected. Some cultural practices may be useful in reducing the disease incidence. Early planting at the onset of the rainy season decreased disease incidence and crop losses. Disease incidence was also reduced by planting at a high population density (Amin et al., personal communication).

Peanut Stunt

Miller and Troutman (1966) observed peanut stunt in Virginia in 1964, and Cooper (1966) observed the disease during the same growing season. Epidemics of peanut stunt developed in North Carolina and Virginia in 1965 and 1966 (Cooper, 1966; Miller and Troutman, 1966; Hebert, 1967). Since then, peanut stunt has been reported on peanuts in Alabama and Georgia (Kuhn, 1971; Rogers and Mixon, 1972). In 1967 Choopanya (1968) found peanut stunt virus (PSV) in white clover throughout most of South Carolina but it was not observed in peanut fields. Peanut stunt has been reported in Japan (Tsuchizaki, 1973), Morocco (Fisher and Lockhart, 1978), and France (Douine and Devergne, 1978).

Symptoms and Host Range. Miller and Troutman (1966) reported that symptoms of PSV were similar to those of groundnut rosette. The most conspicuous symptom is severe dwarfing of all plant parts. The entire plant, a branch, or a portion of a branch may be stunted. Leaves are frequently either curled upward or malformed, and diseased plants are chlorotic. Pod production is reduced, and pods are frequently small and malformed, with a split pericarp wall. Many leguminous plants are susceptible to PSV, as are 1 or more species of plants in the Chenopodiaceae, Compositae, Cucurbitaceae, and Solanaceae. *Vigna unguiculata* is used for maintaining cultures of PSV and as a source of virus for purification. *Vigna unguiculata, Phaseolus vulgaris, Chenopodium amaranticolor,* and *C. quinoa* are satisfactory local-lesion hosts (Mink, 1972).

Causal Agent - Peanut Stunt Virus. Mink et al. (1969) reported that a western strain of PSV consisted of spherical particles ranging from 26 to 30 nm in diameter. It is classified under 'cucumo viruses' on the basis of particle morphology and a distant serological relationship with cucumber mosaic virus. The eastern strain was distinguishable from the western strain on the basis of host range, serological relationships, and particle stability (Mink, 1972).

Echandi and Hebert (1971) reported that PSV from *P. vulgaris* was inactivated between 55 and 60 C and was infectious at a dilution of 1:1,000 but not 1:10,000. Expressed sap was infective after 24 hours but not after 48 hours at room temperature. Mink et al. (1969) reported the following physical properties for the western strain of PSV: The *in vitro* longevity of PSV-W in neutral phosphate buffered homogenates was ca 4 hours at 24 C. Homogenates with 0.01 M sodium diethyldithiocarbamate and 0.01 M cysteine retained most of their infectivity for more than 24 hours at room temperature. The dilution end point of stabilized homogenates was ca 1:3,000, and the thermal inactivation point was 50 C. Particles of PSV-W contained ca 16 % RNA, with a base ratio

of 25.7 % adenine, 24.5 % guanine, 21.0 % cytosine, and 28.8 % uracil after acid hydrolysis. Waterworth et al. (1973) described a procedure for purifying PSV isolated from Tephrosia and a simple procedure for routine serological indexing of peanut plants.

Epidemiology. The PSV is transmitted by seed of infected peanut plants at less than 0.1% (Troutman et al., 1967; Kuhn, 1969). It was transmitted by 0.0038% of seed from symptomless plants obtained from fields with a high prevalence of PSV (Culp and Troutman, 1968a). Hebert (1967) found PSV in 28 to 50 samples of white clover (*Trifolium repens*) collected in North Carolina. Infected white clover was commonly found near fields with PSV-infected peanuts (Hebert, 1967). Tolin et al. (1970) were able to isolate PSV from white clover at any time of the year. Hebert (1967) demonstrated the nonpersistent transmission of PSV by the cowpea aphid (*Aphis craccivora*), the green peach aphid (*Myzus persicae*) and the spirea aphid (*A. spiraecola*). Tolin et al. (1970) reported that *A. craccivora* was the predominant aphid species on peanuts at emergence and thereafter. Although it is of no apparent epidemiological significance, Miller and Troutman (1966) demonstrated PSV transmission with an unidentified species of dodder and by mechanical methods.

Control. Tolin et al. (1970) reported that the incidence of PSV was reduced when peanut fields and white clover were not close together. Culp and Troutman (1968b) rated several hundred *A. hypogaea* varieties, introductions, and breeding lines for their reaction to PSV under field conditions. No immunity was reported, but symptoms were less severe on several entries.

Peanut Clump

Peanut clump has been reported from India (Sundararaman, 1926) and West Africa (Trochain, 1931; Bouhot, 1967a). Germani and Dhery (1973) reported that peanut clump was present in Upper Volta during the summer of 1969. Thouvenel et al. (1974) and Germani et al. (1975) reported that the disease was caused by a virus which they named peanut clump virus (PCV). Reddy et al. (1979) reported clump disease of peanuts in India.

Symptoms and Host Range. PCV infected peanut plants are severely stunted, giving a "rosette appearance", and leaves are much smaller than leaves on healthy plants (Thouvenel et al., 1976; Reddy et al., 1979). Mottle and chlorotic ringspot symptoms appear on newly formed leaves, but symptoms quickly disappear when leaves become dark green. Thereafter, infected plants remain severely stunted as compared with healthy plants. Flowers and pegs are produced, but only very few mature pods with small seed are formed. Thouvenel et al. (1976) reported that localized yellow spots developed on *Chenopodium quinoa* and *C. album* within 2 days after inoculation with PCV. Later the spots became ringspots and line patterns that extended along the veins. Reddy et al. (1979) reported that *Phaseolus vulgaris* French bean and *Canavalia ensiformis* were good diagnostic hosts. PCV has an extremely wide host range (Reddy et al., in preparation).

Causal Agent - Peanut Clump Virus. Germani et al. (1975) and Reddy et al. (1979) reported that peanut clump is caused by a rod shaped virus particle. Thouvenel et al. (1976) reported that PCV consisted of large and small virus particles. Large particles averaged 245 nm, and small particles averaged 190 nm. Particle lengths of 165 and 130 nm were observed by Reddy et al. (personal communication). Thouvenel et al. (1976) studied *in vitro* properties of PCV and noted the dilution end point was between 10^{-4} and 10^{-5}. Infectivity of expressed sap from *C. amaranticolor* leaves was diminished after 10 minutes at 60 C and eliminated after 10 minutes at 64 C. Infected *C. amaranticolor* leaves lost infectivity after air-drying for 37 days. *C. amaranticolor* sap was not infectious after 22 to 27 days at 27 C. Infectivity diminished only slightly in *C. amaranticolor* leaves stored for 15 months at 20 C. The absorption spectrum for PCV exhibited a maximum at 267 nm and a minimum at 250 nm. The probable nucleic acid content of PCV is 4.5%.

Epidemiology. Germani and Dhery (1973) suggested that PCV is soil borne. Thouvenel et al. (1976) confirmed this by planting peanut seed in soil obtained from an area infested with PCV and symptoms developed in 132 of 250 seedlings. No symptoms appeared in 40 container-grown seedlings planted in sterilized soil. Reddy et al. (personal communication) found that soil from infested fields contained stubby root nematodes (*Trichodorus* sp.) and *Pythium* sp., suggesting possible vectors of peanut clump. Thouvenel et al. (1978) demonstrated seed transmission of PCV. Transmission rates of up to 24% and 14% were reported for artificially inoculated plants and naturally infected plants, respectively. Thouvenel et al. (1976) were unable to transmit PCV with *Aphis gossypii* and *A. craccivora*.

Control. Germani and Dhery (1973) and Reddy et al. (1979) reduced the disease incidence with nematicides. This is circumstantial evidence that nematodes may be vectors of PCV. In tests at ICRISAT over 1200 germplasm lines were screened for resistance to PCV in a field where the disease incidence was over 90% during the previous year. Symptoms did not develop in 8 cultivars, and the disease incidence was low in 10 other cultivars (Reddy et al., personal communication).

Cowpea Mild Mottle

Cowpea mild mottle (CMMV) has been observed in Kenya, Nigeria, and Ghana (Brunt and Kenten, 1974). CMMV has also been observed in India (Iizuka et al., 1981) and Thailand (Thongmeearkom et al., 1980).

Symptoms and Host Range. Brunt and Kenten (1974) described symptoms on *A. hypogaea* as "Few necrotic lesions, chlorotic rings or line patterns in inoculated leaves soon followed by systemic leaf chlorosis, leaf rolling, and some veinal necrosis. Infected plants are severely stunted." *Beta vulgaris, Cajanus cajan, Glycine max, Phaseolus vulgaris, Nicotiana clevelandii,* and *Theobroma cacao* are diagnostic hosts (Brunt and Kenten, 1973). *Glycine max* and *N. clevelandii* are useful for culture maintenance and as sources for purification of CMMV. *Chenopodium quinoa* is a good assay host because chlorotic or necrotic lesions are produced within 12 days after inoculation. In the principal natural host (*Vigna unguiculata*), CMMV usually causes mild leaf mottling.

Causal Agent - Cowpea Mild Mottle Virus. CMMV particles are straight or slightly flexuous, and particles tend to fragment easily. Brunt and Kenten (1973) reported that the length of the majority of the particles was 650 nm. In leaf dip preparations, negatively stained CMMV particles are sometimes surrounded by a loose external spiral. More recently Iizuka et al. (1981) deter-

mined the particle length and diameter in crude plant preparations. The particles are about 600 nm in length and 13 nm in diameter. The virus contains a single polypeptide with a molecular weight of 33,000 daltons. Sap from systemically infected *G. max* was infectious on *C. quinoa* at a dilution of 10^{-3} but not at 10^{-4} after 10 minutes at 75 C, but not at 80 C, and after at least 8 days at 20 C or 20 days at 2 C. Lyophilized sap remained infective for at least 4 years.

Epidemiology. Little is known about the epidemiology of CMMV as it relates to *A. hypogaea*. It was not seed transmitted in groundnut (Iizuka et al., in preparation). Brunt and Kenten (1973) reported seed transmission (2 to 90%) in *V. unguiculata, G. max,* and *P. vulgaris,* but not in *N. clevelandii.* CMMV was not transmitted by several aphid species to *A. hypogaea* (Brunt and Kenten, 1973; Iizuka et al., in preparation). Recently it was transmitted by whiteflies (Thongmeearkom et al., 1980). Control measures have not been developed.

Peanut Green Mosaic

Peanut green mosaic (PGMV) was observed in India (Sreenivasulu et al., 1981). Infected plants showed chlorotic spots and vein clearing on young quadrifoliate leaves followed by severe mosaic symptoms. The virus was sap transmissible to 16 species of the Leguminosae, Solanaceae, Chenopodiaceae, Aizoaceae and Pedaliaceae. *Phaseolus vulgaris* French bean was identified as a good local lesion host.

Causal Agent - Peanut Green Mosaic Virus. PGMV was identified as a member of the potato virus Y group on the basis of electron microscopy, aphid transmission, and chemical properties. The virus was serologically distinct from peanut mottle virus (Sreenivasulu et al., 1981).

Groundnut Crinkle

Groundnut crinkle virus (GCV) was observed during 1976-1977 in the Ivory Coast (Dubern and Dollet, 1979). In some instances 90% of the plants were infected with GCV. Infected leaves were crinkled, but the size of leaves and plants was only slightly reduced. Diseased plants flowered and produced seed. The virus was transmitted mechanically to *A. hypogaea* Te3, *Centrosema pubescens, Soja max, Vigna sinensis* Black Eye, *Canavalia ensiformis, Dolichos jacquinii,* and *Psophocarpus tetragonolobus.*

Causal Agent - Groundnut Crinkle Virus. The GCV particles are approximately 650 nm long and 12.0 nm in diameter (Dubern and Dollet, 1979). On the basis of serological tests, it appears to be 1 of the carlaviruses (Harrison et al., 1971).

Groundnut Eyespot

Dubern and Dollet (1978) reported groundnut eyespot in the northern part of the Ivory Coast near Korhogo and demonstrated that it was caused by a filamentous virus consisting of particles of about 750 nm long. Transmission of the virus was accomplished with *Aphis craccivora* and mechanically. The virus probably belongs to the "potyvirus" group.

Peanut Yellow Mottle

Peanut yellow mottle virus (PeYMV) has been observed at 3 locations in southern Nigeria (Lana, 1980). Bright yellow mottle symptoms are produced on seedlings in the field. Infected plants have few or no flowers. Pod and seed size of infected plants is reduced from 9-13%. Under cool conditions, especially in the greenhouse or insect cages, peanut seedlings are symptomless, but symptoms are manifested when plants are exposed to higher temperatures. PeYMV was sap transmissible to 37 of 76 plant species in 9 of 16 families tested. *Chenopodium amaranticolor, C. quinoa,* and *Vigna unguiculata* were useful indicator hosts.

Causal Agent - Peanut Yellow Mottle Virus. The disease is caused by a virus, closely related to okra mosaic virus. Virus particles in a partially purified preparation are isometric and approximately 29 nm in diameter. PeYMV sediments into "top" and "bottom" components during sucrose density gradient centrifugation, with apparent sedimentation velocities at 52 S (top) and 110 S (bottom). Lana (1980) was unable to transmit the virus from peanut to peanut with aphids, beetles, and whiteflies. PeYMV was not seedborne.

Marginal Chlorosis

Feakin (1973) reported that marginal chlorosis has been observed on *A. hypogaea* in Papua and New Guinea. Symptoms appear within 2 or 3 weeks after planting. Symptoms are manifested in the form of yellow leaf margins and crinkling of leaves. Plants are smaller than normal, and pod production is about half that of production of healthy plants. The causal virus is seed and graft transmissible.

Rugose Leaf Curl

Rugose leaf curl (RLC) is widely distributed in clovers in Queensland, Australia, but it is rare in *A. hypogaea* (Feakin, 1973). Puckered leaves on terminal shoots become distorted in shape, are harsh to the touch, and erect in habit. No control measures have been developed for this disease. In the original description of RLC by Grylls (1954), leafhopper *(Austroagallia torrida* Evans) transmission of RLC was demonstrated in 16 species distributed in 8 plant families. Three of the hosts were legumes, but *A. hypogaea* was not 1 of them. RLCV was transovarially transmitted by *A. torrida* but not either mechanically or by dodder. Behncken and Gowanlock (1976) reported that rugose leaf curl disease is caused by a rickettsia-like organism.

Peanut Chlorotic Leaf Streak

Iizuka et al. (1981) first observed peanut chlorotic leaf streak (PCLSV) in India. Chlorotic spots, streaks, and puckering of young leaves and severe stunting of young leaves is characteristic of this disease. Systemic mosaic mottling was observed on the following mechanically inoculated plants: *Canavalia ensiformis, Cyamopsis tetragonoloba, Glycine max, Phaseolus aureus, P. mungo, P. vulgaris, Pisum sativum, Vigna unguiculata, Datura stramonium, Nicotiana benthami-*

ana, N. clevelandii, N. glutinosa, N. rustica, N. tabacum, Petunia hybrida and *Zinnia elegans*.

Causal Agent - Peanut Chlorotic Leaf Streak Virus. The PCLSV consists of isometric particles, 45-50 nm in diameter. Spherical or ellipsoidal intracellular inclusions with many particles in a dense matrix are present in infected peanut leaves. Because of the morphology of virus particles and inclusions, this virus resembles caulimoviruses that contain DNA. However, this virus contained single stranded RNA. The ultraviolet absorption spectrum (minimum absorbance at 245 nm and maximum at 200 nm) was typical of the spectrum for a nucleoprotein.

SOILBORNE DISEASES

Stem Rot

Stem rot of peanuts, a descriptive name coined by Garren (1959), also known as white mold, southern stem rot, southern blight and Sclerotium rot, is caused by *Sclerotium rolfsii*. This fungus, isolated from branches of diseased peanuts in 1911 by Saccardo (1911), is 1 of the most important soilborne pathogens of peanuts. Although yield reductions of over 80% can occur, stem rot is generally characterized by an erratic distribution in a field (Aycock, 1966). Environmental factors directly affect infection and disease development (Garren, 1959; Aycock, 1966). A positive linear regression exists between pod yield losses and the number of plants with symptoms of *S. rolfsii* infection (Garren, 1959), and yields can be negatively correlated with the number of infection loci (Garren, 1964a). A system was recently devised to predict actual yield losses caused by infection with *S. rolfsii* (Rodriguez-Kabana et al., 1975). Comprehensive discussion of stem rot of peanuts has been published (Garren, 1959; Aycock, 1966).

Symptoms. A very early symptom of stem rot is sudden wilting of a lateral branch (Garren, 1959). Leaves on affected branches become chlorotic and turn brown. Adjacent branches subsequently develop symptoms. Occasionally, a few branches on each plant survive. Adventitious roots sometimes form on diseased plants. Sheaths of white mycelium form on the affected branches and on the soil surface (Figure 2A). Under favorable conditions mycelial growth is rapid and spreads to other branches and to other plants in the row. Sclerotia, produced abundantly on affected plant parts and on the soil surface, are at first white and velvety but later become light brown to dark brown and spherical. Mycelium may not be noticed on affected plant parts during periods of drought, but it may then be active below the soil line, causing lesions on underground plant parts. Production of visible mycelium may be limited during periods of high soil moisture, but even then branches are readily attacked by the fungus.

Pegs colonized by *S. rolfsii* have light- to dark-brown lesions (0.5-2 cm long). Peg tissue, shredded as a result of infection, is weak and many pods are shed. Pods are also attacked, with severity ranging from a few rotted pods per plant to total rot of pods.

Causal Organism - *Sclerotium rolfsii* Sacc. [*Pellicularia rolfsii* (Curzi) West]. *Sclerotium rolfsii* was the name Saccardo (1911) gave to an imperfect

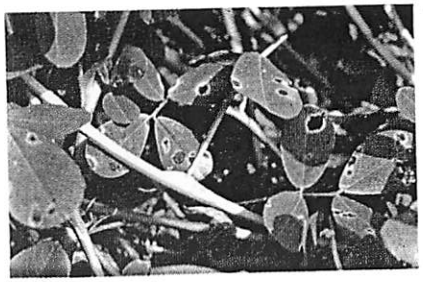
fungus without asexual spores but pathogenic to a wide range of crops. West (1947) studied the basidial stage of *S. rolfsii* in detail and based on characters such as hymenium type, short-celled stout hyphae, basidiospore size and shape, stout basidia, and right-angle branching of the mycelium assigned the name *Pellicularia rolfsii* as the perfect stage of *S. rolfsii*. The taxonomy of *S. rolfsii* was reviewed by Aycock (1966).

Disease Cycle and Epidemiology. The optimum range for mycelial growth of *S. rolfsii* is 30-35 C (Higgins, 1927). Temperatures for preemergent seedling rot caused by *S. rolfsii* (Bell, 1974) and disease development in the field (Aycock, 1966) closely approximate those most favorable for growth in culture. Growth in culture is luxuriant on media enriched with many carbon sources but scanty on media containing few nutrients. Organic substrates in the soil are colonized easily by *S. rolfsii*. Increased inoculum potential and increased disease severity can be positively correlated with the availability of a food base. Debris serving as a food base for *S. rolfsii* can also serve as an infection bridge.

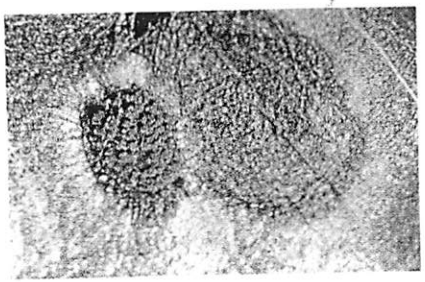
Sclerotium rolfsii penetrates plant tissue directly by the formation of appressoria, and mycelial movement from cell to cell is both intercellular and intracellular (Higgins, 1927). Large amounts of oxalic acid, produced during the infection process, kill host epidermal cells well before penetration by *S. rolfsii* (Higgins, 1927). A large array of enzymes are produced by *S. rolfsii* during pathogenesis (Watkins, 1961; Bateman, 1968; Cole and Bateman, 1969; Van Etten and Bateman, 1969). The respiratory rate of peanut hypocotyls infected with *S. rolfsii* reached maximum levels during symptom development, as did the activity of oxidative enzymes (Amma et al., 1975). Evidence suggests that enzymes secreted by *S. rolfsii* increase permeability of host cells so that electrolytes are released to the fullest advantage of the invading pathogen (Amma et al., 1977). The physiology of parasitism by *S. rolfsii* was reviewed by Watkins (1961).

Soil type is not a limiting factor in the growth of *S. rolfsii* and disease development. However, in heavy soils fungal activity may be limited to the soil surface, while in lighter soils the fungus may be active at greater depths (Boyle, 1961). Soil moisture is important in disease severity and symptom development. During dry periods, *S. rolfsii* does limited damage to above ground plant parts but can cause severe pod rot. The severity of above ground symptoms is positively correlated to soil moisture (Aycock, 1966; Rodriguez-Kabana et al., 1975). Factors that tend to increase or prolong soil moisture, such as dense foliage canopy (Backman et al., 1975), can be directly correlated with disease severity.

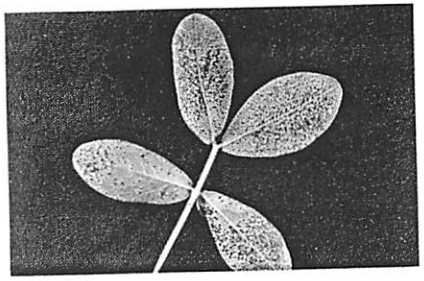
Sclerotia of *S. rolfsii* (0.5-1.5 mm in diameter), produced abundantly on infected plant tissue, retain viability for long periods of time (Garren, 1974). That germination of sclerotia is favored by low humidity may account for increased disease development during dry periods (Boswell, 1958). Drying and rewetting of *S. rolfsii* sclerotia enhanced germination and microbial breakdown of these sclerotia in soil (Smith, 1972). Wetting of field-produced sclerotia did not affect germination in the absence of volatiles of remoistened peanut hay (Beute and Rodriguez-Kabana, 1979b); with these volatiles a 5-fold increase in germination was noted. Volatile compounds, particularly methanol, from remoistened, undecomposed peanut plant tissues stimulated germination of



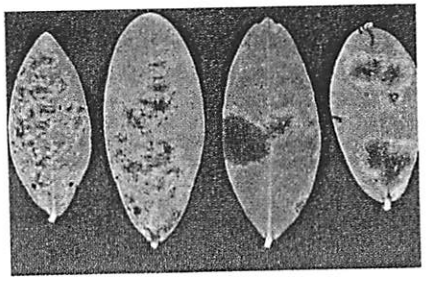
A Leafspot



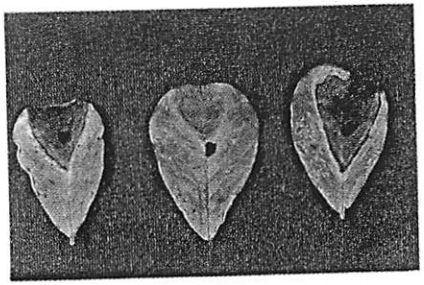
B Late and early leafspot



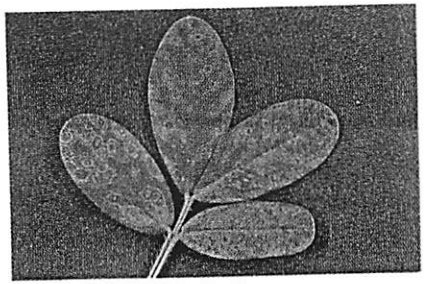
C Rust



D Web blotch



E Scorch



F Spotted wilt

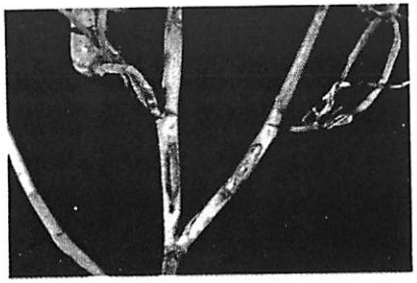
Fig. 1. Fungal and viral diseases of peanut foliage. A. Early leafspot caused by *Cercospora arachidicola;* B. Comparison of early leafspot (brown spot) caused by *C. arachidicola* and late leafspot (black spots covered with tufts of conidia) caused by *Cercosporidium personatum;* C. *Puccinia arachidis* pustules on abaxial leaf surface; D. Web blotch caused by *Phoma arachidicola;* E. Leaf scorch caused by *Leptosphaerulina crassiasca;* and F. Spotted wilt.

A Stem rot

B Sclerotinia

C CBR

D Pod rot Pythium



E Rhizoctonia

F Crown rot

Fig. 2. Symptoms of peanut diseases caused by soilborne pathogens. A. Stem rot caused by *Sclerotium rolfsii;* B. Sclerotinia blight caused by *Sclerotinia minor;* C. Cylindrocladium black rot caused by *Cylindrocladium crotalariae;* D. Pythium pod breakdown caused by *Pythium myriotylum;* E. Rhizoctonia stem canker caused by *Rhizoctonia solani;* and F. Crown rot caused by *Aspergillus niger.*

sclerotia and growth of *S. rolfsii* (Beute and Rodriguez-Kabana, 1979a). Methanol and other volatile stimulants from senescent or dead peanut leaves at the base of the plant may enhance sclerotial germination in the soil to a depth of more than 2 cm and thus may be responsible for an increase in incidence of disease. Nitrogenous amendments, particularly ammonia, significantly decreased the germinability of sclerotia of *S. rolfsii* (Henis and Chet, 1968; Avizohar-Hershenzon and Shacked, 1969).

Control. McClintock (1917) was the first to suggest that stem rot of peanuts might be controlled by the use of resistant cultivars. Many cultivars show tolerance to *S. rolfsii,* but neither immunity nor a high degree of resistance has been found. Growth habit of the peanut (runner vs bunch) was found insignificant in disease development (Garren and Bailey, 1963). In more recent screening trials, stem rot appeared more severe in bunch type peanuts (Muheet et al., 1975). However, a bunch cultivar of peanuts, NC 2, has shown considerable tolerance to *S. rolfsii* (Cooper, 1961). Valencia botanical type peanuts are more susceptible to *S. rolfsii* than spanish botanical type peanuts, which in turn are more susceptible than virginia botanical type peanuts (Garren, 1964a). The literature on resistance of peanuts to *S. rolfsii* has been reviewed (Cooper, 1961; Aycock, 1966).

Severe outbreaks of stem rot of peanuts have occurred regardless of previous cropping history (Garren, 1959). However, disease severity was less and yields were greater in long-term rotation sequences than in short-term rotations (Flowers, 1976). The principle of reducing the incidence of stem rot by manipulation of organic debris with cultural practices has been described (Boyle, 1956, 1961; Garren and Duke, 1958; Garren, 1959, 1961). Since the highest inoculum potential for *S. rolfsii* develops when peanut debris is close to peanut branches, stem rot can be controlled by depriving the fungus of this food base (Garren, 1964a). The concept of control of *S. rolfsii* with cultural practices comprises the following elements: (a) deep plowing to bury surface debris; (b) use of herbicides to minimize cultivation; (c) avoidance of "dirting" during cultivation ("dirting" is the practice of piling soil into the rows to smother seed or grasses therein); and (d) use of effective leafspot fungicides to minimize leaf defoliation. With deep plowing, debris is positioned too far away for infection bridges to occur. Avoiding cultivation or use of nondirting cultivation prevents the formation of new organic debris and the smothering of branches and leaves that could serve as a food base for *S. rolfsii* and initiate disease development. Incidence of disease was greater in hip-rip treatments (subsoiling and bedding without inverting soil and burying crop residue) than in deep plowing treatments (Samples, 1976).

Attempts at chemical control of *S. rolfsii* were not successful until the introduction of PCNB (pentachloronitrobenzene) (Cooper, 1956; Harrison, 1961). There are additional reports of control of stem rot with PCNB (Abd-El-Ghany et al., 1973; Sturgeon, 1973). However, at some locations responses with PCNB have been erratic (Harrison, 1967; Indulkar and Grewal, 1970; Thompson, 1978). Broad-spectrum biocides such as potassium azide applied after peanut seedling emergence can reduce the extent of damage and loss of peanut plants caused by *S. rolfsii* (Rodriguez-Kabana et al., 1972). Carboxin (5,6-dihydro-2-methyl-1,4-oxathiin-3-carboxanilide) was recently demonstrated to be effective against *S. rolfsii* and is now recommended for the control

of stem rot (Diomande and Beute, 1977; Prasad et al., 1977). Dinoseb (2-*sec*-butyl-4,6-dinitrophenol), a commonly used herbicide, reduced the incidence of stem rot under field conditions in Virginia (Chappell and Miller, 1956). These findings, later verified by Backman et al. (1977), showed that dinoseb as well as oxadiazon [2-*tert*-butyl-4-(2,4-dichloro-5-isopropoxyphenyl)-Δ^2-1,3,4-oxadiazolin-5-one] significantly reduced the incidence of stem rot and increased pod yields. Stem rot was also suppressed under field conditions when plants were treated with the herbicide dinitamine [N^3, N^3- diethyl -2,4- dinitro -6- (trifluoromethyl) -1,3-benzenediamine] (Grinstein et al., 1979a). Other herbicides, including paraquat (1,1-dimethyl-4,4-bipyridinium ion) (Rodriguez-Kabana et al., 1967), EPTC (S-ethyl dipropylthiocarbamate) (Rodriguez-Kabana et al., 1970), atrazine [2-chloro-4-(ethylamino)-6-(isopropylamino)-S-triazine] (Curl and Funderburk, 1965), and fluometuron [1,1-dimethyl-3-(d,d,d-trifluoro-*m*-tolyl)urea] (Bozarth and Tweedy, 1971) have also exhibited inhibitory properties against *S. rolfsii* in liquid culture media.

The beneficial and detrimental effects of specific nematicides against *S. rolfsii* have recently been demonstrated. Mycelial growth of *S. rolfsii* and sclerotia production were stimulated in culture by the presence of DBCP and the incidence of stem rot increased in fields treated with this nematicide (Rodriguez-Kabana et al., 1979a). However, other nematicides, including fensulfothion (*o,o*-diethyl *o*-[p-(methylsulfinyl)phenyl]phosphorothionate) and ethoprop (*o*-ethyl S,S-dipropyl phosphorodithioate), have been shown to be fungicidal against *S. rolfsii* (Rodriguez-Kabana et al., 1976a, 1976b). A combination of fensulfothion plus PCNB provided significantly better control of *S. rolfsii* and higher yields than when PCNB was used alone (Thompson, 1978).

Although Cooper (1961) noted that *S. rolfsii* was fairly resistant to parasitism by other soilborne fungi, the potential for biological control has been recently demonstrated. Several species of *Trichoderma* including *T. viride* (Rodriguez-Kabana, 1969), *T. harzianum* (Wells et al., 1972) and *T. lignorum* (Ibrahim et al., 1977) have been shown to be antagonistic to *S. rolfsii*. The efficacy of *T. harzianum* as a biological control agent for *S. rolfsii* under field conditions was first reported by Wells et al. (1972). Control of *S. rolfsii* under field conditions with *T. harzianum* was more recently demonstrated by Grinstein et al. (1979a). Successful methods of delivery of *T. harzianum* to the soil have included the use of diatomaceous earth impregnated with molasses (Backman and Rodriguez-Kabana, 1975) and bran preparations (Grinstein et al., 1979a), but effective delivery of the antagonist remains a serious obstacle to biocontrol. Also, the antagonistic abilities of *T. harzianum* vary greatly between isolates and therefore may influence results (Dennis and Webster, 1971). Stimulatory compounds that increase the parasitism of sclerotia by *Trichoderma* spp. may also help to reduce *S. rolsfii* inoculum in infested soils (Beute and Rodriguez-Kabana, 1979a). On the other hand, reductions in the mycofloral components of the soil, particularly *T. harzianum,* might also predispose plants to invasion by pathogenic organisms such as *S. rolfsii*. The use of benomyl at rates recommended for control of Cercospora drastically reduced populations of *T. harzianum* in the soil but had no effect against *S. rolfsii* (Backman et al., 1975). This selectivity of mode of action is thought to be related to the enhancement of stem rot in peanuts after the application of benomyl. A review of biological control of several plant pathogens, including *S. rolfsii* with *T. harzi-*

anum, was recently published (Chet et al., 1979).

Control of *S. rolfsii* in peanuts by solar radiation was recently demonstrated (Grinstein et al., 1979b). Disease incidence decreased and pod yields increased significantly when soil temperature was increased by mulching moist soil with transparent polyethylene plastic. Katan (1980) recently published a review of solar radiation in disease control.

Sclerotinia Blight

A wilt of peanut caused by *Sclerotinia trifoliorum* was reported in Argentina in 1922 (Marchionatto, 1922). Two species, *S. arachidis* and *S. miyabeana*, were reported attacking peanuts in China in 1933 (Chu, 1933). Another report from China in 1936 identified *S. sclerotiorum* as a causal agent of a peanut wilt disease (Anonymous, 1936). A wilt disease caused by *S. miyabeana* was reported in Taiwan in 1972 (Jan and Wu, 1972). *Sclerotinia minor* was identified as causing a peanut disease disorder in Australia in 1948 (Anonymous, 1948). Frezzi (1960) listed both *S. minor* and *S. sclerotiorum* as causes of a root and pod rot of peanuts in Argentina. Sclerotinia blight was found in the United States in Virginia in 1971 and North Carolina in 1972 (Porter and Beute, 1974) and in Oklahoma in 1972 (Wadsworth, 1979). It has since become widespread and is very serious in Virginia (Porter et al., 1977). Sclerotinia blight has not been found in Georgia, Florida, Alabama, or Texas. A review of Sclerotinia blight of peanut was published recently (Porter, 1980c).

Symptoms. The first symptom of Sclerotinia blight is usually sudden wilting of a single lateral branch (Porter and Beute, 1974). The lateral branches usually become infected along soil contact points. The main branch usually becomes infected by growth of the fungus into the main branch from an infected lateral branch. Foliage on infected branches becomes chlorotic, turns dark brown, and withers; the branch then dies and is defoliated. Thus, these symptoms result in a typical blight. Once infection has been initiated white, fluffy mycelium may develop on the diseased tissue (Figure 2B). The infection process appears to be both intra- and inter-cellular with enzymatic activity concentrating in the middle lamella, so that tissue shredding results. Branch lesions are initially light tan and elongated along the axis of the branch. There is a distinct demarcation zone between the lesion and healthy tissue. Pegs are usually invaded at the soil line. The peg tissue also shreds, resulting in severe pod shed. As lesions develop they appear dry and become dark brown with age. Black, irregularly shaped sclerotia (0.04-3.0 mm) are produced abundantly on all infected plant parts.

Causal Organism - *Sclerotinia minor* Jagger and *Sclerotinia sclerotiorum* (Lib.) de Bary. *Sclerotinia* spp., including isolates producing small sclerotia and those producing large sclerotia, attack a wide host range (Abawi and Grogan, 1979). Classification of *Sclerotinia* spp. has been based in part on host range and sclerotial size. The impracticality of separating *Sclerotinia* spp. on the basis of sclerotial size was demonstrated by Purdy (1955), who showed that the small sclerotial type (*S. minor*), the intermediate sclerotial type (*S. trifoliorum*), and the large sclerotial type (*S. sclerotiorum*) often produced sclerotia of variable sizes. Purdy, therefore, synonymized several species into *S. sclerotiorum*. In Virginia, species of *Sclerotinia* pathogenic to peanuts produce small sclerotia, ranging in size from 0.02-3 mm, similar to those produced by a *Sclerotinia* sp. described by Jagger (1920) as the causal agent of lettuce drop. In Oklahoma, *Sclerotinia* isolates producing small sclerotia and those producing large sclerotia were both taken from diseased peanut fields (Wadsworth, 1979). Apothecia of both species of *Sclerotinia* have been observed in Oklahoma but not in Virginia or North Carolina. Kohn (1979), in a recent attempt to resolve the taxonomic position of the genus *Sclerotinia*, used several taxonomic characters, including the development of free discrete sclerotia, absence of functional conidia, production of ascospores, and orientation of the cells in the outermost layer of the apothecium to delineate 3 species of *Sclerotinia: S. sclerotiorum, S. minor* and *S. trifoliorum*. Using a neotype specimen from peanuts with symptoms of Sclerotinia blight in Virginia in 1974, Kohn identified the organism as *S. minor* and described this species as follows: "Ascospores uniform in size, no segregation in ascus, ectal excipulum at margin of apothecium composed of globose cells; ascospores tetranucleate, length/width ratio of ascospores greater or less than 2; sclerotia formed abundantly throughout the colony, sometimes adhering to form agregate crust and cultures, individual sclerotia .05-2 mm long." Willetts and Wong (1980) recently provided additional evidence to support separation of *S. sclerotiorum, S. trifoliorum* and *S. minor*.

Disease Cycle and Epidemiology. Peanuts become infected with *S. minor* via mycelium from germinating sclerotia (Beute et al., 1975; Wadsworth, 1979). *Sclerotinia minor* usually invades branches, leaflets, or pegs at points of soil contact. The optimum temperature for infection by *S. minor* is 18 C (Imolehin et al., 1980). In Oklahoma, Wadsworth (1979) found a few infection sites some distance from the soil, a finding suggesting the involvement of ascospores in infection. Under moist conditions fallen peanut leaflets or senescing attached leaflets in contact with the soil are easily colonized by mycelium from germinating sclerotia of *S. minor*. This food base enhances disease development but is not necessary for infection, since infection sites are commonly found on branches in contact with the soil but with no such food bases. With some diseases caused by *Sclerotinia* spp., a source of nutrition is a prerequisite for penetration and infection of host tissue (Purdy, 1958). Volatile stimulants from remoistened peanut leaves greatly enhanced the germination of sclerotia of *S. minor* over a wide pH range. Germination was optimum at a pH of 6.5 (Hau et al., 1981).

The severity of Sclerotinia blight can be detected by aerial infrared photography. Sclerotinia blight in peanuts has a unique spectral signature that can be detected on photographs taken at altitudes of ca 20,000 m (Powell et al., 1976a). The severity of Sclerotinia blight as measured on infrared photographs can be correlated with pod losses measured in the field (Porter et al., 1977). Plants in areas on photographs interpreted as being slightly, moderately, or severely affected by *S. minor* had pod losses 2, 5, and 7 times greater, respectively, than plants in areas interpreted as being free of disease. In fields with severely infected plants throughout, peanut losses from *S. minor* can exceed 50% of expected yield.

Mechanically injured peanut foliage is very susceptible to colonization by *S. minor* (Porter and Powell, 1978). Plants injured by tractor tires during pesticide application were invaded at twice the frequency of noninjured plants. At 1 location where Sclerotinia blight was severe, the diseased area increased 152%

after injury by tractor tires. Yield loss is correlated with plant injury. At 2 locations, yields averaged 1,736 kg/ha in injured rows and 2,658 kg/ha in noninjured rows. Infrared photography readily shows much Sclerotinia blight in row middles injured by tractor tires. Studies on the ecology of sclerotia of *S. minor* show that sclerotia are produced abundantly on infected peanuts. Sclerotial populations recovered by sieving were 10 times greater in soil from severely infected portions of the crop than in that from slightly infected portions (Porter et al., 1977). Sclerotial density in the top 2.54 cm of soil may be less than 1 sclerotium per 100 g of soil at time of planting, but may exceed 50 per 100 g of soil immediately after harvest of severely infected crops (Porter, 1980c). In a field with a history of Sclerotinia blight but planted to a nonhost crop for 3 seasons, the viable sclerotial populations declined gradually.

Control. Differences in susceptibility to *S. minor* of 36 peanut cultivars, breeding lines, and plant introductions ranged from slight to severe (Porter et al., 1975a). Florigiant, was the most tolerant to *S. minor* of the cultivars tested. In a 3-year study under severe disease pressure in Virginia, PI 371521 and the breeding line Virginia 71-347 had significantly fewer symptoms of this disease than the other cultivars, breeding lines and plant introductions (Coffelt and Porter, 1982).

The fungicide DCNA (2,6-dichloro-4-nitroaniline) partly controlled Sclerotinia blight in peanuts in Virginia and North Carolina (Beute et al., 1975). Benomyl, applied at high rates, provided some control of Sclerotinia blight (Porter, 1977). Procymidone [3-(3,5-dichlorophenyl)-1,5-dimethyl-3-azabicyclo (3.1.0)hexane-2,4-dione], a fungicide not registered for use on peanuts, almost completely controlled Sclerotinia blight (Porter, 1980b). Pod yields in plots treated with procymidone averaged 4,904 kg/ha; yields were 2,603 kg/ha in the untreated plots. Fungicides with a mode of action similar to that of procymidone, such as Ridomil [metalaxyl; N-(2,6-dimethyphenyl)-N-(methoxyacetyl)-*DL*-alanine methyl ester] and Rovral [iprodione;3-(3,5-dichlorophenyl)-N-(1-methylethyl)-2,4-dioxo-1-imidazolidine-carboxamide], were not nearly as effective against Sclerotinia blight (Phipps, 1980). The value of metham (sodium methyldithiocarbamate) applied in irrigation water for control of Sclerotinia spp. was recently demonstrated (Krikun and Frank, 1981). Some fungicides currently recommended as standard control for leafspot (*Cercospora arachidicola* and *Cercosporidium personatum)* of peanut enhance the severity of Sclerotinia blight. Chlorothalonil applied at rates recommended for leafspot control not only increased the severity of Sclerotinia blight but also significantly reduced pod yield (Porter, 1977). At other field locations captafol as well as chlorothalonil increased the severity of Sclerotinia blight and significantly decreased pod yield (Porter, 1980a). At harvest 2 and 4 times more plants were dead in plots treated with chlorothalonil or captafol, respectively, than in nontreated control plots. Pod yields averaged about 500 kg/ha greater in untreated plots than in chlorothalonil- or captafol-treated plots. Similar responses were noted in field tests with several peanut cultivars (Coffelt and Porter, 1982). The soil microflora of plots treated with chlorothalonil and captafol was not different from that of nontreated plots (Lankow et al., 1980). The increased production of oxalic acid by *S. minor* after application of either chlorothalonil or captafol (over 2.5 times greater in medium amended with chlorothalonil and captafol than in similar nonamended medium) may partly explain

the enhancement of Sclerotinia blight in fields where these fungicides are applied (Hau and Beute, 1981).

Sclerotinia blight of peanuts can be suppressed with dinitrophenol herbicides (Porter and Rud, 1980). Dinoseb and naptalam (N-1-naphthylphthalamic acid) + dinoseb broadcast at 0.84 kg/ha significantly reduced the severity of Sclerotinia blight and increased peanut yields. Crop value was increased by about 18% in herbicide-treated plots. Plant nutrients such as zinc and copper sulfates applied to the peanut foliage significantly suppressed the development of Sclerotinia blight (Hallock and Porter, 1981). These same nutrients applied to the soil had no effect on disease suppression. Other plant nutrients had little or no effect on disease. Soybean (*Glycine max* L.) should not be grown in rotation with peanuts since soybeans are susceptible to both *S. minor* and *S. sclerotiorum* (Phipps and Porter, 1982). Both *Sclerotinia* spp. isolated from soybeans were pathogenic to peanuts.

Cylindrocladium Black Rot

Cylindrocladium black rot (CBR) of peanuts, first discovered in Georgia in 1965 (Bell and Sobers, 1966), was observed later in Virginia (Garren et al., 1971) and Alabama (Rodriguez-Kabana et al., 1974). This disease has been observed in Japan (Misonou, 1973), India (Sharma et al., 1978), and Australia (Colbran, personal communication). It was found in all peanut-producing counties of Virginia and North Carolina by 1976. The reasons for such rapid spread are not known. Spread could not be related to seed stock (Garren, 1973). The affected area of a field may range from less than 1% to over 50% (Lewis et al., 1977). Yields are greatly reduced (Garren et al., 1972), especially on plants infected at time of pegging (Jackson and Bell, 1969). A review of CBR was recently published (Beute, 1980).

Symptoms. The first visible symptoms of infection with *Cylindrocladium crotalariae* are usually on the main branch. The foliage becomes chlorotic and wilts (Figure 2C). The branch often dies. Lateral branches usually are similarly affected, but some may remain unaffected. All below ground parts of the plant can be attacked. Infected roots die and turn black. Frequently, the entire root system is destroyed. The black, fragmented taproot of dying plants loses its bark and branch roots when the plant is pulled from the soil. Occasionally, adventitious roots develop. Dark-brown, slightly sunken lesions form on pegs and pods. Pod lesions may remain discrete, or the entire pod may turn black and rot. Reddish perithecia are produced at the soil line on branches of infected plants.

Development of CBR of peanuts is readily discernible on infrared photographs taken at ca 3,500 m above mean sea level (Powell et al., 1976b). Plants with various stages of disease have unique spectral/spatial signatures in the range of 400-1,075 m (Powell and Porter, 1977). Study of infrared photographs taken during 2 growing seasons showed that CBR was 3 times more extensive in 1975 than in 1974 (Powell et al., 1976c). Such photographs were also used by Lewis et al. (1977) to estimate yield losses from CBR, and losses were much greater in 1976 than in 1974.

Causal Organism - *Cylindrocladium crotalariae* (Loos) Bell and Sobers and *Calonectria crotalariae* (Loos) Bell and Sobers. Loos (1949) described differ-

ences in isolates of *Calonectria* obtained from tea and crotalaria but regarded the isolates as 2 strains or varieties of the same species, i.e., *C. theae* and *C. theae* var. *crotalariae*. Bell and Sobers (1966) noted that the isolates of *Calonectria* obtained from peanuts were morphologically identical to *C. theae* var. *crotalariae* but different from *C. theae*. The stipe of the imperfect form (*Cylindrocladium*) from peanuts has a globose vesicule whereas the stipe of the fungus from tea has a clavate vesicule. On the basis of this distinct, stable morphological characteristic plus other differences described by Loos (1949), Bell and Sobers (1966) proposed to replace *C. theae* var. *crotalariae* with *C. crotalariae* and to call the imperfect form *Cylindrocladium crotalariae*. A description of the fungus is provided by Bell and Sobers (1966).

Disease Cycle and Epidemiology. The entire root system is susceptible to *C. crotalariae*. Mycelium produces infection cushions on the epidermis and penetrates the cortex of fibrous roots in 24 hours (Johnston and Beute, 1975). Sometimes the periderm of hypocotyls is breached and the plants are killed. New periderm laid down in advance of the invading pathogen may limit its spread and allow the plant to recover. *Cylindrocladium crotalariae* is pathogenic to peanuts at soil temperatures from 15-40 C (Bell, 1967a). A soil temperature of 25 C and a moisture content near field capacity were most conducive to infection (Phipps and Beute, 1977a).

Development and ejection of ascospores from perithecia of *C. crotalariae* was optimum at 25 C (Rowe and Beute, 1975a). Repeated discharges occurred within 4 to 6 hours and continued for up to 2 weeks. At first ascospores are forcibly discharged, but as the perithecium ages the remaining ascospores are exuded in a viscous ooze. Ascospores are extremely susceptible to desiccation and thus play only a minor role in disease spread. Rowe and Beute (1975b) found an extreme range of virulence in isolates of *C. crotalariae* from peanut plants. That there is a potential for race development in the fungus is suggested by isolation of *C. crotalariae* from a resistant peanut variety (NC 3033) after 1 cropping cycle (Hadley et al., 1979a).

Microsclerotia, the overwintering propagules of *C. crotalariae*, are produced abundantly on infected roots and can survive for several years in the soil without a host crop. Populations of *C. crotalariae* microsclerotia can be determined by use of selective media (Griffin, 1977) or by elutriation (Phipps et al., 1976). Microsclerotia found in diseased peanut roots are highly variable in size, ranging from 53-88 μm (Rowe et al., 1974). Microsclerotia are extremely susceptible to low temperatures, and populations decline sharply in frozen soil (Phipps and Beute, 1977a). As the inoculum density of microsclerotia increases in the soil, disease severity increases (Hanounik et al., 1977). Phipps and Beute (1977b) correlated microsclerotial density with disease incidence and showed that disease incidence depends on both inoculum density and time. Florigiant, a susceptible variety, was severely diseased in soils with 0.5 microsclerotia per g while 50 microsclerotia per g were necessary to produce severe disease on NC 3033, a resistant variety (Phipps and Beute, 1977b). After harvest, microsclerotial populations increased in plots planted to Florigiant but remained almost static in plots planted to NC 3033 (Phipps and Beute, 1979).

Control. Genetic vulnerability of all virginia botanical type peanuts to *C. crotalariae* has been reported (Wynne et al., 1975; Garren and Coffelt, 1976).

A small-fruited virginia type peanut of spanish ancestory, NC Acc 3033, was resistant to *C. crotalariae* (Wynne et al., 1975; Phipps and Beute, 1977b). Resistance in diallel crosses was primarily due to additive genetic effects, with heritability ranging from 48 to 65% (Hadley et al., 1979b). Germplasm of VGP 1, a cultivar with resistance to *C. crotalariae*, was recently released (Coffelt, 1980). A positive interaction between nematode injury, even on resistant cultivars, and severity of CBR has been demonstrated (Diomande and Beute, 1981).

Fungicidal control of *C. crotalariae* has not been demonstrated, although numerous fungicides have been screened for efficacy. Sodium azide, a wide-spectrum soil biocide, showed promise of controlling CBR in Alabama (Rodriguez-Kabana et al., 1974). Only partial control was obtained with this fungicide in Virginia (Hanounik et al., 1977). Rotations with crops such as corn, wheat, rye, barley, and oats might aid in reducing incidence of CBR, while rotations with crops such as soybean, tobacco, and cotton might increase the disease (Rowe and Beute, 1973; Sobers and Littrell, 1974; Krigsvold et al., 1977). Increases in the severity of CBR were noted in soils containing high organic matter and treated with the herbicides dinitramine and dinoseb (Barron, 1981). Sanitation can be used to minimize infection by *C. crotalariae*. Movement of the pathogen from field to field can be reduced by cleaning tillage equipment (Krigsvold et al., 1977) and by leaving peanut debris on the soil surface for maximum winter kill of microsclerotia.

Pythium Diseases

Pythium spp. cause damping-off, root rot, vascular wilt, and pod breakdown (pod rot) of peanuts. Although most of the diseases are caused by *P. myriotylum*, other *Pythium* spp. are pathogenic to peanuts (Jackson and Bell, 1969). Economic losses due to *Pythium* spp. may range from nil to over 80% and are difficult to define.

Extensive postemergence damping-off (Bell and Minton, 1973) and a serious root rot of older peanut plants (McCarter and Littrell, 1970) caused by *P. myriotylum* were noted in Georgia. In soils inoculated with *P. myriotylum* all peanut seedlings became infected (Wills and Moore, 1973). A vascular wilt of peanuts was observed in Nigeria in 1967 (Perry, 1967) and *P. myriotylum* was isolated from diseased tissue. *Pythium myriotylum* was the causal organism of a wilt disease observed in Virginia (Porter, 1970).

Pod breakdown, a term used by Garren (1966) to describe an in-soil rot of pods attached to an otherwise healthy plant, is usually called "pod rot" (Figure 2D). It is widespread, often causing much economic loss (Garren and Jackson, 1973). Garren (1966), using specific fungicides, showed that *P. myriotylum* is the prime pod breakdown pathogen in Virginia. Several pathogens including *P. myriotylum* (Garren, 1966, 1967, 1970a; Frank, 1968), *Rhizoctonia solani* (Garren, 1970b; Ibrahim et al., 1977), and *Fusarium solani* (Kranz and Pucci, 1963; Mercer, 1977b) can cause pod breakdown. The synergistic effects of *P. myriotylum* and *F. solani* in a pod breakdown complex were demonstrated by Frank (1972a); neither pathogen alone caused pod rot but an interaction of the 2 caused severe pod rot. Similar synergistic interactions also were noted by Garcia and Mitchell (1975a). In Israel (Frank, 1968), *Pythium* spp. are thought to precede *Fusarium* spp. in the pod rot disease complex, while in the United

States (Garren, 1966) *Fusarium* spp. usually precede *Pythium* spp. *Pythium myriotylum* attacks peanut pods at all stages of growth (Frezzi, 1956). *Fusarium* spp. are the dominant fungi isolated from rotted immature and mature pods in some areas of the world (Kranz and Pucci, 1963; Gibbons and Mercer, 1972; Mercer, 1977b; Odunfa, 1979). In Malawi, certain peanut cultivars have a pod rot with a breakdown of the corky outer layers of the pod (Gibbons and Mercer, 1972; Mercer, 1977b).

Symptoms. *Pythium myriotylum* attacks all underground parts of the peanut plant including seed, seedlings, roots, pegs, and pods.

Damping-off. Seed infected with *P. myriotylum* become water soaked and mushy. The first symptom in young seedlings is a rapid total wilt (Jackson and Bell, 1969). Tannish-brown lesions usually form on the hypocotyl and the cotyledon at and below the soil line.

Roots. Fibrous roots especially are vulnerable to *P. myriotylum*. All roots and nodules of severely infected plants are attacked and turn dark brown to black. Cortical tissues disintegrate and slough off readily, leaving a fragmented, nonfunctional stele (Jackson and Bell, 1969). Plants with root rot have stunted growth and wilting.

Vascular Wilt. Wilting of leaflets and petioles on the tip of a single lateral branch is usually the first symptom of this wilt, but under hot, dry conditions, the entire plant wilts (Porter, 1970). Leaflets on infected plants become chlorotic, with adaxial curling beginning at the apical end. Wilted plants usually recover turgidity at night, but when roots are severely infected, survival depends upon the regeneration of lateral roots. Pods usually rot on plants that wilt early in the season but not on plants that wilt later.

Pods. Pod breakdown symptoms caused by *P. myriotylum, R. solani, F. solani,* or a combination of *P. myriotylum* and *F. solani* are often indistinguishable. Both immature and mature pods may be attacked by *R. solani, P. myriotylum,* and *Fusarium* spp. Infected pods have degrees of discoloration from superficial russeting to browning of part or all of the pod, followed by decay (Jackson and Bell, 1969). In moist soil, pods infected by *P. myriotylum* appear watery, become dark brown to black, and rot quickly. Distinct lesions develop on pods infected with *R. solani* and *F. solani,* particularly under dry soil conditions. Pod breakdown caused by *R. solani* usually develops much slower than that caused by *P. myriotylum* (Garren, 1970b; Garcia and Mitchell, 1975a).

Causal Organism - *Pythium myriotylum* Drechs. The genus *Pythium,* established in 1858 by Pringsheim, belongs in the family Saprolegniaceae (Hendrix and Campbell, 1973). *Pythium myriotylum* was described in 1930 by Drechsler (1930). Middleton (1943) compiled information on the host range and separation of species within this genus and showed that numerous plant species were susceptible to *P. myriotylum*. A study of the genus *Pythium* as plant pathogens was made by Hendrix and Campbell (1973).

Disease Cycle and Epidemiology. Species of *Pythium* naturally inhabit the soil and can subsist indefinitely in the soil as saprophytes. Oospores of *P. myriotylum* are probably the primary survival structures (Hendrix and Campbell, 1973). Zoospores and sporangia seem short lived. Zoospores and mycelium of *P. myriotylum* form appressoria and penetrate epidermal cells of peanut pods directly (Jones, 1975). Penetration occurred in 2 hours at 30-34 C, but there was no penetration below 25 C. Pods rotted from mycelium infection but not from zoospore infection. From 15 to 43 oospores per g of soil will result in 50% infection of peanut roots by *P. myriotylum* (Mitchell, 1978). Isolates of *P. myriotylum* vary in ability to cause postemergence damping-off (Bell and Minton, 1973).

Infection of plant tissue by *Pythium* spp. is influenced by soil moisture, soil temperature, pH, cation composition, light, the presence of other organisms, and inoculum density (Hendrix and Campbell, 1973). The optimum temperature for mycelial growth of *P. myriotylum* is 35 C (Littrell and McCarter, 1970). Bell (1967b) demonstrated that under axenic conditions *P. myriotylum* damaged peanut seedlings more at temperatures between 24 and 29 C than at either 18 C or 35 C. *Pythium myriotylum* attacks a wide host range, including grass crops used in rotation with peanuts (McCarter and Littrell, 1968). The incidence of pod rot and the population density of *Pythium* spp. were significantly higher in fields where peanuts had been grown successively (Frank, 1972b). Garren (1970b) showed that an antagonism existed between *P. myriotylum* and *R. solani*. Indigenous populations of *P. myriotylum* dominanted over introduced populations of *R. solani* and caused severe pod breakdown. However, *R. solani* was antagonistic to *P. myriotylum* and prevented or reduced the development of pod breakdown caused by *P. myriotylum* if it was introduced before inoculation with *P. myriotylum* (Garcia and Mitchell, 1975c).

A positive and significant correlation between increasing soil moisture and frequency of Pythium pod rot was demonstrated by Frank (1974). Frequent irrigation of sandy soils increased the severity of pod rot by a combination of *P. myriotylum* and *F. solani,* whereas less frequent and heavier irrigations reduced its severity. The soil fauna, including mites and springtails (Shew and Beute, 1979), nematodes (Garcia and Mitchell, 1975a), and southern corn rootworm (Porter and Smith, 1974), enhances pod breakdown caused by *P. myriotylum*. Synergistic interactions in pod rot were observed when *P. myriotylum* was combined with *Meloidogyne arenaria* (Garcia and Mitchell, 1975a). Preemergence damping-off caused by *P. myriotylum* can be enhanced by either *F. solani* or *M. arenaria* but not *R. solani* (Garcia and Mitchell, 1975c).

Control. Information on resistance of peanuts to damping-off, root rot, and wilt caused by *P. myriotylum* is almost nonexistent. Some resistance to the pod breakdown caused by *P. myriotylum, R. solani, F. solani* and *P. myriotylum* plus *F. solani* has been reported. Porter et al. (1975b) noted that the most widely planted cultivars in Virginia were the most resistant to *P. myriotylum* and *R. solani*. Moderate levels of resistance to *Pythium* spp. and high yield potentials were noted in certain lines grown in *Pythium*-infested soil (Smith and Boswell, 1979). Simpson et al. (1979) recently released a spanish market type peanut with resistance to both *P. myriotylum* and *R. solani*. Frank (1977) reported that Schwarz 21 and Matjan, both spanish type peanuts, and Mwitunde-7, a valencia type peanut, were resistant to pod breakdown caused by *P. myriotylum* and *F. solani*. In evaluating components of resistance to *P. myriotylum,* Frank (1973) noted that equally susceptible cultivars differed in disease incidence and that a low disease incidence does not necessarily mean a high degree of resistance but could imply an escape mechanism.

Since more than 1 organism is involved in pod breakdown, only wide-spectrum fungicides or combinations of fungicides can provide control. Also, nematicides should be used, since nematodes enhance severity of pod break-

down (Garcia and Mitchell, 1975b). Soil fumigants containing methyl isothiocyanate and a nematicide have controlled pod breakdown in North Carolina (Wells, 1968). Metham-sodium applied in irrigation water controlled pod breakdown caused by the *Pythium-Rhizoctonia-Fusarium* complex in Israel (Krikun and Frank, 1981). Pod breakdown in Malawi (Mercer, 1977c), caused by *F. solani,* can be reduced with leafspot fungicides that delay crop maturity so that pods develop in a period of lower rainfall, which is less favorable for fungal proliferation. Pod breakdown caused by *P. myriotylum* can be suppressed significantly by an increased rate of gypsum (Garren, 1964b; Hallock and Garren, 1968; Boswell and Thames, 1976). However, Moore and Wills (1974) found no correlation between high rates of Ca in an artificial medium and pod breakdown incited by either *P. myriotylum* or *R. solani*. The addition of gypsum did not counter the severity of pod breakdown caused by *P. myriotylum* and *F. solani* in Israel (Frank, 1972b). Pod breakdown of peanuts can be increased by the application of K_2SO_4 or $MgSO_4$ at blooming (Hallock, 1973); K_2SO_4 caused more increase in pod breakdown than $MgSO_4$. Gypsum counteracted the adverse effects of K_2SO_4 and minimized the severity of pod breakdown.

Although crop rotation has little effect on pod breakdown, fields where peanuts had been grown for several seasons in succession had significantly more pod breakdown caused by *Pythium* spp. than fields that were fallow for 2 growing seasons (Frank, 1972a). Since soil moisture greatly influences the severity of pod breakdown caused by a combination of *P. myriotylum* and *F. solani,* irrigation might be manipulated, i.e., to be heavier but less frequent (Frank, 1974), to reduce the incidence of pod breakdown.

Rhizoctonia Diseases

Diseases of peanuts caused by *Rhizoctonia solani* such as damping-off, root and stem rot, pod breakdown (pod rot), and foliage blight occur throughout the world. The first record of *R. solani* attacking peanuts was from India in 1912 (Shaw, 1912). A root disease of peanuts caused by *Rhizoctonia* spp. was reported in Santo Domingo in 1929 (Ciferri, 1929). *Rhizoctonia solani* caused much injury throughout the life of peanuts grown in Texas (Ashworth et al., 1961) and is 1 of the most prevalent diseases in Australian peanut fields (Rawson et al., 1972). Bell (1966) isolated this fungus from hypocotyls and senescent cotyledons of peanuts in Georgia; Wills and Moore (1973) noted that it caused a preemergence damping-off and a dry sore shin of peanut seedlings.

From peg penetration to harvest, peanut pods are subject to attack by *R. solani* (Jackson and Bell, 1969). Garren (1970b) demonstrated that *R. solani* was a pathogen of an in-soil rot known as pod breakdown. Seedling diseases and preharvest pod rot of peanuts, caused by *R. solani,* are destructive in Egypt (Ibrahim et al., 1977). Littrell (1974b) noted up to 70% defoliation of the lower part of the plant in a foliar blight of peanuts caused by *R. solani.*

Symptoms. All parts of the peanut plant-seed, seedlings, roots, branches, foliage, pegs and pods-are susceptible to *R. solani.*

Damping-off. Seed are sometimes killed by *R. solani* before or during germination, as are young seedlings. The fungus forms infection cushions on the hypocotyl, penetrates the epidermal and cortical cells, and causes a collapse of the plant tissue. Distinct lesions, often elongated and brown, appear on the hypocotyl. As lesions coalesce or enlarge, the seedling dies.

Root Rot. Peanut roots colonized by *R. solani* usually have individual lesions that are sunken and light to dark brown. The fungus is most active in the outer cortical tissue. Often the taproot is attacked near the soil surface. Once a lesion girdles the primary root, the plant dies and the root system decays.

Mature Plant. Branches, especially those in contact with the soil, are colonized freely by *R. solani* (Figure 2E). Lesions are circular at the site of infection. They may elongate and girdle the stem, and the branch wilts and dies. Lesions are usually sunken, dry, light to dark brown, with distinct elongated rings of zonation. Branch lesions have the appearance of shredded tissue because of the disintegration of all tissues except vascular bundles. Many branches may die. Occasionally, the entire plant is killed.

Pegs. *Rhizoctonia solani* usually invades pegs at or near the soil line. Lesions are sunken and elongate rapidly. Once the peg is girdled, tissue becomes shredded with vascular bundles exposed. The fungus can move down and colonize the pod and subsequently cause rot. The fungus can also move up into the branch, where lesions develop at the point of peg attachment. Pods on pegs infected with *R. solani* are usually lost during harvest.

Pods. Pod breakdown (pod rot) involving *R. solani, Fusarium* spp. and *P. myriotylum* was discussed under Pythium diseases.

Foliage. Leaflets, especially those in contact with the soil, are colonized by *R. solani.* Leaflet lesions are light to medium brown with rings of distinct zonation.

Causal Organism - *Rhizoctonia solani* Kuhn. *Rhizoctonia solani* is usually found without a distinct spore form and thus is put in the group of fungi known as *Mycelia sterilia* (Ainsworth, 1963). The basidial stage, found frequently, has been named *Thanatephorus cucumeris* (Frank) Donk (Donk, 1956), *Corticium vagum* Berk. and Curt., *C. solani* (Prill. and Del.) Bourd. and Galz, and *Pellicularia filamentosa* (Pat.) Rogers (Garren and Jackson, 1973). *Rhizoctonia solani* isolates, including those from peanuts, fall into 2 groups: 1 group with multinucleate hyphal cells, the other with binucleate hyphal cells. The perfect stage of those with multinucleate cells is *T. cucumeris,* while the binucleate isolates have a *Ceratobasidium* perfect state (Parmeter et al., 1967; Taber and Pettit, 1970).

Disease Cycle and Epidemiology. *Rhizoctonia solani* can persist in most soil types for long periods of time and can grow freely without host crops (Blair, 1943; Papavizas and Davey, 1960). Garrett (1956) noted that highly saprophytic fungi usually survive in the soil longer than weakly saprophytic organisms, but prolonged survival of *R. solani* requires some parasitism (Sanford, 1952). Since most soils have organic substrates continually added as crop debris, the potential for long term survival of *R. solani* is unlimited.

The pathogenicity of *R. solani* on peanuts may be due to its ability to produce large amounts of phenolic acids (Sherwood and Lindberg, 1962). As the severity of preemergence damping-off of peanuts increased, production of phenolic acid increased (Reddy et al., 1976). In a host-parasite study the levels of protein, soluble N, protein amino acids, phenols, and B-amylase increased in peanut plants infected with *R. solani,* while the levels of reducing and nonreducing sugars, free amino acids, starch, and phosphorylase decreased (Reddy and Rao, 1978). Only peanuts grown in the summer season in Bangladesh were susceptible to *R. solani* (Khan and Mian, 1974). Under cool temperatures

(21-24 C) 40 to 70% of peanut plants showed signs of disease in Texas (Ashworth et al., 1961). Root rot of peanuts in Mauritius caused by *R. solani* was favored by high humidity, low soil temperatures, and rich humus soil (Felix and Orieux, 1963). *Rhizoctonia solani* is a common companion of the peanut seed and may initiate seedling diseases. Movement of soil or infested plant residue may also facilitate the spread of the fungus (Garren and Jackson, 1973).

Control. Resistance to foliage and root diseases caused by *R. solani* has been noted in some plants but has not been incorporated into acceptable peanut cultivars (Ashworth et al., 1961; Khan and Mian, 1974). Under controlled conditions resistance of peanut seedlings to *R. solani* was noted and could be correlated with resistance noted in field plots (Woodard and Jones, 1980). Resistance to pod breakdown was discussed under Pythium diseases.

Dibromochloropropane (DBCP), a nematicide, provided excellent long-term control of *R. solani* seedling diseases of the peanut at low to moderate inoculum potentials of the fungus (Ashworth et al., 1964a). In Egypt, benomyl and carboxin (Abu-Arkoub, 1973) and a systemic fungicide TCMTB [2-(thiocyanomethylthio) benzothiazole] (Abdou and Khadr, 1974) provided some control of seedling diseases caused by *R. solani*. Other chemical control practices have been suggested (Garren and Jackson, 1973). Control of pod breakdown with fungicides was discussed under Pythium diseases. Cultural practices also aid in controlling peanut diseases caused by *R. solani* (Garren and Jackson, 1973). Seed protectants are available that provide some control of *R. solani* (Jackson, 1963a), but they do not provide control if the fungus is established in the seed cotyledon and hypocotyl (Bell, 1966). Seed rot caused by *R. solani* was greatly reduced in seed treated with antagonists such as *Trichoderma viride* and *Bacillus* sp. (Singh and Chohan, 1974). In field experiments, *T. harzianum* effectively controlled damping-off of peanuts caused by *R. solani* (Elad et al., 1979). Efficacy was greatly improved when *T. harzianum* was added to solarized or fumigated soil.

Fusarium Diseases

Many species of *Fusarium* are ubiquitous on peanut roots, stems, and pods throughout the world. A total of 17 species and varieties of *Fusarium* were isolated from peanut seed and geocarposphere and rhizoplane soil in Israel (Joffe, 1973). However, the exact nature and extent of injury to peanuts by *Fusarium* spp. is not known (Jackson and Bell, 1969). In spite of reports that *Fusarium* spp. do cause considerable damage and greatly reduced yields of peanuts (Miller and Harvey, 1932; Lin, 1959), the economic importance of *Fusarium* spp. as pathogens of peanuts is low.

Fusarium spp. have been associated with seed rot (Jackson and Bell, 1969) and concealed damage (Garren and Wilson, 1951). A wilt of peanut, first described in Georgia in 1932 (Miller and Harvey, 1932), has been found in Taiwan (Lin, 1959), Rhodesia (Rothwell, 1962b), Nigeria (Bailey, 1966), and Malawi (Mercer, 1978). Diseases of both mature and immature pods are sometimes caused by *Fusarium* spp. (Kranz and Pucci, 1963; Gibbons and Mercer, 1972; Mercer, 1978; Odunfa, 1979). The mycoflora of seed and shells of sound mature and overmature pods is sometimes dominated by *Fusarium* spp. (Gilman, 1969; Joffe, 1973). *Fusarium* spp. were readily isolated from peanut hypocotyls and cotyledons (Bell, 1966).

Symptoms. All parts of the peanut plant, including seed, seedlings, roots, stems, pegs, and pods, are attacked by species of *Fusarium*.

Damping-off. Shortly after planting and especially in cool, damp weather, seed are colonized by *Fusarium* spp. from either seed or the soil. The seed generally deteriorate; they become water soaked, and many decay. Under such conditions, seedling emergence is poor and slow. Infected seedlings are stunted, the taproot becomes brown and withers, and the hypocotyl is quickly invaded (Jackson and Bell, 1969).

Wilt. Fusarium wilt, caused mainly by *F. solani* and *F. oxysporum*, is sporadic. Sudden wilting of an entire plant is usually the first evidence of disease. The vascular system of the taproot becomes distinctly discolored, but the other roots of infected plants appear healthy. Hyphae of the fungus are found in vessels of diseased plants (Mercer, 1978). Leaflets turn grayish-green, and sometimes the plants are defoliated before death.

Root Rot. The symptoms on peanut plants with roots infected by *Fusarium* spp. are chlorosis of leaflets, slight wilting, and then death (Miller and Harvey, 1932; Lin, 1959; Jackson and Bell, 1969). The fungus usually attacks the taproot just below the soil surface and causes formation of elongated, sunken, brownish lesions. If the taproot is girdled, the plant wilts and dies. Primary and secondary rootlets also are attacked by *Fusarium* spp., but injury is usually minimal.

Pegs. Pegs are usually colonized at or below the soil line. Distinct lesions develop, and once the peg is girdled, the tissue becomes shredded and weakened.

Pods. Pod breakdown (pod rot) involving *Fusarium* spp., *Rhizoctonia solani*, and *Pythium myriotylum* was discussed under Pythium diseases. Pods infected with *F. oxysporum* and *F. solani* have many seed with "pale testa" and are sometimes characterized by a deep purple pigmentation (Reichert and Chorin, 1942).

Causal Organism - *Fusarium* spp. During the past 50 years several species of *Fusarium* attacking various parts of the peanut plant have been identified. Snyder and Toussoun (1965) reviewed the taxonomy of *Fusarium* spp. and their perfect stages, including *Hypomyces, Nectria, Calonectria* and *Gibberella*. Species identified as pathogenic or associated with peanuts are as follows: *Fusarium solani* is usually associated with seedling rot (Jackson and Bell, 1969; Joffe, 1973). *Fusarium solani* and *F. oxysporum* generally are the causal agents of Fusarium wilt of peanuts (Miller and Harvey, 1932; Lin, 1959). *Fusarium solani, F. oxysporum* and *F. equiseti* are isolated most frequently from pegs (Lin, 1959; Frezzi, 1960; Madaan and Chohan, 1978). *Fusarium roseum* is isolated most frequently from diseased seedlings (Kranz and Pucci, 1963; Bell, 1967b). Although Frank (1972b) and Joffe (1973) noted that *F. solani* dominated the pod mycoflora, others (Kranz and Pucci, 1963; Frank and Palti, 1976) have shown that *F. oxysporum* and *F. equiseti* also can dominate the pod mycoflora. Gilman (1969) and Odunfa (1979) noted the occurrence of *F. semitectum, F. tabacinum, F. heterosporum, F. tricinctum,* and *F. moniliforme* on peanut pods.

Disease Cycle and Epidemiology. *Fusarium* spp. persist indefinitely in the soil as saprophytes. The usual survival structure is the chlamydospore, but conidia and hyphae, produced on soil debris also aid in survival. Many *Fusarium*

spp. that are normally soil saprophytes can become pathogenic to peanuts (Smith, 1970). Where peanuts are cropped continuously, populations of *Fusarium* spp. increase considerably, and the likelihood of disease increases (Odunfa, 1979). Only under conditions very favorable for the growth of the fungus were peanut plants attacked by *F. oxysporum* and *F. roseum* (Bell, 1967b). Wet soil predisposes pods to *Fusarium* spp. (Mercer, 1977b). Injury to the root, either mechanical or chemical, predisposes roots to infection by *Fusarium* spp. (Jackson and Bell, 1969). Many *Fusarium* spp. isolated from peanuts can attack the soft tissue of the seedlings of other crops, though they seldom injure peanuts (Joffe, 1973). Four *Fusarium* spp. were isolated consistently from all subterranean peanut plant parts but did not cause disease symptoms (Jackson and Bell, 1969). It is evident that the *Fusarium* spp., even though abundant in the soil where peanuts are grown, do not often cause major damage of peanuts. Pod breakdown, discussed under Pythium diseases, may be an exception, since severe pod diseases often occur (Mercer, 1978; Odunfa, 1979).

Seed from sound pods are known to be infected with species of *Fusarium* (Gilman, 1969; Joffe, 1973). Seed inoculum may initiate disease during germination and could cause preemergence damping-off. Hypocotyls and cotyledons readily support growth of *Fusarium* spp. (Bell, 1966). Exudates from this rapidly expanding plant tissue may be partly responsible for invasion of the tissue by *Fusarium* spp. Germination of chlamydospores of *F. oxysporum* is predominantly a rhizoplane effect, since germination decreased sharply with increased distance from the root surface (Griffin, 1969).

Control. No cultivars appear resistant to any phase of peanut disease caused by *Fusarium* spp. The incidence of *Fusarium* spp. on hypocotyls and cotyledons was not altered by seed treatment (Bell, 1966). A combination of seed and soil treatment did suppress *Fusarium* diseases of peanut seedlings but not those of pegs in India (Madaan and Chohan, 1978).

Aspergillus Crown Rot

Aspergillus crown rot of peanuts, reported first in the East Indies in 1926 (Jochems, 1926), is now found in all peanut-growing regions of the world (Garren and Jackson, 1973). This disease, also known as collar rot or Aspergillus blight, has caused severe losses in Africa (Gibson, 1953a), Australia (Morwood, 1953), and Iran (Vaziri and Vaughan, 1976). In areas of continuous peanut cropping, losses often exceed 50% (Mathur and Sharma, 1970; Aulakh and Sandhu, 1970b; Chahal et al., 1974). During some years Aspergillus crown rot is a serious problem in Texas (Ashworth, 1963) and New Mexico (Hsi, 1966a). However, this disease causes little loss in Georgia (Jackson, 1962) and Virginia.

Symptoms. *Aspergillus niger* attacks peanuts at all stages, but germinating seed and seedlings are especially vulnerable. Infected seed become pulpy, and sometimes have sooty, black spore masses of the fungus (Gupta and Chohan, 1970a). Infected hypocotyls become water soaked and die. Emerged seedlings are attacked near the soil level. A single branch or the entire seedling may wilt rapidly. The plant may die rapidly. Affected tissue is often covered by black spore masses of the fungus (Figure 2F). Older plants are less susceptible to attack. Additional symptoms of Aspergillus crown rot are given by Garren and Jackson (1973).

Causal Organisms - *Aspergillus niger* van Tieghem and *Aspergillus pulverulentus* (McAlpine) Thom. The most common pathogen of Aspergillus crown rot is *A. niger*. However, *A. pulverulentus* also has been isolated from diseased plants. Under controlled conditions, this fungus was pathogenic to peanuts and caused symptoms similar to those caused by *A. niger* (Chohan, 1965). Both species were characterized by Raper and Fennell (1965).

Disease Cycle and Epidemiology. *Aspergillus niger* persists saprophytically on many substrates. At 18 test locations in Israel, *A. niger* was 1 of 5 dominant fungal species found (Joffe, 1972). In Australia *A. niger* was more prevalent in fields continuously cropped to peanuts than in fields not so cropped (Purss, 1962). A positive relationship was shown between the incidence of crown rot and the number of *A. niger* propagules in the soil (Jackson, 1962; Ashworth et al., 1964b) and between the incidence of crown rot and the prevalence of *A. niger* infection of peanut seed.

Injury to seed, cotyledons, and hypocotyls increased susceptibility of peanut seedlings to *A. niger* (Chohan, 1965). Heat-induced wounds but not mechanical wounds predisposed resistant peanut seedlings to infection (Ashworth et al., 1964b). Factors enhancing infection include high soil and air temperatures, moisture stress, and low light intensity at emergence. In Virginia more propagules of *A. niger* were found in dry soils than in wet soils (Griffin and Garren, 1974). Coleman (1916) showed that *A. niger* can tolerate fairly low levels of soil moisture and can grow well in substrates with high osmotic values. This may explain the high incidence of crown rot in some countries, such as Israel (Joffe, 1972), and its low incidence in others. The generally low soil populations of *A. niger* in Virginia may explain the low incidence of crown rot there (Griffin and Garren, 1974). Crown rot is more prevalent in soils with low organic matter (Morwood, 1953). Although Joffe and Lisker (1970) correlated soil type with the prevalence of pod infection by *A. niger*, Abdalla (1974) could not find this correlation. Joffe (1972) showed that *A. niger* was much more prevalent in the geocarposphere than in the rhizosphere. In a survey of seed stocks in Georgia, Jackson (1963b) found *A. niger* infrequently. He concluded that soilborne not seedborne inoculum initiated Aspergillus crown rot. However, Agnihotri and Goyal (1971) and Vaziri and Vaughan (1976) showed that *A. niger* can be introduced into fields on or in seed and can cause severe disease in fields with no history of crown rot.

Control. In Georgia, virginia type cultivars of runner habit were extremely susceptible to *A. niger* (Jackson, 1962). Spanish type (Ashworth, 1963) and valencia type (Hsi, 1966a) peanuts also are very susceptible to *A. niger*. In India, breeding line U-4-47-7 (Ec21115) was resistant to *A. niger* (Aulakh and Sandhu, 1970b; Chahal et al., 1974). At another location, several peanut lines had some field resistance to *A. niger* (Mathur and Sharma, 1970). The cultivar Ashiriya Mwitunde appeared more tolerant than several other cultivars (Verma, 1971). Germplasm screenings have consistently shown that cultivars with a bunch type growth habit are usually less susceptible than cultivars with a prostrate growth habit to *A. niger*. Some peanut breeding lines have shown promise in disease screening trials throughout the world, but this resistance has not been incorporated into agronomically acceptable cultivars.

Organic mercurial fungicides reduced infection by *A. niger* and greatly improved seedling emergence in the field (Morwood, 1953), but later the same

fungicides seemed to increase the incidence of crown rot (Gibson, 1953b; Purss, 1960; Jackson, 1964). It is speculated that tolerant strains of *A. niger* developed in response to seed treatment with organomercurials and as a result this seed treatment is no longer recommended for control.

Gortner and Kruger (1958) found non-mercury-containing seed protectants such as captan [N-(trichloromethyl)thio-4-cyclohexene-1,2-dicarboximide] and thiram [bis(dimethylthiocarbamoyl)disulfide] effective against *A. niger*. In field studies in Australia (Purss, 1960) and in the United States (Jackson, 1964), a combination of captan and thiram plus an organic mercury compound gave better control than any chemical used singly. However, thiram and captan were the most effective fungicides for *A. niger* on artificially infested seed (Chohan et al., 1966; Sidhu and Chohan, 1971). Ashworth et al. (1964b) showed that fungicides such as captan and thiram did not protect seedlings from low-quality seed against *A. niger* but did protect high-quality, rapidly emerging seedlings.

Charcoal Rot

Charcoal rot, caused by *Macrophomina phaseoli,* is a disease of minor importance in the United States even though it can greatly reduce peanut stands (Hoffmaster et al., 1943). It is 1 of the most common seedling diseases in India, with losses sometimes exceeding 50% (Mathur et al., 1967; Shanmugam and Govindaswamy, 1973a). Charcoal rot is the descriptive name for diseases caused by *M. phaseoli* in a wide host range. On peanuts the disease is called dry rot, ashy stem blight, root rot, Macrophomina root rot, or groundnut root rot. To lessen confusion a better name for the disease in peanuts is "charcoal rot," since this name is so commonly accepted for this disease in other crops.

Symptoms. Peanut seedlings are usually attacked by *M. phaseoli* at the soil line. However, peanut roots, branches, leaves, and pods in all stages of growth are susceptible. Typical charcoal rot occurs in older plants. Signs of infection begin in the taproot or the lower portion of the main stem near the soil line. Infected tissue turns light brown. In the early phase of infection plants do not have aboveground symptoms. However, once the stem is girdled the plant dies, quickly. Shortly before the plant dies, the leaves turn yellow and wilt but usually remain attached to the plant. When death occurs, the entire plant is colonized by *M. phaseoli*. The dead plant, particularly the main branch and taproot, turns black, and these tissues have a profusion of sclerotia. Infested pods turn black, a condition known as black nut. Jackson and Bell (1969) published an excellent summary of symptoms caused by *M. phaseoli*.

Causal Organism - *Macrophomina phaseoli* (Maubl.) Ashby; Syn. *Macrophomina phaseolina* (Tassi) Goid., *Rhizoctonia bataticola* (Taub.) Butler, and *Sclerotium bataticola* Taub. *Macrophomina phaseolina,* the pcynidial stage of *R. bataticola* or *S. bataticola,* was derived from *Macrophoma phaseolina* by Tassi in 1901 according to Goidanich (1947). Based on morphological characteristics of the pcynidial stage of *Macrophoma phaseoli,* Ashby (1927) derived *Macrophomina phaseoli*. The sterile mycelial phase of *M. phaseolina,* first named *S. bataticola* (Taubenhaus, 1913), was later transferred to the genus *Rhizoctonia* (Britton-Jones, 1925). Jackson and Bell (1969) gave reasons for continued use

of *R. bataticola* as the name of the sterile mycelial phase. They also described the taxonomy and morphology of *M. phaseoli* and *R. bataticola*.

Disease Cycle and Epidemiology. According to Garrett (1956), *M. phaseoli* is a soil-inhabiting fungus colonizing only senescent or recently killed plant tissue. High soil temperature (35 C) and low soil osmotic potentials favor the growth of *M. phaseoli* (Odvody and Dunkle, 1979). Charcoal rot development, therefore, is favored by hot, dry weather that reduces plant vigor and favors growth of the fungus (Shokes et al., 1977). Charcoal rot of peanut is most frequent on peanut plants growing under conditions of high soil temperatures and low soil moisture.

Sclerotia of *M. phaseolina* remain viable in the soil for many years (Smith, 1966), particularly in very dry soil; viability is much reduced in water saturated soil (Shokes et al., 1977). Exogenous nutrients increased sclerotial germination at low osmotic potentials, a finding suggesting that nutrients overcome fungistatic factors and promote infection by *M. phaseolina* (Odvody and Dunkle, 1979). Both organic and inorganic N sources are utilized by *M. phaseoli* (Tandon, 1967). Dextrose and asparagine were the best C and N sources, respectively, for the mycelial growth of *M. phaseoli* (Shanmugam and Govindaswamy, 1973b). Mycelial growth was enhanced by the addition of vitamins.

Macrophomina phaseoli is a dominant fungus of peanut shells and seed in irrigated and nonirrigated peanuts (Subrahmanyam and Rao, 1977). In Nigeria, *M. phaseoli* was dominant in the mycoflora of overmature plants (McDonald, 1970), but it was not common in peanut pods in the United States (Jackson, 1965a). Overmature pods were rapidly colonized by *M. phaseoli* (Jackson, 1965a). Riley (1960) reported that *M. phaseoli* was a common component of the pod mycoflora in Tanganyika. *Sclerotium bataticola* was isolated consistently from hypocotyls and senescent cotyledons from tillering peanut plants in Georgia (Bell, 1966). Seed infection by *S. bataticola* was most frequent between 21 and 32 C (Jackson, 1965b). The fungus can be seed transmitted (Mridha and Fakir, 1978).

Control. Mathur et al. (1967) showed differences in susceptibility of peanut cultivars to *M. phaseoli,* but no cultivar was immune. The mortality rate of bunch cultivars was much greater than that of cultivars with a spreading or runner growth habit. Captan and thiram can reduce infection by seedborne *M. phaseoli* and minimize preemergence root rot (Chohan, 1971; Lewin and Natarajan, 1971). Also, PCNB can minimize seedling disease (Bouhot, 1967b). Preemergence rot of peanut seed caused by seedborne inoculum of *M. phaseoli* was best controlled with captafol, but soil drenching with benomyl and PCNB also provided control (Shanmugam and Govindaswamy, 1973a). In biological control studies seedborne pathogens such as *M. phaseoli* have been controlled with the antagonists *Trichoderma viride* and *Bacillus* sp. (Singh and Chohan, 1974).

Diplodia Collar Rot

Diplodia collar rot of peanuts is sporadic throughout the world. Only rarely has it caused much loss (Jacoway and Owen, 1951; Higgins, 1956; Lin,

1959; McGuire and Cooper, 1965). In Australia, the disease is usually confined to areas heavily cropped to peanuts (Rawson et al., 1972). The diseases caused by *Diplodia gossypina* have been called collar rot (Garren and Wilson, 1951), stem blight (Jacoway and Owen, 1951), stem rot (Asuyama and Yamanaka, 1953), Diplodia dry rot (Chorin and Frank, 1961), Diplodia wilt (Lin, 1959), and Diplodia blight (Rawson et al., 1972). Diplodia collar rot (Jackson and Bell, 1969) appears to be the best descriptive name.

Symptoms. Wilting of a lateral branch is the first field symptom of Diplodia collar rot. When the disease is severe, it usually develops rapidly, and the entire plant wilts and dies in a few days. The base of infected plants and the taproots become slate gray to black, and the infected tissue becomes shredded (McGuire and Cooper, 1965). Lesions of greenhouse-inoculated plants have light-brown centers with dark-brown margins (Porter and Hammons, 1975). Branches die when girdled by lesions. Pegs can be infected, with infection spreading up to branches. Black pycnidia develop in the necrotic tissue of infected plants.

Causal Organism - *Diplodia gossypina* (Cke.) McGuire and Cooper. Several species of *Diplodia* have been noted as pathogens of peanut diseases. The synonymy of some of these, including *D. gossypina, D. theobromae, D. frumenti, D. tubericola,* and *D. natalensis* has been suggested on the basis of morphological similarity and lack of host specificity (McGuire and Cooper, 1965). *Physalospora rhodina* (Berk. and Curt.) Cooke has been reported as the perfect stage for most of these species (Voorhees, 1942). No ascogenous stage has been found for *D. gossypina* (McGuire and Cooper, 1965).

Disease Cycle and Epidemiology. *Diplodia gossypina* produced 2 types of spores, single-celled hyaline spores (immature) and 2-celled dark-brown spores (mature) (McGuire and Cooper, 1965). Both spore types cause infection (Brooks, 1944). Single-celled spores are sensitive to desiccation. Mature spores survive in the soil from 1 growing season to the next (Brown, 1971). A secondary spore wall not present in single-celled spores may be associated with the long-term persistence of these spores in the soil (Ekundayo and Haskins, 1969).

Infection by *D. gossypina* in the field is rare unless peanut plants are predisposed to infection by heat (Boyle, 1953; McGuire and Cooper, 1965). In tests in which heat was applied to the stem, *D. gossypina* colonized the heat-damaged cortical tissue and then spread throughout the entire branch (McGuire and Cooper, 1965). Plants irradiated for 10 minutes at 45 C developed heat lesions that were easily colonized by *D. gossypina*. Growth of *D. gossypina* was optimum at 32 C (McGuire and Cooper, 1965). In areas where peanuts are planted on light-colored sandy loam soils, temperatures during hot, dry weather cause heat lesions on peanut branches and predispose them to infection by *D. gossypina*. Predisposition or wounding of plant tissue is not, however, a prerequisite for infection. In the greenhouse, plants of susceptible genotypes were easily colonized by *D. gossypina* (Porter and Hammons, 1975). Infection from soilborne inoculum was through taproots or pegs, and the fungus moved quickly from the pegs into the branches and occasionally throughout the plant. Some plants died. *Diplodia gossypina,* rarely found as a component of the peanut pod mycoflora in the United States, was the dominant fungus found on pods grown in Puerto Rico (Garren and Porter, 1970). Isolates of *D. gossypina* were obtained frequently from unblemished peanut seed from plants growing in infested soil (Porter and Hammons, 1975).

Control. Eight peanut genotypes tested showed no evidence of resistance to field infection by *D. gossypina* (McGuire and Cooper, 1965). In greenhouse studies, Florigiant, now planted on 90% of the peanut acreage in Virginia and North Carolina, is extremely susceptible to *D. gossypina* (Porter and Hammons, 1975). Breeding line F420-100, although not immune, was the most tolerant to *D. gossypina* of all lines tested. Crop rotations should be modified in areas where more susceptible hosts are used in rotation with peanuts. Diplodia collar rot was many times more severe in peanuts following cotton than in those following corn (McGuire and Cooper, 1965). Predisposition of peanut plants to *D. gossypina* by heat injury can be minimized by good Cercospora leafspot control, which results in a dense foliage canopy that shades the soil surface.

Blackhull

Blackhull of peanuts, caused by *Thielaviopsis basicola* was first observed in the United States in 1963 (Mason, 1964). This disease first found only in eastern New Mexico was more severe on valencia type and spanish type peanuts (Mason, 1964; Hsi, 1965; Hsi, personal communication). There are earlier reports of this disease from other countries (Ciccarone, 1949; Frezzi, 1960). Blackhull was found recently on spanish type peanuts in South Africa (Baard et al., 1980; Prinsloo, 1980), where pod losses sometimes exceeded 50%. All commercial cultivars of both spanish and virginia type peanuts currently planted in New Mexico are susceptible to this disease.

Symptoms. Blackhull is characterized by numerous black lesions on the surface of pods. As individual lesions increase in size, they coalesce, and eventually most of the pod surface becomes blackened, so that pod marketability is greatly reduced. Masses of chlamydospores are produced throughout the infected shell tissue. Seed inside infected pods sometimes become discolored, although Mason (1964) found no evidence that *T. basicola* attacked any part of the peanut plant other than the shell. Hsi (personal communication) and Baard et al. (1980) noted that roots and pegs of plants also are susceptible to attack and Tabachnik et al. (1979) found necrosis of the taproot. The blackening or browning of plant tissue from black rot is induced by methyl acetate, a phytotoxin (Tabachnik and Devay, 1980).

Causal Organism - *Thielaviopsis basicola* (Berk. and Br.) Ferr. and *Chalara elegans* Nag Raj and Kend. In a comprehensive treatment in 1975, Nag Raj and Kendrick (1975) noted that the distinctions between the genera *Chalara, Thielaviopsis,* and *Chalaropsis* were arbitrary at best. They proposed that the 3 genera be combined in the genus *Chalara*. The characteristic phialidic state of phialides and phialoconidia was very similar in all 3 genera, and this similarity was called a basis for recognizing only 1 genus. This combination of these 3 genera into 1 genus was suggested first in 1968 (Barron, 1968). A complete mycological description of *C. elegans* is provided by Nag Raj and Kendrick (1975). However, this suggestion to combine these genera is not generally accepted since the criteria for combination is based on the imperfect stage of the fungus (Hsi, personal communication).

Disease Cycle and Epidemiology. *Thielaviopsis basicola* persists in field soils mainly as chlamydospores (Tsao and Bricker, 1966), and it can survive indefinitely as a saprophyte (Nag Raj and Kendrick, 1975). Several hosts of *T. basicola* have been reported (Tabachnik et al., 1979). Using selective media, Hsi (1978) found no significant differences in the chlamydospore populations of *T. basicola* in soils under different cropping sequences. However, blackhull was more prevalent in fields cropped continuously to peanuts than in fields with peanuts rotated with grain sorghum. Crop sequence had no effect on blackhull prevalence in areas where peanuts were grown on calcareous soils (Hsi, 1966b). Significantly greater populations of *T. basicola* were found in fields where blackhull was severe than in fields where the prevalence of blackhull was low.

Growth of *T. basicola* in culture was optimum at 15-16 C (Maier, 1967). Chlamydospores of *T. basicola* survived in the soil at very low soil temperatures (Tsao and Bricker, 1966). High soil and air temperatures suppressed disease development while low soil and air temperatures enhanced disease development (Hsi, 1977). Heat units of the cooler months of May and June were positively correlated with severity of blackhull damage, whereas those of the hotter months of September and October were negatively correlated with disease severity. A positive relationship also existed between disease severity and soil moisture when heavy rains followed irrigation (Hsi, 1977). Blackhull recently has become a major problem of irrigated peanuts in South Africa (Prinsloo, 1980). Growth of several strains of *T. basicola* obtained from peanuts in New Mexico was optimum on media with a pH of 8 (Maier, 1967). Soil with low pH appears to suppress growth of *T. basicola*. *Thielaviopsis basicola* seems not to be seed transmitted, since seed from fields with severe blackhull produced plants with disease-free pods (Hsi, 1965; Baard et al., 1980).

Control. There are no peanut cultivars available that are resistant to *T. basicola*. In-furrow band sprays of benomyl and thiophanate methyl [diethyl(1,2-phenylene bis iminocarbonothioyl)bis(carbamate)] controlled *T. basicola* (Hsi, 1976; Hsi and Ortiz, 1980). Foliar sprays also reduced disease severity. Blackhull is controlled in New Mexico by planting peanuts in rotation with grain sorghum (Hsi, 1965, 1966b). Irrigation should be used judiciously to prevent excessive soil moisture for extended periods of time.

Verticillium Wilt

A vascular wilt of peanuts caused by *Verticillium albo-atrum* was observed in New Mexico by Smith in 1958 and described in 1960 (Smith, 1960). This fungus had been found previously only in diseased peanuts in which *Fusarium* spp. was the primary pathogen (Golovin, 1937; Morwood, 1945). More recently, Verticillium wilt was reported in Australia (Purss, 1961), Israel (Krikun and Chorin, 1966), Oklahoma (Khan et al., 1972) and Virginia in 1974 (Porter, unpublished data). Verticillium wilt is not widespread in the world. However, it can become serious and cause economic losses exceeding 50% (Smith, 1960; Purss, 1961; Khan et al., 1972). A pod rot phase of Verticillium was reported in Argentina (Frezzi, 1965b). *Verticillium albo-atrum* caused severe pod losses in Australia recently (Middleton, personal communication).

Symptoms. Symptoms of Verticillium wilt range from mild chlorosis of leaflets to severe wilt. Leaflets on infected plants usually turn dull green, a change followed by marginal chlorosis and a curling of individual leaflets. The root system of infected plants appears normal except for brown-to-black vascular discoloration. Plants first wilt during the middle of sunny days but usually recover turgidity during the night. Wilt eventually becomes permanent. Leaflets on permanently wilted plants become chlorotic and are shed rapidly. Occasionally, plants die. Roots on dead plants are severely rotted and the vascular tissue of the taproots turns black; this blackening extends up into the vascular tissue of the branches.

Causal Organisms - *Verticillium albo-atrum* Reinke and Berth. and *Verticillium dahliae* Kleb. Verticillium wilt of peanuts is caused by either *Verticillium albo-atrum* or *V. dahliae*. The taxonomic basis for separation of 2 species has been questioned (Jackson and Bell, 1969). Although there is no genetic basis for 2 species (Isaac, 1967), the production of microsclerotia by *V. dahliae* (Isaac, 1949), its temperature requirements for growth (Robinson et al., 1957), and the dark mycelium of *V. albo-atrum* (Isaac, 1967) are used to separate them as distinct ecological groups. Both *V. albo-atrum* (Smith, 1960) and *V. dahliae* (Purss, 1961; Frank and Krikun, 1969) are pathogenic to peanuts. *Verticillium albo-atrum* was the pathogen of the wilt in New Mexico (Smith, 1960), while *V. dahliae* was isolated from wilted peanuts in Virginia (Porter, unpublished data).

Disease Cycle and Epidemiology. *Verticillium albo-atrum* and *V. dahliae* are soil-invading fungi and can survive in the soil for long periods. Without susceptible hosts the inoculum potential of *V. albo-atrum* declines rapidly over 2 to 3 years (Sewell and Wilson, 1966) while the inoculum potential of *V. dahliae* remains high (Purss, 1961). Wilhelm (1950) showed that the percentage of infected tomato plants was directly proportional to the density of microsclerotia in the soil. Also, inoculum distribution in the soil and previous cropping history influenced the severity of tomato wilt. Invasion of susceptible plants by *Verticillium* spp. through the root cortex is followed by a systemic invasion of vascular tissue (Garber and Houston, 1966). Wilhelm (1950) found no relationship between soil type and severity of Verticillium wilt of tomato, but in Israel the peanut disease is much more serious on chalky soils (Frank and Palti, 1976). Verticillium wilt of peanuts was more severe in Australia on fertile soil than on less fertile soil (Purss, 1961).

Control. Populations of *V. albo-atrum* can be suppressed by crop rotation (Sewell and Wilson, 1966), but cropping sequences have no effect on the microsclerotia-producing *V. dahliae* (Purss, 1961). Populations of *V. albo-atrum* are reduced drastically in the absence of a host crop or dicotyledonous weeds. Peanuts in rotation with cotton, okra, and peanuts were more severely infected with *V. dahliae* than peanuts in rotation with grain sorghum (Hsi, 1967). Virginia type peanut cultivars, including Va. Bunch 67 and NC 2, were more resistant to *V. albo-atrum* than either spanish or valencia cultivars (Smith, 1961). Of 28 peanut cultivars screened in Israel for resistance to *V. dahliae* only 1, Mwitunde-3, was resistant (Frank and Krikun, 1969). Selection 65-121 from Schwarz-21 also appeared resistant. Khan et al. (1972) found no resistance in 81 plant entries but noted that various levels of susceptibility to *Verticillium* spp. did exist. Some control of Verticillium wilt of peanuts with fungicides has been reported. *Verticillium dahliae* was controlled with metham

applied continuously through sprinkler irrigation in Israel (Krikun and Frank, 1981).

Botrytis Blight

Botrytis blight of peanuts, caused by *Botrytis cinerea,* first reported in 1914 in Japan (Suematu, 1924), has spread throughout the world. It has been found in South Africa (Dyer, 1951), Russia (Protsenko, 1954), Japan (Sawada, 1958), Tanganyika (Riley, 1960), Venezuela (Mazzani, 1961), Rhodesia (Whiteside, 1960; Rothwell, 1962), the United States (Orellana and Bailey, 1964), and Romania (Puscasu, 1977). All parts of the plant are subject to attack. Damage to peanuts usually is slight. Conditions favoring good development of disease are not often found during the regular growing season. Occasionally outbreaks do greatly reduce yields. The disease is also called "gray mold" or, in South Africa, "Botrytis shoot disease" (Dyer, 1951).

Symptoms. Mechanically wounded tissues or tissues weakened by other pathogens (Rothwell, 1962) or by frost are readily attacked by *B. cinerea.* Plants become infected usually at branch tips but also on branches at soil contact points. If infection of the foliage is severe, the fungus can move rapidly into pegs and pods. The fungus may kill branches or entire plants rapidly under favorable conditions. A characteristic symptom of Botrytis blight is the complete coverage of infected tissue with gray conidia, conidiophores, and mycelium of the fungus. Also, black, irregular-shaped sclerotia are produced abundantly on infected plant tissue.

Causal Organism - *Botrytis cinerea* (Persoon) ex Fries. The genus *Sclerotinia* was originally erected by Fuckel (1870) to include species producing free sclerotia or sclerotia embedded in host tissue in or not in conjunction with conidia. The genus was redefined in 1945 (Whetzel, 1945) to include species producing a true sclerotium not embedded in host tissue and species not producing a conidial state and with hyaline conidia. The taxonomy and morphological characteristics of the genus *Botrytis* are given by Gilman (1957).

Disease Cycle and Epidemiology. *Botrytis cinerea* overwinters as sclerotia in the soil. Sclerotia may survive for several years in the soil. Under favorable conditions the sclerotia germinate to form mycelia or conidiophores and conidia. Apothecia have been reported from sclerotia taken from peanut fields (Garren and Jackson, 1973). Infection of the peanut plant by mycelium is near the soil surface and seems to be aided by contact between dead leaves and living plant tissue (Rothwell, 1962). Conidia, produced profusely on infected tissues, are widely dispersed by wind currents. Under favorable conditions, conidia germinate to form mycelia, which penetrate and infect host tissue, especially wounded tissue. Low night temperature (15-20 C) with heavy dews, rainfall, or both are prerequisite for infection of peanuts by *B. cinerea* (Higgins, 1956; Orellana and Bailey, 1964). These are usually conditions of late fall, when most peanuts are already harvested or are ready to be harvested.

Control. There are no peanut cultivars known to be resistant to *B. cinerea.* Fungicides such as benomyl, chlorothalonil, and DCNA are effective against *B. cinerea* in other crops. Disease threat could be reduced by planting early maturing cultivars that would escape frost and by minimizing plant wounding during production.

Rhizopus Diseases

Rhizopus, a soil-inhabiting saprophyte, was first associated with seed rot of peanuts in the United States in 1943 (Atkinson, 1943) and has long threatened the peanut industry of Australia (Purss, 1962). This fungus, cosmopolitan in most soil types where peanuts are grown, causes seed rot and preemergence seedling rot throughout the world. The disease occasionally has been found on emerged seedlings (Bell, 1966, 1967b; Gupta and Chohan, 1970b). *Rhizopus* spp. were the organisms primarily responsible for seed decay, seedling rot, and seed decay in storage in India (Srivastava and Saksena, 1974). Losses from diseases caused by *Rhizopus* spp. are difficult to assess, but losses from 1 to 60% have been reported (Jackson and Bell, 1969). Stand counts were reduced 80% in the Sudan by fungi including *Rhizopus* (Clinton, 1962). *Rhizopus* spp. are the dominant fungi isolated from rotted peanut seed (Mercer, 1978) and from sound seed (Bell, 1966).

Symptoms. Under favorable conditions peanut seed are rapidly decayed by *Rhizopus.* Planted seed turn dark brown, become pulpy, and can decay within 36 to 96 hours. Loose mats of mycelium with clinging soil particles often envelop decaying seed. Necrotic lesions are found on the plumule and cotyledonary branches (Bell, 1966). The plumule can be completely destroyed and covered with a mass of fungal mycelium and spores. Infected seedlings are stunted and often die.

Causal Organism - *Rhizopus* spp. Three *Rhizopus* spp., including *R. arrhizus, R. stolonifer* and *R. oryzae,* are pathogenic to peanuts. The morphology and taxonomy of these species were reviewed by Jackson and Bell (1969).

Disease Cycle and Epidemiology. *Rhizopus* spp. produce resting spores, zygospores or chlamydospores that can survive in soil for many months. The fungus can live indefinitely as a saprophyte in the soil. Wounds, although not necessary, do enhance the infection process. Spores of *Rhizopus* spp. are airborne and soilborne and the fungus may be seedborne. *Rhizopus arrhizus* is most active and causes most severe pod damage at 31 to 37 C (Gupta and Chohan, 1970b). Necrosis of peanut seedlings was maximum at 35 C (Bell, 1967b). Infection of seed inside surface-disinfested pods inoculated with *R. stolonifer* was maximum at 32 C (Jackson, 1965b). A saturated environment is not a prerequisite for infection. Once *Rhizopus* has become established in a peanut pod, decay may proceed even at low humidity.

Control. Seed infection with *Rhizopus* is minimized by proper harvesting and curing and by reducing injury. Deep burial of organic material helps to reduce activity of soil saprophytes in the fruiting zone. *Rhizopus* is usually seedborne (Frank, 1969; Gupta and Chohan, 1970b), so it can be controlled with seed treatment (Bell, 1966). Several fungicides, alone or in combination, reduced the isolation frequency of *R. stolonifer* from 54% to an average of 3% (Bell, 1966). However, in some areas *Rhizopus* is 1 of the fungi least affected by seed treatment (Mercer, 1978). Blends of fungicides may be superior to single fungicides (Bell, 1968). The beneficial effects of planting fungicide-treated seed, especially damaged seed and seed with low viability, in soil infested with *Rhizopus* was demonstrated by Bell (1968).

Bacterial Wilt

Bacterial wilt of peanut was first observed in the East Indies in 1905 (Breda de Hahn, 1906). The disease in the United States was first reported in 1912 (Fulton and Winston, 1914). This disease, also called slime disease, has now been found everywhere that peanuts are grown (Garren and Jackson, 1973; Weber, 1973). Although bacterial wilt is overall a disease of minor importance, occasional losses up to 40% have been reported (Simbwa-Bunnya, 1972).

Symptoms. Leaves on peanut plants infected with *Pseudomonas solanacearum* suddenly lose turgidity. Wilt symptoms usually are evident on a few scattered plants in a field but can sometimes affect plants over large areas (Simbwa-Bunnya, 1972). Usually, wilt symptoms occur on only 1 side of the plant. Leaflets turn light green, become chlorotic, and usually curl at the tips. Also, marginal necrosis of leaflets may develop. With time, leaflets turn brown but are usually not shed. Infected plants may have adventitious roots.

The roots of infected plants rot. The vascular system of the taproot becomes discolored, and bacteria can be isolated from the vascular tissue, which extends into the main stem and lateral branches. Pods usually remain free of bacteria but are shed readily (Breda de Hahn, 1906).

Causal Organism - *Pseudomonas solanacearum* (E. F. Sm.). Many scientific names have been applied to the pathogenic bacteria causing wilts of many hosts (Smith, 1939; Kelman, 1953). Among these names are: *Bacillus solanacearum, Phytomonas solanaceara, Bacillus nicotianae, Bacillus musae, Erwinia nicotianae, Xanthomonas solanacearum*, and *Phytomanas solanaceara*. The classification system of Bergey's Manual (Buchanan and Gibbons, 1974) gives *P. solanacearum* as the proper name for the bacterial wilt pathogen. A detailed description of the taxonomy and morphology of *P. solanacearum* was provided by Kelman (1953). Several races, strains and pathotypes of *P. solanacearum* have been identified (Buddenhagen and Kelman, 1964).

Disease Cycle and Epidemiology. *Pseudomonas solanacearum* is a soil-inhabiting bacterium that can persist for many years in some soil types. It is found throughout the world, especially in areas where high soil temperatures prevail. High soil moisture favors its pathogenicity. Penetration of peanut roots has not been thoroughly studied, but roots may become infected through insect and nematode wounds, lenticels, or rifts in the root cortex made by secondary roots (Kelman, 1953). After entry the bacteria quickly become localized in the water-conducting tissue. The unique relationship between host and pathogen existing from time of initial entrance of bacteria into the plant and the occurrence of wilt symptoms has been described (Buddenhagen and Kelman, 1964). The xylem tracheae of infected plants become filled with bacteria that will return to the soil at death of the plant or in debris left at harvest.

Isolates of *P. solanacearum* vary in virulence and in host specificity. Isolates from peanuts vary considerably in their ability to cause disease (Dukes et al., 1965). Race 3 of *P. solanacearum*, indigenous in soil of recently cleared land in Georgia, was highly virulent to several hosts but not to peanuts (Dukes et al., 1965). Several biotypes, some pathogenic and some not pathogenic to peanuts, were described in Uganda (Simbwa-Bunnya, 1972).

Control. Potential sources of resistance in peanuts to *P. solanacearum* have been identified. In Uganda, plant introductions (PI 341884, PI 341995 and PI 341886) obtained from the United States were immune to *P. solanacearum*. Schwarz 21, a peanut cultivar resistant to *P. solanacearum* (Winstead and Kelman, 1952) was susceptible to certain of its races (Jenkins et al., 1966). Other methods that provide partial control include crop rotation and use of pathogen-free seed stock (Garren and Jackson, 1973).

Yellow Mold, Aflaroot, and Aflatoxin

Aspergillus flavus and related species are ubiquitous throughout the peanut-growing areas of the world. The fungus thrives in the soil as a saprophyte, but peanut seed, seedlings, and pods are subject to attack (Gibson and Clinton, 1953). Yellow mold, a name coined by Jackson and Bell (1969), is a disease of ungerminated peanut seed and seedlings; this name was proposed to distinguish this disease from other, similar diseases such as Rhizopus seedling and seed rot, and crown rot caused by *A. niger*. Seedling infection with *A. flavus* is characterized by necrotic lesions on the emerging plumule and cotyledons. Seedlings sometimes die; however, the economic importance of *A. flavus* as a pre- and postemergence pathogen is low.

Aflaroot, a disease of peanut seedlings that is associated with the presence of toxins produced by *A. flavus* in the transpiration stream, has been reported in India (Chohan and Gupta, 1968; Aujla et al., 1974). Plant cotyledons are infected and overall plant growth is reduced.

Asperigillus flavus attacks peanuts as they mature in the soil and causes them to become moldy and sometimes contaminated with mycotoxins. Aflatoxin, a carcinogenic metabolite produced by *A. flavus* on moldy peanuts, has had a tremendous impact on the entire peanut industry and consuming public. This mycotoxin is discussed in Chapter 13.

Symptoms. Seed attacked by *A. flavus* become shriveled and decay quickly. Cotyledons of germinating seed are usually invaded first, and the emerging radicle and hypocotyl decay rapidly (Garren and Jackson, 1973). Cotyledons have necrotic spots, and infection loci are characterized by the presence of yellow-green spore masses. Plants infected with *A. flavus* have sparse roots and are chlorotic and temporarily stunted. A most characteristic symptom of infection in the field is vein-clearing of leaflets (Aujla et al., 1974). The radicle of surviving infected plants lacks secondary roots, a condition described by Chohan and Gupta (1968) as aflaroot. Normal plant growth usually resumes once the infected cotyledons are shed (El-Khadem, 1968). On maturing plants moldy peanut pods, characterized by the presence of yellow-green spore masses, are often observed during windrow drying, especially during periods of unfavorable drying conditions. Occasionally, moldy pods are observed in the soil, still attached to the plant.

Causal Organism - *Aspergillus flavus* Link ex Fries and *Aspergillus parasiticus* Speare. The taxonomy and morphology of the genus *Aspergillus* were described by Raper and Fennell (1965). Species delimitation is often difficult, since the taxonomic criteria of different species intermingle. Group species such as the *A. flavus* group are now used to designate 11 species of Aspergillus (Raper and Fennell, 1965), including *A. flavus* and *A. parasiticus*, the domi-

nant species associated with peanuts.

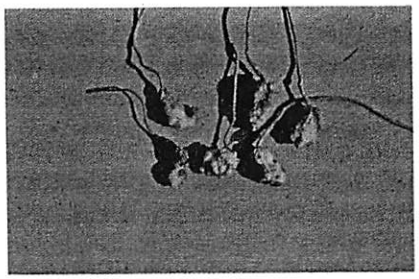
Disease Cycle and Epidemiology. *Aspergillus flavus,* characterized by the profuse production of yellow-green conidia and sclerotia, can thrive indefinitely in the soil and on debris as a saprophyte. This fungus is not known for its ability to invade intact, actively growing plant tissue. However, plants can be colonized when conditions are more favorable for the growth of *A. flavus* than for the growth of its competitors. Many environmental factors, including soil moisture, temperature, and relative humidity affect the epiphytology of *A. flavus* in peanut pods. Biological factors, including damage to pods and improper drying of pods increase the chances for contamination. Other factors affecting the growth and sporulation of *A. flavus* are listed by Garren and Jackson (1973). Soil populations of *A. flavus* range from less than 1 propagule per g of soil (Griffin and Garren, 1974) to 10^4 propagules per g of soil (Bell and Crawford, 1967; McDonald, 1969). Some peanut seed from mature pods are colonized by *A. flavus* before harvest. However, most contamination and subsequent accumulation of aflatoxin occur after peanuts are harvested and before they are dried. Occasionally, pods removed from the soil during droughty periods contain much visible *A. flavus* and sometimes aflatoxin.

Resistance of peanut seed to invasion by *A. flavus* has been reported (Mixon and Rogers, 1973; Tavasolian, 1977; Bartz et al., 1978; Kushalappa et al., 1979). Heritability of resistance is thought to be associated with seed coat thickness (La Prade and Bartz, 1972), smaller hila and a cellular arrangement of the palisade layer of the seed coat (Taber et al., 1973), wax accumulation on seed coat (La Prade et al., 1973), intact seed coats (Mixon and Rogers, 1975), thickness and structure of the peanut shell (Zambettakis, 1977), and presence of tannins (Sanders and Mixon, 1978).

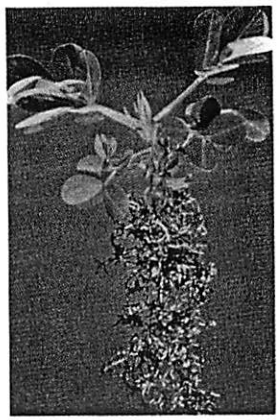
Control. Planting high quality seed treated with seed protectants minimizes in-soil rot of germinating seed. Harvesting procedures that minimize pod damage and proper drying of pods greatly reduce the chances of infection by *A. flavus.* Resistance of peanut seed to invasion by *A. flavus* has ben reported but has not yet been incorporated in agronomically acceptable cultivars. In the United States, the possibility of aflatoxin-contaminated peanuts reaching the consuming public is minimized by a strict marketing agreement supervised by the U. S. Department of Agriculture (Dickens, 1977). Fungicides such as captafol aid in suppressing aflaroot, but otherwise control for this disease has not been studied (Aujla et al., 1974).

DISEASES CAUSED BY NEMATODES

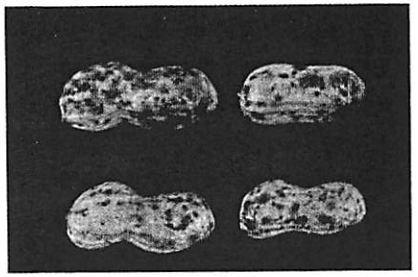
The peanut plant is subject to attack by a variety of plant parasitic nematodes. Yield losses caused by these parasites are such that in some areas of the world, cultivation of the crop cannot be maintained without nematode control. The principal species involved are in the genera *Meloidogyne, Pratylenchus, Belonolaimus,* and *Macroposthonia* (formerly *Criconemiodes*). Symptoms of nematode attack on peanuts result from a reduced and damaged root system, which deprives the plant of adequate nutrition. Affected plants lack vigor and have a reduced ability to withstand drought, obtain nutrients from the soil, and generally withstand adverse conditions. Plants are stunted, with unthrifty growth, yellowish foliage, and, in severe cases, typical nutrient-deficiency



A Root-knot

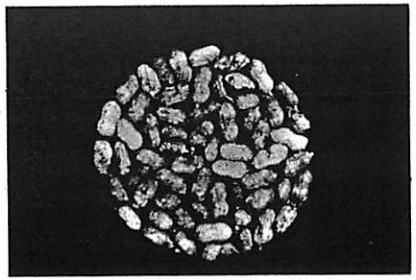


B Bushy roots



C Lesion of pods

D Ring nematode



E Pod lesion, Ring

F Sting nematode

Fig. 3. Symptoms of peanut diseases caused by nematodes. A. Severe galling caused by *Meloidogyne arenaria* on pods of Florunner peanuts; B. *M. hapla* galls on roots of young peanut plants; C. Shell lesions caused by *Pratylenchus brachyurus;* D. Root pruning caused by ring nematodes (*Macroposthonia ornata*), with healthy plant on left; E. Lesions on pods caused by *M. ornata;* and F. Reduced root system and characteristic necrotic spots on peanut root tissue caused by sting nematode (*Belonolaimus* spp.).

symptoms. In contrast to other diseases of peanuts, nematode damage does not cause aboveground symptoms that are distinct enough to permit identification of the responsible nematode species.

Root-Knot Nematodes

The root-knot nematodes (*Meloidogyne* spp.) are the most important nematode species limiting yields in peanuts. Because of the conspicuous "galls" and "warts" caused by these nematodes on the root system of the plant, the seriousness of the damage they caused was recognized early. Thus, in the southern United States, root-knot nematodes were first reported in Alabama in 1947 (Wilson, 1948), in North Carolina in 1949 (Cooper, 1950), and in Georgia in 1950 (Machmer, 1951). Recognition by Chitwood (1949) that root-knot nematodes were not 1 species (*Heterodera marioni*) but a number of species that could be separated morphologically permitted accurate studies on distribution patterns and the significance of the species in relation to diseases of peanuts.

The principal species of root-knot nematodes attacking peanuts are *M. arenaria* (Neal) Chitwood, the peanut root-knot nematode, and *M. hapla* Chitwood, the northern root-knot nematode (Taylor and Sasser, 1978). In addition, *M. javanica* (Treub) Chitwood has also been reported to attack peanuts (Martin, 1961; Minton et al., 1969a). The distribution of *M. arenaria, M. hapla,* and *M. javanica* is worldwide (Taylor and Sasser, 1978). Thus, *M. hapla* has been reported on peanuts in Israel (Minz, 1956), Australia (Alexander, 1963), Zimbabwe (Martin, 1958, 1961), and South Africa (van der Linde, 1956), and *M. arenaria* has been found in Israel (Orion and Cohn, 1975), Zimbabwe (Martin, 1958, 1961), Senegal (Netscher, 1975), the Mediterranean area and many other parts of the world (Taylor and Sasser, 1978). Taylor and Sasser (1978) state that "the part of the world between 35° S and 35° N latitudes is widely infested by 3 species of *Meloidogyne* adapted to continuous existence in warm countries, namely, *M. javanica, M. incognita,* and *M. arenaria.* North of 35° latitude in the northern hemisphere the most common *Meloidogyne* species is *M. hapla.*"

In the United States, the northern root-knot nematode (*M. hapla*) has traditionally been considered the important root-knot nematode of peanuts in the northern peanut-growing region (North Carolina and Virginia), while *M. arenaria* has been considered most important in the southern peanut-growing areas of Alabama, Florida, and Georgia (Taylor and Buhrer, 1958). However, this regional distribution is not absolute, and recent surveys suggest it may not be accurate. Motsinger et al. (1976) reported *M. hapla* as a serious pest in southern-grown peanuts. In Georgia 6.7% of the peanut fields contained *Meloidogyne* spp. and 69% of them were *M. hapla.* Similarly, in Alabama 40% of the soil samples examined contained root-knot nematodes; *M. hapla* is found frequently even though the predominant species is *M. arenaria* (Ingram and Rodriguez-Kabana, 1980).

Symptoms. Peanuts infected with *M. arenaria* commonly develop enlarged roots and pegs, which develop into galls of various sizes (Figure 3A). The galls result from an internal swelling of the root tissue; thus they can be distinguished from *Rhizobium* nodules, which are mostly appended laterally to the root. Galls may attain a diameter several times that of the adjacent root. Pods also become infected and develop knobs, protuberances, or small warts. Galls on roots, pegs, and pods sometimes begin to deteriorate by the time of peanut maturity. Development of the root system is commonly much reduced.

Symptoms of damage caused by *M. hapla* are similar to those caused by *M. arenaria.* Roots, pegs, and pods may all be galled, but individual galls are smaller than those caused by *M. arenaria.* Infected roots tend to form branches near the point of nematode invasion (Figure 3B). This frequently produces a dense, reticular (bushy) type of root system.

Infection and Spread. Peanut root-knot nematodes exist in the soil as egg masses, infective second-stage larvae, and adult males. The infective larvae emerge from the eggs, move freely through the soil, and penetrate suitable portions of the roots, pegs, or pods. After penetrating the plant tissue, the larvae lose their mobility and feed on adjacent plant cells. Under favorable conditions, larvae develop into enlarged mature females, which, when at maturity, produce large numbers of eggs in a gelatinous matrix (egg masses). Egg masses may remain in the roots or be extruded into the soil. As a result of the feeding, cells of the root, peg, or pods increase in size and number to form galls, knobs, or warts. The eggs hatch, and the new, second-stage larvae enter the soil surrounding the root; thus the life cycle is completed. The time required to complete the cycle depends primarily on soil temperature and moisture. Under the temperature and moisture conditions usually prevalent in peanut fields, 2 or more cycles occur during each season. Root-knot nematodes in a field can be distributed through their own movement but they are then very localized. However, crop debris containing galls may be widely spread by farming operations or running water.

Root-Lesion Nematodes

Root-lesion nematodes (*Pratylenchus* spp.) were first implicated as the causal agents of plant damage by Steiner (1945). He believed that these nematodes probably caused more crop loss than other nematode species and reported finding them in injured peanut roots in Holland, Virginia (Steiner, 1949), and also on peanuts growing near Fairhope, Alabama. Boyle (1950) found peanuts from several locations in Georgia with the characteristic pockmarks associated with damage by a *Pratylenchus* sp., which Steiner identified as *P. leiocephalus.* This species was changed to *P. brachyurus* (Godfrey) Filipjev and Schuurman-Stekhoven. *Pratylenchus* spp. are ubiquitous in the peanut-growing areas of the United States. They were in 37% of the soil samples in Minton's survey of Alabama peanut fields (Minton et al., 1963) and in 83.9% of the samples examined in a more recent survey of the state (Ingram and Rodriguez-Kabana, 1980). Alexander (1963) found that 2 out of 14 peanut fields surveyed in South Carolina were infested with these nematodes; Motsinger et al. (1976) reported them in 16.9% of Georgia's fields, and they are common in Florida and Texas (Boswell, 1968; Dickson and Waites, 1978), where severe damage has occurred.

Symptoms. Root-lesion nematodes attack pegs and pods as well as roots. Roots of infected plants are restricted in length and total volume, and tend to be discolored. A good description of the damage caused by *P. brachyurus* to pea-

nuts was given by Good et al. (1958), who found (Figure 3C) that "lesions on mature shells, pericarp, were purplish-brown and could be distinguished from soil microbial decomposition by their darker color and distinct boundaries, which did not fade gradually into healthy surrounding tissue, as with microbial decomposition." Miller and Duke (1961) described the damage caused by *P. brachyurus* as "small brown lesions on the fruit, giving it a speckled appearance if the lesions are numerous." Miller and Duke also found that fungi and bacteria attack dead tissue in the pegs and fruit and, under certain conditions, cause peg rot and seed decay. Heavily attacked plants were found to be slightly stunted, with an unthrifty yellow-green color and reduced root systems.

Because of the involvement of bacteria and fungi in the decomposition of root tissue invaded by *Pratylenchus* spp., for a number of years these nematodes were thought to cause damage providing "avenues of entrance" into plant roots for secondary invaders that caused necrosis of adjacent tissue and prevented recovery (Steiner, 1945). However, Boswell (1968) found significant correlations between numbers of *P. brachyurus* present and reduction of peanut yields. He noted also distinct differences between the lesions produced by *P. brachyurus* and those produced by *Rhizoctonia solani*, a fungal pathogen commonly associated with lesion nematode damage. According to Boswell (1968), lesions due to *R. solani* had definite margins, and the epidermal tissue was necrotic throughout the lesion, whereas the lesions caused by the nematode were characterized by their blotchy appearance and indistinct margins (a result of the light shell surface with the darker necrotic parenchyma showing underneath), which gave a diffuse appearance to the necrotic area. He found that lesions caused by the nematode began as pinpoint tan to light brown areas, on the surface, and the affected areas became larger and darker as the nematode fed and reproduced. Once the nematode penetrated through the epidermal tissue, it preferentially attacked the parenchymatous tissue between and around the network of vascular tissue.

Critical examination by Boswell (1968) of damaged shells from peanut fields showed that nematode-type lesions were present as well as lesions of *R. solani*. He also found other lesions with the appearance of *P. brachyurus* lesions but with a more general surface discoloration; these lesions contained both *P. brachyurus* and *R. solani*, the nematode being mostly in the margins of the lesions, with fungal hyphae predominant in the darker areas. The 2 organisms were not found within the same cells. *Pratylenchus brachyurus* was also found in association with the mycelia of other fungi, most often *Fusarium* and *Penicillium* spp.

Infection and Spread. Root-lesion nematodes are migratory endoparasites, and both adults and larvae can infect roots, pegs, and pods. The nematodes enter peanut tissues directly, and once inside they feed on parenchymatous tissue. An interesting and important property of *P. brachyurus* is its ability to survive extremes of temperature and moisture when protected within organic debris. This property permits the parasite to overwinter and provides an excellent means for dissemination (Graham, 1951; Good et al., 1958; Feldmesser and Rebois, 1965; Koen, 1967). This aspect of the life cycle of *P. brachyurus* explains reports that the nematode population decreases under peanut cultivation, when populations are estimated by soil extraction techniques (Sasser, 1951; Good et al., 1954; Johnson et al., 1974). Good et al. (1958) showed that when pegs and pods are used as the sources for extraction, the parasite is easily found and that numbers recovered from peanut shells are 6 to 8 times those in the root. Frequently several hundred nematodes can be dissected from a single shell lesion. These investigators also found large numbers of *P. brachyurus* in elongating and mature pegs.

Pegs are greatly weakened through the combined activities of root-lesion nematodes and secondary invading fungi. Consequently, when subjected to mechanical stresses, as in digging and shaking operation, the pegs often break, and the pods fall from the vine (Good et al., 1958). These nematodes remain alive throughout natural or artificial drying in winter storage. Furthermore, after shelling the shells continue to be an important nematode reservoir and means of spread. The protection afforded by the shell is such that ground shells used as diluents in certain preparations may transit the living nematode (Good et al., 1958).

Ring Nematodes

Ring nematodes in the genus *Macroposthonia* (formerly *Criconemoides*) have been associated with peanut damage since 1950. Machmer (1953) attributed yellowing of peanut plants to a ring nematode. *Macroposthonia ornata* (Raski) de Grisse and Loof is ubiquitous in the peanut-growing areas of the United States. Motsinger et al. (1976) found that 97% of the Georgia peanut fields contained this species and that often *Macroposthonia* was the only nematode genus present. Alexander (1963) found ring nematodes in 43% of samples from South Carolina fields, and in Alabama Minton et al. (1963) reported them in 64% of the fields; in a more recent survey of Alabama *Macroposthonia* spp. (mainly *M. ornata*) was found in 83% of the fields sampled (Ingram and Rodriguez-Kabana, 1980). Johnson et al. (1974), Kinloch (1974), and Kinloch and Lutrick (1975) have shown that peanuts support high populations of ring nematodes. These nematodes are exclusively ectoparasitic. Graham (1955) demonstrated in greenhouse experiments that ring nematodes caused a reduction of plant height and weight in spanish peanuts accompanied by considerable root decay. A study by Minton and Bell (1969) showed that *M. ornata* reproduced on Starr and Argentine peanut cultivars. The parasites were attached to all underground parts of the host (Figure 3D, E), causing lesions and discoloration, with yield reductions of 50%.

Sting Nematodes

Sting nematodes (*Belonolaimus gracilis* Steiner and *B. longicaudatus* Rau) have been known to be economically important in peanuts for some time. Holdeman (1955) reported that *B. longicaudatus* was associated with the plants in Virginia, North Carolina, South Carolina, and Georgia, and that peanuts sustained damage attributable to this nematode in Virginia and South Carolina. Minton and Hopper (1959) reported that *B. longicaudatus* was found in a single field in Alabama. Later (Minton et al., 1963), *Belonolaimus* spp. were found in 3% of Alabama peanut fields surveyed; however, Ingram and Rodriguez-Kabana (1980) failed to find the nematodes in peanut fields in the state in a 1979

survey. Alexander (1963) reported finding sting nematodes in 1 out of 14 peanut fields surveyed in South Carolina, but Motsinger et al. (1976) failed to find them in samples from Georgia peanut fields.

Results of greenhouse tests (Holdeman and Graham, 1953) have demonstrated that *B. gracilis* multiplies in valencia peanuts. The nematode causes roots to become gnarled and stubby, with the taproot frequently being the only root remaining (Owens, 1951). Feeding by *B. gracilis* causes tiny lesions along the taproot (Owens, 1951), and plants are chlorotic, with stubby, sparse root systems. Roots and pods have small, dark necrotic spots (Figure 3F) caused by the feeding of the nematode (Owens, 1951). The sting nematode, like the ring nematode and unlike the root-knot or root-lesion nematode is, for the most part, an ectoparasite; it is rarely found internally in roots or pods.

Although spanish peanuts have been reported to be poor hosts for *B. longicaudatus*, studies in North Carolina by Sasser et al. (1967, 1968) showed a significant correlation between numbers of *Belonolaimus* spp. and reduction in yield of spanish peanuts. More recently, Sasser et al. (1975) reported a higher correlation between peanut damage and numbers of *B. longicaudatus* than between damage and numbers of *M. ornata, M. hapla, Helicotylenchus dihystera* (Cobb) Sher, *Trichodorus christiei* Allen, or *P. brachyurus*. Soil texture is the limiting factor in the distribution of *B. longicaudatus* (Robbins and Baker, 1974). Miller (1972a) found that this nematode occurred characteristically in soils with sand contents of 84-94%. So dependent is this species on soil texture that Miller has proposed using the sand and water content of a soil to predict the survival of this nematode in any given field. Reports from other workers generally have confirmed his findings.

Other Nematodes

Several other species of nematodes have been reported to parasitize peanuts, but their importance is uncertain. The banana race of *Radopholus similis* (Cobb) Thorne, has been found (O'Bannon et al., 1971) to be more pathogenic to peanuts than the citrus race, but, despite the widespread occurrence of this species in tropical and subtropical areas of the world, it has not been recognized as causing a problem in peanuts. Germani (1970) recovered *Aphasmatylenchus straturatus* from the roots of peanuts in Upper Volta and later found it in roots of chlorotic peanuts (Germani, 1972).

The stubby root nematode, *Paratrichodorus (N) christiei* (Allen) Siddiqi, has been reported as a parasite of peanuts (Coursen et al., 1958); however, other studies have indicated that the parasite declines under peanut cultivation or fails to increase in peanut fields (Johnson et al., 1974; Kinloch and Lutrick, 1975).

Species of *Aphelenchus* and *Aphelenchoides* have been found feeding on peanut pods and roots (Lordello and Zamith, 1960) and are frequently associated with the root system of peanuts. Both of these genera, however, contain many mycophagous species, and the ones observed in association with peanuts might have been feeding on fungi in the rhizosphere or geocarposphere of the plant. An exception is *Aphelenchoides arachidis* (Bos, 1977), an endoparasite found in the testa of peanuts, which causes discoloration of seed, enhances the infection of seeds by fungi, and reduces seedling emergence (Bridge et al., 1977; McDonald et al., 1979).

Dagger nematodes (*Xiphinema* spp.) are found fairly consistently in peanuts and damage their roots. Schindler (1954) reported that galls and curly-tips developed on roots of peanuts growing in soil infested with *X. diversicaudatum* (Micoletzky) Thorne. Ingram and Rodriguez-Kabana (1980) found dagger nematodes in 19.6% of soils in a 2-year survey of peanut fields in Alabama. However, the numbers found were very low and did not change significantly throughout the growing season.

Plant parasitic nematodes in general, other than those described, have been reported to be associated with peanut roots, but the nature of the associations and their importance to crop yield have not been fully explored (Heyns, 1962; Colbran, 1964; Ali et al., 1969; Fortuner and Amougou, 1973; Germani and Luc, 1973; Germani, 1979). It is clear, however, that such nematodes are not widespread in peanut fields and are probably of little economic importance on a worldwide basis.

Population Dynamics

Relatively little information is available on population dynamics of plant parasitic nematodes in peanut fields. The recent survey of fields by Ingram and Rodriguez-Kabana (1980) provides information pertaining to Alabama. Peak populations of species of *Macroposthonia, Pratylenchus,* and *Meloidogyne* increased through the season (April-September); the numbers were highest in late July to September and lowest between January and early June. These results support those of Kinloch (1974) and Johnson et al. (1974), which showed that numbers of *M. ornata* increased with peanut crop development in Florida and Georgia, respectively, with peak populations in July. Also, Good et al. (1954) and Johnson et al. (1974) demonstrated population increases of *Pratylenchus* spp. in peanuts through the growing season.

A study of the vertical distribution of plant parasitic nematodes in Alabama peanut fields (Ingram and Rodriguez-Kabana, 1980) revealed that numbers of aphelenchoid nematodes, *Macroposthonia* spp. and *Pratylenchus* spp. during the growing season were generally either highest in the 0-15 cm depth or similar in the 0-15 cm and 15-30 cm sections of the soil profile. Significant date times depth interactions indicated that vertical distribution for these nematodes may depend on seasonal weather conditions. Data on the distribution of *Meloidogyne* spp. showed that numbers of these nematodes, when determined by bioassay of tomato cultivar Rutgers were not significantly different at the 2 depths, but when determined by soil extraction (flotation-sieving method) they reflected a significant date times depth interaction, which indicates variation according to seasonal weather conditions. The study showed significant numbers of plant parasitic nematodes to be present throughout the year at the 15-30 cm depth. In a recent study in Florida, Garcia (1976) found that gelatinous egg masses of *M. arenaria* were present at soil depths of 0-75 cm from August (3 months after planting of peanuts) through October. Egg masses could be recovered from November (1 month after harvest) through July but since none contained viable eggs, *M. arenaria* probably overwinters as second-stage larvae. Garcia (1976) also reported that although larvae were found throughout the year at all sampling depths studied (0-75 cm), numbers at soil depths of 0-15 cm and 15-30 cm decreased rapidly beginning 2 months after peanut harvest. At deeper

levels in the soil, numbers of larvae also decreased, but less rapidly than in the more superficial layers. These findings tend to support Potter's (1967) view that populations located at the lower depths may be overwintering populations. Nematodes located at these lower depths could escape control efforts and serve as sources of infestation throughout the season and possibly in the succeeding season. The numerous date times depth interactions found by Ingram and Rodriguez-Kabana (1980) do not suggest a general trend of population movement to lower depths.

Sampling And Detection

The correct sampling time for nematode analyses is critical for accurate diagnosis and for recommending control measures to farmers. In this respect, a knowledge of the population dynamics of nematode species in any field is fundamental. As noted earlier, studies on population dynamics in peanut fields are limited. The available information (Good et al., 1954; Johnson et al., 1974; Kinloch, 1974; Ingram and Rodriguez-Kabana, 1980) indicates that numbers of plant parasitic nematodes in peanut fields in the southeastern United States are highest between July and September. Therefore, the probability of detecting any nematode species will be greater if samples are taken during this period. This practice, while not helpful for the current season, permits the establishment of expected levels of infestation for the following season. Samples collected during the winter or early spring invariably contain very low numbers of plant parasitic nematodes and require some form of bioassay to establish the level of infestation for root-knot or lesion nematodes (Boswell, 1968; Ingram and Rodriguez-Kabana, 1980).

A number of soil extraction methods are available for determining nematode numbers in soil (Carter, 1945; Southey, 1970). These methods are satisfactory for evaluating nematode levels during the growing season. However, when nematode numbers are low, bioassay procedures are more suitable (Ingram and Rodriguez-Kabana, 1980). For migratory endoparasites such as the lesion nematodes, an estimate of the soil population may reflect only a small portion of the population present in a field. Ingram and Rodriguez-Kabana (1980) found that for lesion nematodes a corn bioassay was the most sensitive method. Similarly, because of the relatively low number of larvae of *Meloidogyne* spp. found in the off-season in peanut fields, bioassay with plants of tomato cultivar Rutgers was the most sensitive method for detecting them.

As indicated by the work of Fox and Phipps (1980), perhaps the most accurate prediction on nematode numbers is one based on a good knowledge of the crop history of fields. Maintenance of good records can help to identify problem fields and avoid the indiscriminate use of nematicides or costly rotations.

Interactions between Nematodes and Other Peanut Pathogens

Several studies have indicated interactions between nematodes and other peanut pathogens. The presence of *P. brachyurus* in peanut fields increased the occurrence of *Aspergillus flavus* in seed (Jackson and Minton, 1968). Minton et al. (1969b) and Minton and Jackson (1969) found a significantly greater incidence and density of *A. flavus* in peanut seed inoculated with both the fungus

and *M. hapla*, but also reported no significant differences in incidence and density of *A. flavus* in shells or in the total incidence of all fungal propagules (*A. flavus* and other fungi) in shells and seed. A study on the possible interaction between *A. flavus* and *M. arenaria* in Argentine peanuts (Bell et al., 1971) showed that pods from nematode-inoculated plants were heavily galled, but the incidence of infection by *A. flavus* and other fungi was not affected. They concluded that *M. arenaria* damage to peanut pods did not affect incidence of *A. flavus* infection.

Garcia and Mitchell (1975c) studied the interactions of *Pythium myriotylum* and *M. arenaria* in preemergence damping-off and pod rot of peanuts. Their results showed a synergistic interaction of *P. myriotylum* with *M. arenaria*.

Boswell (1968) conducted critical inoculation experiments to determine the significance of the suspected (Steiner, 1945; Ashworth et al., 1961) interaction between *P. brachyurus* and *R. solani*. He showed that the 2 organisms could produce morphologically distinguishable lesions on peanut shells but were never found within the same cell. In field material, *P. brachyurus* and *R. solani* were sometimes found within the same lesion, but, as in greenhouse material, the 2 microorganisms were not found in the same cell. The author noted that *P. brachyurus* in field samples was found in association with mycelium of other fungi, most often species of *Fusarium* and *Penicillium*.

An interaction between *M. ornata*, *M. hapla*, and *Cylindrocladium crotalariae* (causal agent of black rot of peanuts CBR) has been suggested by Diomande and Beute (1979). Using 2 peanut cultivars, NC 3033 (CBR resistant) and Florigiant (CBR susceptible), they found in greenhouse experiments that CBR severity increased in the presence of *M. hapla* on both cultivars and that *M. ornata* could increase the disease syndrome on Florigiant but not on NC 3033.

Sclerotium rolfsii and root-knot nematodes may interact in peanut fields where both pathogens occur at sufficiently high levels of infestation. Yields were higher when combinations of fungicides and appropriate nematicides were applied to soil than when fungicides were used alone (Rodriguez-Kabana et al., 1977a). Work by Beute and Rodriguez-Kabana (1979a) revealed that dormant sclerotia of *S. rolfsii* can be "triggered" to germinate by volatile compounds emanating from dead or dying peanut tissues. This type of vegetable material may be more available in fields heavily infested with root-knot nematodes or other plant parasitic nematodes than in fields without the nematodes. The suspected interaction then would be indirect but dependent on nematode damage to the plant.

An interaction between a virus and nematodes has been reported. Merny and Mauboussin (1974) demonstrated that fumigating with DD (mixture of chlorinated C_3 hydrocarbons including 1, 3-dichloropropene, 1,2-dichloropropane and other related hydrocarbons) prevented clump in peanuts, a viral disease, and also eliminated the nematodes. They suggested that possibly more than 1 species of nematodes was acting as vector of the virus and that particular attention should be paid to *Longidorus siddiqii*.

Nematode Control

Nonchemical Control. Ideally, control of nematodes in any crop should be based primarily on the use of resistant varieties alone or in combination with

proper rotation crops and with cultural techniques that reduce infestation of soil to an economically tolerable level. A great deal of effort has been expended by researchers in attempts to identify sources of nematode resistance in peanuts. Edwards (1956) reported the cultivars Natal Common and Kumawu Erect to be highly resistant to root-knot nematodes. Castillo et al. (1973) found 8 of 235 lines of *A. hypogaea* only moderately susceptible to *M. hapla*, and 4 of 12 accessions of wild *Arachis* species exhibited resistance. However, the search for resistance to *M. arenaria* has been unsuccessful. Although Miller and Duke (1961) reported that a peanut of "a foreign introduction with a purple skin" was resistant to *M. arenaria*, Miller (1972b) later reported no resistance to the nematode in 2,000 peanut introductions in field plots in Virginia. More recently, Minton and Hammons (1975), using severity of galling as an indicator of resistance, corroborated Miller's findings; they tested 512 entries, including cultivars, breeding lines, and plant introductions, and found no resistance to *M. arenaria*. However, peanut responses to *M. arenaria* or indeed to other species of *Meloidogyne* may vary according to the races or even isolates of the species tested (Sasser, 1966; Kirby et al., 1975; Taylor and Sasser, 1978). Minton et al. (1969a) found a Georgia isolate of *M. javanica* capable of parasitizing peanuts; *M. javanica* was not parasitic on peanuts in Sasser's (1954) original scheme of differential host responses for separation of the species of *Meloidogyne*. In addition, Minton (1963) reported significant differences in infectivity and pathogenicity to peanuts between morphologically identical populations of *M. arenaria*. Also, Netscher (1975), working with Senegal isolates of *M. arenaria* and *M. javanica*, found 7 peanut cultivars that exhibited a high degree of resistance to these nematodes. At present no commercially available peanut cultivar in the United States has any significant level of resistance to *Meloidogyne* spp. Clearly, further work is needed to find sources of resistance and to characterize the responses of existing commercial cultivars to races and isolates of root-knot nematodes.

Relatively little information is available on resistance of peanuts to lesion nematodes. Boyle (1950) reported that lesions were less obvious on heavy shelled virginia type than in spanish type peanut pods. Minton et al. (1970) found that lesions caused by *P. brachyurus* were not as conspicuous on pods of Virginia Bunch 67 and Georgia 186-28 as on Florigiant, Early Runner, Argentine, and Starr cultivars; shell tissues of Virginia Bunch 67 and Georgia 186-28 were less sensitive to the nematodes, to microbial degradation that followed nematode invasion, or to both. Smith et al. (1978) recently found that 2 peanut introductions, PI 295233 and PI 290606, had signficantly less pod discoloration and lower numbers of *P. brachyurus* from shell extractions than the commercial spanish cultivars Starr and Spancross or Florunner peanuts. There is thus some evidence to suggest the existence of sources of resistance to *P. brachyurus* in peanuts. Because of the economic significance of this nematode and its widespread distribution, efforts to incorporate resistance in commerical cultivars should be increased. Testing for resistance to lesion nematodes in peanuts has been very limited and is not comparable to the effort devoted to determining sources of resistance to *Meloidogyne* spp.

Research on resistance to other plant parasitic nematodes is lacking. A report by Miller (1972b) indicated no resistance in peanuts to *B. longicaudatus*.

Rotations. Rotation of peanuts with other crops can significantly decrease

levels of infestation with plant parasitic nematodes in soils. This concept was explored as early at 1952 by Cooper (1952) in a North Carolina field infested with *M. hapla*. He used a split block design in which soybeans, corn, cotton, and peanuts were planted in both fumigated and nonfumigated plots; the entire area was planted to peanuts the next year. The study showed that the preceding crop other than peanut reduced nematode infestation and increased yields in both fumigated and nonfumigated blocks. Also, within rotation treatments, rootknot indices were lower and yields higher in the fumigated than in the nonfumigated blocks. Results from a study with spanish peanuts in Texas (Thames and Langley, 1967) indicated that *M. arenaria* could be controlled (based on dollar returns per hectare) by rotation with *Sorghum vulgare* cultivar Early Hegari. More recent work by Fox and Phipps (1980) analyzed results obtained with a predictive nematode assay program in Virginia. The analysis indicated rotation effects on nematicide recommendations in 11, 29, and 49% of the 1980 peanut fields without potential nematode problems after the 1979 culture of peanut, soybean, and corn, respectively. The data also indicated that the culture of peanut resulted in higher populations of root-knot and ring nematodes than the culture of soybean or corn.

Chemical Control. The lack of resistance to plant parasitic nematodes in commercial peanut cultivars has made control of the parasites dependent on the routine use of nematicides. Two types of nematicides are widely used in peanuts: fumigants and nonfumigants (those with contact or systemic properties).

The earliest and principal nematicides used in peanuts were formulations containing the fumigants 1, 3-dichloropropenes (DD, 1, 3-D), 1, 2-dibromoethane (ethylene dibromide, EDB), and 1, 2-dibromo-3-chloropropane (DBCP). A mixture of 1, 3-dichloropropene, 1, 2-dichloropropane and other hydrocarbons (DD) was the first nematicide (Carter, 1943, 1945). Among the first reports on the use of dichloropropenes in peanuts was that of Cooper (1952), who found them effective alone and in combination with chloropicrin (trichloronitromethane) for control of *M. hapla* in North Carolina. In various formulations, these materials have been used for control of root-knot, lesion, and other nematodes in peanuts (Miller and Duke, 1961; Thames and Langley, 1964; Boswell, 1968; Dickson and Mitchell, 1974). Because of their intrinsic phytotoxicity to peanuts, dichloropropenes must be injected into soil before planting. This requirement, together with the relatively high dosages (40 to 100 L/ha) required for effective control, limited their widespread use in peanuts. This limitation became particularly evident when other fumigant nematicides were developed that were effective at lower dosage and were not phytotoxic to peanuts when injected at planting (ethylene dibromide and DBCP). Recently, a new formulation containing 92% 1, 3-dichloropropenes (Telone II) has become available, but it has not been any more effective than the old DD formulation (Rodriguez-Kabana, unpublished data).

McBeth and Bergeson (1955) described a new nematicide, DBCP, with a lower vapor pressure and considerably more effective than DD. On peanuts the new fumigant was effective at dosages of 7 to 14 L/ha and was nonphytotoxic when injected into the soil at planting time (Good and Steele, 1959). Because of its effectiveness and relatively low cost, DBCP became the standard nematicide for use on peanuts, and considerable research was devoted to determining

the best ways to use it. Good and Steele (1959) in Georgia applied DBCP as a liquid injected into the soil. They found this form of application to be more effective than broadcast treatments with a 10% granular formulation. Also, they reported that preplant row applications of DBCP were superior to broadcast and postemergence applications. Rodriguez-Kabana et al. (1979b) reported on other methods of application for DBCP.

In the United States and throughout the world, DBCP is effective against a wide range of plant parasitic nematodes in peanuts (Boswell, 1968; Dickson and Mitchell, 1974; Minton and Morgan, 1974; Germani, 1979; Rodriguez-Kabana et al., 1979b). In addition to its nematicidal properties, DBCP is fungicidal against soilborne pathogens (Rodriguez-Kabana and Curl, 1980), notably *Pythium ultimum* (Brodie, 1961) and, more important for peanuts, *R. solani* (Ashworth et al., 1964a). As indicated by Boswell (1968) some of the beneficial effects of applying DBCP to peanuts were not due entirely to control of nematodes, particularly in fields with pod rot complexes. There is also good evidence that DBCP enhances development of endomycorrhizal fungi and does not affect nodulation by *Rhizobium* spp. (Walker et al., 1976; Germani, 1979). A negative aspect of the use of DBCP on peanuts was revealed recently (Rodriguez-Kabana et al., 1979a); the fumigant increased the incidence of stem rot caused by *Sclerotium rolfsii* and stimulated germination of dormant sclerotia of *S. rolfsii*. Even more serious were DBCP's reported inhibitory effect on production of human sperm and its suspected carcinogenicity (Anonymous, 1978). These findings resulted in its elimination from use in peanut fields in the United States.

Elimination of the use of DBCP in peanuts prompted a search for alternative fumigant nematicides with comparable efficacy and cost. Ethylene dibromide and combinations of ethylene dibromide plus chloropicrin injected at planting were found to be almost as effective as DBCP for control of *M. arenaria* in Alabama (Rodriguez-Kabana et al., 1979c). Maximal yield responses in infested fields were obtained with rates in the range of 14 to 19 L/ha, somewhat higher than those required with DBCP. Formulations of ethylene dibromide containing chloropicrin offered no advantage over those with ethylene dibromide alone. Planting-time applications of ethylene dibromide were superior to midbloom applications of the fumigant, both for control of root-knot nematodes and for maximum peanut yield. In very sandy soils, at-plant or preplant applications of DBCP or ethylene dibromide were less effective in controlling *M. arenaria* in peanuts than combination treatments of at-plant and at-pegging applications of systemic nematicides (Dickson and Waites, 1978). In heavier soils, however, the use of the complementary postemergence treatment did not significantly improve yield (Minton and Bell, 1981).

A number of fumigant nematicides such as dazomet (tetrahydro-3,5-dimethyl-2H-1,3,5-thiadiazine-2-thione), metham-sodium, and carbon disulfide are available for use in peanuts but have not been studied adequately; as with the dichloropropenes, use of these nematicides requires a waiting period after application because of their inherent phytotoxicity (Rodriguez-Kabana et al., 1977b). Recently, metham-sodium was used in Israel in through-the-line application in irrigated fields to control nematodes and pod rot organisms (Krikun et al., 1981). Because of its convenience, this form of application should be further explored, particularly with materials of low mammalian toxicity or in situations with only low risk of contamination from runoff water of poisoning of animals (Rodriguez-Kabana et al., 1977b).

Beginning in the late 1950's a series of nonfumigant compounds were introduced that had nematicidal and insecticidal properties. Some, such as fensulfothion [0,0-diethyl 0-4-(methyl sulfinyl)phenyl phosphorothioate] and ethoprop (0-ethyl S,S-dipropyl phosphorodithioate) were contact nematicides with no significant systemic properties; while others, such as aldicarb [2-methyl-2-(methylthio) propionaldehyde O-(methylcarbamoyl)oxime], carbofuran (2,3-dihydro-2,2-dimethyl-7-benzofuranyl methylcarbamate), oxamyl (methyl N', N'-dimethyl-N-[(methylcarbamoyl)oxy]-1-thiooxamimidate), and phenamiphos[ethyl 4-(methylthio)-*m*-tolyl isopropylphosphoramidate], combined direct (contact) toxicity to nematodes and systemic absorption in significant quantities by plants while retaining their nematicidal properties, either as the parent compound or as some metabolite in the plants. Because of their dual role as nematicides and insecticides, these compounds have been the subject of numerous comparative studies with traditional fumigant nematicides that have low insecticidal activity. A good evaluation of the dual role of these nematicides in peanuts was provided by Minton and Morgan (1974) from several years of field experiments in Georgia with Starr, Florigiant, and Florunner peanuts. The effectiveness of aldicarb, carbofuran, ethoprop, fensulfothion, phenamiphos, and oxamyl was determined against thrips (*Frankliniella* spp.), the lesser cornstalk borer [*Elasmopalpus lignosellus* (Zeller)], leafhoppers (*Empoasca* spp.). the corn earworm [*Heliothis zea* (Boddie)], and the rednecked peanut worm [*Stegasta bosqueela* (Chambers)], and against lesion (*P. brachyurus*), ring (*Macroposthonia* spp.), and northern root-knot (*Meloidogyne hapla*) nematodes. Minton and Morgan (1974) found that carbofuran and aldicarb controlled thrips, but fensulfothion, phenamiphos, and oxamyl gave a lower level of control or less consistent control. Damage by the lesser cornstalk borer was not reduced by any treatment. Carbofuran, unlike any other compound, completely controlled leafhoppers in 1 experiment. None of the nematicides was effective against the corn earworm and the rednecked peanut worm. All chemicals provided some measure of nematode control. It was noted that significant nematode control usually increased yields; although thrips were controlled the increase in yield was attributable to nematode control. This conclusion was substantiated by the finding that yields from plots treated with DBCP, which does not control insects, were usually among the highest. Perhaps because effective insect control did not always translate into significant yield increases, DBCP, a cheap nematicide, remained the preeminent nematicide for use in peanuts for almost 2 decades.

The experiments of Minton and Bell (1981) and Minton and Morgan (1974) and other workers (Sasser et al., 1966; Rodriguez-Kabana et al., 1977a, 1980; Rodriguez-Kabana and King, 1979) showed that peanut yields with nonfumigant nematicides can be equivalent to those obtained with fumigant nematicides such as DBCP and ethylene dibromide at similar broadcast dosages of 4 to 7 kg of active ingredient per ha.

Recently, because of the toxicological considerations mentioned earlier, DBCP and other halogenated hydrocarbon nematicides either have been eliminated from use or are under review by the U. S. Environmental Protection

Agency (EPA). The possibility of losing these fumigants for use in peanuts has renewed interest in the use of nonfumigant contact and systemic nematicides. Nonfumigant nematicides offer advantages over traditional fumigants in that they do not need to be injected into soil, so planting problems associated with the use of injectors are eliminated (Rodriguez-Kabana et al., 1977b). Most nonfumigant nematicides are available in granular and emulsifiable formulations (Rodriguez-Kabana et al., 1977b; Hammond and Rodriguez-Kabana, 1978). These formulations allow for easy application either by spraying or with granular applicators mounted on the planter equipment. However, the best way of using these materials, particularly those with systemic properties is not known. This lack of knowledge was emphasized recently by the finding that the traditional methods of thorough incorporation into the soil used for some systemic nematicides offered no advantage and could indeed reduce the effectiveness of aldicarb, phenamiphos, and oxamyl against *M. arenaria* (Rodriguez-Kabana and King, 1979). Further, some nematicides, such as oxamyl, are effective against *M. arenaria* when applied in liquid formulation to a depth of 10 cm in the seed furrow (Rodriguez-Kabana et al., 1980). The subject of application techniques and correct time for delivery of nonfumigant systemic nematicides to achieve maximal effectiveness is 1 on which information is severely lacking.

There are relatively few reports on nontarget effects of nonfumigant contact and systemic nematicides. Carbofuran has been shown to exert a temporary inhibitory effect on endomycorrhizal fungi in Florunner peanuts (Backman and Clark, 1977). Ethoprop and fensulfothion show activity against *S. rolfsii* and other soilborne fungal pathogens. Rodriguez-Kabana et al. (1976a, 1976b) found that ethoprop inhibited growth of *S. rolfsii* and *R. solani* on potato dextrose agar (PDA) but did not significantly affect growth of *Trichoderma*, a genus antagonistic to the pathogens. Ethoprop also inhibited growth of *R. solani* and *S. rolfsii* in soil and enhanced invasion of *S. rolfsii* colonies by *Trichoderma* spp. The activity of ethoprop against *S. rolfsii* was equivalent to that of the standard soil fungicide PCNB, and field applications of ethoprop to peanuts at blooming time consistently reduced damage attributable to *S. rolfsii*. Fensulfothion was also found (Sasser, 1951; Rodriguez-Kabana et al., 1976b) to inhibit growth of both *S. rolfsii* and *R. solani* on PDA, but, in contrast to ethoprop, it did not stop mycelial development of *S. rolfsii* in soil and only reduced the production of sclerotial initials by the fungus. Fensulfothion did not affect the rate of development of *Trichoderma* spp. on colonies of *S. rolfsii*. Field studies with peanuts revealed that fensulfothion applied at blooming time reduced damage by *S. rolfsii* during the early part of the season, but this reduction was not apparent at harvest. In Georgia, Thompson (1978) also reported that fensulfothion reduced the incidence of damage by *S. rolfsii* in peanut fields. These findings on the effects of ethoprop and fensulfothion on *S. rolfsii* have permitted the development of formulations combining PCNB with the nematicides to assure consistent control of stem rot in peanuts. Although it was used for a number of years in Georgia and Alabama for control of *S. rolfsii*, PCNB had not given satisfactory yield responses, in spite of its effectiveness against the pathogen. Use of PCNB increased populations of *Pratylenchus* and other parasitic nematode species (Boswell, 1968; Adams et al., 1979), so that the yield responses expected from control of *S. rolfsii* were offset by damage from the enhanced nematode populations. The combination of PCNB with fensulfothion or ethoprop has resulted in consistent control of stem rot and parasitic nematodes and has significantly increased yields.

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Chapter 11

GROWTH PHYSIOLOGY

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Great progress has been made in acquiring knowledge of the physiology and biochemistry of plants. This basic information, along with that from other disciplines such as soil science, plant pathology, genetics, and entomology, provides a basis for improved crop management. Increased yields and better quality are the goals of agriculturists, but these are the complex end-products of a series of biological processes and reactions.

Until recently very little effort has been devoted to peanut physiology investigations. The effort has increased, but information on peanuts is deficient in comparison with other crops. However, progress is being made, and recent research developments will be discussed from the basic view to indicate their implications for improved peanut cultivars and crop management where applicable.

Major topics in this chapter are germination and seedling growth, photosynthesis and growth analysis, growth regulators, environmental factors, nitrogen fixation, and tissue culture. Also, some specific environmental effects on physiological processes discussed in this chapter will be included in their respective sections. Mineral nutrition was reviewed by Reid and Cox (1973) and in Chapter 6 of this volume. Early work on environmental factors was reviewed briefly by Gregory et al. (1973).

GERMINATION AND SEEDLING GROWTH

Life cycles in the plant kingdom begin and end with seed. Seed are the "thread of life" connecting successive plant generations. Continuity between generations is tenuous since seed are easily damaged physically and physiologically by unfavorable conditions during or after seed maturation. Gregory et al. (1973) previously reviewed peanut seed and seedling morphology. Only a brief description is given here. Reed (1924) described the mature peanut seed as a straight embryo, consisting of 2 fleshy cotyledons, a short hypocotyl, and a plumule all enclosed by a thin testa. Examination of the plumule by Yarbrough (1949) revealed a main axis and 2 cotyledonary lateral axes. He indicated that the mature seed contained 9 or more embryonic leaves on the main and lateral axes. In some recent research (Maeda, 1970, 1972, 1973) fewer leaf primordia were found in the embryo, indicating possible genotype differences in this characteristic of peanut seed. A dominant feature of peanut seed is the protruding tip of the hypocotyl-radicle axis. This protruding and relatively unprotected radicle is a major site of injury during harvesting and handling that may predispose the seed to subsequent physiological deterioration. Peanut seed are among the world's most delicate seed to handle in commerce.