Chapter 13

Peanut Diseases

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Introduction

The chapter on diseases in the original monograph on peanuts (91) noted that until the 1930's there was little interest in, or work on, diseases of peanuts in the United States. Peanuts became a priority crop during World War II and this changed the emphasis in research on peanut diseases. Instead of a concentrated study of a few diseases the war years saw much surveying to determine which diseases limit peanut production in the United States. At present (1970) peanut disease research is again concentrated on a few most important diseases. But the situation which prevailed in the late 1960's was different from that of the late 1930's. Perhaps there are now 10 persons engaged in peanut disease research in the United States for every one person engaged in this field of research 30 years ago.

The aflatoxin problem which came to the forefront in the mid-1960's is largely responsible for this great increase in research personnel assigned to peanut diseases. However, aflatoxin deserves, and has, a chapter of its own in this monograph. Likewise, physiological diseases of peanuts, such as mineral deficiencies, are more appropriately treated elsewhere in this monograph.

One presumes that the peanut disease picture has changed considerably since 1951 when the first peanut monograph was published (91). A review of the literature shows

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this is not true unless the aflatoxin problem is falsely considered a "peanut disease." With the possible exception of stem rot caused by *Sclerotium rolfsii* Sacc. (77), all peanut diseases which were of major importance in 1950 remain so in 1970. From the worldwide viewpoint leafspot still takes its toll; and, in limited areas, rosette, bacterial wilt, etc. still take their toll. Fortunately, peanut rust (36) has shown no tendency to increase in importance. Of the newly recognized peanut diseases studied since 1951, a few, such as pod rot or pod breakdown (83) and aspergillus crown rot (123), are important in some areas. However, and again fortunately, the peanut stunt virus disease (51) and many other "new" peanut disorders have not yet justified much concern.

Recently Jackson and Bell prepared and published a technical handbook on diseases of peanuts caused by fungi (138). Their literature reviews began where those of the chapter on diseases (91) in the first monograph stopped. The sections herein on diseases caused by fungi are condensed and updated from the Jackson and Bell handbook. The handbook was published in 1969, thus these sections required little in the way of updating. Readers of this revised monograph can find a more technical discussion and a more thorough review of literature on peanut diseases caused by fungi in the handbook. The sections herein on virus and nematode diseases required much updating from those of the 1951 monograph (91). Practically speaking, these are new sections.

When a publication is the brainchild of two or more persons, someone has to be listed as the first author. The author named first above prepared this introduction, therefore it is his privilege to point out that Jackson is as much the senior author as is Garren. This chapter has two senior authors.

CERCOSPORA LEAFSPOTS

Cercospora arachidicola Hori and *Cercospora personata* (Perk. & Curt.) Ellis & Everhart

Cercospora leafspots (tikka, viruela, peanut Cercosporosis, Mycosphaerella leafspots, brown leaf spot, "leaf spot") are probably the most serious diseases of peanuts on a worldwide scale. Economic losses from cercospora leafspots are estimated to be from 15 to 50% of the yield in many areas of the world. In the United States, where chemical control measures are generally used, the average annual loss for 1951 to 1960 is estimated at 10% (262). Distribution of cercospora leafspots is shown in Commonwealth Mycological Institute (CMI) maps 152 and 166. Members of the genus *Arachis* are the only commonly reported hosts for the two pathogens.

Symptoms

Infection of leaflets by *C. arachidicola* may be noted first as small chlototic spots which enlarge and become brown to black, subcircular, 1 to 10mm or more in diameter. A chlorotic halo surrounding each lesion has been reported (141, 278) to be a characteristic feature, but a halo is not always present. When present, halos are more distinct on the adaxial leaflet surface. Sporulation is at first epiphyllous, and later amphigenous. During prolonged periods of rainfall coalescense of lesions is common. Petioles and stems are commonly attacked and exhibit dark, elongate lesions with indistinct margins and a somewhat superficial appearance.

C. personata (Cercosporidium personatum (Beck. and Curtis) Deighton) infection initiates symptom development that is similar to that of *C. arachidicola.* Differences noted by Jenkins (141) were that halo formation was limited to the adaxial surface

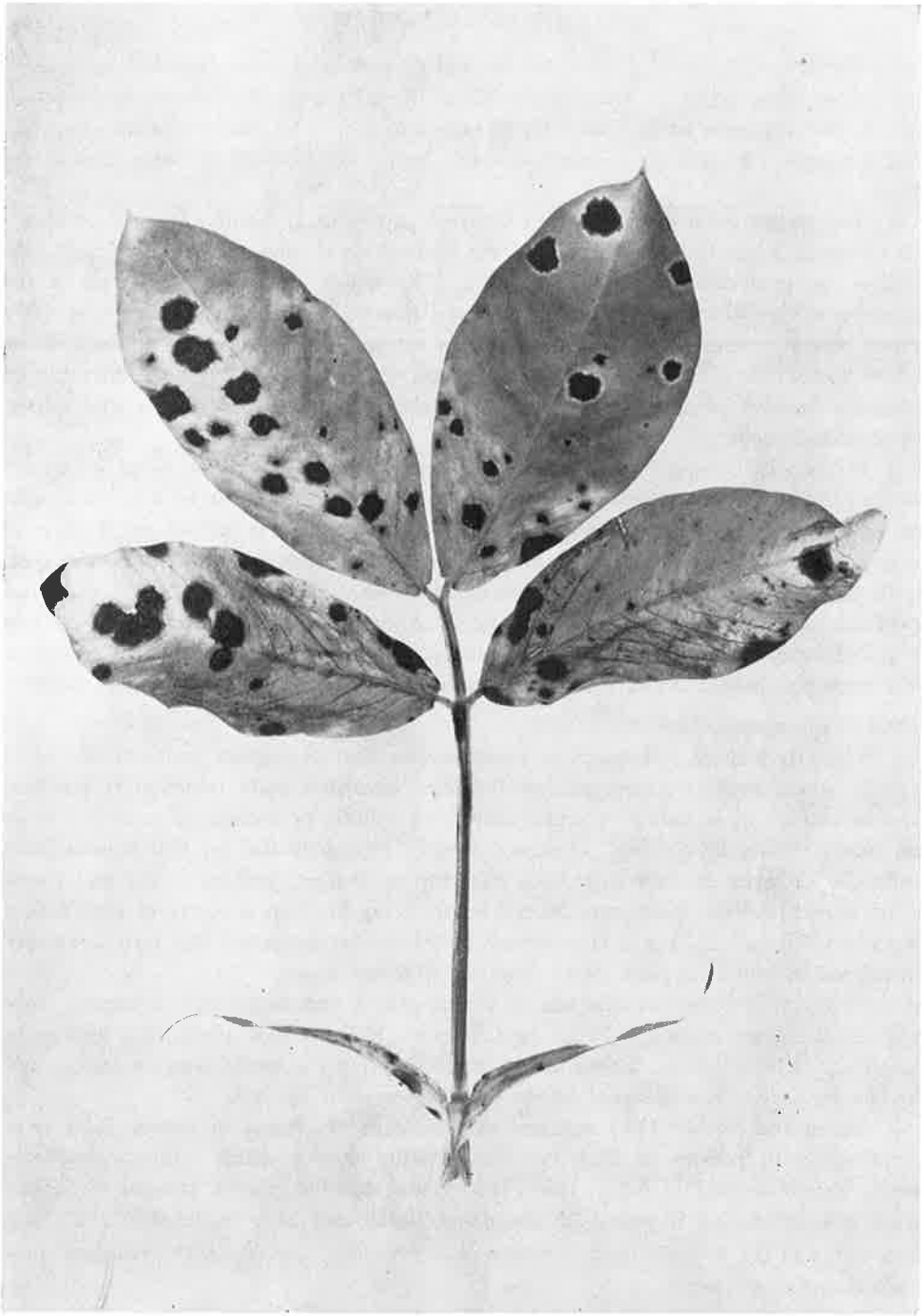

Figure 1. The leafspot caused by *Cercospora arachidicola.*

around mature spots, lesions were darker, sporulation was predominantly hypophyllous at first and later amphigenous, with fruiting structures often arranged in concentric circles, and with the stromatic tufts visibly raised above the lesion surface.

CAUSAL ORGANISMS

The sexual stages of *C. arachidicola* and *C. personata* were described by Jenkins (142) as *Mycosphaerella arachidicola* W. A. Jenkins and *Mycosphaerella berkeleyii* W. A. Jenkins, respectively. This stage of each fungus may be involved in initial spread, but the asexual stage is most commonly seen during development of the disease in the field.

The geographical distribution of the two pathogens is similar, but the incidence of either pathogen differs markedly. In the United States most reports list *C. arachidicola* as the predominant species (141, 182, 278) which usually is found early in the growing season whereas *C. personata* is found later and is less abundant. Frezzi (70) noted that the occurrence of the two species was more closely related to host differences than to the period of the growing season. *C. arachidicola* was more frequent on common varieties of *A. hypogaea* while *C. personata* was found mostly on wild species in plant collections.

Miller (182) in his study of parasitism of the peanut leafspot fungi found the range of temperatures for growth of three races of *C. arachidicola* to be 2 to 35°C with an optimum of 25 to 32°C. Three races of *C. personata* had a growth range of from 4 to 34°C with an optimum of 25 to 30°C. Growth of both species in culture is slow, with *C. personata* being the slower growing. Das (60) reported that *C. personata* had cardinal temperatures of 23, 27, and 32°C. Abdou (1) found that light was not required for sporulation in culture of *C. arachidicola* but was necessary for *C. personata.* His work also indicated that the nutritional requirements for these two species differed.

Dissemination and infection

There is general agreement in many reports that cercospora leafspots are more serious where peanuts follow peanuts. In these situations early infection is common and the source of inoculum is presumably from conidia or ascospores produced in or on peanut debris in the field. However, Frezzi (70) demonstrated that conidia have sufficient longevity to carry over from one crop to another. Jenkins (141) and Frezzi (70) determined that ascospores formed in persisting litter are a source of early season inoculum. Shanta (237) and Hemingway (109) further suggested that mycelia survive in the soil as well as in plant debris from the previous season.

Inoculum is blown or splashed on leaves giving rise to primary infections. Subsequent spores are carried by wind, rain, insects (112), or machinery, thus leading to secondary infection cycles. Leaves are susceptible during the entire growing season, and lesions are usually first observed on the lower leaves near the soil.

Jensen and Boyle (143) reported that amounts of disease in peanut fields were correlated with periods of high relative humidity during which temperatures were nearly always in the 20's (°C). Lyle (163) found that the greatest number of conidia were detected during a period of abundant rainfall and high minimal (22°C) and maximal (35°C) temperatures. Infection was correlated directly with inoculum production during this period.

Conidia of both species germinate to form one to several germ tubes which grow over the leaf surface and through open Stomata. Penetration may also occur directly through the lateral faces of epidermal cells (141). With *C. arachidicola* the intercellular mycelium kills cells in advance of its growth and hyphae then became intracellular (141). *C. personata,* in contrast, does not kill cells in advance of its intercellular hyphae but produces botryose haustoria within host cells (141, 278).

CONTROL

Varietal Resistance

One possible underlying physical basis for resistance has been reported by Hemingway (110). He selected Kanyoma and Mwitunde as examples of resistant varieties and in a comparison with less resistant varieties, e.g., Natal Common, found that leaf thickness and dark green color were related to higher resistance. Lesion enlargement was slower for varieties with thicker leaves. He reported that most infections occurred through stomata and the resistant varieties had smaller stomata than susceptible varieties. D'Cruz and Upadhyaya (62) compared stomatal frequency and aperture size with susceptibility ratings for six species of *Arachis*. *A. hypogaea,* the most susceptible, has the largest stomata (averaging 17 u) but the lowest frequency. Gibbons and Bailey (93) working with *C. arachidicola* found that three wild species of *Arachis* were resistant and that resistance was associated with the small size of the stomatal apertures on the lower leaf surface.

Abdou (1) recently reported that sources of resistance can be found in wild peanuts. Immune or highly resistant strains were found but in some instances the resistance displayed is limited to only one of the two species of *Cercospora.* There was a strong relationship between stomatal width and frequency of wide stomata and infection. Hemingway (110) reported that resistance was related in some way to differences in the branching pattern of the host. Higgins (112) noted that resistance to leafspot is associated with lack of fruit-set so that highly resistant selections, species, etc., are commonly agronomically unacceptable.

Cultural and Chemical

From the comments on dissemination of the pathogens it is obvious that crop rotation is of primary importance in avoiding early season infection. Also practices designed to remove or bury debris (see Stem Rot control) or volunteer plants enhance other control measures.

Chemicals have been widely used for control of cercospora leafspots. The nature of the materials used depends largely on the economic status of the peanut farmer and the nature of the research information available to him. Sulfur is perhaps the most widely used chemical since it is fungitoxic and miticidal. The addition of a copper compound to sulfur results in a mixture of greater fungicidal properties. Organic fungicides are also used in leafspot control.

STEM ROT

Sclerotium rolfsii Saccardo

The disease of peanut caused by *Sclerotium rolfsii,* commonly called stem rot, southern blight, white mold, sclerotium blight, sclerotium rot, sclerotium wilt, root rot, or foot rot, has been reported from all peanut-growing areas of the world. The Fungus is distributed throughout the world and is particularly prevalent in warmer climates. The disease as it occurs in the United States ranks with cercospora leafspots as being of major importance in peanut culture. Reports from other countries, e.g., Argentina and Rhodesia, indicate that the disease is of major importance although serious outbreaks may occur only sporadically. In the United States an estimated 7.5 percent of the peanut crop is lost annually to the disease (262). Garren (77) in 1959, estimated the losses to peanut growers in the southern United States as 10 to 20 million dollars annually. The use of new effective control procedures during the past five years

has reduced losses substantially, but an estimation of current losses is not available. More than 189 plants, including some bryophytes and pteridophytes, are reported hosts (273) of the fungus. Aycock (12) recently published an exhaustive listing of reported hosts that represent nearly 100 plant families, most of which are herbaceous plants or seedlings of woody species.

SYMPTOMS

Stems

We use the common name "Stem Rot" because stem infection is the most frequent and serious manifestation of the disease. Under optimum conditions for disease development, the earliest sympton is a sudden wilting of a branch, usually a semi-decumbent outer branch. Leaflets rapidly become chlorotic to light green then turn brown as they desiccate quickly. Subsequently adjacent branches become infected and wilt. Wilting is caused by invasion of stems at or near the soil line. White mycelium of the fungus forms in copious layers on affected stems, particularly if a heavy canopy of leaves is present to maintain high humidity at the base of the plant (Fig. 2). Growth of the mycelium over the soil surface or along bits of organic debris is extremely rapid in the basal area of a plant so that all branches are usually involved within a few days. Dead

Figure 2. Stem rot caused by *Sclerotium rolfsii.* Felt of white mycelium and cluster of light brown sclerotia.

plants tend to remain upright in the row. The infected areas of the stem become shredded and the sheathing mycelium quickly produced abundant spherical sclerotia (Fig. 2). Sclerotia are at first white, velvety and succulent but later become light brown and hard, with a finely sculptured surface.

Garren (77) reported that if unusually wet weather occurs during the course of disease development the mycelial sheaths are not prominent and the affected stem bases are covered with elongate, lenticular, eroded lesions with tan to red corky excresences. Development of adventitious roots may temporarily prevent death of the branch. Conversely, during periods of drought, lesions caused by *S. rolfsii* may occur on the stem just below the soil surface. Garren reported such lesions to be brown, lenticular and about 0.5 cm long. Leaflets on infected plants may be smaller than normal and have a bronze cast.

Pegs

Growth of mycelia in the area of branch divergence leads to infection of pegs. The excursions of superficial mycelia are not confined to the basal branch area of the plant if sufficient leaf litter or other debris is present. Thus the pegs of procumbent varieties as well as bunch types readily become infected. It is a matter of small concern if pegs on a dying plant become diseased but infection of pegs can occur independently of stem infection. Peg infection leads to development of light to dark brown lesions 0.5 to 2 cm long and eventual tissue shredding and pod loss.

Roots

Primary and secondary root infection is far less common than stem or peg infection. Garren (77) noted that he had not seen an authentic case of root rot of peanut caused by the fungus. Singh and Mathus (241) reported a root rot caused by *S. rolfsii* but did not describe symptoms in detail. Similarly Dubey (64) reported on the "root-rot" disease but presumably this name did not apply to specific symptoms. Ashworth *et al.* (6) reported only occasional tap root infection under their conditions of inoculation in greenhouse culture.

Pods and Kernels

Ashworth, *et al.* (6) found that pods and kernels were less frequently attacked than stems or pegs. Lesions on young pods of Spanish peanut were orange-yellow to light tan and were light brown to black and zonate on older pods. Kernels in advanced stages of decay were shriveled and covered with wisps of fungus mycelium. General surveys of mycoflora of kernels have provided evidence that *S. rolfsii* is not a predominant seed-invader under common cultural and environmental conditions (126). Although it is usually listed among seed-borne fungi, its incidence is very low. A malady known as blue damage is caused by the fungus *(see* Blue Damage) but these symptoms differ from those produced by active rotting of kernels.

CAUSAL ORGANISM

Taxonomy and Morphology

The taxonomy of the fungus was reviewed by West (273), Garren (77), and Aycock (12). The name *Sclerotium rolfsii* was applied by Saccardo (223) characterizing the fungus as having no known asexual spores and belonging to this heterogenous form genus. The basidial stage was subsequently known from culture only and was assigned the name *Corticium rolfsii* (Sacc.) Curzi. In 1947 West (272) found

the basidial stage occurring naturally on *Ficus pumila* L. and proposed the name *Pellicularia rolfsii* (Curzi) West.

The size, shape, color, and surface texture of sclerotia are quite variable among isolations of the fungus. Presumably because the sclerotia are so prominent and variable, they have been used as the basis for transient taxonomic distinctions of species, e.g., *S. delphinii* Welch, and strains.

Physiology

Higgins (111) established that *S. rolfsii* in culture virtually would not grow below 8 or above 40°C (Below 46°F or above 104°F). At 42°C (108°F) the fungus did not grow but was still alive after 48 hr. Temperatures of —2 to —10°C (30 to 14°F) killed mycelium and germinating sclerotia but not dormant mature sclerotia. These early data by Higgins have been repeatedly confirmed (Watkins (267)). Higgins also found that growth of *S. rolfsii* occurred *in vitro* over a pH range of 1.4 to 8.8. The temperature relationship to growth, and particularly to survival of sclerotia, was thought by Higgins (111) to be the primary factor limiting the geographic distribution of *S. rolfsii.*

Higgins (111) found that *S. rolfsii* produced large amounts of succinic and oxalic acids and he provided evidence to show that oxalic acid was of primary importance in the infection process. Cooper (50) concluded that strains of the fungus vary greatly in pathogenicity and virulence. Loss of virulence in culture has been noted frequently.

S. rolfsii grows well over a range of concentrations of nitrogen and calcium compounds that probably exceeds ranges normally found in soils. This suggests that the reported disease control effects from calcium and nitrogen compounds probably involve an increased resistance or growth rate in the host rather than decreased growth by this pathogen (267).

Because the soil surface is the prominent site of growth and pathogenesis of the fungus, the importance of oxygen in growth and persistence of the fungus has been frequently questioned. Flados (68) found that in pure culture *S. rolfsii* grew in atmospheres containing even trace amounts of oxygen. When cultures were allowed to become contaminated, growth of contaminants under low oxygen concentrations exceeded that of *S. rolfsii* and tended to restrict growth of this fungus. However, in tubes of soil *S. rolfsii* remained near the surface unless the soils were autoclaved. The conspicuous concentration and activities of *S. rolfsii* near the soil surface may be related more closely therefore, to the presence of an adequate food base and abundance of competitive or antagonistic fungi in the soil than to oxygen concentration. Antagonism may also be increased by high soil moisture leading possibly to development of *S. rolfsii* on or near the soil surface in contrast to extensive growth below the surface (68) although the fungus is capable of growth through the soil for considerable distances from its food base. Formation of sclerotia and active growth of mycelia usually seem to have an inverse relationship. Observations that outbreaks of stem rot of various crops are common and particularly destructive following dry periods seemingly involves the conditions surrounding germination of sclerotia. Watkins (266) and Boswell (23) both found that sclerotia germinated poorly when stored under humid conditions but that after a few days storage at a low relative humidity, germination was much improved. Boswell further concluded that the low relative humidity led to drying and superficial cracking of the sclerotial rind. Subsequently he showed that other treatments which resulted in mild scarification of the sclerotial surface improved germination.

Infection

Higgins (111) described the process by which oxalic acid, secreted by the fungus hyphae, kills host epidermal cells well in advance of penetration by the fungus. The mycelium develops specialized bulbous holdfasts that attach the fungus to the host surface before penetration. The fungus hyphae grow into host cells at points well behind the widening margin of killed tissue and pass from cell to cell. Thus any plant, as Higgins stated, with an epidermis permeable to oxalic acid solutions is susceptible to attack and if living hosts are absent growth may continue on plant debris in the surface soil. Boyle (29) termed the fungus *necrotrophic* and pointed out that the basically saprophytic nature explained why crop rotation had been an unsuccessful method of control or disease reduction. Starting with a sclerotium that had survived the rigors of adverse growing conditions, Boyle (30) suggested that in total absence of natural media for saprophytic growth the sclerotium may not have sufficient reserve energy to establish its mycelium in a living host. This suggestion, while largely hypothetical, seems to be pragmatically sound, based on the usual low disease incidence in experimental field plots where surface debris is practically nil. Reasoning further, Boyle (30) suggested that the presence and abundance of organic debris that can be used by the fungus for saprophytic growth therefore becomes of great importance in providing a growth connection between germinating sclerotia and the host plant. Whether or not this conception of the growth and pathogenic establishment of the fungus is wholly correct, cultural practices based partly on this idea have proven exceptionally successful in control of the disease.

CONTROL

Varietal Resistance

Reyes (220) reported in 1936 that rather signficant differences in susceptibility existed among peanut varieties. His data suggest that such differences were related to growth habit, the semi-decumbent or bunch types being more susceptible than runner types. Cooper (50) summarized Reyes' data and gave information from his own research indicating that the varietal differences found were somewhat consistent from year to year. Garren and Bailey (86) reported that the slight differences in disease recorded for the bunch variety, Virginia Bunch 46-2, as compared with the runner variety, Virginia 56R, were not enough to justify saying that either was more susceptible. Conventional cultivation made disease easier to detect in the bunch variety than in the runner, and offers a possible explanation for the persistence of the idea of greater resistance in runner peanuts. Garren (79) later concluded that, in comparative terms, Valencia was highly susceptible, Spanish was susceptible, Virginia Bunch 46-2 was intermediate in suceptibility, and NC-2 was resistant. Studies of the qualitative and quantitative aspects of inheritance have not been made, hence these important genetic data are lacking.

Cultural and Chemical

Cultural practices for control of stem rot have proven to be extremely successful. Based on the concepts of Boyle (29, 30), a large body of experimental evidence testifies to the benefits of (31) deep plowing to bury surface litter, (77) a flat or, at most, slightly raised bed area with movement of soil away from the row during cultivation, and (23) control of cercospora leafspots to prevent leaf drop and subsequent accumulation of dead leaves at the bases of plants. Boyle and Hammons (31) reviewed these points in detail and related them to other facets of peanut culture such as use of

pre-emergence herbicides. Garren (77) reviewed the history of peanut stem rot control efforts in detail and presented abundant evidence to show that deep covering or burial of organic matter before planting and nondirting cultivation procedures controlled stem rot. The effectiveness of deep plowing according to Garren (77, 79) is to remove from the soil surface a medium highly conducive to the growth of the pathogen rather than the actual total removal of the pathogen. By avoiding the movement of soil up around the bases of plants during cultivation (nondirting), formation of new organic debris by injury and smothering branches and leaves is prevented and the fungus has no weakened or dead tissue as a food base. Cultural practices may be only partially successful unless they are done with precision, as recommended by Shepherd (238).

Chemical control of *S. rolfsii* of peanut has been economically feasible and successful in some areas. Cooper (49) reported the successful use of pentachloronitrobenzene (PCNB) when applied to the soil before planting in North Carolina and Harrison (106) obtained beneficial effects with the same chemical in Texas. Jackson, *et al.* (139) showed that some fumigants applied before planting were of considerable value in reducing plant loss from stem rot. The efficiacy of PCNB and other non-volatile fungicides has not been sufficient in Georgia to warrant recommendation for control of stem rot.

RHIZOCTONIA DISEASES

Rhizoctonia solani Kuhn

This pathogen is capable of infecting all principal organs of the peanut plant. The fungus persists for long periods in the soil and is widely distributed in all major peanut-growing areas of the world. The economic importances of the disease are impossible to determine with accuracy, since even in areas where disease estimations have been made rhizoctonia diseases are grouped anonymounsly with other root, stem and pod diseases (262). Peanut losses due directly to *Rhizoctonia* probably are substantially greater than might be assumed by judging only from world literature on the subject. Several hundred plant species, including many economic crop plants are susceptible to this fungus. Root and stem rots are the common symptoms found on most hosts.

Symptoms

Fruit

Peanut fruit from the time of soil penetration by the elongating gynophore-bearing peduncle (pegs) to harvest, are exposed to the soil-borne fungus *R. solani. R. solani* can be isolated from many of the pegs or small pods which become brown or black at the tip and then rot or wither. Therefore it is assumed that these symptoms resulted from the infection by *R. solani.* This is probably a correct assumption for Ashworth, *et al.* (6) and Garren (84, 85) have experimentally obtained the needed scientific proof that *R. solani* can cause lesions on and a rot of peanut fruits. Kranz and Pucci (152) surmised that fungi other than *R. solani* can also cause this "blight" of pegs and young fruit of peanuts.

Pods during all stages of development probably are susceptible to infection by *Rhizoctonia* and will exhibit varying degrees of discoloration, from slight superficial russeting to browning of the entire pod and decay of the contents. Other fungi may also invade pods after, or simultaneously with, *Rhizoctonia* thus tending to mask or

alter symptoms. Perhaps for this reason a variety of lesion colors and textures have been reported as symptomatic of this disease, but dark brown to black decayed areas are at least circumstantial evidence of *Rhizoctonia* damage. Ashworth, *et al.* (6) in Texas have conducted a thorough study of *Rhizoctonia* on Spanish peanut in the field and greenhouse. They reported that pod lesions were characteristically dark brown and angular from the beginning of pod infection with occasional raised areas due to the presence of sclerotia. They also noted that this type lesion is similar to those reported in Georgia by Good, *et al.* (100), who attributed such lesions primarily to the nematode, *Pratylenchus brachyurus* (Godfrey, 1929) Goodey 1951. In the Texas study nematodes were not necessarily involved in the production of such lesions, although in limited field samples, both *P. brachyurus* and *R. solani* were frequently associated in these angular dark shell lesions. Angular dark pod lesions are not, therefore always symptomatic of *R. solani.*

Pegs are commonly attacked and lesions are brown to black, varying from slight to extensive sunken areas along the peg. Seed within pods are readily infected as the fungus grows over and through the pod in the soil. In many cases both seed and pod are sufficiently decayed to be mechanically excluded from the harvested crop. When infected seed are harvested, they frequently have discolored, faded, or stained seed coats. In varieties with pink testae the area of discoloration is often light brown to gray.

Garren (85) recently showed that *Rhizoctonia* can cause rot of Virginia peanut fruits in the soil which can be distinguished from pod breakdown caused by *Pythium myriotylum* Drechs. by laboratory analyses only. He also reported evidence from greenhouse studies (84) that there is competition between *Rhizoctonia* and *Pythium* when both are present in the same soil. Certain limited conditions must prevail before *Rhizoctonia* can become a major cause of a fruit rot because *P. myriotylum* usually invades peanut fruits rapidly.

Seedlings

Invasion of germinating seed by seed- or soil-borne inoculum may lead to preemergence death which, unless many plants are killed, is usually unnoticed. In emerged plants lesions are most frequent on the hypocotyl as sunken, elongate, dark brown areas 2 to 3 cm long. A rapid invasion and browning of the entire hypocotyl sometimes occurs. Similar lesions develop on the tap roots. Rotting of lateral roots may accompany these symptoms, but seldom occurs alone. The apical meristem may also become infected as Ashworth, *et al.* (6) found when infected seed were used in greenhouse experiments.

Mature Plants

Plants in various stages of development may be attacked with gradual disintegration of roots, first noted as sunken dark brown cankers along the primary roots and progressive total browning of secondary roots. Stem symptoms are equally common and occur at or near the soil line. Infected areas are dry, sunken, dark brown and may extend several cm along the stem. Girdling of the stem marks final stages of disease development and at this point plants may exhibit a "wire-stem" appearance, i.e., the uninfected tissue above the girdling is of noticeably greater diameter than the infected stem. Such terminal symptoms are common with many hosts of *R. solani.* During excessively moist weather or, as Frezzi (71) noted, in thickly planted stands the fungus may spread up into the branches for a short distance. Branch lesions become brown and may appear "shredded" due to disintegration of tissues around vascular bundles. Gross field symptoms of root, stem, or branch infection are excessive wilting followed by death of one to many branches or the entire plant.

We have observed infection of lower leaves during periods of prolonged rainfall. Brown speckled or blotchy areas develop but this symptom does not persist due to rapid progressive killing of leaf tissue.

CAUSAL ORGANISM

Because *R. solani* is a serious and ubiquitous pathogen of many crops it has been studied intensively for many years. During this period the fungus has acquired several scientific names for the basidial stage. Although it may be assumed that ultimately only one name will be considered correct, the matter is not clear at this time. *Corticium vagum* Berk. and Curt., *Corticium solani* (Prill. and Del.) Bourd, and Galz., *Pellicularia filamentosa* (Pat.) Rogers, and *Thanatephorus cucumeris* (Frank) Donk are names currently used.

Morphology and Physiology

The mycelia of *R. solani* isolates in culture vary in color and growth rate. Characteristically, the forward extension of hyphae is accompanied by development of branches that diverge almost perpendicularly to the main hyphal axis. Hyphal development in and on peanut tissues is sometimes evident as tawny strands. The fungus can produce brown to black, flattened, irregular sclerotial structures in or on some host tissues but these are not often found when peanuts are attacked. The sexual stage develops on the surface of soil or plant parts as a relatively conspicuous white hymenial layer covered with basidia. Two or more basidiospores are borne at the apex of each basidium and these spores can germinate to form thalli of *R. solani.*

Strains of *R. solani* vary in their capacities to persist or grow under various conditions of soil carbon dioxide content or temperature. Although exceptions are found, generally disease development is favored by temperatures between 19 and 36°C (66 and 97°F) and moderate soil moisture, rather than extremely wet soil.

Dissemination and Infection

The pathogen persists in the soil for long periods where, in absence of live hosts, it lives saprophytically on bits of organic debris. Its spread is therefore facilitated by the movement of even small quantities of soil or plant residue that contain mycelia or sclerotia of the fungus. Basidiospores, carried by air currents or water, also serve to disseminate the fungus. In addition to soil-borne inoculum the fungus commonly is carried in peanut seed (7) and is thus proximate to the emerging seedling after infected or infested seed are planted.

Infection occurs through wounds or directly through intact surface tissue. In the latter case, work on *R. solani* parasitism of other crops has shown that cushion- or finger-like structures are typically produced by branches of hyphae in contact with the host surface and penetration of host follows this development.

CONTROL

Varietal Resistance

Genetically determined resistance of cultivated peanuts to *R. solani* has not been conclusively demonstrated and putatively resistant selections (91) have found no useful place in presently grown varieties.

Cultural and Chemical

The cultural practices found effective for control of *Sclerotium rolfsii* (see Stem Rot) were noted by Higgins (112) to be effective in control of *R. solani.* The prac-

tices consist of complete burial of litter and nondirting during subsequent cultivations. The principal benefit from litter burial presumably is the removal of infested debris from the upper soil layer where growing plant parts are located. Crop rotations are important in control of rhizoctonia diseases. Crops that are relatively susceptible to the pathogen — such as bean, soybean, cotton, southern pea, peanut — should not be followed by a peanut crop. Summer or winter grain crops and pasture grasses, on the other hand, are better choices as an antecedent crop. The implication that only resistance (hence decrease of debris-borne inoculum) of a particular crop determines its suitability is not warranted in view of recent information on fungistatic effects of certain crop residues (61, 204). Smith and Ashworth (245) reported that *R. solani,* unlike the soil microflora as a whole, was depressed by soil amendments of rice hulls and oak sawdust. Water extracts of amended soil were found to inhibit growth of the fungus. They suggested that competition among the soil microflora and direct toxic effects were responsible for the benefits from the soil amendments.

Control of seed-borne *R. solani* has been successful in large measure by use of chemical seed treatments. Jackson (125) showed that several fungicides were effective in eradicating most of the fungi (including *R. solani)* in test seed lots, prior to planting. However, the list of acceptable chemical treatments changes rapidly and the latest list should be used in selecting treatments.

Economic control of *R. solani* in the soil is a more difficult problem. Effective chemical treatments might be considered essential if virtual crop failure resulted when they were not used. However, it is impossible to predict with accuracy the peanut losses that will be sustained in a given area during a given growing season. Hence use of a soil treatment may be an unprofitable endeavor when disease incidence is low. Jackson, *et al.* (139) reported that *R. solani* and other peanut pathogens were controlled through use of volatile fumigants. Ashworth, *et al.* (8) also have reported long-term inhibition of the fungus with a fumigant. Here again the latest list of acceptable fumigants should be used.

Non-volatile fungicides with relatively low water solubility have been used to control early attacks of *R. solani.* Among such fungicides, pentachloronitrobenzene (PCNB) inhibits growth of *R. solani* (134, 245) and is of value in some peanut growing areas as a pre-plant soil-incorporated treatment for control of seedling diseases. Despite many experimental evaluations over the past 15 years PCNB and other soil fungicides have not been of clear value in some areas, and therefore, have not been recommended.

It may be that conditions will rarely be right for *Rhizoctonia* to be the major cause of peanut pod breakdown (85). This may be due partially to the fact that rotation, cultural practices, etc. are keeping *Rhizoctonia* under control in most peanut fields. Garren (81) found that three out of five years in Virginia high rates of PCNB actually increased pod breakdown rather than decreased it. The assumption is that the PCNB kept in check the *Rhizoctonia* and perhaps other competitors of *P. myriotylum.*

ASPERGILLUS CROWN ROT

Aspergillus niger van Tieghem
Aspergillus pulverulentus (McAlpine) Thom

Aspergillus crown rot was reported on peanuts in 1926 in Java by Jochems (144). It was later reported from Australia, India, and the United States. It is likely that aspergillus

crown rot is found in all peanut growing areas of the world. The economic importance of the disease is difficult to estimate. A uniform stand reduction of 10 to 20% possible would not affect final yield significantly. A greater, or less uniform stand depletion would probably compel replanting with its attendant costs and delay or lead to important yield loss.

SYMPTOMS

Seed can be attacked as soon as they are placed in a moist soil environment, but more commonly seed germinate and the succulent elongating shoots are attacked and rapidly killed. The hypocotyl becomes watersoaked and light brown and is soon covered by black masses of spores. The first symptom in emerged seedlings is usually a rapid wilting of the entire plant or its branches, especially during dry weather. At this stage, the hypocotyl and tissues of the cotyledonary node are partially rotted. Diseased tissue is dark brown and intact at first and later becomes lighter in color and shredded. Necrosis and shredding of tissue may extend up into the branches. Development of spores, particularly on and around the cotyledons, occurs prior to wilting but sporulation is often sparse on shredded necrotic tissue. When infection of the hypocotyl takes place well below the cotyledons, plants may wilt temporarily and eventually recover. Such recovery occurs frequently during periods of high soil moisture and is possible because of rapid growth of roots above the infection site.

CAUSAL ORGANISM

According to Jacin and Nema (121), Jochem's (144) 1926 report specified *A. pulverulentus* as the cause of crown rot of peanut. Inspection of Jochem's report shows that only *A. niger* is mentioned. However, Boedijn (21) later identified Jochem's fungus as *A. pulverulentus* which is grouped closely with *A. niger*. Chohan (39) reported that isolates of *A. pulverulentus* and *A. niger* were equally effective in causing aspergillus crown rot.

The optimum temperature for radial growth of *A. niger* in culture was reported by Gibson (96) to be 37°C. Jackson (130) found that an isolate from peanut produced most growth weight at 32°C. The fungus persists and grows on a variety of organic and inorganic materials and is quite tolerant of copper (218). Gibson (97) reported that organic mercury compounds when used alone as seed treatments, greatly increased the incidence of aspergillus crown rot. This report was affirmed by Purss (213) after several year's field trials in Australia, by Schmutterer (232) in Sudan, and by Jackson in Georgia (124, 128). Gibson (97) found that mercury-tolerant strains of *A. niger* isolated from African soils were distinguished from mercury-sensitive strains both by a great and rapid development of acidity in culture and by a greater virulence in the production of crown rot.

Dissemination and Infection

A. niger is found abundantly in soils and plant debris. Jackson (123) reported that great abundance of *A. niger* propagules in soil was directly related to high disease incidence in plants growing in the soil. Ashworth, *et al.* (5) also found a positive relationship between disease and the number of particles of a given soil from which *A. niger* could be isolated.

The pathogen is carried on the seed surface and in or under the tissues of the testae (127). Sites of infection are also abundant in the cotyledons or radicle-plumule

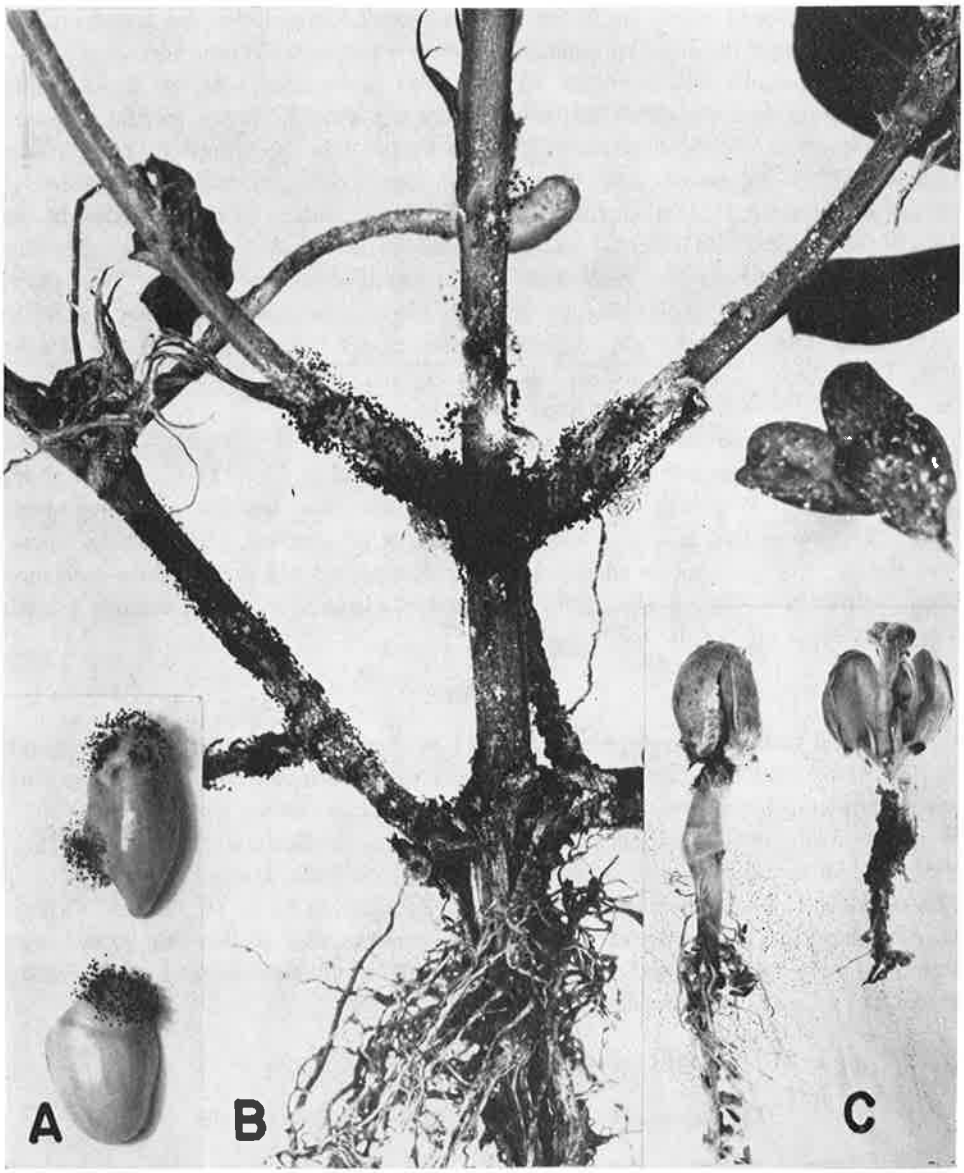

Figure 3. Seed and crown rot caused by *Aspergillus niger*. A. Fungus growing from infected seeds. B. Necrosis and shredding of stem at groundline, plus mass of black conidia. C. Infections of elongating hypocotyl and primary root.

of the seed (129). Ashworth, *et al.* (5) found, in their study of Spanish peanut, that superficial contamination of seed with *A. niger* was more common than deep-seated infection. Seed became infected during the last days of maturation in the soil and in subsequent harvesting and handling procedures. Infection of kernels in undamaged pods in the field may occur prior to maturity (135). If pods are harvested and dried promptly, presumably the numbers of infected kernels decrease due to death of hyphae or spores. However, if harvested peanuts are allowed to remain damp for long periods or to dehydrate after becoming dry, *A. niger* may invade most of the kernels. Jackson

(126) surveyed seed stocks in Georgia and concluded that soil-borne inoculum was of more importance in initiating aspergillus crown rot, than seed-borne inoculum.

Infection usually occurs within 10 days after germination. As the hypocotyl of the germinating seed elongates and comes in contact with soil-borne inoculum, lesions develop rapidly due to direct penetration of the hyphae into the hypocotyl or cotyledons. Jackson (123) suggested that cotyledonary infection, that commonly results in growth of the fungus down into the hypocotyl, was important in the initiation of the disease. The majority of infected plants succumb in less than 30 days after planting. Gibson (96) found that the peak death rate occurred about the 17th day after planting and that the period of dying of emerged plants extended from about 10 to 30 days and he inferred that many slightly infected plants recovered during the first 30 days. The occasional occurrence of aspergillus crown rot in older plants is probably the result of light or arrested infections becoming active later.

High soil and air temperatures may predispose plants to infection. Gibson (96) found that greater infection occurred among plants raised at 30 to 37°C than in plants raised at 20°C even though the postinoculation temperature was the same. Ashworth, *et al.* (5) showed that etiolation predisposed plants to infection, and delay in emergence due to deep planting or chemical toxicity also caused a greater disease incidence. Wounds from heat injury healing slowly predisposed plants to infection, whereas wounds from mechanical injuries did not.

CONTROL

Chemical control of aspergillus crown rot has been achieved with seed treatments. In view of the unusual effect of organic mercury seed treatments, non-mercurial organic compounds have found great use in areas where aspergillus crown rot is serious (101). Most commonly used materials are tetramethyl thiuram disulfide (thiram, TMTD); N-[(thrichloromethyl) thiol-4-cylohexane-1, 2-dicarboximide (captan); and N-[(1,1,-2,2,tetrachloroethyl) sulfenyl]-*cis*-4-cyclohexene-1,2-dicarboximide (difolatan). Organic mercury materials possess excellent eradicative qualities and if they are mixed with captan or other non-mercurial fungicides they provide excellent control of aspergillus crown rot and many other seedling diseases.

PEPPER SPOT AND LEAF SCORCH

Leptosphaerulina crassiasca (Sechet) Jackson & Bell

A disease of peanut foliage, caused by *L. crassiasca (Pleospora crassiasca* Sechet) was reported from Madagascar in 1955 (235). A year later Yen, *et al.* (280) reported that a similar disease in Taiwan, which they called leaf scorch was caused by *Leptosphaerulina arachidicola* Yen, Chen and Huang. Pepper spot or leaf scorch symptoms caused by *L. crassiasca* have been reported from Georgia, Texas, India, and Argentina. The only reported hosts of the fungus are species of *Arachis.* Frezzi (73) notes that the rhizomatous species *A. burkartii* Handro, *A. glabrata* Benth, *A. hagenbeckii* (Harms) Hoehne, and *A. hypogaea* are susceptible. Graham and Luttrell (105) reported *A. monticola* Krap. and Rigoni and *A. hypogaea* as hosts.

The disease cannot be considered serious from a world view. The common names of this disease, describe the two principal, distinct, and often apparently unrelated symptoms of the disease. 'Leaf scorch' has been applied also to symptoms caused by *Phomopsis, Macrophoma* and *Colletotrichum* (73).

SYMPTOMS

Symptoms, which are confined to leaves, are seen as minute necrotic flecks or marginal necrosis. Pepper spots are dark brown to black lesions, usually less than 1 mm in diameter, irregular to circular in outline, and occasionally depressed. The spots usually occur, discretely over the leaflet surface and are visible from both sides of the leaflet. Lesions do not rapidly enlarge with age, but when numerous, lesions tend to coalesce giving the leaflet surface a netted appearance. In such cases leaflets soon die. However, smaller number of lesions do not have an obvious deleterious effect on leaflet color or persistence. After infected leaflets become detached and die, the fungus grows rapidly throughout the leaf and ascocarps are formed in profusion.

Leaflets with scorch symptoms become chlorotic and then necrotic at discrete points along the margins. The necrotic tissue becomes dark brown and a chlorotic zone commonly develops along the edges of necrotic tissue. Necrotic tissue tends to fragment along the leaflet margins, presenting a tattered appearance.

Pepper spot and leaf scorch symptoms are very often obscured or confused by the occurrence of cercospora leafspots. Scorch lesions frequently coalesce and encompass these leafspots and, in addition, pycnida of *Macrophoma* and *Phomopsis* are often found with ascocarps of *L. crassiasca.*

CAUSAL ORGANISM

The fungus has cardinal temperatures for growth *in vitro* on potato dextrose agar of about 8, 28, and 35°C (280). Field observations of the prevalence of pepper spot and leaf scorch in some areas suggest that the mechanisms for spread of the fungus are quite efficient. No reports are available to document the manner in which ascospores of the fungus are disseminated. Graham and Luttrell (103) and Luttrell and Boyle (162) comment on the rather forceful way in which ascospores are ejected in great numbers.

The infection process is poorly understood. Frezzi (73) reported that ascospores at 25 to 28°C in water germinated in 2 hours. Yen, *et al.* (280) found that temperature and moisture were very important factors in ascospore germination. At 28°C and 100% relative humidity (RH) germination was 96% but at 25°C and 98.5% RH it was only 33%. Pettit, *et al.* (207) reported that ascospores become closely attached to the leaflet surface and germinate when free water is available.

CONTROL

Protectant fungicides have not been used in any field trials reported in the literature. Yen, *et al.* (280) present comparisons of laboratory tests using captan, thiram and other fungicides. McGill and Samples (166) suggest that fungicides and spray programs effective in control of cercospora leaf spots will also control pepper spot.

DIPLODIA COLLAR ROT
Diplodia gossypina Cooke

Diplodia collar rot (diplodia blight, collar rot) is sporadically serious on peanut in the southern United States. The disease has also been reported from Israel (42) Venezuela, Australia, and South Africa (138). The casual fungus is distributed throughout the world and lives as a saprophyte and as a wound parasite on many crops.

D. gossypina also causes a disease of peanut seed *(see* Concealed Damage). Citrus, cotton, sweetpotato, peach, alfalfa, and camellia are a few of the other hosts of the fungus.

The common name *collar rot* was proposed by Garren and Wilson (91) to describe symptoms of an undetermined cause. They presented evidence to show that the cause was probably a fungus. Although aspergillus crown rot was not recognized as a problem in the United States until recently, it seems probable that the disease had been present in previous years and may have been part of the collar rot disease described by Garren and Wilson (91). In 1965 losses attributed to a combination of collar rot and crown rot in the United States were estimated at 1% of the crop (262). Instances of 25 to 50% stand loss have been reported (112, 120).

SYMPTOMS

Seedlings or maturing plants are attacked at or near the soil level and the fungus quickly invades the stem. The first obvious symptom of the disease is a rapid wilting of branches or the entire plant. Infected plants usually die within a few days during warm weather. Stem lesions become grayish-brown to black and extend toward the taproot. Necrotic stems tend to become shredded. Infection of a branch, particularly of a large plant, may result only in death of the branch. In such instances progress of the infection toward the main axis of the plant is slow. Numerous embeded pycnidia develop in the necrotic tissue and are seen as minute, black, pimple-like dots.

CAUSAL ORGANISM

Morphology and Physiology

In a review of the taxonomy of the collar rot *Diplodia* McGuire and Cooper (168) presented comparisons of many common species of *Diplodia* and concluded that there was no tenable basis for maintaining five separate taxa. Consequently they proposed that the correct name for the casual fungus was, on the basis of priority, *D. gossypina.*

McGuire and Cooper's (168) isolate of *D. gossypina* had cardinal temperatures of 8, 32, and 40°C (46, 90, 104°F) for growth on agar medium. Optimum temperature for growth has been used as a taxonomic criterion in *Diplodia* (168) but fails in this service because of the wide range of variation among isolates with morphological and pathological similarities.

Dissemination and Infection

Although the literature indicates that *D. gossypina* is not usually considered a soil inhabitant, it clearly is able to persist in organic debris in the soil for long periods.

Garren and Porter (90) in a comparison of the mycoflora of mature, cured peanut fruits from Puerto Rico and Virginia found up to 24% of the shells of peanuts from Puerto Rico were infested with viable *D. gossypina.* This fungus was not found in the Virginia-grown peanuts.

Comparison of effects of corn and cotton crop debris on the incidence of diplodia collar rot in a following crop of peanuts have shown that the disease is up to nine times more severe when peanuts follow cotton (168). Limited information on soybean (138) suggests that this crop also leads to a substantial disease increase in a following peanut crop. McGuire (167) found *D. gossypina* sporulating abundantly on stem and boll debris in cotton fields during the winter and the fungus was able to persist until the following summer. The severity of the disease is thus influenced by

the previous crop and the susceptibility of the prior crop is an important consideration in rotation planning.

Hyphae in debris, and spores of the fungus can be moved by running water, splashing or blowing rain, or by cultivation in the proximity of peanut stems. Infection rarely occurs unless plants have been predisposed by heat injuries. Prior to the reports of Boyle (27, 28) and McGuire (167, 168) concerning the role of heat injury, attempts to reproduce diplodia collar rot by inoculating healthy or mechanically wounded stems generally failed. Boyle (28) showed that intense heat generated by incident and reflected sunlight could injure tender peanut stem tissues, causing a heat canker. Such injuries generally either healed slowly or became infested by fungi but heat cankers *per se* did not commonly cause widespread death of seedlings. McGuire (167), after comparing eight wound-inoculation techniques, found that only heat injury from sunlight or electric infrared heat lamps, followed by inoculation with the fungus, consistently resulted in development of infection. This predisposition of the stem mainly, but not exclusively, by sunlight-induced heat injury seems to be an indispensable condition for infection in the field. Hot dry weather is reported to favor infection, presumable because it also favors heat injury.

The fungus rapidly colonizes heat-injured tissue and grows mostly intercellularly through the cortical parenchyma. After infection is initiated in moribund tissue, adjacent unwounded tissues are readily invaded.

CONTROL

McGuire and Cooper (168) found no appreciable resistance to diplodia collar rot in the commercial varieties NC-2, Va. 56R, NC-4X, Ga. 119-20. Since the disease is related closely to heat injury, varietal resistance to heat injury might be pertinent. Boyle (28) suggested that heat canker is more prevalent in Runner than in Spanish varieties because the speed and profusion of leaf development is greater in Spanish varieties, thus providing shade to the stem earlier during growth.

Crop rotation schemes in which cotton, soybean, and perhaps other crops are avoided before planting peanuts should be adopted. Deep burial of litter may be useful, particularly if good rotations cannot be established. Boyle (28) suggested that heat canker could be reduced by planting peanut rows so that plants tended to shade each other. He reported that a finely clodded soil surface was most favorable for reducing reflective sunlight energy.

To date of this writing there is no published information to indicate that seed-borne inoculum is of great importance in the initiation of diplodia collar rot. However, as yet unpublished results of D. M. Porter and K. H. Garren indicate both a high degrees of differences in susceptibility among recently introduced commercial varieties of peanuts to diplodia collar rot and that much of the collar rot of highly susceptible varieties had its origin in seed-borne inoculum. Seed treatments, particularly those with eradicative vapor activity, might be expected to reduce seed-borne inoculum.

RUST

Puccinia arachidis Spegazzini

Rust of peanut is a serious disease in areas of the world where it is endemic or occurs regularly. As it was in 1951 (91), the disease is still restricted to the West Indies, northern South America, areas of Argentina, Uruguay, and Paraguay, the Caspian Sea

region of Asia, and portions of the United States. Muller (193) has reported it from Central America also. The disease is endemic in the West Indies and continues to hinder commercial peanut production in these islands (67). In the United States rust has been reported from every major production area. However, as Wells (269) and Higgins (112) reported, the fungus apparently does not overwinter in the United States but blows in from subtropical areas. The resulting infection patterns in the United States are quite erratic. When rust becomes established early in wet seasons, economic losses may be locally serious, whereas late season establishment of the disease does not cause great losses (269). The fungus attacks *Arachis hypogaea* and has been reported on *A. marginata* Gardn. (35), *A. nambyquarae* Hoehne, and *A. prostrata* Benth. (271).

SYMPTOMS

McVey (169) followed the development of symptoms after inoculating plants in the greenhouse. Infection appeared 8 to 10 days after inoculation as whitish flecks on the abaxial (lower) surface of the leaflets. Yellowish-green flecks appeared on the adaxial (upper) surface about 24 hours later; simultaneously uredinal pustules became visible on the abaxial surface within the whitish flecks. These pustules enlarged and ruptured within 48 hours of their appearance. Pustules on the abaxial surface appeared opposite some of those on the adaxial surface a short time later and ruptured after a similar time period. All of the speciments examined by Jackson and Bell (138) had more sori on the lower surface. Individual sori are 0.3 to 0.6 mm in diameter, circular, and often surrounded by leaf tissue that is dull green to light brown. Coalescence of infection sites is common leading to elongate or variously irregular patches of sori. Erumpent pustules vary in color from dark orange when young to dark brown at maturity. The plant tissue surrounding the visible sites of infection becomes nectoric and desiccated in irregular patches and eventually leaflets may curl and drop off. In our specimens leaflet necrosis is usually more prominent on the adaxial surface opposite concentrations of sori. Infection of leaflets, petioles, and stems is common.

Rust usually occurs with cercospora leafspots and the symptoms and effects of each of the diseases become indistinct.

CAUSAL ORGANISM

Taxonomy and Morphology

Spegazzini described *Puccinia arachidis* in 1884. Other names were subsequently proposed, but Arthur (4) eventually accepted the name as correct. Only the uredinial and telial stages are known. Garren and Wilson (91) noted that the telial stage was reportedly rare but Higgins (112) cited Gaurch in Uruguay as reporting abundant telia on certain specimens. In the United States only the uredinial stage has been found.

Dissemination and Infection

Objective information showing that rust outbreaks in the United States are due to wind-borne inoculum from the West Indies or other areas where the fungus overwinters is lacking. However, by analogy with pathogen dissemination in cereal rusts and the fact that there is considerable summer air movement northward form the tropics, the presumption is probably correct. Dissemination on seed was suggested by West (271) and Garren and Wilson (91). Spread of the pathogen within fields is facilitated by wind movement or blowing rain.

The temperature range at which infection may occur has not been reported in the literature, but McVey (169) maintained temperatures of 22 to 25°C (72 to 77°F) at night and 30 to 43°C (86 to 109°F) during the day in his successful inoculation experiments. The occurrence of the majority of uredinia and telia on the abaxial leaflet surface is a characteristic of the disease but not an indication of greater susceptibility of the abaxial surface to infection. McVey (169) showed that infection occurred directly through the adaxial surface but that the number and rate of development of uredinia were greater on the abaxial surface.

McVey (169), Castellani (36), and others have observed that rust is not prevalent in the field until plants are about six weeks old even though fields may contain rusted volunteer plants as a source of inoculum. However, McVey found that plants of any age were susceptible in his greenhouse inoculations. Wet weather seems a prerequisite for serious widespread field infection as noted by Wells (269) but experimental evidence is not available to show the degree of moisture necessary.

CONTROL

Although experiments on chemical control have been carried out for years in the West Indies where rust is the major factor limiting peanut production, the latest reports from the area (36, 56) indicate successful treatment has not yet been found.

Mazzani and Hinojosa (176) classified 254 varieties for their resistance to rust under field conditions. Their classification system was based on subjective ratings of R_0 to R_4, the latter indicating that 75% or more of the leaves were covered with uredinia. An entry from Peru, Tarapota, was highly resistant (class R_0). Thirteen varieties were rated R_1, 115 were R_2, 101 were R_3, and 24 were R_4. McVey (169) checked seven entries (Tennesssee Red, Early Runner, Argentine, NC 4x, PI 259746, PI 259747, and a local Valencia type) in a field test in Puerto Rico and all were highly susceptible.

FUSARIUM ROOT, STEM, AND POD DISEASES

The species of *Fusarium* reported to be pathogenic to various parts of the peanut plant include *Fusarium oxysporum* Schlecht. emend Snyd. & Hans., *F. solani* (Mart.) App. & Wr. emend Snyd. & Hans., *F. roseum* (Lk. ex Fr.) emend Snyd. & Hans., *F. tricinctum* (Cda.) Sacc. emend Snyd. & Hans., *F. moniliforme* Sheld. emend Snyd. & Hans. As judged from the pertinent literature linking *Fusarium* and peanut, the evidence required to assign a primary pathogenic role to any fusaria has been largely lacking. Root and stem diseases have been attributed to *F. solani* f. sp. *phaseoli* (Burk.) Snyd. & Hans. by Miller and Harvey (180), to *F. oxysporum* by Rothwell (221), and to *F. oxysporum* and *F. solani* by Frezzi (71). Pod and kernel diseases have been reported to be caused by *F. solani, F. tricinctum, F. moniliforme,* and *F. roseum* (132, 152).

Diseases of peanut caused by fusaria are found throughout the world. Peanut is only one of many hosts on which most of the species listed above are found. The economic importance of the diseases on peanuts, particularly diseases of roots and stems, is slight. Losses due to pre- and post-digging invasions of pods and kernels may be substantial, but estimations of such losses have not been made.

SYMPTOMS

When germinating seedlings are attacked shortly before emergence, a general deterioration results, and tissues become gray, watersoaked, and are often overrun with strands of hyphae. An injury to the tap root, such as that caused by mechanical or chemical damage to the seed or seedling radicle may predispose seedlings to infection. *Fusarium solani* has been isolated from many peanut seedlings showing a dry root rot. The lower end of the tap root becomes brown to reddish-brown, withers, and often curls. Secondary roots become brown and slough off. The disease progresses up the tap root to the hypocotyl. Hypocotyl invasion is often noted only after plants have permanently wilted due to loss of main and secondary root. If plants survive root invasion long enough for adventitious roots to develop above the lesion, the infected plant may survive, particularly if weather conditions are favorable for rapid root growth.

In a detailed report Miller and Harvey (180) outlined the features of a disease of maturing peanut plants caused by *F. solani* f. sp. *phaseoli.* Maximum infection occurred when the hosts were about two months old. The first symptoms were chlorosis and wilting followed by death of the plants. Initial infection of the root just below the crown was seen as small, elongate, slightly sunken, brown lesions. Lesions enlarged to girdle the root about 1-2 cm along the root axis and the cortical tissue became shredded.

Fusarium wilt of peanut caused by *F. oxysporum* was reported from Rhodesia by Rothwell (221). Wilting and development of grayish-green leaves were the first symptoms noted. No external symptoms were seen in freshly dug plants but brown discoloration of tissues beneath the cortex was evident.

Symptoms of pod infection by various species of *Fusarium* are not usually characteristic enough to permit diagnosis by visual inspection (81). In fact the inevitable presence of other fungi on the pod surface, some of which may be pathogenic, can cause symptoms which cannot be attributed to any single fungus species. However, Reichert and Chorin (219) mention the diagnostic value of the violet-whitish color imparted to pods by the growth of *Fusarium* sp.

CAUSAL FUNGI

Taxonomy and Morphology

The species concept of *Fusarium* promulgated by Snyder and Hansen (248, 249, 250) and reviewed recently by Snyder and Toussoun (251) provides a basis for interpreting literature cited in this section on Fusarium diseases:

(1) *F. solani* f. sp. *phaseoli* = *F. martii* Apel and Wr. var. *phaseoli* Burk. as cited in (180).

(2) *F. oxysporum* = *F. angustatum* Sherb. and *F. vasinfectum* Atk. as cited in (71).

(3) *F. roseum* = *F. scirpi* Lambr. and Fautr., *F. scirpi* var. *acuminatum* (E and E) Wr., and *F. equisiti* var. *bullatum* (Sherb.) Wr. as cited in (9); *F. equisiti* (Cda) Sacc. as cited in (145); and *F. recticulatum* Mont. as cited in (212).

(4) *F. tricinctum* = *F. sporotrichioides* Sherb. as cited in (11).

Dissemination and Infection

Species of *Fusarium* persist for long periods in the soil as chlamydospores and as living hyphae in plant debris. Spores form readily on invaded plant parts and are a common inoculum source and a principal form in which the fungi are disseminated.

Garren and Wilson (91) were unable to demonstrate the pathogenicity of *Fusarium* spp. Bell (15) obtained evidence of pathogenicity of *F. oxysporum* and *F. roseum* under favorable conditions for these fungi. Other experiments have shown that root discoloration increases during growth and the apparent root mass at maturity decreases in the presence of *F. oxysporum* and *F. solani.* Miller and Harvey (180) noted that *F. solani* f. sp. *phaseoli* most seriously affected young plants during hot, dry weather. When moisture was sufficient for good plant growth, the damage from *Fusarium* was negligible. In many etiological aspects, this disease of peanuts resembles dry root rot of bean.

F. oxysporum, F. solani, F. roseum or *F. moniliforme* often can be isolated from peanut roots, pegs, pods, or kernels. These species apparently flourish on the subterranean parts of the plant without causing obvious disease symptoms. If their roles are primarily parasitic, the effects of this parasitism have yet to be detected. Pathogenesis, as detailed above, is obvious in some instances but the ubiquity of fusaria on peanut organs and the rarity of discrete disease symptoms suggest that these fungi are pathogenic only under rarely occurring circumstances.

CYLINDROCLADIUM BLACK ROT

Cylindrocladium crotalariae (Loos) Bell & Sobers

Cylindrocladium black rot was first recognized as a disease of peanut in 1965. The pathogen, *Cylindrocladium crotalariae* (Loos) Bell & Sobers, has been isolated from roots, hypocotyls, pegs, pods, and seeds of diseased plants (17). Species of the genus *Cylindrocladium* were widespread in soil, and conidia are air-borne for undetermined distances in splashing or blown rain or irrigation water. *C. crotalariae* is known to cause lesions on leaves of eucalyptus seedlings, *Crotalaria spectabilis* Roth (16), and tea (76). It also causes a collar rot of *Crotalaria anagyroides* L. (76) and is pathogenic to leaves and all subterranean parts of peanut.

SYMPTOMS

The first symptoms on diseased plants in the field are chlorosis and wilting of the leaves on the main axis, followed by chlorosis and wilting of the remaining foliage and blighting of the leaf tips and margins. The main axis often is more extensively affected than the lateral branches. Hypocotyls and tap roots are necrotic and blackened, with necrosis usually terminating near the groundline. Frequently, the entire root system of a diseased plant is destroyed, leaving a blackened and fragmented hypocotyl. Adventitious roots often develop on diseased plants near the groundline. Dark, slightly sunken lesions occur on pegs and pods. Lesions on pods are usually discrete, but occasionally the entire pod is affected. Reddish-orange perithecia of the sexual stage, *Calonectria crotalariae* (Loos) Bell & Sobers, are occasionally visible just above the groundline on moribund stems. These structures are a positive sign of the pathogen that may be seen in the field. Testa of infected seeds exhibit faint and profuse stippling with minute tan specks.

CAUSAL ORGANISM

Optimum growth of the fungus occurs at 26 to 28°C (17). Conidia are produced abundantly on agar containing 0.5% glycerol plus 0.5% phytone and on the

surface of moist eucalyptus leaves (252). Perithecia are formed readily on oatmeal and peanut meal agar, on sterilized peanut stems plated on water agar, and on sterilized, moistened oat and millet seed.

Gadd (76) and Wolf (277) in studying foliar diseases caused by other *Cylindrocladium* spp., stated that damage was most severe under conditions of high relative humidity and abundant free water on foliage. Cylindrocladium black rot of peanut has been observed only in areas of heavy clay soils where the moisture holding capacity is great and waterlogging is common when abundant rainfall occurs.

C. crotalariae was pathogenic to peanut plants inoculted and incubated at soil temperatures of 15 through 40°C. Necrosis of subterranean parts was more intense at 25 to 40°C than at 15 to 20°C (16). Fifty- and 90-day-old plants were more susceptible and were damaged more extensively by the fungus than were the 14-day-old seedlings (16). Infection apparently can occur any place on the subterranean structures. However, in the field it seems to occur most frequently on the tap root and progresses upward infecting the lateral roots, hypocotyl, pegs and pods, and usually terminates near the soil line.

BOTRYTIS BLIGHT

Botrytis cinerea (Persoon) Fries

In many parts of the world species of *Botrytis,* principally *B. cinerea,* have been reported to cause blight of peanuts characterized by infection of leaves, stems, and subterranean organs. Higgins (112) and Orellana and Bailey (200) reported the disease in the United States. It has been reported from Venezuela, the Ukraine, Japan, Tanzania, Rhodesia, and Malawi (91, 138) and this attests to the world distribution of the fungus. The host range of *B. cinerea* includes a great variety of agronomic and horticultural crops. The economic importance of botrytis blight is slight, presumably because the climatic conditions that favor blight outbreaks are not normally encountered in most peanut-growing areas.

SYMPTOMS

Blackened diseased stems are covered with a profusion of grayish conidia and immature infected pods bearing dark sclerotia. The fungus attacks leaves and stems causing a rapid decay of these organs. Infected tissues soon become sparsely covered with dark gray mycelia, conidiophores and conidia of the fungus. The infection progresses rapidly down into pegs and fruit. Flattened or plano-convex, black, irregular-shaped sclerotia develop on decayed stems and pods (Fig. 4).

CAUSAL ORGANISM

Taxonomy and Morphology

The ascomycetous stage of *B. cinerea* was reported (274) as *Botryotinia fuckeliana* (DeBary) Whetzel (=*Sclerotinia fuckeliana* (DeBary) Fuckel). *Botryotina* Whetzel was established to segregate some species, formerly regarded as *Sclerotinia (S. convoluta* Drayton, *S. fuckeliana, S. porri* Bayman Thoe Kingma, and *S. ricini* Godfrey), on the basis of differences in structure of sclerotia. The conidial stages of all *Botryotinia* spp. are *Botrytis* of the cinerea type, but sufficiently different to warrant separate species names (274).

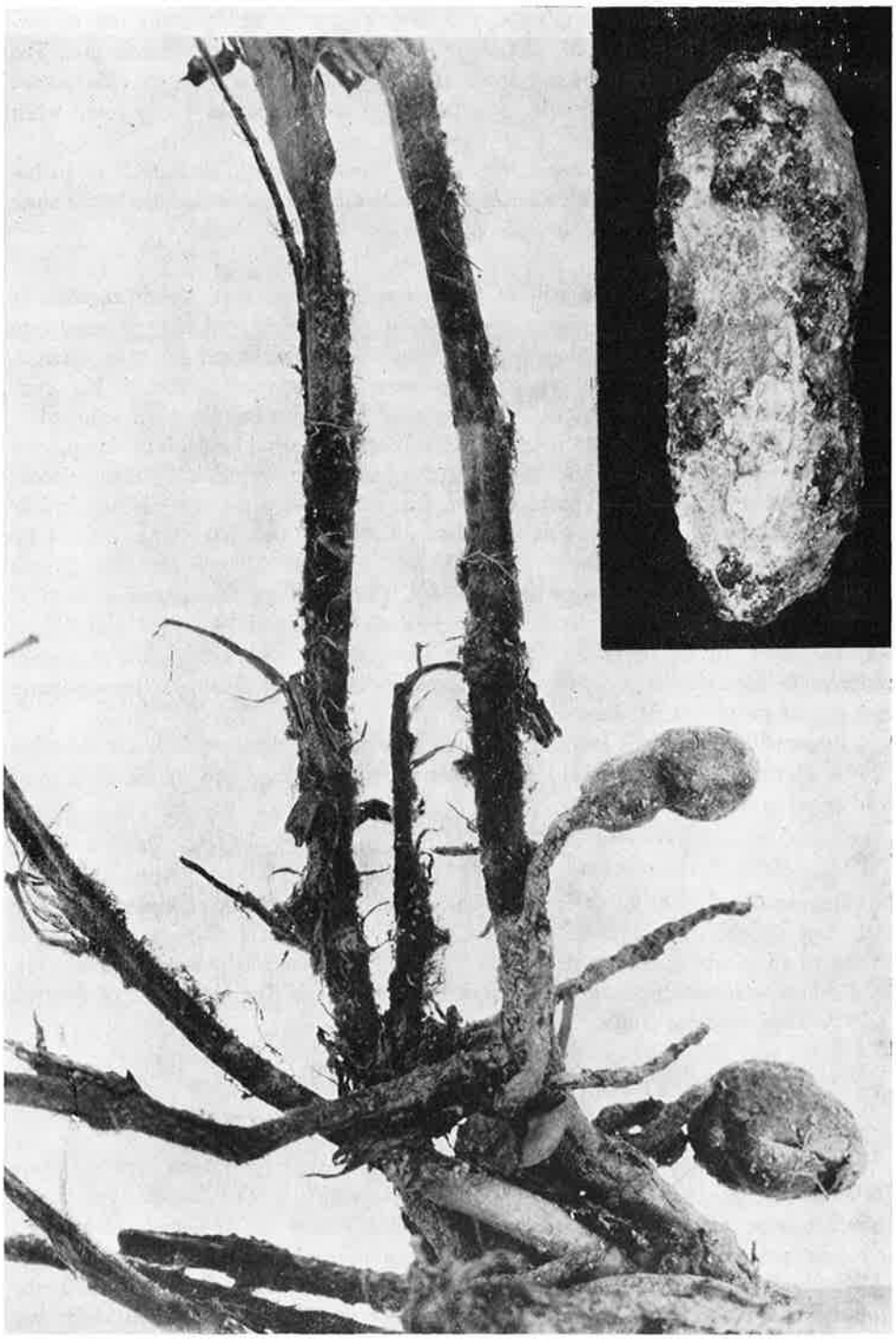

Figure 4. Botrytis blight caused by *Botrytis cinerea.* Gray mycelium and spores on stems. Enlargement showing sclerotia on a pod. (Photo courtesy R. G. Orellana and W. K. Bailey.)

Although *Botrytis cinerea* (Persoon) Fries is extremely variable, any one of several descriptions, such as that of Gilman (99) may be used in its identification. The term "grey mold" is an excellent descriptive name for the organism. Tissues in advanced stage of the disease are covered with grey spores that are released in a grey cloud when the tissue is jarred.

Several reports in the literature *(see* Minor Root and Stem Diseases) of pathogenesis by species of *Sclerotinia* probably have little relationship to botrytis blight since the symptoms usually described are only those of root and collar rots.

Dissemination and Infection

B. cinerea forms abundant conidia which can be disseminated by air currents or water. Sclerotia of *B. cinerea* persist in soil or in plant debris and may germinate to form mycelia, or support conidiophores and conidia superficially on sclerotial surfaces. Sclerotia of some *Botrytis* spp. are known to germinate to form apothecia. However, in the case of *B. cinerea* on peanut, such apothecial formation has not been reported.

Higgins (112) noted that botrytis blight occurred during periods of damp, cool weather in late fall. Orellana and Bailey (200) found that 'Argentine' peanuts inoculated with *B. cinerea* and incubated at several temperatures were severely attacked at 15°C (59°F) but no symptoms developed at 27 ± 2°C (80°F). At 20°C (68°F) blighting was less severe than at 15°C (59°F). Jackson (122) found that growth of *B. cinerea in vitro* over a range of 12 to 36°C (54 to 98°F) was maximum at 20°C (68°F) and mycelial weight decreased progressively at 16 and 24°C (61 and 75°F). Growth below 16 or above 24°C (61 or above 75°F) was scanty. The optimum temperature for infection of peanut may therefore be related closely to the optimum temperature for growth *in vitro.*

Presumably the fungus requires no wounds or necrotic tissue to facilitate infection (200). However, Rothwell (221) noted that the disease developed in the vicinity of dead leaves in contact with stems.

CONTROL

Regulation of planting dates to prevent plants from growing or maturing during cool, wet weather would largely control this disease. Where this is not possible protective fungicidal sprays or dusts would probably be beneficial. One such fungicide, 2,6-dichloro-4-nitroaniline, has been found to be quite effective in control of *Botrytis* and *Sclerotinia* on some crops.

VERTICILLIUM WILT AND POD ROT

Verticillium albo-atrum Reinke and Berth. and *Verticillium dahliae* Kleb.

Verticillium wilts or pod rots (floury rot) on peanut have been reported from the United States (246), Australia (214), and Argentina (72). The two species reported to cause peanut diseases have very wide host ranges which include plants such as cotton, potato, tomato, eggplant, and tobacco and many weeds. Peanut wilt is not widely distributed in the United States but in some areas (246) a large portion of the crop may become affected. Purss (214) states that the disease in Australia is widely recognized and generally thought to be of little importance. He suggested that yield reductions of 14 to 64 percent, as he found in his studies, are of considerable economic importance to peanut growers. Pod rot was recognized by Frezzi (72) in Argentina where in some cases, 58 percent of the pods were destroyed.

SYMPTOMS

Wilt symptoms are usually seen about flowering time as a dull green or chlorotic discoloration of portions of lower leaflets. As the disease progresses many leaflets over the entire plant become withered and brown and fall from the plant. Infected plants may progressively lose foliage until they die, but unless unusually dry weather prevails, infected plants do not die rapidly or exhibit severe overall wilting. If adequate moisture is present infected plants remain alive but are stunted with sparse foliage and are relatively unproductive. Brown to black vascular discoloration can be found in the root, stem and petioles in advanced stages of the disease.

Floury rot of peanut fruit, as described and illustrated by Frezzi (72) is characterized by dark, blackened, rotted pods which are usually sprinkled with white powdery patches composed of masses of conidia of *Verticillium* sp.

CAUSAL ORGANISM

Taxonomy and Morphology

Considerable controversy centers around the validity of maintaining separate taxa for *V. albo-atrum* and *V. dahliae.* Equally cogent opinions support the separation or the synonomy of the two. We have necessarily treated them as two distinct species because they are given as such in peanut literature.

Survival and Dissemination

Verticillium is able to persist for many years in field soil in absence of known host plants. Menzies and Griebel (178) studied survival of *V. dahliae* in uncropped soil and suggested that pseudosclerotia were able to germinate, grow saprophytically and produce conidia in the soil. After a few such cycles sclerotia tended to become depleted of endogenous reserves but could still produce a few conidia. Evans, *et al.* (66) reported that *V. albo-atrum (V. dahliae)* produced abundant microsclerotia in dead cotton plants and that these sclerotia served to disseminate the fungus in bits of plant debris, and contributed greatly to the soil inoculum. Peanut plant debris has not been scrutinized as a source of inoculum, but the report by Purss (214) suggests that infected plants are chief agents of fungus spread. Purss (214) commented that until recently wilt occurred in small patches usually associated with stationary threshing sites, but with the advent of mobile field harvesting equipment peanut debris is spread over the field and the distribution of infection has become more uniform.

Verticillium was found in the 0 to 6 inch and 6 to 12 inch strata of 19 of 20 soils assayed by Wilhelm (275). The fungus was recovered from 3 of 20 soils at a depth of 30 to 36 inches. The 0 to 12 inch layers contained three to four times the degree of infestation as deeper layers. Wilhelm (275) found no relationship between verticle distribution of the inoculum and soil type, climate, or cropping history. However, an experimental infection index was related to past crop history.

Purss (214) commented on the prevalence of wilt on more fertile soils in contrast to poorer agricultural soils and tentatively suggested that the level of nitrogen in better soils aided in disease development. Although verticillium wilt is generally more common on crops grown in neutral or alkaline soils, Wilhelm (276) contended that *Verticillium* was not greatly affected by the soil reaction within the range at which hosts will grow.

Weeds are known to play an important part in the persistence of *V. dahliae* in Australia. *Tagetes minuta* L., *Anoda cristata* Schlecht. and *Xanthium pungens* Wallr.

are listed by Purss (214) as important weed hosts. Wilhelm (276) lists *Solanum nigrum* L. as a prevalent host.

CONTROL

Varietal Resistance

Smith (247) grew peanut varieties on infested sites previously cropped to cotton and found that Valencia and Spanish type peanuts were more susceptible than bunch types. 'New Mexica Valencia' was highly suceptible and 'Georgia Bunch 182-28' was very resistant. The resistant varieties all became infected but vascular discoloration was restricted to the roots and crown.

Chemical and Cultural

Adequate control of *Verticillium* spp. in infested soil has been achieved in a few crops (peppermint, potato) with certain volatile fumigants used on a broadcast or row treatment basis (240). Non-volatile and relatively insoluble fungicides have not proven of much value.

Rotation schedules have been found to be of some benefit. Purss (214) suggested that severely infested land be cropped to grass for an extended period because the longevity of the fungus made the usual maize-peanut rotation relatively ineffective. Hsi (118) found that peanuts following cotton, okra, or peanuts developed severe wilt, whereas less severe wilt occurred following grain sorghum or alfalfa. From the report of Menzies and Griebel (178) one might infer that clean fallow with occasional plowing during dry periods would lead to a significant depletion and death of soil-borne inoculum.

Long term rotation schedules employing clean fallow and a series of non-hosts are possibly the best control measures available now. Field sanitation, such as burning or removing infested plant debris would be beneficial through reduction of inoculum.

BLACKHULL

Thielaviopsis basicola (Berk. and Br.) Ferraris

Blackhull is a disease that occurs sporadically on Valencia peanuts in the Portales area of New Mexico but is not a reported problem in other peanut growing areas of the United States (116). Rotted peanuts and pods with symptoms resembling those of blackhull were pictured by Ciccarone (45). Frezzi (71) reported that *T. basicola* and many other fungi had been isolated but Mason (173) and Hsi (116) seem to be the only researchers who have obtained scientific proof that *T. basicola* actually causes a discoloration (disease) of peanut pods. *T. basicola* has been reported on various crops all over the world, but reports of peanut as a host are uncommon.

SYMPTOMS

Infections of *T. basicola* occur on the external sclerenchymatous shell tissue during the development of the fruit and are first seen as minute black dots (173). As the incidence of infections increase the shell tissue becomes blackened due to the aggregation of chlamydospores in the developing shell. Dark scabrous patches develop where great numbers of lesions have coalesced. The fungus grows throughout shell tissue and produces masses of chlamydospores. The internal shell and testae of kernels often show brown discolorations although Mason (173) was not able to culture the fungus

from discolored kernel tissue. Ciccarone (45) reported that *T. basicola* caused black sooty spots on pods which became irregularly confluent and involved one-half or more of the shell. The kernels eventually had similar symptoms. Despite the role of *T. basicola* as a root rotting agent of most of its hosts, peanut roots are not attacked (173).

CAUSAL ORGANISM

Morphology and Physiology

A good characterization of the fungus *T. basicola* may be found in the book on tobacco diseases by Lucas (160).

The cardinal temperatures for growth *in vitro* are 8, 22 to 28, and 35°C (46, 72, and 96°F). Optimum growth in culture occurs over a pH range of from 4.0 to 6.2 (160).

Dissemination

T. basicola is widespread in the soil and persists indefinitely as a soil saprophyte (160). Mason (173) diagrammed a suggested life cycle to explain the way the fungus persisted under peanut culture. Chlamydospores in the endocarp of the fruit overwinter in the soil in unharvested peanuts. As the pods deteriorate during the ensuing season chlamydospores germinate and produce mycelia which grow along the new pod surface and penetrate into the endocarp. Peanuts are not the only crop that apparently maintain a high level of inoculation in the soil, as Hsi's (117) work with cotton rotations indicates. Hsi (116) indicated that no constant relationship has been found between seed from pods showing blackhull and subsequent disease incidence when these seeds are planted.

CONTROL

Hsi (116) reported that 50 to 60 strains of Valencia peanuts had been tested for their reaction to blackhull but that no consistent host differences were found.

In his studies of blackhull Hsi (116, 117) has summarized factors which appear to decrease blackhull severity. They are: High seasonal temperatures, low middle-season rainfall, light-textured soil with good drainage, acid soil, no excess irrigation water, crop sequences of peanuts following grain sorghum, broomcorn, small grains, or fallow, a late planting date, and sound viable, treated seed.

RHIZOPUS SEED AND SEEDLING ROT

Rhizopus arrhizus A. Fischer,
Rhizopus oryzae Went & Prin. Geerl. Gerlings,
and *Rhizopus stolonifer* (Ehrenberg ex Fries) Vuillemin

Rhizopus seed and pre-emergence seedling rot (seed-bed rot, ground rot, soft rot, crown rot, damping off, pre-emergence damping-off) is a serious disease of peanut that is often not recognized. Species of *Rhizopus* have been associated with rotted, ungerminated seed and dying, emerging seedlings throughout the world where peanuts are grown commercially. The casual fungi are thoroughly cosmopolitan in soil, as facultative parasites, and in air.

Although the disease is fairly restricted to unemerged seedlings, the fungi have been isolated from emerged seedlings and older plants and in at least one instance pathogenesis to emerged seedlings was positively established (15). Accurate estimates

of peanut losses due to species of *Rhizopus* are not available, because most reports merely have related fungi isolated from dead seed or pre-emergence damped-off seedlings.

SYMPTOMS

Seed and pre-emerged seedlings attacked by *Rhizopus* are rapidly decayed in about 36 to 96 hours after planting when soil moisture and temperature are favorable. At this stage a loose mat of mycelium with clinging soil particles enveloping each seed is often observed. Decay is most rapid when infected seed are planted as the fungi apparently revive and become active as soon as the seed begin to hydrate.

The plumule and cotyledonary lateral branches of emerged seedlings growing in axenic culture were attacked occasionally and partially to completely destroyed by *R. oryzae* and *R. stolonifer* (15). Necrosis usually stopped at or just below the cotyledonary axis. Mats of mycelium and black spores may be seen on necrotic tissues.

CAUSAL ORGANISM

Physiology

R. arrhizus grows in culture over a temperature range of 18 to 37°C (284). Growth was extensive at 37°C but characteristic morphological features were most apparent at 26°C. Zycha (284) stated that *R. oryzae* grew in culture and formed dense mycelial mats in the temperature range of 30 to 40°C. Bell (15) reported that the maximum infective temperature of *R. oryzae* probably exceeds 35°C.

Maximum dry weight was produced at 26°C by *R. stolonifer* in stationary liquid cultures of 2% malt extract (130). Kernels inside intact, surface disinfested shells were most rapidly invaded by *R. stolonifer* at 26°C; however the total number of kernels infected was slightly greater at 32°C. No kernels were infected at 38 and 44°C.

Conflicting reports exist concerning the moisture requirements of *R. stolonifer*. Heintzeler (108) stated that spores of the fungus germinated only when the RH was 80% or less but Schmiederknecht (231) found that a RH of 100% was optimum for spore germination. Griffin (104) stated that the minimum RH for germination of spores at 20°C was 84%. Kouyeas (151) determined that 99.6% RH was optimum for maximum growth and the minimum threshold value for growth was 92%.

Dissemination

Spores of *Rhizopus* are air-borne and often comprise a large percentage of the air spore load. *Rhizopus* is also soil-borne and is frequently recovered from field and forest soils, particularly in the rhizosphere by soil dilution, soil plating, and buried slide techniques.

Species of *Rhizopus* are seed-borne and peanut seed in particular are frequently infected with these fungi. Planting infected seed promotes an increased inoculum and leads to reduction in emergence and stand.

Saksena (224) found that *R. stolonifer* was more abundant in the upper 6 inches of soil than at lower depths. In one instance a *Rhizopus* sp. was isolated more frequently from acid than from alkaline soils (194). Martin and Graham (172) stated that these fungi developed most profusely immediately after the addition of organic matter to soil and waned when soluble components were exhausted. *R. stolonifer* was found to be an important factor in binding soil particles together and promoting flocculation (263).

R. stolonifer and *R. arrhizus* produced zygospores and chlamydospores in culture which remained viable for 18 and 58 months, respectively (209). Atkinson (10) determined that *R. stolonifer* remained viable in dried soil cultures after five years.

CONTROL

Proper treatment of seed peanuts with broad spectrum and vapor action fungicides is an effective and economical way of controlling rhizopus seed and pre-emergence seedling rot. Seed treatment is usually beneficial regardless of seed quality and is particularly valuable when seed stocks are highly infested with *Rhizopus* spp. or are physically damaged.

PYTHIUM DISEASES

Pythium myriotylum Drechsler and *Pythium* spp.

INTRODUCTION

Pythium pod rot occurs erratically in temperate and semi-tropical regions and in semi-arid and arid regions where extensive irrigation is practiced. Other less clearly defined diseases of peanut caused by *Pythium* spp. are post emergence damping-off, root rot, and wilt of older plants. One of the primary casual agents, *P. myriotylum,* is distributed widely in the warmer climates. In addition to reports in the United states it has been reported as a root and fruit rot and damping-off pathogen of various hosts in other areas of the world.

Economic losses due to pythium diseases are difficult to assess because extensive reports are lacking. The pod rot phase is apparently much more important on the large fruited-seeded "Virginia" type of peanuts than on "Spanish" or "Valencia" types. Garren (82) proposed that the pod rot of Virginia peanuts caused by *Pythium* sp. and possibly by *Rhizoctonia solani* was important enough to be given the distinctive name "pod breakdown." By 1968 pod breakdown was recognized as a cause of major losses to peanut growers in North Carolina and Virginia, and Wells (270) stated that pythium pod rot is highly important in North Carolina in terms of monetary loss to growers. In Georgia, McGill (165) observed the disease in occasional fields in counties containing a large peanut acreage. Losses within an individual field ranged from almost nil to about 80% of the fruit set. Garren (78, 80, 83) demonstrated that pythium pod rot caused losses approaching 450 lb/A in certain fields in Virginia. He developed the concept (78) that certain fields have a definite, though fluctuating, potential for pythium pod rot; whereas in other fields pod rot was caused mainly by *R. solani* (see Rhizoctonia Diseases).

Garren (83) demonstrated the pathogenicity of *P. myriotylum* to peanut pods. In one study detached pods of 'Virginia Bunch 46-2' were inoculated with an isolate of the fungus from rotted pods, and all pods were decayed within seven days. In another study attached and detached pods of 'Virginia Bunch 46-2' and 'Dixie Spanish' were inoculated, and all pods were decayed within eight days. The fungus was reisolated from decayed pods in both tests.

In Northern Nigeria, Perry (205) reported that *P. myriotylum* is suspected of causing a vascular wilt of maturing peanut plants. The fungus was consistently isolated from discolored stelar tissue that had been surface disinfested. Jackson and Bell (138) observed a similar malady in Tifton, Georgia, in plots of maturing breeding lines. In

some lines 7 to 15% of the plants were affected. The vascular tissue was discolored and *P. myriotylum* was isolated regularly from surface disinfested pieces of stelar tissue.

Other *Pythium* spp. are known to be pathogenic to peanut. According to Rangaswami (215), Wager in 1931 reported from South Africa that *P. ultimum* Trow caused a root rot of peanut, and Shaw in 1936 and Edson and Wood in 1937 in North Carolina isolated a *Pythium* sp. (identified as *P. ultimum* by Wood and Nance in 1938) from peanuts having a root rot (138). Frezzi (70) in Argentina showed that *P. debaryanum* Hesse was pathogenic to the variety 'White Santa Fe' in both greenhouse and field tests. Plants were exposed by placing inoculum in the fruiting zone. The result was 25 to 30% rotted pods. Necrosis was evident four to six days after inoculation and extended over the entire surface of the pods in 10 to 15 days. On mature fruit, necrosis was limited to the shell, but immature fruit were completely destroyed.

Frezzi (70) found that *P. irregulare* Buisman and *P. ultimum* were responsible for rot of both immature and mature fruit. The former fungus was more predominant in May, June, and July. Frank (69) surveyed peanut fields in Israel and he stated that pythium pod rot caused by *Pythium* spp. was found frequently and caused substantial losses.

SYMPTOMS

Pod Rot

Both immature and mature pods may become infected. In either case the first symptoms are light browning and extensive watersoaking of the tissue. Infection spreads rapidly and in two to four days the entire pod appears watery with a brown-black necrosis (Fig. 5). Immature pods are usually completely destroyed. Garren (80) states that a rapid and general breakdown of the shell occurs in mature fruit. Seeds in mature fruit show various degrees of watersoaking and brown-black necrosis.

Pegs may be infected and destroyed by *P. myriotylum* as they contact wet soil. When this type of infection is extensive, plants removed from the soil have many rotted and blackened peg tips.

Damping-off

The first symptom is a rapid and total wilt. Frequently water-soaked necrotic tissue can be seen on the hypocotyl and cotyledonary lateral branches at the groundline. An elongate, slightly sunken, tan-brown lesion may partially or completely encircle the stem and extend upward 2 to 4 cm. Diseased seedling frequently topple over at the groundline.

Vascular Wilt

The first noticeable symptom is wilting of one or more branches. This is followed rapidly by chlorosis and scorching of the foliage on the wilted branches, with necrosis beginning at the margins of the leaflets, and rapidly extending inward until the entire leaflet and soon the entire leaf is dry and crinkled. The entire plant seldom wilts at once. The petiolules frequently become dry but the petioles often remain green. Perry (205) stated that the vascular system of a wilted plant, particularly in the taproot-hypocotyl region, shows a dark brown-black discoloration when sectioned longitudinally. The discoloration extends from the hypocotyl downward 6 to 16 cm, and to a lesser distance upward into the main axis and lateral branches. Xylem of severely wilted plant is often shredded in the hypocotyl region. Perry (210) found the xylem walls of diseased roots were discolored, and the vessels frequently contained aseptate fungal hyphae which sometimes filled the lumens. Hyphae were sparse in the lower

parts of the root where discoloration was less intense. Porter (211) made what seems to be the first study in which a wilt of peanuts was artificially produced by infesting soil in which the peanuts were growing with a *Pythium* sp. *(P. myriotylum)*. He found that the small fibrous roots were heavily attacked and most of them destroyed.

Root Rot

Root rot frequently occurs in conjunction with pod and peg rot and while it is reported to sometimes occur in mild forms in conjunction with vascular wilt in Southeastern United States (138) it was regarded by Porter (211) as the primary cause of the wilting of Virginia peanuts.

Where root rot occurs separately from pod and peg rot it may become evident from about the early-bloom through the early-mature growth stages. The First noticeable symptoms are stunted growth and loss of natural luster grading into chlorosis of the foliage. Diseased plants often wilt during the day and recover partially at night. Large plants are seldom killed but rarely recover fully from extensive infection.

The smaller fibrous roots are often extensively decayed. Primary and secondary roots and the terminal portion of the taproot are particularly or completely destroyed. Total volume of the root system is greatly reduced. The individual roots are light tan

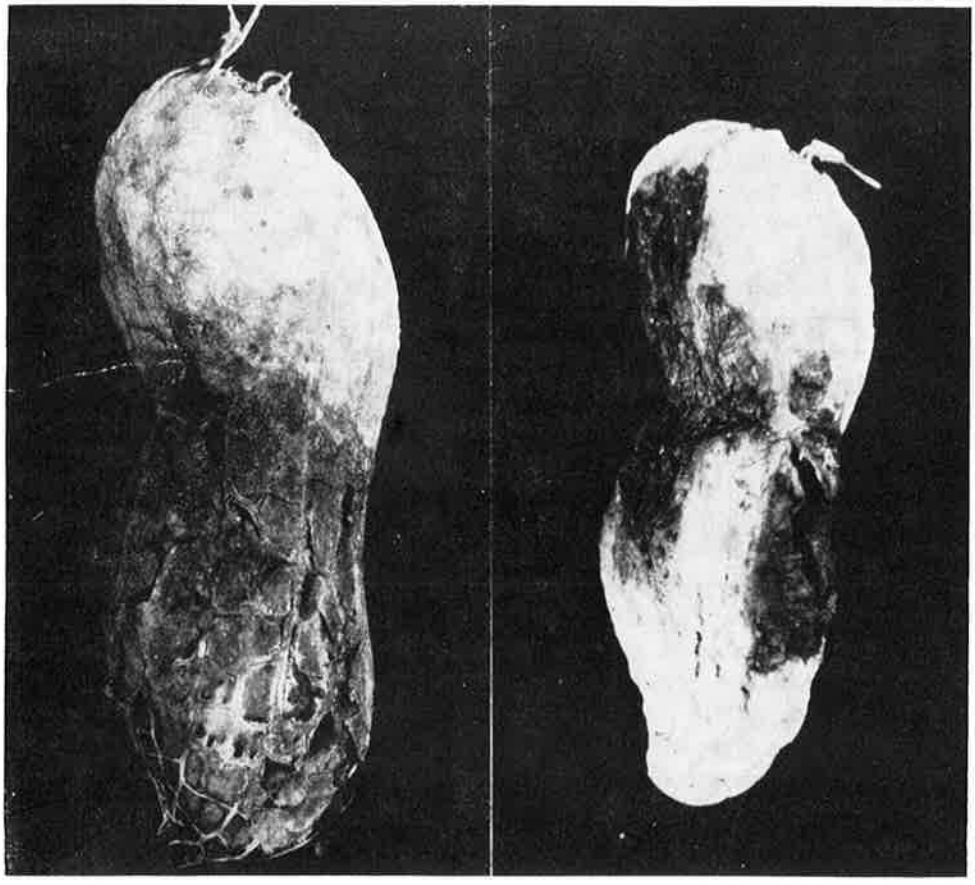

Figure 5. Close-ups of two pods typical of those rotted by *Pythium myriotylum* in Virginia. Sometimes *Rhizoctonia solani* also can be isolated from the type of pod rot shown on the right.

to dark brown, sometimes shading into black, and the entire root system has a soggy and clumped appearance. Cortical tissues disintegrate rapidly and can be readily sloughed-off leaving a fragmented and nonfunctional stele.

Physiology of the casual organisms

Middleton (179) studied the relation between temperature and growth of *P. myriotylum* (which seems to be the most important *Pythium* sp. on peanuts from the world-wide viewpoint). He found it grew best at temperatures between 34 and 37°C (90 and 99°F) and that it would not grow at temperatures below 10°C (50°F) or above 43°C (110°F). He stated that isolates of the fungus from different locations had the same temperature requirements. Littrell and McCarter (158) found that isolates from different hosts and locations varied somewhat in cardinal temperatures. On V-8 juice agar the average optimum temperature range of nine isolates was 33 to 35°C. A maximum of 43°C was evidence for five of nine isolates and 41°C for the other four. The minimum for all isolates was near 11°C.

Thus, by and large, pythium diseases of peanut seem to be associated with high soil and ambient air temperatures in both greenhouse and field. Garren (83) found that pythium pod rot was more severe on detached and attached pods of 'Virginia Bunch 46-2' and 'Dixie Spanish' at 32 and 39 than at 22 and 27°C. Peanut seedlings cultured and inoculated with *P. myriotylum* under axenic conditions exhibited more extensive necrosis and damping-off at 24 and 29 than at 18 and 35°C (15). Plumules were more extensively damaged than roots at all temperatures.

High soil moisture seems to predispose peanut to infection by *Pythium* spp. Frezzi (70) stated that high soil moisture was positively related to pythium pod rot. Frank (69) determined that numerous light irrigations applied at weekly intervals increased the intensity of pythium pod rot, as compared with equal, total amounts of water applied every two weeks; plots irrigated weekly had 40% diseased pods, whereas those irrigated biweekly had 26 percent.

When the soil is warm and contains free water, *P. myriotylum* may produce abundant motile zoospores. These, in turn, are readily transportable by moving water, and they can also swim for limited distances. Oospores and hyphae are likely present both in diseased host tissue and the surrounding soil environment. These structures may be transported by moving water and by cultivation and harvesting equipment.

CONTROL

Frank (69) reduced pythium pod rot in arid areas of Israel by reducing irrigation frequency from weekly to biweekly. He also found that the disease was reduced after the blossoming stage by allowing the top soil to dry for periods exceeding two weeks.

Garren (80) stated that, in Virginia, applications of high rates of $CaSO_4$ (landplaster, gypsum) to the soil surface over the fruiting zone at peak flowering decreased rotted pods at harvest from 8% with no gypsum to 1.5% with 2000 lb/A. Yield increased correspondingly by 450 lb/A. Deep plowing to bury organic litter was practiced in conjunction with the applications of gypsum. Garren, *et al.* (87) believe that control with the gypsum results from depletion of inoculum and increasing physiological resistance of the pods to infection. Garren (81) also obtained substantial reductions in pythium pod rot with sodium-p(dimethylamino)benzene diazosulfonate, a pythium-specific fungicide.

Wells (270) stated that applications of gypsum in North Carolina have been partially to largely ineffective. He obtained from 80 to 2058 lb/A increases in yield

with soil fumigation, by using a broad spectrum fumigant which combined nematocides and methylisothiocyanate. Wells has also used soil fungicides with varying degrees of success. The most effective combination, a *Pythium*-specific plus a broad spectrum material, increased the monetary return of $170.49 per acre over the control.

Seed treatment with broad spectrum and vapor action fungicides provides a means of partially controlling the post emergence damping-off phase.

MACROPHOMINA DISEASES

Macrophomina phaseoli (Maublanc) Ashby
(Rhizoctonia bataticola (Taub.) Butler)

This fungus has been reported to cause wilt, root rot, stem rot (dry rot, charcoal rot, ashy stem blight) and leaf spots of seedlings and older plants and to be an important cause of gross deterioration of shells and kernels (blacknut). In many instances it is an important cause of concealed damage which results from growth of the fungus within the testa between the inner flat faces of cotyledons (25). *M. phaseoli* is found throughout the world on a large number of hosts and in soil mycoflora. Such crops as bean, cowpea, sunflower, sweet potato, cantaloupe, tomato, corn, and sorghum are hosts, as well as many weeds. It has been studied extensively as a cause of a black rot of sweet potatoes (258). The economic importance of the disease in maturing peanut plants is slight. The deterioration of fruit beginning at maturity in the soil may be economically serious in some areas. Bouriquet and Jaubert (25) reported an instance of 34 percent loss of peanut seed in Senegal. Peanut diseases caused by *M. phaseoli* have been reported from Gambia, Argentina, Venezuela, India, Israel, and the United States. (See references cited by Jackson and Bell (138).)

SYMPTOMS

Stems and Roots

Watersoaked necrotic areas develop on stems at the groundline. As the disease progresses infected areas become dull brown and extend up the stem into the branches and down into the roots. If the initial lesion girdles the stem, wilting follows. However, wilt symptoms are not characteristic of a vascular wilt disease but result from girdling of stem or destruction of root tissue. When stems are girdled plants die and the pathogen invades aerial portions of the branches with great rapidity. A blackening of the entire, partially defoliated stems may be seen. Sclerotia of *M phaseoli* develop profusely in invaded plant parts and pycnidia are found in some instances. The abundant sclerotia give invaded tissues a sooty appearance.

Although root rot has been given as a characteristic symptom of *M. phaseoli* infection of peanut (219), details of progressive disease development are lacking. In our experience root rots that may be attributed to this pathogen have always been associated with stem rotting. However, root infection can occur independently (219) and leads first to a blackening and later to complete rot of the taproot.

Fruit

The fungus may be cultured from the shell surface, shell tissue, and kernels from intact pods that show no visible evidence of its presence (137). When peanuts are physically damaged before or after harvest or when inclement weather prevents prompt harvesting, *M. phaseoli* will grow rapidly throughout shells and into kernels. This

abundant growth and sclerotia development commonly is evident as a blackening of internal and external shell surfaces.

Leaves

Leaf symptoms that have been reported consist of large marginal zonate spots in which pycnidia are found. Infection of leaflets is apparently rare as judged by the lack of published information.

CAUSAL ORGANISM

Taxonomy and Morphology

Jackson and Bell (138) gave a detailed review of the taxonomy and morphology of this organism and stress their reasons for calling the sterile state of *M. phaseoli* "Rhizoctonia" instead of "Sclerotium" (222). Readers interested in these aspects are referred to pages 92 through 95 of Jackson and Bell (138).

Physiology

Norton (196) gave 30 to 35°C (86 to 95°F) as the temperature range optimum for growth of *R. bataticola in vitro.* Livingston (159) found that a pH range of 5 to 8 was suitable for rapid growth. He also found that the fungus was able to grow on a wide variety of carbohydrate and nitrogen sources.

Dissemination and Infection

The fungus persists in the soil for long periods as actively growing mycelia or dormant sclerotia. The role of chlamydospores in persistence of the fungus has not been reported. Several studies have shown that growth and survival of *R. bataticola* in soil, on plant parts, and in mixed cultures are greatly influenced by other microorganisms (92, 196, 198). Jackson (131) found that *R. bataticola* invasion of hydrating peanut pods was halted by concomitant growth of *Aspergillus flavus* (Lk.) Fries and that propagules of invading *R. bataticola* were killed by growth of *A. flavus.*

Dissemination of the fungus is effected by movement of infested plant debris and soil. Since shells and kernels are important sites of infection, the fungus is probably disseminated widely by these plant parts (24, 126, 133).

In Georgia infection of peanut fruit is mainly restricted to maturity or postmaturity phases of peanut culture. In artificially dried peanuts, the fungus was significantly more abundant when peanuts were dried slowly rather than rapidly. Harvest impact damage before drying had no apparent effect on the incidence of infection (135, 136).

Infection of seedlings was more rapid and severe at 29 and 35°C (84 and 95°F) than at 18 or 24°C (64 or 73°F) (15). In every case plumules were invaded more frequently than roots. Livingston (159) found that the fungus entered plants (corn and sorghum) through the fine fibrous roots and grew intercellularly through the root system to the stem. Similar findings are lacking in peanut. Infection of older plants is well documented in the literature (138), but in the Southeastern United States root or stem diseases of peanut which are clearly initiated by *R. bataticola* are rare. The explanation for this lack of pathogenicity by a fungus that is known to be abundant in our soils is possibly due to climatic or varietal factors. Livingston (159) found that either low soil temperature or high soil moisture prevented occurrence of root and stalk rot in sorghum and corn. The disease was favored by low soil moisture and high temperatures. Similarly, Norton (196) found that guayule was most often attacked during hot, dry periods and he suggested that these conditions had a debilitating effect

on the host thereby increasing susceptibility. Norton found that application of water almost competely controlled the disease even though high temperatures prevailed. Hoffmaster, *et al.* (114) believed that the fungus was moderately and variably aggressive and invasion was favored by devitalization characteristic of plants subjected to environmental extremes, wounds, or attacks by other fungi. By these criteria areas that receive ample rainfall (such as the Southeastern United States) might therefore be relatively free from this disease.

CONTROL

The scarcity in the Southeastern United States of root and stem rots caused by *R. bataticola* may be due unknowingly to the use of resistant varieties. Bouhot (24) reported differences in susceptibility to pod infection in four numbered varieties and Mathur, *et al.* (174) recently found two of four tested varieties to be resistant to root rot.

Rotation schemes have not been extensively tested, but Livingston (159) reported no satisfactory results from short-term rotations. The reduction of soil-borne inoculum in this way would possibly be inadequate because of the saprophytic nature of the fungus and its wide host range. Various comments (114, 159, 196) concerning effects of host vigor and environmental conditions as they affect disease potential suggest that an effective control program must include measures that enable plants to maintain good growth, such as adequate fertilization, irrigation, and pest control.

Bouhot (24) found pentachloronitrobenzene of value in reducing fruit infection. Although much work has been done in the United States with soil fungicides, the disease of peanut caused by *R. bataticola* have been so minor that little data have been collected on chemical control of this fungus.

Seed treatment fungicides eradicate or suppress growth of *R. bataticola* in kernels and provide effective control during germination (14). However such control is ephemeral as Bell (14) showed and *R. bataticola* in the soil is not controlled for long periods.

PHYLLOSTICTA LEAFSPOT

Leafspots of peanut caused by *Phyllosticta* spp. cause minor damage and are found throughout the world. Such diseases have been reported from Rhodesia, Senegal, India, Argentina, Venezuela and the United States (175, 239, 243).

SYMPTOMS

Frezzi (71) describes the lesions as circular to oval, 1.5 to 5 mm in diameter, with definite borders, halos absent, reddish brown on the perimeters becoming lighter or tawny in the centers. At times leaflets become perforated, leading to a shot-hole aspect.

Chevaugeon (37) found lesions predominantly near the tips of leaflets, along margins, and extending along the midrib. Unlike Frezzi (71), Chevaugeon noted a chlorotic halo surrounding spots which were pale brown to reddish-brown with a dark brown perimeter. Rothwell (221) also noted wide chlorotic zones around the circular, tan lesions.

Rao (217) described lesions caused by *Phyllosticta arachidis-hypogaea* V. Rao as irregular in outline, mostly marginal and apical, scattered, dark-brown, and epiphyllous.

Comparative size and shape of lesions in his drawings suggest a disease quite unlike the leafspot pictured by Frezzi (71). Differences in reactions of peanut varieties might account for some variation in symptoms. However, such a large disparity may well be the result of different pathogens.

Vasant Rao (217) described *P. arachidis-hypogaea* as the cause of a leafspot in India. *P. sojaecola* Massal was found to cause a leafspot in Africa.

PHOMOPSIS DISEASES

Phomopsis sojae Lahman

Several reports in the literature refer to peanut leaf and stem diseases caused by *Phomopsis* or the related sexual stage, *Diaporthe.* In the United States *Diaporthe sojae* Lehman was reported to be the cause of stem blight of peanut by Atkinson (9). Luttrell (161) found *D. phaseolorum* (Cke and Ell.) Sacc. var. *sojae* (Lehman) Wehmeyer on dead stems and stipules of peanut plants collected in several locations in Georgia. Frezzi (73) found a *Phomopsis* sp. to be the cause of leaf scorch of wild peanuts in Argentina and he was able to obtain infection of cultivated varieties with this fungus.

SYMPTOMS

No characteristic stem symptoms were reported by Atkinson (9). Luttrell (161) noted that dense parallel rows of pycnidia gave the dead stems a blackened appearance. Phomopsis is usually associated with *Leptosphaerulina crassiasca, Cercospora* spp., or *Colletotrichum* spp. in marginal nectoric leaflet lesions. Such lesions are brown to black, often with a distinct chlorotic zone between healthy and necrotic tissue, and commonly advance from the leaflet tip to the petiole along a wedge-shaped infection front. Pycnidia of *Phomopsis* spp. are usually found in rows paralleling the midribs or smaller veins. *Phomopsis* spp. have been isolated from discrete lesions in the center of leaflets. Lesions are small, circular to irregular, 1 to 10 mm in diameter or length. Centers of lesions become papery, white to light brown, with pycnidia developing in dead tissue. Lesions are surrounded by a distinctive reddish-brown margin.

CAUSAL ORGANISM

Frezzi's (73) *Phomopsis* sp. was determined to be the conidial stage of *D. sojae,* the same fungus reported by Atkinson (9). *D. phaseolorum* var. *sojae,* a derivation from *D. sojae* representing the same taxon, was found by Luttrell (161) on dead peanut stems. The pycnidial state of *D. phaseolorum* var. *sojae* is *Phomopsis sojae.*

ANTHRACNOSE

Colletotrichum spp.

Anthracnose of peanut is reportedly caused by three named species of *Colletotrichum.* Small (242) in 1926 was perhaps the first to record the presence of an unnamed species of *Colletotrichum* on peanut in Uganda although Sawada (229) in Taiwan collected specimens from peanut in 1909. In 1952 Chevaugeon (37) reported *C. mangenoti* Chevaugeon from peanut in Africa and Silvestre (239) later reported this species as being important in Senegal. Wallace (264) recorded an anthracnose fungus similar to *C. capsici* (Syd.) But. & Bis. in Tanzania. In India *Colletotrichum*

sp. was found on peanut by Melta (177). Larsh (155) reported W. W. Ray's diagnosis of *Colletotrichum* sp. in stem lesions from peanuts collected in Oklahoma. Frezzi (73) described a species of *Colletotrichum* that occurs infrequently in Argentina on wild species of Arachis. Saksena, *et al.* (225) have recently reported that *C. dematium* (Pers. ex Fr.) Grove is the cause of a leaf spotting disease in India.

SYMPTOMS

Chevaugeon (37) described lesions caused by *C. mangenoti* as brownish gray, visible on both leaflet surfaces, and rarely on petioles or stems. His illustrations show marginal, elongate to circular lesions. Silvestre (239) noted that lesions, caused by the same species, were large, involving up to half the leaflet. Sawada (229) described leaflet lesions caused by *C. arachidis* Sawada as scattered, 3 to 6 mm large, circult to irregular, with grayish white centers surrounded by dark brown borders.

Saksena, *et al.* (225) described the symptoms caused by *C. dematium* as small water-soaked yellow spots which become dark brown and enlarge to 1 to 3 mm in diameter. Under favorable conditions the spots grow rapidly, become irregular, and spread over the entire leaflet. The disease may gradually extend into petioles and branches and cause death of the entire plant. Acervuli are abundant in diseased tissue.

Frezzi's (73) illustration of symptoms of *Colletotrichum* sp. on *Arachis* sp. shows an apical leaf scorch similar to that caused by *Leptosphaerulina crassiasca* (Sechet) Jackson & Bell. He noted that affected tissue is very dark, and firm and does not tend to disentegrate.

CAUSAL ORGANISM

The morphology of *C. mangenoti, C. arachidis,* and a species reported by Frezzi is given in the report by Jackson and Bell. The possible relationships of these species to other species of *Colletotrichum* are discussed in this same report.

SCAB

Sphaceloma arachidis Bitancourt and Jenkins

Scab (verrugose) was first recognized in Brazil prior to 1940. The disease occurred in severe proportions in 1938 but was less severe in following years. There is no reference to a recent serious outbreak of scab in Brazil. However, Ojeda (199) reported recently the occurrence of the disease in the province of Corrientes, Argentina. CMI map 231 shows scab confined to a small area of Brazil corresponding generally to the state of Sao Paulo. *S. arachidis* is presumably pathogenic only on species of *Arachis.*

SYMPTOMS

Bitancourt and Jenkins (20) described symptoms on leaves as small spots, round to irregular, visible on both surfaces, with sunken centers and raised margins, spreading or frequently distributed just beside or on both sides of the principal vein, at times confluent. On the upper leaflet surface spots become tan (tillene buff) with narrow, brown, marginal lines. Lesions are frequently covered with continuous velvety layers of grayish olive conidiophores and conidia of the pathogen. Conidia later fall away exposing dark brown to black acervuli. On the under surface, the spots are pinkish brown to red, at times with a brown margin. On petioles and branches lesions are

more numerous and larger, oval, prominent, measuring to 3 mm in length, at times coalescing in more or less extensive areas, causing distortion of branches and petioles. Cruz, *et al.* (56) and Ojeda (199) reported that the disease affected all aerial and tender parts of the plant. Lesions appear like stains of cankerous growths which may cover more than 80% of the stem making them appear very wavy or sinuous.

CONTROL

Ojeda (199) reported a high degree of resistance in the varieties 'Guayurü' and Overo and some resistance in Colorado Manfredi and 'Prudente INTA'. 'Manfredi 1', 'Manfredi 68', 'Manfredi Champaqui', 'Blanco Rio II', 'Blanco Santa Fe', and races of the subspecies *fastigiata* Waldron were susceptible.

YELLOW MOLD

Aspergillus flavus (Link) Fries

Yellow mold occurs sparsely throughout the peanut production areas of the world. The casual fungus, *Aspergillus flavus,* is reported most commonly inhabiting pods and seed. The fungus has an extremely wide host range throughout the plant kingdom.

In terms of pathogenesis *A. flavus* generally is restricted to seed of various hosts. However, since it is pathogenic to emerging peanut seedlings Jackson & Bell (138) proposed that the common name 'yellow mold', be inclusive of seed and pre-emergence seedling rot. The name denotes masses of yellow-green spores produced on infected tissue and prevents confusion with 'crown rot' caused by *Aspergillus niger* and 'Rhizopus seed and seedling rot'.

Gibson and Clinton (98) stated that *A. flavus* caused a dry rot of cotyledons prior to emergence. This condition was most noted at seven to nine days after planting. Clinton (46) determined that germinating seed were readily infected and destroyed. Infections of emerging seedlings generally occurred about seven days after planting. Seedlings were killed prior to emergence but after emergence, death seldom occurred. However, the fungus may persist in senescent cotyledons at least 31 days after planting (14). Plumules of seedlings inoculated with the fungus in axenic culture were extensively damaged (15). The fungus appears to be concentrated on pod surfaces and in the testae.

SYMPTOMS

Seed and unemerged seedlings attacked by *A. flavus* are reduced to a shriveled, dried, brown or black mass within four to eight days after planting. During this time masses of yellow-green spores may be seen. After six to eight days the seed is indistinguishable from surrounding soil. Decay is most rapid when infested seed are planted where the fungus becomes active as the seed hydrate. Cotyledons of germinating seed are usually invaded first and, under favorable conditions, the emerging radicle and hypocotyl are decayed rapidly. If conditions become unfavorable for disease development after the initial infection, the fungus may quiesce and persist in the cotyledons until they are completely decomposed.

CAUSAL ORGANISM

Physiology

The interaction of temperature and moisture are major factors controlling growth of *A. flavus* on artificial and natural substrates. Jackson (130) found that a maximum dry weight was produced at 38°C on malt extract broth and growth declined slowly at 21°C and sharply at 16 and 44°C. Sporulation was abundant from 21 to 38°C. Maximum infection of whole pods occurred at 32°C, and infection declined slowly at 16°C and sharply at 44°C. Bell (15) obtained disease indices of 14, 29, 50, and 54, in a range of 0 to 100 on inoculated seedlings in axenic culture at 18, 24, 29 and 35°C, respectively. Tandon and Chauman (256) found that the thermal death point of single spore cultures on Czapek's medium was 54°C. No growth occurred at 6°C and maximum dry weight was produced at 20°C, with a slight reduction up to 30 and a considerable one at 37°C.

Inoculated pods were hydrated up to 6 days at three temperatures (133). Infected seeds rose from 0 to 3% at 26°C between the fourth and sixth day. At 32°C infection increased from 0 to 15% between the second and fourth day, and to 42% the sixth day. At 38°C infection rose from 0 to 38% the second and fourth days, and to 57% the sixth day.

Growth and sporulation of *A. flavus* is affected by light. Tatarenko (257) found that intense white light retarded sclerotial formation. Weak light stimulated conidial formation. Darkness increased mycelial growth. After prolonged cultivation in darkness the fungus lost the ability to produce conidia and degenerate forms occurred. In partially degenerate cultures conidial formation was enhanced by cultivation in weak light and the effect was transmitted to subcultures.

In ecological studies *A. flavus* has shown antagonism to certain other fungi. Jackson (131) showed that when peanut shells and kernels were simultaneously inoculated with *A. flavus* and *Rhizoctonia bataticola,* growth and spread of the latter were reduced. Growth of *R. bataticola* on malt agar also was competitively reduced. When Norton (198) paired the two fungi at opposite ends of soil columns, *A. flavus* inhibited but did not overgrow *R. bataticola.* Nain and El-Esawy (195) stated that *A. flavus* from the rhizosphere of cotton was antagonistic to *R. solani* in culture. Bedi (13) reported that sclerotia of *Sclerotinia sclerotiorum* (Lib.) DeBary were invaded and killed by the fungus when buried in compost heaps. Minton and Jackson (190) demonstrated an interaction between *A. flavus* and *Meliodogyne arenaria* (Neal, 1889, Chitwood, 1949) in infestations of peanut pods.

Dissemination

A. flavus produces abundant conidia, sclerotia, and hyphal fragments, all of which are easily transportable by natural and man-made agents. The fungus will grow, to some degree, on practically any plant material in, on, or above ground. It also grows reasonably well in practically any type of soil. Lily (156) reported that *A. flavus* was a rapid recolonizer of steam sterilized soil in contact with non-sterile soil.

CONTROL

The indications are that *A. flavus* is a relatively weak and minor pathogen on growing peanut plants. Cultural practices which promote vigorus growth and control other disease and insect pests would probably control *A. flavus.* However, when the mature plants are brought above ground, the fruit becomes highly susceptible to infection by the fungus. Harvesting procedures which damage the pod or seeds or

both greatly increase the chances of seed infection. Such practices should be avoided. Proper curing by artificial means and subsequent cool, dry storage have been effective in preventing proliferation of the fungus on harvested peanuts.

Treating seed peanuts with broad spectrum and vapor action fungicides is a highly effective economical means of controlling *A. flavus* in seed bed. Soil treatment with fungicides and broad spectrum fumigants, while partially effective in controlling the fungus on peanut fruit, is currently not practical or economical.

POWDERY MILDEW

Oidium arachidis Chorin

Powdery mildew of peanut foliage was reported from Israel (41) in 1961. The disease had been seen in the coastal plain of Israel as early as 1941 but its occurrence is very sporadic. There is no other detailed reference to powdery mildew of peanut although Hirata (113) lists *Erysiphe communis* (Wallr.) FR. and *Erysiphe pisi* (PC) Sh-Amans as having been reported on peanut in Mauritius, Portugal, and Tanganyika.

Initial symptoms occur in midsummer and as the disease progresses the upper surfaces of leaflets become covered with large spots, the centers of which can be distinguished by the brownish appearance of necrotic tissue. The superficial hyaline mycelium usually spreads radially and bears abundant conidiophores and oidia.

A temperature of about 25°C (77°F) favored rapid and intense development of the disease in Israel (41). Two varieties were checked for resistance, one seemed more resistant than the other (41).

MELANOSIS

Melanosis of peanut leaves was described in detail by Frezzi (71) but has not been reported from any country other than Argentina. The disease has occurred at times in an intense and widespread form on peanuts, especially on one variety. Its importance is minor.

Symptoms consist of very small irregularly circular, oval, or elongate, dark brown spots, solitary or confluent, 0.5 to 1 mm in diameter for circular lesions and up to 1.5 mm long for elongate lesions. At times they are numerous enough to cover the entire abaxial leaflet surface giving the appearance of being covered with fly specs. Lesions are slightly submerged at first becoming elevated and crust-like with age. Defoliation does not occur even in severe cases.

Isolations from lesions often yielded a *Macrosporium* sp. and *Alternaria* spp. (71). Although the latter fungi were more frequent, inoculation experiments showed that only *Macrosporium* sp. produced the original symptoms. Jackson and Bell (138) suggested that the fungus illustrated and described by Frezzi (71) was *Stemphylium botryosum* Wall instead of *Macrosporium* sp.

MINOR ROOT, STEM, AND POD ROTS
SCLEROTINIA ROOT AND STEM ROTS

Sclerotinia minor Jagger and *Sclerotinia sclerotiorum* (Lib.) DeBary

Root, stem, and pod rots have been reported to be caused by species of *Sclerotinia,* but the rarity of such reports and the apparent lack of importance of these pathogens suggests that they are not economically signficant.

Frezzi (71) lists *S. minor* and *S. sclerotiorum* as causes of root and pod rot in Argentina and *S. minor* was found on peanut in Australia (3). A report from China (2) lists *S. sclerotiorum* on peanut and suggests that this fungus and *Sclerotinia miyabeana* (Hanzawa?) are essentially similar. Two species of doubtful validity are given in the literature as being pathogens of peanut. Chu (44) mentions *S. miyabeana* and *S. arachidis* (Hanzawa?) as pathogens of peanut in China. Garren and Wilson (91) commented that both species were published privately by Hanzawa in Japan and were regarded by mycologists as invalid, presumably on the basis of the manner of publication.

PHYMATOTRICHUM ROOT ROT

Phymatotrichum omnivorum (Shear) Duggar

This disease, often called Texas root rot, has been reported on peanuts from the Southwestern United States (91). The pathogen has a wide host range and persists in the soil for long periods. The information summarized by Garren and Wilson (91) seems to include most of the pertinent literature.

MINOR LEAFSPOTS

Several foliage diseases have been reported in the literature to be cause by fungi that have not been found often in peanut growing areas of the world. In most instances reports do not indicate that the fungi are proven pathogens of peanut and the extent or importance of disease is not noted.

A leaf disease of peanut caused by *Pestalotiopsis arachidis* Satya was reported from Bhopal, India by Satya (228). Infected leaves had dark brown circular spots surrounded by yellow halos. Lesions on the abaxial leaflet surface were marked by concentric rings, and black spherical acervuli were prominent. Satya (228) gave no indication that the fungus had been used in inoculation experiments to prove its pathogenicity.

Frezzi has reported (73) leaf scorch (quemadura) of wild *Arachis* species caused by *Macrophoma* sp. Lesions are very dark and composed of firm nectotic tissue. His illustration of symptoms show marginal necrosis along the apical portions of leaflets. Pycnidia are reported to be very similar to those of *Phomopsis* sp. (cf. *Diaporthe phaseolorum* var. *sojae)*, but the size of conidia are different, being 6.5 to 9 x 17 to 27u elliptical, elongate, continuous, hyaline, and guttulate. An illustration of the fungus is included in Frezzi's report.

ASCOCHYTA LEAFSPOT

Ascochyta arachidis Woronichin was reported (279) in 1924 from dying leaves of *A. hypogaea.* In 1969 Frezzi (74) reported this fungus as the cause of a leafspotting disease of peanut in Argentina. Frezzi also described the sexual stage of this fungus as *Mycosphaerella argentinensis* Frezzi.

MINOR SEED AND SEEDING DISEASES

Penicillium, spp.

Species of *Penicillium* are very prominent in the endocarpic and geocarpospheric mycoflora of peanut (137, 145, 216). Studies on the pathogenicity of *Penicillium* spp.

to peanut are few and limited in the number of species tested. Morwood (192) in 1953 associated *Penicillium* spp. with root rot, and Gibson and Clinton (98) classed *Penicillium* spp., and particularly *P. funiculosum Thom* and *P. caryophyllum* Dierckx., as fungi which caused soft rot of the seed. *Penicillium* spp. caused a small amount of 'concealed damage' to peanut seed but in this regard were much less important than species of *Diplodia* and *Sclerotium* (88, 197).

Ward and Diener (265) inoculated peanut seed with *P. citrinum* Thom and *P. meleagrinum* Biourge, respectively, and found that these fungi caused a slight loss in organic matter, degredation of sucrose, decrease in total oil, and increases in free and unsaturated fatty acids.

We conclude from both scope and results of the few investigations reported that *Penicillium* spp. are relatively unimportant as peanut pathogens, and pathogenesis is manifested mainly in the seedbed. These fungi can be controlled adequately by seed treatment with broad spectrum and vapor-action fungicides.

BLUE DAMAGE

A characteristic discoloration of the testa and cotyledons of Kernels of peanut, especially Spanish varieties, was reported by Garren *et al.* (89) and Garren and Wilson (91) to be caused by the action of oxalic acid. *Sclerotium rolfsii* Sacc. was found to produce sufficient oxalic acid while growing in shells and over the surface of kernels to cause blue damage.

Norton (197) subsequently reported that cultural filtrates of certain fungi and selected chemicals could cause intense blueing of the testa. He found that filtrates of *Aspergillus niger, A. flavus,* and *S. rolfsii* caused blue damage. Oxalic acid; sodium, potassium, and ammonium chlorides; and kojic acid caused a blue discoloration when mixed with kernels and sand and incubated in a pH range of 2.2 to 7. Norton (197) considered that blue damage was partly related to acid soil and the presence of certain ions. In his tests Runner types were not as severely injured as Spanish peanuts.

Blue damage is characterized by the appearance of bluish gray or bluish black discoloration of the testa which appears in the form of streaks, light to dark patches, or circular patterns.

CONCEALED DAMAGE

The term 'concealed damage' was originally applied to seed damage from fungi that was not visible until the kernel was broken open. The characteristic symptom as pictured by Garren and Wilson (91) and Garren and Higgins (88) was deterioration of the inner faces of cotyledons and frequently the presence of mycelial growth between the cotyledons. 'Concealed' or 'hidden' damage as now used by the United States peanut industry refers to any kind of damage that is not visible externally, e.g., fungal growth, boron or calcium deficiency symptoms, and mechanical damage.

Concealed damage caused by fungi was once a serious problem of peanut production in the Southeastern United States but the problem is of minor importance now. The reasons for the decreased incidence of this problem are not wholly known but apparently the replacement of susceptible varieties such as Southeastern Runner with Dixie Runner and other new varieties has been partly responsible.

VIRUS DISEASES OF PEANUTS

General

History. In 1907 a "curl" disease of peanut was reported from what is now called Tanzania (283). According to Storey and Bottomley (254) a similar or identical disease was noted in South Africa about 1909. They (254) applied the name "rosette" to the disease in 1925 and regarded rosette as definitely a virosis and transmitted the disease. Rosette was reported from equatorial Africa in 1926 (33), and was reported extensively from the African region thereafter. In 1945, rosette was noted as one of the three most important diseases of peanuts in Rhodesia, South Africa (115).

Virus diseases other than rosette have been reported from other areas (105, 268). Mosaic was reported on peanuts in Argentina in 1936 (253) and in China in 1939 (281). In 1941 Costa (55) described a "ring spot" of peanut in Brazil as a virosis. Cooper in 1950 (47) regarded virus diseases as unimportant in North Carolina but recognized a severe and a minor mosaic as transmittable viroses and a ringspot as a fairly definite virosis. Cooper in 1966 (51) first reported a potentially destructive virus disease called "stunt" of peanuts in the North Carolina, Virginia peanut belt. Ringspot virosis of peanuts and a peanut disease caused by the tomato spotted wilt virus have been reported from several areas and have been studied in South Africa (148, 149). Several peanut viroses have been reported from Australia (191), South Africa (150), and India (236). At present, however, there are only four described diseases of peanuts definitely connected with viruses which attract more than passing attention—rosette, mosaic, ringspot, and stunt. Of these only rosette and stunt were of much concern to the World's peanut growers in the late 1960's.

ROSETTE

Importance. Several reports include statements which may be used in evaluating the importance of the virus disease called "rosette." This information is condensed in Table 1.

Table 1. Estimates of importance of Rosette

Year of report	Area reported on	Observations on importance of rosette	Reference
1907	Tanzania	Serious loss	(283)
1945	South Africa	One of 3 most important diseases	(115)
1926	Gambia	78% infection, yield decrease 66%	(157)
1937	French West Africa	75 to 80% loss	(203)
1937	Ivory Coast	Vine weight loss 61% Pod weight loss 81%	(210)

Description. Rosette is characterized by a "condensation" of the plant. Petioles and internodes are shortened, giving the plant a typical rosette or clumped appearance. Storey and Bottomley in 1928 (255) gave a detailed description of peanut rosette as it was then recognized, and the following description is condensed from their report:

The whole plant is severely stunted. Some leaves, especially younger ones, are more or less chlorotic and faintly mottled. The first leaves formed after initial infection are pale yellow with dark green veins. Successive leaves are smaller, curled and distorted, uniformly yellow, and without green veins. Usually most of the leaves turn green and eventually appear almost normal. Yield depends upon time of infection.

If infection is early, small, sessil flowers which do not open may be found, but they do not mature into fruits. If plants are infected after seeds begin forming low yields may be obtained. The virus can be transmitted by aphids or mechanically. It is not seed-borne. It seems not to be transmitted by nematodes.

An earlier observation indicated a general deterioration of infected plants before symptoms became evident. This was offered as an explanation for reduced numbers of nuts and many small pods on plant with minor expressions of symptoms (33). Nuts from rosetted plants also had a lower shelling percentage (34).

According to Hull and Adams in 1968 (119) the many different types or strains of rosette which have been described can be grouped into two main types: A. *Chlorotic Rosette* which predominates in East and Southern Africa. B. *Green Rosette* which predominates in West Africa. Klesser (150) described these as follows: "*Chlorotic Rosette:* The plant is extremely stunted and rosetted, and the leaves are uniformly chlorotic." "*Green Rosette:* The plant is similarly stunted and rosetted, but apart from occasional isolated chlorotic flecks, the leaves are a normal green."

Studies carried on cooperatively but independently by Hull in Cambridge, England, and by Adams in Malawi in Africa (119) may help to clarify some of the confusion about the many types of rosette and some of the anomalies of its spread by the aphid (*Aphis craccivora* Koch). According to Hull and Adams (119) the aphid spreads the virus in a "pesistent" and "circulative" manner. They demonstrated that peanut rosette is caused by a complex of two viruses, only one of which can be transmitted by aphids. They found many peanut plants did not develop rosette after exposure to aphids which had been feeding on rosetted plants. Often the plants which did not develop rosette after exposure to the aphids contained a virus that restored aphid transmissibility when introduced into plants containing the virus which cannot be spread by aphids.

To quote Hull's and Adams' conclusions "Groundnut rosette is a complex of two viruses. One virus, which is manually but not aphid-transmissible, caused the symptoms normally found in groundnut rosette-infected plants. We suggest that this virus be called groundnut rosette virus (GRV). The other virus in the complex, the aphid-transmitted virus, is symptomless in groundnut and we suggest that this virus be called groundnut rosette assistor virus (GRAV)."

Pathogenicity. Several factors have been reported to influence the pathogenicity of the rosette virus. These factors, may be effective on the peanut plant or on the aphid vector. For instance, conditions promoting aphid infestation early in the peanut growing season also usually promote development of much rosette. Booker (22) noted that early or June planting of peanuts in Nigeria reduced the development of rosette as compared with peanuts planted in July. He related this to fewer of the aphids on the peanuts planted in June.

Berchoux (18) showed that, in the upper Volta, the earlier rosette symptoms appeared in a plant the lower the yield of that plant. For example, plants that showed symptoms before 40 days after planting had an average yield of less than a gram; plants that showed symptoms between 56 and 70 days after planting had average yield of 5.3 grams; plants showing symptoms after 101 days had average yield of 18.3 grams; Gibson, *et al.* (94) reported much the same response for peanuts in Malawi.

Possibly many of the apparent seasonal effects are actually effects of variations in soil moisture. Rosette has been reported more prevalent (34) and spreading more rapidly (33) in dry seasons. Some earlier observations indicated that denser vegetative coverings in peanut fields made for less severe rosette. This, of course, could have

been an indirect effect of variation in soil moisture. However, several later studies, of which Booker's (22) is typical, discounted this and pointed out that closer spacing reduced the percentages of rosetted plants but had no effect on the total damage done by the disease.

Control. In a summarizing report of 1966 Gibbons, *et al.* (95) reviewed the latest information on control of rosette: "Although some control can be obtained by cultural methods the ideal solution would be to produce peanuts resistant to the disease. This is important in view of the reluctance of African peanut farmers to adopt new methods, and because peanuts are often produced on very small farms which are difficult to merchandise because of tribal and economic reasons." According to Gibbons, *et al.* (95) the only true resistance (not immunity but a high degree of resistance) to rosette in Africa is in lines which originally came from West Africa but which have been widely tested in Africa. In the late 1960's there seemed to be several extensive breeding programs in Africa aimed at improving peanut varieties having resistance to rosette.

PEANUT STUNT

A stunting disease of peanuts obviously caused by a virus or a combination of viruses was first noted in North Carolina and Virginia in 1964 (51, 183). It reached epidemic proportions in 1965, and again in 1966 (51, 107, 183) but by 1969 it had decreased in severity until only an occasional affected plant could be found in the great majority of peanut fields of the two states.

During the period in which peanut stunt was epidemic proof was obtained that it was caused by a virus or viruses. Numerous attempts to relate it to peanut rosette failed but a close relationship to a virus disease of beans *(Phaseolus* spp.) was established.

SYMPTOMS

Peanut stunt is characterized by severe dwarfing and malformation of foliar parts and pronounced suppression of fruit development (183). On affected plants mature, normal leaves range from a few to over one half of the leaves per stem. Apical to the normal leaves the leaves are stiff, somewhat erect with light green leaflets which are less than half normal size and apically pointed (51). On moderately to severely stunted plants those fruits which develop are small, misshapen, and frequently the pericarps are split (183).

In 1966 Virginia fields which had 25% of plants evidently affected with stunt had an average yield of 2400 lb. per acre. Fields with 50% stunt had a yield of 1600 lb. per acre and those with almost all plants with some degree of evident stunt had a yield of 700 lb. per acre. The market grade components of "fancy pods," extra large kernels, and sound mature kernels were correspondingly reduced by the stunt infection (57).

When peanut plants are inoculated before they are 50-60 days old few seed are produced. Inoculation at 80-100 days after planting decreases the number of seed produced, but does not reduce size or weight of seed (153).

THE VIRUS

Peanut stunt virus disease can be transmitted by mechanical means, by grafting, by dodder *(Cuscuta* spp.), by the green peach aphid [*Myzus persicae* (Sulz.)] (183)

and by other aphids *(Aphis* spp.) (107).

The peanut stunt virus can be separated from the peanut mottle virus by passage through tobacco *(Nicotiana* spp.). The peanut mottle virus was found in 78% of field plants having the peanut stunt virus, but in artificial inoculation studies the stunt produced when both viruses were inoculated into plants was no more severe than that produced by the peanut stunt virus alone (153).

OTHER HOSTS OF PEANUT STUNT VIRUS

The peanut stunt virus has been tested on several genera of cultivated legumes and it seems to have a wide host range in the legumes. Up to 1967, it had not been found damaging snap beans *(Phaseolus vulgaris* L.) in the field, but greenhouse studies with 5 varieties of beans showed it to have potential to cause severe damage to snap beans (282). In 1967 a disease of snap beans in the commercial bean producing area of North Carolina was attributed to the peanut stunt virus (107) but damage apparently was slight.

However, beginning in 1967 studies (185, 186) have shown that there is a distinct strain of the peanut stunt virus (called the "Western strain") in the commercial bean producing area of Washington and perhaps other Western states. The economic importance of this "Western strain" on beans apparently has not been determined. Physical and chemical properties of the peanut stunt virus, Western strain, suggest it is a strain of cucumber mosaic virus. The serological data, however, connect it with the Southeastern peanut stunt virus. Futher study suggested peanut stunt virus, Western strain, is related to cucumber mosaic virus through tomato aspermy virus (184). Such relationship may hold for peanut stunt virus—Southern strain.

White clover *(Trifolium repens* L.) is the over-wintering reservoir of the peanut stunt virus. In 1967 the virus was found in 29 of 58 samples of white clover from the peanut region of North Carolina. The virus was not found in 61 samples of 21 other species of plants in the vicinity of infected peanuts (107).

In a study on the genus *Trifolium* no infection was obtained in 4 species after inoculation with the peanut stunt virus. There was successful infection in 13 species of *Trifolium* with the typical symptoms of reduced leaves and stems. In addition white clover *(T. repens)* also had reduced flowers (40).

Hebert (107) found that three species of aphids transmitted peanut stunt virus readily, but one species did not. The cowpea aphid, *Aphis craccivora* (Koch), seemed the main transmitter.

Kuhn (153) concluded that the virus is transmitted through seed in less than 0.1% of seeds. Troutman, *et al.* (261) concluded that peanut stunt virus is transmitted through seeds. Yield of seeds large enough to be used for planting is very low in plants affected with stunt and seeds that transmit stunt virus tend to be low in vitality and give rise to seedlings that emerge late and are low in vigor. Seed transmission of peanut stunt virus, therefore, seems unlikely to be a major economic factor in spread of stunt virus. Culp and Troutman (58) later concluded that seed transmission through seeds from symptomless plants is very low, in fact only 0.0038%. Circumstantial evidence indicated the two seeds involved came from plants erroniously classified as disease free.

CONTROL

If peanut stunt becomes a serious disease control measure will have to involve control of the cowpea aphid and other aphids in peanut fields as well as eradication of white clover near peanut fields.

Culp and Troutman (59) studied several hundred peanut varieties, introductions, and breeding lines and rated them for natural infection. None were immune. Several showed less severe damage than others. One breeding line showed least damage in several tests.

SLIME DISEASE — BACTERIAL WILT

Pseudomonas solanacearum (E. F. Sm.) E. F. Smith

Importance. "Slime disease" is a general name for a wilt-tpye disease of a number of cultivated plants. Slime disease is caused by a bacterium *Pseudomonas solanacearum* (146). Slime disease of peanuts, the first recorded important disease of peanuts, was observed in the East Indies around 1905 with losses of at least 25% (32). The disease was investigated extensively in the East Indies thereafter until 1937 when a gradual decrease in the importance of the disease was noted (91). Slime disease of peanuts was reported, without estimates of importance, from various regions but in South Africa the disease became of sufficient importance for an extensive study to be made in 1930 (164).

In the United States bacterial wilt of peanuts is of minor importance. The disease was noted in North Carolina in 1912 when about 15% of Spanish peanuts on soil known to be infested were diseased (75). Wartime plant disease surveys reviewed by Garren and Wilson (91) reported some bacterial wilt of peanuts in the United States. Experimental host range studies made in North Carolina in 1917 substantiated the general conclusion that the disease is relatively unimportant on peanuts in the United States since peanuts were placed in the "very slightly susceptible" class (91).

Description. As slime disease of peanuts was observed in the East Indies, attacked plants usually wilted rather suddenly with leaves on dead plants sometimes remaining green (32, 202). Slight, early infections, however, were usually overcome (202). Apparently the disease developed primarily in patches and general attacks over an entire field were very rare (32). In contrast to this are the descriptions given from the United States where the disease appears to be much milder (91). The attack of the causal organism is centered in the conducting cells of the roots and stems (32). One diagnostic characteristic is a large number of dead roots (202). Bacterial colonies form throughout the root, main stem and lower branches (32). These colonies are evident as streaks of brown or black discolorations (32). The original point of entrance is possibly an insect wound or a lenticle (32). The infected tissue is finally blackened with extensive plugging and necrosis. If young plants are attacked the pods are invaded and remain small (202) or become wrinkled and develop a spongy decay (32). Shells of well-developed fruits have been found to contain the bacteria (202). When relatively mature plants are attacked there is no evidence of an invasion of the fruit (32).

When not otherwise evident the infection may be detected in cross sections of stems and roots. Dark-brown spots are usually evident in the cut xylem and pith regions (32) though healthy appearing plants may be filled with bacteria without any discoloration of the vessels (202).

Organism and pathogenicity. Since 1911 *Pseudomonas solanacearum* has seemed definitely established as the pathogen of the bacterial wilt of peanuts. Inoculation tests have established that a typical slime disease is produced when peanuts are inoculated with *P. solanacearum* isolated from peanuts or other plants (91, 164). In 1953 Kelman (154) published a general and thorough report on the organism and the widespread disease and damage it causes.

With a number of plants reportedly susceptible the existence of different strains of the bacterium seems likely. Early observations in the East Indies suggested the existence of a strain equally pathogenic to peanuts, tobacco and tomatoes (32, 234) and another strain more pathogenic to eggplant, potatoes and local species (234).

In the United States, *P. solanacearum* has been investigated most frequently in connection with the "Granville wilt" of tobacco, and bacterial wilt of peanuts was first noted on peanuts grown in rotation with tobacco (75). The bacterium from tobacco was successfully cross-inoculated into peanuts. Further studies showed that numerous species of cultivated plants and weeds are susceptible to the bacterium (244). In South Africa cross-inoculation tests indicated that tomatoes and only one variety of tobacco were partially susceptible to the bacterium attacking peanuts (164). These results suggest the existence of different strains of the bacterium in the three widely separated peanut-producing areas, and this may explain the apparent unimportance of the slime disease of peanuts in the United States. More recently in the United States the existence of strains differing in pathogenicity to tobacco and peanuts has been demonstrated (147).

Factors most frequently suggested as affecting the pathogenicity of *P. solanacearum* on peanuts are soil type, soil moisture, and rotation practices. The virulence of the organism on peanuts in the East Indies was found to be higher on more moist soils, on heavy clay soils, and on soils planted to peanuts for several successive years (202). Continuous cultivation on irrigated soils resulted in an apparent increase in infections in dry seasons (233). In South Africa repeated cropping to peanuts increased the severity of the disease which was apparently restricted to the heavier loamy soils (164). This emphasis on soil texture and drainage suggests that sandy soil may be an important factor in making bacterial wilt relatively unimportant in the United States.

Control. Planting of a resistant variety is the most convenient means of controlling the slime disease of peanuts. From selection work in the East Indies has come the variety 'Schwarz 21' which has resistance to the disease and which has resulted in a considerable decrease in loss from bacterial wilt in that area (202).

A few attempts have been made to control *P. solanacearum* by soil treatment. Those treatments which might be applied to peanuts offer little hope (63, 209). Application of sulfur to East Indies soil gave no beneficial results on peanuts (63).

The control measures recommended in addition to the use of resistant varieties of peanuts are:

A. Seed Treatment; the bacterium can be seed-borne (202).

B. Planting on light, well-drained soil (164, 202).

C. Rotation with crops which seem to be resistant to *P. solanacearum* such as sweet potatoes, grains and certain legumes.

D. Variation of the rotation to prevent building up other disease-producing organisms in the soil to the extent that the effects on peanuts will be more detrimental than that of *P. solanacearum.*

NEMATODE DAMAGE TO PEANUTS

The damaging effect of nematodes on developing peanut plants has been recognized increasingly during the past two decades, but information in the scientific literature is limited mostly to research accomplished in the Southern United States. Reports from other peanut-growing sections of the world are scanty and very generalized. Species which are known to attack peanut over certain rather broad geographic areas, e.g. the Southern United States, may be present in other parts of the world, but the assumption that damage to peanut will result from the species is questionable. Damage estimates are now based on limited information and the presence of large numbers of parasitic nematodes does not necessarily result in economic damage to peanuts (259). Species of nine genera of plant parasitic nematodes have been reported (259) causing injury to peanuts. Species of genera not included here may have peanut in their host range but this may be a relationship of negligible economic importance.

ROOT-KNOT NEMATODE

Meloidogyne arenaria (Neal, 1889) Chitwood, 1949

and *Meloidogyne hapla* Chitwood, 1949

The peanut root-knot nematode *(M. arenaria)* and the Northern root-knot nematode *(M. hapla)* are widely distributed in the Southeastern United States and *M. arenaria* is found on peanut westward to Texas (170, 206, 259, 260). Species of the genus are reported to attack peanut in Africa (65). The economic importance of the disease is generally slight when one considers the entire peanut-growing acreage of the United States. However, severe economic losses can and have resulted from the disease in many instances.

A great variety of other plants are attacked by these two species of nematodes, including both economic and native plants. Peanut is resistant to attack by other root-knot nematode species.

SYMPTOMS

Above-ground symptoms of attack by the peanut root-knot nematode are often confined to a slight yellowing of the foliage and almost imperceptible stunting of the entire plant. In instances of more severe infection, plants become noteably stunted and yellow and more susceptible to death during very dry weather. Roots and pegs are commonly seen to be enlarged at many points along their length into variously sized galls. The galls are usually symmetrical with respect to the longitudinal axis of the root thereby distinguishing them from nodules which are mostly laterally appended to the root. Galls may reach a diameter of several times the normal adjacent root diameter. Pods are infected also and develop knobs, protuberances or small warts. Galls on roots, pegs and pods sometimes begin to deteriorate by maturity. Necrotic tissue in such cases may be colonized by a variety of fungi including *Aspergillus flavus* Link. *ex.* Fries, but no evidence has been obtained to suggest that aflatoxin content is subsequently greater in these peanuts (188, 190). The extent of the root system is commonly much reduced.

The Northern root-rot nematode causes similar above-ground symptoms. Roots, pegs, and pods are galled also but the individual galls are smaller than those caused by *M. arenaria.* In addition infected roots tend to form branches near the point of in-

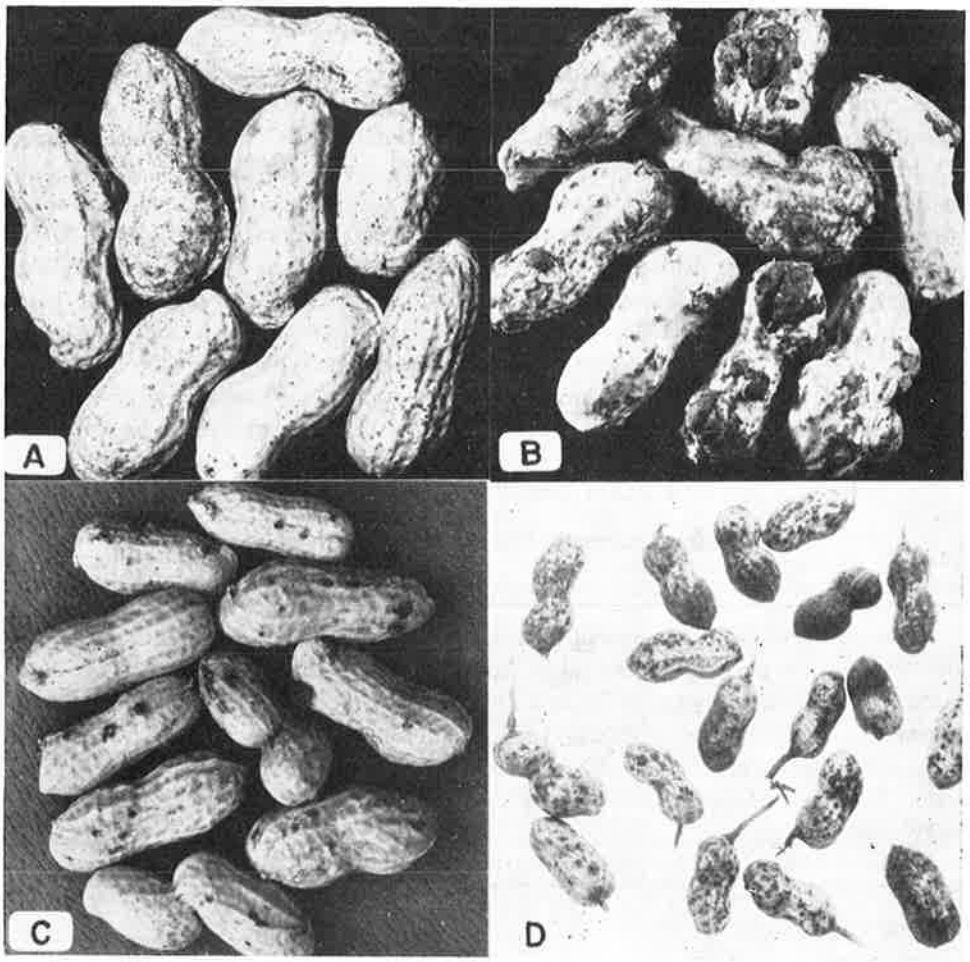

Figure 6. Damage to peanut pods from severe intestation of: A. Sting nematodes; B. Peanut root-knot nematodes; C. Northern root-knot nematodes; D. Root-lesion nematodes. Photographs courtesy L. I. Miller, Va. Agric. Expt. Sta.

vasion. This results in a dense, reticulate (bushy) type of root system. Reduction of the normal yield of pods is a usual consequence of moderate to severe infection by both species (Fig. 1 B and C). Total crop loss may occur when infection is severe.

INFECTION AND SPREAD

Root-knot larvae exist in the soil and move freely to any adjacent plant root. With the aid of penetrating mouth-parts they invade peanut roots, pegs, or pods. Within the root the larvae lose their mobility and begin to feed on the tissues of the root. As a result of the feeding and physiological activities of the nematode, cells of the root, peg, or pod increase abnormally both in size and number. When the female nematode reaches maturity, large numbers of eggs eventually develop within or outside the root. Ultimately, the eggs hatch and the new larvae enter the soil surrounding the root; the cycle leading to infection is thereby completed. The time required for this cycle is controlled primarily by soil temperature and moisture. Under the temperature and

moisture conditions which are suitable for peanut production two or more cycles are usually possible during the time required to produce the crop (43).

Differences in pathogenicity between morphologically similar populations of *M. arenaria* were reported by Minton (187). Two populations varied considerably in their ability to infect and reproduce in peanut. Such differences, however, have not been a basis for enduring sub-specific distinctions in these peanut pathogens (38, 43).

The spread of root-knot nematodes by their own movement through the soil is very limited. However, crop debris containing galls may be spread by farming operations or running water for considerable distance in a field. It is possible in this way to transport nematodes to new areas. Free-living larvae may be disseminated by most agencies which move moist soil.

CONTROL

Differences in resistance to root-knot nematode infection exist among peanut varieties. Edwards (65) lists 'Natal Common' and 'Kumawu Erect' as possessing a high degree of resistance. However, most widely grown varieties in the world are susceptible.

The use of chemicals applied to the soil to greatly reduce populations of root-knot larvae has been quite successful. Numerous reports (43, 48, 260) list specific effective chemicals. Other methods of control are reviewed at length by Christie (43).

ROOT-LESION NEMATODE

Pratylenchus brachyurus (Godfrey, 1929) Filip. & Stek., 1941

The root-lesion nematode (lesion nematode, smooth-headed lesion nematode, meadow nematode) causes up to about 20% in yields of peanuts when it occurs in relatively high populations. Yield is lowered as a result of the reduction in plant vigor caused by restriction of root systems and as a consequence of peg infections that tend to cause abnormal shedding of mature pods at harvest. *P. brachyurus* is found in all peanut growing areas of the Southern United States. The wide host range of this nematode includes tobacco, peach, okra, bean, and most grasses and small grains.

A second species of the genus, *P. zeae* Graham, 1951 (43) is associated with peanuts but does not cause injury. Boyle (26) quotes G. Steiner as finding *P. brachyurus* (syn. *P. leiocephalus)* and another unnamed species of *Pratylenchus* that were infecting peanut root specimens from Georgia.

SYMPTOMS

Peanut plants exhibit varying degrees of stunting and chlorosis depending on the magnitude of root infestation. In severe cases, plants become stunted to half normal size and develop marked chlorosis (100). Symptoms commonly appear in plants in somewhat circular and restricted areas of a field. 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. Pegs exhibit brown diffuse lesions. Pod symptoms vary from a few brown to black angular lesions per pod to many brown to black lesions with diffused margins which discolor most of the pod (Fig. 6 D).

INFECTION AND SPREAD

Precise information regarding the infection of peanut by *P. brachyurus* is not available. However, members of the genus are known to be vagrant parasites and

presumably both adults and larvae can infect roots, pegs, and pods (43). Pegs and pods support larger populations than roots (100). The nematodes enter peanut tissue by direct penetration and, once within the organ, they feed on the parenchyma tissue. Often many individuals are found in a confined area, as is especially the case with pods. The destruction caused by parasitic activities of these nematodes is usually magnified by the combined activity of the nematodes and the secondary soil fungi. This has been reported in the case of *Rhizoctonia* (see *Rhizoctonia* Diseases), *Aspergillus flavus,* and other fungi (140).

Pegs are greatly weakened by combined activities of root lesion nematodes and secondary fungi. When they are subjected to mechanical stresses in digging and shaking they often break and the pods fall from the vine (100). Masses of individuals of *P. brachyurus* are found in discrete pod lesions but infrequently penetrate into the kernel. These nematodes remain alive throughout natural or artificial drying and winter storage. Furthermore, after shelling the shells continue to be an important nematode reservoir and means of spread (100). The protection afforded by the shell is such that ground shells, when used as diluents in certain fertilizer preparations, may transmit the living nematode (100).

CONTROL

Root lesion nematodes are readily controlled by several nematocides. In many cases, the economic benefits from using chemical control may be questioned. Reduction of soil populations by rotational schemes is distinctly possible and beneficial. Good cultural practices tend to reduce the obvious damage caused by *P. brachyurus* when the nematode is present in small numbers.

STING NEMATODE

Belonolaimus longicaudatus Rau, 1958

Sting nematodes cause serious damage to peanuts in Virginia and North Carolina. Even though this species occurs in other peanut growing areas of the Southern United States and damages other crops it does not damage peanuts except in the two states. Cooper and Sasser (52, 53) reported yield reduction of more than one ton per acre and that very striking yield increase results from the use of small amounts of preplant nematocide. The nematode has a very wide host range which includes many hosts that presumably are more suitable for population increase, such as rye, wheat, oats, soybean, cotton, southern pea and corn (43, 154).

Symptoms of attack by sting nematodes are stunted, chlorotic plants which have stubby, sparse roots. Roots and pods may exhibit small dark necrotic spots which are caused by the feeding of this nematode (201). The sting nematode, in contrast to root-knot or root lesion nematodes is an ectoparasite and is rarely found inside roots or pods (Fig. 6 A).

Control of sting nematode has been studied in some detail (52, 53, 54, 181, 226, 227) and methods which involve preplant application of chemicals have been used successfully. Much work is now (1971) under way on crop rotations and control of sting and other nematodes. Recommendations should soon be forthcoming.

RING NEMATODE

Criconemoides ornatum (Raski, 1952) Raski, 1958

Criconemoides rusticum (Micoletzky, 1915) Taylor, 1936

Two species of ring nematodes have been reported on peanut (259). Machmer (171) reported symptoms of pronounced chlorosis in peanut in Georgia caused by an unnamed species of ring nematode. Recently *C. ornatum* was reported from peanut in Georgia (189). Graham (102) found that a ring nematode caused stunting and weight loss in peanut plants. Very little else has been published concerning the ring nematode and its relationship to peanut.

OTHER NEMATODES

A listing of nematodes found on crops in the Southern United States (259) records the presence of several other genera and species that cause injury to peanut.

We have referenced two other reports that expand only slightly the information about the following species.

Helicotylenchus sp. a spiral nematode, is given without commentary save that it is associated with injury in Texas. The reniform nematode, *Rotylenchus reniformis* Lindford and Oliveira, 1940 is given by Birchfield (19) as a parasite of peanut. *Trichodorum christiei* Allen, 1957, was reported from Alabama and *Tylenchorhynchus* sp. from Texas. *Xiphinema americanum* Cobb, 1913 has been reported to injure peanuts in Alabama and Texas. Schindler (230) lists peanut as a host of *Xiphinema diversicaudatum* (Micoletzky, 1927) Thorne, 1939.

NOTE: One of the authors of this chapter is an employee of the U. S. Department of Agriculture. It is necessary, therefore, to make the following statements.

1. "Mention of a trademark or proprietary product does not constitute a guarantee or warranty of the product by the U. S. Department of Agriculture, and does not imply its approval to the exclusion of other products that may also be suitable."

2. "All agricultural chemicals *recommended* for use in this chapter had been registered by the U. S. Department of Agriculture and none had had registration withdrawn at the time the manuscript was completed. If any chemical *recommended* in this chapter is used it should be applied *only* in accordance with the directions on the manufacturer's label as registered under the Federal Insecticide, Fungicide, and Rodenticide Act.

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