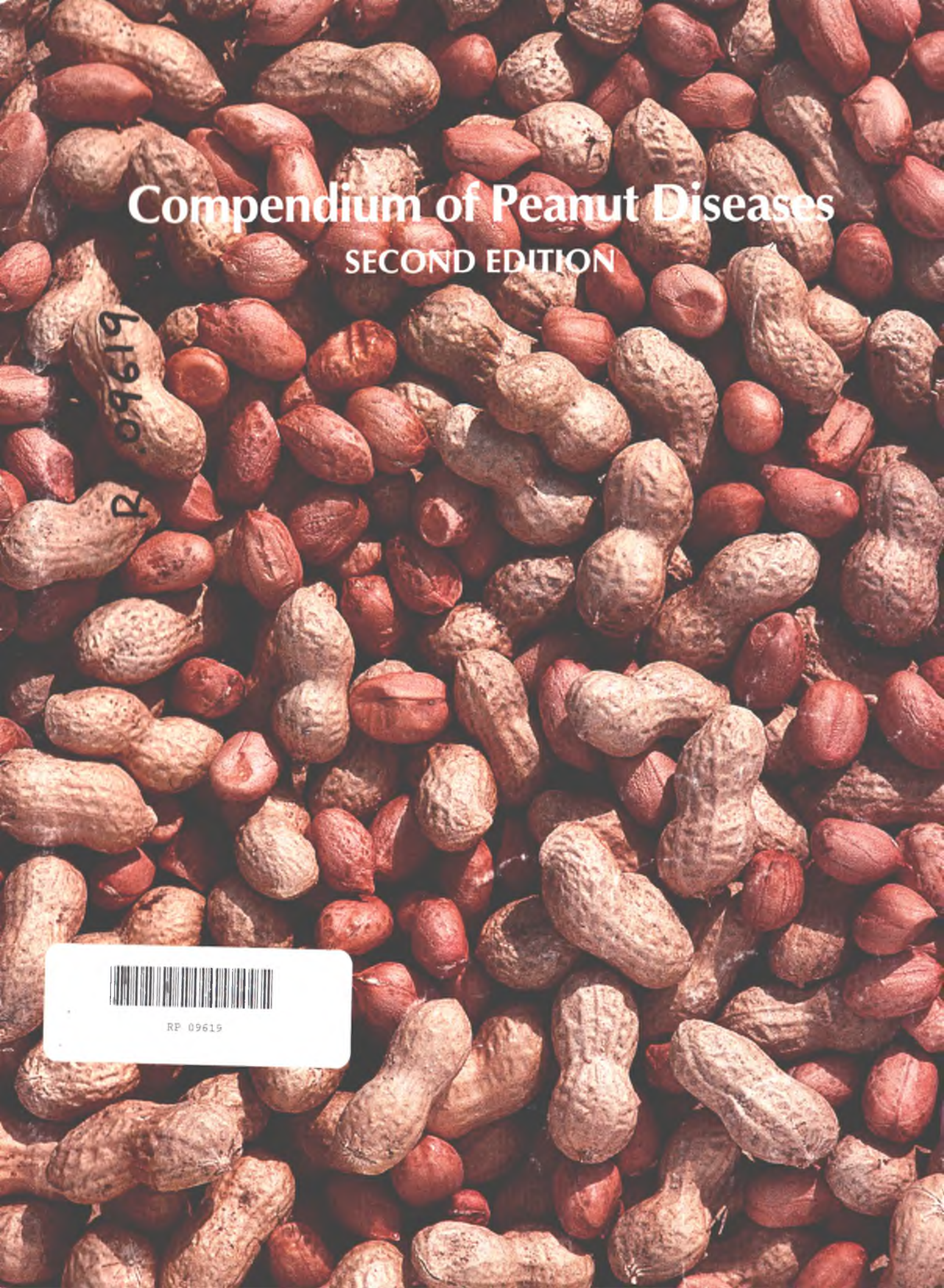

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Compendium of Peanut Diseases

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Preface

This compendium was written by and for individuals worldwide who have a direct interest in peanut production as affected by plant and seed abnormalities. The technical data on specific causal organisms, etiologies, and control strategies for known peanut disorders or abnormalities should serve as a valuable resource for those interested in the economical production of top-quality peanuts free of undesired contaminants.

To further broaden the appeal of the compendium to those involved in extension and scientific activities and to agribusiness and crop specialists, experts from throughout the world have participated in preparing this edition. In fact, authors from India, the People's Republic of China, Malawi, Australia, Israel, and South Africa have prepared sections in the areas of their expertise. Thus, the second edition of this compendium is truly international in scope and should be even more useful to people in all segments of the peanut industry.

For reference and clarity, this compendium is divided into five parts. Part I covers biotic diseases caused by fungi, bacteria, nematodes, viruses, and phytoplasmas; Part II, abiotic diseases caused by environmental stresses such as drought, herbicide injury, nutrient imbalances, and air pollution; Part III, diseases and injury caused by insects and arthropods; and Part IV, miscellaneous detrimental as well as beneficial organisms. Since future disease control appears to depend heavily upon disease resistance, Part V, Management of Peanut Diseases, contains sections on management strategies, genetic modification, and disease- and insect-resistant cultivars already available for use by growers and in breeding programs.

The authors who contributed to specific sections of this compendium are noted at the end of their sections. The time and effort put forth by each author are appreciated. These valuable contributions will enhance the acceptance of this compendium.

The editors especially thank the following individuals who reviewed the entire manuscript: J. P. Damicone, Oklahoma State University, Stillwater; T. B. Brenneman, University of Georgia, Tifton; and J. Fletcher, Oklahoma State University, Stillwater.

We also thank those who contributed photographs and illustrations. Where possible, proper credit is given.

The editors gratefully acknowledge the contributions of the secretarial staff including N. T. Whitfield and R. M. Waldo. Special thanks to F. S. Wright, R. A. Taber, and the Department of Plant Pathology at Auburn University.

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Contents

Part IV. Other Organisms

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Introduction

The peanut (*Arachis hypogaea* L.) is cultivated throughout the world; India and China are the largest producers (Table 1). The United States, the world's third largest producer, produces more peanuts per cultivated unit area than any other country. Production of peanut in the United States is limited to nine states (Table 2), of which Georgia and Texas are the largest producers. More than 675,000 ha (1.7 million acres) were harvested in the United States in 1992 with an average yield of 2,877 kg/ha (2,567 lb/acre) and a value approaching $1.3 billion.

The peanut plant is unusual because it flowers aboveground and pods containing one to five edible seed are produced below ground. Seed from the pods are eaten raw or cooked. Peanut seed are very nutritious and high in calories and contain 25% protein. They may be boiled, broiled, roasted, fried, ground into peanut butter, or crushed for oil. Peanut seed also contain fats, carbohydrates, fiber, vitamin E, niacin, folacin, calcium, phosphorus, magnesium, zinc, iron, riboflavin, thiamine, and potassium. Most of the peanut crop produced in the United States is shelled and sold as peanut butter, salted peanuts, and confections. Peanut oil is of high quality and contains unsaturated fats such as oleic and linoleic acids.

TABLE 1. Peanut Production, 1992[a]

Country	Area Planted (ha)	Production (metric tons)
India	8,750,000	7,500,000
China	2,975,000	6,200,000
Senegal	872,000	724,000
United States	816,000	2,235,000
Indonesia	645,000	920,000
Burma (Myanmar)	550,000	500,000
Zaire	530,000	380,000
Sudan	530,000	400,000
Nigeria	480,000	220,000
Cameroon	320,000	140,000
Argentina	190,000	400,000

[a] Data from Agricultural Statistics, 1992, U.S. Government Printing Office, Washington, DC.

The Peanut Plant

The cultivated peanut plant (*A. hypogaea*) (Fig. 1) is an erect or prostrate, sparsely hairy, annual legume, 15–60 cm high or higher. It has a well-developed taproot with many lateral roots. Roots are usually devoid of hairs and a distinct epidermis.

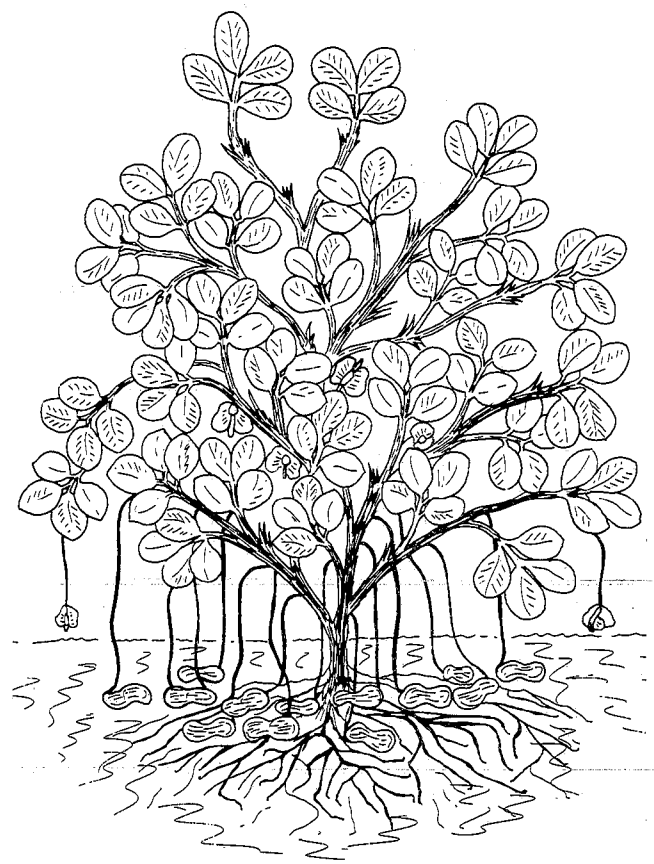

Fig. 1. The peanut plant.

TABLE 2. Peanut Production in the United States, 1992[a]

State	Area Planted		Yield		Production (metric tons)
	Acres	Hectares	lb/acre	kg/ha	
Alabama	237,000	95,912	2,505	2,781	268,157
Florida	88,000	35,628	2,530	2,836	91,858
Georgia	675,000	273,169	2,705	3,032	825,758
New Mexico	21,000	8,498	2,760	3,094	26,416
North Carolina	153,000	61,918	2,660	2,982	184,605
Oklahoma	100,000	40,470	2,410	2,702	107,131
South Carolina	14,000	5,666	2,500	2,803	14,742
Texas	308,000	124,696	2,330	2,612	308,514
Virginia	94,000	38,041	2,755	3,088	116,218

[a] Data from Agricultural Statistics, 1993, U.S. Government Printing Office, Washington, DC.

Peanut leaves are pinnate, with two opposite pairs of leaflets 2–5 cm long. The petioles are 3–7 cm long. Flowers are borne on inflorescences located in the axils of leaves but never at the same nodes as vegetative branches. Nodes have one to many flowers. Flowers have two calyx lobes, an awllike one opposite the keel and a broad, four-notched one opposite the back of the standard. The five yellow petals consist of a yellow to orange standard, two yellow to orange wings, and two petals. The flower has 10 monadelphous stamens.

Flowers appear 4–6 weeks after planting. Self-pollination occurs in the closed keel of the flower at sunrise. The flower withers 5–6 hr after opening.

Within about 1 week after fertilization, a pointed, needlelike structure (the carpophore), commonly called the "peg," develops and elongates quickly. The fertilized ovaries are located behind the tip of the peg. The peg grows into the soil to a depth of 2–7 cm. The tip orients itself horizontally, the ovary enlarges rapidly, and pod growth begins.

The mature pod (fruit) is an oblong, indehiscent legume (1–8 × 0.5–2 cm) containing one to five seed. The dry shell (pericarp) of the mature pod is reticulate and has one to 15 longitudinal ridges. Of the pod weight, 20–30% is shell. Mature seed (incorrectly termed kernels) are cylindrical or ovoid and measure 1–3.5 × 0.5–1.5 cm. The seed coat varies among cultivars in color; it can be white, pink, red, purple, shades of brown, or variegated. Seed have two large cotyledons, an epicotyl with three meristems, a hypocotyl, and a primary root. Seed weight varies from about 0.2 to 2 g.

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(Prepared by D. M. Porter)

Origin of the Peanut

The peanut (*Arachis* spp.), indigenous to South America, is a self-pollinating, indeterminate, herbaceous legume. Sixty-nine species have been identified (see Taxonomy of the Genus *Arachis*). Of these, only six have been cultivated to any extent: *A. hypogaea* L., *A. villosulicarpa* Hoehne, and *A. stenosperma* Krapov. & W. C. Gregory for their edible seed and *A. repens* Handro, *A. pintoi* Krapov. & W. C. Gregory, and *A. glabrata* Benth. for forage and ground cover. *A. villosulicarpa* is cultivated only by the native people of western Brazil. The most widespread cultigen of the genus *A. hypogaea* is found in the tropical and subtropical areas on every continent except Antarctica.

The genus *Arachis* originated in the central part of Brazil, probably predating the present Amazonian forest by several millennia. The pod, fleshy root systems, many plant forms and leaf structures, and an immense potential to recover from environmental stress have all evolved as a result of the wide range of climatic changes imposed on *Arachis* spp. since the early origins of the genus. The ability to survive in harsh environments is in large part the result of the specialized root systems and the pods. Agents capable of physically moving soil containing pods, such as water, are the most likely means of effectively distributing species within regions.

A. hypogaea was likely first domesticated in the valley of the western Paraguay River in the Chaco region of South America. Archeological evidence suggests that peanuts similar to some of today's germ plasm were cultivated by the residents of South America 3,000–3,700 years ago. In spite of its long history of cultivation, *A. hypogaea* has not been found in the wild. *A. hypogaea* is an allotetraploid thought to have been developed from chromosome doubling of a cross between two diploid wild species. Although several proposals have been made as to which species are the progenitors of *A. hypogaea*, conclusive evidence has not yet been presented.

Equally important to peanut improvement is the clarification of the origin of the subspecies within *A. hypogaea*. Whether the subspecies are the results of genetic divergence over a long period or of different crosses among species has not been determined.

Peanuts were being grown extensively, some under irrigation, when the first European explorers came to South America. These explorers were responsible for introducing peanuts into Africa, Asia, Europe, and the Pacific islands. Peanuts were introduced into the United States from Africa, the Caribbean islands, and Spain by traders. Beginning in about 1800, peanuts were planted near seacoast towns including Wilmington, North Carolina; Charleston, South Carolina; Norfolk, Virginia; and Savannah, Georgia. The first commercial production of peanuts in the United States is thought to have been near Waverly, Virginia, in 1844.

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(Prepared by T. A. Coffelt and C. E. Simpson)

Taxonomy of the Genus *Arachis*

Carolus Linnaeus described the cultivated peanut *Arachis hypogaea* L. in 1753. The first taxonomic treatment of the genus, including five species, was published in 1841. During the next 100 years, approximately 10 additional species descriptions appeared. Much confusion was introduced into the *Arachis* taxonomy during this time, and a vast increase in new species and accessions occurred with the 1959–1961 collections. Delays in describing these materials resulted in the use of several undescribed species names (*nomen nudum*) in the literature. From 1976 to 1993, the International Board for Plant Genetic Resources funded collection efforts in the primary center of origin of *Arachis*. New species were collected; and by 1994, the number of species approached 70, only 23 of which were officially described.

A summary of the taxonomy of the primary cultivated species is shown in Table 3. *A. hypogaea* was divided into two subspecies and six botanical varieties. Subspecies *hypogaea* includes variety *hypogaea*, which contains the virginia and runner market types in the United States peanut trade. Variety *hirsuta* also belongs to this larger-seeded, slower-maturing, prostrate, plant-type group. *A. hypogaea hypogaea hirsuta* has small hairs on both surfaces of the leaflets, stipules, petioles, and stems. Subspecies *fastigiata* contains four varieties: 1) *fastigiata*, the valencia market type (United States trade); 2)

vulgaris, the United States spanish type; 3) *peruviana*, the deeply reticulated pod type from northern Peru; and 4) *aequatoriana*, which has a deeply reticulated pod with very hairy plant parts.

Sixty-eight wild *Arachis* species and their assignment to nine taxonomic sections have been described. The most significant characters distinguishing *Arachis* species are their underground structures including pods (fruits), rhizomatous stems, root systems, and hypocotyls. These variations, coupled with autogamous reproductive systems, agametic reproduction, and the limited means of seed dispersal, can be attributed to the genetic variability that gives rise to noticeable infertility in crosses between species within sections and near total infertility in crosses between species from different sections.

A few major differences between the new taxonomy and what is commonly found in the pre-1994 literature include the following:

1. Two new varieties of cultivated peanut, both in the subspecies *fastigiata*, have been described: var. *peruviana* and var. *aequatoriana* (Table 3).
2. *A. chacoensis* (or *A. chacoense*) Krap. et Greg., *nom. nud.*, does not appear in the new revision. This accession (GKP-10602), included under the name *A. diogoi*, was collected downstream (Paraguay River) from accessions of *A. diogoi*. Morphologically, it is difficult to distinguish between accessions.
3. *A. duranensis* includes all old accessions that were called *A. spegazzinii* Krap. et Greg., *nom. nud.*
4. *A. pusilla* Benth. is assigned to the section *Heteranthae*, not *Triseminalae*. Assignments were based on very young seedlings, not mature plant parts.

The evolutionary and phylogenetic relationships between the nine different taxonomic sections make it evident that the genetic distances separating the sections are far from being of the same magnitude. The presumably older sections (*Triseminalae, Trierectoides, Erectoides, Extranervosae,* and *Heteranthae*), except for *Erectoides*, are much more isolated from the remaining sections and each other than those believed to be of more recent origin (*Procumbensae, Caulorhizae, Rhizomatosae*, and *Arachis*).

Section *Arachis* is by far the largest, containing about 39% of the species described. Species of this section appear to be invading geographical areas occupied by species of other sections. They grow intermixed with populations of *Extranervosae* in the upper Paraguay River basin, occupy common ground with section *Procumbensae* in the Gran Pantanal and central Bolivia, and grow with *Heteranthae, Extranervosae*, and *Rhizomatosae* in eastern and northeastern Brazil. They have reached the shores of La Plata and the southeastern coast of Brazil and grow from Salta in northwestern Argentina to the Tocantines in northeastern Brazil.

The 27 species of section *Arachis* will provide most of the genes for the improvements in cultivated peanut in the near future because those from the primary and secondary gene pools are accessible without DNA technology. Utilization in peanut-breeding programs of genes in the tertiary gene pool (i.e., outside section *Arachis*) will probably require DNA technology.

TABLE 3. Taxonomic Assignment of the Cultivated Peanut Species *Arachis hypogaea* L.[a]

Subspecies	Variety	U.S. Market Type	Primary Area of Origin
hypogaea	*hypogaea*	Virginia Runner	Southern Bolivia and northern Argentina
	hirsuta	...	Peru
fastigiata	*fastigiata*	Valencia	Peru, Brazil, and Paraguay
	peruviana	...	Peru
	aequatoriana	...	Ecuador
	vulgaris	Spanish	Paraguay, Uruguay, and Brazil

[a] Source: Krapovickas and Gregory, 1994.

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(Prepared by C. E. Simpson and T. A. Coffelt)

Peanut Diseases

Most widespread peanut diseases of today were noted and described during the early days of commercial peanut production. As the peanut became more economically important in world agriculture, emphasis in production shifted from minimal input to intense input, which resulted in high yields and improved seed quality. As the peanut went from a low-value to a high-value crop and production intensity increased, both old and new diseases prevailed. The development and severity of peanut diseases depend on complex interactions among the host, the pathogen, and the environment (Fig. 2).

Pesticides, cultural practices, and resistant cultivars have been developed to aid in the control of specific pathogens. However, disease is the main factor limiting peanut production in many parts of the world. In the United States, economic losses caused by reduced yields and the preventive measures taken to control disease cost many millions of dollars annually.

Biotic Agents

Any abnormality of the peanut plant (foliage, roots, pods, and seed) is considered to be a disease. Diseases are caused by

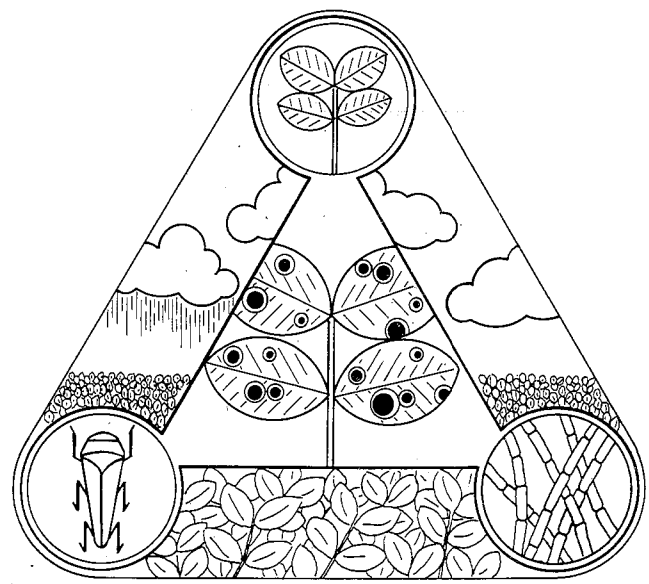

Fig. 2. Interacting factors commonly associated with disease development in peanut plants.

infectious (biotic) agents or noninfectious (abiotic) agents. Biotic agents that cause diseases of major importance include fungi, bacteria, nematodes, viruses and viroids, and phytoplasmas (mycoplasmalike organisms [MLOs]) (Fig. 3).

Fungi. Fungi, the most numerous of all peanut pathogens, are simple, filamentous organisms lacking chlorophyll (Fig. 4). They depend on the oxidation of organic matter for food rather than on energy obtained from sunlight. While most live as saprophytes, some fungi are parasites, obtaining their food from other living plants, such as the peanut. Approximately 50 genera of fungi are causal agents of peanut diseases. Some fungi penetrate the host directly; others penetrate through natural openings or wounds. Once inside the host tissue, fungi grow intercellularly and intracellularly.

Many genera of fungi produce asexual spores in large numbers on infected plant parts (Fig. 4A and B). Such spores are usually short lived and provide inoculum for secondary infections. Sexual spores (produced, usually in lower numbers, by sexual fusion) are thick walled and serve as primary overwintering propagules. These sexual spores often provide the primary inoculum for the initial infection of the host. Spores of both types are spread by air, water, animals, and machinery. Fungi also survive by forming resting bodies: chlamydospores, sclerotia, or oospores (Fig. 4C). These survival propagules can persist in the soil for many years. The filamentous threads characteristic of fungi are called hyphae or mycelia (Fig. 4D).

Bacteria. Bacteria are one-celled, prokaryotic organisms (Fig. 5). They are characterized by a single chromosome, lack mitochondria or endoplasmic reticulum, and have no organized nucleus. They obtain food by saprophytic means from decaying plant and animal material or by parasitic means from living plants. Some bacteria are cocci (spheres), some are rods, and others are comma shaped to spiral. Some have flagella for motility. Most plant-parasitic prokaryotes have rigid, distinct

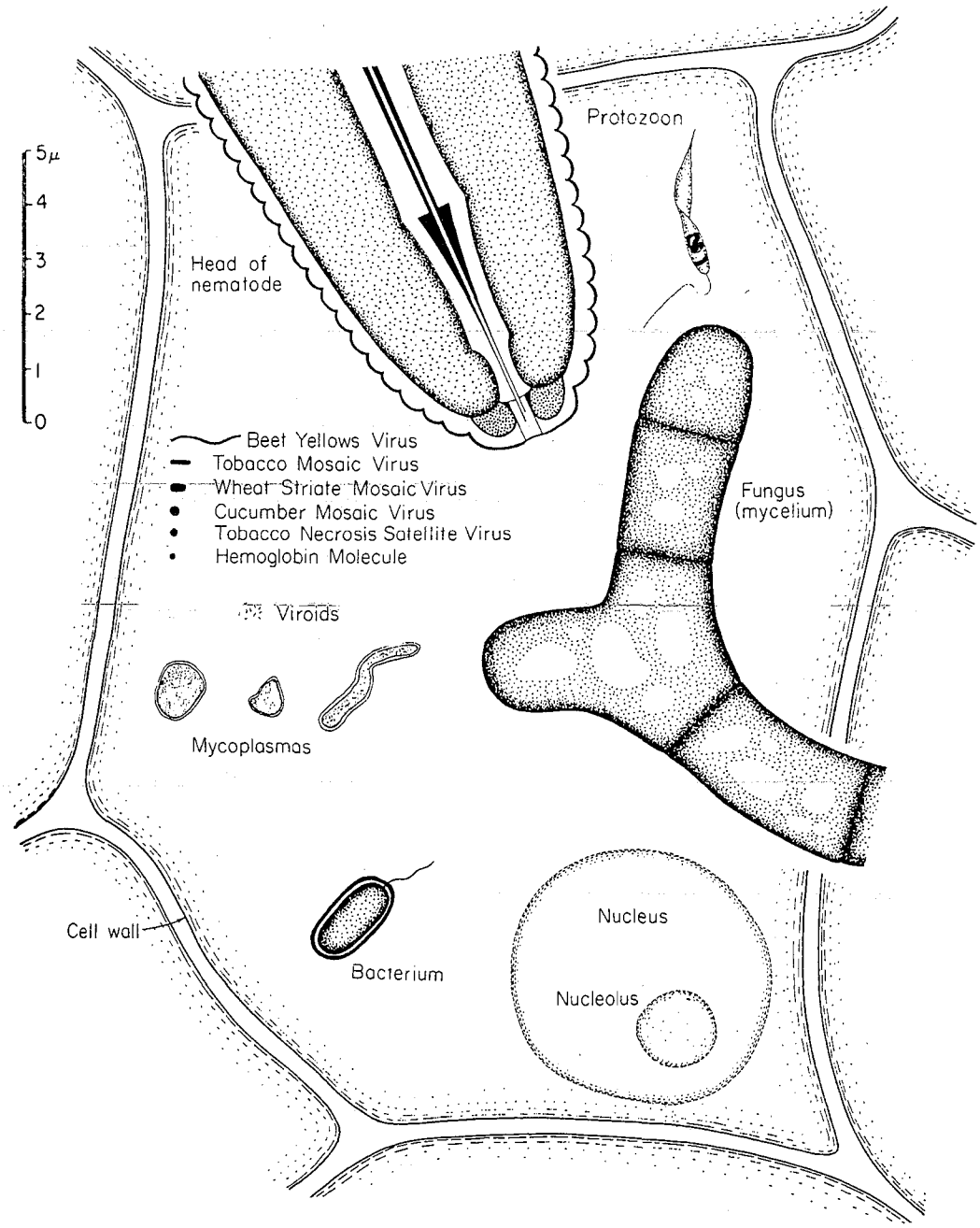

Fig. 3. Shapes and sizes of certain plant pathogens in relation to the size of the plant cell. (Reprinted, by permission, from G. N. Agrios, 1988, Plant Pathology, 3rd ed., Academic Press, San Diego, CA)

cell walls. However, phytoplasmas (Fig. 6), a group of extremely minute prokaryotes that cause plant diseases, do not have distinct cell walls and are pleomorphic.

Bacteria are spread by water, insects, machinery, and humans. Some cause local infections, and some travel throughout the plant via the vascular system, causing systemic infections. Through the activity of enzymes and/or toxins, bacteria kill plant cells and use their contents for food. Bacteria survive in the soil, on plant debris, and on plant hosts.

Nematodes. Nematodes are unsegmented roundworms, sometimes referred to as eelworms (Fig. 7). They are found in soils, in fresh and salt waters, and on plants and animals. The vast majority of nematode species are nonparasitic.

Most plant-parasitic nematodes are slender and vary in length from a few tenths of a millimeter to 2 mm. Usually males and females of the same species are alike in shape, appearance, and size. Sometimes males are slightly smaller. A distinguishing characteristic of plant-parasitic nematodes is the presence in the anterior (head) region of the body of a stylet, a needlelike organ that the nematode inserts into plant cells. Through it, the nematode injects enzymes that aid in cell digestion, resulting in injury to the plant.

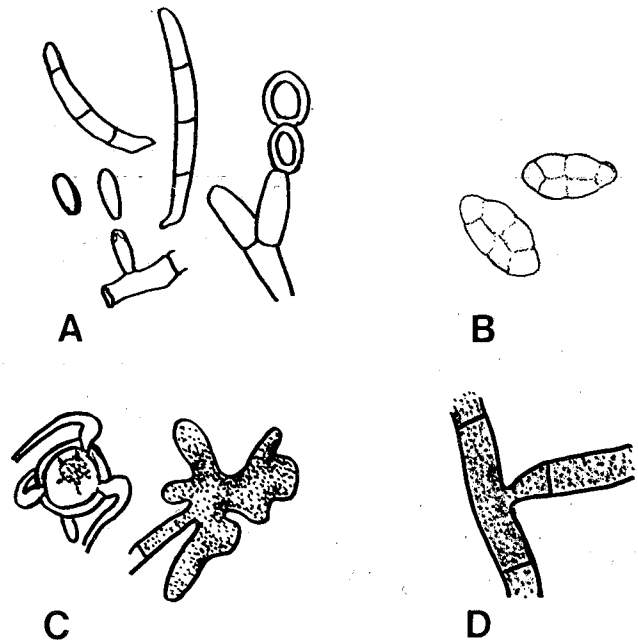

Fig. 4. Representative structures of four fungal pathogens of peanut. **A,** Spores of *Fusarium* spp.; **B,** spores of *Leptosphaerulina crassiasca*; **C,** resting bodies of *Pythium* spp.; and **D,** hyphae of *Rhizoctonia solani*. (Courtesy R. Taber)

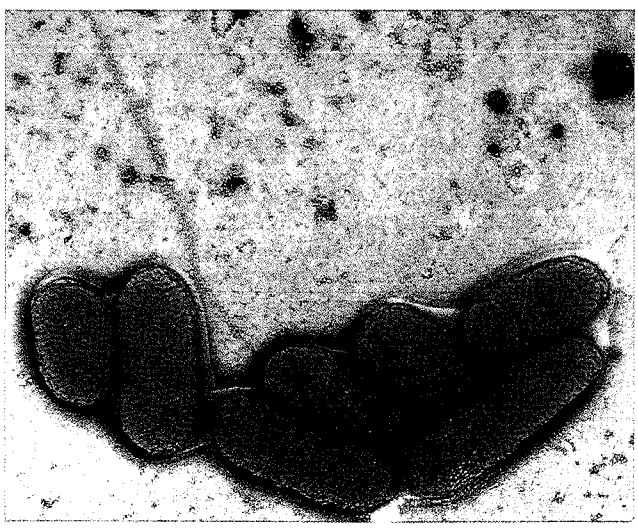

Fig. 5. Cells of the pathogenic bacterium *Pseudomonas solanacearum* (20,000×). (Courtesy R. Gitaitis)

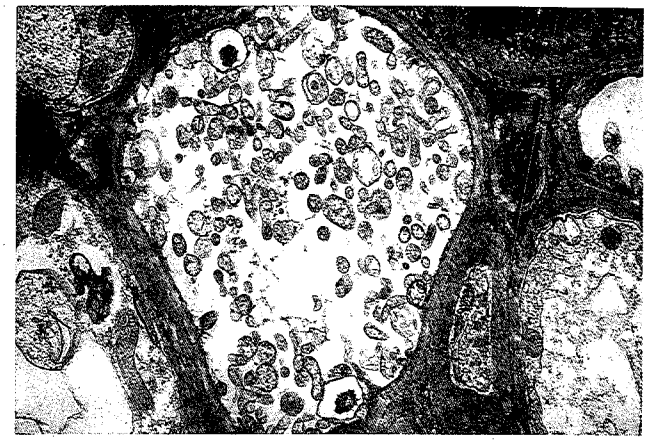

Fig. 6. Phytoplasmas (mycoplasmalike organisms) in phloem. (Courtesy D. Errampalli and J. Fletcher)

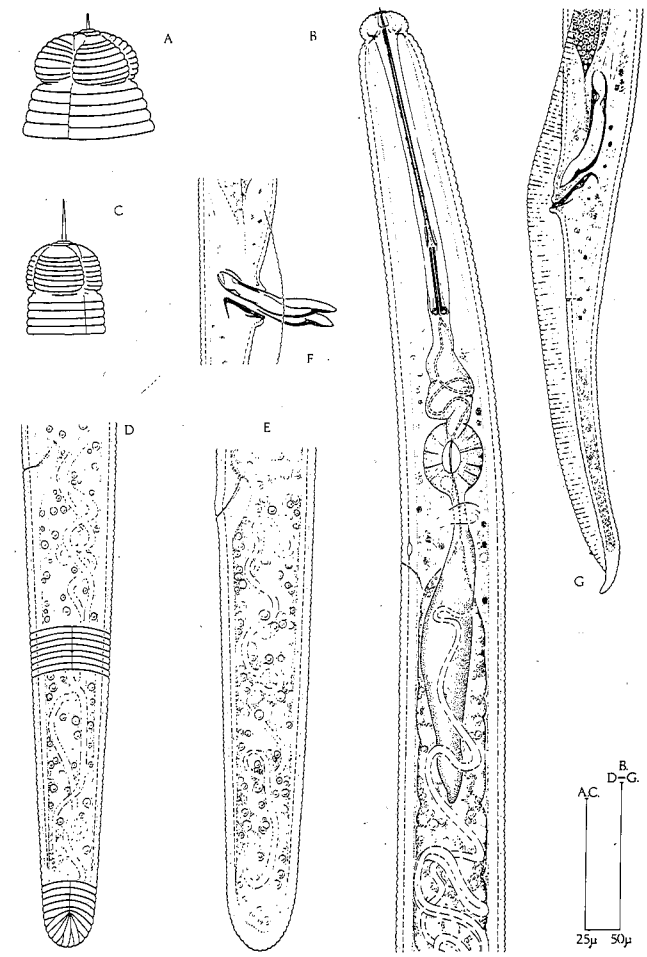

Fig. 7. The sting nematode, *Belonolaimus longicaudatus*. **A,** Female head; **B,** female anterior region; **C,** male head; **D and E,** female tails, with intestine extending into caudal cavity, serpentine lateral canals, and (in **D**) annulation of phasmid region and terminus; **F,** male cloacal region, with spicules extended; and **G,** male tail. (Reprinted, by permission, from C.I.H. Descriptions of Plant-Parasitic Nematodes, Commonwealth Institute of Parasitology; © 1986 C.A.B. International)

Some plant-parasitic nematodes (ectoparasites) feed on the epidermal and cortical cells of roots and never actually enter the roots. Others (endoparasites) penetrate the root tissue and either become sedentary for the rest of their life cycle or migrate after a period of feeding. Sedentary endoparasites, such as root-knot nematodes, show pronounced sexual dimorphism. The females, once they have become established in the plant tissue, lose their slender form and become lemon shaped, pear shaped, or spherical; the adult male remains slender and wormlike.

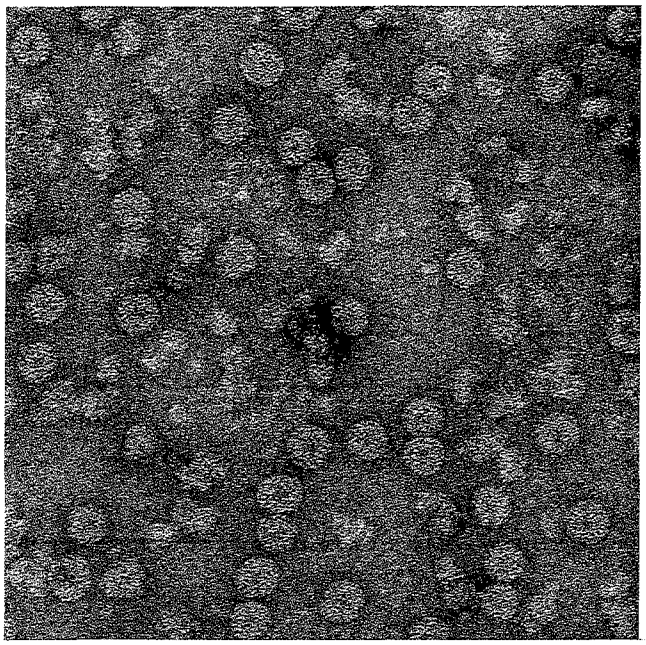

Fig. 8. Peanut stunt virus particles. (Courtesy S. Tolin)

Viruses. Viruses are obligate parasites. They are submicroscopic entities (Fig. 8) not differentiated into cells but composed of a nucleic acid center surrounded by a protein coat. These particles replicate only in a living host plant or in insect vectors. They are transmitted by insects, nematodes, fungi, dodder, and mechanical means; some are transmitted by seed.

Viruses may survive from one growing season to the next in weed hosts, volunteer plants, insect vectors, or seed of the primary host.

Phytoplasmas. A phytoplasma is a prokaryote that lacks a firm outer wall. These organisms commonly occur in conducting tissue such as phloem. Common symptoms of phytoplasma infection include yellowing, internode shortening, phyllody, proliferation of axillary shoots, sterility, virescence, and reduction in root growth. Phytoplasmas can be spread by insects and seed.

Abiotic Agents

Diseases caused by abiotic agents occur wherever peanuts are grown. Pathogens are not associated with these diseases, which are noninfectious. Abiotic diseases interfere with the normal physiological processes of the plant, including those associated with the leaflets, branches, roots, pods, and seed. These diseases may be caused by an excess or lack of a certain substance, e.g., soil moisture (too little moisture results in drought stress; too much moisture results in drowning). Abiotic diseases of the peanut can also be caused by factors such as nutritional imbalance, soil pH, pesticides (too much may cause burn), air pollutants, radiation, and frost.

(Prepared by D. M. Porter)

Part I. Biotic Diseases

Diseases Caused by Fungi

Alternaria Leaf Spot

Foliar diseases of peanut caused by *Alternaria arachidis* Kulk. and *A. alternata* (Fr.:Fr.) Keissl. have been reported in India. Symptoms of Alternaria leaf spot include orange brown lesions in interveinal areas of leaves. These lesions often extend to veins and veinlets (Plate 1).

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(Prepared by D. H. Smith)

Anthracnose

Anthracnose has been reported on peanut in Argentina, India, Senegal, Taiwan, Tanzania, Uganda, and the United States. Although anthracnose has been observed in peanut-production areas of various countries, it is a disease of minor importance.

Symptoms

Brownish gray lesions, marginal to elongate, form on both leaf surfaces and infrequently on petioles and stems of peanut plants infected with *Colletotrichum mangenoti.*

Small (1–3 mm in diameter), water-soaked, yellow spots appear on plants infected with *C. dematium.* Older spots are dark brown. Spots sometimes enlarge rapidly, become irregular, and cover the entire leaf. Petioles are frequently colonized by *C. dematium,* and plants may be killed.

Causal Organisms

C. mangenoti Chevaugeon, *C. arachidis* Sawada, and *C. dematium* (Pers.) Grove are pathogens of *Arachis* spp. *C. dematium* produces circular, erumpent, brown to black acervuli, 75–135 µm in diameter. Setae are black with two to seven septa and 78–146 µm long. Conidiophores (21–28 × 2–4 µm) are hyaline, simple, and erect. Conidia (19–30 × 2.5–4.0 µm), which are produced in pink or creamy masses, are unicellular, hyaline, falcate, and bluntly tapered.

C. arachidis has black, epiphyllous, sparsely scattered acervuli arising from pseudoparenchymatic, dark brown stromata. Conidiophores (13–15 × 4.5 µm) are cylindrical, unicellular, short, and hyaline. Setae are few and black. Conidia (10–15 × 4.5–6 µm) are elliptic, rounded at the apex, rounded or obtuse at the base, hyaline, and unicellular.

C. mangenoti has acervuli that are flattened, lenticular, or elliptic; rose to black; 67–160 µm in diameter; and subepidermal, becoming erumpent. Setae are rigid, erect to subflexuous, continuous or septate, none to numerous, attenuate, brown, and 62–215 µm long. Conidiophores (13.0 × 3.75 µm) are hyaline, cylindrical, granulose or guttulate, and continuous, and both ends are rounded.

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Jackson, C. R., and Bell, D. K. 1969. Diseases of peanut (groundnut) caused by fungi. Ga. Agric. Exp. Stn. Res. Bull. 56.

(Prepared by D. H. Smith)

Aspergillus Crown Rot

Aspergillus crown rot of peanut was first reported from Sumatra in 1926. The pathogen had been reported in 1920 to cause pod and seed discoloration of peanut. Aspergillus crown rot is now an important disease that is probably established in all major peanut-growing areas of the world.

Stand losses caused by the crown rot fungus are variable and difficult to assess. Stand losses in individual fields may be as high as 50% but usually vary from trace levels to 1%.

Symptoms

Seedlings and young plants are very susceptible to infection (Fig. 9). Infection of young plants usually results in high

Fig. 9. Crown rot of peanut seedlings caused by *Aspergillus niger.* Arrows indicate fruiting structures. (Courtesy K. Garren)

mortality rates (Plate 2). As plants mature, they become less susceptible, and the mortality rate declines. In some years, plants may be killed by the fungus throughout the growing season. Seed rot and preemergence damping-off are common phases of the disease, but the most obvious symptom is sudden wilting of young plants. A dark brown discoloration of the vascular tissues is evident in the crowns and roots of wilted plants. The hypocotyls of infected plants also may be swollen. Infection of seedlings commonly occurs in the cotyledons or hypocotyls shortly after germination. Disease progresses rapidly, and infected plants often die within 30 days after planting. Others may survive longer periods, and death of individual limbs or entire plants may occur later in the season. Decayed roots and hypocotyls are often covered with black masses of mycelia, conidiophores, and conidia (Plates 3 and 4). On spanish cultivars, a major symptom of Aspergillus crown rot of older plants is the dead central stem, which is often broken at the soil line.

Causal Organism

Aspergillus niger Tiegh., the causal organism, is ubiquitous in field soils throughout the world. Sometimes *A. pulverulentus* (McAlpine) Thom, a probable mutant of *A. niger*, is also isolated from diseased plants. Colonies of *A. niger* grow well at 25°C on a variety of media, producing abundant large, black, conidial heads that reach 700–800 µm in diameter. Conidiophores are variable, measuring 1.5–3.0 mm × 15–20 µm. Conidia, globose at maturity, are 4.0–5.0 µm in diameter. Sclerotia may be produced by some strains.

Epidemiology

A. niger is widely distributed in soils throughout the world. Growth and sporulation of the fungus are usually favored by warm, moist conditions. Fewer propagules are found in very wet soils than in dry soils. Soil type has not been correlated consistently with prevalence of crown rot, but the disease often is more prevalent in soils low in organic matter.

A. niger is seedborne. The infestation levels of certain seed lots often exceed 90%. Seedlings from such seed usually produce a high percentage of infected plants. However, soilborne inoculum may serve as the primary inoculum. Also, *A. niger* is more prevalent in fields continually cropped to peanut than in fields planted to nonhost crops. Outbreaks of Aspergillus crown rot are sporadic. Predisposition appears to be a major factor in the development of the disease. Drought stress and high temperatures early in the season are associated with crown rot outbreaks. Other adverse conditions such as extreme fluctuations in soil moisture and temperature, poor seed quality, seedling damage from pesticides, insect feeding on roots and crowns, and factors that delay seedling emergence are associated with disease.

Control

All commonly grown cultivars are susceptible to the crown rot fungus. Cultivars that fruit in a bunch pattern usually are more susceptible than runner types. Resistance to *A. niger* has been reported, but it has not been incorporated into agronomically acceptable cultivars. Fungicide seed protectants may provide some control when used where conditions and practices favor rapid germination and seedling emergence.

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(Prepared by H. A. Melouk and J. P. Damicone)

Black Hull

Black hull is an extensive, often cosmetic, black discoloration of peanut shells and pegs that was first observed in the United States in 1960 on New Mexico valencia peanuts (*A. fastigiata* var. *fastigiata*). New Mexico peanut producers have been disproportionately affected by black hull because this state is the major United States producer of peanuts grown for in-shell marketing. Black hull can affect all commercial peanut cultivars but is of economic significance during severe epidemics or on peanuts marketed in-shell. The disease was first reported from Italy in 1949 and has been reported from Argentina, the United States, and most recently (during the 1980s) from South Africa, where severe outbreaks have occurred.

Symptoms

Symptoms and signs of this disease are most commonly found on the external parts of the peanut shell as small, discrete, black spots that often coalesce into large, dark lesions covering almost the whole pod (Fig. 10 and Plate 5). Dark lesions also may be found on the pegs and roots under severe disease pressure, causing significant yield losses in quantity as well as quality. The dark lesions are formed by the production of dark chlamydospores on and in host tissue. The seed coat and seed may also become infected and discolored, leading to seed transmission of the disease.

Causal Organism

The fungus that causes black hull produces two spore types: dark-walled chlamydospores and hyaline or subhyaline to pale

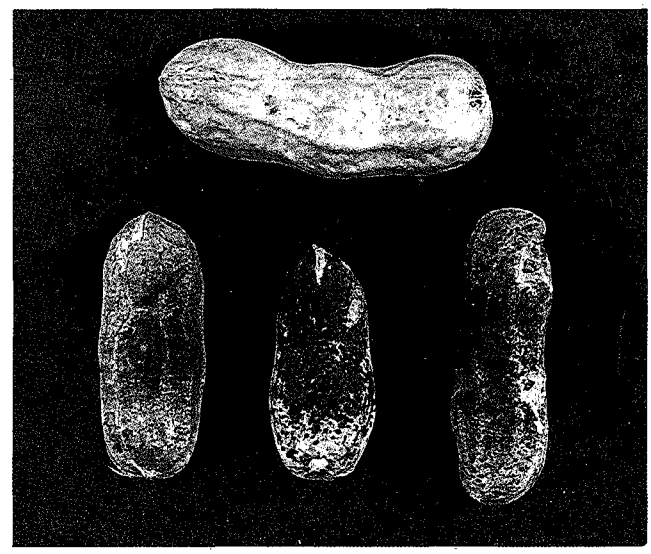

Fig. 10. Symptoms of black hull, caused by *Chalara elegans*, on valencia peanut pods. (Courtesy D. H. Smith)

brown mitospores (enteroblastic phialospores) (Fig. 11). The classification of this fungus and the nomenclature of the two spore stages has been confused since 1910 and remains confused today. The fungus was originally described as *Torula basicola* Berk. & Br. in 1850 from *Pisum* and *Nemophila* spp., entirely on the basis of the chlamydospores. In 1910, the fungus was transferred to *Thielaviopsis basicola* (Berk. & Broome) Ferraris, but this transfer was applicable only to the chlamydospore stage. The mitospore stage remained effectively unnamed until 1975, when both the chlamydospore and mitospore stages were named *Chalara elegans* Nag Raj & Kendrick. The subsequent usage and confusion over the status of these names has led to these stages being erroneously referred to as synanamorphs and the names used as synonyms. Literature on this fungus and the black hull disease is found under both names. However, the only name available for both the mitospore (phialospore) and chlamydospore stages is *Chalara elegans* Nag Raj & Kendrick. Confusion also has occurred in phytopathological literature because of the common occurrence of the ascomycete *Thielavia basicola* Zopf with *C. elegans*. Early literature reported that these fungi were genetically related, and this misconception has persisted into more recent literature. *Thielavia basicola* is not related to *Thielaviopsis basicola* or to *C. elegans*.

C. elegans has distinct sporogenous cells that are borne on simple, erect phialophores, which are cylindrical to subcylindrical, three- to five-septate (occasionally aseptate), subhyaline to pale brown, and 70–95 µm long. The sporogenous cells are swollen at the base with a characteristic cylindrical barrel (collarette). The sporogenous cell is 55–80 µm long, the base is 20–40 × 6–9.5 µm, and the collarette is 25–40 × 3.5–5 µm. The mitospores are enteroblastic phialospores: the first spore is holoblastic, and the following spores are delimited in a retrogressive fashion inside the original wall of the sporogenous cell, leaving a collarette at a fixed sporogenous locus. The mitospores are extruded in chains and are cylindrical to barrellike with truncate or obtuse ends; aseptate; and hyaline, subhyaline, or pale brown (Fig. 11). The chlamydospores are thallic (usually thallic-endoarthritic), intercalary or characteristically terminal, usually in chains of five to seven, and often in rosettes of three or more chains in culture. The terminal chlamydospore is rounded, and the rest are short cylindrical, unicellular, dark brown or amber, and 6.5–14 × 9–13 µm. Chlamydospore chains are 24–55 µm long (Fig. 12). New Mexico isolates of *C. elegans* grow optimally at temperatures of 15.5–19°C and at pH 6.8–7.6.

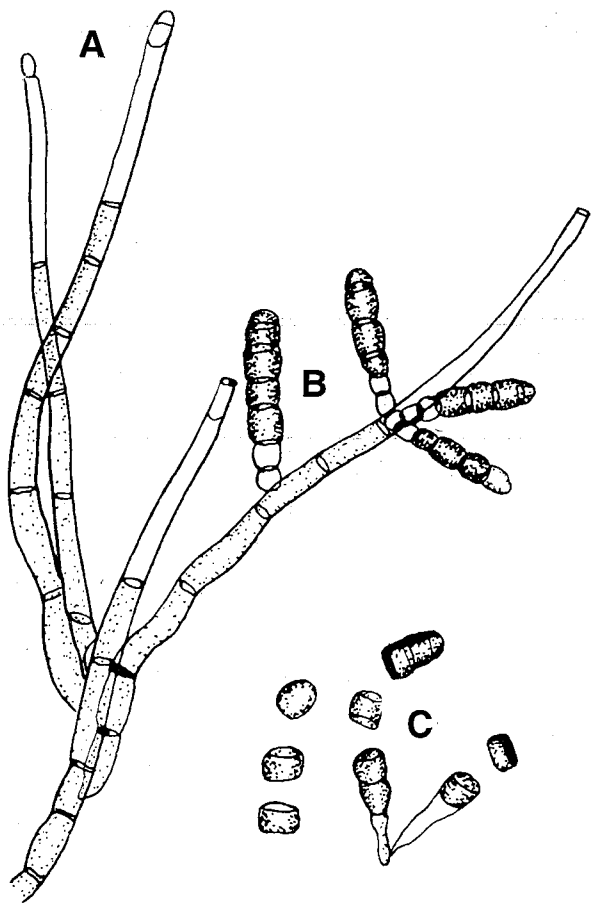

Fig. 11. *Chalara elegans*. **A,** Endoconidial cells and emerging endoconidia; **B,** chlamydospores; and **C,** chlamydospores breaking into segments. (After W. W. Gilbert. Reprinted from Compendium of Alfalfa Diseases, The American Phytopathological Society, 1979)

Disease Cycle

Black hull severity varies from year to year because of climatic variability and changing crop-management practices. *C. elegans* persists indefinitely as chlamydospores in host residue and soil and as a competitive saprophyte growing on soil organic matter. The black hull fungus does not have a known meiotic spore stage (perfect state) and passes through a simple life cycle and only slightly more complex disease cycle. The chlamydospores are the overwintering structures, which germinate to initiate primary infections. The chlamydospore germ tubes infect the peanut directly or indirectly through phialospores, which can be produced rapidly on susceptible tissue and therefore likely act as important sources of secondary inoculum. The fungus penetrates by means of an infection peg formed under a small aggregation of hyphae, although appressoria have also been observed. The infection peg enlarges into a cylindrical structure, which gives rise to short, budlike cells from which the fungus colonizes the host tissue in an inter- and intracellular fashion. Chlamydospore and phialospore production can occur within 72 hr after infection. It is the abundance of the dark-walled chlamydospores on the surface and within the intercellular spaces of the sclerenchymatous mesocarp that gives the black hull disease its name.

Initial inoculum arises from infected hulls and other plant parts left in the field from previous seasons. Factors increasing black hull severity include high soil pH, low temperatures late in the season, overly moist conditions arising from excessive irrigation or rainfall, high-density plantings, the monoculture of peanut, and rotation to peanuts after cotton or sweet potatoes.

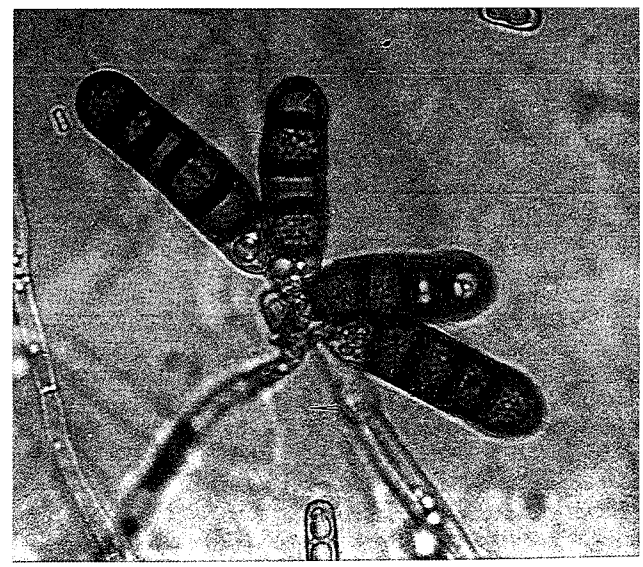

Fig. 12. Chlamydospores of *Chalara elegans*. (Courtesy D. Hsi)

Control

No commercial resistance to black hull is currently available. The disease has decreased in incidence and severity in New Mexico during the past few years because of improved rotation and irrigation practices instigated to control web blotch, another disease favored by cool, moist conditions. Recommendations for control include rotation from peanuts to small grains in 3- to 4-year cycles, use of clean seed, and wider row spacing. The fungicides benomyl and thiophanate-methyl have been shown to reduce black hull incidence. Moving peanut production to new land is no more effective than a good rotation schedule.

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(Prepared by C. M. Liddell)

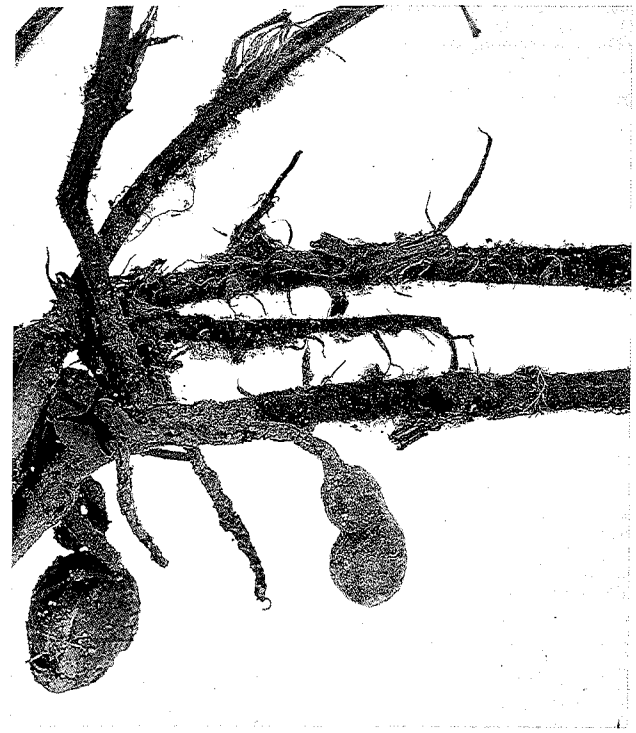

Fig. 13. Botrytis blight on pegs, pods, and stems. (Courtesy K. Garren)

Botrytis Blight

Botrytis blight has been reported in most of the peanut-producing countries of the world. However, damage is usually slight, since environmental conditions conducive to disease development usually are not present during the growing season. Reports of disease occurrence serious enough to greatly reduce yields are rare.

Symptoms

All parts of the peanut plant, above and under the ground, are subject to attack by this soilborne fungus. Plants injured by frost or with reduced vigor from attacks by other pathogens are especially prone to infection, which usually begins on aerial plant parts. Branch tips and branches in contact with the soil surface are especially susceptible.

Peanut leaflets are sometimes infected without any evidence of disease on other plant parts. Infection is characterized by the formation of one to numerous distinct leaf spots on adaxial leaflet surfaces (Plate 6). Leaf spot size may exceed 10 mm in diameter. Conidiophores and conidia are produced sparingly on both adaxial and abaxial leaflet surfaces (Plate 7). A *Gliocladium* species has been constantly associated with the leaf spot fungus and parasitizes conidia, conidiophores (Plate 8), and sclerotia of *Botrytis.*

Under favorable environmental conditions, the causal organism colonizes plant parts rapidly, causing wilt and death of individual limbs or entire plants (Fig. 13 and Plate 9). The fungus moves rapidly from aerial plant parts into underground parts. Botrytis blight is characterized by the profuse production of conidia (Plates 10 and 11) and sclerotia (Fig. 14) on infected plant parts.

Causal Organism

Botrytis cinerea Pers.:Fr. bears conidia on the tips of erect conidiophores that cover the surface of the host substrate (Fig. 15 and Plate 10). Conidia (9–12 × 6.5–10 µm) are ellipsoid to ovoid and one celled with almost hyaline walls. Conidiophores are

Fig. 14. Sclerotia of *Botrytis cinerea* on pods and a stem. (Courtesy K. Garren)

usually unbranched, septate, and 11–23 µm thick. Projections form at the tips, and from these, conidia are formed so abundantly that the infected plant parts become dustlike and gray (Plate 11). Conidia are disseminated readily by air currents. Sclerotia (1–5 mm long) are hard, black, and irregularly shaped (Figs. 14 and 16). They are rounded on the upper surface and flat or concavely depressed on the underside when attached to infected substrate (Fig. 14). The ascomycetous stage of *B. cinerea, Botryotinia fuckeliana* (de Bary) Whetzel, is rarely observed.

Disease Cycle

Temperatures below 20°C accompanied by heavy dews or excessive rainfall are prerequisites for infection by *B. cinerea.* Senescing, frost-injured, and mechanically injured plant parts also are prone to colonization. Organic debris, such as defoliated leaflets and abscised flower parts, on the surface of the soil serves as a food base and aids in the infection process. *B. cinerea* overwinters in the sclerotial form in the soil. Although apothecia have been reported, the primary inoculum source

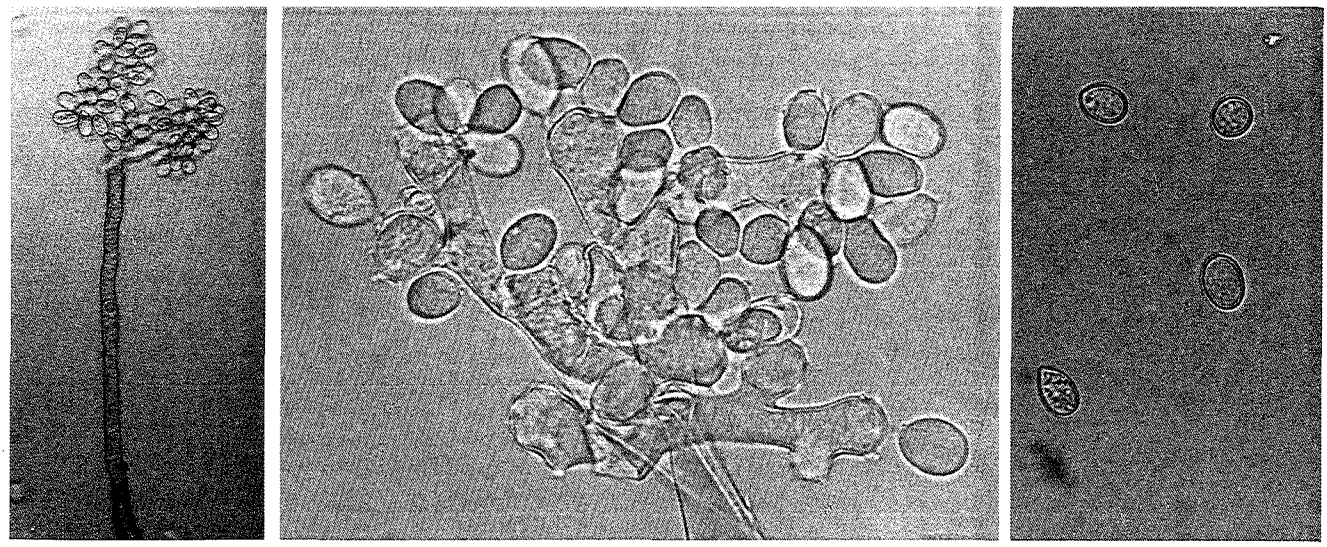

Fig. 15. Conidia of *Botrytis cinerea.* Left and center, conidia on conidiophores. (Reprinted from Compendium of Potato Diseases, The American Phytopathological Society, 1981)

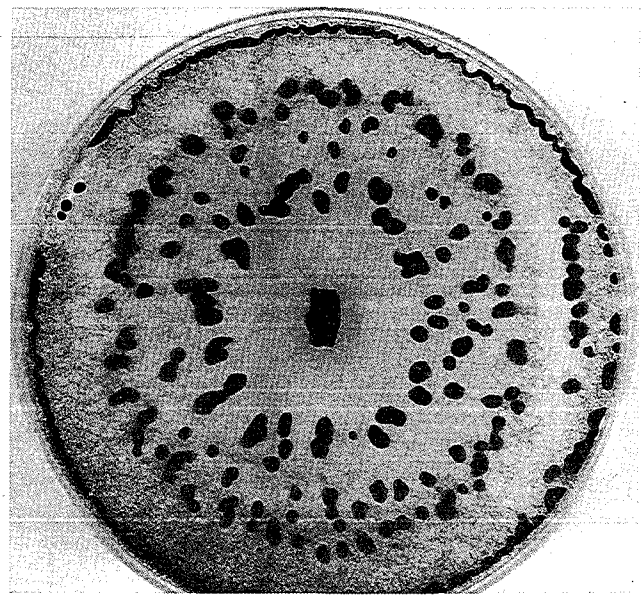

Fig. 16. Culture of *Botrytis cinerea* on potato-dextrose agar showing sclerotia.

appears to be mycelium originating from germinating sclerotia or omnipresent conidia. Conidia are dispersed by wind and rain.

Control

Foliar sprays with fungicides such as benomyl and chlorothalonil offer some protection against *B. cinerea.* The use of early-maturing peanut cultivars might lessen frost damage and thereby reduce disease severity.

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(Prepared by D. M. Porter)

Charcoal Rot

Charcoal rot, caused by the common soilborne fungus *Macrophomina phaseolina,* is widely distributed in most peanut-producing countries. The fungus is distributed throughout the world and causes diseases in a wide range of crops. In peanut, it is responsible for seed and seedling rots, wilt, root and stem rots, leaf spot, rotting of developing pods and seed, and concealed damage. The term "charcoal rot" is used to describe the damage done to roots and stems of seedlings and older plants. Charcoal rot is of only minor importance in the United States, although occasionally it can greatly reduce plant stands. In India, it is the most common disease of peanut seedlings.

Symptoms

The occurrence of water-soaked lesions on the hypocotyl near the soil surface is a characteristic sign of this disease. After the hypocotyl is girdled, the seedling dies. Similar symptoms are observed on older plants at the soil line, although all plant parts at all stages of growth are susceptible. Stem and root lesions appear water soaked at first, but infected tissues later become a dull light brown (Plates 12 and 13). The infection extends down into the taproot and up into the stem and branches (Plate 14). When lesions girdle the stem, the plant wilts, and the fungus rapidly colonizes the branches, which turn brown and die. The dead tissues rot and turn black as sclerotia of the fungus develop profusely. Roots, pegs, and pods also rot and become covered with sclerotia. In some cases, the disease is at first restricted mainly to the roots, which become rotted and blackened and the taproot shreds. The foliage of such plants turns yellow and wilts, and the typical symptoms of stem blight and charcoal rot appear.

Causal Organism

M. phaseolina (Tassi) Goidanich, the pycnidial state of *Rhizoctonia bataticola* (Taubenhaus) E. J. Butler or *Sclerotium bataticola* Taubenhaus, was derived from *Macrophoma phaseolina* by Tassi in 1901. The sterile mycelial phase of *M. phaseolina* was first named *S. bataticola* by Taubenhaus but was later transferred to the genus *Rhizoctonia.*

Pycnidia of *M. phaseolina* (100–200 μm in diameter) are membranous to subcarbonaceous, first immersed and then at least partially erumpent, and globose or flattened globose with

inconspicuous truncate ostioles. Their walls are composed of several layers of dark, thin-walled, angular cells, 9 µm in diameter, and are lined with a hyaline layer two or three cells thick bearing simple, rod-shaped conidiophores, 10–15 µm long. Conidia (14–33 × 6–12 µm) are single celled, hyaline, and elliptic or oval.

Disease Cycle

Charcoal rot is both seedborne and soilborne. Mycelium in seed and mycelium and sclerotia in plant debris in the soil are the primary sources of inoculum (Fig. 17). The sclerotia can remain viable in dry soil for many years but rapidly lose viability in very wet soils.

M. phaseolina is commonly present in peanut seed and pods and can readily be disseminated by their movement. High soil temperatures (about 35°C) and low soil osmotic potential reduce plant vigor and favor growth of the fungus and development of charcoal rot. Fungal growth in pods is increased by rain after harvest. Damage during harvesting and shelling predisposes pods and kernels to injury from this pathogen.

Control

Crop rotation is generally ineffective in reducing soil inoculum because the fungus can grow saprophytically and has a wide host range. However, rotation of peanut with rice for 2–3 years may reduce the soilborne inoculum of *M. phaseolina.* Crop sanitation (e.g., burning crop residues) may help reduce disease levels. Providing adequate fertilizer and soil water to ensure good crop growth should reduce charcoal rot development. Frequent irrigation to keep the soil wet reduces the viability of sclerotia.

Seed treatment with fungicides such as captan and thiram can reduce seedborne infection and provide the germinating seed some protection from invasion by the fungus from soilborne inoculum. Soil drenching with pentachloronitrobenzene (PCNB) can give some control of the disease. No immune or highly resistant peanut genotypes are available.

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(Prepared by V. K. Mehan and D. McDonald)

Choanephora Leaf Spot

Choanephora sp. has been observed on peanut leaves in the Philippines, Thailand, Senegal, and Uganda. Brown lesions originate at the leaflet margin and spread over the entire leaflet. Abundant sporulation occurs on both leaflet surfaces and down the petioles. Defoliation of infected leaflets may occur.

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(Prepared by R. A. Taber)

Cylindrocladium Black Rot

Cylindrocladium black rot (CBR) of peanut was first observed in Georgia in 1965. Soon thereafter, CBR was recog-

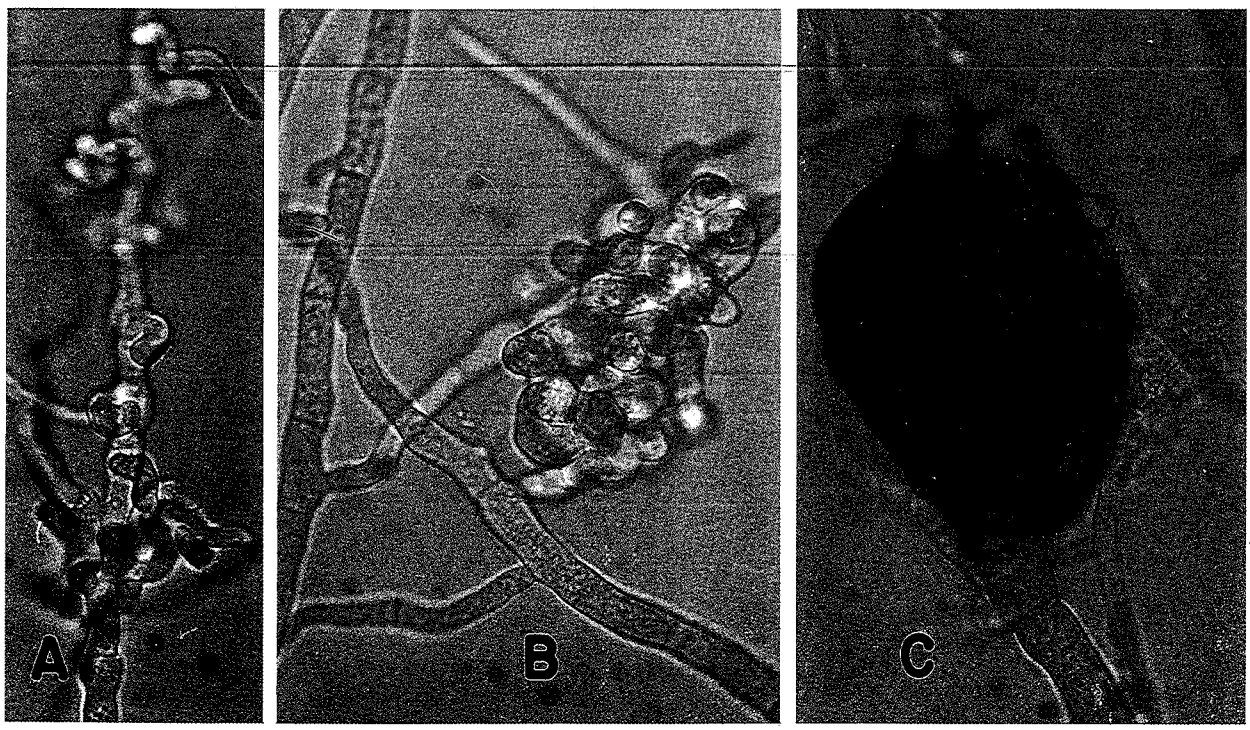

Fig. 17. Sclerotial development of *Macrophomina phaseolina.* **A,** Proliferation of a single hypha; **B,** aggregation of several hyphae; and **C,** mature sclerotium. (Reprinted, by permission, from Jackson and Bell, 1969)

nized in other peanut-producing areas of the United States and in Japan, India, and Australia. The disease has been a cause of major concern, particularly in Virginia and North Carolina because of its widespread occurrence and chronic threat to peanut production in these states. In other parts of the United States, outbreaks of CBR have remained static, and yield losses are generally secondary in importance to those caused by other soilborne diseases.

Symptoms

The first visible symptoms of CBR are chlorosis and wilting of leaves on the erect, primary stem of a plant. Entire plants may wilt and die at an alarming rate when prolonged periods of high soil moisture are followed by a period of moisture stress. In the absence of ideal conditions for disease development, plants may develop only a chlorotic, stunted appearance. When CBR is seen for the first time in a field, the diseased plants are usually observed in one or more localized spots (Plate 15). Aboveground symptoms commonly include chlorosis, wilt, stunted growth, and death.

All below-ground plant parts may develop symptoms of CBR. Hypocotyls, primary and secondary roots, and pods become black and necrotic (Plate 16). The taproot is often necrotic and severely decayed in plants with aboveground symptoms.

A diagnostic sign of CBR is the occurrence of small, reddish orange perithecia of the pathogen in dense clusters on stems (Plate 17), pegs, and occasionally pods. These fruiting bodies develop on tissues just above and below the soil surface during periods of wet, humid weather. If perithecia are not found on diseased plants, tissue samples must be assayed in a laboratory to positively identify the disease as CBR. These structures are sometimes confused in the field with the somewhat smaller, rounder perithecia of *Neocosmospora* sp., a common saprophyte that colonizes dead plant parts that are in contact with soil.

Causal Organism

Numerous publications since 1966 have referred to the causal organism as *Cylindrocladium crotalariae* (C. A. Loos) D. K. Bell & Sobers (teleomorph *Calonectria crotalariae* (C. A. Loos) D. K. Bell & Sobers). Recently, the name was changed to *Cylindrocladium parasiticum* Crous, Wingfield, & Alfenas (teleomorph *Calonectria ilicicola* Boedijin & Reitsma). The fungus grows well on potato-dextrose agar, producing light yellow to white weblike aerial mycelium and a burnt orange to dark brown submerged growth. Conidiophores are borne laterally on a main axis or stipe that terminates in a hyaline, globose swelling (vesicle) measuring 6–13 µm in diameter (Fig. 18). Conidiophores develop reniform or sometimes

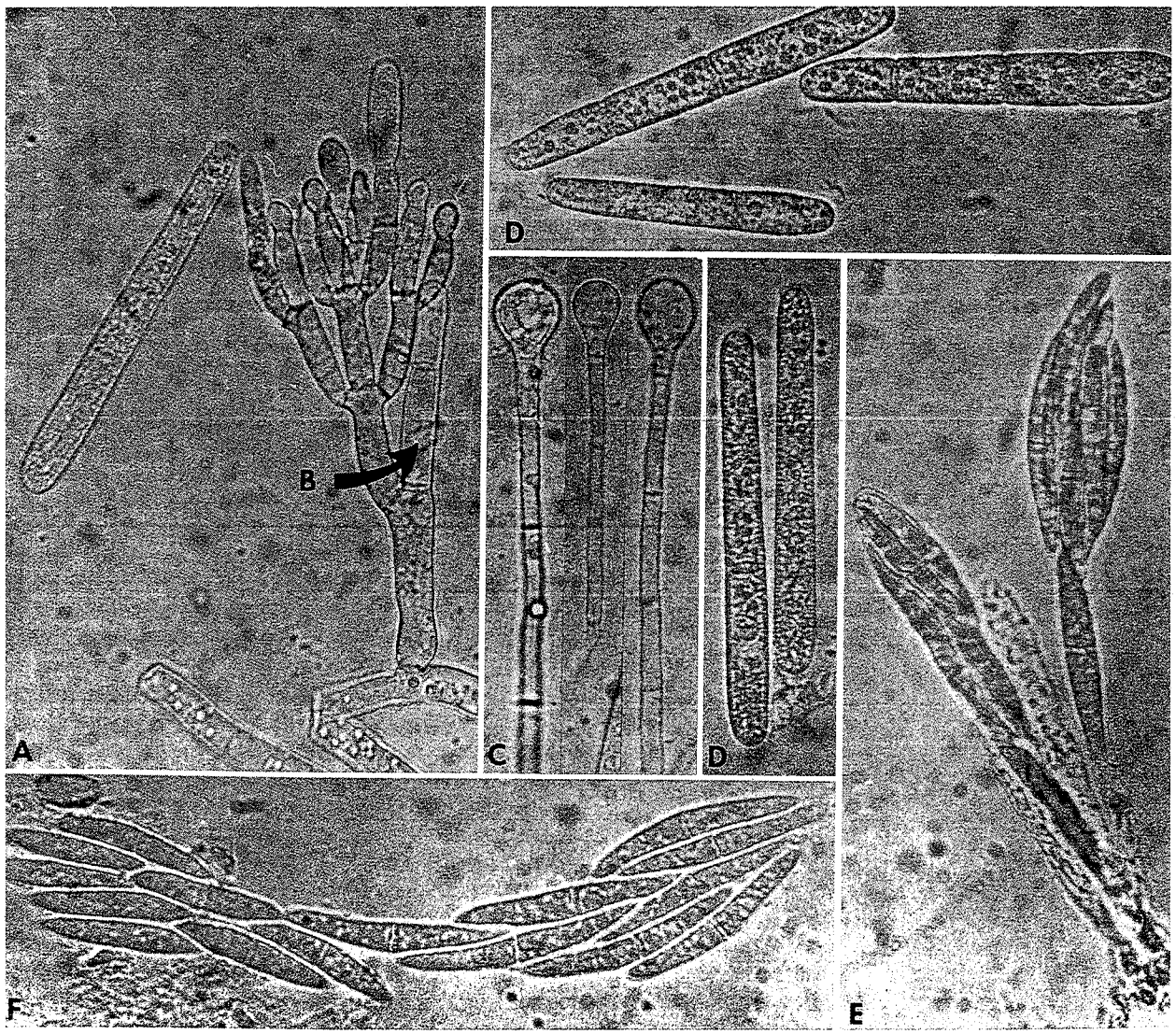

Fig. 18. **A,** Conidiophore, **B,** immature stipe, **C,** vesicles, and **D,** conidia of *Cylindrocladium parasiticum*; **E,** asci and ascospores and **F,** ascospores of *Calonectria ilicicola*. (Reprinted from Bell and Sobers, 1966)

doliform phialides on tertiary branches. Conidia (58–107 × 4.8–7.1 μm) arise by budding from the apexes of phialides and are hyaline, cylindrical, and rounded at both ends and most often have three septa.

Perithecia (Fig. 19) (320–465 × 290–370 μm) form on host tissues and in axenic cultures incubated under lights and are subglobose to oval or obovate and orange to red. Asci are hyaline and contain eight fusoid to falcate, mostly one-septate ascospores (34–58 × 6.3–7.8 μm).

Microsclerotia of *C. parasiticum* are readily visible in cortical tissues and *Rhizobium* nodules of infected peanut roots with the use of histochemical clearing agents (Fig. 20). These propagules are burnt orange to dark brown and are composed of a dense cluster of cells that resemble chlamydospores. Microsclerotia develop abundantly in axenic cultures of *C. parasiticum* on media with high carbon-nitrogen ratios.

Disease Cycle

Microsclerotia of *C. parasiticum* are responsible for overwintering and long-term survival of the fungus in soil. Microsclerotia in crop debris expelled from peanut combines may be carried long distances by prevailing winds (Fig. 21). As infected tissues decompose, microsclerotia are released into the soil and disseminated by tillage equipment. Of the crops commonly grown in rotation with peanut (i.e., soybean, tobacco, cotton, corn, and small grains), only soybean serves as a host of *C. parasiticum* and thereby can further increase populations of microsclerotia in field soil. Soil temperatures have a profound influence on survival of microsclerotia. Cold winters that freeze soil water in the plow layer and sustained periods of cold at or below 5°C can result in a marked reductions in populations of viable microsclerotia in infested fields.

Field studies have shown that microsclerotia of *C. parasiticum* occur in clumps or clusters in the field rather than in a random or uniform distribution. Studies relating inoculum density and disease incidence have demonstrated that the number of observed infections on roots and the level of symptom expression by plants are directly proportional to microsclerotial densities in soil. Soil temperatures of 20–25°C and moisture levels near field capacity are most conducive to infection and rot of peanut roots by *C. parasiticum*. Root infection is suppressed markedly at low soil moisture levels and soil temperatures of 30°C and above.

The primary infection court for *C. parasiticum* on peanut is believed to be near the root tips. Root exudates in this region probably trigger microsclerotial germination and subsequent infection processes. After germination, intercellular penetration of the cortex occurs within 12–24 hr and penetration of the root stele may occur within 48 hr. As necrosis develops in infected roots, hyphae of the fungus begin to produce microsclerotia. Perithecia of *C. parasiticum* may develop on the surfaces of diseased roots and basal stems near the soil surface during moist periods. Ascospores are forcibly discharged into the air when perithecia first mature. In the final phase of development, ascospores exude in a viscous ooze from the ostiole, which may facilitate dispersal by rain or possibly insects. Because of the late-season development of ascospores and the rapid loss of viability with desiccation, their epidemiological importance is believed to be of minor significance.

Although *C. parasiticum* is easily isolated from freshly dug peanut seed, the frequency of its detection in commercially harvested seed is usually quite low. The viability of the fungus in seed is reduced by curing and storage practices and by fungicide seed treatments. The risk for seedborne spread of CBR has been difficult to measure quantitatively but should not be discounted as a factor in pathogen dispersal.

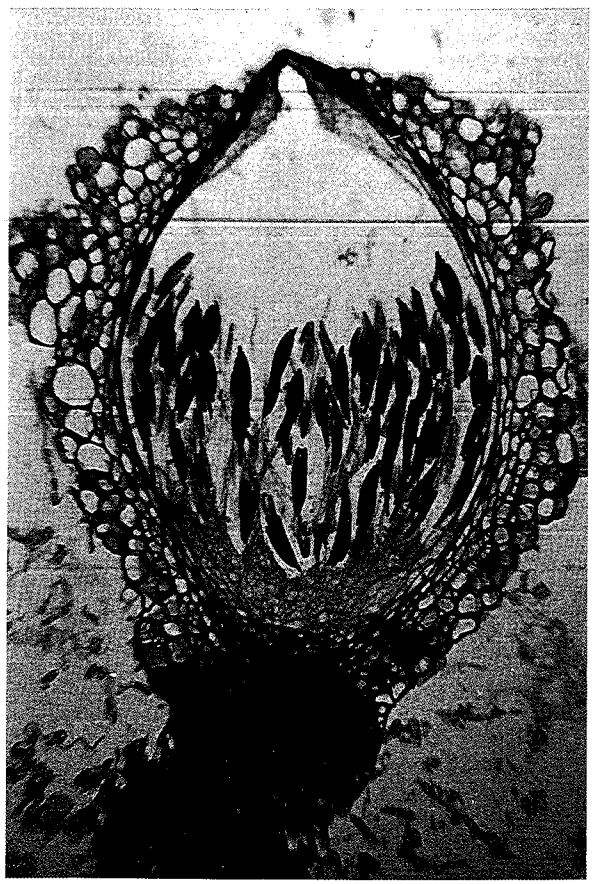

Fig. 19. Perithecium of *Calonectria ilicicola* containing asci and ascospores. (Courtesy R. Rowe)

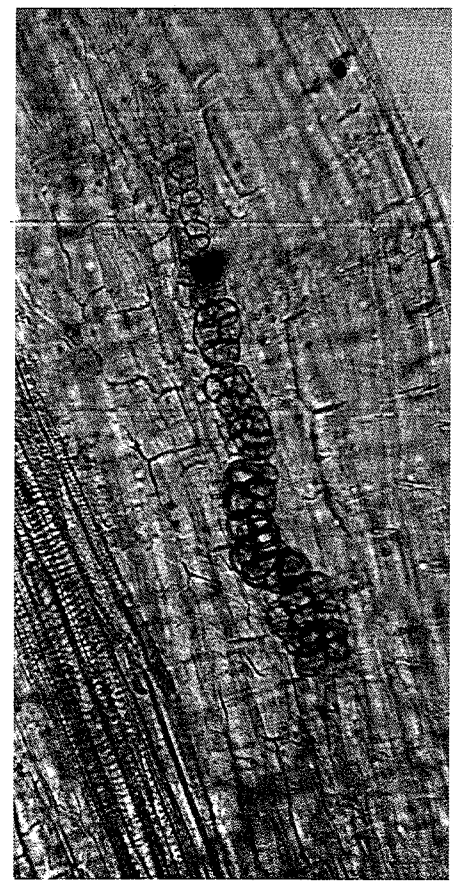

Fig. 20. Microsclerotium of *Cylindrocladium parasiticum* in the cortex of a peanut root. (Courtesy R. Rowe)

Control

The development and release of disease-resistant cultivars of peanut continue to make a significant contribution to CBR management. In general, spanish cultivars are most resistant to CBR, valencia cultivars are the most susceptible, and virginia cultivars are moderately susceptible. Although CBR-resistant lines of each type have been described and released, only one commercial cultivar (NC 10C) of the virginia type offers partial resistance at this time. Carefully planned crop rotation and cultural practices are necessary in deploying a CBR-resistant cultivar. The severity of CBR increases when roots are parasitized by the northern root-knot nematode (*Meloidogyne hapla*) and the ring nematode (*Criconemella ornata*). An increase in the severity of CBR on peanut may also result from root injury caused by certain preplant herbicides. Soils relatively high in organic matter and with a greater capacity for moisture retention generally favor disease development. Low soil temperatures and high levels of soil moisture early in the growing season are most conducive to CBR. Warm soil and low soil moisture are generally unfavorable for root infection.

Because of cooler soil conditions and the probability for higher levels of soil moisture, peanuts planted early are more vulnerable to CBR damage than those planted late. Crop rotation with nonhosts such as corn, small grains, cotton, or tobacco may help reduce the incidence of CBR, whereas rotations with leguminous crops such as soybean can result in increased populations of *C. parasiticum* in the soil. Tillage practices that maximize exposure of microsclerotia to winter temperatures may reduce populations significantly. Such practices include removal and destruction of peanut haulm (hay), omission of a winter cover crop, and no soil tillage until spring. Movement of the pathogen from field to field can be minimized by cleaning field implements and combines to remove adhering soil and plant debris.

An early spring application of metham sodium at 36 kg/ha (Vapam, 10 gallons per acre) is widely used for CBR control in heavily infested fields in Virginia and North Carolina. The fumigant is applied through chisel shanks under each row (8–10 in. deep) at least 2 weeks prior to planting and when soil temperatures are at or above 15°C. At the time of application, rows are bedded to facilitate the alignment of planters over sites of fumigant placement. Control is achieved by killing the fungus in the zone of taproot growth. Lateral roots outside the zone of fumigant activity may become infected, but disease progress from these points of infection generally fails to result in plant destruction or significant loss of yield.

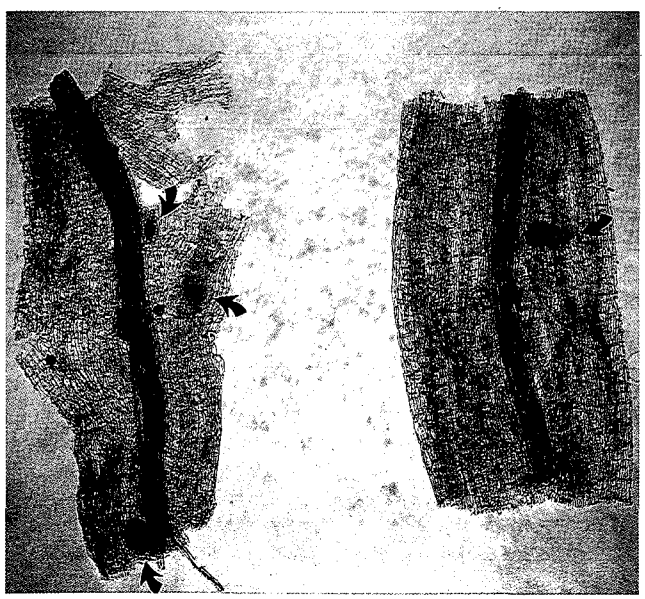

Fig. 21. Microsclerotia (arrows) in windblown debris from a combine. (Courtesy R. Rowe)

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(Prepared by P. M. Phipps and M. K. Beute)

Delimited Shell Spot

Delimited shell spot (DSS), a blemish of peanut pods, is limited to the Mediterranean region. Affected peanut crops are frequent in poorly aerated soils. Spots are abundant when a legume is included in the crop rotation. DSS greatly reduces the quality of the pods. Also, various fungi frequently invade the pod cavity through the necrotic lesions and cause seed decay.

Symptoms

A spot originating as a transitory and inconspicuous tiny depression occurs in or on the surface of the fleshy, lignifying pod shell. A typical spot (approximately 5 mm in diameter) develops as lignification proceeds. The spot becomes necrotic, dry, and light beige to pale grayish beige and is delineated from the unspotted areas of the shell by a distinct, dark tan margin. Cells below the spot are rich in starch granules and are thin walled, whereas in unaffected cells, starch granules gradually disappear and the cell walls thicken. Spots frequently are aggregated in clusters (Plate 18). Secondary invaders often alter spot appearance: it may darken in the center, become necrotic, or expand concentrically (Plate 19 and Fig. 22).

Fig. 22. Pods with delimited shell spots.

Causal Agents

The etiology of DSS is, for the most part, uncertain. That the causal agent, or group of agents, is biotic can be demonstrated by control with physical and chemical, broad-spectrum soil treatments. Nematodes are not involved in the DSS complex. Inoculation of peanut plants with microorganisms that were isolated from plants with DSS symptoms (at different developmental phases of pods and lesions) did not cause DSS. Statistical analysis of spot distribution on individual pods proved that the causal factor does not attack the shell randomly. Moreover, the clustering of spots is reminiscent of an arthropod feeding around its first probe. Indeed, when various selective biocides were applied to soils with a history of DSS, several insecticides and acaricides reduced spot numbers significantly.

Small populations of *Rhizoglyphus* mites were consistently associated with DSS. However, infestation of pods with laboratory-grown gnotobiotic populations of *Rhizoglyphus* sp. did not induce DSS development. The following is hypothesized: 1) in nature, *Rhizoglyphus* sp., or another microarthropod, may serve as a vector for a submicroscopic parasite of endemic legumes that causes local lesions to the pod shell of the *Arachis* plant; 2) in cryptoaerobic microsites that prevail at pod-to-soil interfaces in poorly aerated soil, even tiny wounds do not heal immediately, and each spot expands until arrested by the surrounding tissue; therefore, the number of spots increases with time.

Control

To reduce the risk of DSS development, poorly aerated soils should be avoided and crop rotation should exclude leguminous crops or weeds. Winter cereals cut green (for fodder) preceding peanuts reduce DSS abundance significantly. Deep plowing improves soil aeration and further reduces DSS. Mechanized formalin applications to the seedbed (3,000 liters per hectare of a 37% formaldehyde solution) were found to be cost effective and reduced DSS. Experimentally, seedbed solarization of metham-treated soil was highly effective in reducing DSS, but it is not cost effective. Nevertheless, in severely affected sites, all these measures do not eliminate DSS unless they are used in combination with resistant genotypes. These genotypes combine shallow fruiting (thus partially escaping cryptoaerobic conditions) with a hardy pod shell and bear few spots. Unfortunately, yield of these genotypes is low, but they have potential as sources of resistance in breeding programs.

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(Prepared by Z. R. Frank)

Diplodia Collar Rot

Diplodia collar rot usually occurs sporadically throughout the world, rarely causing economically important losses. In 1993, however, collar rot was severe on several farms in Virginia (Plate 20). Diseased areas often exceeded one acre in size. In such areas, almost all plants exhibited typical collar rot symptoms. Yield reductions caused by collar rot usually are less than 1%, but reductions of 25% or more have been reported. In Virginia in 1993, yield losses in severely diseased fields exceeded 75%.

Symptoms

The causal agent, a common soilborne saprophyte, is usually associated with the peanut plant only as a wound parasite or as a secondary pathogen. Peanut seedlings and older plants, including pods, are subject to attack (Plate 21). Wilting of a lateral branch or the entire plant is usually the first symptom of disease in older plants. Wilt develops rapidly, and the plant usually dies within a few days. Lesions that develop on aboveground stems are characterized by elongated necrotic areas with light brown centers and dark brown margins. Infected roots become slate gray to black and shred easily. Pycnidia embedded in the infected host tissue resemble small, erumpent, black dots (Plate 22). The fungus also causes concealed damage of peanut seed that is not visible on the external surface. Originally, the term "concealed damage" was restricted to damage caused by quiescent, natural, pod mycoflora. However, it now encompasses mechanical damage, damage associated with calcium and boron deficiencies, and damage caused by some pathogenic fungi such as *Diplodia gossypina*.

Causal Organism

Although *Lasiodiplodia theobromae* (Pat.) Griffon & Maubl. and *Botryodiplodia theobromae* Pat. have been associated with peanuts exhibiting symptoms of collar rot, *Diplodia gossypina* (Cooke) probably is the causal agent of collar rot of peanut. Simple or compound pycnidia occur either singly or in groups on the surfaces of necrotic tissues. They may appear immersed or erumpent, and they possess a prominent ostiole. Pycnidia range in diameter up to 400 µm. Conidia (Plate 23) are borne on short conidiophores. Mature pycnidiospores (17–34 × 10–18 µm) are elliptic with one septum. Mature, two-celled conidia are brown and lack longitudinal striations.

Disease Cycle

Mycelium and mature conidia of *D. gossypina* can remain dormant in soil and plant debris for long periods. Germination and infection occur when a suitable substrate becomes available. Peanut plant tissue predisposed by heat stress is more subject to colonization by *D. gossypina* than is similar tissue not so predisposed. However, predisposition is not a prerequisite for infection. Primary infection may be caused by mycelium originating from germinating, mature conidia or mycelial fragments. Upon penetration, the fungus grows from one cell to another throughout the cortical parenchyma. Hot, dry weather favors disease development.

D. gossypina can be isolated from up to 10% of seed from fields exhibiting severe disease symptoms. Seed-treatment fungicides reduce the incidence of *D. gossypina* but do not eradicate it, thus implying the possibility of seed transmission. The causal organism also can be isolated from seed from apparently healthy plants not exhibiting aboveground disease symptoms.

Control

Rotations with nonhost crops can reduce disease incidence. Although resistance to *D. gossypina* is not available in commercial cultivars, some breeding lines, including F 334 AB-14 and Florispan Runner, possess high degrees of tolerance to this disease and are available for use in breeding programs. By manipulating row orientation and controlling foliage diseases so that foliage is maintained to provide shade throughout the growing season, one can minimize heat injury to plant basal stems and perhaps reduce disease incidence.

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(Prepared by D. M. Porter and P. M. Phipps)

Early and Late Leaf Spots

Many of the diseases of peanut have a limited geographic range, but the two major foliar diseases, early and late leaf spots, occur wherever peanut is grown. Other common names for these diseases include Cercospora leaf spots, tikka leaf spots, peanut cercosporosis, Mycosphaerella leaf spot, and brown leaf spot. In some areas, early leaf spot is the predominant disease, and in others, late leaf spot is predominant. For example, early leaf spot was the predominant foliar disease of peanut in the southeastern United States during the 1960s and 1970s, but late leaf spot predominated there during the 1980s. Recently, early leaf spot has again become predominant in some areas of this region. Similar changes in the relative incidence and importance of early and late leaf spots have been observed in other areas of the world.

When fungicide sprays are not used, pod yield losses of up to 50% are common. Losses to late leaf spot as high as 70% have been recorded in research plots where the disease was not controlled. The quantitative relationship between these foliar diseases and pod yield loss is related to defoliation and time of harvest, as shown in Figure 23. For a given level of defoliation, pod yield loss increases with delays in harvest.

Symptoms and Signs

Although the diseases are called leaf spots, the symptoms of both diseases develop on petioles, stipules, stems, and even pegs during the later stages of an epidemic. Lesions are first visible about 10 days after spore deposition as small, chlorotic flecks. These flecks become darker lesions, enlarging to 1–10 mm in diameter (Plates 24 and 25). Mature, sporulating lesions may be apparent by about 15 days after spores are deposited. Early and late leaf spots often look very similar on the upper (adaxial) surfaces of leaflets, but early leaf spot usually has a prominent yellow halo (Plate 25) that is often less conspicuous or absent from late leaf spot lesions. The presence, absence, or distinctiveness of a halo, however, is not a reliable characteristic for distinguishing between early and late leaf spot. On some cultivars (e.g., Florunner) early leaf spot lesions are typically light tan to reddish brown on the undersides (abaxial surfaces) of leaflets, whereas late leaf spot lesions will usually be dark brown to black. This characteristic also may be unreliable on many cultivars because lesions often differ in color on the abaxial surface. The most reliable method of distinguishing between the two leaf spots is identification of conidia by microscopic examination. Sporulation of the early leaf spot fungus is more prevalent on the adaxial surfaces of leaflets, whereas sporulation of the late leaf spot fungus occurs more often on the abaxial surfaces. Conidia are sometimes absent from early leaf spot lesions but are generally present on late leaf spot lesions. When present, conidia are often sparse and light in color on early leaf spot lesions, but conidia on late leaf spot lesions will usually be dark and borne in tight clusters arranged in concentric rings. If leaf spots are actively sporulating, distinction between early and late leaf spot is often possible with a 10× hand lens.

Neither early nor late leaf spot is likely to be confused with other foliar diseases, but symptoms caused by certain phytotoxic pesticides are very similar to leaf spot symptoms. This is particularly a problem where early-season herbicides are used for management of weeds or where systemic insecticides are used for thrips control. Spots caused by pesticide injury are usually slightly lighter in color in the center, and there is no sporulation on the spots. Leaf spots caused by pesticide injury are also less clearly defined on the abaxial leaf surface.

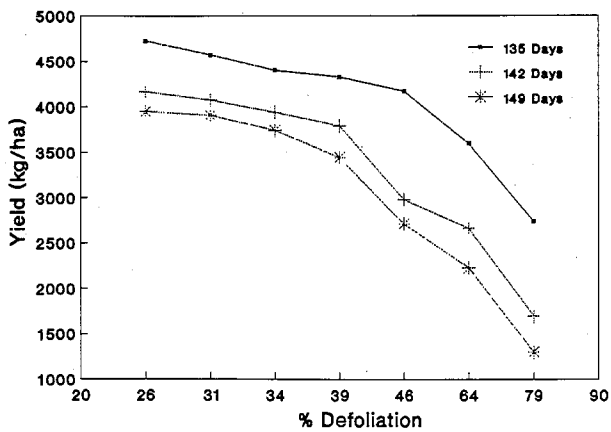

Fig. 23. Effects of defoliation caused by leaf spot diseases on yield of peanut. Note that yield loss is greater for a given level of defoliation when harvest is delayed.

Causal Organisms

Early leaf spot. The anamorph of early leaf spot, *Cercospora arachidicola* S. Hori, is commonly present on lesions. Fruiting of *C. arachidicola* is amphigenous; however, conidia form primarily on the adaxial surfaces of lesions. Stromata are dark brown and up to 100 µm in diameter. Pale olivaceous or yellowish brown conidiophores (15–45 × 3–6 µm) form in dense fascicles (Fig. 24), five to many in number. Conidiophores are darker at the base, mostly once geniculate, unbranched, and septate. Subhyaline conidia (35–110 × 3–6 µm) are olivaceous, obclavate, and often curved. Each has three to 12 septa, a truncate base, and a subacute tip (Fig. 25). The teleomorph of the early leaf spot pathogen, *Mycosphaerella arachidis* Deighton, as described by Jenkins, is rarely observed on peanut.

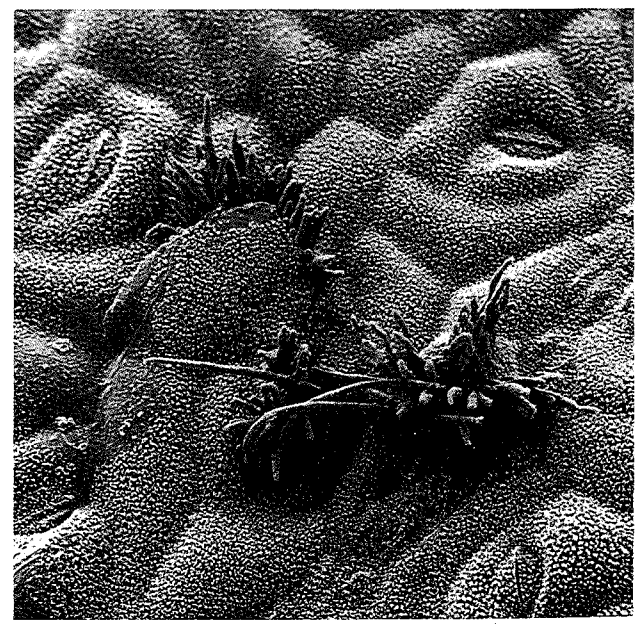

Fig. 24. Conidiophores of *Cercospora arachidicola*, the cause of early leaf spot, erupting from the surface of a peanut leaf. (Courtesy N. Kokalis-Burelle)

Late leaf spot. The anamorph *Cercosporidium personatum* (Berk. & M. A. Curtis) Deighton is known by several other names in the literature. The most recent is *Phaeoisariopsis personata*, proposed by von Arx in 1983 on the basis of small synnemata or long conidiophores and less thickened and darkened, bulging scars. This name change has not been widely accepted, and *Cercosporidium personatum* is preferred. The anamorph as commonly observed on late leaf spot lesions is amphigenous with fruiting on both sides of the leaflet, but sporulation is more common on the lower surface. Dense, pseudoparenchymatous stromata are up to 130 µm in diameter (Fig. 26). Conidiophores (10–100 × 3.0–6.5 µm) are numerous, pale to olivaceous brown, smooth, and one to three geniculate and have conspicuous conidial scars 2–3 µm wide. Conidia (20–70 × 4–9 µm) are medium olivaceous, cylindrical, obclavate, usually straight or only slightly curved, rounded at the apex, and not constricted; they have one to nine (mostly three or four) septa, a wall that is usually finely roughened, and a base shortly tapered with a conspicuous hilum (Figs. 25 and 27). Conidiophores commonly form dense fascicles in concentric rings. The teleomorph, *Mycosphaerella berkeleyi* Jenk., like that of the early leaf spot pathogen, is rarely observed on peanut.

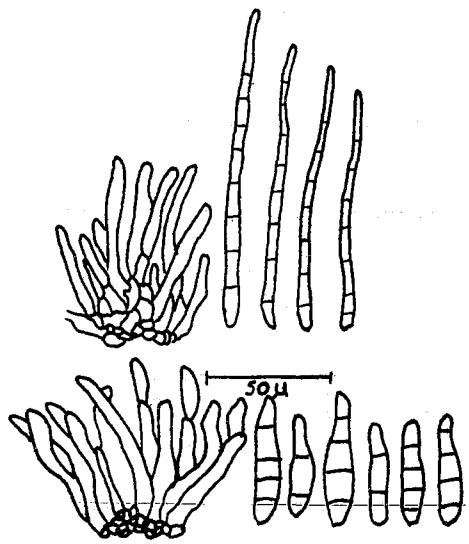

Fig. 25. Conidiophores and conidia of *Cercospora arachidicola* (top) and *Cercosporidium personatum* (bottom). (Reprinted, by permission, from Subrahmanyam et al., 1982)

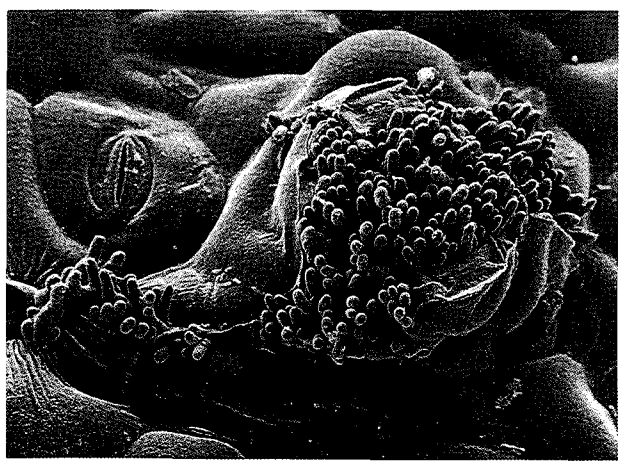

Fig. 26. Conidiophores of *Cercosporidium personatum*, the cause of late leaf spot, erupting from the surface of a peanut leaf. (Courtesy N. Kokalis-Burelle)

Disease Cycle

Disease cycles for early and late leaf spots are presented in Figure 28. Conidia produced on crop residue in the soil are the main source of initial inoculum. However, ascospores, chlamydospores, and mycelial fragments are potential inoculum sources. Mycelium in lesions on stems, petioles, and pegs is more likely to overseason than that on leaflets and is therefore a probable means of survival of the leaf spot pathogens from one season to the next.

Release of conidia of *C. arachidicola* is favored by temperatures of 20–24°C when relative humidity is greater than 90%. Spore production is favored by long periods of leaf wetness, and epidemics are favored by temperatures greater than 19°C and relative humidity that exceeds 95% for extended periods. Conidia germinate, forming one to several germ tubes, which enter open stomata or penetrate the epidermal cells directly. Under favorable conditions, lesions may develop within 6–8 days. *Cercosporidium personatum* produces intercellular, botryose haustoria, but *C. arachidicola* does not produce haustoria. Maximum late leaf spot infection occurs when temperatures are about 20°C and relative humidity exceeds 93% for more than 12 hr or with continuous leaf wetness periods of 10 hr. Few infections occur if temperatures are above 28°C, if relative humidity is high for less than 12 hr, or if leaf wetness periods are less than 10 hr. Late leaf spot lesions occur about 10–14 days after infection. Although late leaf spot usually has a longer incubation period than early leaf spot, it may cause more severe damage over a shorter period of time because of the capacity of *Cercosporidium personatum* to produce more spores per lesion than *C. arachidicola*.

Conidia are dispersed by wind, splashing water, and insects. Peak dispersal periods for conidia occur at dew dry-off in the morning and at the onset of rainfall. Although vertical dispersal of *C. arachidicola* conidia to 2.7 m above the soil surface has been reported, long-distance dispersal of *C. arachidicola* and *Cercosporidium personatum* conidia has not been well documented.

Management

Two management strategies are employed to reduce the threat of leaf spot epidemics. The first is to reduce initial inoculum by crop rotation and burial of peanut crop residue with a moldboard plow. Crop rotation for 2–3 years out of peanut is preferred, and this alone may provide as much as a 2- to 3-week delay in development of a leaf spot epidemic (Fig. 29). Volunteer peanut plants in the nonhost crop should be destroyed to prevent inoculum buildup and carryover. Because of the potential for a rapid rate of increase of leaf spot diseases, crop rotation alone is insufficient for their control.

Fig. 27. Conidia of *Cercosporidium personatum* germinating on the surface of a peanut leaf. (Courtesy N. Kokalis-Burelle)

The primary management strategy involves the use of tactics that reduce the rate of spread of leaf spot diseases. Multiple applications of fungicidal sprays (Plate 26) are usually required to keep leaf spot diseases below damaging levels. Benomyl, chlorothalonil, copper hydroxide, mancozeb, sulfur, propiconazole, and tebuconazole are examples of fungicides that either have been used or are being used for management of early and late leaf spots.

Fungicides are applied with tractor-mounted boom sprayers, fixed-wing aircraft, helicopters, sprinkler irrigation systems,

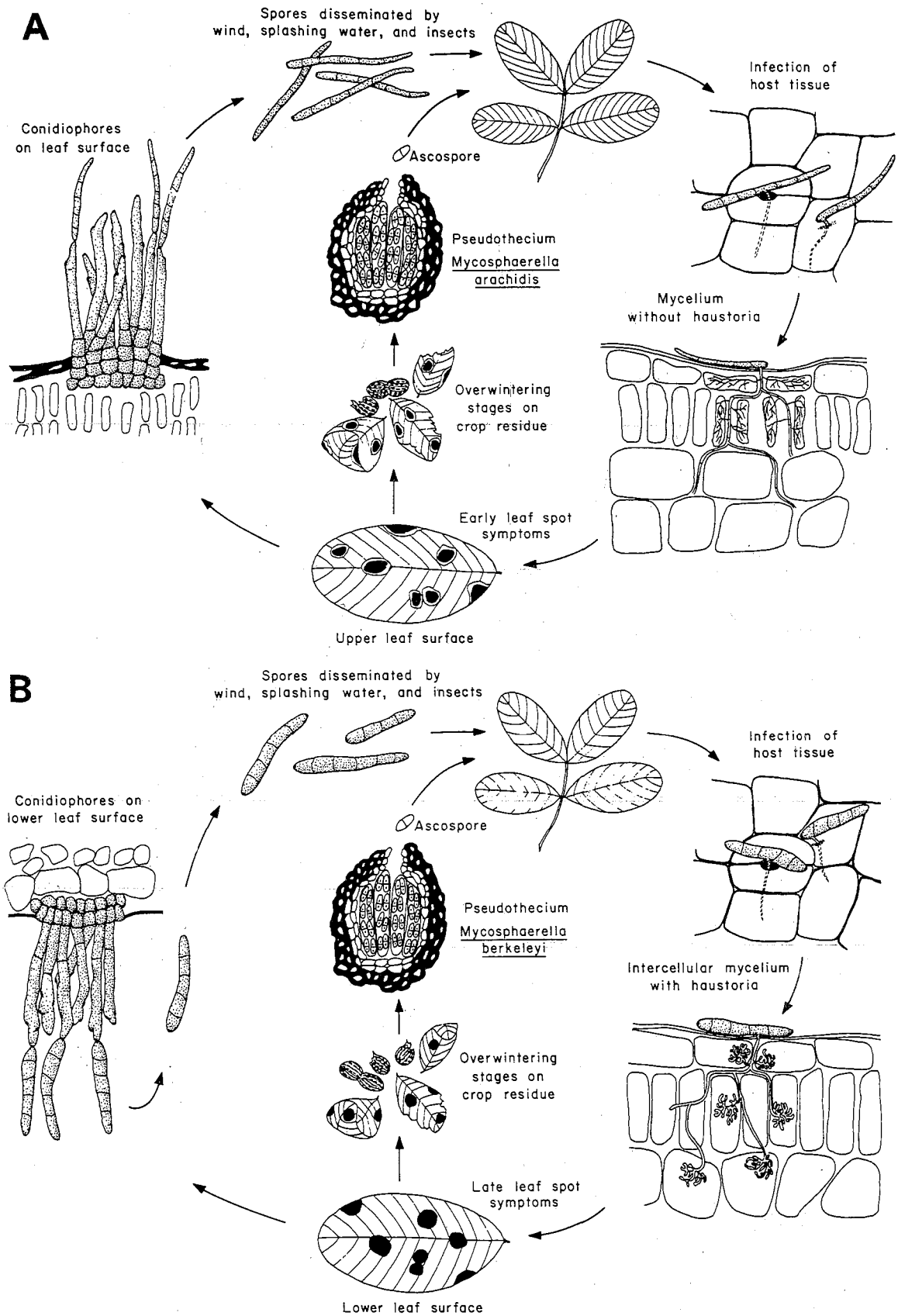

Fig. 28. Disease cycles of **A,** early leaf spot, caused by *Cercospora arachidicola,* and **B,** late leaf spot, caused by *Cercosporidium personatum.* (Prepared by Nancy Browning)

and underslung spray booms on pivot irrigation systems. Generally, application volumes of 5 gallons per acre are recommended for aircraft and 15 or more gallons per acre for ground sprayers. Chemigation is the least effective method because less fungicide is deposited on foliage than with other methods.

Disease-forecasting systems based on temperature and relative humidity, temperature and leaf wetness, or simply the number of rain events and rainfall probabilities are in use in some peanut-production areas. Requirements of the systems vary from the use of an in-field, computer-controlled weather station to a simple rain gauge. Scheduling fungicide applications with a forecasting system allows applications on an as-needed basis when environmental conditions are favorable for rapid disease development. Such methods are in contrast to the traditional application of fungicides on a calendar schedule, beginning at 30–40 days after planting and continuing at 10- to 14-day intervals until 14–21 days before the anticipated date of harvest.

Indiscriminate application of fungicides for control of early and late leaf spots may result in undesirable effects. For example, benomyl-tolerant strains of *C. arachidicola* and *Cercosporidium personatum* developed in the southeastern United States within 3 years after benomyl was registered for use in the United States. The use of chlorothalonil for control of foliar diseases may also increase the severity of Sclerotinia blight where that disease is a problem. In addition, some fungicides suppress development of twospotted spider mites, while other fungicides contribute to increased populations.

New fungicides with systemic activity have been developed. In particular, the ergosterol-biosynthesis-inhibiting (EBI) fungicides work well against leaf spot diseases. Propiconazole has good efficacy against early leaf spot, a little less against late leaf spot, very little against stem rot (*Sclerotium rolfsii*), and none against peanut rust (*Puccinia arachidis*). Therefore, propiconazole is not recommended late in the season where late leaf spot and/or rust is prevalent. In contrast, tebuconazole has good efficacy against this entire spectrum of diseases. It is possible to substitute an EBI for a protective fungicide for several applications during the season and thereby control a broader spectrum of diseases or to use lower rates of the protective fungicide mixed with an EBI full-season to attain control of foliar and some soilborne diseases of peanut. Systemic EBI fungicides are not recommended for full-season use alone because they may increase the risk that resistant populations of the leaf spot pathogens will develop.

Partially resistant cultivars may also be used to reduce the rate of spread of leaf spot epidemics. A few high-yielding cultivars with moderate resistance to early and/or late leaf spots and desirable agronomic traits have been developed, but this resistance is not complete. Components of resistance to leaf spot diseases that may delay disease progress under field conditions include extended latent period, decreased sporulation, smaller lesions, reduced infection frequency, reduced necrotic area of leaves, reduced defoliation, and fewer lesions on stems.

The best disease-management strategy for leaf spot diseases should integrate several of the above tactics into a program adapted to the cultivars and cultural practices of a given area.

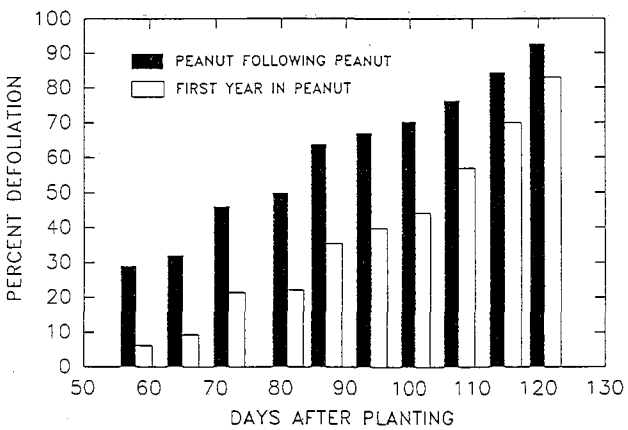

Fig. 29. Effect of crop rotation on defoliation caused by late leaf spot. Note that when peanut follows peanut, defoliation reaches damaging levels (about 30%) 3–4 weeks sooner than when peanut is in the first year or when it follows several years of nonhost crops.

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(Prepared by F. M. Shokes and A. K. Culbreath)

Fusarium Diseases

Fusarium spp. are ubiquitous in the soil of peanut-growing areas. They are commonly isolated from roots (even those of nonsusceptible plants), seed, and the hypocotyls and cotyledons of germinating seed. *Fusarium* spp. affect peanut plants sporadically; and until recently, *Fusarium*-related epidemics were rare, except for pod rots, in which *Fusarium* spp. are part of the complex of causal pathogens. *Fusarium* spp. of soilborne origin frequently penetrate the pod and colonize the seed coat.

Symptoms

In seedlings, preemergence damping-off caused by *Fusarium* spp. results in the young plant roots and hypocotyls becoming gray, water soaked, and frequently overrun by mycelium. Chlamydospores of *F. oxysporum* can germinate in the rhizosphere of young roots.

An infected seedling is stunted, the taproot becomes brown, and the hypocotyl is quickly invaded. In an older seedling, up to the age of about 1 month, dry rot of the taproot, caused by *F. solani*, may occur and spread to secondary roots.

A syndrome of Fusarium root rot and slow wilting may appear in adult plants. Symptoms include the folding of leaflets against each other during dry periods, chlorosis of leaflets, and slight wilting. At this stage, elongate, slightly sunken, brown lesions are found on the taproot near the crown. As the lesions enlarge to girdle the taproot, wilting progresses. Leaves turn brown and die gradually. In moist soil, plants may form adventitious roots around the crown early enough to escape terminal wilting, but plant development and pod yield are impaired. In

the advanced stage of the disease, all tissues near the lesions are invaded by *F. solani*. Hyphae of this fungus are sometimes observed in vessels of the root, even at the earliest phase of disease expression. At this stage, no other organisms can be isolated, but some appear later. Thus, *F. solani* has been implicated in the cause of the disease, although inoculation tests have been inconsistent in verifying the pathogenicity of this fungus.

Fusarium wilt appears sporadically in peanut fields. During sudden wilting (Plate 27), leaves of an entire plant turn grayish green, and during dry weather, the canopy becomes dry, brittle, and bleached. If the wilt is less sudden, leaves turn yellow, and sometimes plants are defoliated before death. In both cases, taproots show vascular browning; however, secondary roots and rootlets appear healthy. These symptoms, in particular the sudden wilting, implicate *F. oxysporum* in the peanut wilt complex.

Peg lesions usually develop on the underground part of the peg, mainly near the soil line, where the high soil temperatures that often prevail can injure the epidermal cells of the pegs and predispose them to soil-inhabiting fungi. Wound parasites, including some *Fusarium* spp., can be isolated from the lesions, which may girdle and rot the peg. As a result, the peg tissues weaken and pods separate from the plant at harvest and are lost.

Pod diseases may take the form of pod blight, pod rots, or external blemishes. Pod blight affects primordial, (i.e., very young) pods. *F. solani* and *F. scirpi* are two of a series of fungi that have been isolated from blighted pods soon after peg penetration of the soil. Both species are pathogenic to young pods. Pod blight is considered a cause of death of many primordial pods that die early in their development.

Maturing pods affected by a dry rot attributed to various *Fusarium* spp. may show grayish, pinkish, or whitish violet coloration (Plate 28). However, these visible symptoms are not proof of the presence of *Fusarium* spp., nor are they proof that a *Fusarium* sp. is the major causal organism. In Libya, *F. solani* and *F. scirpi* have been reported as the causes of a similar pod rot.

External blemishes of the shell and seed coat on whole, mature pods may be caused by a variety of fungi, including *F. solani* and *F. oxysporum*. Under controlled conditions, only *F. oxysporum* incited this symptom.

A common and devastating pod rot, commonly referred to as peanut pod rot complex, is thought to be caused by several pathogens including *Rhizoctonia* spp. (see Peanut Pod Rot Complex) and the *Pythium* disease complex (see *Pythium* Diseases). *F. solani* plays a role in the development of this rot, both as a predisposing factor and as one of the saprophytic microflora that aggravate the final breakdown of the pod. *F. solani* alone is unable to cause an epidemic. Pods affected by the pod rot complex typically have coalescing (confluent) chocolate brown dots and spots.

Causal Organisms

About 17 species and varieties of *Fusarium* have been isolated from the soil around peanut roots or pods and from the root, collar, pegs, and pods, including shells and seed. Of these, only members or close relatives of the following species have been reported as responsible for peanut diseases: 1) *F. solani* (Mart.) Sacc., in particular *F solani* f. sp. *phaseoli* (Burkholder) W. C. Snyder & H. N. Hans.; 2) *F. oxysporum* Schlechtend.:Fr.; 3) *F. equiseti* (Corda) Sacc. and *F. scirpi* Lambotta & Fautrey; 4) *F. tricinctum* (Corda) Sacc.; 5) *F. moniliforme* J. Sheld.; and 6) *F. avenaceum* (Fr.:Fr.) Sacc.

All *Fusarium* spp. produce microconidia, macroconidia, and chlamydospores. Chlamydospores (7–11 µm in diameter) are borne singly or in chains (Fig. 30). Microconidia (5–12 × 2–3.5 µm) are hyaline, nonseptate, and oval to ellipsoid or cylindrical (Fig. 31). Macroconidia (Figs. 31–33) are hyaline with three to five septa and pointed ends. They are produced on phialides arranged sparsely or in sporodochia.

Disease Cycle

Fusarium spp. live saprophytically in soil and reproduce on plant debris. Conidia are abundant but short lived. Chlamydospores are the persistent survival structures. Symptomless carriers actively maintain *Fusarium* spp. during seasons when nonhost crops are planted. Seedborne inoculum and hyphae sheltered in slowly disintegrating debris also carry the pathogen over from one season to another. Seedborne inoculum is frequent, even on the seed coats of sound seed from sound pods.

Injury to seed predisposes seedlings to *Fusarium* spp. that are in soil and carried on the seed coat. Epidemics of seedling damping-off, and the resulting poor stands, occur when soil temperatures are too low for rapid emergence. If in addition soils are wet and insufficiently drained, seed may decay at the beginning of germination.

Injury to roots predisposes seedlings to infection by *Fusarium* spp. Slow wilting most seriously affects young plants during hot, dry weather. Without water stress, damage levels are low.

Fusarium spp. are facultative xerophytes that can grow actively under dry conditions. Nevertheless, moist soil enhances

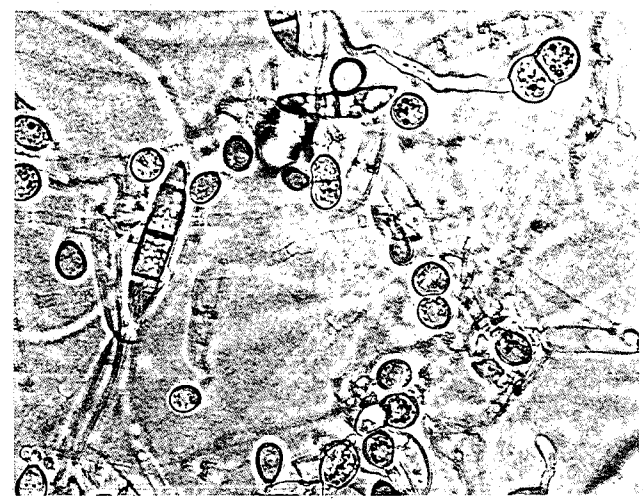

Fig. 30. Chlamydospores of *Fusarium solani*. (Courtesy Fusarium Research Center, Department of Plant Pathology, The Pennsylvania State University, University Park)

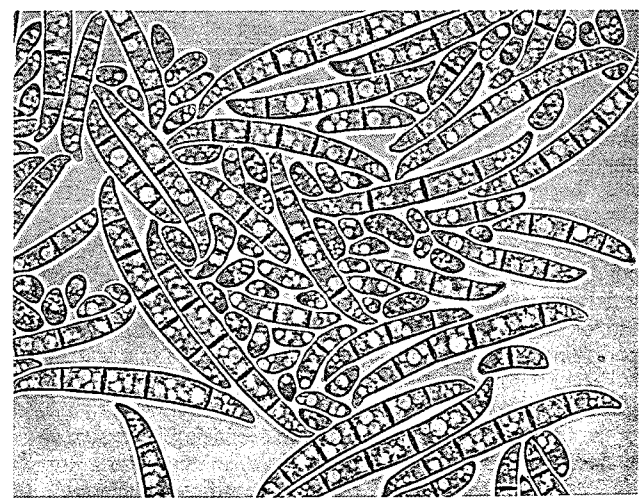

Fig. 31. Macroconidia and microconidia of *Fusarium solani*. (Courtesy Fusarium Research Center, Department of Plant Pathology, The Pennsylvania State University, University Park)

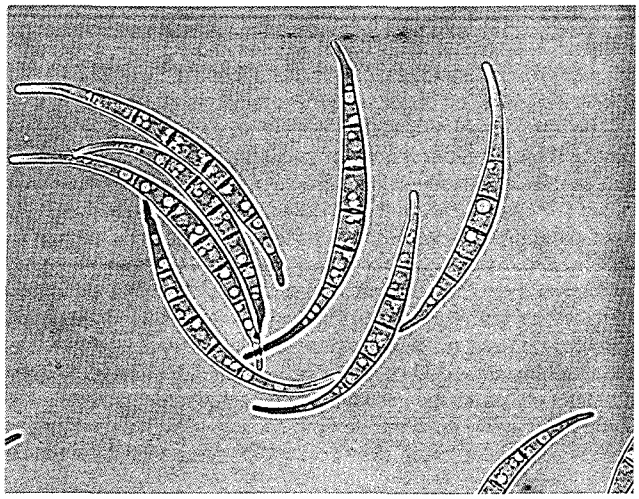

Fig. 32. Macroconida of *Fusarium roseum*. (Courtesy Fusarium Research Center, Department of Plant Pathology, The Pennsylvania State University, University Park)

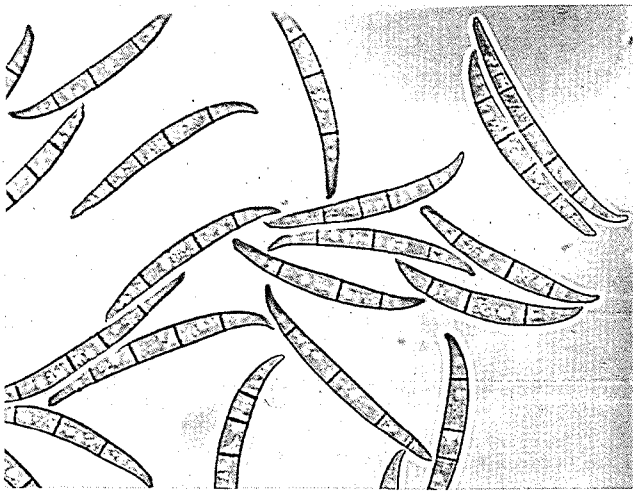

Fig. 33. Macroconidia of *Fusarium oxysporum*. (Courtesy Fusarium Research Center, Department of Plant Pathology, The Pennsylvania State University, University Park)

pod rot, possibly because of poor soil aeration (the shortage of oxygen predisposes the pod to pathogens, including *Fusarium* spp.).

Under continuous peanut cultivation, the population densities of *Fusarium* spp. in the soil increase considerably. This abundance in the soil is rarely accompanied by a disease epidemic, with the exception of pod rots.

Control

Fusarium spp. have been isolated from hypocotyls and cotyledons of fungicide-treated seed. Seed dressings are not highly efficient against *Fusarium* spp., but they significantly reduce damping-off diseases because of their effect on other pathogens.

Crop rotation may retard a quick buildup of *Fusarium* propagules; nevertheless, it does not efficiently reduce populations of the fungus enough to avert *Fusarium*-incited diseases.

Avoiding peanut cultivation in poor, acid soils and improving general soil fertility by organic amendments (manure) may reduce incidence of *Fusarium*-incited root rot.

Cultivation in well-drained soil will minimize pod rots. Overhead irrigation permits better control of irrigation intensity and duration and of subsequent drainage and drying of the topsoil than do other irrigation methods. Infrequent irrigation with adequate amounts of water are preferable to frequent irrigation with smaller amounts because the former regime allows the topsoil, which contains the pods, to dry.

Soil treatments with sublethal doses of the biocide metham sodium and solarization (solar pasteurization beneath transparent tarps) selectively reduce soil fungal populations. These treatments reduce *Fusarium* populations and increase total pod yield, apparently by reducing the incidence of peg blight and pod rot.

Breeding for resistance to peanut diseases caused by *Fusarium* spp. has not been systematically pursued. However, the rarity of epidemics caused by *Fusarium* spp. in peanut suggests that in field nurseries, *Fusarium*-diseased plants either die or are discarded; thus, lines selected on the basis of other characteristics have incidentally been given field resistance to several *Fusarium* spp.

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(Prepared by Z. R. Frank and Y. Ben-Yephet)

Melanosis

Melanosis of peanut leaves has been reported in Argentina and is referred to as Stemphylium leaf spot in the United States.

Symptoms

Lesions are dark brown, 0.5–1.0 mm in diameter, and usually circular. Solitary or confluent, they appear only on the abaxial leaflet surface and are at first slightly submerged but later become raised and crustlike.

Causal Organism

The causal organism, *Stemphylium botryosum* Wallr., produces spores that are terminal, solitary, muriform, pale to dark brown to olivaceous, and sometimes minutely verruculose or echinulate. They are borne on conidiophores with terminal swellings, which become, through percurrent proliferation, intercalary.

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(Prepared by R. A. Taber and D. H. Smith)

Myrothecium Leaf Blight

A leaf blight of peanut caused by *Myrothecium roridum* has been observed in India and Thailand. *M. gramineum* has also been reported as a leaf blight pathogen in India. Both pathogens infect a wide range of host plants.

Symptoms

The two pathogens produce similar symptoms on infected peanut leaves. Lesions are round to irregular, 5–10 mm in diameter, with tan centers and brown margins surrounded by chlorotic halos. The centers of these lesions become thin, papery, and light tan. Lesions coalesce to give affected leaves a blighted appearance. Abundant olive green to black fruiting bodies, often arranged in circular rings, are formed on necrotic areas of both leaf surfaces (Plate 29).

Causal Organisms

The conidia of *M. roridum* Tode:Fr. are hyaline, one celled, elongated, and 4.7–11.7 × 1.2–3.5 µm. Conidia of *M. gramineum* Lib. are 5.5–14.0 × 3.0–5.0 µm with stiff, acute setae mixed with smaller, torsive ones.

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Mahrshi, R. P. 1983. *Myrothecium gramineum*—A new report on groundnut from Rajasthan. Indian Phytopathol. 36:728.

(Prepared by P. Subrahmanyam)

Neocosmospora Foot Rot

Foot rot has been observed in peanuts in Taiwan and South Africa. The causal organism, *Neocosmospora vasinfecta* E. F. Sm., has been observed colonizing aboveground plant parts and is also pathogenic to pod hulls and seed. Pods exhibit discolored internal tissues and later decompose. Diseased plants are stunted with yellow lower leaves and frequently defoliate and senesce prematurely. There are no control measures available.

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(Prepared by D. M. Porter)

Olpidium Root Discoloration

Root discoloration of peanut caused by *Olpidium brassicae* has been reported in the Indian states of Andhra Pradesh, Gujarat, and Punjab and in Texas in the United States.

Symptoms

Lightly infected roots remain apparently healthy, but when infection is advanced, the root cortex becomes brown to black. The pathogen is restricted to the peripheral layers of the cortex of infected roots.

Causal Organism

Plasmodia of *O. brassicae* (Woronin) P. A. Dang. are thin walled, cylindrical to rounded, and 10–22 × 15–45 µm and have densely granulated protoplasm. Zoosporangia are variable in size (8–32 µm in diameter) with a single exit tube. Resting spores are spherical (10–27 µm in diameter) and consist of thick, stellate exospores and thin, smooth endospores (Fig. 34). Zoospores are spherical and have a single posterior, whiplash flagellum.

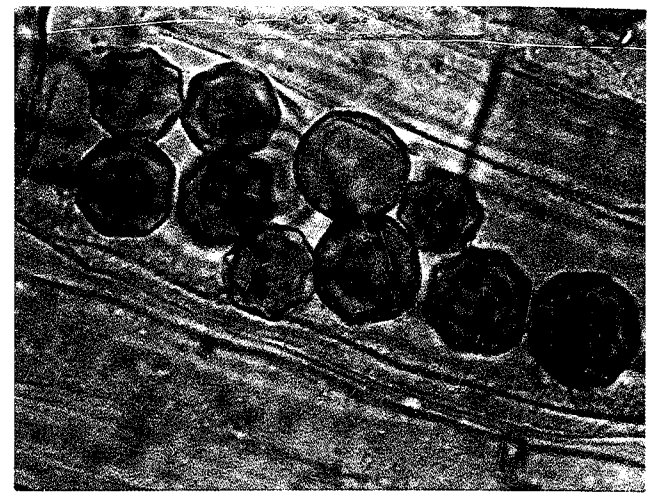

Fig. 34. Endospores of *Olpidium brassicae* with typical stellate wall surfaces.

O. brassicae is widely distributed, particularly in temperate regions. It is parasitic on roots of several phanerogams and is a vector of several soilborne plant viruses.

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Subrahmanyam, P., and McDonald, D. 1980. Occurrence of *Olpidium brassicae* on groundnut. Trans. Br. Mycol. Soc. 75:506-509.

(Prepared by P. Subrahmanyam)

Peanut Pod Rot Complex

Peanut pod rot (pod breakdown) is a sporadic but common disease of peanut that causes serious losses throughout all peanut-growing regions of the world. In 1954, the condition was described in Georgia as black pod, and it has been prevalent in Israel since 1959. In 1964, a preharvest pod (fruit) rot of peanut in Virginia was described and referred to as pod breakdown. Others have since referred to what appears to be the same malady as the peanut pod rot complex. Losses are variable and appear to be related to cultivar, the pathogen involved, and nutrition.

Symptoms

Symptoms of the pod rot disease complex vary depending on the location, season, and pathogens involved. Deterioration or rot of fully developed pods is the first sign of disease. Pods develop either a tan to brown, dry decay or a greasy, black, wet decay, depending on the pathogens and environmental conditions (Plate 30). Many pods, both sound and rotted, may remain in the soil after digging, the result of weakened or decayed pegs.

There are no aboveground symptoms of pod rot, except that severely affected plants may be darker green and exhibit prolonged flowering. The root system generally is not infected, and the reduced demand for carbohydrate from the loss of the fruit usually increases the vigor of the foliage. Plants with the greatest degree of pod rot at or near harvest will appear to be the most vigorous and provide no indication of serious disease losses below the soil surface.

Causal Organisms

This overview of the peanut pod rot disease complex focuses on *Pythium myriotylum* Drechs., *Rhizoctonia solani* Kühn, and *Fusarium solani* (Mart.) Sacc., since there is considerable evidence indicating that the complex involves these three fungal species. In Libya, pod rot is caused mainly by *F. solani* and other *Fusarium* spp. *Fusarium* spp. have been implicated in Pythium-Rhizoctonia pod rot in the United States. In Virginia, *Fusarium* spp. have been reported to precede *P. myriotylum*. A three-step progression of the pod rot complex has been described in Israel. In step 1, *F. solani* predisposes pods to sporadic infection by *P. myriotylum*. Step 2 involves pod colonization by *P. myriotylum* and a rapid increase in pod rot. In step 3, *F. solani* and saprophytic organisms cause the disintegration of pods and there is a sharp reduction or disappearance of *P. myriotylum*.

Soil fauna often plays an important role in the pod rot complex. The feeding of insects and nematodes impacts the severity of pod rot caused by *P. myriotylum*, *R. solani*, and *F. solani*. In studies conducted in Texas, pod damage was influenced by the feeding of insect larvae. In Virginia, pods injured by larvae of the southern corn rootworm (*Diabrotica undecimpunctata howardi* Barber) were more susceptible than uninjured pods to infection by various fungi. In Florida, pods exposed to moderate, combined inoculum levels of the peanut root-knot nematode (*Meloidogyne arenaria* (Neal) Chitwood) and *P. myriotylum* sustained 31% more decay than those exposed to *P. myriotylum* alone. In North Carolina, soil mites (*Caloglyphus* spp.) were associated with more than 50% of decaying pods where *P. myriotylum* was the primary fungal pathogen.

Nutrition

The severity of pod rot associated with the pod rot complex appears to be related to the presence of a specific pathogen or multiple pathogens and a wide range of environmental factors, such as the presence or absence of a specific plant nutrient. The first symptoms of peanut pod rot complex include a collapse of pod tissues, which are usually attacked by various soil fungi causing the characteristic dark discoloration of the pod. The pod rot complex appears to be more common on large-seeded virginia peanuts than on the smaller-seeded runner and spanish types. Large amounts (2 t/ha) of gypsum ($CaSO_4 \cdot 2H_2O$) applied to peanuts at bloom significantly reduces the severity of pod breakdown and maximizes pod yield. High rates of gypsum (1–3 t/ha) applied to Virginia Bunch 46-2 also reduced pod breakdown in 2 of 3 years and increased pod yields and the percentage of sound, mature seed. Gypsum increases the amount of calcium and decreases the amount of potassium in pods. Applications of $MgSO_4$ (1.3 t/ha) and K_2SO_4 (1–2 t/ha) increase pod breakdown and the amount of potassium in pods but decrease or tend to decrease pod yield and calcium levels in pods. The vulnerability to pod-breakdown pathogens is reduced in pods containing 0.20% or more calcium. However, similar treatments of gypsum did not suppress pod rot in Oklahoma.

Studies have been conducted in Georgia to determine the effects of peanut cultivar and rate of gypsum on pod rot in soils naturally low and naturally high in calcium. Pod rot did not occur on any cultivar in any treatment in the high-calcium soil. In the low-calcium soil, severe pod rot occurred on plots receiving no gypsum, but the severity decreased for all cultivars as the rate of gypsum increased. Cultivars with high calcium requirements (e.g., Early Bunch) were more susceptible to pod rot than cultivars less dependent on calcium fertilization (e.g., Florunner).

An evaluation of pathogen-specific fungicides for the control of the pod rot complex pathogens failed to distinguish which pathogen was involved. Although *Pythium*, *Rhizoctonia*, and *Fusarium* spp. were isolated from soil and decaying pods and pegs throughout the growing season, no consistent differences were found among treatments for soil populations or isolation frequency from decaying pods. Plots treated with a calcium source were generally higher in pod yield and grade and exhibited a lower incidence of pod rot. There was a positive correlation between the concentrations of most elements in pods and pod rot, except in the case of calcium, for which concentration was negatively correlated with amount of pod rot.

The key factor in the reduction of peanut pod rot (pod breakdown) in the studies conducted in Virginia and Georgia was the increased concentration of calcium in the pods. In Texas, where high sodium levels exist in the water, pod rot of peanut can be reduced with applications of gypsum. Although water relations in the peanut pod rot system have not been studied extensively, it is well accepted that calcium must be in an aqueous form to be absorbed by the fruit. Since peanut pods develop in the soil, they could be very susceptible to colonization by microorganisms after predisposition by a nutrient imbalance or deficiency. Striking similarities between the peanut pod rot complex and blossom-end rot of tomato and pepper are evident.

Control

Current recommendations for control of the pod rot complex in the eastern United States include high rates of gypsum applied at flowering time. Nutritional imbalances in the pod-development zone are discouraged. Large-seed peanuts tend to be more susceptible than small-seed cultivars. Pesticides such as pentachloronitrobenzene (PCNB) and metalaxyl, which have activity on *Rhizoctonia* and *Pythium* spp., respectively, are being used. Although chemicals such as PCNB and metalaxyl have sometimes been recommended for the management of pod rot, little or no economic gain has been demonstrated.

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Csinos, A. S., Gaines, T. P., and Walker, M. E. 1984. Involvement of nutrition and fungi in the peanut pod rot complex. Plant Dis. 68:61-65.

Filonow, A. B., Melouk, H. A., Martin, M., and Sherwood, J. 1988. Effect of calcium sulfate on pod rot of peanut. Plant Dis. 72:589-593.

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Porter, D. M., Smith, D. H., and Rodríguez-Kábana, R. 1982. Peanut plant diseases: Soilborne diseases. Pages 348-378 in: Peanut Science and Technology. H. E. Pattee and C. T. Young, eds. American Peanut Research and Education Society, Yoakum, TX.

(Prepared by A. S. Csinos and D. K. Bell)

Pepper Spot and Leaf Scorch

Pepper spot and leaf scorch have been reported in Angola, Argentina, Burkina Faso, India, Madagascar, Malawi, Maur-

itius, Mozambique, Niger, Senegal, Swaziland, Thailand, Taiwan, Uganda, the United States, Vietnam, Zambia, and Zimbabwe. The diseases do not usually contribute to pod yield losses.

Symptoms

Pepper spot lesions are dark brown or black (Plate 31), less than 1.0 mm in diameter, and irregular to circular and may be depressed. They form on the adaxial leaf surface and slowly enlarge.

Leaf scorch (Plate 32) usually appears at the leaflet apex, followed by the development of a wedge-shaped lesion with a vivid yellow zone adjacent to its advancing margin. Lesions caused by *Cercospora arachidicola* or *Cercosporidium personatum* are sometimes observed in necrotic areas of tissue. Therefore, it appears that *Leptosphaerulina crassiasca* is well adapted to secondary colonization of peanut leaf tissue.

Causal Organism

L. crassiasca (Sechet) C. R. Jackson & D. K. Bell is the causal pathogen of both conditions. Only the teleomorphic state of this homothallic fungus has been reported. Mycelial hyphae are septate with uninucleate cells that give rise to fertile hyphae with binucleate or multinucleate cells. Asci contain eight ascospores, which are initially uninucleate. Mature ascospores are multinucleate with three to five transverse septa and zero to two longitudinal septa. The ascoma is a uniloculate pseudothecium with a papillate, ostiolar neck. Asci are bitunicate, and ascoma ontogeny is typical of the *Dothidea* type of development.

Pseudothecia (Fig. 35) contain eight to 20 asci, each 50–80 × 25–55 µm. Ascospores (23–40 × 11–17 µm) are oblong to ellipsoidal, hyaline, and pale yellow to light brown at maturity (Figs. 36 and 37).

Disease Cycle

Pseudothecia form abundantly in necrotic leaf tissue. Peak dispersal periods of forcibly ejected ascospores occur at the end of the dew period and at the onset of rainfall. Germ tubes form appressoria with subsequent direct penetration of epidermal cells.

Control

Leaf scorch and pepper spot are controlled quite effectively with fungicides such as chlorothalonil.

Selected References

Graham, J. H., and Luttrell, E. S. 1961. Species of *Leptosphaerulina* on forage plants. Phytopathology 51:680-693.

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Pettit, R. E., Taber, R. A., and Harrison, A. L. 1968. *Leptosphaerulina-Cercospora* complex on peanuts in Texas. (Abstr.) Phytopathology 58:1063.

Porter, D. M., Garren, K. H., Mozingo, R. W., and van Schaik, P. H. 1971. Susceptibility of peanuts to *Leptosphaerulina crassiasca* under field conditions. Plant Dis. Rep. 55:530-532.

Subrahmanyam, P., and Smith, D. H. 1991. Variability in pathogenicity and symptomatology of *Leptosphaerulina crassiasca* on peanut. Plant Dis. 75:1266-1269.

(Prepared by D. H. Smith and R. A. Taber)

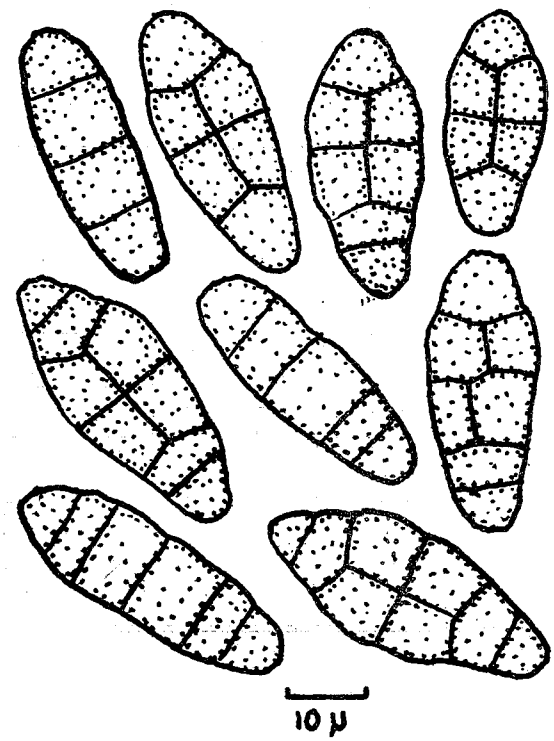

Fig. 36. Ascospores of *Leptosphaerulina crassiasca*. (Reprinted from Graham and Luttrell, 1961)

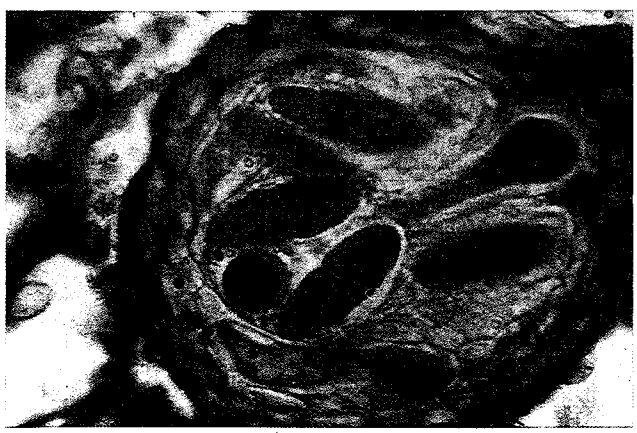

Fig. 35. Pseudothecia of *Leptosphaerulina crassiasca*.

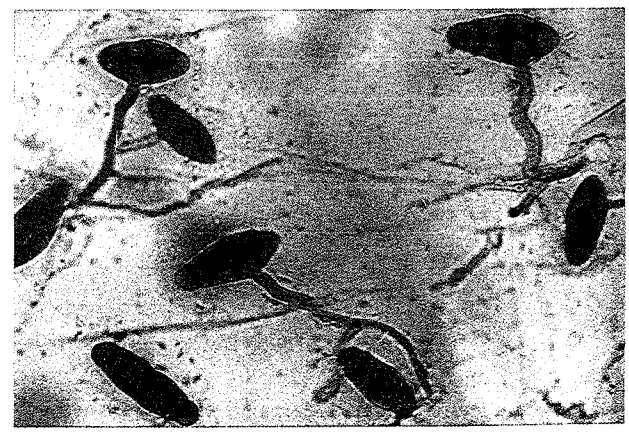

Fig. 37. Germinating ascospores of *Leptosphaerulina crassiasca*.

Pestalotiopsis Leaf Spot

Pestalotiopsis leaf spot has been observed on peanut leaves in India.

Symptoms

Lesions are dark brown and circular. They are surrounded by yellow halos and are commonly restricted to either side of the midrib. Acervuli containing the dark conidia form near the center of diseased tissue.

Causal Organisms

Two species, *Pestalotiopsis arachidis* Satya and *P. neglecta* Thuem., have been reported in India. The distinction between these species and the genus description are incomplete. Conidia have four septa and are fusiform, tapering to the base, with hyaline appendages.

Selected Reference

Gupta, D. K. 1988. New host records from India. Indian Phytopathol. 41:506.

(Prepared by R. A. Taber and D. H. Smith)

Phanerochaete omnivora

A yellow orange, resupinate, hydnaceous basidiomycete has been observed on peanut plants and soil in several counties in Texas. Basidiocarps were commonly effused and poorly developed in patches; however, weather conditions during the 1989 growing season were favorable for extensive development of the fungus. It sporulated profusely on plants diagnosed to be infected with cotton root rot as well as plants not exhibiting symptoms of this disease. It was identified as *Phanerochaete omnivora* (Shear) Burdsall & Nakasone, a fungus that is saprophytic on wood in the forests and desert areas of the southwest and is associated with a white rot of hardwoods. This is the first report of its being associated with an herbaceous plant. Although the orange teeth and rhizomorphs of the south Texas isolates resemble those of *P. chrysorhiza* (Torr.) Budington & R. L. Gilbertson, an eastern species, the micromorphology (including basidiospore size and ability to grow at 36°C) is characteristic of *P. omnivora.* Rhizomorphs of the fungus extend down into the soil for several feet. The fungus appears to be superficial on the surface of the plant, although pathogenicity studies are incomplete.

Selected References

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Taber, R. A., Smith, D. H., Burdsall, H. H., Jr., Black, M. C., and Crumley, C. R. 1990. Association of *Phanerochaete* with peanut in Texas. (Abstr.) Phytopathology 80:438.

(Prepared by R. A. Taber)

Phomopsis Blight

Phomopsis leaf and stem diseases have been reported in the United States, and a leaf scorch caused by a *Phomopsis* sp. has been reported in Argentina.

Symptoms

Parallel rows of pycnidia develop on dead peanut stems infected with *Phomopsis* sp. The stems become blackened. A scorch symptom similar to that of Leptosphaerulina leaf scorch is often observed on the leaflets. *Phomopsis* spp. are often isolated from lesions caused by other fungi such as *Colletotrichum* sp., *Cercospora arachidicola,* and *Cercosporidium personata.*

Causal Organism

Phomopsis sojae Lehman is the causal organism. Conidiomata are eustromatic and separate or aggregated to confluent and have short, ostiolate beaks. Two forms of conidia are produced. Alpha conidia (4.5–9.8 × 1.1–3.9 µm) are one celled, hyaline, rounded at both ends, and usually biguttulate. Beta conidia (9–27 × 0.8–1.8 µm) are long, filiform, nonseptate, and straight or more often hamate.

Selected References

Frezzi, M. J. 1955. "Quemadura" de las hojas causada por *Leptosphaerulina arachidicola* y otros hongos, en manies silvestres (grupo rizomatoso) de distinta procedencia. Rev. Invest. Agropecu. Ser. 5. 2:13-24.

Jackson, C. R., and Bell, D. K. 1969. Diseases of peanut (groundnut) caused by fungi. Ga. Agric. Exp. Stn. Bull. 56.

(Prepared by R. A. Taber and D. H. Smith)

Phyllosticta Leaf Spot

Phyllosticta leaf spot occurs in many countries, including the United States, Pakistan, India, China, Niger, Burkina Faso, Thailand, and the Philippines. Occasionally, it is destructive in Argentina and Zimbabwe.

Symptoms

Lesions (0.5–5 mm) are usually circular and have reddish brown borders with light brown to tan centers (Plate 33). The light center often drops out with age, and then the lesion appears as a shot-hole.

Causal Organisms

Phyllosticta arachidis-hypogaea V. G. Rao forms scattered, dark brown pycnidia (Plate 34), which are spherical, thick walled, and 69.3–172.3 µm in diameter. Conidiospores (5.2–6.9 × 2.2–3.2 µm) are elongate, hyaline, and nonseptate.

P. sojaecola C. Massal. forms numerous pycnidia (105–175 µm) in concentric circles. Conidiospores (7.1–9.8 × 2.1–3.2 µm) are hyaline and ellipsoid or claviform.

Control

Fungicides applied for early and late leaf spot control often control Phyllosticta leaf spot.

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Jackson, C. R., and Bell, D. K. 1969. Diseases of peanut (groundnut) caused by fungi. Ga. Agric. Exp. Stn. Bull. 56.

Patil, A. S. 1987. Leaf spot of groundnut caused by *Phyllosticta arachidis-hypogaea* Rao in Maharashtra state, India. J. Maharashtra Agric. Univ. 12:205-209.

Subrahmanyam, P., Bosc, J., Hassane, H., Smith, D. H., Mounkaila, A., Ndunguru, B. J., and Sankara, P. 1992. Groundnut diseases in Niger and Burkina Faso. Oleagineux 47:119-133.

Zhang, M., and Wang, Y. X. 1991. Effect of agricultural antibiotic 120 against peanut Phoma blight (caused by *Phyllosticta* sp.). Chin. J. Biol. Control 7:175-176.

(Prepared by R. A. Taber and D. H. Smith)

Phymatotrichum Root Rot

Phymatotrichum root rot of peanut, which occurs in the calcareous soils of the southwestern United States and Mexico, is reported under the names Phymatotrichum root rot, cotton root rot, and Ozonium root rot. It is not widespread in the Southwest because peanut is not often cultivated in calcareous soils.

Symptoms

Wilted plants with attached leaves first occur in small, scattered patches. Patches enlarge, with newly wilted plants occurring at the leading edges of diseased areas. The taproots of affected plants are severely decayed. The surfaces of decayed roots are frequently covered with tan to light brown mycelial strands (rhizomorphs). Diseased areas may expand to up to an acre in size. Tan to buff-colored spore mats often appear on the soil surface at the edges of diseased areas after rain, but these are transient and dissipate after 2–3 days.

Causal Organism

Phymatotrichum omnivorum Duggar (syn. *Phymatotrichopsis omnivora* (Duggar) Hennebert) produces rhizomorphs on infected roots and conidia in spore mats. Rhizomorphs and acicular (cruciform) hyphae are useful for identification of this fungal pathogen (Plate 35). Globose, single-celled conidia are 45–55 µm in diameter (Plate 36). *Hydnum omnivorum* Shear and *Sistotrema brinkmannii* (Bres.) J. Eriksson are reported to be teleomorphs of this fungus. *P. omnivorum* sclerotia are present in soil at depths of 30–75 cm.

Disease Cycle

P omnivorum survives in soil as sclerotia for many years and is well adapted for survival in alkaline, poorly aerated, black clay soil. It survives also on weed hosts. However, it does not survive at temperatures below 0°C. Propagules of this pathogen can be disseminated with farm equipment.

Control

Partial disease management is possible with deep plowing and crop rotation with grain sorghum. Fungicides are not effective for management of Phymatotrichum root rot. Peanut and other susceptible crops such as alfalfa and cotton should not be planted in infested fields.

Selected Reference

Lyda, S. D. 1978. Ecology of *Phymatotrichum omnivorum.* Annu. Rev. Phytopathol. 16:193-209.

(Prepared by D. H. Smith)

Powdery Mildew

Powdery mildew (Fig. 38) has been observed on peanut in India, Israel, Mauritius, Portugal, and Tanzania. The causal pathogen, *Oidium arachidis* Chorin, is limited mainly to the adaxial leaf surface. Oidia are 31–44 × 13–15 µm. Conidiophores produce one to two oidia under drought stress conditions, but chains of three or four oidia develop under humid and calm conditions. Subspherical, pyriform haustoria form in epidermal cells. Rapid development of powdery mildew at 25°C has been reported in Israel.

Selected Reference

Chorin, M., and Frank, Z. 1966. *Oidium arachidis* Chorin, powdery mildew of groundnut foliage. Isr. J. Bot. 15:133-137.

(Prepared by D. H. Smith)

Pythium Diseases

Several species of *Pythium* may cause pod breakdown (pod rot), preemergence and postemergence damping-off, vascular wilt, and root rot diseases of peanut. All species are cosmopolitan in soils and attack a wide range of crop plants.

Pod breakdown (Plate 37), a term used to describe an in-soil rot of pods, is usually called pod rot. The disease is widespread in peanut-growing areas of the world, often causing economic losses. Such losses caused by *Pythium* spp. may be 0–80% but are difficult to define, since infection by these pathogens usually does not result in well-defined, aboveground symptoms. *P. myriotylum* is considered the major pod-rotting pathogen in North Carolina, Virginia, and other peanut-growing areas. However, several pathogens, including *Pythium* spp., *Rhizoctonia solani*, and *Fusarium solani*, can cause pod breakdown singly or in combination, i.e., in a pod breakdown complex. A complex involving *P. myriotylum* and *F. solani* is necessary for pod rotting to occur in Israel. Similar synergistic interactions also were noted with pathogens isolated from peanuts in Florida. In Israel, *Pythium* spp. are thought to precede *Fusarium* spp. in the pod rot complex, whereas in the United States, *Fusarium* spp. reportedly precede *Pythium* spp. A more complete discussion of the etiology of pod rot is found in the section Peanut Pod Rot Complex.

Environmental conditions are frequently important in determining the severity of preemergence and postemergence damping-off, vascular wilt, and root rot alone or in combination. *P. myriotylum* isolates vary widely in virulence as well as in differential pathogenicity to seedlings, roots, and pods of peanut cultivars. *P. myriotylum* is generally considered a warm-soil pathogen that is favored by constantly moist soil conditions; however, pod rot epidemics have been observed in soil drier than the wilting range for mesophytes.

Symptoms

In preemergence or early postemergence rot of peanut seedlings caused by *P. myriotylum*, the predominant symptoms include a black root rot, in which the roots are rapidly decayed, and the collapse of the top of the plant, generally without apparent invasion of the stem above the ground. The cotyledons and primary roots are often covered with a loose, white mycelial mat if moisture is available. Seedlings that emerge are stunted; leaves are light green and rapidly become necrotic. Root systems are water soaked and clumped. Cortical tissues

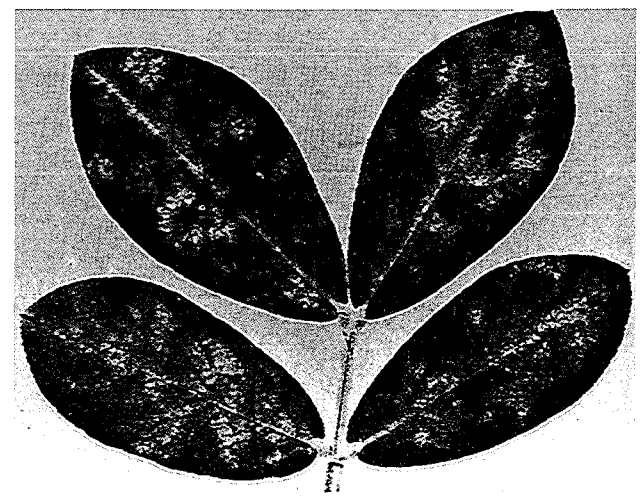

Fig. 38. Powdery mildew on an upper leaf surface. (Reprinted, by permission, from Chorin and Frank, 1966)

rapidly become brown and disintegrate, leaving a nonfunctional vascular skeleton.

Fibrous roots are especially susceptible to decay by *P. myriotylum*; however, all root tissues and nodules can be infected and ultimately turn dark brown to black during decay (Fig. 39). The total root system usually is greatly reduced by deterioration of lateral and branch roots. Cortical tissues disintegrate and slough off readily, leaving a fragmented, nonfunctional vascular system. Plants with root rot generally are stunted and readily wilt during moisture stress. Wilted plants may recover turgidity and outgrow the disease if conditions are conducive to growth.

Occasionally, peanut plants without obvious Pythium root rot symptoms wilt suddenly (Plate 38) and quickly die. Shortly after the appearance of wilt symptoms, leaflets become chlorotic or light green. Leaflet margins pucker, and adaxial curling or rolling occurs, starting at the apical end of the leaflet. Some leaflets eventually fold, turn brown gradually, and shed prematurely. Plants with these symptoms usually have some evidence of root deterioration. In addition, in advanced stages of infection, the vascular tissue of the taproot is dark brown from the tip to several centimeters into the stem. The discoloration extends into the primary and lateral branches of the stem and, in some cases, may be found in the vascular tissue of the petioles. Pods usually rot on plants that wilt early in the season but not on plants that wilt later.

Causal Organisms

Although *P. myriotylum* Drechs. is the dominant pathogen associated with peanut disorders caused by *Pythium*, other species, including *P. aphanidermatum* (Edson) Fitzp., *P. debaryanum* Auct. non R. Hesse, *P. irregulare* Buisman, and *P. ultimum* Trow, also are pathogenic to the peanut.

Pythium spp. are characterized by the presence of coenocytic mycelium, from which develop asexual reproductive structures (sporangia) that differ in size and shape among species (Fig. 40). The sporangia of *P. myriotylum* may be terminal or intercalary and may consist of simple or branched portions of mycelia. Sporangia germinate by producing either a germ tube or zoospores. Smooth-walled oogonia (average diameter 26.5 µm) are produced abundantly and borne most often on hyphal tips. Antheridia, usually three to six per oogonium, are declinous and crook necked. Oospores (12–37 µm in diameter) do not fill the oogonium. Each has a single reserve globule, a wall up to 2 µm thick, and pale golden contents.

Disease Cycle

Species of *Pythium* naturally inhabit the soil and can subsist indefinitely as saprophytes. *P myriotylum* has a wide host range, including grass crops used in rotation with peanut. The incidence of pod rot and the population density of *Pythium* spp. in soil, however, are significantly higher in fields in which peanut is grown successively than in fields in which crops are rotated.

Oospores are the primary survival structures of *P. myriotylum* in soil. Zoospores and sporangia are short lived. Mycelia of *P myriotylum*, produced by zoospores or germination of oospores, form appressoria and directly penetrate epidermal cells of peanut pods (Fig. 41). Penetration occurs in 2 hr at 30–34°C, but no penetration occurs below 25°C.

Pythium spp. are not restricted in their ability to infect peanut tissues but vary in pathogenic ability when infecting diverse tissues. Pathogenic ability is also variable within species. Infection of plant tissue by *Pythium* spp. is influenced by soil moisture, soil temperature, pH, cation composition, light, the presence of other organisms, and inoculum density.

The optimum temperature for mycelial growth of *P. myriotylum* is 35°C. In Florida, inoculum densities of 15–43 oospores per gram of soil resulted in 50% infection of peanut roots by *P. myriotylum*. In Oklahoma, preplant densities of three to 20 oospores per gram of soil were reported to have increased approximately 100-fold after 67 days; populations then declined to approximately 30 oospores per gram of soil at harvest time. Although seven cultivars tested in Oklahoma varied in their susceptibility to infection by *Pythium* spp., no effect was noted when cultivars and mean pathogen population were compared over a whole season. Use of the soil fungicide metalaxyl was reported to suppress *Pythium* population peaks at midseason, but disease incidence and final inoculum density were not significantly reduced at harvest.

A significant positive correlation between increasing soil moisture and incidence of Pythium pod rot has been demon-

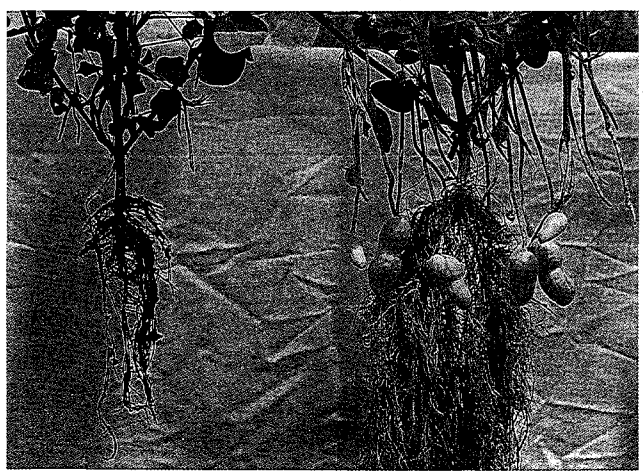

Fig. 39. Roots affected by *Pythium myriotylum* (left) and healthy roots (right). (Courtesy D. M. Porter)

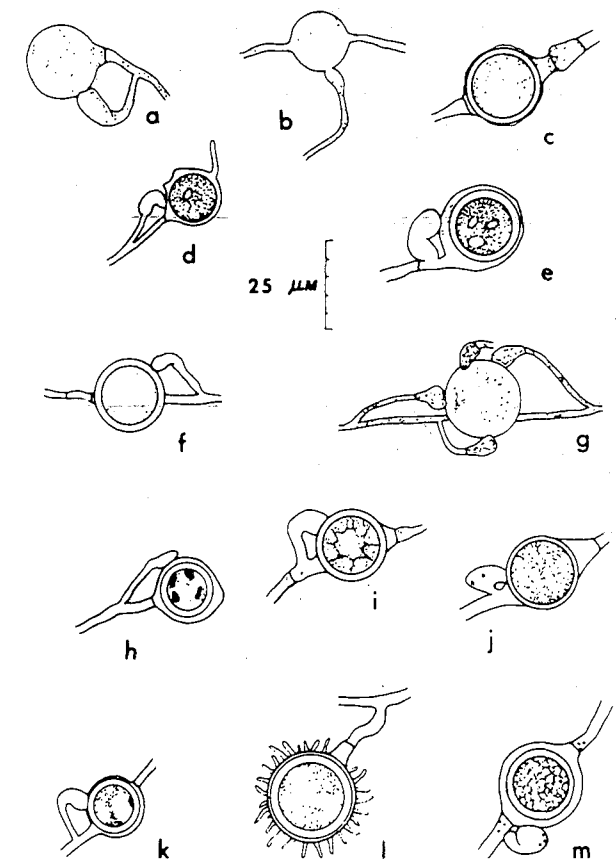

Fig. 40. *Pythium* sex organs. **a–g,** Different types of antheridia; **h, i, k, and l,** oogonia; and **j and m,** oospores in oogonia. (Reprinted, by permission, from Robertson, 1980)

strated in Israel. Frequent irrigation of sandy soils increased the severity of pod rot caused by a combination of *P. myriotylum* and *F. solani*, whereas less frequent and heavier irrigations reduced pod rot severity. Pod rot incidence and/or severity also may increase after injury resulting from feeding by diverse soil fauna. Soilborne mites and springtails have been implicated in the spread of *P. myriotylum* from infected to healthy peanut pods in North Carolina. Wounds created by southern corn rootworm feeding were involved in increased pod rot incidence in Virginia. The peanut root-knot nematode, *Meloidogyne arenaria*, may increase the incidence of both pod rot and preemergence damping-off of peanut caused by *P. myriotylum* in Florida.

Control

Some resistance to pod rot caused by *P. myriotylum* has been reported. The most widely planted peanut cultivars in Virginia are the most resistant to *P. myriotylum*. High yield potential and moderate levels of resistance to *Pythium* spp. have been reported in certain peanut lines grown in *Pythium*-infested soil in Texas. The spanish variety Toalson has resistance to both *P. myriotylum* and *R. solani*. In Israel, spanish and valencia peanuts resistant to pod rot caused by *P. myriotylum* and *F. solani* have been developed. Evaluation of components of resistance to *P. myriotylum* has shown that equally susceptible cultivars differ in disease incidence and that a low disease incidence does not necessarily mean a high degree of resistance but could imply the existence of an escape mechanism.

Wide-spectrum fungicides or combinations of fungicides often are needed to control pod rot. Use of effective nematicides can be important, since some types of nematodes intensify pod rot in certain locations. Soil fumigants containing methyl isothiocyanate and a nematicide have provided some control. Metham sodium applied in irrigation water controls pod rot in Israel.

Pod rot caused by *P. myriotylum* can be significantly suppressed in some areas by the application of high dosages of gypsum. However, the addition of gypsum to peanut plants did not reduce the severity of pod rot caused by *P. myriotylum* or *F. solani* in Israel. Application of K_2SO_4 or $MgSO_4$ to peanut plants at blooming increased pod rot.

Control of *Pythium* diseases of peanut through field management has been difficult. Traditional crop rotations are reported to have little effect on controlling pod rot in peanuts; however, fields in which peanut has been grown for several seasons in succession have significantly more pod rot caused by *Pythium* spp. than fields that are fallow for two growing seasons.

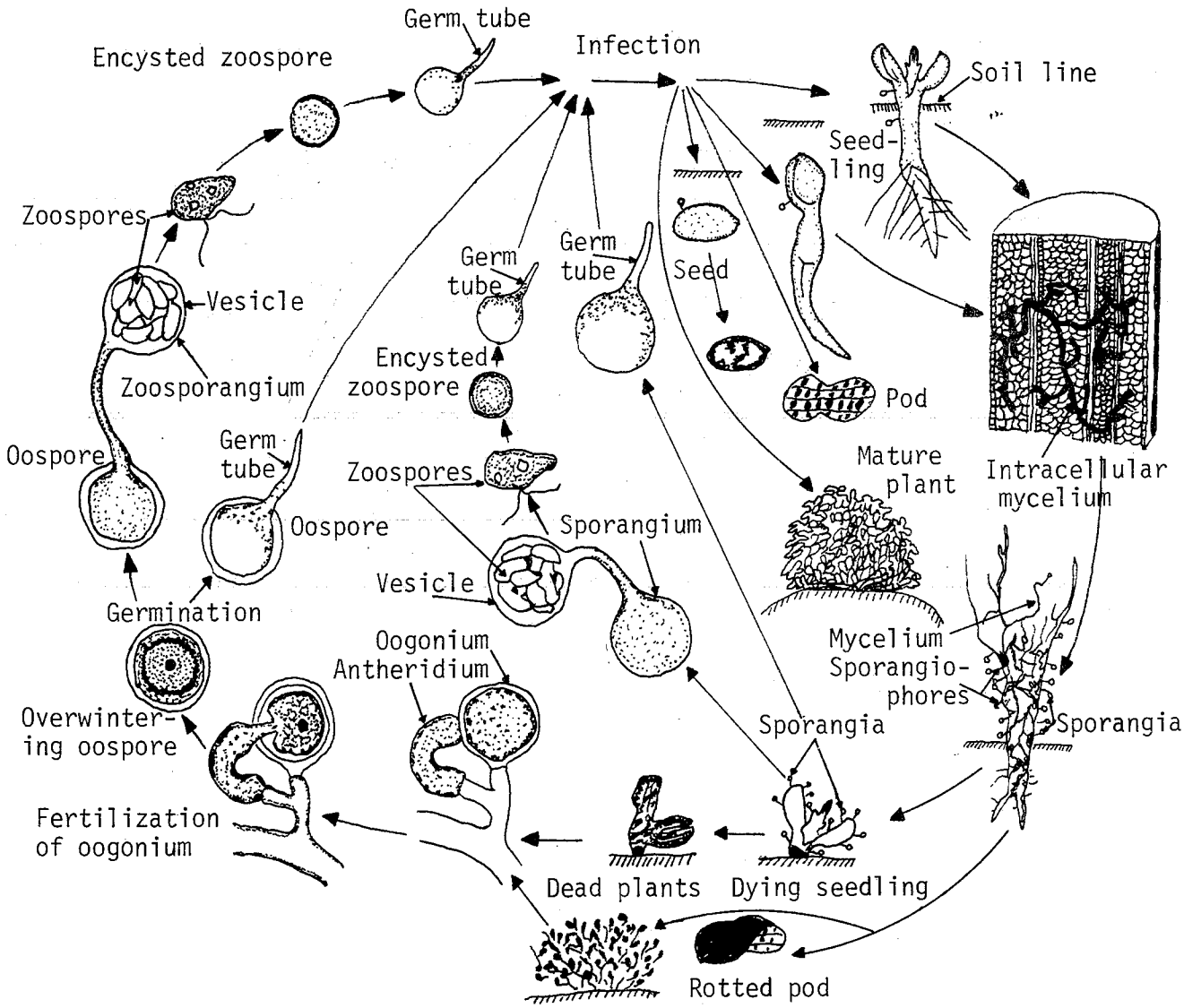

Fig. 41. Life cycle of *Pythium*. (Modified and reprinted, by permission, from G. N. Agrios, 1978, Plant Pathology, 2nd ed., Academic Press, New York. Prepared by A. J. Jaks)

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(Prepared by M. K. Beute)

Rhizoctonia Diseases

The peanut plant is susceptible to diseases caused by *Rhizoctonia solani* from planting until harvest. The fungus is responsible for rotting pods and any aboveground portions of the plant in close proximity to the soil as well as for seed decay, preemergence and postemergence damping-off of seedlings, and root and hypocotyl rot. Seed decay and damping-off can be serious, especially in fields not involved in crop rotation or when conditions are not favorable for seedling development. Replanting occasionally may be necessary. Root and hypocotyl rot can reduce yields. In the southeastern United States, Rhizoctonia limb rot has become a major problem, particularly in irrigated fields. Annual losses in Georgia exceed several million dollars.

Symptoms

Peanut seed are sometimes decayed prior to emergence by seedborne or soilborne *Rhizoctonia* spp. Such decay may be difficult to distinguish from that caused by other seed-rotting pathogens. Symptoms on emerged seedlings are more distinctive and include dark, sunken, "sore-shin" lesions just below the soil line (Plate 39). As the fungus colonizes the hypocotyl, the lesions become darker and larger. Under optimum conditions for disease development, the fungus will cause enough damage to the hypocotyl and roots to cause plant death (Plate 40). Under less favorable conditions, lesions will be more restricted and the plants will survive. Colonization of roots by *Rhizoctonia* spp. may cause small, brown lesions on secondary or tertiary roots or involve severe necrosis of the entire root system. Older plants tend to have sunken, brown or black lesions on the upper taproots.

Symptoms of *Rhizoctonia* infection on aboveground plant parts can be dramatic. Although the disease is commonly referred to as Rhizoctonia limb rot, symptoms may be evident on stems, leaves, or pegs. Lower branches in contact with the soil are usually the first to become infected. Lesions are initially small and light to dark brown with a distinctly zonate or target pattern (Plate 41). They often form near the terminal end of stems, particularly when stems have been injured by tractor traffic (Plate 42). These lesions may expand inward toward the crown of the plant. Additional lesions may form on stems, either from direct infection or from fungal growth up infected pegs or leaves (Plate 43). Girdling of the stem is sufficient to cause the loss of any pods between the lesion and the stem terminal. Pods formed on the outer limbs are more likely to rot or be shed at harvest, but the pathogen can also damage the primary pod crop (Plate 44).

R. solani frequently destroys the tips of pegs or colonizes pegs near the soil line (Plate 45). The fungus may subsequently colonize attached pods at any time or simply cause their loss at harvest by severing the peg. *Rhizoctonia* is a major component of the peanut shell mycobiota, and shed pods remaining in the soil have higher populations of *Rhizoctonia* spp. than those harvested successfully. Infected pods have dry, dull-colored, light to dark brown lesions (Plate 46) in contrast to the dark, greasy-appearing lesions characteristic of *Pythium myriotylum* infections. In fact, *Rhizoctonia* spp. may play a vital role in peanut pod rot (see Peanut Pod Rot Complex).

Causal Organism

R. solani Kühn (teleomorph *Thanatephorus cucumeris* (A. B. Frank) Donk) is found wherever peanut is cultivated and can be readily isolated from plants or soil. A severe foliar blight caused by *R. solani* anastomosis group 1 (AG-1) has been reported, and AG-2 isolates are sometimes weakly virulent. However, AG-4 is the most common pathogen of peanut (Fig. 42). *R. solani* AG-4 is highly virulent on seed and seedlings as well as on stems and foliage of mature plants.

Disease Cycle

R. solani is well adapted for long-term survival in the soil because of its ability to survive saprophytically on plant debris and form abundant sclerotia in infected plant parts. Large amounts of pods (630–778 kg/ha) are often left in the soil after harvest. Isolates of *R. solani* AG-4 have been recovered from peanut pods after 2 years in the soil, but populations fall sharply during the first year. The pathogen also has an extremely wide host range consisting of approximately 500 plant genera, although AG-4 is found primarily on members of Chenopodiaceae, Leguminosae, and Solanaceae. This enables the fungus to infect and reproduce not only on various rotated crops, but also on a multitude of weed species (Plate 47). The addition of organic matter to the soil or exudates from a susceptible host will stimulate sclerotia to germinate. The fungus can penetrate the plant directly through the intact cuticle and epidermis, by infection cushions, or may enter through natural openings or wounds. Colonization of the plant is aided by production of cellulolytic and pectic enzymes. Mechanical injury to stems has been clearly linked to increased limb rot severity. Therefore, tractor traffic should be kept to a minimum, especially after the plants have closed the row middles. Also, overhead irrigation and excessive fertilization have been shown to increase limb rot severity. Disease severity is probably most damaging either early season, when it promotes excessive foliar growth, or late season, when the disease progresses most rapidly because of prolonged moisture under the dense plant canopy.

Control

Rotation with grass crops reduces severity of many peanut diseases, including those caused by *R. solani* AG-4, although brace roots of corn are reported to be symptomless carriers of AG-4. Other cultural practices such as deep plowing and nondirting cultivation (i.e., dirt is not thrown into the crown of the plant) are helpful in managing *Rhizoctonia*-induced diseases.

The recent registration of tebuconazole offers an effective chemical control for Rhizoctonia limb rot. Other fungicides

such as flutolanil or fluazinam may be registered also. These treatments will also reduce losses to *Rhizoctonia*-induced pod rots but may not be active against other pod-rotting organisms such as *Pythium myriotylum* and *Fusarium solani*. Foliar sprays of flutolanil have been shown to reduce the incidence of *R. solani* in peanut pods left in the soil. Adequate calcium nutrition is known to be essential for pod rot management. Combination chemical treatments containing carboxin, PCNB, and captan provide some control of *R. solani* on seed and seedlings, although stand reductions can still be significant in cold, wet soils. Resistance to limb rot has not been available in large-seeded peanut cultivars. Some resistance has been reported in spanish-type peanuts. The recently released runner cultivar, Georgia Browne, has good partial resistance to limb rot.

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(Prepared by T. B. Brenneman)

Rust

Rust is an economically important disease in most peanut-producing countries of the world and causes substantial yield losses, particularly if the crop is also attacked by the leaf spot pathogens *Cercospora arachidicola* and *Cercosporidium personatum*. During recent years, combined attacks by rust and leaf spot have caused severe crop losses in many countries of Asia and Africa and have all but eliminated commercial peanut production in the Caribbean region and Central America. In the People's Republic of China, rust caused a 49% reduction in pod yield and lowered the 100-kernel weight by 19%. Artificially induced rust epidemics caused up to 79% reduction in pod yield in India. The disease is not a major limiting factor in peanut production in the United States, with the exception of southern Texas, where rust causes severe economic losses during some years. Losses measured at two locations in Texas were 77 and 86% from foliar diseases and 50 and 70% from rust alone. Establishment of the disease early in the growing season causes reduced pod fill and necessitates early harvesting. In addition, hay yields are drastically reduced.

Symptoms

Rust can be easily recognized when the orange pustules (uredinia) appear on the lower surfaces of peanut leaves and then rupture to expose masses of reddish brown urediniospores (Plates 48 and 49). In highly susceptible cultivars, the original pustules may later be surrounded by colonies of secondary pustules. Pustules may later be formed on the upper surfaces of the leaflets opposite those on the lower surfaces. The pustules, which develop on all aerial plant parts except flowers, are

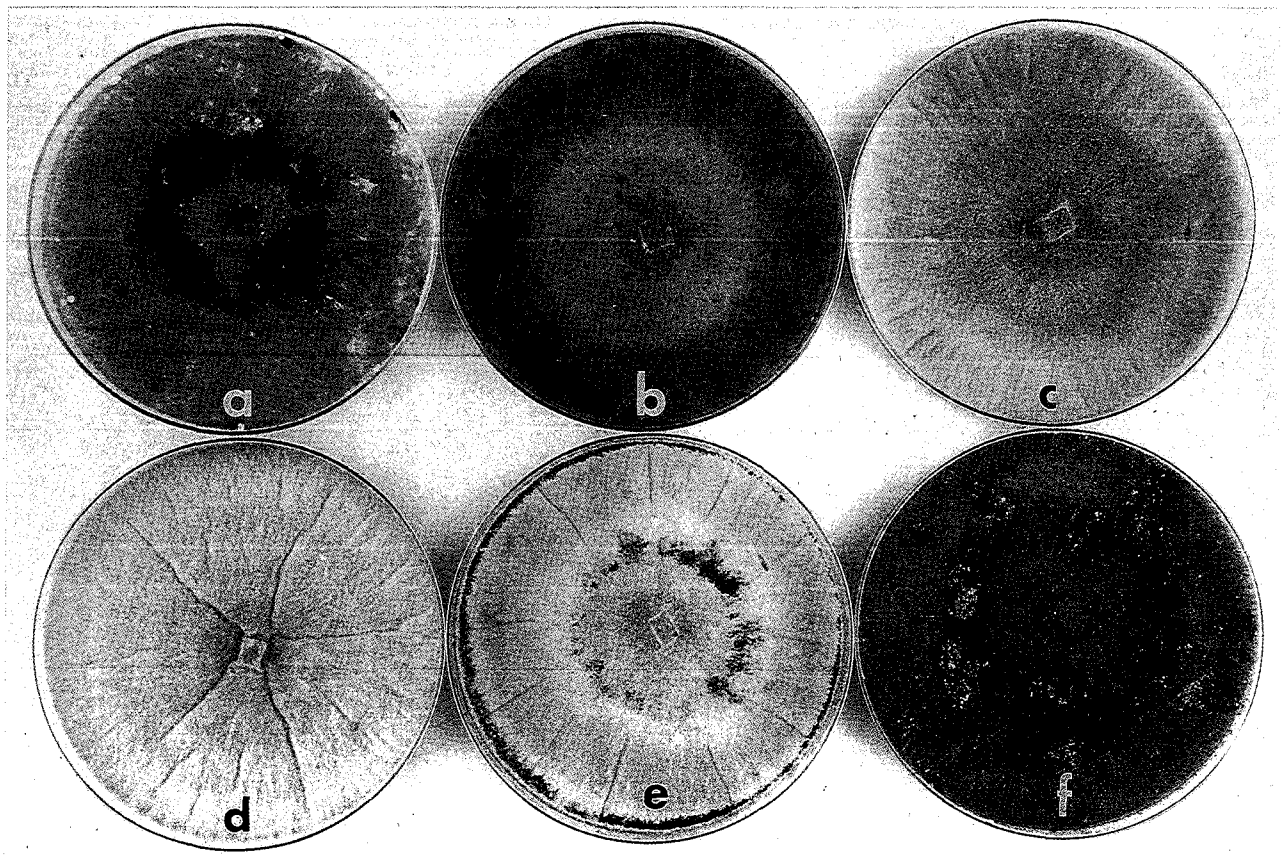

Fig. 42. **a–c,** Multinucleate anastomosis groups of *Rhizoctonia solani* and **d–f,** binucleate *Ceratobasidium* anastomosis groups of *Rhizoctonia*-like fungi from peanut. (Courtesy D. Bell)

usually circular and 0.5–1.4 mm in diameter. Pustules may also form on shells of developing pods. Unlike the rapid defoliation associated with leaf spots, leaves infected with rust become necrotic but remain attached to the plant. Heavily infected plants often appear pale green.

Causal Organism

Puccinia arachidis Speg. is the causal organism of peanut rust. The uredinial stage is the predominant and most commonly observed. The uredinia are pustular, scattered or irregularly grouped, and round, ellipsoid, or oblong. They are subepidermal in origin; covered by a thin, membranous, netlike peridium; and blisterlike when immature, becoming erumpent, powdery, and dark cinnamon brown when mature. The ruptured epidermis is conspicuous. Urediniospores (Fig. 43) are broadly ellipsoid or obovoid (23–29 × 16–22 µm), have brown walls 1–2.2 µm thick, and are finely echinulate, with echinulae 2–3 µm apart (Fig. 44). Urediniospores usually have two germ pores, which are nearly equatorial, often forming in flattened areas.

Telia, chiefly occurring on the lower sides of peanut leaves, are scattered, prominent, naked, pulvinate, and chestnut brown or cinnamon brown, becoming grayish from the germination of spores. A ruptured epidermis is prominent. Teliospores (Fig. 45) are oblong, obovate, ellipsoid, or ovate with a rounded to acute and thickened apex. They are constricted in the middle, tapering gradually at the base or tapered and rounded at both ends; smooth walled; predominantly two celled but sometimes have one, three, or four cells; 38–42 × 14–16 µm; light or golden yellow or chestnut brown; 0.7–0.8 µm thick at the sides; and 2.5–4.0 µm thick at the top. The apical thickening is almost hyaline. The pedicel is thin walled, hyaline, usually collapsing laterally, and up to 35–65 µm long but is usually detached at the spore base. Teliospores germinate at maturity without a dormancy requirement.

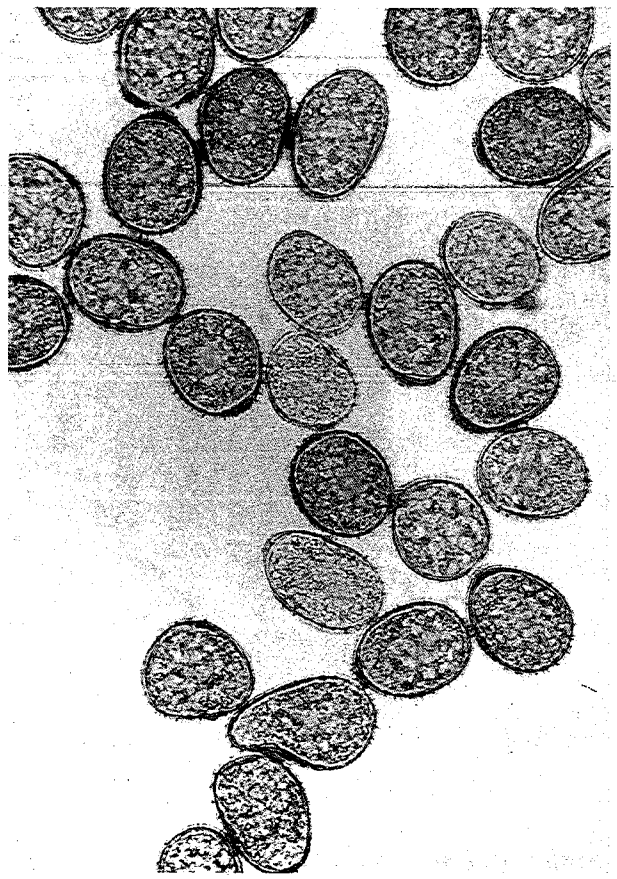

Fig. 43. Urediniospores of *Puccinia arachidis*.

Spermagonia, aecia, metabasidia, and basidiospores have not been reported for *P. arachidis*.

Because there is no knowledge of spermagonia, aecia, and hosts that basidiospores will infect, the life cycle of peanut rust is unknown and the taxonomic position of the fungus is obscure.

Disease Cycle and Epidemiology

Urediniospores are the main, if not the only, means of dissemination of this pathogen. There are a few authentic records of the occurrence of teliospores in South America but none from other countries. The pathogen is highly host specific. There are no records of any collateral hosts of peanut rust outside the genus *Arachis*. Urediniospores are short lived in infected crop debris in the tropics, and the fungus is unlikely to survive from season to season under postharvest conditions that include a fallow period of more than 1 month between successive peanut crops. The pathogen may survive from season to season on volunteer peanut plants. Long-distance dissemination of the pathogen may be by airborne urediniospores, movement of infected crop debris, or movement of pods or seed, the surfaces of which are contaminated with viable urediniospores. There is no reliable evidence of peanut rust being internally seedborne, and there is no authenticated report of rust being spread by germ plasm exchange. Spread of the organism within fields is facilitated by wind, rain splash, and insects. Urediniospores can remain viable for several months when stored at a low temperature (–16°C), but at a high temperature (40°C), they lose viability within 5 days. The thermal death point of urediniospores is 50°C for 10 min. The optimum conditions for germination of urediniospores include temperatures of 20–25°C and low light. Temperatures of 20–30°C and free water on the leaf surfaces favor infection and subsequent disease spread. Plants of all ages are susceptible. The incubation period varies from 7 to 20 days, depending on environmental conditions and host genotype. Intermittent rains with mean relative humidity above 87% and temperatures between 23 and 24°C for several days favor disease initiation. Continuous dry periods with temperatures greater than 26°C and relative humidity below 75% delay rust infection and reduce disease severity.

Control

Wherever possible, field management should include a fallow period of at least 1 month between successive peanut

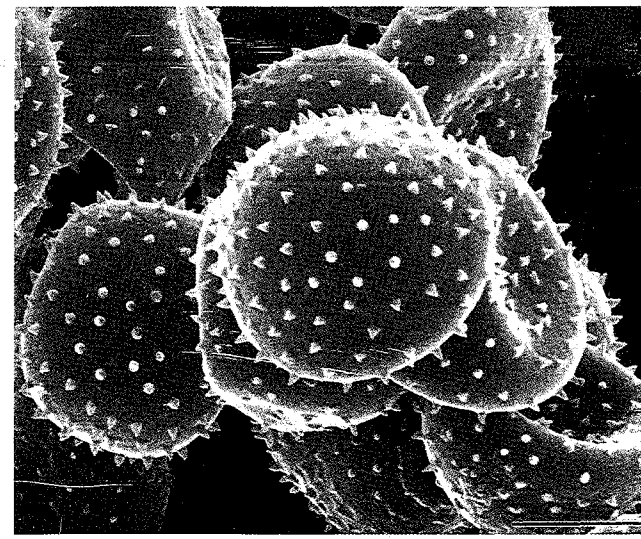

Fig. 44. Urediniospores of *Puccinia arachidis* with echinulation. Bar = 15 µm. (Courtesy R. A. Taber)

crops. Eradication of volunteer peanut plants during this period is important in reducing the primary source of inoculum. If cropping systems permit, time of sowing should be adjusted to avoid infection from outside sources and to avoid environmental conditions conducive to the onset of an epidemic. Existing plant-quarantine procedures should suffice to prevent spread of the pathogen on pods or seed externally contaminated with rust spores to areas where the disease is absent.

Several fungicides and mixtures of fungicides have been tested for control of rust or, more often, for control of rust and leaf spot together. The dust formulations (copper, sulfur, and copper plus sulfur) that were commonly used for control of leaf spot in the United States up to the 1960s also controlled rust, but sprays of Bordeaux mixture and dithiocarbamates were even more effective. The structurally related fungicides benomyl and carbendazim are effective against leaf spot but ineffective against rust. Tridemorph is effective against rust but not against leaf spot. Chlorothalonil and tebuconazole are effective against both rust and leaf spot. It is obvious that any fungicide treatment applied for control of rust must also be effective against leaf spot, because the diseases frequently occur together.

Prior to 1977, there were only a few reports of research on genetic resistance to peanut rust, but the rapid spread of rust during the early 1970s and the increasing cost of disease management with fungicides have resulted in increased research on genetic resistance to peanut rust. At the ICRISAT (International Crops Research Institute for the Semi-Arid Tropics) Asia Center in India, the world collection of more than 13,000 germ plasm accessions was screened for resistance to rust during the period from 1977 to 1992, and more than 120 rust-resistant germ plasm lines have been identified. Most of the currently available rust-resistant genotypes originated in Peru, which is believed to be one of the secondary "gene centers" of cultivated peanut.

Most of the rust-resistant germ plasm lines are primitive land races and have undesirable pod and seed characters. In recent years, several high-yielding, agronomically superior lines, with high levels of resistance to rust and moderate levels of resistance to late leaf spot, have been developed and released for cultivation in India (e.g., ICGVs 86590, 87157, 87160, Girnar 1, and ALR 1). ICGV 87160 has also been released in Myanmar (Burma). High levels of resistance and immunity to peanut rust have been found in wild *Arachis* spp. Cytogenetic research aimed at incorporating the rust resistance from wild *Arachis* spp. into the cultivated peanut is in progress in various countries. At the ICRISAT Asia Center, several stable, tetraploid or near-tetraploid lines derived from crosses between the cultivated peanut and wild species have been developed.

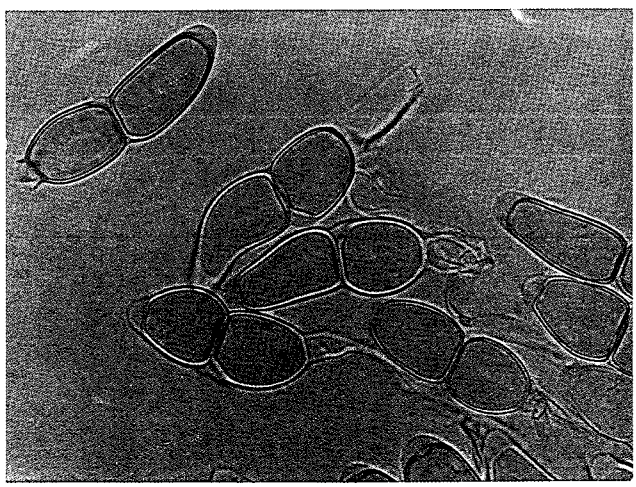

Fig. 45. Teliospores of *Puccinia arachidis*. (Courtesy J. F. Hennen)

The rust resistance available in the cultivated peanut is the "slow-rusting" type, i.e., resistant genotypes have an increased incubation period, decreased infection frequency, and reduced pustule size, spore production, and spore viability. On the basis of field scores, rust resistance in cultivated peanut is reported to be governed by two or three duplicate recessive genes. On the contrary, in diploid *Arachis* spp., rust resistance appears to be partially dominant. In crosses involving both cultivated and interspecific derivatives, rust resistance was found to be controlled by both additive and nonadditive gene action. Rust resistance in most genotypes is stable over a wide range of geographical locations except in a few locations, indicating possible variation in the pathogen.

Several mycoparasites of the peanut rust pathogen have been reported, and mycophagous insects may feed on urediniospores of peanut rust. However, no serious attempts have been made to use any of these organisms in biological control of peanut rust at the field level.

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(Prepared by P. Subrahmanyam)

Scab

Peanut scab was first observed in São Paulo, Brazil, in 1937 with subsequent reports in Brazil during 1941 and 1961 and in the Argentinian provinces of Corrientes (1966) and Córdoba (1975). Córdoba produces 99% of the peanut crop in Argentina. Scab has also been reported in the Chiba prefecture of Japan and in Swaziland. The mode of distribution of the scab pathogen to Africa, Asia, and South America has not been determined.

Symptoms

Symptoms first appear on leaves and petioles near the top of the plant. Numerous small, chlorotic spots, usually less than 1 mm in diameter, often form on the adaxial and abaxial leaf surfaces and are either uniformly distributed or clustered near the midvein. Spots on the adaxial leaf surface are light tan with raised margins and sunken centers, while spots on the abaxial surface are darker and not raised. Spots have a maximum diameter of 2 mm and coalesce near the midvein. Plant tissue becomes necrotic and torn, and leaf margins curl upward, resulting in additional tearing of tissue.

Spots on petioles and stems are larger and more irregular than spots on leaves and lead to development of cankers (Plate 50). As the disease progresses, lesions coalesce, plants are stunted, petioles and stems are sinuous, and lesions become corky and cover nearly all plant parts (Fig. 46 and Plate 51). Plants with numerous cankers appear to be burned.

Causal Organism

Sphaceloma arachidis Bit. & Jenk. is the causal pathogen of peanut scab. Acervuli (300 × 45 µm) are amphigenous, numerous, effuse, and sometimes pulvinate and erumpent. Conidiophores (8–11 × 3–5 µm) are hyaline, globose, and conical and arranged in aggregations resembling a palisade. Conidia (9–17 × 2.5–3 µm) are hyaline and mainly unicellular. Microconidia are approximately 1 µm in diameter. Growth of *S. arachidis* on potato-dextrose agar is slow at 22°C and occurs in convoluted colonies with dark red areas. On host tissue, lesions are dark olive with a velvet appearance.

Control

Several cultivars including Colorado Manfredi, Colorado Commun, and Colorado Irradialis INTA have partial resistance to *S. arachidis*. In Brazil, 15 of 639 breeding lines were resistant.

Because the pathogen survives in crop residue, crop rotation may reduce disease progress. Effective disease management can be obtained with benomyl.

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(Prepared by D. H. Smith)

Fig. 46. Symptoms of scab caused by *Sphaceloma arachidis*. Note characteristic sinuous stem. (Courtesy L. Giorda)

Sclerotinia Blight

Sclerotinia blight, first observed on peanut plants in Argentina in 1922, is now present in most peanut-producing countries of the world. It was first observed in the United States in Virginia in 1971, thereafter spreading to North Carolina, Oklahoma, and Texas. By 1982, Sclerotinia blight was considered the most important disease of the peanut in Virginia and Oklahoma (Plate 52). Yield losses of 10% are common. In areas of fields showing severe symptoms of disease, 1,500–2,000 kg of pods per hectare often remain in the soil after harvest. In such fields, pod losses often exceed 50% of expected yield.

Symptoms

The first obvious symptom of Sclerotinia blight is the rapid wilting or flagging of the tips of infected branches. Initial infections are characterized by small, light green, water-soaked lesions on stems near the soil line. Older lesions appear bleached or straw colored with a distinct demarcation zone between infected and healthy tissues. Foliage of infected branches becomes chlorotic, turns dark brown, and withers. Once the stem is girdled, the branch dies. These symptoms result in the blight appearance for which the disease is named.

White, fluffy mycelium develops on diseased tissue (Plate 53), especially during periods of high humidity. Another symptom characteristic of Sclerotinia blight is the shredding of infected branch and peg tissue (Plate 54). Severe peg infection results in significant pod losses at harvest.

Sclerotial production on and in infected plant parts is a characteristic sign of this disease. Sclerotia form on infected branches (Plate 55), leaflets, pegs, and pods and inside branches, pegs, pods, and roots (Plate 56).

Causal Organism

Sclerotinia minor Jagger is the causal agent; however, on rare occasions *S. sclerotiorum* (Lib.) de Bary also is found (Fig. 47). In artificial inoculations, either species can produce typical blight symptoms. Apothecia of *S. minor* are rarely observed in soils during the growing season (Plate 57) but appear during February and March or midfall on the soil surface. Apothecia are pale orange to white and have concave or flat tops. A single sclerotium can produce one to several apothecia, which range up to 6 mm or more in diameter. Asci contain eight hyaline ascospores measuring 8–17 × 5–7 µm.

The mycelium of *S. minor* is white and fluffy. Sclerotia have a black outer rind and a white inner cortex. They are small (0.5–3 mm), black, and irregularly shaped. Spermatia, produced on phialides, are globose, hyaline, and 3–4 µm in diameter. Their role in disease development is not known.

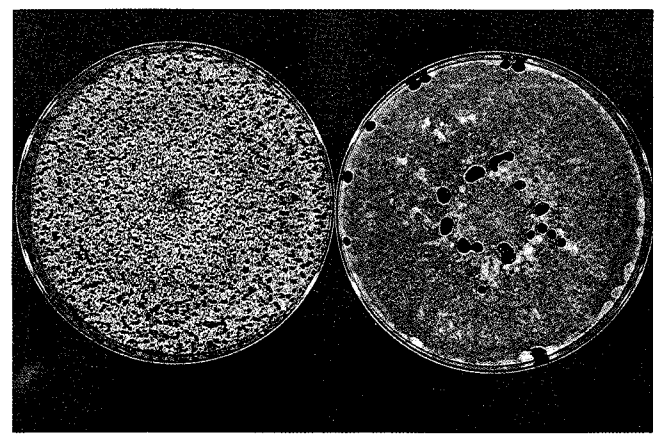

Fig. 47. Cultures of *Sclerotinia minor* (left) and *S. sclerotiorum* (right) growing on potato-dextrose agar.

Disease Cycle

S. minor overwinters as sclerotia. Viable sclerotia have been found in soil throughout the plow layer (the top 20 cm) of fields with a previous history of Sclerotinia blight but not planted to peanuts for 4 years. Under favorable environmental conditions (17–21°C, high soil moisture, and relative humidity above 95%), sclerotia of *S. minor* germinate myceliogenically. The life cycle of *S. minor* is provided in Figure 48. Plant tissues, branches, pegs, pods, and leaflets near or in contact with the soil and lying adjacent to a germinating sclerotium are infected by fast-growing, white mycelia. Before it penetrates the peanut stem, *S. minor* produces infection cushions (Plate 58). As disease progresses, infections occur in the plant canopy above the soil surface. Senescing or mechanically injured leaflets and stems can be colonized readily by *S. minor*. However, this is not a prerequisite for infection, since disease can become severe in healthy, vigorously growing plants.

Infection by *S. minor* is favored by cool conditions (18°C), moist soils, and high relative humidity (95–100%). Optimum sclerotial germination occurs at a pH of 6.5. Under favorable environmental conditions, white, fluffy mycelia can be seen on infected plant parts as well as on the soil surface. Sclerotia are produced abundantly on infected plant parts. The number of sclerotia at harvest often exceeds one sclerotium per 4 g of soil in areas exhibiting severe symptoms of Sclerotinia blight. Although sclerotial counts decline throughout the winter, more than enough viable sclerotia survive to propagate the disease during the following year. One sclerotium per 100 g of soil is sufficient to cause severe disease. Transmission of *S. minor* through peanut seeds is considered negligible, provided seeds are properly picked (which includes removal of damaged seed), screened (15/64-in. screen), and treated with a recommended seed treatment.

Control

High-quality peanut seed treated with chemical protectants should be planted to ensure against the low possibility of seed transmission of *S. minor*. Minimizing the injury of peanut vines by machinery is highly recommended. The use of resistant cultivars is highly desirable and profitable. Recently released peanut cultivars including Virginia 81B, Virginia 93B, Tamspan-90, and Southwest Runner possess resistance to *S. minor*. The use of fungicides can also aid in reducing losses. Excessive irrigation is not advised at any time during the growing season when cool conditions prevail. Some fungicides applied for the control of leaf spot (*Cercospora arachidicola* and *Cercosporidium personatum*) may increase the severity of Sclerotinia blight. Sclerotia are often colonized and destroyed by an array of soilborne fungi. *Trichoderma* spp., *Gliocladium* spp., and *Penicillium* spp. can be easily isolated from sound sclerotia and might be used as biocontrol agents (Plate 59).

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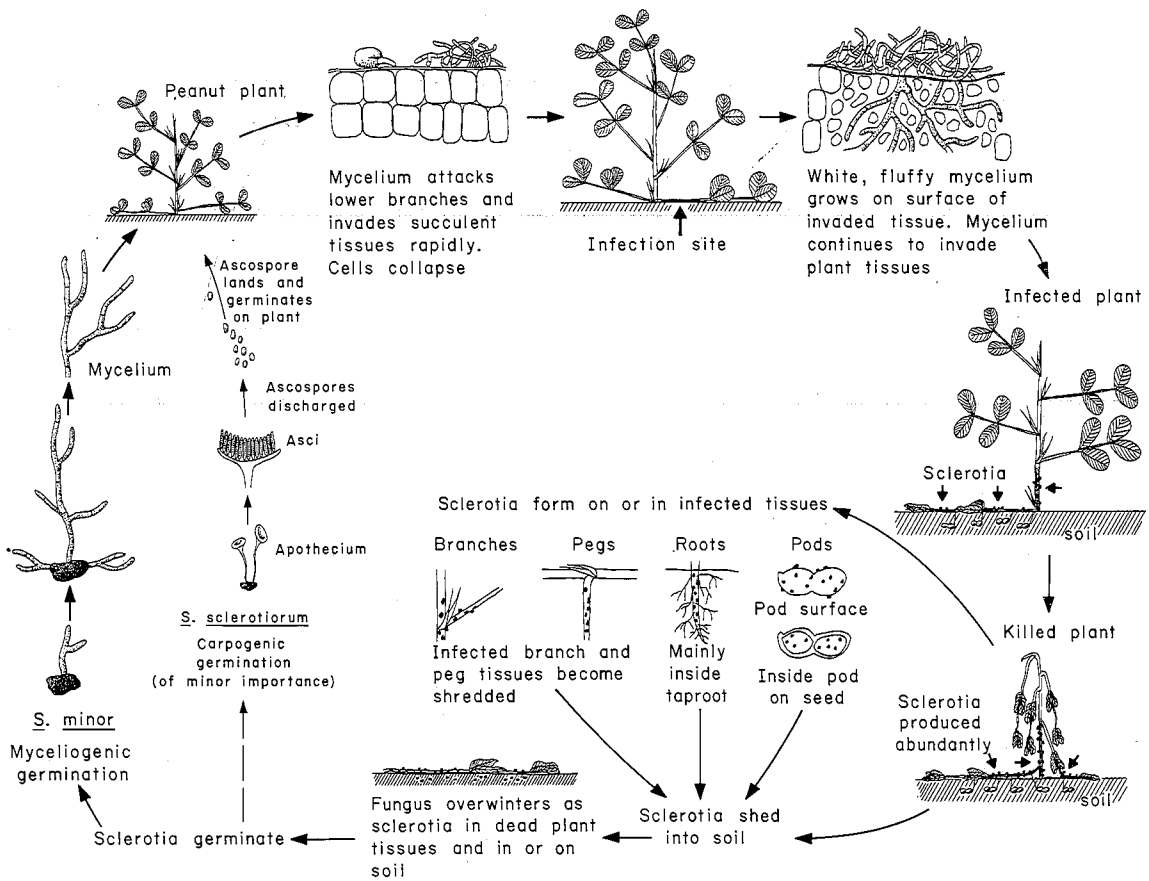

Fig. 48. Disease cycle of *Sclerotinia minor*. (Modified and reprinted, by permission, from G. N. Agrios, 1978, Plant Pathology, 2nd ed., Academic Press, New York. Prepared by Nancy Browning)

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(Prepared by D. M. Porter and H. A. Melouk)

Stem Rot

Stem rot of peanut, also known as white mold, southern stem rot, southern blight, and Sclerotium rot, is found in virtually all major peanut-growing areas of the world. Damage is easily seen during warm, wet weather, since a single infection site will often kill several adjacent plants. Disease incidence is often determined by counting the number of disease loci (30 cm or less of row length with dead or diseased plants). There is a strong negative correlation between numbers of disease loci per unit row length and yield, but the slope of the line varies from year to year, primarily because damage to pods and stems is environmentally driven. Yield losses typically do not exceed 25% but may be as great as 80%. This disease causes the greatest yield losses of all diseases of peanut in the United States. Losses to stem rot average 7–10% annually in the southeastern United States but are usually much lower in the southwest and Atlantic states.

Symptoms

Stem rot does not usually occur until midseason when the foliage has covered the row middles. The first obvious symptom of the disease is the yellowing and wilting of a lateral branch, the main stem, or the entire plant. *Sclerotium rolfsii* produces large amounts of oxalic acid, a phytotoxin that produces a purple stain on seed and is responsible for chlorosis and necrosis of foliage during the early stages of disease development. Sheaths of white mycelium can be seen at or near the soil line around affected plant parts (Plate 60). The mycelium grows rapidly under favorable environmental conditions and quickly spreads to other branches and plants. Spherical sclerotia (0.5–2.0 mm in diameter), produced abundantly on affected plant parts and the soil surface, are initially white but later turn dark brown (Plates 61 and 62). Lesions produced on the basal areas of branches and on pegs are initially light brown, become dark brown as disease develops, and do not extend more than 2–4 cm above the soil surface. Infected pods are usually rotted and tan to brown with a wet, spongy texture and may occur on plants without any visible aboveground symptoms (Plate 63). Infected pods often will be covered with soil clinging to mycelium on the pod surface (Plate 64).

Causal Organism

Stem rot is caused by *S. rolfsii* Sacc. The fungus grows well on a wide range of culture media and is characterized by the presence of white mycelium and hard, round, brown sclerotia, which are larger when produced in potato-dextrose agar cultures than in the field (Fig. 49). The fungus does not produce asexual spores. The basidial stage, *Athelia rolfsii* (Curzi) Tu & Kimbrough, is rarely seen in the field or in culture. When it does develop, *A. rolfsii* produces an exposed hymenium bearing clavate basidia and hyaline, pyriform basidiospores (1.0–1.7 × 6–12 µm). Considerable diversity in traits such as growth rate, virulence, and sclerotial production have been found, even among single-basidiospore strains (presumed homokaryons) from one parental isolate. At least 42 different mycelial compatibility groups have been used to classify *S. rolfsii*, although recent studies of peanut isolates from Texas indicate the presence of many more.

Disease Cycle

S. rolfsii has a broad host range of more than 200 plant species. The pathogen can colonize either the living plant or plant debris. Disease severity in sugar beets (*Beta vulgaris* L.) is a function of sclerotial population size; but in peanuts, the populations required to produce severe disease are so low that current quantification methods are insufficient to measure them. Deeply buried sclerotia survive a year or less, while those near the soil surface remain viable for many years.

Because *S. rolfsii* has a high demand for oxygen, overwintering sclerotia are activated only when they occur in the upper regions of the soil. In soils that crack deeply when dry (e.g., vertisols), oxygen can penetrate deep into the soil profile allowing pod and root rots that would not occur if the same soils were wet. Sclerotia also germinate in response to alcohols and other volatiles released from decomposing leaves. Shed leaves can also serve as a bridge to facilitate plant-to-plant spread.

Control of peanut leaf spot can indirectly increase stem rot severity. Maintaining a complete canopy creates a moist subcanopy environment that is conducive to disease development and may also intercept fungicides, directly or indirectly affecting *S. rolfsii*.

Control

Control of stem rot begins with prevention of inoculum buildup. Deep plowing serves to bury crop debris, which serves as a saprophytic food base, and sclerotia, which do not survive as well deeper in the soil. Cultivation for weed control will reduce disease buildup if growers are careful to prevent the movement of soil onto the plant crown or lateral stems. Crop rotation is a very effective practice, but long rotations (3–4 years) are required if a severe infestation has already developed. Grass crops such as corn, grain sorghum, or pasture grasses are particularly effective. Weed control is essential in any rotation, including fallow, because of the wide host range of the fungus.

Pesticides can influence the development of stem rot. Benomyl can actually increase the severity of stem rot by reducing levels of *Trichoderma* spp., a natural biological control agent. The dinitro herbicides and the insecticides ethoprop, fensulfothion, and chlorpyrifos are all reported to suppress the severity of stem rot. Chlorpyrifos has been used extensively for this purpose and also provides control of the lesser cornstalk borer, *Elasmopalpus lignosellus* (Zeller).

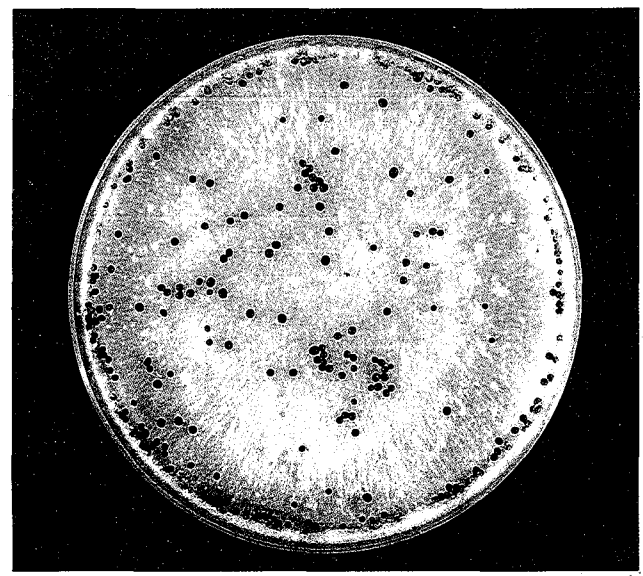

Fig. 49. Culture of *Sclerotium rolfsii* on potato-dextrose agar with characteristic sclerotia. (Courtesy D. M. Porter)

PCNB and carboxin have been used for many years in the United States to suppress stem rot. They offer no more than 50% control and are used as either granules or liquid delivered via irrigation water. Tebuconazole, registered for use on peanuts in the United States in 1994, provides a level of control superior to that achieved with previously registered products. Tebuconazole and several other triazole-type, sterol-inhibiting fungicides have given greater than 80% control of stem rot at seasonal use rates of less than 1.0 kg/ha. These treatments are applied either in a block at midseason or as full-season tank mixes with chlorothalonil. The triazoles generally have excellent activity against foliar pathogens as well, and propiconazole was registered for that purpose in 1994. Propiconazole has been shown to be most active against stem rot when applied via chemigation, but control is still moderate at best. Flutolanil is a systemic fungicide that is not a triazole but also offers excellent control of stem rot. This experimental fungicide is usually applied as a tank mix with chlorothalonil once or twice during midseason. It has little activity against leaf spot, and therefore full rates of chlorothalonil are required. Several other new fungicides have shown promise for stem rot control but are not yet registered. Both disease control and the related yield increases can be dramatic with these products where disease incidence is high (Plate 65).

Biological control with the antagonistic fungus *Trichoderma harzianum* Rifai has successfully suppressed stem rot severity, with control levels similar to those of PCNB. Commercialization of this agent has not been successful because of the difficulty of delivering a viable formulation to the field.

Until recently, only low levels of tolerance to stem rot had been reported in peanut germ plasm. Generally, genotypes with a more erect growth habit had less disease than cultivars with a spreading growth habit. However, Southern Runner produces a heavy, spreading canopy, and its disease incidence is about one-half that of Florunner, the current predominant cultivar. Georgia Browne has resistance comparable to that of Southern Runner, and recent studies have identified germ plasm with even higher levels of resistance.

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(Prepared by P. A. Backman and T. B. Brenneman)

Thermophilic Fungi

Thermophilic fungi have been isolated from peanut and peanut field soils in nine counties in Texas and three counties in Oklahoma. Thermophiles are defined as those fungi that have a maximum temperature for growth at or above 50°C and a minimum temperature at or above 20°C. Species isolated include *Mucor pusillus* Lindt, *Humicola lanuginosa* (Griffon & Maubl.) Bunce (syn. *Thermomyces lanuginosus* Tsiklinsky), *Talaromyces* (*Penicillium*) *dupontii* (Griffon & Maubl.) Emerson Apinis, *Thermoascus aurantiacus* Miehe, *Malbranchea pulchella* Sacc. & Penzig var. *sulfurea* (Miehe) Cooney & Emerson, *Aspergillus fumigatus* Fresen. (considered to be thermotolerant), *Thielavia albomyces* (Cooney & Emerson) Malloch & Cain, *Sporotrichum* sp., and *Chaetomium* sp. as well as numerous actinomycetes and bacteria. Discoloration of shells and seed is associated with several of these fungi.

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(Prepared by R. A. Taber)

Verticillium Wilt

The first report of Verticillium wilt in peanut was from Asia in 1937. The disease was reported in Australia in 1945 and in the United States (New Mexico) in 1985. It occurs in other peanut-growing countries, and although yield reductions of 60% have been reported in Argentina, it is not considered a serious worldwide production constraint. Verticillium wilt of peanut is found in all peanut-producing areas of the United States and has increased since 1970, but the disease is of economic importance only in New Mexico and Oklahoma. The occurrence of Verticillium wilt in Oklahoma in 1994 was widespread and severe in some fields. Data on yield losses in peanut in the United States caused by Verticillium wilt are not yet available.

Symptoms

The first symptoms of Verticillium wilt may appear as early as flowering but generally occur during pod fill (Plate 66). Early symptoms appear on leaves and consist of marginal chlorosis, loss of turgidity, and curling. As foliar symptoms progress, general yellowing, marginal leaf necrosis (Plate 67), wilting, defoliation, stunting, and dehydration of infected plants precede plant death. Symptoms intensify most rapidly under conditions of moisture stress and daytime air temperatures above 26°C. With adequate moisture, infected plants may exhibit moderate wilt symptoms and live to maturity. Infected plants usually mature earlier than noninfected plants.

Internal symptoms consist of light brown to tan vascular discoloration in crowns and stems (Plate 68). When wilt symptoms are severe, vascular discoloration also occurs in the roots and petioles (Plate 69). In a greenhouse study, the root mass of *Verticillium*-infected plants of the spanish cultivar Tamnut-74 was reduced by 50%.

Causal Organisms

Verticillium dahliae Kleb., a soilborne fungus, is considered the primary causal agent of Verticillium wilt of peanut. However, *V. albo-atrum* Reinke & Berthier has also been implicated as a pathogen. Both species are widespread and are the two most important vascular wilt pathogens within this genus. They attack a wide range of hosts in more than 36 plant families, including many important herbaceous and woody plants. The two species have different temperature requirements and morphology. *V dahliae* grows at 32°C and forms melanized microsclerotia that range in size from 50 to 200 µm (Fig. 50A). *V. albo-atrum* grows better at temperatures below 32°C and does not produce microsclerotia but does form thick-walled, dark mycelium (Fig. 50B).

The conidial stages of *V. dahliae* and *V. albo-atrum* are similar. When grown on potato-dextrose agar, *V. dahliae* produces white, fluffy, aerial mycelium with slender, often-branched conidiophores, which are usually arranged in whorls. Conidia (3 × 6.5 µm) are hyaline, unicellular, ovoid

to ellipsoid, and borne singly or in small clusters apically (Figs. 51 and 52). Microsclerotia form in 1 week at 20°C. White mycelial sectors, often devoid of microsclerotia, sometimes develop.

Disease Cycle

V dahliae survives in soil mainly as pigmented microsclerotia capable of withstanding environmental stress over prolonged periods, sometimes for several years. Microsclerotia are formed abundantly on or in all infected plant parts, including stems, pegs, pods, and roots (Plate 70). When infested peanut debris decomposes, microsclerotia are released into the soil, either freely or embedded in debris pieces, and remain dormant until peanut or other host root exudates stimulate them to germinate. Infection occurs through the roots, and the fungus spreads throughout the vascular system. A recent field observation in Oklahoma on a runner-type cultivar suggests that the interaction of the northern root-knot nematode and the Verticillium wilt pathogen can produce severe wilt symptoms.

V. dahliae spreads within and between fields by movement of soil or infested debris carrying microsclerotia on farm machinery from infested to noninfested areas. The pathogen can also be spread by wind and water movement of infested soil and infected tissue. Another possible means of dissemination of *V. dahliae* is by infected peanut seed. One to four percent of peanut seed harvested from field-grown plants that exhibited 50% wilt incidence carry *V. dahliae*. However, seed transmission of *Verticillium* in peanut has never been documented.

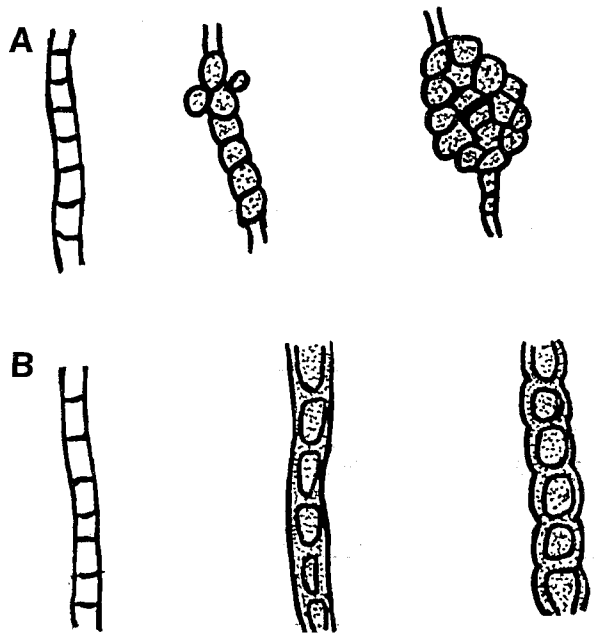

Fig. 50. Formation of resting structures. **A**, Microsclerotia of *Verticillium dahliae* and **B**, dark mycelium of *V. albo-atrum*.

Control

Severity of Verticillium wilt is increased by high temperatures and moisture stress. Infested fields should be irrigated more frequently to reduce wilt symptoms and allow plants to mature. Use of *Verticillium*-free seed also is recommended. Weed control might aid in alleviating the incidence of Verticillium wilt, since some weeds are also susceptible to *V. dahliae*.

Peanut following nonhost crops such as grain sorghum or Sudan grass develop less wilt than peanut following susceptible crops such as cotton, okra, or peanut. Control of root-knot nematodes may reduce severity of wilt symptoms. Removing and burning infected crop residues reduces the inoculum density of the pathogen in field soil.

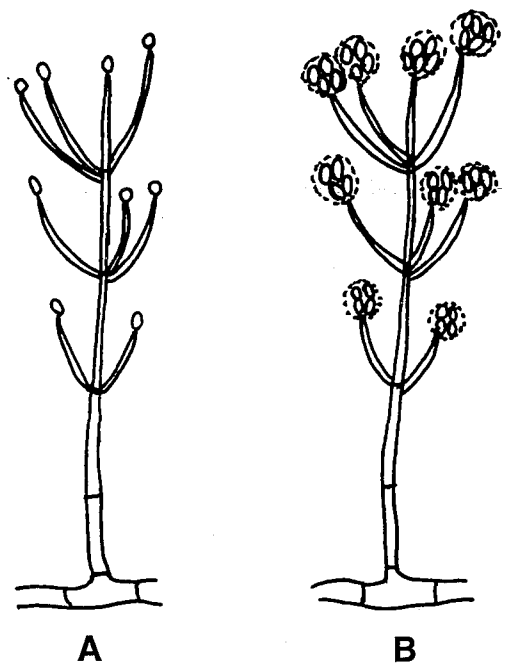

Fig. 51. Verticillate branching of *Verticillium* conidiophores in water mount (**A**) and bearing clusters of conidia (**B**).

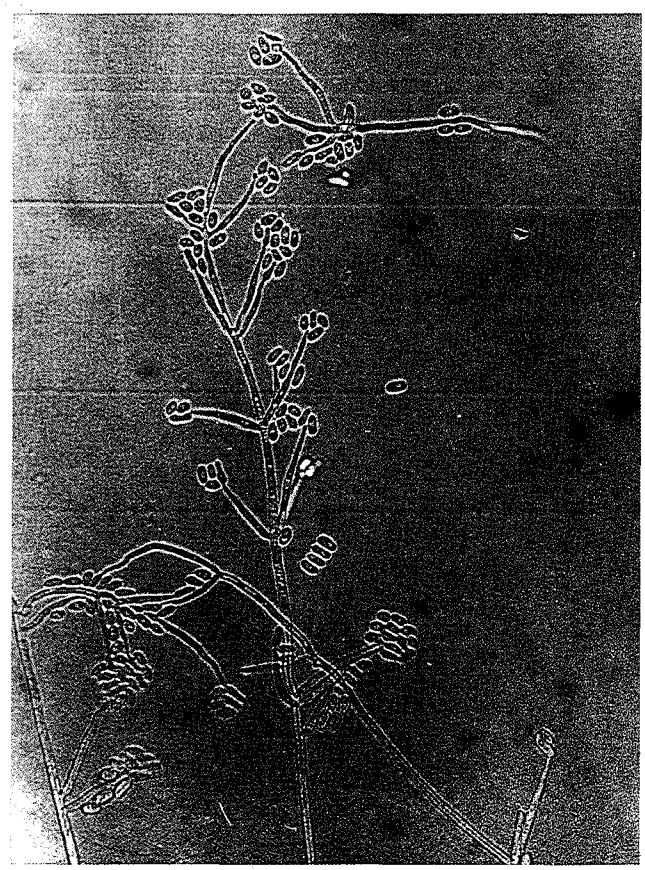

Fig. 52. Clusters of conidia on conidiophores of *Verticillium albo-atrum*. (415×) (Courtesy F. Uecker)

Chemical control of Verticillium wilt of peanut has not been effective in the United States. In Israel, the disease has been effectively controlled in sandy soil with metham sodium applied by sprinkler irrigation.

No cultivars of peanut are resistant to Verticillium wilt. Valencia and spanish peanuts are more susceptible to Verticillium wilt than bunch or runner types. In Israel, where Verticillium wilt is a problem, several breeding lines resistant to *Verticillium* have been developed.

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(Prepared by H. A. Melouk and J. P. Damicone)

Web Blotch

Web blotch of peanut occurs in many peanut-growing areas throughout the world, although its economic importance varies from country to country. It has been reported from Angola, Argentina, Australia, Brazil, Canada, China, Japan, Lesotho, Malawi, Mauritius, Nigeria, South Africa, Swaziland, Zambia, Zimbabwe, the United States (initially in Texas and subsequently in Florida, Georgia, New Mexico, Oklahoma, and Virginia), and the former Soviet Union. It is one of the most important foliar diseases of peanut in the Vaalharts and Natal regions of South Africa; in Zimbabwe, especially at high elevations and on irrigated, long-season cultivars; and in the United States on valencia-type peanuts in western Texas and New Mexico. In Zimbabwe, losses of approximately 10% of yield can be directly attributed to web blotch. Losses in New Mexico reach 50% in some years, and the disease can have a heavy impact on the quality of valencia peanuts marketed in-shell.

Web blotch is often referred to as net blotch because of the netlike appearance of the lesions. It is also known as Phoma leaf spot, Ascochyta leaf spot, and muddy spot.

Symptoms

The first signs of web blotch are small, dark brown or tan blotches (the color depends on the type of peanut) or netlike brown lesions (Plate 71) on the adaxial surfaces of leaves under a dense canopy or under conditions of high humidity. These blotches become more numerous and enlarge to form dark brown, roughly circular lesions with irregular margins (Plate 72). As lesions mature, they become darker with dull, rough surfaces and may completely cover the upper surface of the leaf. At this stage, small blotches may be visible on the lower surface. The leaf becomes brittle and is liable to disintegrate and detach from the plant (Plate 73).

Causal Organism

Web blotch is caused by *Phoma arachidicola* Marasas, G. D. Pauer, & Boerema. The nomenclature of the anamorph is confusing, because the fungus was previously assigned to the genus *Ascochyta (A. arachidis* Woronichin and *A. adzamethica* Schoschiaschvili). The teleomorph has also been assigned to various taxa, including the genera *Mycosphaerella (M. arachidicola* Jenk. non Chochrjakov and *M. argentinensis* Frezzi), *Didymosphaeria (D. arachidicola* (Choch.) Alcorn, Punith., & McCarthy), and *Didymella (D. arachidicola* (Choch.) Taber, Pettit, & Philley), or considered to be none of these. Confusion over placement of the teleomorph centers around interpretation of the identity of sterile elements in the pseudothecium (Fig. 53) and differences in opinions on development of pigmentation of the ascospores. *Didymella arachidicola* is the most commonly used holomorph classification.

Pycnidia of *P. arachidicola* are pale to dark brown, separate, globose to flask shaped, ostiolate, amphigenous, and immersed in the necrotic leaf spots. Pycnidiospores are not readily visible but can be seen distinctly in cleared (in KOH) and stained leaf tissue (Fig. 54). Size varies with the substrate and isolate, but in general, they are 85–240 µm in diameter. Conidia are formed in basipetal succession on short, conidiogenous cells and, under humid conditions, are exuded through the ostiole as light-colored droplets. Conidial sizes vary with the substrate and septation. One-celled, obovoid conidia (4–9 × 2.5–4 µm) are produced in culture, while on host material, spores may be larger (7–18 × 3–6 µm) and a large proportion of them are septate. Conidial sizes are also influenced by temperature—the lower the temperature the larger the conidia. Colonies of *P. arachidicola* on malt agar at 25°C are dark grayish olive. They may be loose and felty or closely appressed at the periphery. The hyphae are 2–8 µm in diameter, olivaceous to brown, and

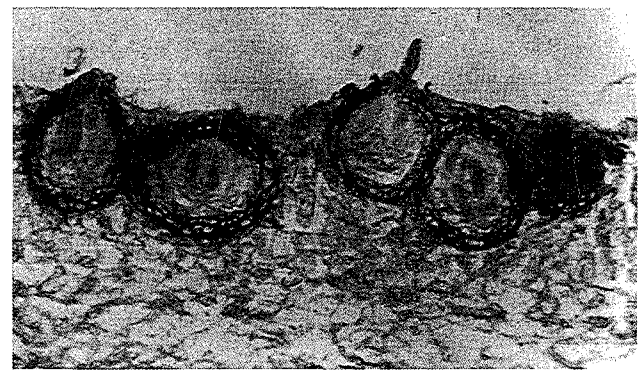

Fig. 53. Pseudothecia of the web blotch fungus, *Phoma arachidicola*. (Courtesy R. Taber)

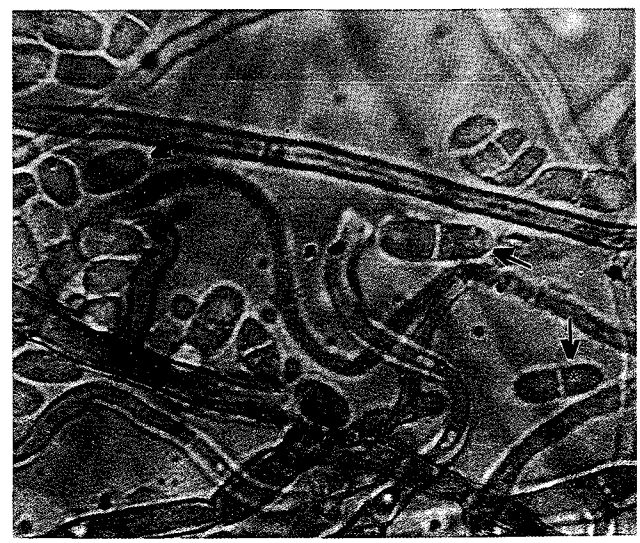

Fig. 54. Pycnidiospores (arrows) of *Phoma arachidicola*. (Courtesy R. Taber)

septate. Some isolates form dark, one- to multicelled chlamydospores (7–19 × 8–20 µm) both on aerial and submerged mycelia. Colonies on potato-dextrose agar are appressed and creamy white. A dark brown color develops as the fungus produces pigmented chlamydospores either in clusters or in chains. If the temperature is lowered to 18°C under near-ultraviolet light, concentric rings of pycnidia appear after 5 days in cultures on either medium. The teleomorph is not common in naturally infected tissue or in culture but can be induced to form on detached leaflets under high humidity by some isolates.

Pseudothecia (65–154 µm in diameter) are dark brown, subglobose to globose, short beaked or unbeaked, and usually immersed in the substratum. Cell walls are mostly isodiametric and angular to round. Asci are hyaline, cylindric to somewhat clavate, mostly with a differentiated foot, eight spored, and distichous. Ascospores (13–17 × 4.5–6.5 µm) have one septum and are smooth and at first hyaline, becoming dark with maturity. The upper cell of the ascospore is broader and more sharply tapered than the lower.

Disease Cycle

Web blotch development is generally more severe under cool, moist conditions and is more common on irrigated than on rain-fed crops. Prolonged leaf wetness periods at temperatures of 15–21°C favor disease development. *P. arachidicola* survives in infected crop residues or on volunteer peanut plants. Pycnidia and, in some cases, pseudothecia develop on fallen leaves and provide the initial inoculum to infect subsequent peanut crops. Germ tubes penetrate the cuticle directly, and small infection pegs form near the germinated spores. Networks of individual hyphae ramify between the cuticle and the epidermis and kill adjacent cells, resulting in the web symptom. Hyphae can also penetrate subepidermal tissue, and proliferation of hyphae and subsequent extensive cell necrosis produce typical blotch symptoms.

Control

Crop rotation and management of infected crop debris and volunteer peanut plants assist in eliminating primary sources of inoculum. Under conditions that favor severe web blotch infection, foliar fungicide sprays are very effective. A computer model to predict disease onset for efficient fungicide application has been developed. When present, early leaf spot, which usually appears before web blotch, provides some control of web blotch because it induces the production of phytoalexins in peanut leaves. Germ plasm resistant to web blotch is being included in breeding programs, but no commercial cultivars are yet available.

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(Prepared by D. L. Cole and C. Liddell)

Yellow Mold and Aflatoxin

Invasion of plant products by the yellow green molds *Aspergillus flavus* and *A. parasiticus* is a worldwide problem, especially in tropical and subtropical latitudes. Peanuts, corn, cotton seed, and tree nuts are particularly susceptible to invasion, both in the field and during storage. The toxicity of these fungi was first recognized in Great Britain in 1960 when turkey poults fed a protein supplement containing Brazilian peanut meal developed a syndrome originally called turkey X-disease. Investigations of the symptoms led to the discovery of the highly carcinogenic aflatoxins B_1, B_2, G_1, and G_2; B_1 is the most toxic to mammals. It has subsequently been postulated that cyclopiazonic acid, another mycotoxin produced by *A. flavus*, may have been responsible for several of the symptoms in the early reports of turkey X-disease not characteristic of aflatoxin poisoning.

The International Agency for Research on Cancer has classified aflatoxin as a probable human carcinogen. The U.S. Food and Drug Administration permits maximum aflatoxin levels of 20–300 and 20 ppb in peanut products destined for animal and human consumption, respectively. In other countries, acceptable maximum aflatoxin levels for foods range from 0 to 50 ppb (5 ppb is the most common) and maximum levels for animal feeds from 10 to 200 ppb.

Peanut seed and their products are assayed for aflatoxin by grinding a sample and using a solvent mixture such as methanol and water for extraction. Aflatoxin is then directly measured by use of an immunoassay; or the extract is passed through a column, and aflatoxin is quantified by thin-layer chromatography or high-performance liquid chromatography.

Symptoms

Symptoms of severe drought stress in peanut plants, including permanent wilting of the foliage, receding of the canopy between rows, and shedding of leaves, indicate conditions favorable for preharvest aflatoxin contamination of the seed. Harvested pods may reveal extensive insect damage, sometimes with visible yellow green sporulation of the causal fungus at the points of injury. Infected seed often exhibit areas of brown or yellow discoloration that may also be associated with external sporulation of the fungus (Plate 74). However, considerable invasion and aflatoxin contamination commonly occur without visible sporulation. Concealed damage, in which the inner lumen between the cotyledons is filled with conidial heads, can be detected only by splitting the seed in half (Plate 75).

High levels of infection by *A. flavus* in peanut seed may result in preemergence rotting of the seed and seedlings, a condition known as yellow mold. Brown, necrotic lesions with sporulating *A. flavus* are present on the cotyledons, radicles, and hypocotyls of ungerminated and germinated seed. Emerged seedlings also exhibit necrotic lesions on the cotyledons, and the plants are stunted and chlorotic and have poorly developed root systems.

Causal Organisms

Conidia-bearing structures (conidiophores) of *A. flavus* Link:Fr. and *A. parasiticus* Speare arise from septate, vegetative hyphae. The nonseptate, rough-walled stipe is swollen terminally to form a globose to subglobose vesicle. Phialides may be borne directly on the vesicle (uniseriate condition) or

may arise from metulae that cover the vesicle surface (biseriate condition). The vesicle, metulae when present, phialides, and chains of conidia comprise the head (Fig. 55). The heads of *A. parasiticus* are predominantly uniseriate. In *A. flavus*, seriation is more variable; usually at least 20% of the heads are biseriate, but a small percentage of isolates are almost entirely uniseriate. Conidia are globose to ellipsoidal and 3–6 µm in diameter. The texture of the conidial wall is the best means of distinguishing the two species: conidia of *A. flavus* are nearly smooth to finely roughened while those of *A. parasiticus* are distinctly roughened. In addition, when colonies are grown on Czapek's agar medium, conidia of *A. flavus* are yellow green to gray green en masse whereas those of *A. parasiticus* are dark green (Plate 76). Isolates of either species may produce dark brown to black sclerotia. A third, related aflatoxin-producing species, *A. nomius* Kurtzman, Horn, & Hesseltine, has been reported only rarely from peanut commodities. Morphologically, *A. nomius* resembles *A. flavus*, differing primarily in the formation of elongate sclerotia.

A. flavus typically produces aflatoxins B_1 and B_2 in 40–80% of isolates, whereas nearly all isolates of *A. parasiticus* produce aflatoxins B_1, B_2, G_1, and G_2. Furthermore, *A. flavus* often produces cyclopiazonic acid, which has not been reported from *A. parasiticus*.

Disease Cycle

Inocula of *A. flavus* and *A. parasiticus* are present in soil as conidia and sclerotia and in plant debris as mycelium. Populations of the two species together in fields under peanut cultivation may exceed 5,000 colony-forming units per gram of soil. These fungi are saprophytic on plant debris in soil and are potential facultative parasites of peanut seed. *A. flavus* appears to be more aggressive than *A. parasiticus* in the preharvest invasion of seed, although more aflatoxin may be attributed to *A. parasiticus* (on the basis of the presence of G aflatoxins) in peanut seed than in aboveground crops such as corn and cotton seed.

The two most important conditions that favor preharvest invasion and aflatoxin contamination of peanut seed are excessive heat (a mean soil temperature of 27–30°C) and prolonged drought stress during the last 3–6 weeks of the growing season. The leaf canopy of the peanut plants recedes during drought, which further increases soil temperatures and evaporation of soil moisture. As the peanut seed lose moisture and eventually attain a water activity of approximately 0.95, phytoalexin synthesis ceases and fungal growth is no longer inhibited. Hence, preharvest peanut seed are most susceptible to invasion and aflatoxin contamination at water activities of 0.90–0.95. Below a water activity of 0.90, fungal growth is greatly restricted because of the reduced availability of moisture but ceases only when the water activity is less than 0.80. Mature peanut seed apparently have an additional resistance factor, which may account for the greater invasion by *A. flavus* and *A. parasiticus* as well as the higher levels of aflatoxin in smaller, immature seed.

Much of the aflatoxin in preharvest peanut seed can be attributed to insect damage, particularly the activities of termites in Asia and Africa and the lesser cornstalk borer, *Elasmopalpus lignosellus* (Zeller), in the United States. Damage by lesser cornstalk borers is favored by the hot, dry conditions that also promote aflatoxin contamination. Wounding of pods involves either direct penetration, in which the seed is often damaged, or external scarification. Insects transmit *A. flavus* and *A. parasiticus* to the wound sites, and damage to the seed by their feeding encourages rapid colonization by aflatoxigenic fungi. Seed may also become infected without visible damage to the pod. In such instances, the route of invasion is not understood, although it has been suggested that invasion first occurs in the ovary of the aerial peg or, alternatively, by way of the funiculus of the developing pod.

Peanut seed that become contaminated under conditions of drought stress may show an increase in aflatoxin when plants are dug and inverted in the windrow for drying if the seed become rehydrated by rainfall for an extended period. Otherwise, noncontaminated seed from sound pods do not appear to be invaded during windrow drying. Improper storage of the seed may further increase the risk for aflatoxin production. Conditions important for aflatoxin formation during storage include moisture, temperature, and the condition of the seed. In general, aflatoxin is produced at relative humidities greater than 83% (seed moisture content of 10.5–11.0%) and at temperatures of 12–42°C (optimum 20–35°C).

Control

Irrigation alleviates drought stress of plants and is the best control measure for minimizing aflatoxin contamination in the field. Where irrigation is not available, the problem may be reduced by early harvest during drought before contamination becomes extensive. Control of insects also lowers the incidence of damaged seed that contain high levels of aflatoxin, but drought conditions limit the use of most insecticides, which require moisture to be effective. Preemergence rotting of seed and seedlings caused by *A. flavus* is best avoided by planting noninfected, high-quality seed.

Proper storage in the warehouse will prevent further contamination of peanut seed with aflatoxin. Seed should be protected from rehydration caused by intense insect activity, leaky roofs, or moisture condensation resulting from temperature fluctuations. Good ventilation and adequate roof and wall insulation will limit condensation in the warehouse.

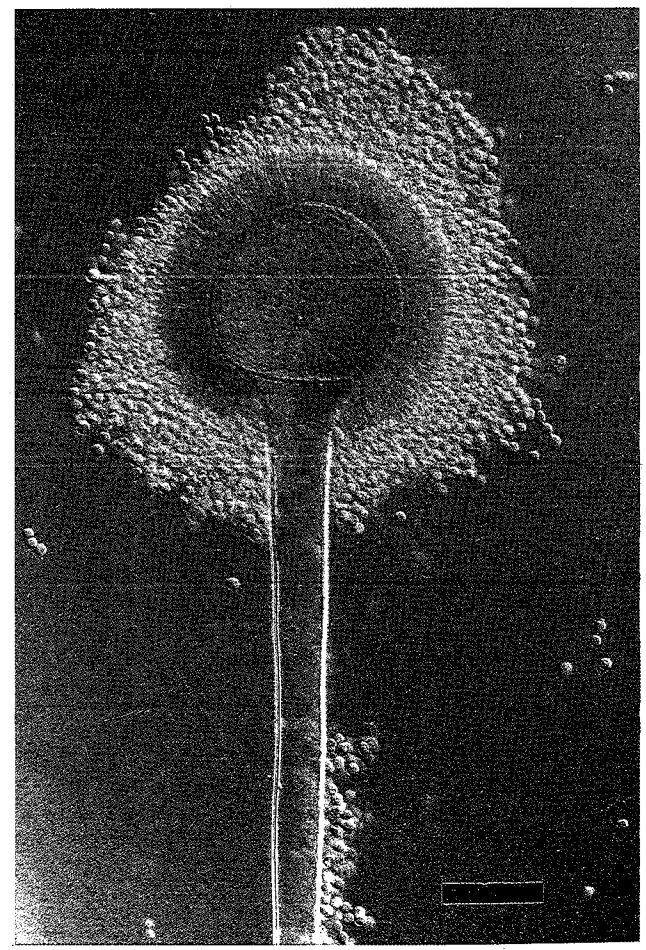

Fig. 55. Conidiophore of *Aspergillus flavus*.

Aflatoxin is not uniformly distributed in a contaminated seed lot, and early removal of high-risk seed, such as those that are damaged, immature, or loose (shelled during combining operations), can eliminate more than 95% of the aflatoxin during processing. Methods of removing high-risk seed include 1) removal of loose seed and small pods with a high-capacity belt screen; 2) separation of small, immature seed after shelling by use of vibratory screens; 3) density separation, in which lighter, aflatoxin-contaminated seed are separated on gravity tables; 4) electronic color sorting, which removes seed discolored by fungal colonization; and 5) blanching, in which the outer seed coat, or skin, is removed to further expose discolorations that can be detected by electronic color sorters. Seed from these high-risk categories are then diverted from the edible market to oil stock.

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(Prepared by B. W. Horn and J. I. Pitt)

Zonate Leaf Spot

Zonate leaf spot, a foliar disease of minor importance, has been observed in India, Thailand, and the United States. *Cristulariella moricola* (Hino) Redhead (syn. *C. pyramidalis* A. M. Waterman & R. P. Marshall) is the causal agent. The sclerotial state is *Sclerotium cinnamomi* Sawada. Necrotic lesions on leaves are 2–12 mm in diameter (Fig. 56). Small lesions have light brown centers surrounded by a ring of necrotic tissue. A zonate pattern is visible on both leaf surfaces (Plate 77). Pyramidal heads (conidia) (Fig. 57) form on both surfaces of necrotic leaves. There may be many pyramidal heads per lesion.

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(Prepared by D. H. Smith)

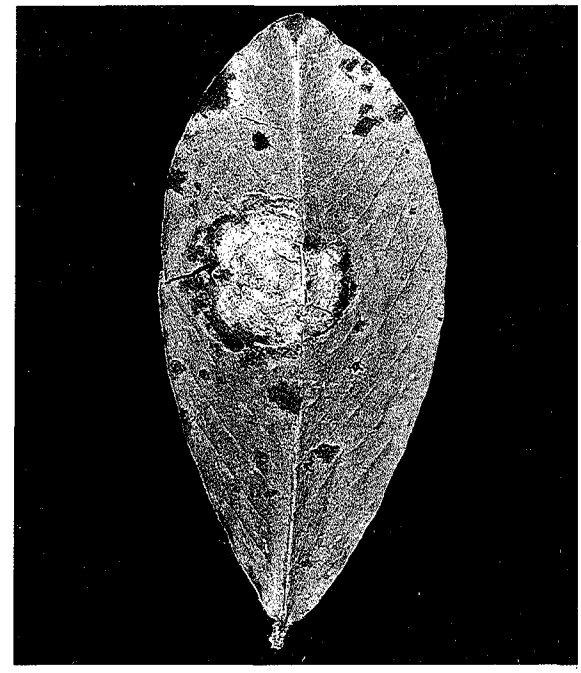


Fig. 56. Zonate leaf spot on the upper surface of a peanut leaflet.

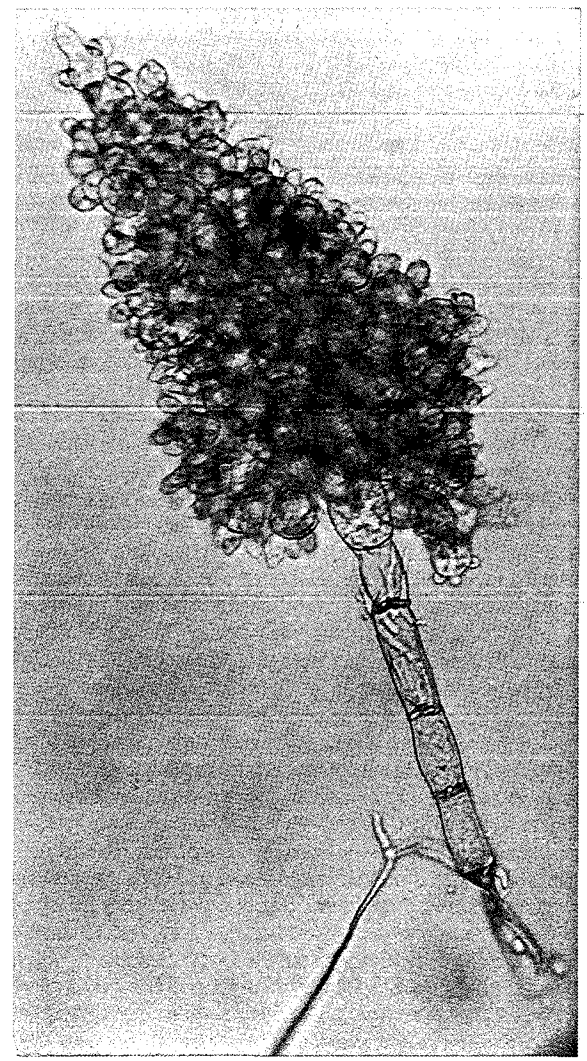
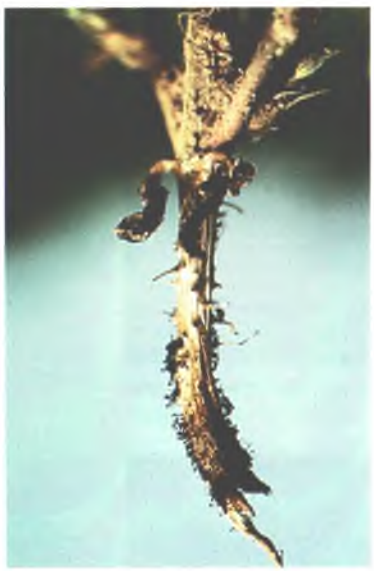


Fig. 57. Pyramidal head of *Cristulariella pyramidalis*. (Courtesy A. Latham)

Color Plates

1. Alternaria leaf spot. (Courtesy D. H. Smith)

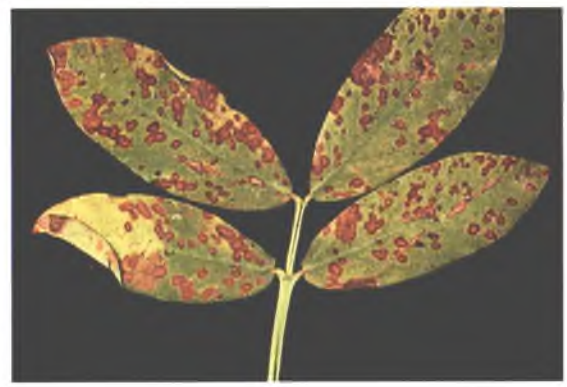
2. Early stages of Aspergillus crown rot. (Courtesy D. M. Porter)



3. Sporulation of *Aspergillus niger* on a shredded taproot. (Courtesy D. M. Porter)

4. Subterranean sporulation of *Aspergillus niger*. (Courtesy P. Phipps)

5. Symptoms of black hull, caused by *Chalara elegans*. (Courtesy D. Hsi)



6. Leaf spot symptoms caused by *Botrytis cinerea*. (Courtesy D. M. Porter)

7. Conidia and conidiophores of *Botrytis cinerea*. (Courtesy D. M. Porter)

8. *Gliocladium roseum* associated with mycelium of *Botrytis* spp. (Courtesy D. M. Porter)

9. Field symptoms of Botrytis blight. (Courtesy D. M. Porter)

10. *Botrytis cinerea* sporulating on a stem, petiole, and stipule. (Courtesy D. M. Porter)

11. *Botrytis cinerea* fruiting structures on a stem. (Courtesy D. M. Porter)

12. Foliar symptoms of charcoal rot. (Courtesy J. Damicone)

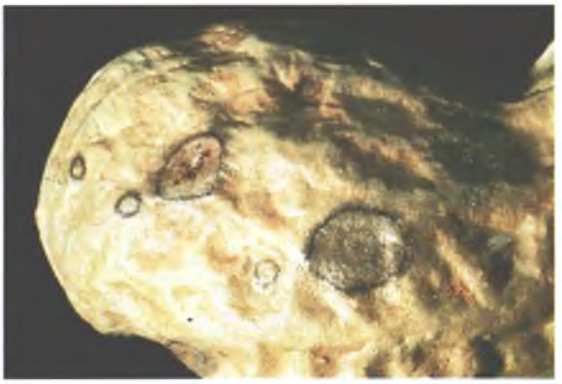
13. Foliar symptoms of charcoal rot. (Courtesy J. Damicone)

14. Taproot symptoms of charcoal rot. (Courtesy J. Damicone)

15. Field symptoms of Cylindrocladium black rot. (Courtesy P. Phipps)

16. Roots, pegs, and pods infected with *Cylindrocladium parasiticum.* (Courtesy P. Phipps)

17. Reddish orange perithecia of *Calonectria ilicicola* in a plant crown at the soil surface. (Courtesy P. Phipps)

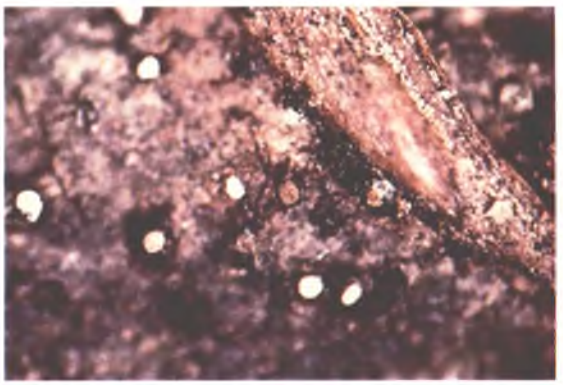


18. A cluster of delimited shell spots. (Courtesy Z. R. Frank)

19. Pods with primary spots and spots with concentric rings caused by secondary invaders. (Courtesy Z. R. Frank)

20. Field symptoms of Diplodia collar rot. (Courtesy D. M. Porter)

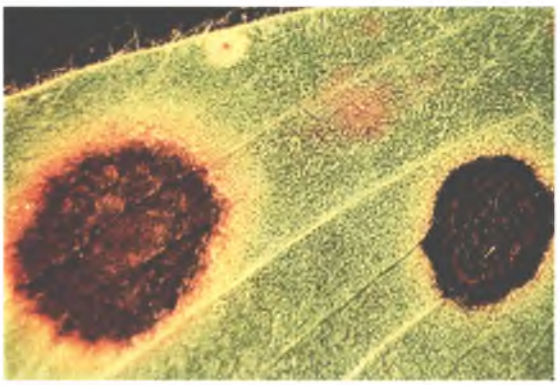
21. Symptoms of *Diplodia gossypina* on pods. (Courtesy D. M. Porter)



22. Pycnidia of *Diplodia gossypina* embedded in host tissue. (Courtesy D. M. Porter)

23. Conidia and conidiophores of *Diplodia gossypina*. (Courtesy D. M. Porter)

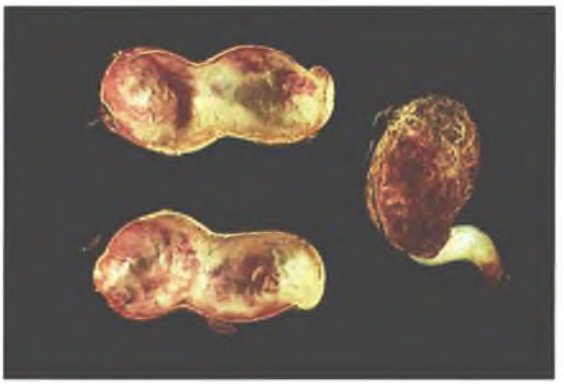
24. Early (brown lesions) and late (black lesions) leaf spots on a lower leaf surface. (Courtesy D. H. Smith)



25. Early (left) and late (right) leaf spot. (Courtesy D. H. Smith)

26. Aerial infrared photograph of fields with good leaf spot control (dark red, right) and defoliation caused by poor or no leaf spot control (brown, left). (Courtesy F. M. Shokes)

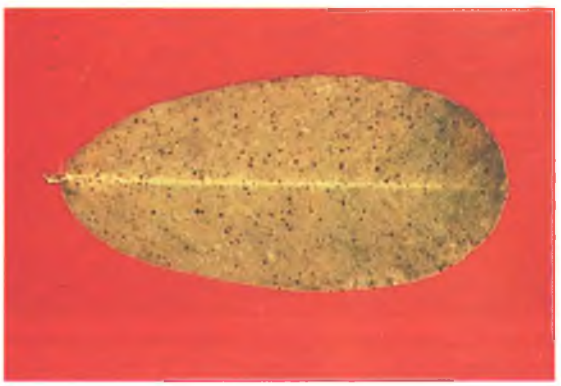
27. Sudden wilt caused by *Fusarium* sp. (Courtesy Z. Frank)



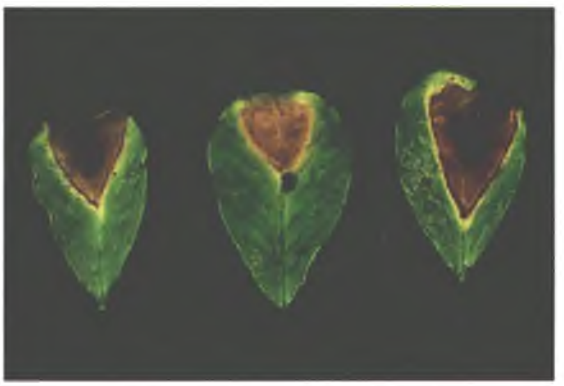
28. Pink pods caused by *Fusarium solani.* (Courtesy D. H. Smith)

29. Leaf blight of peanut caused by *Myrothecium roridum.* (Courtesy P. Subrahmanyam)

30. Symptoms caused by the pod rot complex. Several pathogens can be isolated from a single pod. (Courtesy A. S. Csinos)

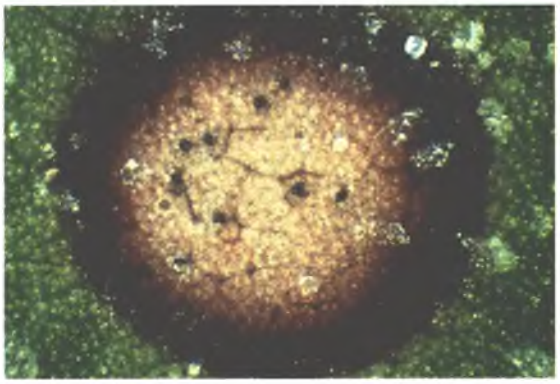


31. Pepper spot caused by *Leptosphaerulina crassiasca.* (Courtesy D. M. Porter)

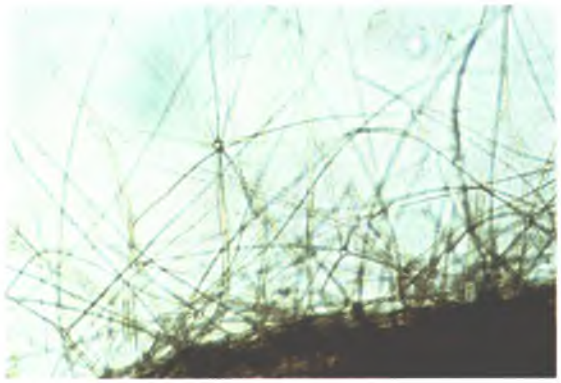


32. Leaf scorch caused by *Leptosphaerulina crassiasca.* (Courtesy D. H. Smith)

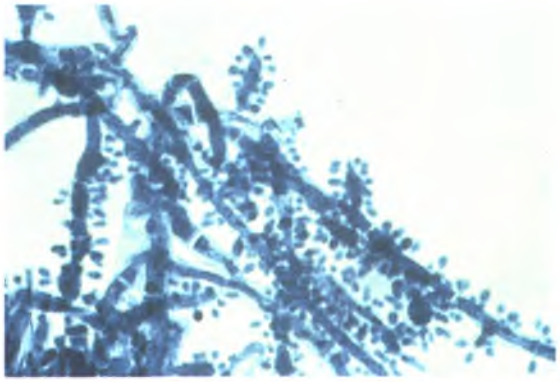
33. Phyllosticta leaf spot. (Courtesy D. H. Smith)



34. Phyllosticta leaf spot with pycnidia. (Courtesy D. H. Smith)



35. Acicular branching and hyphal strands of *Phymatotrichum omnivorum.* (Courtesy R. Taber)



36. Spores of *Phymatotrichum omnivorum.* (Courtesy R. Taber)

37. Pythium pod rot symptoms on virginia peanuts. (Courtesy D. M. Porter)

38. Wilt caused by *Pythium myriotylum.* (Courtesy D. M. Porter)

39. Young seedlings with sore shin caused by *Rhizoctonia solani* (right) and control (left). (Courtesy D. Bell)

40. Rhizoctonia lesions on young plants. (Courtesy D. M. Porter)

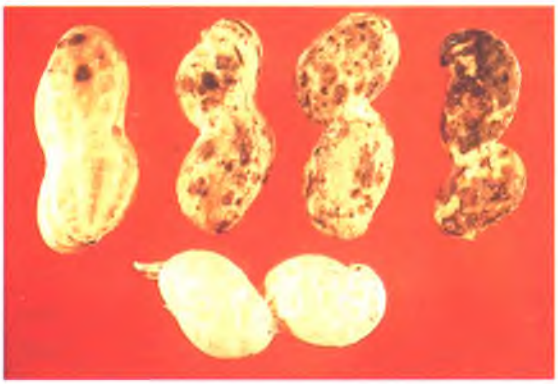
41. Stem lesions caused by *Rhizoctonia solani* AG-4. (Courtesy T. B. Brenneman)

43. Stem lesion caused by *Rhizoctonia solani* AG-4. (Courtesy T. B. Brenneman)

42. Rhizoctonia limb rot symptoms on plants damaged by tractor tires. (Courtesy T. B. Brenneman)

44. Severe Rhizoctonia limb rot symptoms and loss of pods near the taproot. (Courtesy T. B. Brenneman)

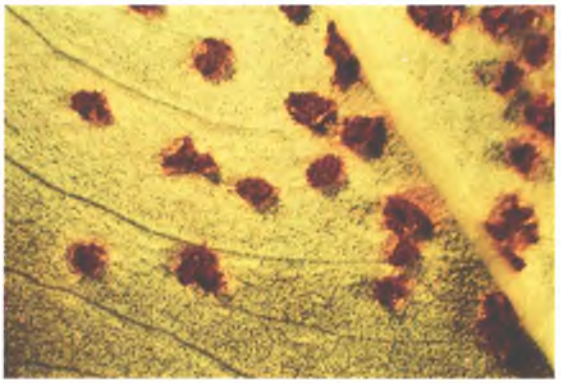
45. Peg damage on plants infected with *Rhizoctonia solani.* (Courtesy D. M. Porter)



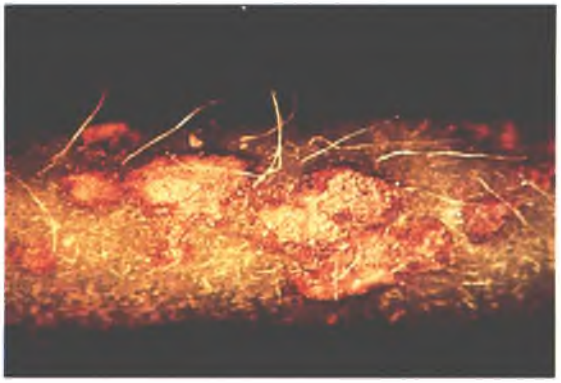
46. Pod rot caused by *Rhizoctonia solani.* (Courtesy D. M. Porter)

47. Symptoms of *Rhizoctonia solani* AG-4 infection of pigweed, a common weed. (Courtesy T. B. Brenneman)

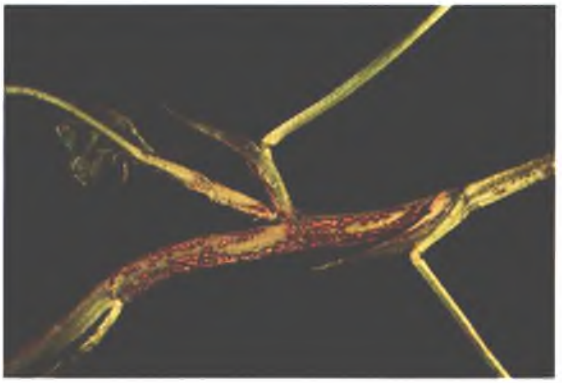
48. Rust pustules on a lower leaf surface. (Courtesy P. Subrahmanyam)



49. Rust pustules on a lower leaf surface. (Courtesy P. Subrahmanyam)



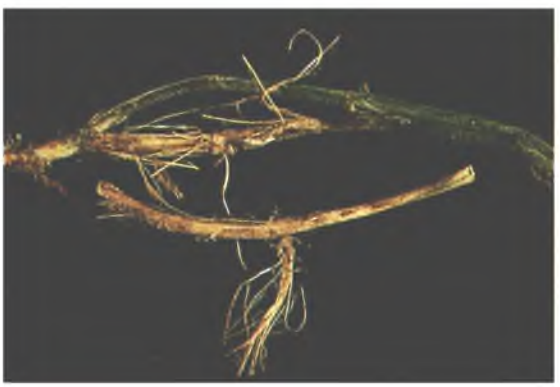
50. Symptoms caused by *Sphaceloma arachidis* on a stem. (Courtesy L. Giorda)



51. Scab symptoms caused by *Sphaceloma arachidis*. (Courtesy L. Giorda)

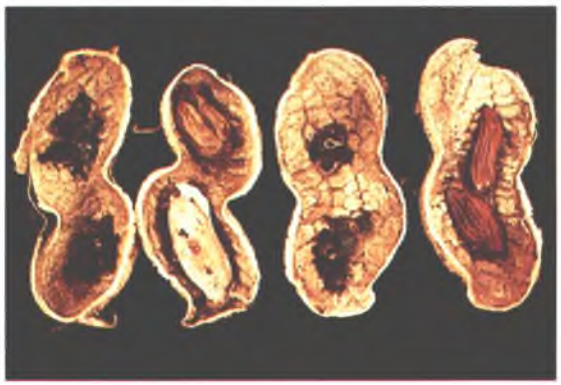
52. Field symptoms of Sclerotinia blight. (Courtesy D. M. Porter)

53. Fluffy, white mycelium of *Sclerotinia minor*. (Courtesy D. M. Porter)



54. Shredding of plant tissues caused by *Sclerotinia minor*. (Courtesy D. M. Porter)

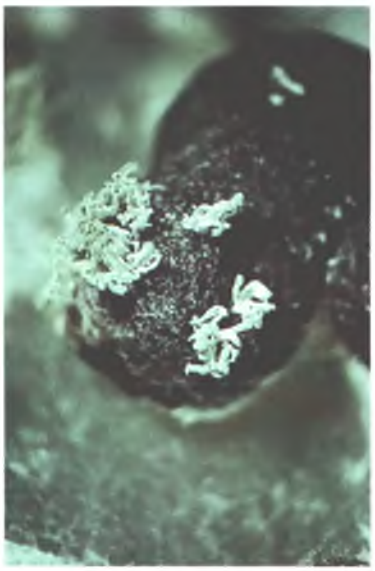
55. Sclerotia of *Sclerotinia minor* on infected branches. (Courtesy D. M. Porter)



56. Sclerotia of *Sclerotinia minor* in pods. (Courtesy D. M. Porter)

57. Apothecia from germinating sclerotia of *Sclerotinia minor*. (Courtesy D. M. Porter)

58. Infection cushion of *Sclerotinia minor* on peanut stems 18 hr after inoculation. (Courtesy H. A. Melouk)



59. Sclerotia of *Sclerotinia minor* colonized by *Trichoderma* sp. (Courtesy D. M. Porter)

60. Stem rot caused by *Sclerotium rolfsii* with actively growing, white mycelium. (Courtesy T. B. Brenneman)

61. Sclerotia of *Sclerotium rolfsii* on peanut stems and debris. (Courtesy D. M. Porter)

62. Active mycelium and young sclerotia of *Sclerotium rolfsii* on a single peanut peg. (Courtesy T. B. Brenneman)

63. Sclerotial initials on pods colonized by *Sclerotium rolfsii*. (Courtesy T. B. Brenneman)

64. Right, plants killed by early *Sclerotium rolfsii* infection and left, plants with active stem and pod rot. (Courtesy T. B. Brenneman)

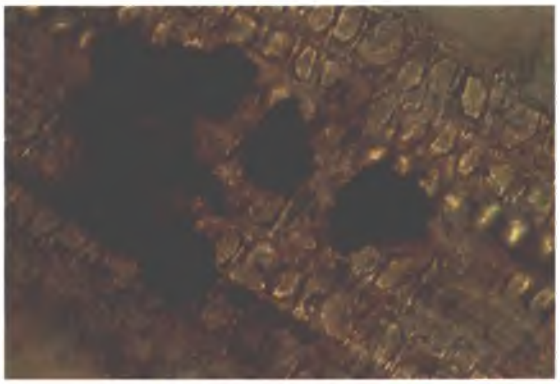
65. Inverted peanuts treated for stem rot with a fungicide (right) and untreated peanuts (left). (Courtesy T. B. Brenneman)

66. Chlorosis and marginal leaf necrosis in *Verticillium*-infected peanut plants. (Courtesy J. P. Damicone)

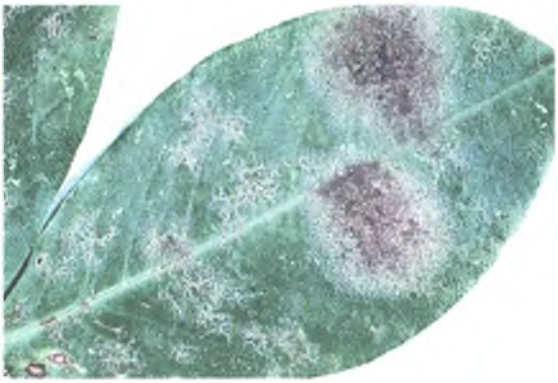
67. Marginal necrosis on leaves of plants infected with *Verticillium dahliae*. (Courtesy D. M. Porter)

68. Vascular discoloration of the taproot of a plant infected with *Verticillium dahliae*. (Courtesy D. M. Porter)

69. Brown discoloration of the vascular elements in leaf petioles from *Verticillium*-infected plants. (Courtesy J. P. Damicone)



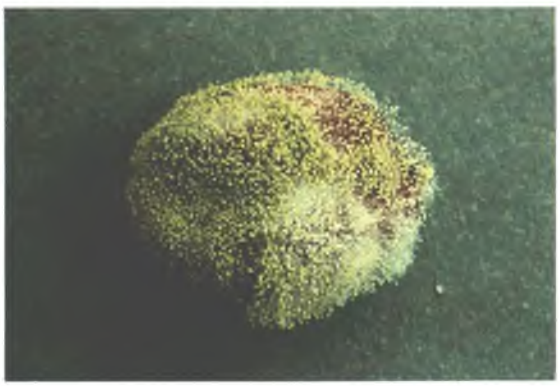
70. *Verticillium dahliae* microsclerotia in root tissue. (Courtesy H. Melouk)



71. Early web blotch symptoms on leaves. (Courtesy D. L. Cole)

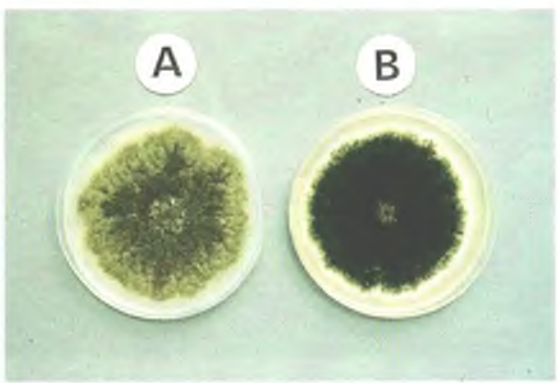
72. Symptoms of advanced web blotch on a leaf. (Courtesy D. L. Cole)

73. Plants exhibiting severe symptoms of web blotch. (Courtesy D. L. Cole)

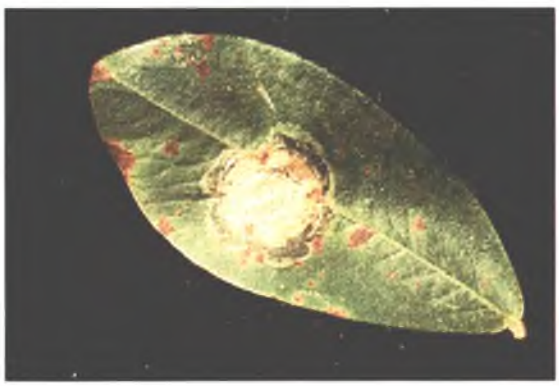


74. *Aspergillus flavus* sporulating on a peanut seed. (Courtesy B. Horn)

75. Concealed damage in peanut seeds that have been split in half to reveal sporulating *Aspergillus flavus*. (Courtesy B. Horn)



76. A, *Aspergillus flavus* and **B,** *A. parasiticus* on Czapek's agar medium after 7 days at 30°C. (Courtesy B. Horn)



77. Zonate leaf spot caused by *Cristulariella pyramidalis*. (Courtesy D. H. Smith)

78. Bacterial leaf spot of peanut caused by *Pseudomonas* sp. in India. (Courtesy P. Subrahmanyam)

79. Early symptoms (flagging) of bacterial wilt. (Courtesy D. M. Porter)

80. Dead branch of a plant infected with *Pseudomonas solanacearum*. (Courtesy D. M. Porter)

81. Advanced stage of bacterial wilt caused by *Pseudomonas solanacearum*. (Courtesy D. M. Porter)

82. Vascular discoloration of a taproot infected with *Pseudomonas solanacearum*. (Courtesy D. M. Porter)

83. Experimental plots with peanut cultivars resistant and susceptible to bacterial wilt. (Courtesy A. C. Hayward)

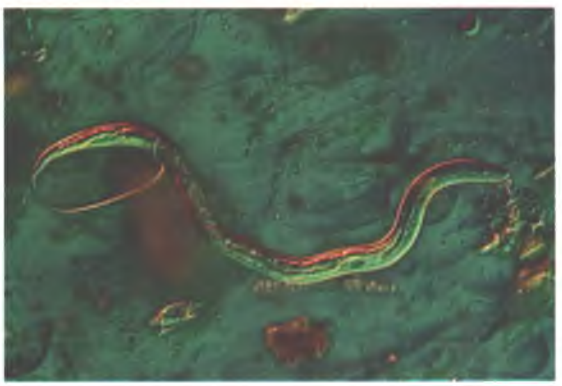
84. Roots and pods with galls caused by *Meloidogyne arenaria*. (Courtesy D. H. Smith)

85. Left, pods from untreated soil with galls or "warts" caused by *Meloidogyne arenaria* and right, pods from nematicide-treated soil. (Courtesy W. Horne)

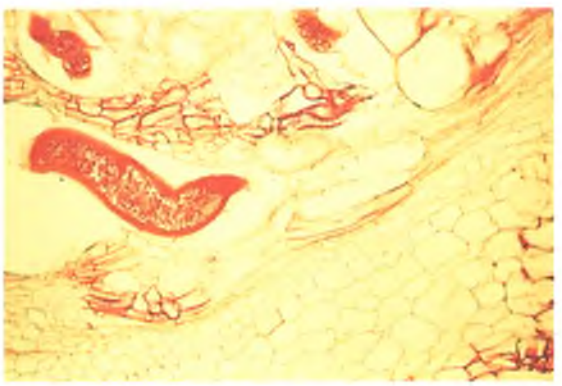
87. Left, plants growing in soil treated with a nematicide for control of *Meloidogyne arenaria* and right, plants in untreated soil. (Courtesy R. Rodríguez-Kábana)

86. Root damage caused by *Meloidogyne hapla*. (Courtesy R. Rodríguez-Kábana)

88. Eggs of *Meloidogyne arenaria*. (Courtesy N. Kokalis-Burelle)



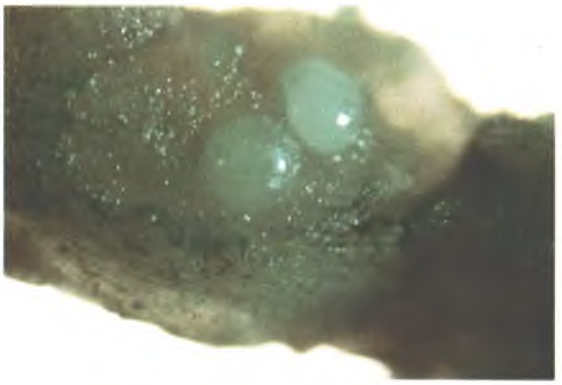
89. *Meloidogyne arenaria* juvenile emerging from an egg. (Courtesy N. Kokalis-Burelle)



90. Giant cells induced by female *Meloidogyne arenaria* in root tissue. (Courtesy W. Horne)

91. *Meloidogyne arenaria* male. (Courtesy N. Kokalis-Burelle)

92. Globose, pyriform females of *Meloidogyne arenaria* extracted from root tissue. (Courtesy N. Kokalis-Burelle)



93. Mature *Meloidogyne arenaria* females in root tissue. (Courtesy N. Kokalis-Burelle)

94. Symptoms caused by lesion nematodes on pods. (Courtesy J. L. Starr)

95. Stunting of plants in a peanut field infested with sting nematodes (*Belonolaimus longicaudatus*). (Courtesy A. Allison)

96. Root systems damaged by *Belonolaimus longicaudatus*. (Courtesy A. Allison)

97. Symptoms caused by *Ditylenchus africanus* on peanuts, including discoloration of the pod, discoloration of the seed testa, and premature seed germination. (Courtesy C. Venter)

98. *Ditylenchus africanus*, which feeds on peanut and on a wide variety of fungi, including *Penicillium* spp. (Courtesy C. Venter)

99. Chlorotic rings caused by the tomato spotted wilt virus. (Courtesy D. H. Smith)

100. Plants with flaccid, drooping petioles caused by the peanut bud necrosis virus. (Courtesy D. V. R. Reddy)

101. Chlorosis, leaf distortion, and line pattern caused by the tomato spotted wilt virus. (Courtesy D. H. Smith)

102. Left, healthy seeds and right, seeds infected with the tomato spotted wilt virus. (Courtesy D. H. Smith)

103. Left, healthy plants and right, plants infected with the Indian peanut clump virus. (Courtesy D. V. R. Reddy)

104. Mosaic mottling and chlorotic rings on plants infected with the peanut clump virus. (Courtesy D. V. R. Reddy)

105. Symptoms of chlorotic rosette. (Courtesy P. Subrahmanyam)

106. Chlorotic rosette symptom caused by the groundnut rosette virus. (Courtesy D. V. R. Reddy)

107. Plants infected with the mosaic rosette virus. (Courtesy D. V. R. Reddy)

109. Inward curling of leaf edges and interveinal depression caused by the peanut mottle virus. (Courtesy D. V. R. Reddy)

108. Mosaic symptoms caused by the peanut mottle virus. (Courtesy D. V. R. Reddy)

110. Stripes along the veins caused by the peanut stripe virus. (Courtesy J. W. Demski)

111. Green blotches caused by the peanut stripe virus. (Courtesy D. V. R. Reddy)

112. Mild mottle symptoms caused by the peanut stripe virus. (Courtesy J. W. Demski)

113. Plant infected with the peanut stunt virus. (Courtesy S. A. Tolin)

114. Stunted plants with leaflets showing rolled edges and necrosis induced by the cowpea mild mottle virus. (Courtesy D. V. R. Reddy)

115. Symptoms of chlorosis caused by the cucumber mosaic virus. (Courtesy Z. Xu)

116. Distorted and puckered leaflets with necrosis caused by the groundnut streak mosaic virus. (Courtesy P. Subrahmanyam)

117. Yellow spots caused by the peanut yellow spot virus. (Courtesy D. V. R. Reddy)

118. Symptoms of witches'-broom caused by a phytoplasma. (Courtesy D. V. R. Reddy)

119. Drought-stressed plants. (Courtesy D. H. Smith)

120. Symptoms of drought stress. (Courtesy D. M. Porter)

121. Frost injury. (Courtesy D. M. Porter)

122. Frost injury. (Courtesy D. M. Porter)

123. Variegated leaves, a genetic abnormality. (Courtesy D. M. Porter)

124. Variegated leaflet, a genetic abnormality. (Courtesy D. M. Porter)

125. Hail damage. (Courtesy R. Sturgeon)

126. Dicamba injury. (Courtesy J. Beasley)

127. Paraquat injury. (Courtesy J. Everest)

128. Chloroacetamide injury to seedlings. (Courtesy C. Swann)

129. Acifluorfen injury. (Courtesy G. Sullivan)

130. Dinitroaniline injury to seedlings. (Courtesy G. Sullivan)

131. 2,4-DB injury. (Courtesy G. Sullivan)

132. Injury caused by *s*-triazine. (Courtesy C. Swann)

133. Metribuzin injury. (Courtesy C. Swann)

134. Vernolate injury. (Courtesy C. Swann)

135. Bentazon injury. (Courtesy C. Swann)

136. Norflurazon injury. (Courtesy G. Sullivan)

137. Lightning injury. (Courtesy S. Thompson)

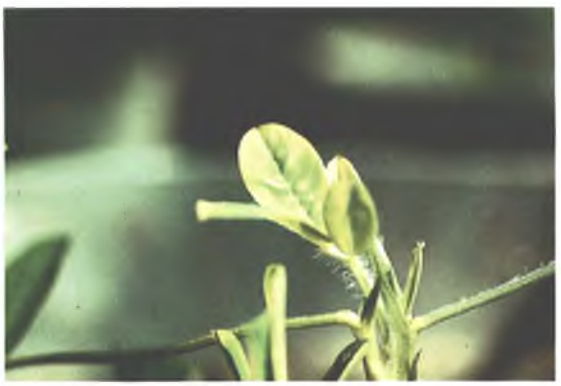
138. Nitrogen deficiency. (Courtesy R. Henning)

139. Calcium deficiency. (Courtesy A. Narayanan)

140. Sulfur deficiency. (Courtesy A. Narayanan)

141. Iron chlorosis. (Courtesy D. H. Smith)

142. Manganese deficiency. (Courtesy R. Henning)



143. Chlorotic leaves caused by zinc toxicity. (Courtesy R. Henning)

144. Plants stunted by zinc toxicity. (Courtesy P. Phipps)

145. Stem splitting caused by zinc toxicity. (Courtesy D. M. Porter)

146. Field symptoms of copper deficiency. (Courtesy L. Tripp)

147. Leaves with marginal chlorosis caused by copper deficiency. (Courtesy C. Gray)

148. Boron toxicity. (Courtesy M. Walker)

149. Leaf injury caused by ozone. (Courtesy A. Heagle)

150. Larva of the fall armyworm, *Spodoptera frugiperda*, on a peanut leaf. (Courtesy R. E. Lynch)

151. Beet armyworm, *Spodoptera exigua*, larva on a peanut leaf. (Courtesy S. Poe)

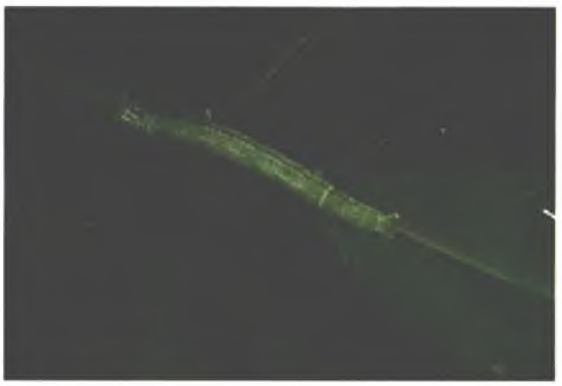
152. Adults of the beet armyworm, *Spodoptera exigua*. (Courtesy S. Poe)

153. *Spodoptera litura* larva feeding on a peanut leaf. (Courtesy ICRISAT)

154. Egg masses of the tobacco armyworm, *Spodoptera litura*, on a peanut leaf. (Courtesy ICRISAT)

155. Larva of the corn earworm, *Helicoverpa zea*, feeding on peanut. (Courtesy R. E. Lynch)

156. Larva of the gram pod borer, *Heliothis armigera*, feeding on a peanut leaf. (Courtesy ICRISAT)



157. Larva of the velvetbean caterpillar, *Anticarsia gemmatalis*, on peanut. (Courtesy S. Poe)

158. Larva of the groundnut leafminer, *Aproaerema modicella*, feeding on a peanut leaf. (Courtesy ICRISAT)

159. Larva of the red-headed hairy caterpillar, *Amsacta albistriga*, on peanut. (Courtesy ICRISAT)

160. Adult red-headed hairy caterpillar, *Amsacta albistriga*. (Courtesy ICRISAT)

161. Adult and cast nymphal skin of the potato leafhopper, *Empoasca fabae*. (Courtesy S. Poe)

162. Adult of the groundnut jassid, *Empoasca kerri*, on peanut. (Courtesy ICRISAT)

163. Hopper burn caused by potato leafhopper feeding. (Courtesy J. Stewart)

164. Immature and adult tobacco thrips, *Frankliniella fusca*. (Courtesy R. Howell)

165. Cupping and crinkling of peanut leaves caused by thrips injury. (Courtesy D. H. Smith)

166. Aphids feeding on peanut terminals. (Courtesy D. M. Porter)

167. *Aphis craccivora* feeding on peanut flowers. (Courtesy ICRISAT)

168. Damage to peanut caused by twospotted spider mites, *Tetranychus urticae*. (Courtesy J. C. Smith)

169. Adult of the sweetpotato whitefly, *Bemisia tabaci*, on peanut. (Courtesy R. E. Lynch)

170. Larva of the lesser cornstalk borer, *Elasmopalpus lignosellus*. (Courtesy T. P. Mack)

171. Female lesser cornstalk borer moth. (Courtesy T. P. Mack)

172. Male lesser cornstalk borer moth. (Courtesy T. P. Mack)

173. Peanut pod externally scarified by the lesser cornstalk borer. (Courtesy R. E. Lynch)

174. Southern corn rootworm adult, the twelve-spotted cucumber beetle. (Courtesy J. C. Smith)

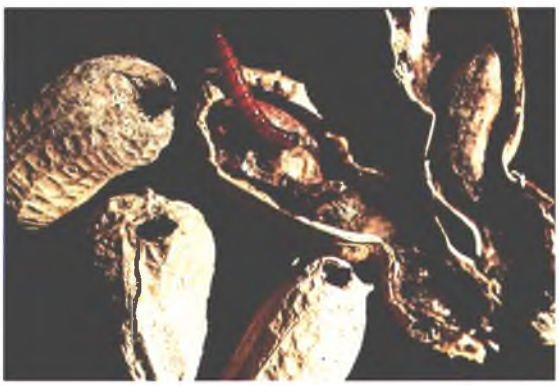
175. Larva of the southern corn rootworm, *Diabrotica undecimpunctata howardi.* (Courtesy R. E. Lynch)

176. Adult beetles, *Lachnosterna serrata,* an important white grub pest of peanut. (Courtesy ICRISAT)

177. Larva or white grub of *Lachnosterna serrata.* (Courtesy ICRISAT)

178. Termites, important pests of peanut in Africa and Asia. (Courtesy ICRISAT)

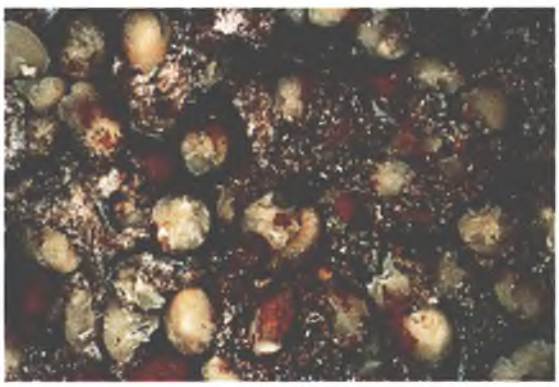
179. Peanut pods externally scarified by termites. (Courtesy ICRISAT)



180. Wireworm larva and damage to a peanut pod. (Courtesy R. Howell)

181. Adult Indianmeal moth, *Plodia interpunctella*. (Courtesy L. Zettler)

182. Adult rice moth, *Corcyra cephalonica*. (Courtesy ICRISAT)



183. Larva and damage caused by the rice moth, *Corcyra cephalonica*. (Courtesy ICRISAT)

184. Adults and larvae of the red flour beetle, *Tribolium castaneum*. (Courtesy L. Zettler)

185. Larva of the red flour beetle, *Tribolium castaneum*. (Courtesy L. Zettler)

186. Larva of the groundnut bruchid, *Caryedon serratus*, emerging from a peanut pod. (Courtesy ICRISAT)

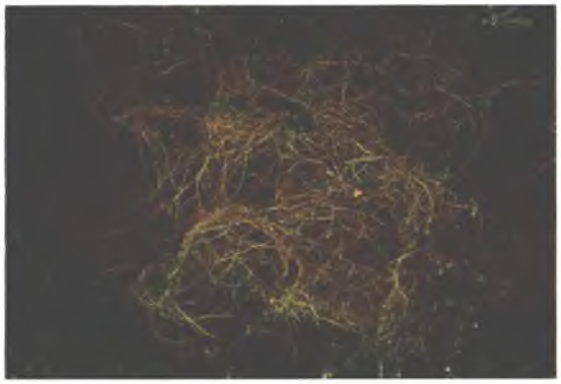
187. The lygaeid bug *Elasmolomus sordidus*, a pest of stored peanut. (Courtesy ICRISAT)

188. *Alectra vogelii*, a root parasite of peanut. (Courtesy P. Subrahmanyam)

189. The connection between *Alectra vogelii* and the peanut roots. (Courtesy D. H. Smith)

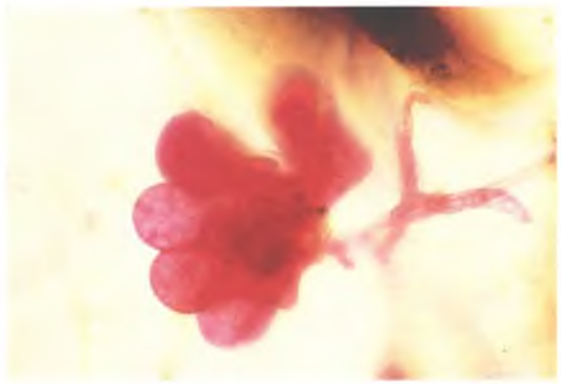
190. *Striga hermontheca*, a root parasite of peanut in West Africa. (Courtesy K. V. Ramaiah)

191. *Striga gesnarioides*, a parasite of peanut in Mozambique. (Courtesy D. R. Butler)



192. Dodder (*Cuscuta campestris*), a parasite of peanut in the United States. (Courtesy D. H. Smith)

193. Azygospore of *Gigaspora* sp. (Courtesy D. M. Porter)

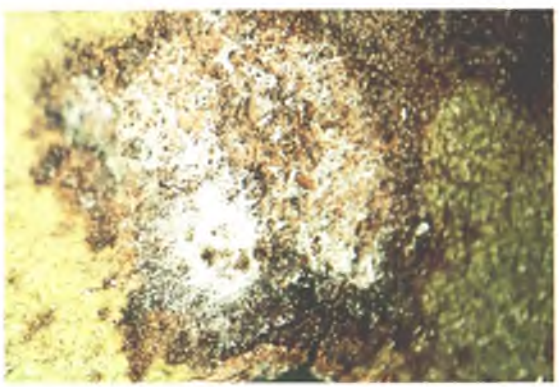


194. Extramatrical vesicles of *Gigaspora* sp. (Courtesy D. M. Porter)

195. Roots of two peanut cultivars nodulated by *Rhizobium* sp. (Courtesy J. Wynne)

196. Plants inoculated with an effective *Rhizobium* strain (NC 123), uninoculated plants, plants that received nitrogen fertilizer, and plants inoculated with an ineffective *Rhizobium* strain (TAL 420). (Courtesy J. Wynne)

197. Agar imprint of the abaxial surface of a field-collected peanut leaf colonized by a variety of bacteria (nutrient agar contained chlorothalonil to inhibit fungal growth). (Courtesy H. W. Spurr)



198. Leaf spot caused by *Cercosporidium personatum* colonized by *Dicyma* sp. (Courtesy D. M. Porter)

Diseases Caused by Bacteria

Bacterial Leaf Spot

Bacterial leaf spot of peanut, caused by an unidentified species of *Pseudomonas*, has been observed in India, Vietnam, and Zimbabwe.

Symptoms

Lesions, which are small, circular to irregular, and light brown, frequently occur on the lower leaves of young plants. At early stages of disease development, the lesions are water soaked and prominent on upper surfaces of leaflets. On the lower surfaces of the leaflets, lesions become visible only after the spots on the upper surfaces are well developed. The lesions enlarge, become irregular, and may develop chlorotic halos. When lesions are fully developed, their centers are light brown with dark brown margins (Plate 78). Under favorable conditions for disease development, the lesions coalesce, and the leaflets become chlorotic and shed prematurely.

Bacterial colonies grown on D4 medium are pale white, circular, raised, and 1–2 mm in diameter. The bacterium (0.5–0.8 × 1.0–1.3 μm) is gram negative, nonfluorescent on King's medium, and rod shaped and has one or two polar flagella.

Selected References

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Raghava Reddy, J., McDonald, D., and Joshi, N. C. 1989. Bacterial diseases of ICRISAT legume crops and methods in plant bacteriology. Pages 37-55 in: Rep. Int. Legumes Pathol. Training Course, 8th. Int. Crops Res. Inst. Semi-Arid Trop. Legumes Pathol. Progr. Rep. 10.

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(Prepared by P. Subrahmanyam)

Bacterial Wilt

Bacterial wilt of peanut, caused by *Pseudomonas solanacearum,* was first reported in Indonesia in 1905 and since then has been reported in many regions throughout the world. The first report of the disease in the United States was in 1915 from Granville County, North Carolina, which had previously experienced a high incidence of bacterial wilt of tobacco. Bacterial wilt of peanut was responsible for heavy losses in Georgia in 1913 but is now of minor importance in the United States. By contrast, the disease is a major constraint to peanut production in several Asian and African countries, notably China, Indonesia, Malaysia, Uganda, and Vietnam.

In Indonesia, bacterial wilt of peanut is most severe in western Java, southern Sulawesi, and southern Sumatra and is also important in central and eastern Java, Bali, and North Sulawesi. In China, the disease is most severe in 16 southern and central provinces, where it is estimated that more than 200,000 ha of peanut fields are infested with the wilt pathogen. The annual incidence of the disease is estimated to be 4–8% on resistant cultivars and 10–30% on susceptible ones in some regions, and the annual loss in pod yield caused by the disease is estimated to be approximately 36,000 t. The disease is widespread in the major peanut-growing areas of both northern and southern Vietnam and is most severe in dryland cropping systems, especially on sandy upland or riverbank soils. Peanut is probably less susceptible to bacterial wilt than solanaceous hosts such as tomato, potato, tobacco, and eggplant, except where peanut is intensely cropped under environmental conditions conducive to the disease and highly virulent strains of *P. solanacearum* occur. Bacterial wilt is regarded as a potential threat to peanut production in several warm, humid areas of the world as production expands into new areas or cultural practices change.

Symptoms

Infection of young plants can result in the sudden wilting of stems and foliage, although leaves on dead plants remain green (Plate 79). Wilt symptoms can be observed 2–3 weeks after sowing. The first sign of the disease is a slight drooping or curling of one or more leaves. In mature plants or in cultivars that are not highly susceptible, a gradual decline causes the foliage to turn yellow. Wilt and death of single branches (Plate 80) or of the entire plant (Plate 81) may follow. Alternatively, the plant may show signs of recovery. The root systems of infected plants display numerous discolored and dead roots. Dying branches often curl to form a "shepherd's crook." Diagnostic characteristics for this disease are a dark discoloration in the xylem and pith (Plate 82) and a milky white ooze composed of masses of bacteria that exudes from cut ends of stems placed in water.

Causal Organism

P. solanacearum (Smith) Smith (syns. *Burkholderia solanacearum* (Yabuuchi et al.) and *Ralstonia solanacearum* (Yabuuchi et al.)) is an aerobic, gram-negative, rod-shaped bacterium that does not form spores and accumulates poly-β-hydroxybutyrate as a carbon reserve. Although the bacterium does not produce fluorescent pigments, it can produce a brown, diffusible pigment on agar media containing tyrosine. The organism does not grow at 41°C, and it cannot utilize arginine and betaine as sole carbon sources. The bacterium is unable to hydrolyze starch, and gelatin is liquefied weakly or not at all. There is variation in nitrate metabolism. Asian isolates from peanut produce gas from nitrate, whereas those from the Americas reduce nitrate to nitrite but without gas production. The optimum temperature for growth is 30–35°C.

P. solanacearum is heterogeneous in phenotypic properties, such as ability to utilize specific carbon sources, and has been classified into five biovars on the basis of differences in oxidation of particular hexose alcohols and disaccharides. Biovars 1, 3, and 4 have been reported as pathogens of peanut. In the United States, the disease is caused by biovar 1, whereas in those Asian and African countries for which there is published information, it is caused by biovar 3 or 4. On the basis of hosts of origin and host range, isolates of *P. solanacearum* have been tentatively divided into five races. The isolates from peanut are identified as race 1.

The classification of strains of *P. solanacearum* has been greatly advanced by DNA analysis. Restriction fragment length polymorphism (RFLP) analysis has been used to differentiate *P. solanacearum* into RFLP groups by using nine probes to regions of the chromosomal DNA associated with virulence and the hypersensitive response. Similarity coefficients for all

pairwise combinations of RFLP groups show two major divisions into which biovars and races can be placed (Table 4). The same two divisions, one including biovars 1 and 2 and the other biovars 3, 4, and 5, are also obtained by sequencing of the approximately 1,540 nucleotides in the 16S rRNA genes.

The present nomenclature does not reflect this fundamental difference at the genetic level. It is conceivable that divisions I and II represent subspecies of *P. solanacearum*. It is of quarantine significance that strains pathogenic to peanut are found in both divisions, biovar 1 of division II in the southeastern United States and biovars 3 and 4 of division I in Asia and Africa (Table 5). Division II may have originated in the Americas and division I in Asia.

In culture, *P. solanacearum* undergoes spontaneous mutation involving multiple changes in phenotype; this phenomenon has become known as phenotype conversion. These pleiotropic mutants show reduced extracellular polysaccharide production and endoglucanase activity and virulence and increased motility. Highly motile, avirulent forms can rapidly dominate the virulent form, especially in unaerated liquid media. Although virulent forms are usually nonmotile, they may possess single polar flagella, which characteristically are straight rather than wavy. Avirulent and virulent strains can be differentiated by growing the organism on a tetrazolium medium. Colonies of virulent strains are fluidal and irregular in shape and have white to pink centers that darken with age. Avirulent colonies are round, butyrous, and uniformly red, even at early stages of growth.

Maintenance of virulence in culture requires continuous selection of the fluidal wild-type colony form from turbid suspensions kept at 15–20°C or at room temperature (about 25°C). Unselected cultures rapidly become dominated by the butyrous colony form, giving rise to many reports in the literature of loss of virulence by *P. solanacearum* in culture.

In Indonesia and China, most of the peanut isolates belong to biovar 3, which is more virulent to peanut than biovar 1 occurring in the United States. Peanut isolates are reported to be more virulent on peanut than are isolates from other host crops. Strains of *P. solanacearum* differ greatly in their virulence on peanut. In China, seven pathotypes have been identified on the basis of their pathogenicity on six indicator cultivars having different levels of wilt resistance. All six cultivars were susceptible to pathotype 7 and moderately resistant to pathotype 6. Strains from southern China are generally more virulent to peanut than those from northern regions. Differences in virulence among isolates from peanut have been observed in Indonesia, but the degree of specialization to different cultivars does not warrant designation of pathotypes.

TABLE 4. Classification of *Pseudomonas solanacearum*[a]

	Division I	Division II
Biovars[b]	3, 4, and 5	1 and 2
Number of groups	16	12
Similarity coefficient (%)		
Within division	78 ± 9	62 ± 19
Between divisions	13.5	

[a] Based on restriction fragment length polymorphism involving nine DNA probes to chromosomal DNA into 28 groups.
[b] Representatives of race 1 (Cook et al., 1989) are found in divisions I and II, while races 2 and 3 are found in division II.

TABLE 5. Classification of strains of *Pseudomonas solanacearum* from peanut

		RFLP[a] Analysis	
Country	Biovar	Division	Groups
Indonesia	3	I	ND[b]
Papua New Guinea	3	I	ND
Philippines	3	I	ND
People's Republic of China	3	I	ND
	4	I	11 and 17
Sri Lanka	3	I	ND
Uganda	3	I	ND
	4	I	ND
United States	1	II	ND

[a] Restriction fragment length polymorphism.
[b] No data available.

Disease Cycle

Bacterial wilt is a soilborne disease. Continuous cropping of a susceptible host or weed hosts will favor long-term survival of the pathogen. Some of the most common weed hosts in peanut fields in Asia are *Ageratum conyzoides* L., *Crassocephalum crepidioides* (Benth.) S. Moore, *Crotalaria juncea* L., and *Croton hirtus* L. Infected crop residues also serve as primary sources of inoculum.

Bacterial wilt is profoundly influenced by environmental conditions, particularly soil temperature and moisture content. High levels of soil moisture not only aid in the survival of the bacterium, but also increase bacterial dissemination and disease development. Invasion of peanut occurs through wounds or natural openings in the roots followed by invasion of the water-conducting tissues, bacterial multiplication, blockage, and interference with water transport. Root injury by soil insects and nematodes provides points of entry for the bacterium and may increase disease severity. Soil temperatures above 25°C (at a soil depth of 5 cm) and high soil moisture favor the development of bacterial wilt. The disease is exacerbated by soil temperatures greater than 30°C (air temperatures greater than 25°C) for 10 days. Optimum temperatures for wilt development are 28–33°C. In greenhouse studies in Australia with biovar 3, wilt severity was most pronounced under diurnal temperature regimes of 35/30°C and 30/25°C and was slight or absent under regimes of 25/20°C and 20/15°C.

Reports on the incidence and severity of bacterial wilt in different soil types are contradictory. In Indonesia, the disease has been most prevalent and severe in clay soils; in China, it is predominant in sandy soils and relatively unknown in clay or loam soils. The disease also occurs in red lateritic soils. In Malaysia, the disease was shown to be more severe in clay soil than in two types of sandy soils at the same moisture level. Disease severity increased significantly with an increase in moisture for each of three different soil types. Continuous planting of susceptible cultivars in wet soils leads to a rapid buildup of inoculum.

Studies in China, Indonesia, Malaysia, and Vietnam have shown that seed transmission occurs at a rate of 4–15%, the highest rates occurring with freshly harvested seed. Infected seed is a potential source of primary inoculum, especially for disease-free areas. However, there is rapid loss of viability of the bacterium as peanut seed dry to a moisture content below 9%. In areas where well-dried seed is used, seedborne infection is unlikely to occur.

Control

Crop rotations are effective in reducing losses caused by bacterial wilt but do not give complete control of the disease. Rotations of peanut with crops that are immune or highly resistant to *P. solanacearum* and with nonhost crops such as rice, corn, soybean, and sugarcane are effective measures. Rotation of peanut with rice for 1–2 years or with other nonhost crops such as sugarcane for 2–5 years is the most effective cultural control measure. Other cultural control measures include flooding peanut fields for 15–30 days before sowing and improving soil drainage. Sowing dates can be adjusted to avoid periods of high temperature or ample soil moisture, conditions that favor bacterial infection and disease development. For example, in the Hubei Province of China, early sowing in mid-April results

in much lower incidence and severity of bacterial wilt than sowing in June. Other measures include burning of crop residues, removal of weeds, and cleaning of farm tools after operations in infested fields. Chemical control measures have not proved successful. In view of the potential for seedborne transmission, seed movement should be strictly controlled to avoid spreading the pathogen to disease-free areas.

The use of resistant cultivars is the most effective and practical method to control bacterial wilt. Peanut is the first crop in which resistance has been successfully used against *P. solanacearum* (Plate 83). The high level of resistance in the cultivar Schwarz 21, first identified in Indonesia, has held up for more than 50 years, and strains capable of overcoming this resistance have not evolved. A series of resistant cultivars has been released and used in production in China since the 1980s.

Field screening under uniformly high disease pressure in wilt-infested plots at Cikeumeuh, western Java; Jambegede, eastern Java; Indonesia; and Hong An, Hubei Province, China, has proved to be a useful way to identify sources of resistance. Wilt-resistant lines have approximately 80–95% plant survival compared with less than 10% survival in susceptible cultivars in infested fields. Lines showing wilt incidence of less than 10% are considered highly resistant and those with 10–20% incidence moderately resistant. Host-pathogen-environment interactions may be responsible for the variability in disease reactions of some lines at different locations. For example, the resistant Indonesian cultivars Gajah, Kidang, Macan, and Banteng were only moderately resistant in some areas of China.

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(Prepared by A. C. Hayward and L. Y. He)

Diseases Caused by Nematodes

Root-Knot Nematodes

Root-knot nematodes (*Meloidogyne* spp.) are the nematodes causing the greatest yield losses in peanut. The conspicuous root "galls" and "warts" caused by these nematodes resulted in their early recognition as serious pathogens. Yield losses in fields infested with the peanut root-knot nematode range from 20 to 90%. Root-knot nematodes can limit development of *Rhizobium* nodules and aggravate damage caused by other soilborne plant pathogens. In most fields of the southeastern United States, continuous culture of the peanut results in increased damage from root-knot nematodes and associated pathogens.

Symptoms

Peanut plants infected with the root-knot nematode commonly develop enlarged roots and pegs, which appear as galls of various sizes. The galls result from an internal swelling of the roots and can be distinguished from *Rhizobium* nodules, which are appended to the side of the root. Galls may attain a diameter several times that of the adjacent root. Pegs and pods also become infected and develop galls (Fig. 58). Galls on roots, pegs, and pods (Plates 84 and 85) sometimes begin to deteriorate by the time of crop maturity. Development of the root system is often significantly reduced.

Symptoms of damage caused by the northern root-knot nematode (*M. hapla*) are similar to those caused by the peanut root-knot nematode (*M. arenaria*). Roots, pegs, and pods become galled by both species, but individual galls caused by *M. hapla* are smaller than those caused by *M. arenaria* (Fig. 59). Roots infected with *M. hapla* often form branches near the point of nematode invasion, producing a dense, bushy root system (Plate 86).

Peanut plants infected with root-knot nematodes may show various degrees of stunting and chlorosis. Severely infected runner-type cultivars typically show limited growth, resulting in poor ground coverage at midseason (Plate 87). Root growth is restricted and the vascular elements of infected tissues are

Fig. 58. Pod galls caused by *Meloidogyne arenaria*. (Courtesy K. Garren)

disrupted, resulting in poor water and nutrient transport. Infected plants show a tendency to wilt during periods of water stress.

Damage from *M. arenaria* is aggravated by stem rot (caused by *Sclerotium rolfsii*) and pod and peg rots. In some fields, there is a close association between densities of *M. arenaria* juveniles in soil and incidence of stem rot (Fig. 60).

Causal Organisms

The principal species of root-knot nematodes attacking peanuts are the peanut root-knot nematode (*M. arenaria* (Neal) Chitwood) and the northern root-knot nematode (*M. hapla* Chitwood). Two races of *M. arenaria* are morphologically indistinct and are separated on the basis of ability to infect and reproduce on Florunner peanut (race 1) or the failure to do so (race 2). In addition, the javanese root-knot nematode (*M. javanica* (Treub.) Chitwood) has been reported to attack peanut and produce symptoms similar to those of *M. arenaria.*

M. arenaria, M. hapla, and *M. javanica* are distributed worldwide. The area between 35°S and 35°N latitudes is widely infested by three species of *Meloidogyne* adapted to continuous existence in warm climates: *M. arenaria, M. incognita* (Kofoid & White), and *M. javanica.* The most common species of root-knot nematode north of 35° latitude in the northern hemisphere is *M. hapla.*

Root-knot nematodes exist in the soil as egg masses, infective second-stage juveniles, and adult males. Individual eggs are elongate and ovate (30–60 × 75–113 µm) (Plate 88). The infective second-stage juveniles emerge from the eggs and move freely through the soil (Plate 89). Juveniles are slender (430–470 µm long) with a stylet length of 10 µm. The infective juveniles penetrate roots, pegs, or pods and move intercellularly and intracellularly to a region near the vascular tissue. There they lose their mobility and begin to feed on adjacent plant cells. Giant cells are induced by feeding (Plate 90). Under favorable conditions, the sedentary juveniles swell and develop into males (Plate 91) or enlarged (1–2 mm long), white, globose-pyriform, mature females (Plate 92), which produce large numbers of eggs (200–1,500 per female) in a gelatinous matrix. These egg masses may remain in the roots (Plate 93) or be extruded into the soil (Fig. 61). After eggs hatch, the new second-stage juveniles enter the soil surrounding the roots, completing the life cycle. The cycle is influenced significantly by soil temperature and moisture, but under normal conditions, two or more cycles may occur during each season. Crop debris containing galls may be spread by farming operations or running water, producing widely distributed infestation sites.

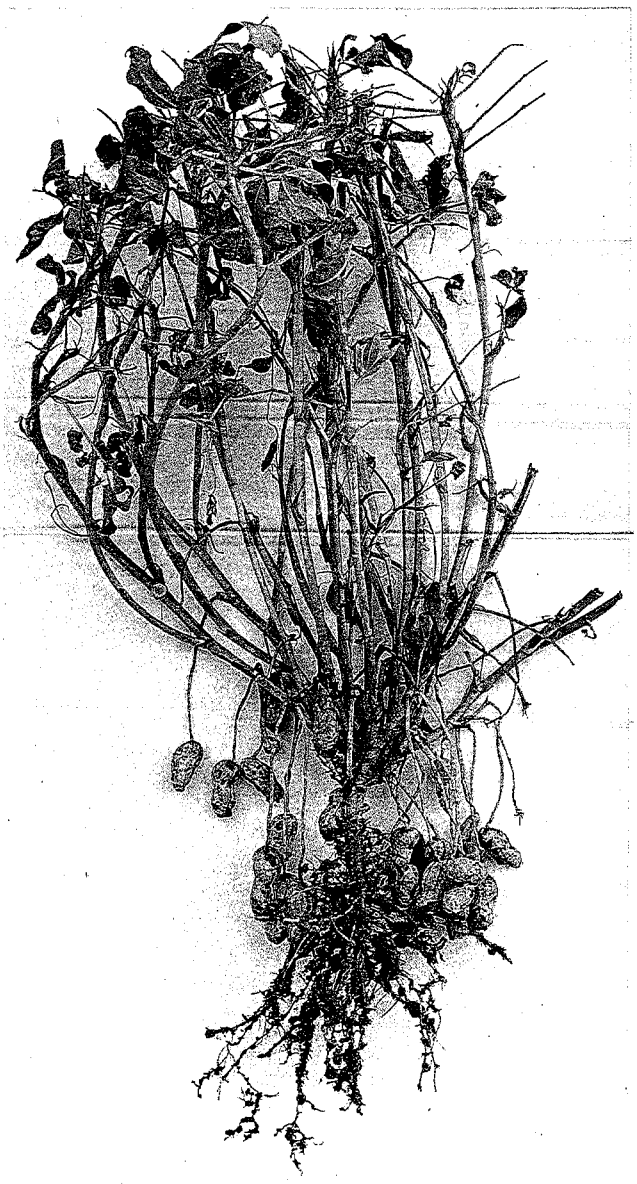

Fig. 59. Pod and root galls caused by *Meloidogyne arenaria.* (Courtesy K. Garren)

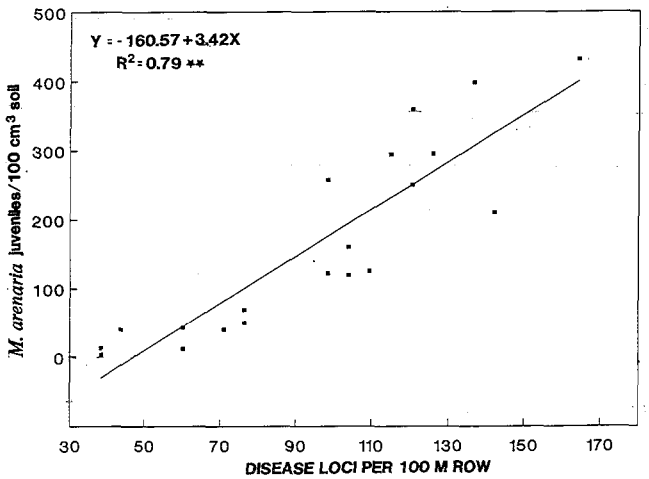

Fig. 60. Relationship between incidence of stem rot, caused by *Sclerotium rolfsii*, and soil population densities of *Meloidogyne arenaria* juveniles determined near harvest in Alabama.

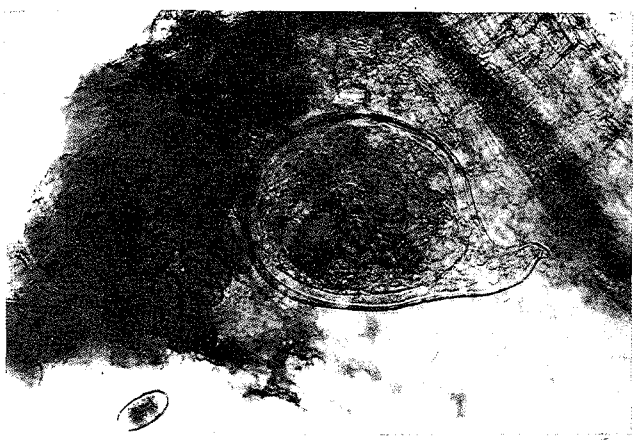

Fig. 61. *Meloidogyne arenaria* female and egg mass in root tissue. (Copyright J. Eisenback, used by permission)

Root-knot nematodes can be distributed in a field through their own movement but remain very localized.

Choosing the correct sampling time for nematode analysis is critical for accurate diagnosis and selection of control measures. Identification of *Meloidogyne* spp. is based on perineal patterns (Fig. 62), differential host responses, size of juveniles, male morphology, and other characteristics. More recently, molecular techniques such as polymerase chain reaction and DNA fingerprinting have been used successfully to identify species of these nematodes. Population levels of root-knot nematode juveniles in the soil vary widely throughout the year. In Alabama and other regions in the southeastern United States, the number of juveniles of *M. arenaria* in soil increases through the growing season (April–September), reaching a maximum (more than 200/100 cm^3 of soil) in late July to September and a minimum (fewer than 20/100 cm^3 of soil) between January and early June. The up-rooting of peanut plants causes a steep decline in the density of juveniles in the soil after harvest. Typically, numbers of juveniles during March and April are less than 10% of those observed at harvest the previous year. Numbers of nematodes also change significantly in relation to soil depth (Fig. 63). In Alabama, significant numbers of juveniles are present throughout the year at a depth of 15–30 cm. In Florida, gelatinous egg masses of *M. arenaria* are present at soil depths of 0–75 cm from August through October; however, egg masses recovered from November (1 month after harvest) to July contained no viable eggs, suggesting that *M. arenaria* probably overwinters as second-stage juveniles. These observations indicate that the probability of detecting root-knot nematodes in any peanut field will be greater if samples are taken during the last 2 months of the growing season. This practice, while not helpful for the current season, permits the establishment of expected levels of infestation for the following season. Samples collected during the winter or early spring (off-season) invariably contain very low numbers of plant-parasitic nematodes and require some form of bioassay to establish the level of infestation with root-knot nematodes.

Fig. 62. Perineal pattern of adult *Meloidogyne arenaria* female. (Courtesy J. Eisenback)

Control

Ideally, control of parasitic nematodes should be based primarily on the use of resistant cultivars, alone or in combination with proper rotational crops and cultural techniques that reduce soil infestation to a tolerable level. Presently, no commercially available cultivars are resistant to root-knot nematodes; however, a number of selections from crosses of *Arachis hypogaea* with wild-type peanut species (e.g., *A. glabrata*) are resistant to *M. arenaria* and have acceptable agronomic characteristics. There are significant differences in susceptibility to *M. arenaria* and *M. hapla* among peanut cultivars. Also, for equally susceptible peanut cultivars, those with late maturity dates sustain greater populations of *M. arenaria* than those with earlier maturity dates.

Continuous culture results in a gradual and steady decline in yield (Fig. 64). Yield losses of 100–150 kg/ha can be expected for each year of continuous culture. This trend in yield decline in response to continuous culture cannot usually be reversed with application of available nonfumigant nematicides. Rotation of peanut with other crops can significantly decrease levels of infestation of root-knot nematodes in soils. Rotations of peanut with cotton, corn, sorghum, and some soybean cultivars are effective in controlling or suppressing *M. arenaria* or *M. hapla*. Although corn and sorghum serve as hosts for *M. arenaria*, the parasite does not develop as well on these crops

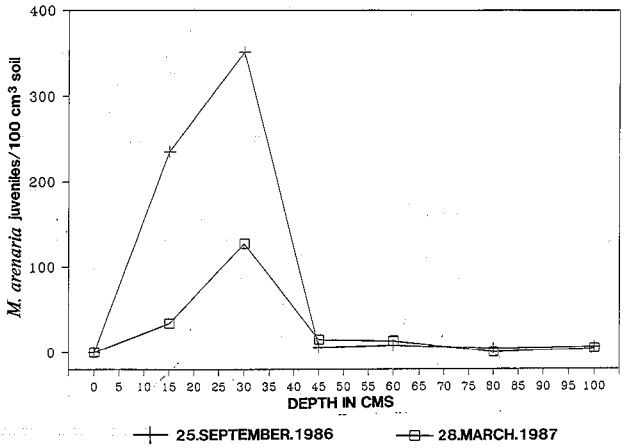

Fig. 63. Relationship between soil depth and *Meloidogyne arenaria* juvenile population density in an Alabama field at harvest (September 1986) and off-season after winter fallow (March 1987).

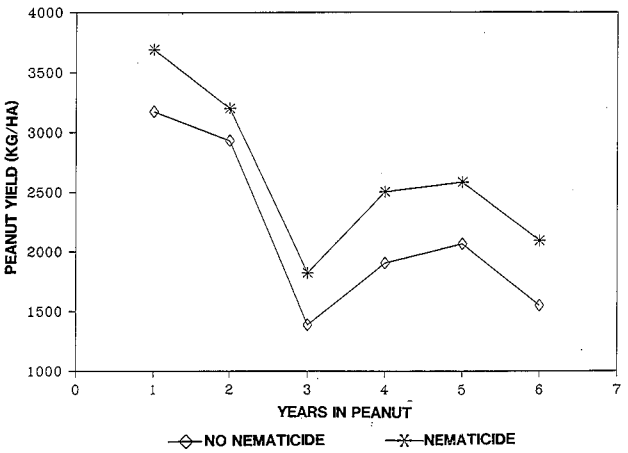

Fig. 64. Effects of number of years of continuous culture of peanut on yield in the southeastern United States.

as on the peanut plant, so soil infestation can be reduced. The reaction of individual cultivars to the nematode should be determined before they are used as rotational crops. Cotton is an excellent rotational crop for the management of *M. arenaria* in peanut since it is not a host to the nematode and its use results in significant reductions in the incidence of root knot and stem rot (*S. rolfsii*). Rotations of peanut with Bahia grass (*Paspalum notatum*) or velvet bean (*Mucuna deeringiana*) are also very effective for the management of root knot and associated diseases such as stem rot and pod rots. Other effective rotational crops are castor bean (*Ricinus communis*), sesame (*Sesamum indicum*), American joint-vetch (*Aeschynomene americana*), partridge pea (*Cassia fasciculata*), and hairy indigo (*Indigophora hirsuta*). Choice of a rotational crop is dependent on the economics and logistics of the production system.

Nematodes are attacked by a wide variety of microorganisms and invertebrates including viruses, bacteria, fungi, other nematodes, protozoa, mites, and insects. *Pasteuria penetrans*, a bacterial parasite of nematodes present in many peanut fields, has shown promise as a biological control agent against several plant-parasitic nematodes.

Two types of nematicides are widely used in peanut production: fumigants and nonfumigants with contact or systemic properties. The fumigants dibromochloropropane (DBCP) and ethylene dibromide (EDB) were very effective for control of root-knot nematodes and permitted continuous peanut culture without significant decline in yield. However, health and environmental problems associated with these chemicals resulted in the removal of DBCP (in 1978) and EDB (in 1981) from agricultural use in the United States, but EDB is still used in agriculture in some countries. Other fumigant nematicides available for use in peanut are 1,3-dichloropropene (1,3-D) and metham sodium. These fumigants must be applied 1–2 weeks before planting to avoid phytotoxicity. 1,3-D is most effective when applied in the row at rates of 50–75 liters per hectare. Metham sodium decomposes in soil, generating nematicidal methyl isothiocyanate; maximal peanut yield responses are obtained with in-the-row rates of 150–250 liters per hectare. Efficacy of metham sodium is variable and depends on the application methods and soil moisture and temperature.

Nonfumigant systemic nematicides available for use in peanut production are aldicarb, carbofuran, and phenamiphos. These nematicides are most effective when applied at planting at rates of 2–3 kg of active ingredient per hectare. For best results, the application should be performed in a band 17–25 cm wide with light (2–4 cm deep) incorporation into the soil. Other nonfumigant nematicides used with peanuts are ethoprop and fensulfothion.

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(Prepared by N. Kokalis-Burelle and R. Rodríguez-Kábana)

Root-Lesion Nematodes

Root-lesion nematodes were first observed on peanut plants in the United States in 1945. They have subsequently been found in all peanut-production areas of the United States and in most peanut-producing countries. Although these nematodes are often found in fields with a history of peanut production, they are not always associated with economic losses.

Symptoms

Lesion nematodes are migratory endoparasites that attack peanut roots, pegs, and pods, feeding primarily on parenchyma cells (Fig. 65). Nematode feeding activity causes substantial tissue destruction and results in the development of necrotic lesions (Fig. 66). Under high disease pressure from large nematode population densities, root lesions coalesce, causing a general discoloration of the roots. Damaging population densities of lesion nematodes are most frequently associated with coarsely textured, sandy soils.

Pod lesions begin as tan to brown pin-point areas on the pod surface; as the nematodes feed and reproduce, the affected areas enlarge and become darker (Plate 94). Older lesions are characterized by a blotchy appearance and indistinct margins, which are caused by the darker necrotic parenchyma showing

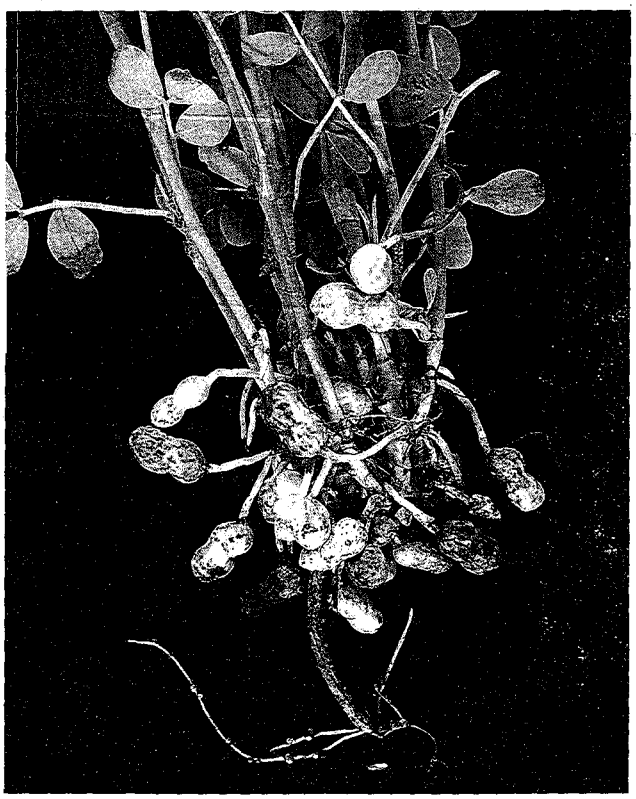

Fig. 65. Roots, pegs, and pods with necrotic lesions caused by *Pratylenchus brachyurus*.

through the outermost cells of the pod and making the necrotic area look diffuse. Infection of pegs also results in the development of necrotic lesions. Such lesions weaken the pegs, and pods are shed prematurely during the harvesting process.

Symptom development, damage to pods and pegs, and suppression of plant growth are related to initial nematode population densities and rate of population increase. Population densities in excess of 5,000 nematodes per gram of fresh tissue have been observed in association with severe crop damage. Root-lesion nematodes may also affect the percentage of sound mature seed, seed weight, and value.

Other microorganisms may colonize nematode-damaged tissues. The combined effects of nematodes and associated microorganisms can result in additional losses from decreased pod yield and reductions in yield quality.

Causal Organisms

Two species of lesion nematodes, *Pratylenchus brachyurus* (Godfrey) Filipjev & Schuurmans-Stekhoven and *P. coffeae* (Zimmermann) Schuurmanns-Stekhoven, attack peanut plants (Figs. 67 and 68). *P. brachyurus*, first reported in 1945, is the most common and widely distributed species. Some evidence suggests that populations of *P. brachyurus* may differ in virulence (aggressiveness). *P. coffeae* has been reported only from India, where affected plants have a sickly appearance and patchy, stunted growth. Other species of lesion nematodes have been associated with peanut but have not been observed to cause economic loss.

Disease Cycle

P. brachyurus survives extremes of temperature and moisture when the nematodes are within root and pod debris. Active nematodes can be recovered from infected pods for several months after harvest. It is likely that dissemination of the nematode occurs through movement of infected pods and root debris.

All developmental stages of the nematode are capable of infecting roots, pegs, and pods. Initial infection may occur immediately after germination of planted seed. The nematode requires 4–5 weeks to complete its life cycle at optimal temperatures (24–28°C), and several generations can occur in a single growing season. Because all developmental stages are infective, egg production by fecund females may begin within a few days of infection. Root lesions also can be seen within a few days of the initial infection. Discrete generations have not been observed in field populations because of the mix of life stages that infect the developing root system.

Control

Identification of the nematode species within a particular field and determination of population density are basic to developing appropriate management programs. The most accurate estimates of nematode population densities are obtained from samples collected during the autumn when densities are expected to be at maximum levels. It is critical that both soil and root populations be determined for this migratory endoparasite. Nematode extraction from pod, peg, and root tissues provides a more accurate estimate of population densities for lesion nematodes than soil analysis.

Soil treatment with fumigant or nonfumigant nematicides is effective in controlling lesion nematodes. It is often beneficial, when initial nematode population densities are high, to split nematicide applications and make the first application prior to or at planting and the second at the time of peg initiation. With lower initial nematode population densities, the at-pegging

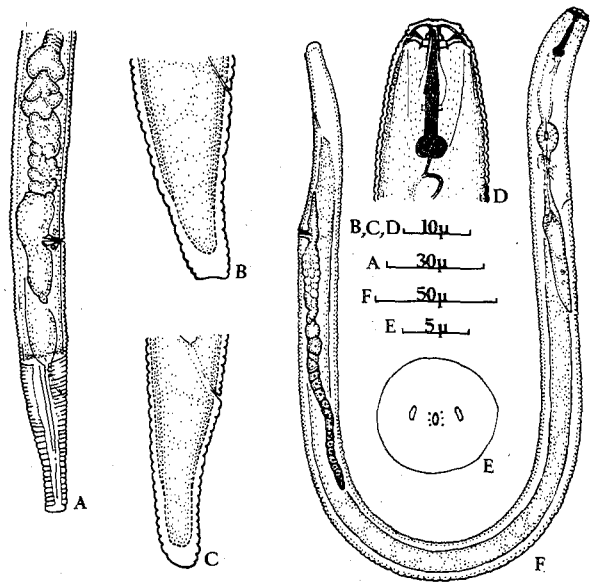

Fig. 67. *Pratylenchus brachyurus* female. **A,** hind end; **B and C,** tail tips; **D,** head-end profile; **E,** head, face view; and **F,** entire female. (Reprinted, by permission, from C.I.H. Descriptions of Plant-Parasitic Nematodes, Commonwealth Institute of Parasitology; © 1986 C.A.B. International)

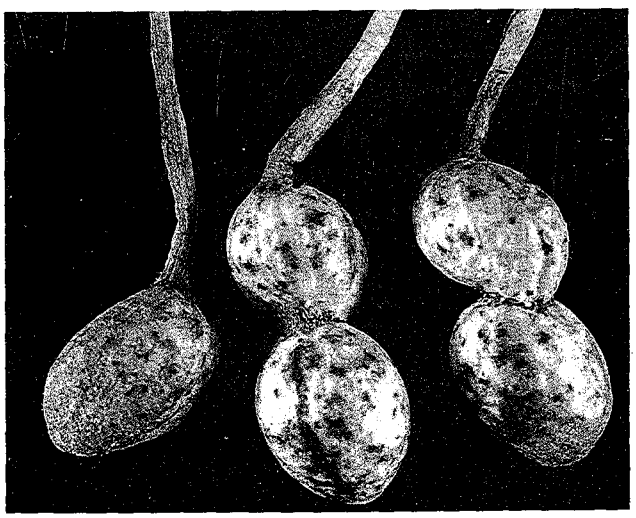

Fig. 66. Pods and pegs with lesions caused by *Pratylenchus brachyurus.*

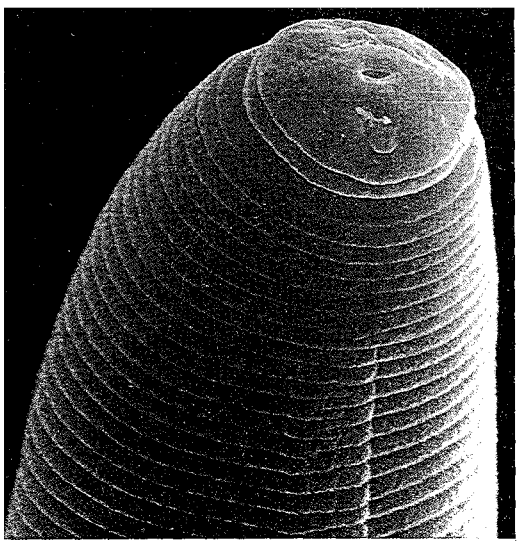

Fig. 68. *Pratylenchus brachyurus* cephalic region. (Reprinted, by permission, from McClure and Stowell, 1978)

application may provide economic control. Comparisons of nematicide efficacy under conventional full tillage and that with minimum tillage operations indicate that tillage does not affect efficacy. Rotation is usually less effective and more difficult than chemical control because lesion nematodes have a wide host range that includes numerous crop and weed species.

Moderate levels of host resistance have been identified but have not been incorporated into agronomically suitable peanut genotypes.

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(Prepared by T. E. Boswell and J. L. Starr)

Sting Nematodes

Sting nematodes (*Belonolaimus* spp.) have been known for some time to be economically important pathogens of peanut in the United States. These nematodes are present in Virginia, North and South Carolina, Georgia, Alabama, and Florida but are of greatest importance in Virginia and the Carolinas. Yield losses can be slight or very severe (Plate 95). Sting nematodes have not been found in peanut-growing areas outside the United States.

Symptoms

Sting nematodes cause peanut roots to become gnarled and stubby; frequently the taproot is the only remaining root (Plate 96). Feeding by these nematodes causes tiny lesions along the taproot, and plants become chlorotic with stubby, sparse root systems. Roots and pods have small, dark, necrotic spots (Fig. 69) caused by nematode feeding. Sting nematodes, unlike root-knot or root-lesion nematodes, are for the most part ectoparasites; they are rarely found internally in roots or pods.

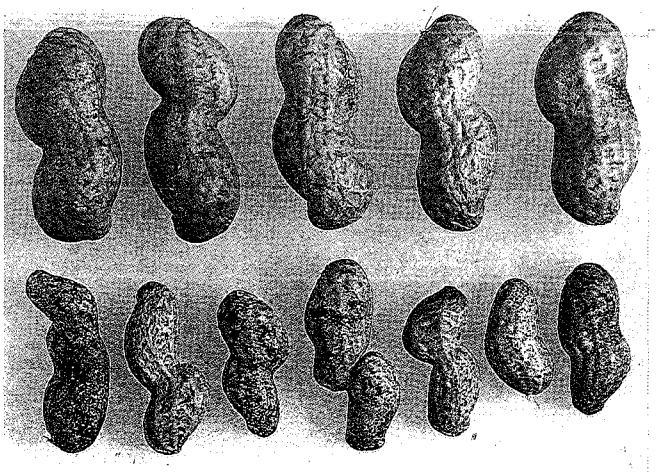

Fig. 69. Pods with lesions caused by the sting nematode, *Belonolaimus longicaudatus* (bottom), and unaffected pods (top). (Courtesy K. Garren)

Causal Organisms

Sting nematodes parasitic on peanut plants are *B. gracilis* Steiner or *B. longicaudatus* Rau. Before 1958, when *B. longicaudatus* was described, all records on sting nematodes referred to *B. gracilis* as the causal agent of disease on peanut. However, these records probably referred to *B. longicaudatus*, which is the more common of the two species in the southeastern United States.

Males and females of this nematode are morphologically similar. They range in length from 2 to 3 mm and have a strongly striated cuticle. The stylet is very long (100–140 µm).

Disease Cycle

Sting nematodes are migratory ectoparasites restricted to soil types with greater than 84% sand. The nematodes occur mainly in the upper 30 cm of soil, and their numbers fluctuate during the season. Sting nematodes are most active when soil temperatures are 20–34°C and reproduce fastest at 30°C. Infested areas of a field vary in size and shape, but the boundary between diseased and healthy plants is usually well defined, indicating an aggregated distribution.

Little is known about the life cycle of these nematodes. Sting nematodes in all stages of development are apparently capable of feeding and causing damage.

Control

No commercially available peanut cultivars are resistant to this nematode. Sting nematodes have a very wide host range, which includes corn and other grasses, legumes (including soybean), cotton, several vegetable crops, and sunflower. This wide host range precludes the use of crop rotation as a practical control method. Fortunately, sting nematodes are easily controlled with fumigant and nonfumigant nematicides; dosages required are generally lower than those needed to control root-knot nematodes. Some nematicides, such as fensulfothion, that are effective against root-knot nematodes also afford good control of sting nematodes.

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(Prepared by N. Kokalis-Burelle and R. Rodríguez-Kábana)

Ring Nematodes

An unidentified species of ring nematode was first reported on peanut plants in a field in Georgia in 1953 and then in South Carolina in 1955. This same nematode has been found since then in a high percentage of the peanut fields in Georgia and is widely distributed in the peanut-producing region of the United States. Losses caused by the ring nematode have not been well defined. Relatively large numbers of nematodes may be associated with peanut roots, although little discernible loss in yield or quality occurs. However, in North Carolina, negative correlations were obtained between population levels and peanut yields. Also, in field experiments in North Carolina, positive

correlations were obtained between population levels of ring nematodes and symptoms of Cylindrocladium black rot of peanut plants; in greenhouse tests, presence of the ring nematode increased the incidence of the disease on the Florigiant cultivar but failed to affect disease severity on NC 3033, a resistant breeding line.

Symptoms

Obvious damage to peanut plants is seldom caused by the ring nematode, and large populations are usually necessary to produce symptoms. Peanut plants growing in heavily infested field soil have been described as chlorotic, and the condition has been called "peanut yellows." In microplots inoculated with about 10,000 nematodes per plant, the roots and pods of peanut cultivars Argentine and Starr were severely discolored with brown necrotic lesions (Fig. 70A–C). Small, necrotic lesions were usually superficial, but necrosis in large lesions extended deep into the roots and pods. Many root primordia and young roots were killed, resulting in limited numbers of lateral roots. Pod yields from nematode-infested plants were half those of healthy plants. In microplot tests in North Carolina, as few as 178 freshly introduced *Criconemella ornata* per 500 cm^3 of soil stunted peanut plants.

Causal Organism

C. ornata (Raski and Luc) (syns. *Macroposthonia ornata* (Raski) De Grisse & Loof and *Criconemoides ornatus* Raski) are stout, fusiform nematodes about 0.36–0.44 mm long. They have a thick cuticle with deep fissures separating approximately 87–92 annules. The life cycle has not been reported. *C. ornata,* generally considered ectoparasitic, feeds by thrusting its stylet into an epidermal cell or into the cell of the cortex directly beneath the epidermis (Fig. 70D). *C. ornata* feeds on peanut roots, pegs, and pods.

Control

No known peanut cultivars have resistance to *C. ornata.* Some of the crops grown in rotation with peanut (such as cotton, soybeans, corn, and tobacco) reduce population levels of this nematode. Fumigant and organophosphate nematicides are usually effective in control.

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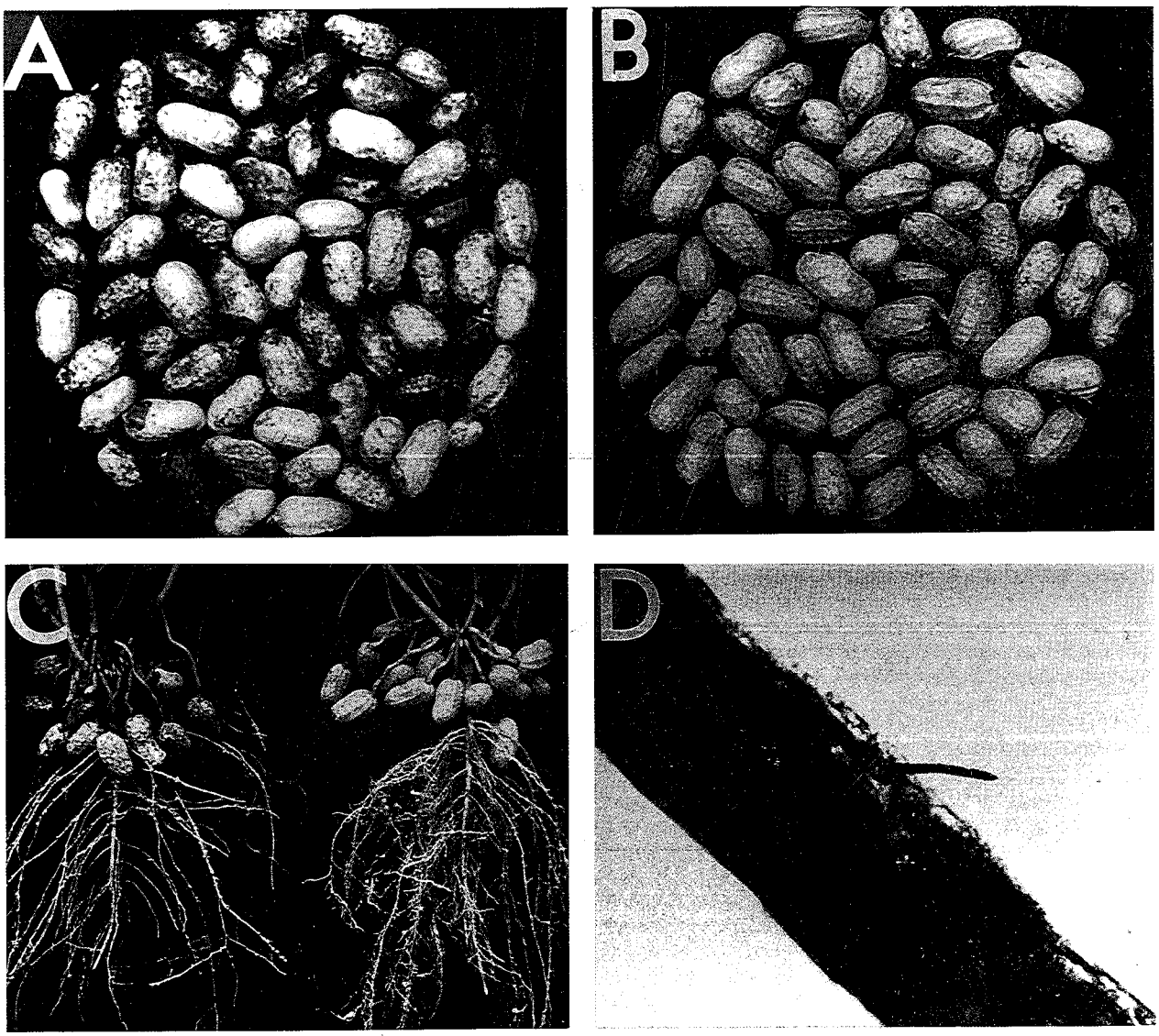

Fig. 70. **A,** Peanut pods inoculated with *Criconemella ornata*; **B,** uninoculated peanut pods; **C,** peanut roots and pods inoculated with *C. ornata* (left) and uninoculated roots and pods (right); and **D,** *C. ortata* partially embedded in a peanut root. (Reprinted, by permission, from Minton and Bell, 1969)

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(Prepared by N. A. Minton)

Peanut Pod Nematodes

Damage caused by *Ditylenchus africanus* was observed on peanut pods in South Africa as early as 1967; but because it was confused with black hull, the first official report of the nematode did not appear until 1988. This nematode has been found in all major peanut-production areas of South Africa in approximately 75% of all fields. *D. africanus* has not been reported outside South Africa, although unpublished observations report its occurrence in neighboring southern African countries.

Symptoms

The peanut pod nematode enters peanut pegs at the connection with the pod and penetrates various layers of the hull. The first visible symptom is a gray, bruiselike discoloration of the pod at the peg connection. The seed coat becomes discolored, and seed germinate prematurely (Plate 97). Those nematodes that enter the hull exocarp migrate along the pod to the distal end, causing a typical broad band of discoloration along the sides of the pod. Many of the nematodes, however, penetrate to the hull endocarp and migrate through the seed micropyle into the seed coat and embryo. No visible damage is caused to the roots.

Breakdown of the pod may lead to early germination of up to 25% of the seed. Seed weight may also be suppressed by 20–50%. The greatest economic impact is to the market value of the crop. Discolored seed, according to South African regulations, are downgraded from first to second grade or even to oil stock. The price decrease to second grade is approximately 15% and to oil stock approximately 65%.

Causal Organism

D. africanus Wendt was first classified as *D. destructor* but differs from the latter species in host plant specificity, characteristics of morphology, and restriction fragment length polymorphisms (RFLPs) of ribosomal DNA. In South Africa, it is commonly referred to as the peanut pod nematode.

Males and females of the peanut pod nematode are morphologically similar, slender, and approximately 0.9–1.0 mm long. The stylet is weak and approximately 8.7–8.9 μm long.

Disease Cycle

The peanut pod nematode is a migratory endoparasite that completes its life cycle from egg to adult in 8 days at 30°C. The life cycle is slower and eggs are less viable at lower temperatures. The nematode enters the pods soon after pegging, approximately 49 days after planting. It remains within the hulls and seed and reproduces until harvest. At harvest, approximately 90% of the nematode population in or around a plant is found within the pods. Only a small percentage is found in the soil and roots. Around the time of harvest, large numbers of eggs are laid in both the hulls and seed. Nematodes in the hulls may enter anhydrobiosis. Many of the rotten pods and hulls remain in the soil, permitting survival from year to year. Infested seed carry the nematode from field to field. Planting infested seed in clean soil or clean seed in infested soil will result in economic loss in yield.

Control

Although the peanut pod nematode does not appear to cause economic damage to other crops, it survives on a wide range of rotation crops including corn, sunflower, and tobacco. Since the nematode also feeds on many weeds and fungi, including *Penicillium* spp. (Plate 98), commonly found in peanut fields, sanitation is one of the first steps in control. The use of nematode-free seed is also important, and current research projects are directed toward this goal. A recently released commercial cultivar, Kwarts, has resistance to this nematode and will become more widely used when seed becomes available.

In South Africa, treatments with nematicides registered for use against the peanut pod nematode include applications of phenamiphos at planting, aldicarb at either planting or pegging, and oxamyl at pegging. These nematicides reduce the numbers of nematodes, but their cost effectiveness is often unreliable, particularly in dryland production.

Recently, soils suppressive to the peanut pod nematode were identified in regions where peanuts have been grown in continuous culture for 10–30 years. A total of 13 species of nematophagous fungi have been isolated from these soils. Two *Arthrobotrys* species and two *Monacrosporium* species are the most commonly found and easily cultured. These fungi will be tested for their potential for biocontrol of *D. africanus*.

In fields infested with *D. africanus*, farmers are encouraged to practice early harvesting. Severity of pod symptoms correlates closely with seed quality. Pods with early symptoms receive a second grade. Although yield increases with time, seed quality continues to decrease. Regular inspection for the first symptoms of nematode infection is required to determine a timely harvest.

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(Prepared by C. Venter)

Other Nematodes

Several other species of nematodes parasitize peanut, but their occurrence is isolated and their importance uncertain. The testa nematode, *Aphelenchoides arachidis* Bos, has been described on peanut only in northern Nigeria. *A. arachidis* is a facultative endoparasite found mainly in the parenchymatous tissue and around the tracheids of the seed, testae (seed coats), shells, roots, and hypocotyls. Heavily infested seed coats become discolored and translucent with dark vascular strands.

Seed coats may become unevenly thickened. All stages of the nematode are found throughout the seed coat until the end of the growing season, when mainly juvenile stages are found. *A. arachidis* predisposes seed to invasion by soilborne fungal pathogens such as *Rhizoctonia solani, Sclerotium rolfsii, Macrophomina phaseolina,* and *Fusarium* spp., leading to reduced seedling emergence. *A. arachidis* devalues the crop by reducing seed quality.

A. arachidis can survive desiccation in stored pods for up to 12 months, while infected pods that are sun dried in the field before storage contain no active nematodes. Adult nematodes have been isolated from volunteer plants. Control of this nematode is mainly by preventive treatments that include immersing seed in 60°C water and sun drying pods after harvest. *A. arachidis* can be disseminated in infected seed and therefore has the potential to become a widely distributed pest.

Aphasmatylenchus structuratus Germani is a migratory endoparasite and ectoparasite of peanut that has been reported only in southwest Burkina Faso in western Africa. The nematode causes chlorosis, stunting, reduced development of the root system and *Rhizobium* nodules, and yield reduction. *A. structuratus* survives the dry season adjacent to roots of the karite tree (*Butyrospermum parkii* L.) and does not enter into anhydrobiosis. This nematode spreads rapidly and parasitizes other economically important leguminous plants in Burkina Faso.

Scutellonema cavenessi Sher has been found in northern Nigeria, Senegal, and Mali. This nematode causes chlorosis, reduced root growth, and reduced *Rhizobium* nodulation. *S. cavenessi* is active during the rainy season and enters into anhydrobiosis when soil moisture drops to approximately 0.2%. The extent of yield loss from this nematode is not known, but the application of nematicides increases yield 20–220%. *S. cavenessi* has been found associated with most cultivated plants in Senegal and Mali. Leaving fields fallow between crops provides excellent control but is not economically practical.

Tylenchorhynchus brevilineatus Williams (syn. *T. indicus* Siddiqi) has been reported only in the Kalahasti area and Nellore district of Andhra Paradesh, India. This nematode causes a brownish black discoloration of the pod surface and reduced pod size. Pegs and young pods may have brownish yellow lesions with slightly raised margins. *T. brevilineatus* can be controlled and yields increased with application of aldicarb or carbofuran.

Dagger nematodes (*Xiphinema* spp.) are found consistently in peanut and often damage roots, producing galls and curly tips. Populations of dagger nematodes in peanut fields are typically very low and stable throughout the growing season, making the damage they cause relatively unimportant.

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(Prepared by N. Kokalis-Burelle
and R. Rodríguez-Kábana)

Diseases Caused by Viruses

Tomato Spotted Wilt and Peanut Bud Necrosis

The peanut bud necrosis virus (PBNV) and tomato spotted wilt virus (TSWV), both tospoviruses, cause economically important diseases in peanut. The distinction between PBNV and TSWV has been recognized only recently. The disease referred to as "bud necrosis," now shown to be caused by either TSWV or PBNV, was previously attributed only to TSWV. TSWV and PBNV cannot be distinguished by symptoms alone on peanut or other hosts. TSWV is widely distributed in the Americas, Australia, Africa, and Europe, whereas PBNV appears to be restricted to southern and southeastern Asia. Nevertheless, the thrips vectors of TSWV and PBNV occur in most peanut-growing countries. Therefore, in surveys for the occurrence of virus diseases in peanut, assays for both PBNV and TSWV should be conducted.

Symptoms

Symptoms caused by TSWV and PBNV in peanut are variable. They may appear in young leaflets in the form of chlorotic spots or a mild mottle (Plate 99) that develops into necrotic and chlorotic rings and streaks. Bud necrosis symptoms appear to be governed by ambient temperatures; when temperatures are above 30°C during the day, petioles bearing fully expanded leaflets with initial symptoms (as described above) usually become flaccid and droop (Plate 100). This symptom is followed by necrosis of the terminal bud. When a relatively young plant is infected, the necrosis spreads toward the base of the plant, resulting in its death. Secondary symptoms include stunting and proliferation of axillary shoots. Leaflets produced on the axillary shoots are reduced in size and show puckering, mosaic mottling, and general chlorosis (Plate 101). Secondary symptoms are most common on early-infected plants, giving them a stunted and bushy appearance. Plants infected later may also be stunted, but the symptoms may be restricted to a few branches or to the apical parts of the plants. Seed from early-infected plants are small and shriveled, and the seed coats are red, brown, or purple with mottling (Plate 102). Although late-infected plants may produce seed of normal size, the seed coats are often mottled and cracked.

Causal Agents

TSWV and PBNV are found in all parts of affected plants. Clusters of virus particles are often found in the cisternae of the endoplasmic reticulum. Individual particles are 80–120 nm in diameter and are covered with projections resembling spikes (Fig. 71). TSWV and PBNV have an extremely low thermal inactivation point (45°C for 10 min) and short longevity in vitro (less than 5 hr at room temperature). These properties can be used, in conjunction with others, to identify TSWV and PBNV.

It is difficult to isolate TSWV or PBNV from infected plant tissues. Additionally, neither TSWV nor PBNV is highly immunogenic. Polyclonal antibodies produced for an isolated virus may react with healthy plant extracts. Therefore, extreme care should be taken in using polyclonal antisera and in interpreting the results of serological tests. Polyclonal or mono-

clonal antibodies are widely used to study relationships among tospoviruses and to diagnose disease.

Host Ranges

TSWV and PBNV have extremely wide host ranges that include more than 370 species of plants in more than 50 families.

Transmission

Both TSWV and PBNV are mechanically transmissible. Only chilled extracts containing antioxidants such as mercaptoethanol or thioglycerol are suitable for transmitting the virus by mechanical inoculations. Both TSWV and PBNV are transmitted by thrips. Probable vectors of TSWV in the United States are *Frankliniella fusca* (Hinds) and *F. occidentalis* (Pergande); *Thrips palmi* (Karny) transmits PBNV in India. The viruses are acquired only by insect larvae but may be transmitted by larvae or adults. TSWV multiplies in its thrips vector. Neither PBNV nor TSWV is transmitted by seed in peanut.

Control

Cultural practices such as early seeding, use of high-quality seed treated with an approved seed protectant, and sowing at the recommended rate and spacing to give optimum plant population can reduce the incidence of TSWV and PBNV. The seedbed should be well prepared, and soil moisture should be sufficient to ensure good germination and seedling establishment. Given good growing conditions, the crop will rapidly develop a close canopy and will not be as attractive to the thrips vector as a patchy crop. The incidence of TSWV and PBNV under these conditions is much reduced. Because of the extremely wide host ranges of TSWV and PBNV and their vectors, it is not practical to control the disease by destroying weed reservoir hosts. Intercropping one row of a quick-growing cereal crop such as sorghum or pearl millet with each three rows of peanut can reduce disease incidence. Removal of infected plants will create gaps in the field that may lead to an increase in the percentage of infected plants.

Good sources of resistance to both TSWV and PBNV have been identified. The cultivar Southern Runner typically has 50% lower disease incidence than the susceptible cultivar Florunner. Peanut genotypes resistant to PBNV and *T. palmi* include ICGV 86029, ICGV 86031, and ICGV 86388. Southern Runner is also resistant to PBNV. Since the application of insecticides increases disease incidence of both PBNV and TSWV, it is not advisable to apply insecticides for control.

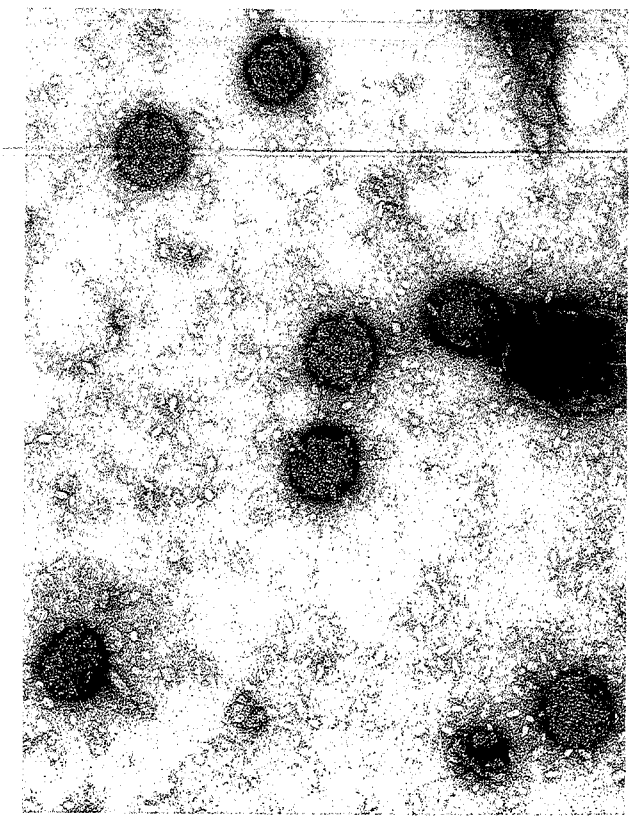

Fig. 71. Particles of peanut bud necrosis virus covered with spikes or projections.

Peanut Clump and Indian Peanut Clump

Peanut clump occurs in the Indian subcontinent and in western Africa (Senegambia, Burkina Faso, Niger, and the Ivory Coast). The pathogen, a soilborne virus, causes severe crop losses. Symptoms of peanut clump resemble those of green rosette (see Groundnut Rosette). As a result, it is likely that these two diseases have been confused.

Studies on genome organization have revealed that isolates from India and western Africa are two distinct viruses. The virus from western Africa, referred to as peanut clump virus (PCV), and that from India, Indian peanut clump virus (IPCV), are not serologically related. Differences in genome organization between PCV and IPCV are apparent, and the complete nucleotide sequences of both genome RNAs are available.

Symptoms

Diseased plants are severely stunted and dark green (Plate 103). The disease occurs in patches in the field and recurs more or less in the same position in the same field in successive peanut crops. The symptoms first appear on young plants as mottling, mosaic, and chlorotic rings on newly emerged quadrifoliates (Plate 104). Subsequently, infected leaves turn dark green with or without faint mottling. Early-infected plants are severely stunted and may produce flowers, but any pods formed are not well developed. Plants infected later are also stunted and have shortened internodes and dark green leaflets. These plants may produce pods, but seed weights may be reduced by up to 60%.

Causal Agents

PCV and IPCV have rod-shaped particles 24 nm in diameter; there are two predominant lengths, approximately 185 and 250 nm (Fig. 72). The particles of PCV and IPCV each contain a single polypeptide species with a molecular mass of 24 kDa.

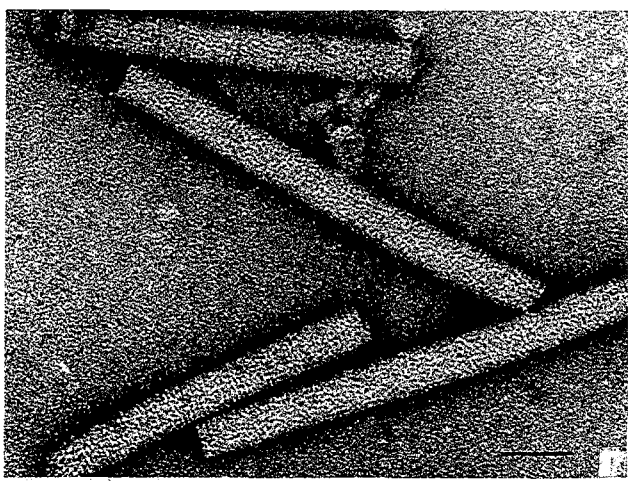

Fig. 72. Peanut clump virus particles.

Both IPCV and PCV are currently known to occur in several serologically distinct variants. Among the IPCV variants, three distinct serogroups have been identified. Variants can also be distinguished on the basis of their reaction on several hosts. RNA1 of IPCV has been found to contain sequences conserved among the variants and will be utilized to produce nucleic acid probes capable of detecting several IPCV variants.

Host Ranges

PCV and IPCV have extremely wide host ranges, which include many monocotyledonous and dicotyledonous plants.

Transmission

IPCV and PCV are readily sap transmissible. Both are transmitted by *Polymyxa graminis*. Both viruses are transmitted by seed in peanut (more than 6% frequency). Since IPCV is present in seed coats of all seed from infected plants, the seed coats should be removed before ascertaining seed transmission. IPCV is seed transmitted in cereal crops such as finger millet, foxtail millet, and pearl millet but not in sorghum.

The occurrence of IPCV is correlated with ambient air temperatures. When ambient air temperatures are below 25°C, only negligible IPCV incidence is observed. Therefore, in the tropics, crops grown after the rainy season in locations where temperatures are lower than 25°C escape infection. High temperatures during the summer season followed by monsoon rains appear to favor natural transmission.

Control

IPCV incidence is higher when peanut crops are rotated with susceptible cereal crops. The incidence of virus can be reduced by the application of soil biocides. Soil solarization (during hot summer months for a period of 2 months) reduces IPCV incidence. None of more than 9,000 *Arachis hypogaea* genotypes showed resistance to IPCV when tested on infested soils. Since the genomes of PCV and IPCV have been sequenced, there are excellent prospects for utilizing viral genes to induce resistance by unconventional methods in *A. hypogaea*.

Groundnut Rosette

Groundnut rosette disease was first reported from Tanzania in 1907. Many epidemics of rosette were subsequently recorded in Africa. One major epidemic in 1975 caused nearly $250 million in crop losses in Nigeria alone.

Rosette has also been reported in Argentina, India, Indonesia, and the Philippines. In Africa, rosette disease is restricted to countries south of the Sahara.

Symptoms

Three types of rosette disease, chlorotic, mosaic, and green, are recognized on the basis of symptoms. Chlorotic rosette occurs throughout Africa south of the Sahara. The disease first appears on young leaflets as faint mottling with a few green islands. Leaflets produced subsequently are pale yellow with green veins. Plants infected when young produce progressively smaller, chlorotic, curled, and distorted leaflets. When older plants are infected, symptoms may be restricted to a few branches or to the apical portion of the plants. Plants infected early are severely stunted (Plate 105) with thickened stems. Early infection causes severe reduction in the number and size of pods.

Green rosette disease occurs in western Africa and Uganda. Young leaflets show mild chlorotic mottling and isolated flecks. Symptoms are masked in older leaflets, but leaflets are reduced in size, show outward rolling, and are not distorted. Plants infected early are severely stunted and are a darker green than healthy plants (Plate 106), somewhat resembling plants infected with peanut clump virus.

Mosaic rosette occurs only in eastern and central Africa. Young leaflets show conspicuous mosaic symptoms (Plate 107), which resemble those of chlorotic rosette except that stunting is less pronounced.

Causal Agents

Rosette disease of peanut is caused by a complex of two viruses and a satellite RNA. Diseased plants contain a mechanically transmissible virus, groundnut rosette virus (GRV), which is classified as an umbravirus. GRV depends on the groundnut rosette assistor virus (GRAV), a luteovirus, for transmission by *Aphis craccivora*. GRAV causes no obvious symptoms in peanut on its own. Variants of the satellite RNA are responsible for the different forms of rosette (chlorotic, green, and mosaic). These variants differ appreciably in nucleotide sequence, as do forms of the satellite from different parts of Africa.

GRAV, a typical luteovirus, can be detected by polyclonal antisera produced against GRAV and by antisera to some other luteoviruses, such as potato leafroll virus (PLRV). It can also be detected by some monoclonal antibodies to PLRV. No virus particles have been reported for GRV, and so no antiserum is available. Infected plants contain abundant infective single-stranded RNA.

Host Range

Peanut is the only natural host known for both GRAV and GRV, though alternate hosts probably play an important part in perpetuating the inoculum between crop seasons. Most species become infected with only one virus or the other. Host range tests with GRV can be done also by mechanical inoculation. GRV cultures, with or without the satellite RNA, induce necrotic lesions in host plants but not systemic infections. Necrotic rings are also produced on inoculated leaves. Most sensitive test plants produce mild veinal chlorosis or necrosis on the first systemically infected leaves, which is followed by a faint mottle and moderate stunting.

Transmission

The viruses associated with all three types of rosette disease are transmitted by *A. craccivora* in a persistent or circulative manner. By analogy with other umbraviruses, it is suspected that GRV and its satellite RNA are encapsidated in GRAV coat protein. Peanut is considered to be the main source of inoculum from which the initial spread of the rosette disease occurs. Neither GRAV nor GRV is seed transmitted in peanut. Because rosette-infected plants survive longer than healthy plants, they are not normally harvested and serve as an important source of inoculum. Volunteer plants may also be a source of inoculum. It is likely that *A. craccivora* colonizes these rosette-infected plants and that moving rainy fronts are responsible for the dissemination of the aphids. Alate and apterous aphids are involved in the secondary spread. Diagnostic aids for the various components of rosette have recently become available and should permit epidemiological studies on rosette disease in Africa.

Control

Rosette disease can be effectively controlled by cultural practices. These include destroying all volunteer and unharvested infected plants, planting early in the season and at a high seeding rate, maintaining plant stands, and applying insecticides at the correct time. Excellent sources of resistance to rosette disease are available in peanut germ plasm. Early-maturing, rosette-resistant genotypes recently have been identified and are being used in the development of rosette-resistant, early-maturing cultivars. Since the coat protein gene of GRAV has been sequenced and constructs suitable for transformation have been prepared, prospects for producing rosette-resistant cultivars by insertion of the coat protein genes into peanut are good.

Peanut Mottle

Peanut mottle virus (PeMoV) was first reported in the United States in 1961 and is currently present in all the major peanut-growing countries. Its widespread distribution is probably the result of dispersal of infected seed. An economic loss of 5–6% from PeMoV was estimated in Georgia. In field tests in India, susceptible cultivars suffered yield losses of up to 40%. Because of its worldwide distribution and potential for causing economic losses, PeMoV is considered to be of global economic importance.

Symptoms

Young leaflets show mild mottle symptoms or a mosaic of irregular, dark green islands (Plate 108). In older leaflets, mosaic symptoms are not obvious but can be seen by transmitted light. In some genotypes, conspicuous interveinal depressions and an inward curling of the edges of leaflets are apparent (Plate 109). Infected plants are slightly stunted. PeMoV reduces both numbers and size of pods on infected plants.

Causal Agent

PeMoV belongs to the potyvirus group. Particles are flexuous rods that measure about 750 ×12 nm (Fig. 73). The coat protein has an apparent molecular mass of 32–36 kDa. The thermal inactivation point is 55–64°C, and the longevity in vitro is 1–2 days at room temperature.

High-quality polyclonal antisera and monoclonal antisera have been produced for PeMoV. They do not react with the peanut stripe virus or the peanut green mosaic or groundnut eyespot potyviruses that occur on peanut.

Host Range

PeMoV occurs in several important legume crops, including peanut and soybean, and weeds.

Transmission

PeMoV is readily transmitted by mechanical sap inoculation and is seed transmitted at rates of 0–8.5%. PeMoV is also seed transmitted in mung bean and cowpea but not in soybean. The virus can be detected in peanut seed by enzyme-linked immunosorbent assay. PeMoV is transmitted in a nonpersistent manner by *Aphis craccivora*, *A. gossypii*, *Myzus persicae*, *Hyperomyzus lactucae*, *Rhopalosiphum padi*, and *R. maidis*.

Control

Peanut seed appears to be the primary source of inoculum. Since many aphid species can transmit the virus, it is spread rapidly to nearby plants. To avoid the disease, the planting of virus-free seed is important. Genotypes in which PeMoV is not seed transmitted have been identified and utilized in conventional breeding programs to produce acceptable, high-yield breeding lines that do not transmit the disease via seed. Although resistance to PeMoV has been identified in wild species of *Arachis*, it has not yet been transferred to *A. hypogaea*.

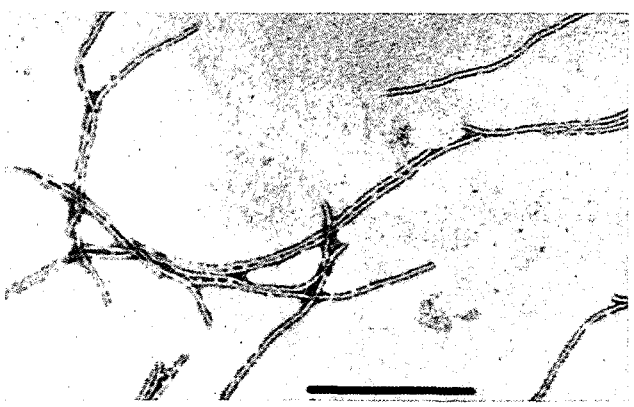

Fig. 73. Peanut mottle virus particles. Bar = 500 nm. (Courtesy C. Kuhn)

Peanut Stripe

The peanut stripe virus (PStV) was first reported in the United States in 1983, having entered the country in peanut seed imported from China. PStV has been present in Southeast Asia since the early 1970s but has been often misidentified as peanut mottle virus (PeMoV). Peanut stripe poses a serious threat to peanut production in southern and southeastern Asia.

Symptoms

Several symptom variants of PStV are known. The name peanut stripe virus was first given to an isolate that induced discontinuous, dark green stripes along the lateral veins of young leaflets (Plate 110). However, the most widely distributed variant causes irregular green blotches on young leaflets that persist as the leaflets age (Plate 111). A variant that induces chlorotic rings surrounding blotches on young leaflets was reported from Thailand and Indonesia. The most widely distributed isolate in China induces a mild mottle symptom (Plate 112).

Causal Agent

PStV belongs to the potyvirus group. Although its particles resemble those of PeMoV, PStV is serologically distinct from PeMoV. However, comparison of available full-length potyvirus nucleic acid sequences indicates that PStV is closely related to the soybean mosaic virus.

Host Range

Natural hosts in the field are *Centrosema pubescens*, *C. macrocarpum*, *Calopogonium caeruleum*, *Crotalaria striata*, *Desmodium siliquosum*, and *Pueraria phaseoloides*. A number of hosts can be systemically infected with PStV by sap inoculation. Since PStV does not produce local lesions on the *Phaseolus vulgaris* cultivar Topcrop, this host can be used to distinguish it from PeMoV.

Transmission

PStV is transmitted by sap inoculation and by many aphid species in a nonpersistent manner. Different symptom variants of PStV have different aphid transmission frequencies. Seed transmission of PStV can be as high as 37% when the seed are derived from plants inoculated before flowering. Seed transmission frequency in naturally infected plants is normally less than 5%. The virus is readily detected in seed by enzyme-linked immunosorbent assay.

Control

Of approximately 10,000 peanut genotypes evaluated for resistance to PStV in Indonesia, none was resistant. However, some genotypes showed only mild symptoms, and some took a longer time than the susceptible check to show overt symptoms. The PStV genome has been sequenced, and the potential exists for utilizing viral coat protein genes to incorporate resistance into *Arachis hypogaea*.

In areas where PStV is established, it occurs at high incidence, resulting in the harvest of virus-contaminated seed. The common practice of using seed from the previous season's crop assures the continuous presence of PStV in the field. Therefore, production and distribution of virus-free seed should be given a high priority in efforts to contain the spread of PStV.

Peanut Stunt

Peanut stunt was first observed in the United States in 1964. It was economically important in the southeastern United States in various forage legumes and beans, but it is now only a minor disease. The peanut stunt virus (PSV) occurs naturally in peanuts from Sudan and China, where it can cause crop losses of up to 75%.

Symptoms

In the United States, PSV causes severe dwarfing of the entire plant or of one or more branches. In China, the virus does not cause severe stunting. Shortening of the petioles, reduction in the size of leaflets, chlorosis, and malformation are observed in the United States, China, and Sudan (Plate 113). Plants infected early in the growing season produce very few pods, and these are misshapen and frequently have a split pericarp wall. The viability of seed from such pods is markedly reduced. The virus causes epinasty with systemic mosaic and malformation in cowpea (cultivar Blackeye). Systemic symptoms produced by PSV in peanut, beans, and cowpea can be used to distinguish it from the cucumber mosaic virus.

Causal Agent

PSV belongs to the cucumovirus group. The particles are 25–30 nm in diameter and encapsidate three single-stranded RNAs. Two serologically distinct isolates from the United States, PSV-E from the eastern region and PSV-W from the western region, and three serotypes from China, PSV-T, PSV-2, and PSV-B2, have been reported.

Host Range

PSV has a wide host range. It produces local lesions on *Chenopodium amaranticolor* and *C. quinoa.*

Transmission

PSV is transmitted by sap inoculation and in a nonpersistent manner by three aphid species, *Aphis craccivora, A. spiraecola,* and *Myzus persicae.* It is seed transmitted at the lowest frequency of all the other known seed-transmitted peanut viruses. Up to 0.01% of large seed from plants infected late in the season may contain the virus. Up to 0.2% of small seed from less severely stunted plants may be infected.

Control

Since some forage legumes, such as white clover, are the primary source of inoculum, peanuts should not be planted in fields located near such legumes. Roguing of infected plants from crops intended for seed production is recommended. Currently, there are no peanut genotypes resistant to PSV.

Cowpea Mild Mottle

The cowpea mild mottle virus (CPMMV) is widely distributed in Asia and Africa but has not been reported in the United States. CPMMV incidence in peanut does not exceed 5%. Because of its wide distribution, potential to cause severe crop losses, and occurrence at high incidence in peanut crops intercropped or grown adjacent to crops such as soybean or cowpea, incidence of CPMMV should be routinely monitored in countries where it is endemic.

Symptoms

Initial symptoms on young leaflets are veinclearing followed by downward rolling of the leaflet edges and veinbanding. Subsequently, necrosis of leaflets and petioles occurs. Plants are severely stunted and are conspicuous because of the rolled edges and veinbanding of the leaflets (Plate 114).

Causal Agent

CPMMV is a member of the carlavirus group of plant viruses. The particles are slightly flexuous rods, 15 nm in diameter and 610 nm long (Fig. 74). The thermal inactivation point of CPMMV is 75–80°C. High-quality polyclonal antisera to CPMMV also react with the groundnut crinkle virus reported from the Ivory Coast. CPMMV may be serologically related to several aphid-transmitted carlaviruses.

Host Range

The virus produces local lesions and systemic symptoms on many hosts.

Transmission

CPMMV is readily sap transmissible. The whitefly, *Bemisia tabaci,* transmits the virus in a nonpersistent manner. CPMMV is not seed transmitted in peanut.

Control

Peanut crops should not be planted adjacent to crops such as cowpea and soybean, which are frequently colonized by *B. tabaci* and are highly susceptible to infection by CPMMV in areas where it occurs.

Cucumber Mosaic

Natural occurrence of the cucumber mosaic virus (CMV) in peanut has been reported only from China. The disease caused by CMV is referred to as peanut yellow mosaic and is currently recognized as economically important in the northern regions of China. CMV has caused crop losses of up to 40%.

Symptoms

Initial symptoms are chlorotic spots and upward rolling of young leaflets. Subsequently produced leaflets show a yellowing of the lamina with green stripes along the lateral veins (Plate 115). Occasionally, leaflets are deformed and plants are moderately stunted. The severe yellowing and mottling symptoms observed on young plants are not apparent on older plants.

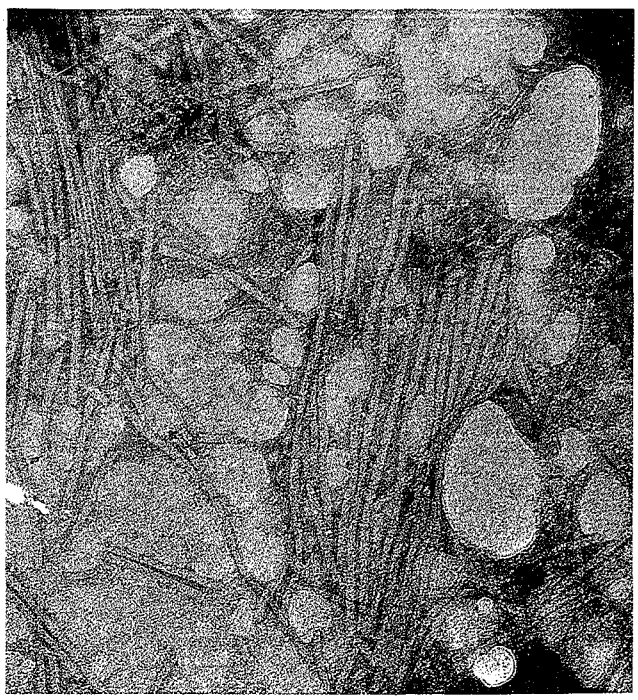

Fig. 74. Cowpea mild mottle virus particles.

Causal Agent

Virus particles are 29 nm in diameter. Many strains of CMV occur in crop plants. Two strains of CMV naturally infect peanut in China. A strain of minor importance, CMV-CS, is serologically related to the peanut stunt virus. The predominant strain, CMV-CA, is serologically related to CMV-D but is distinct from CMV-CS.

Host Range

CMV strains have wide host ranges. CMV-CA can infect 31 plant species in six families by sap inoculation.

Transmission

CMV-CA is easily transmitted by sap inoculation and by several aphids. Seed transmission is up to 4% in peanut.

Control

Seed from infected crops should not be planted. Cultural measures such as mulching with transparent plastic sheets and roguing out diseased seedlings at early stages of crop growth can reduce disease incidence. No resistance to CMV has been identified in the cultivated peanut.

Peanut Chlorotic Streak

Peanut chlorotic streak was first observed in 1977 in Andhra Pradesh, India. In subsequent surveys, the causal agent, the peanut chlorotic streak virus (PClSV), was found to be widely distributed in India. It was reported to be a new member of the caulimovirus group. Recently, a symptom variant of PClSV, which is referred to as the "veinbanding isolate" and has minor differences in the physical map of the genome, has also been reported.

Symptoms

Charaeteristic symptoms appear on young peanut leaflets as oval, chlorotic streaks along the veins. Leaflets are reduced in size, and early-infected plants are stunted. Symptoms are not distinct on older leaflets.

Causal Agent

Purified virus particles of PClSV are 52 nm in diameter. Purified virus contains two polypeptides with molecular masses of 58 and 51 kDa. PClSV DNA has recently been fully sequenced, and the majority of the gene products indicate significant relationships to other caulimoviruses. In enzyme-linked immunosorbent assays, PClSV does not react with cauliflower mosaic, figwort mosaic, or soybean chlorotic mottle viruses.

Host Range

The host range of PClSV is unusually diverse compared with other caulimoviruses. Chlorotic lesions with necrotic centers produced on cowpea are very characteristic. Systemic infection occurs on several *Nicotiana* species, *Datura stramonium*, *Glycine max*, *Petunia* × *hybrida*, *Spinacea oleracea*, and *Vigna radiata*.

Transmission

PClSV is readily mechanically transmissible. It is not transmitted by *Aphis craccivora*, *Myzus persicae*, or *Bemisia tabaci* and is not seed transmitted.

Control

The field incidence of PClSV does not exceed 1%. However, the veinbanding variant of PClSV was observed at an incidence exceeding 20%. No control measures are currently available.

TABLE 6. Viruses That Naturally Infect Peanut[a]

Name	Taxonomic Group	Family	Distribution
Cowpea chlorotic mottle virus	Bromovirus	Bromoviridae	United States
Cowpea mild mottle virus	Carlavirus	NA[b]	China, India, Indonesia, Ivory Coast, Nigeria, Thailand, Philippines, Papua New Guinea, Sudan
Groundnut crinkle virus	Carlavirus	NA	Ivory Coast
Peanut chlorotic streak virus	Caulimovirus	Pararetroviridae	India
Peanut chlorotic streak virus (veinbanding isolate)	Caulimovirus	Pararetroviridae	India
Cucumber mosaic virus	Cucumovirus	Bromoviridae	China
Peanut stunt virus	Cucumovirus	Bromoviridae	Sudan, Japan, Spain, United States
Peanut clump virus	Furovirus	NA	Niger, Burkina Faso, Ivory Coast, Senegal
Indian peanut clump virus	Furovirus	NA	India, Pakistan
Groundnut yellow mosaic virus (bean golden yellow mosaic virus)	Geminivirus	Geminiviridae	India
Tobacco streak virus	Ilarvirus	NA	Brazil
Groundnut rosette assistor virus	Luteovirus	NA	All of Africa south of the Sahara
Sunflower yellow blotch virus	Luteovirus	NA	Malawi, Kenya, Zambia, Tanzania
Groundnut veinal chlorosis virus	Rhabdovirus	Rhabdoviridae	India, Indonesia
Groundnut chlorotic spotting virus	Potexvirus	NA	Ivory Coast
Bean yellow mosaic virus	Potyvirus	Potyviridae	United States
Groundnut eyespot virus	Potyvirus	Potyviridae	Ivory Coast
Passion fruit woodiness virus	Potyvirus	Potyviridae	Australia
Peanut green mosaic virus	Potyvirus	Potyviridae	India
Peanut mottle virus	Potyvirus	Potyviridae	Worldwide
Peanut stripe virus	Potyvirus	Potyviridae	Brazil, China, India, Indonesia, Japan, Malaysia, Philippines, Myanmar, Thailand, Taiwan, Vietnam, United States
Peanut bud necrosis virus	Tospovirus	Bunyaviridae	India, Nepal, Sri Lanka, China, Taiwan, Indonesia, Thailand
Peanut yellow spot virus	Tospovirus	Bunyaviridae	India, Thailand
Tomato spotted wilt virus	Tospovirus	Bunyaviridae	North America, South America, South Africa, Nigeria
Groundnut yellow mottle virus	Tymovirus	NA	Nigeria
Groundnut rosette virus	Umbravirus	NA	All of Africa south of the Sahara

[a] Listed alphabetically by taxonomic group.
[b] Not yet assigned.

Other Viruses

Many other viruses, including those that cause groundnut streak mosaic (Plate 116), groundnut crinkle, groundnut eyespot, peanut green mosaic, peanut yellow spot (Plate 117), peanut yellow mottle, groundnut streak, marginal chlorosis, and rugose leaf curl, have been associated with peanuts throughout the world. Although these pathogens often cause sporadic infections, yield losses can be significant.

Known viruses that can infect peanut under natural conditions, their geographical distributions, and the taxonomic groups to which they belong are listed in Table 6.

Witches'-Broom

Witches'-broom is caused by a phytoplasma (formerly called a mycoplasmalike organism). Unlike viruses, phytoplasmas are cellular organisms related to bacteria. The disease is characterized by stunting and excessive proliferation of shoots from axils (Plate 118). Plants are bushy in appearance. Leaflets are pale yellow and small. Pegs tend to grow upward, and pod yields are severely reduced.

A high incidence of witches'-broom on peanut has been observed in parts of Taiwan, Indonesia, and the Philippines. The pathogen is also known to occur in India, Thailand, China, and Papua New Guinea. A polyclonal antiserum has been produced for the detection of phytoplasma by use of enzyme-linked immunosorbent assay and can be used to distinguish witches'-broom from viral diseases characterized by severe stunting and a bushy appearance.

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(Prepared by J. W. Demski and D. V. R. Reddy; description of cucumber mosaic prepared by Z. Xu)

Part II. Abiotic Diseases

Drought Stress

At the onset of drought, peanut leaflets on affected plants curl and become lighter in color than normal (Plates 119 and 120). If the drought continues, leaflet curling becomes more pronounced and the abnormal coloration becomes even more distinct. Eventually, leaflets turn brown and abscise. Wilted plants usually recover, provided water becomes available. However, if plants remain wilted for a prolonged period of time, they will not recover. Pegs, either because of continued dry soil or pathogenic activity, weaken under severe drought stress. Pods attached to weakened pegs often shed during harvest, especially if the soil is dry and hard. Shallow planting often produces nonvigorous plants that lack deep roots and are extremely sensitive to drought conditions.

(Prepared by D. M. Porter)

Frost Injury

Exposure of peanut plants to below-freezing temperatures during the spring or fall can damage foliage. Tender, young seedlings can be killed if subfreezing temperatures prevail for several hours. Exposure of mature plants to subfreezing temperatures during the fall results in various degrees of damage. Symptoms range from marginal and tip necrosis to death of individual leaflets, branch terminals, and all aboveground plant parts (Plates 121 and 122). Necrotic tissue is readily invaded by both saprophytic and parasitic fungi. With severe injury, defoliation may be extensive. If plants are not carefully examined, frost injury can be mistaken for symptoms of a biotic disease.

Seed in freshly dug pods are also subject to freeze injury. The cotyledons of frost-injured seed are off-white, water soaked or translucent, and off-flavored. Seed have a rubbery texture. Severely damaged seed can be used only for oil stock.

(Prepared by D. M. Porter)

Genetic Disorders

Necrotic Etch

Necrotic etch, a foliar disease of unknown origin, was first observed in Georgia in 1962. Leaflet lesions, characterized at first by the death of three to five cells, are usually surrounded by chlorotic zones of cells. The lesion center appears over a tertiary vein. The lesion expands rapidly; and within 2 days, the necrotic tissue doubles in size. The lesion moves rapidly through tertiary and secondary veins but not through the leaflet midvein. Irregular, zonate lesions that occur on both sides of the leaflet often approach 1 cm in diameter.

The causal agent of necrotic etch has not been established. Attempts to isolate fungi, bacteria, and viruses from necrotic tissue have been unsuccessful. Disease expression is genetically controlled, and affected plants breed true for this condition. Since necrotic etch is inherited as a qualitatively controlled recessive trait, this disorder is considered to be of genetic origin.

Chlorophyll Deficiency

Plants with chlorophyll deficiency ranging from complete albinism to partial deficiency are noted occasionally in peanut fields. Albino plants die shortly after germination. Variegated leaflets, sometimes called chimeras, have areas of normal green tissue as well as areas that lack chlorophyll (Plates 123 and 124). These chlorophyll deficiencies are genetic in origin.

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(Prepared by D. M. Porter)

Hail Injury

Peanut plants at all stages of growth are occasionally damaged by hail. Such plants are characterized by shredded or tattered leaves, parts of which may remain attached to the petiole while dislodged parts fall to the ground (Plate 125). Unless damage is very severe, plants usually recover and pod yields are not noticeably reduced. Under severe conditions, stems may appear bruised. Bruised areas often become necrotic, which can predispose plants to disease.

(Prepared by D. M. Porter)

Herbicide Injury

Herbicides are chemicals used to control, suppress, or kill plants or severely interrupt their normal growth. Selective herbicides are chemicals that are more toxic to certain plant species (weeds) than to others (species that include the crop). Herbicides registered for use on peanut are selective because they usually cause only slight to moderate toxic effects on the crop while controlling the weeds. Severe crop injury may result from use of excessive rates or from herbicide interaction with the environment or other pesticides. Herbicide injury may also result from herbicide carryover from the previous crop, from drift, and from a wide array of application errors such as use of the wrong chemical, overlapping application, nonuniform soil incorporation, or improper timing of application

(Table 7). Herbicide injury is shown in the following plates: dicamba (126), paraquat (127), chloroacetamide (128), acifluorfen (129), dinitroaniline (130), 2,4-DB (131), *s*-triazine (132), metribuzin (133), vernolate (134), bentazon (135), and norflurazon (136).

The presence of distinct injury patterns is often helpful in diagnosing the source of herbicide injury. Overdose patterns related to sprayer swath or direction of sprayer operation, bands of injury related to treatments banded the previous year, and patterns related to areas treated with particular tanks of spray mix can often provide evidence to identify the source of the problem. Patterns related to improper mixing techniques or inadequate agitation are also readily identifiable.

TABLE 7. Symptoms and Control of Herbicide Injury to Peanuts

Herbicide Classification and Names	Symptoms	Control	Remarks
Benzoic Dicamba (Banvel)	Epinasty of stems, leaves, and petioles; splitting of stems and petioles; shortening of internodes; leaves often strap-like in appearance	Avoid drift. In application to adjacent crops, use ground equipment and drift-control measures as noted on label.	Peanut yield loss may occur if damage is sufficient to cause splitting of stems and petioles.
Bipyridyliums Paraquat (various trade names)	Irregularly shaped white to light brown spots or blotches with dark brown margins	Use correct rate to minimize effects and apply at appropriate growth stage.	Peanut plants usually recover from injury without yield loss.
Chloroacetamides Alachlor (Lasso) Metolachlor (Dual)	Swelling of hypocotyl; curling of hypocotyl; roots thickened and stubby in appearance; delayed seedling emergence and crop development; occasionally, failure of seedlings to emerge	Use correct rate, and apply uniformly.	Injury is usually associated with heavy rainfall or irrigation soon after preemergence application, with low seed vigor, or both.
Diphenyl ether Acifluorfen (Blazer)	Leaflets with bronze, speckled burn spots; leaflet crinkle; stem and petiole burn. In severe cases, many injured leaflets may drop.	Use correct rate, and apply uniformly.	A contact herbicide injury is usually most severe when temperature exceeds 32°C and humidity is high.
Dinitroanilines Benefin (Balan) Pendimethalin (Prowl) Trifluralin (Treflan) Ethalfluralin (Sonalan)	Delayed seedling emergence and crop development; swollen hypocotyl; secondary roots thickened and stubby. Meristem of primary root may die. Leaves may be small. Plant may wilt readily under moisture stress.	Use correct rate. Apply and incorporate uniformly. Adhere to label directions concerning incorporation depth.	Injury is usually associated with non-uniform application or incorporation or with use of excessive rates. Factors other than dinitroanilines may cause swollen hypocotyl symptoms; however, roots will usually be normal.
Phenoxys 2,4-DB (Butoxone, Butyrac) 2,4-D (numerous trade names)	Epinasty of stems, leaves, and petioles; occasional splitting of stems and petioles. 2,4-DB frequently causes base of peanut leaflet to become chlorotic and roll downward, resulting in elongated appearance. Seed from plants treated with 2,4-DB are often misshapen, with one cotyledon larger than the other. In affected seed, the larger cotyledon may fold around the smaller, and the radicle is frequently very prominent.	Avoid drift of 2,4-D to peanut plants. Use correct rate of 2,4-DB, and apply uniformly. Do not apply 2,4-DB to peanut plants under moisture stress.	Peanut injury with 2,4-DB is most likely to occur when high rates are applied under conditions of moisture stress.
Substituted ureas Fluometuron (Cotoran) Diuron (Karmex)	Interveinal chlorosis. In severe injury, entire leaflet becomes chlorotic, and necrosis proceeds inward from leaflet tip and margins.	Observe crop rotation restrictions noted on herbicide labels. Apply uniformly and at correct rates.	Carryover of these herbicides is common. It is most likely when pH is high and high rates are used.
s-Triazines Atrazine (Atrazine, AAtrex) Simazine (Simazine, Princep)	Interveinal chlorosis. In severe injury, entire leaflet becomes chlorotic, and necrosis proceeds inward from leaflet tip and margins.	Observe crop rotation restrictions noted on herbicide labels. Apply uniformly and at correct rates.	Carryover of these herbicides is common. It is most likely when pH is high and high rates are used.
Other triazines Metribuzin (Lexone, Sencor)	Bleaching of leaflet at top and/or margins	Avoid aerial application near peanut fields.	Metribuzin does not present a carryover hazard to peanut plants.
Thiocarbamate Vernolate (Vernam)	Delayed seedling emergence. On newly emerged seedlings, leaflets of youngest leaves are sealed at the margins. Affected seedlings are often stunted.	Use correct rate. Apply and incorporate uniformly.	Injured stands usually recover without yield reduction. Injury is most likely under cool conditions on coarse-textured soils with moisture stress during seedling emergence.
Other herbicides Bentazon (Basagran)	Leaflet speckling; mottling or occasionally marginal or complete chlorosis. Affected leaflets are retained within the crop canopy.	Use correct rate and apply uniformly. Avoid application with crop oil concentrates except where necessary to increase effectiveness of weed control.	Peanut plants usually recover from injury without yield loss.
Norflurazon (Zorial)	Veinal chlorosis (bleaching) of leaves or in severe instances chlorosis of entire leaflets	Use correct rate. Apply to soil surface prior to crop emergence.	Peanut plants usually recover from injury without yield loss.

Injury caused by herbicide drift may result from movement of spray particles or vapors from treatment sites adjacent to peanut fields. Drift damage is usually most severe in areas nearest the application site and declines in intensity with distance from that site. Drift damage usually can be detected in sensitive vegetation in and around both the field of origin and the contaminated site.

Herbicide injury resulting from either herbicide carryover or improper soil treatments is often associated with soil conditions. Where excessive dosages of herbicides are used, injury is usually most severe in coarse-textured (sandy) soils low in organic matter. Herbicide carryover is usually associated with high clay content or high organic matter content or both.

Herbicide injury problems can be minimized by using the correct product at the proper rate and time and using recommended application techniques.

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(Prepared by C. W. Swann)

Lightning Injury

Plant injury by lightning occurs infrequently. When it does, it is spectacular and may be confused, without close observation, with an initial locus of a potentially pandemic disease such as rust (*Puccinia arachidis*). Injury is characterized by the death of peanut plants in isolated, round spots within the field (Plate 137). Plants not killed may have black streaks of necrosis running along their branches. The demarcation between uninjured and injured plants in the field is usually distinct. At the fringe of the strike, plants with varying degrees of injury are scattered among unaffected plants.

(Prepared by D. M. Porter)

Nutrient Imbalances

A nutritional imbalance in peanut plants is usually expressed in patterns of abnormal growth. The symptoms, whether from deficiency or toxicity, depend on the severity of the conditions. To produce clear symptoms of known cause, researchers have occasionally used solution culture studies. Some of the descriptions in this section are from such observations. Symptoms in plants grown in the field may be much less distinct and more difficult to diagnose.

Nitrogen

Nitrogen deficiency is characterized by varying degrees of general chlorosis (Plate 138). Young plants not yet adequately colonized by rhizobia may become lighter green than normal. In more severe cases, the entire leaf becomes a uniform, pale yellow, and stems may be thin and elongated. As the plant develops, the lower, older leaves are most affected and drop from the plant. Growth is stunted, and stems may become reddish. In the field, foliar chlorosis can result from lack of nodulation, from ineffective nitrogen fixation induced by molybdenum deficiency (usually associated with extreme soil acidity), from translocation of the limited supply of nitrogen to developing pods late in the season, or from waterlogged conditions.

Phosphorus

Plants deficient in phosphorus are stunted; leaf size is especially reduced. Affected leaves may first become bluish green and then, as they become thickened and leathery, a dull, dark green. In time, the older leaves turn orange yellow (their veins may be reddish brown). The entire leaf becomes brown and finally drops. Stems may become purplish because of an accumulation of anthocyanins.

Potassium

Potassium deficiency is expressed in chlorosis of the leaves, beginning at leaflet margins. Some chlorosis may be interveinal, but most yellowing occurs at the leaf edges. These regions change to reddish brown and then become necrotic or scorched. Severely affected older leaves are shed. Leaf margins may curl upward somewhat, and the tips of branches may redden and die.

Calcium

Peanut is especially sensitive to calcium deficiency. In solution culture, a lack of this nutrient is expressed not only quickly, but with marked effects. Roots are severely affected, becoming short, stubby, and discolored. Young leaves soon wilt, apical buds die, and regions of the petiole break down. Stem elongation ceases, so plants are stunted. In less severe cases, leaves are small and plants take on a bushy appearance. Leaves may develop numerous brown spots or pitted areas, resulting in a bronze color (Plate 139). Flowering and fruiting are inhibited.

In the field, calcium deficiency is expressed more commonly by abnormal fruiting. Extreme deficiency of calcium in the fruiting zone results in no pods being formed. Cultivars vary, however, in their sensitivity to a lack of this nutrient in the fruiting zone. With a less extreme deficiency, the seed often abort and only the shells develop, resulting in empty pods that have been called "pops." Seed that do develop often have a darkened plumule (Fig. 75). The viability of seed, including those that do not have darkened plumules, is directly related to their calcium concentration.

Magnesium

Leaves deficient in magnesium exhibit interveinal chlorosis. The yellowing begins at the margins and extends toward the midrib. The edges may become orange and crinkle or curl. Older leaves develop necrotic areas and then drop from the plant.

Sulfur

Terminal growth of the peanut plant is affected by lack of sulfur. Under deficient conditions, root development is restricted and new leaves become pale green or yellow (Plate 140). Leaf chlorosis resembles that caused by lack of nitrogen, except that because of the greater immobility of sulfur within

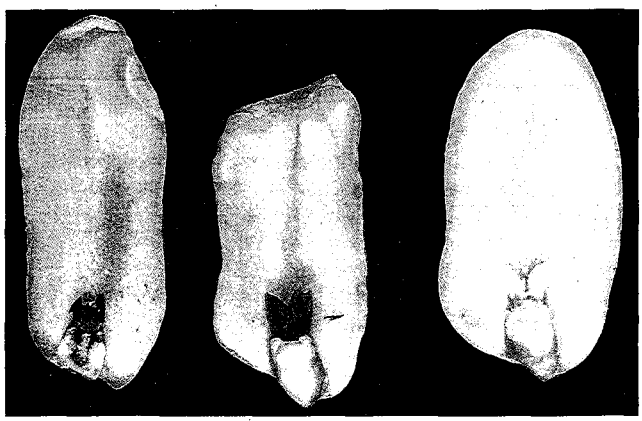

Fig. 75. Healthy cotyledon and plumule (right) and darkened plumules exhibiting calcium deficiency symptoms (left and center). (Courtesy D. Hallock)

the plant tissues, it occurs predominately at growing points. Plants deficient in sulfur may be stunted but appear quite upright because of reduced branching.

Iron

Iron deficiency occurs in calcareous soils with alkaline pH. It may develop so rapidly that intermediate or mild conditions are difficult to observe. Young leaves exhibit interveinal chlorosis and may have somewhat crinkled margins. Very soon, however, these young leaves become very pale yellow, almost white (Plate 141). Even the petiole is very light in color. Affected leaves then develop brown spots or necrosis on the laminae. In the field, plant growth may be restricted and leaf size reduced.

Manganese

Manganese deficiency is expressed by interveinal chlorosis in the leaves (Plate 142). Symptoms range from mild, in which leaves are light green and the regions immediately adjacent to the veins and the veins themselves remain green, to severe, in which the entire interveinal area is chlorotic. After a period of interveinal chlorosis, some bronzing may occur; then older leaves develop necrotic spots and drop from the plant. Stems are slender and weak. Fruiting, and thus yields, are reduced. Manganese deficiency occurs in soils inherently low in this nutrient, especially if they have been limed more than necessary.

Excess manganese, associated with high-manganese soils and very low pH, results in manganese toxicity symptoms. Leaves have slight interveinal chlorosis near the leaf margin, and small brown spots develop in the affected regions. Flowering and rate of maturation are delayed, so pod development is impaired.

Zinc

Lack of zinc results in interveinal chlorosis of recently matured leaves. In severe cases, they will later turn red brown and then drop from the plant. However, retarded growth is the dominant deficiency symptom. Internodal length is reduced; plants are stunted; terminal growth is retarded; and new leaves develop very slowly. Terminal leaflets are small, thickened, leathery, and exceptionally dark green. Yields are reduced.

Yields are also restricted by zinc toxicity. Severely affected plants exhibit chlorosis (Plate 143) and stunting (Plate 144). Stems and petioles become purplish. Lesions resulting in a split stem (Plate 145) occur at the plant's base, and progressive necrosis may cause the plant to die prematurely.

Copper

Under copper deficiency conditions, young leaves become deformed and are greenish yellow or chlorotic (Plate 146). Terminal leaflets are small, and their margins may curl inward, giving a cupped appearance (Plate 147). Yellowish white spots may occur in affected regions. Necrosis develops in the tips and margins, progressing inward until the petiole drops. The bud areas are affected, resulting in stunted plants with short branches. Yields are reduced.

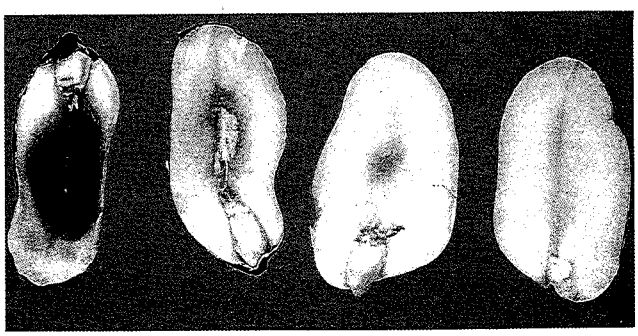

Fig. 76. Cotyledons with hollow heart symptoms (left two) caused by boron deficiency compared with cotyledons from healthy plants. (Courtesy D. Hallock)

Molybdenum

Deficiency of molybdenum may occur if the soil is extremely acid. The symptom produced, however, is that of nitrogen deficiency. Apparently, the critical level of molybdenum needed for nitrogen fixation is greater than the level needed for other physiological processes.

Boron

Severe deficiency of boron causes leaves to turn deep green. Water-soaked areas may develop, and lesions may occur on leaves, petioles, and stems. Growth is restricted: terminal leaves become small and deformed, internode length is reduced, and secondary branching occurs, making the plant appear stumpy and short. The lower branches may split. Flowering and pod production are reduced. The pods of certain varieties may exhibit fine cracks. Lateral root growth is restricted, and root tips become swollen.

The most common boron deficiency symptom in field-grown peanut plants occurs in the pod. Seed do not develop properly. The inner face of the cotyledon is depressed in the center, and that region often turns brown, especially when roasted. The symptom has been termed "hollow heart" (Fig. 76).

Boron toxicity symptoms are likely to occur if more than 0.5 kg of boron per hectare is applied. Leaflet margins become chlorotic and then necrotic (Plate 148). In severe cases the leaves shed from the plant.

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(Prepared by N. L. Powell and F. R. Cox)

Ozone Damage

Ozone, sulfur dioxide, and fluorides are the major air pollutants likely to affect peanuts. Ozone is by far the most important air pollutant because it occurs throughout peanut production areas at concentrations that cause foliar injury and decrease yield. Classical foliar symptoms caused by sulfur dioxide or fluorides have been described for crops grown near industrial point sources. However, injury from sulfur dioxide or fluorides has not been documented for peanuts.

Sources

Ozone is formed in the troposphere by complex photochemical reactions between oxides of nitrogen and hydrocarbons that originate chiefly from gasoline engines and the burning of other fossil fuels. These primary pollutants are transported long distances by regional weather patterns. Concentrations of ozone are likely to be high enough to injure peanuts during calm, sunny days when primary pollutants from urban areas are present. Seasonal concentrations of ozone in the southeastern peanut-production areas are higher than in most other areas of the United States because of the frequent transport of nitrogen oxides and hydrocarbons from urban areas and the frequent periods of bright sunshine.

Symptoms

Ozone enters leaves through stomata during normal gas exchange. As strong oxidants, ozone or secondary products resulting from oxidation by ozone (e.g., free radicals) cause a variety of symptoms that appear within 2 days after exposure. Most symptoms are subtle and difficult to separate from those caused by normal senescence (e.g., chlorosis). Sometimes, however, a distinct, light tan, powdery appearance (fleck) and/or tiny, darkly pigmented areas (stipple) can occur on upper leaf surfaces between the veins. Both of these symptoms appear mostly on middle-aged leaves and are caused by the death of individual cells. With time and continuing daily exposure to ozone, these symptoms are gradually blurred by chlorosis and other symptoms of general senescence. Flecking, stippling, and symptoms of early senescence on peanut leaves occur in the field and have been duplicated by controlled exposures to ozone. Field chamber experiments have shown that ambient concentrations of ozone can decrease peanut yield in the southeastern United States.

A healthy peanut leaf from a plant grown in a field chamber receiving charcoal-filtered air (i.e., reduced ozone concentrations) and a leaf from a plant grown in the open that has injury caused by ozone are shown in Plate 149.

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(Prepared by A. S. Heagle)

Part III. Insects and Arthropods

Insects and related arthropod pests occupy every conceivable niche on the peanut plant. Insects feed in terminal buds and flowers; on leaves, roots, and pods; behind leaf axils and petioles; on plant fluids by inserting their mouthparts into cells or directly into the nutrient transport system; in tunnels that they form in the leaves, main stem, lateral branches or roots; and on or in peanut pods and seed. They damage the plant directly by removing photosynthate that would otherwise be used for vegetative or reproductive plant growth; damaging cells in photosynthetically active tissue; removing foliage that produces photosynthate; feeding on developing pegs, pods, and seed; damaging the root-hypocotyl region by the removal of periderm, cortex, and phloem tissue; and increasing the rate of water loss from an injured, stressed plant. To add to this biological complexity, arthropod pests generally do not occur in single-species groups but in groups of several species.

Equally important as the direct damage to plants by insects is the indirect damage that may result from the injury they cause. Insects are vectors of plant pathogens such as the groundnut rosette, bud necrosis, and tomato spotted wilt viruses. In addition, injured plant tissue is vulnerable to secondary infection by pathogens such as *Aspergillus flavus* and *Sclerotium rolfsii*. Economic losses attributable to plant pathogens transmitted by insects often exceed losses caused by the insects themselves. For example, the peanut rosette virus, transmitted by *Aphis craccivora* Koch, was credited with decimating peanut production in West Africa in 1975. Likewise, the plant viruses that cause bud necrosis and tomato spotted wilt are transmitted by thrips and cause significant crop losses in Asia and the United States, respectively.

The feeding by multiple species of insect pests can best be understood by grouping the insects into feeding guilds based on the plant's physiological response to the injury they cause. There are six categories of plant injury or crop damage caused by insect feeding: stand reduction, leaf-mass removal, assimilate removal, water balance disruption, pod destruction, and architectural modification.

For the purpose of this discussion, insects and other arthropods that cause damage to peanut are divided into the following guilds: foliage feeders, intracellular feeders, root and pod feeders, and stored-product feeders. A general description of the injury caused by arthropod pests and its physiological effect on the plant is presented with a brief description of selected species in each guild.

Foliage Feeders

The foliar feeding guild of insects that injure peanut is composed primarily of immature lepidopterans. Plant injury by these larvae is diverse. Examples include the groundnut leafminer, *Aproaerema modicella*, which initially feeds between the epidermal layers of the leaf and forms mines while feeding on the mesophyll tissue; the rednecked peanutworm, *Stegasta bosqueella* (Chambers), which feeds almost exclusively within a developing terminal; and the corn earworm, *Helicoverpa zea*, which consumes terminals, young foliage, flowers, and immature pegs. The major mechanism of yield reduction in legumes such as peanut, caused by feeding of foliar insects, is the removal of photosynthetically active tissue, which thereby reduces the production of photosynthate. Many lepidopterous defoliators of peanut prefer to feed on young leaves and terminals found in the upper plant canopy. The upper canopy is more active in light interception and production of photosynthate than shaded leaves of the mid- and lower canopy.

Armyworms

Several species of armyworms are major defoliators of peanut, including the fall armyworm, *Spodoptera frugiperda* (J. E. Smith) (Plate 150), and the beet armyworm, *S. exigua* (Hübner) (Plates 151 and 152), in the United States; the tobacco armyworm, *S. litura* (Fabricius) (Plate 153), in Asia; and the African armyworm, *S. littoralis* (Boisduval), in Africa and western Asia. Armyworm moths lay masses of 20–1,000 eggs on the leaves and stems of peanut (Plate 154). The egg masses often are covered with body scales and silk webbing, giving them a green to golden bronze color. Newly emerged larvae feed cryptically on the undersides of leaflets, in terminals, or between the leaf petioles and stems while older larvae feed openly on the plant on terminals and younger leaves. Older *S. litura* larvae feed primarily at night and hide under debris or soil clods at the base of the plant during the day. The fall armyworm prefers to feed on young leaf tissue and consumes about 100 cm^2 of foliage during larval development, 80% of which is consumed during the last two instars. Fall armyworm larvae require almost twice as much foliage to complete development when feeding on older foliage than on younger foliage. In India, defoliation of peanut by *Spodoptera* larvae has a greater effect on yield during the dry season (December–April) than during the rainy season (June–October). This difference in susceptibility to injury probably occurs because of the plant's inability to compensate for the effects of defoliation during the short, cooler, dry season.

Armyworm larvae feed on a wide variety of cultivated and wild host plants. Mature larvae may reach a length of 3–4 cm, have a characteristic inverted Y pattern on their head capsules, and vary from light green to brown or black with longitudinal stripes along their sides. These insects are called armyworms because young larvae feed together on a host and crawl en masse to adjacent fields after all the foliage in one area has been consumed.

Helicoverpa (*Heliothis*) spp.

Helicoverpa larvae are plant pests in all temperate, tropical, and subtropical regions of the world. In the New World, the corn earworm, bollworm, or tomato fruitworm (all approved common names), *Helicoverpa zea* (Boddie) (Plate 155), and the tobacco budworm, *Heliothis virescens* (Fabricius), are the most important pest species. In Africa and Asia, the most important pest species is the old world bollworm or gram pod borer, *Heliothis armigera* (Hübner) (Plate 156). On peanut, the

eggs are laid singly, primarily on the undersides of leaves in the outer plant canopy. Larvae of all these species feed on flowers and pods of numerous hosts including cotton, okra, pigeonpea, peanut, soybean, and cowpea. Early instars feed in leaf terminals while older larvae feed openly on terminals and young foliage. *Helicoverpa* larvae reach a length of almost 4 cm and may be rose pink, yellow green, brown, or almost black on their dorsa, with alternating light and dark longitudinal stripes on their sides and a lighter color on their undersides. Spiny projections are also quite noticeable on the surfaces of *Helicoverpa* larvae. A single corn earworm larva consumes 175–200 cm^2 of peanut foliage during development, of which 75–97% is consumed by the last two instars. In India, *H. armigera* feeds primarily on flowers, and its greatest effect on peanut is to extend the fruiting period of the plants.

Velvetbean Caterpillar

The velvetbean caterpillar, *Anticarsia gemmatalis* Hübner, is a tropical to subtropical New World pest. It does not overwinter in peanut-producing regions of the United States and survives the winter only in the most southern latitudes. The moths migrate northward each year. Damage by the velvetbean caterpillar (Plate 157) in the United States is most severe in northern Florida and southern Georgia, where high larval densities often occur on peanut late in the growing season. Eggs are usually laid singly on the undersurfaces of leaves. Larvae feed openly on the plant, initially consuming terminals and young foliage. With increased larval age, this preference declines, and larvae consume leaves of all ages. They are voracious feeders on peanut, soybean, kudzu, and velvetbean and may rapidly and completely defoliate plants. A larva consumes approximately 100 cm^2 of leaf tissue during its development.

Larvae of the velvetbean caterpillar reach a maximum length of 4.5–5 cm and are characterized by a yellow head capsule and a body color ranging from light green to black with yellow to white longitudinal stripes along the entire length of the body. The last pair of prolegs projects backward, and the legs are prominent when larvae are at rest. When larvae are disturbed, they often drop to the ground and thrash from side to side in rapid, twitching contortions characteristic of the insect.

Groundnut Leafminer

The groundnut leafminer, *Aproaerema modicella* Deventer, is a primary pest of peanut in India and Southeast Asia. The hosts of this leafminer are primarily legumes, and peanut and soybean are among its most important crop hosts. Moths deposit eggs singly on the undersides of leaves or on petioles and stems. First-instar larvae tunnel into the leaflet and feed on the mesophyll between the upper and lower epidermis, forming blotch mines. Severe infestations may result in complete loss of photosynthetic tissue and defoliation as leaflets turn brown, shrivel, and desiccate. Third-stage larvae leave their mines, web two or more leaflets together, and continue to consume foliage as they complete their development (Plate 158). At maturity, larvae reach a length of 6–8 mm and pupate within webbed leaflets. Populations of leafminers increase during the rainy season and may become severe pests during the pod-filling stage. Problems caused by this pest may intensify when irrigation allows peanut production to extend beyond the rainy season into the dry season. Moths then move from fields with mature plants to fields with immature plants, which are particularly susceptible to damage by this species.

Hairy Caterpillars

Arctiid larvae of the genus *Amsacta* are among the most important defoliators of peanut in India, although they produce only sporadic losses on peanut. The red-headed hairy caterpillar, *A. albistriga* Walk. (Plate 159), is most important in southern India, and *A. moorei* (Butler) is most important in northern India. These insects are named "hairy caterpillars" because of the numerous, long hairs on the bodies of older larvae. Hairy caterpillars have one generation per year. Moths are brownish white with a wing span of 40–50 mm (Plate 160). They emerge shortly after the first rains of the rainy season and lay clusters of 50–100 eggs in or around any available plant. As a peanut seedling emerges, young larvae move and feed en masse on the undersides of leaves. Older larvae disperse through the field and feed on terminals, leaves, and flowers. Larvae reach a length of 5 cm and may completely defoliate all peanut plants in a field before migrating and feeding in an adjacent field.

Intracellular Feeders

Peanut hosts a number of intracellular feeders including aphids, leafhoppers, thrips, mites, and whiteflies. These insects generally have rasping or piercing-sucking mouthparts and directly damage plants by consuming photosynthate. Several of the intracellular feeders, especially thrips and aphids, are vectors of plant pathogens. Feeding by the potato leafhopper, *Empoasca fabae*, on leaves reduces photosynthesis and increases respiration. A reduction in photosynthesis occurs immediately after *E. fabae* feeds. The duration of photosynthesis reduction is related to the length of the feeding period, the stage of plant development, and the stage of leafhopper development. Injured plants partially recover photosynthetic activity, but the reduction is permanent, affects all stages of plant development, and is most pronounced in bloom and postbloom growth stages. In plants damaged by the twospotted spider mite, *Tetranychus urticae*, both photosynthesis and transpiration are reduced in severely damaged cells while moderate damage may increase transpiration and cause development of smaller, deformed leaves with a lower chlorophyll content. Increased leaf transpiration also increases water loss, resulting in plant stress and closure of leaf stomates.

Leafhoppers

Several species of leafhoppers, especially members of the genus *Empoasca*, are pests of peanut. These include the potato leafhopper, *E. fabae* (Harris) (Plate 161), in North America; the groundnut jassid, *E. kerri* Pruthi (Plate 162), in India; and *E. facialis* Jacobi and *E. dolichi* Paoli in West Africa. Both adult and nymphal leafhoppers feed primarily on the undersides of peanut leaflets or leaves by inserting their mouthparts into the midribs and extracting plant fluids. Adult leafhoppers are 3–5 mm long, wedge shaped, and light green to yellow. Leafhopper feeding causes leaves and leaflets to turn yellow from the point of injury to the tip in a typical V shape (Plate 163), probably as a result of the injection of salivary toxin or toxins before feeding. The symptom, called "hopper burn," may become so severe that the injured area dies. In the United States, the potato leafhopper does not survive the winter in northern latitudes and overwinters only in mild climates along the gulf coast. Adults disperse northward during the growing season on wind currents associated with weather fronts, reaching as far north as Canada by early June. In the United States, injury to peanut by leafhoppers is generally most severe during June and July; in India, injury is most severe during August and September of the rainy season and February and March of the dry season.

Thrips

In the United States, the tobacco thrips, *Frankliniella fusca* (Hinds) (Plate 164), is the most abundant species of thrips on peanut, while in Southeast Asia, *Scirtothrips dorsalis* Hood and *Thrips palmi* (Bagnall) are the most abundant species on this crop. Thrips are extremely small (1.5–2 mm), delicate insects that feed in peanut leaf buds and flowers. Eggs are depos-

ited in plant tissue and hatch in 5–7 days. Immature stages resemble the adults but lack the fringed wings. Immatures are pale yellow to white, while adults range from light yellow to gold to black. Thrips have rasping mouthparts and feed by scraping the upper surfaces of developing terminals and imbibing the exuded fluid. Thrips injury to terminals results in deformed leaves that are crinkled and slightly cupped upon emergence (Plate 165). Thrips damage to peanut foliage is most severe during the first 30 days after plant emergence. Once plants begin to bloom, most thrips are found in the peanut flowers.

Results from several studies indicate that thrips injury to peanut does not significantly decrease pod yield. However, two recent findings have altered opinion on the importance of thrips. Thrips injury and herbicide injury to seedlings interacted to significantly reduce main stem height, canopy width, yield, and value of peanut. Secondly, and probably most importantly, thrips transmit the bud necrosis and tomato spotted wilt viruses, both of which cause important diseases in peanut. However, control of thrips does not reduce virus incidence.

Aphids

Several aphid species have been reported on peanut, but the cowpea or groundnut aphid, *Aphis craccivora* Koch, is the most important. This aphid occurs in the United States, Asia, and Africa, but it is a major vector of the groundnut rosette virus only in Africa. The adult aphid is black, rounded, and slightly oblong with brown legs, a prominent cauda, and thin, black cornicles. They reproduce asexually, and nymphs are dark brown. As populations increase on peanut, winged aphids are produced and disperse to form new colonies. Nymphs and adults feed primarily on new tissue including leaf buds and unfurling leaves (Plate 166), pegs, and flowers (Plate 167) by inserting their piercing-sucking mouthparts into the phloem and extracting sap.

Twospotted Spider Mite

The twospotted spider mite, *Tetranychus urticae* Koch, is a major pest of peanut in the United States, especially in Virginia and North Carolina. The adult mite is approximately 0.5 mm long and light green to yellow with two black dots on the dorsum. The mites injure plants by inserting their piercing-sucking mouthparts into plant cells and sucking out the contents. They overwinter as diapausing females or, in areas with a mild winter, as actively reproducing adults. In the spring, mites initially feed on early hosts and then become established on corn. Populations increase rapidly on tasseling corn and then disperse as it begins to senesce. Dispersal from senescing corn occurs during flowering and pod development on peanut, stages that are favorable for mite establishment and population increase (Plate 168). Mite outbreaks in peanut are thought to be induced by application of fungicides and insecticides; the interaction between the two pesticides reduces the number of natural enemies that regulate mite populations on peanut.

Whiteflies

The sweetpotato whitefly, *Bemisia tabaci* (Gennadius), is a relatively new pest of peanut in the United States, but it has been a pest of peanut in India for many years. The first whitefly infestations on peanut in the United States coincided with the identification of a new strain, B, that devastated vegetable crops in Florida, California, Arizona, and Texas during 1987 and 1988. Strain B was also found on peanut in Georgia. Recent research on strain B of the sweetpotato whitefly has shown that it may be a new species, and the names silverleaf whitefly and *B. argentifolii* Bellows & Perrin have been proposed. This whitefly is a serious threat to peanut because of its ability to increase its population rapidly, its occurrence on the undersides of leaves where it is difficult to control with insecticides, its resistance to a number of insecticides, and its role as a vector of plant viruses.

The silverleaf whitefly lays its eggs on the undersides of peanut leaves, and immatures can be found on both the upper and lower leaf surfaces, which does not occur on most other hosts. The nymphs are distributed equally among the tetrafoliates of a peanut leaf and pass through four nymphal instars. They are most abundant on leaves three, four, and five, and then abundance declines as leaf age increases. Adult whiteflies are white and approximately 2 mm long and are found on the lower leaf surfaces (Plate 169). Plant damage is by direct removal of photosynthate from the phloem and by the production of honeydew, which drips onto leaves below the nymphs and is colonized by a sooty mold, *Capnodium* spp., that reduces light interception and photosynthesis.

Root and Pod Feeders

Several soil-inhabiting insects and other arthropods are key pests of peanut worldwide and represent a diverse group of species, including white grubs, wireworms, earwigs, ants, lepidopterous larvae, termites, and millipedes. These pests feed on roots, stems, and pods, causing stunting; decreased leaf area; diminished root, pod, and seed dry weight; reduced xylem pressure; reduced photosynthate flow in the phloem; decreased nitrogen fixation; and wilting of plants caused by insufficient transport of water. In addition, injury to plants by insects predisposes them to invasion by secondary pathogens such as *Aspergillus flavus*.

Lesser Cornstalk Borer

The lesser cornstalk borer, *Elasmopalpus lignosellus* (Zeller) (Plate 170), is among the most important insect pests of peanut in the United States. This insect is a pest of numerous crops, including peanut, when soil moisture is deficient; it seldom causes damage when soil moisture is high. This insect also is more important as a pest on peanut grown in sandy soil than in heavier soil that has a higher organic content and greater water-holding capacity. Soil moisture affects every stage of *E. lignosellus*. Under wet conditions, moths lay eggs singly on the plant and larvae feed between leaf axils and stems or in flower buds. Under dry conditions, moths lay eggs just below the soil surface around the base of the plant and larvae feed under leaves touching the ground or on the main stem at or just below the soil surface. Larvae form feeding tubes in the soil from which they feed on roots and developing pods. They also may tunnel through the main stem or lateral branches of plants. Moths are approximately 12.5 mm long; females are charcoal gray to black, often with brown markings toward the anterior (Plate 171), and males are buff to light yellow with charcoal gray bands on their wings (Plate 172). Young larvae are bright red, and older larvae have dark mahogany and blue green alternating bands around their bodies and may reach 2 cm in length. Larvae prefer to feed on immature peanut pods and damage developing seed when they penetrate. Older larvae feed externally on more mature pods by scarifying the exocarp without actually penetrating the pod (Plate 173). This external damage to pods is sufficient to enhance infection by *A. flavus* and the formation of aflatoxin.

A model for predicting an infestation by the lesser cornstalk borer (LCB) has been developed. The model is used to calculate LCB days, i.e., the number of hot, dry days (on which the temperature is at or above 35°C and rainfall is less than 2.54 mm) minus the number of cool, wet days (on which the temperature is less than 35°C and rainfall is at or above 2.54 mm). Scouting for LCB damage is recommended when the running total of LCB days approaches zero. Values below zero indicate that the LCB is unlikely to be a problem in peanut, while values of 10 or greater represent conditions under which outbreaks of this insect are likely to occur.

Southern Corn Rootworm

The southern corn rootworm, *Diabrotica undecimpunctata howardi* Barber, is a major pest of peanut in Virginia and North Carolina and of peanut grown on poorly drained soils in the southeastern United States. The adults, often called spotted cucumber beetles, are about 6–6.5 mm long and are easily recognized by the twelve spots on their greenish yellow elytra (Plate 174). Adults prefer to oviposit at the bases of plants in moist, dark soil with moderate levels of organic material and clay. The presence of weeds among peanut plants increases oviposition. The larvae are slender, fragile, and white with dark brown to black head capsules and anal plates and may reach a length of 12.5–18.0 mm at maturity (Plate 175). Adult rootworm beetles feed above the ground on peanut leaves, and larvae feed below ground on roots or developing pegs and pods. Larvae make almost cylindrical entry holes and feed on the immature pods and developing seed. Damage by rootworm larvae also predisposes pods to invasion by fungi that induce pod rot. Three to four generations may occur each year in the southern latitudes, while only one generation occurs in the northern latitudes.

White Grubs

White grubs are the larvae of scarab beetles and are considered among the most important pests of peanut worldwide, especially in the developing nations of Africa and Southeast Asia. Several species of the genera *Lachnosterna*, *Adoretus*, *Anomala*, *Eulepida*, *Leucopholis*, and *Schizonycha* feed on peanut roots and pods. Adult beetles, called cockchafers, are fairly large, 18–20 × 6 mm. They emerge from the soil at dusk during the first few weeks of the rainy season. They often congregate on trees to mate and feed (Plate 176) before returning to the soil to lay their eggs. Eggs are laid singly or in small clusters 5–15 cm below the soil surface. Larvae are C shaped and white with brown to black head capsules and anal plates (Plate 177). Larvae of some species are up to 50 mm long and 20 mm in diameter. Older (third-instar) larvae may feed on the taproots, resulting in patches of stunted, wilted, or dead plants. Most injurious species of white grubs on peanut have one generation per year.

Termites

Termites (Plate 178) of many genera, especially *Odontotermes* and *Microtermes*, are major pests of peanut in Africa and Asia. Termites tend to be a major problem on peanut during periods of insufficient rainfall, whereas white grubs are more of a problem in those areas where soil moisture is adequate for proper plant growth. Harvester termites feed at the bases of stems and, like beavers, can "fell" the whole plant. Other species cover a plant with soil and feed on the leaves, but the greatest damage is caused by species that feed on pods or tunnel into the taproot, main stem, or lateral branches. Termites damage pods in two ways: by externally scarifying the pod without pod penetration (Plate 179) and by penetrating the pod and feeding on the seed. Damage to pods is most severe late in the growing season when they are left in the soil past optimum maturity or during periods of insufficient rainfall. Damage by termites reduces yield and, equally important, enhances *A. flavus* invasion and aflatoxin contamination of seed.

Wireworms

Wireworms, immature stages of click beetles, are increasingly important pests of peanut in the southeastern United States. Several species of the genus *Conoderus* have been collected from peanut; *C. scissus* (Schaffer) and *C. amplicollis* (Gyllenhal) are encountered most frequently. Wireworms have life cycles lasting 1–6 years, depending on the species. They tend to be more of a problem in moist soils and during years of higher than normal rainfall. Adult click beetles emerge from the soil during spring to early summer and lay eggs in the soil close to a plant host. Wireworm larvae are slender, hard-bodied insects with three pairs of inconspicuous legs on the front half of the body. They range in color from yellow to light brown and at maturity average 15–25 mm in length. Larvae feed on all underground parts of the peanut plant, but they are especially damaging to the pods (Plate 180). Wireworms make jagged entry holes in the pods and feed on the developing seed, often completely consuming the seed and leaving empty pods.

Millipedes

Millipedes are a serious pest of peanut in several developing nations, particularly in western Africa. Of the 13 species of millipedes that damage peanut in Senegal, *Peridontopyge* spp. are the most prevalent. Millipedes spend the dry season deep in the soil below stumps and in or under termite mounds. During the rainy season, more than 50% of the millipedes are found in the upper 10 cm of soil, whereas during the dry season, more than 90% are found below 10 cm. They emerge from the soil shortly after the first substantial rains of the wet season and feed on seedling plants, including peanut, often reducing plant density by up to 20%. They also feed on developing pods and may reduce yield by 30–40%. Millipedes primarily attack immature, developing peanut pods, while termites attack more mature pods.

Stored-Product Feeders

Peanuts in storage are attacked by a variety of stored-product pests that can rapidly reduce seed quality. More than 100 species of insects and related arthropods infest stored peanuts. Most stored-peanut pests penetrate the pod and feed on the seed. However, several species have difficulty penetrating undamaged peanut pods, and thus seed damage tends to be more severe in damaged or cracked pods. Other pest species easily bore through the pod to feed on the protein- and oil-rich seed. Heavy infestations with stored-product insects may leave damaged seed or seed contaminated with frass, webbing, insects, or insect parts, all of which can make the product unsuitable for human consumption.

Indianmeal Moth

The Indianmeal moth, *Plodia interpunctella* (Hübner), is an important pest of stored peanut worldwide. Moths are readily distinguishable by the unusual color of the forewings; the front half of the forewing is white to gray while the outer one-half is reddish brown to purple with a copper luster (Plate 181). The wings are folded close to the body when the moth is resting, and each is marked with a prominent, reddish brown band. Adult moths have a wing spread of nearly 19 mm and a length of 8–13 mm when the wings are folded. Females lay eggs singly or in small groups. Larvae feed on shelled seed or on seed in damaged or cracked pods. Larvae form a silken, matted web on the seed; and under severe infestation, the entire surface of seed may be covered with webbing. At maturity, larvae average about 15 mm in length and are light yellow to creamy white, often with a pinkish hue. Damage is in the form of partially consumed seed, cast skins, and webbing on the pods and seed.

Rice Moth

The rice moth, *Corcyra cephalonica* (Stainton), like many other stored-product pests, is considered a secondary pest of stored peanut because it is unable to penetrate and damage seed in sound pods. Moths of this species are gray brown to tawny with a wing span of 14–24 mm and a length of 12–15 mm with the wings folded along the sides of the abdomen (Plate 182). Veins in the wings are slightly darkened. Eggs are laid singly, and larvae feed on loose, shelled seed or on seed in cracked pods (Plate 183). Larvae form silken tubes that are attached to the seed on which they feed. They also spin a dense, tough, silken cocoon for pupation. A mature larva is

about 15 mm long and white to dirty gray with numerous long hairs and a dark brown head and pronotum.

Flour Beetles

The red flour beetle, *Tribolium castaneum* (Herbst), and the confused flour beetle, *T. confusum* Jacquelin du Val, average 3–4 × 1.5–2.0 mm and are oblong and slightly flattened, reddish brown beetles that attack stored peanut worldwide (Plate 184). The two species are very similar in appearance, habits, and life history. An adult may lay up to 450 eggs among peanut pods and seed. Larvae are yellowish white with a brown head capsule and forked anal plate (Plate 185). Both adults and larvae feed on the surface of peanut seed and burrow into the seed. As a result of this feeding, seed become powdery and dusty and are unfit for human consumption. This damage also increases the percentage of split seed when pods are shelled.

Groundnut Bruchid

The groundnut bruchid, *Caryedon serratus* Oliver, is an important pest because it can attack unshelled, undamaged peanut pods. It is a major pest of peanut in Asia and Africa. The adult is 4–7 × 5 mm, dark gray to brown, and slightly mottled and has large hind legs. Eggs are laid on the pods, and larvae chew directly through the eggshell and pod to feed on the seed. Thus, damage is not visible externally on the pods without careful observation. Often the first evidence of infestation is a hole cut in the pod by a larva before it pupates; the hole allows an adult to emerge from the pod. Larvae sometimes emerge and pupate outside an infested pod (Plate 186). Infestation may occur shortly after plants are inverted and while the pods are drying in the windrow or while the pods are stored in the open during the dry season.

Pod-Sucking Bug

The "Wang," *Elasmolomus sordidus* (Fabricius), which may attack peanut pods while they are drying in the field or during storage, is a widespread pest of peanut in India. The adult is a dark brown, 10- × 2-mm, typical lygaeid-shaped bug with long legs and antennae (Plate 187). The adult pierces a pod with its long, slender mouthparts and feeds on the oil in the enclosed seed. This injury causes the seed to become wrinkled with dark spots and increases the probability of rancidity. In the field, the eggs are laid either in the soil or on vines, and in storage they are laid among the pods. First-instar nymphs have bright red abdomens, while later instars become progressively darker.

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(Prepared by R. E. Lynch, J. A. Wightman, and G. V. Ranga Rao)

Part IV. Other Organisms

Parasitic Flowering Plants

Alectra vogelii

Alectra vogelii Benth. (Scrophulariaceae) is a root parasite of peanut and several other leguminous crop plants. It has been reported in various countries in Africa (Angola, Burkina Faso, Malawi, Mozambique, Nigeria, Swaziland, Zambia, and Zimbabwe). A high disease incidence (about 90%) has been reported in Burkina Faso and Malawi. *A. picta* Hemsl. parasitizes peanut in glasshouse experiments.

A mature plant of *A. vogelii* reaches a height of about 0.5 m with stems branching out at the base (Plate 188). Flowers are a prominent lemon yellow with horseshoe-shaped stigmata. Roots are orange and poorly developed. The connection between the parasite and the peanut roots can be seen by carefully removing the soil in the root zone (Plate 189).

Parasitized peanut plants become stunted, and yields are reduced. The potential pod yield loss has been estimated at about 40% in Nigeria.

Striga spp.

More than 60 species of *Striga* (Scrophulariaceae) have been reported as parasites of several cereal and leguminous crop plants. *S. hermontheca* Benth. has been reported on peanut in West Africa and *S. gesnarioides* (Willd.) Vatke (witchweeds) on peanut in Mozambique and on *Arachis repens* in Nigeria.

S. hermontheca is a cross-pollinated species with wide variation in plant type and floral morphology. It is an annual, erect herb reaching a height of about 0.5 m (Plate 190). Leaves are green and 20–60 mm long. Flowers are sessile, irregular, and bright pink. The calyx is distinctly five ribbed, and the corolla tube, 11–17 mm long, bends characteristically at an angle immediately over the tip of the calyx. Bracteoles are 2–3 mm wide. The fruits (capsules) contain vast numbers of minute seed.

S. gesnarioides is an annual, erect herb reaching a height of about 0.15 m (Plate 191). Leaves are scalelike, rarely exceed-

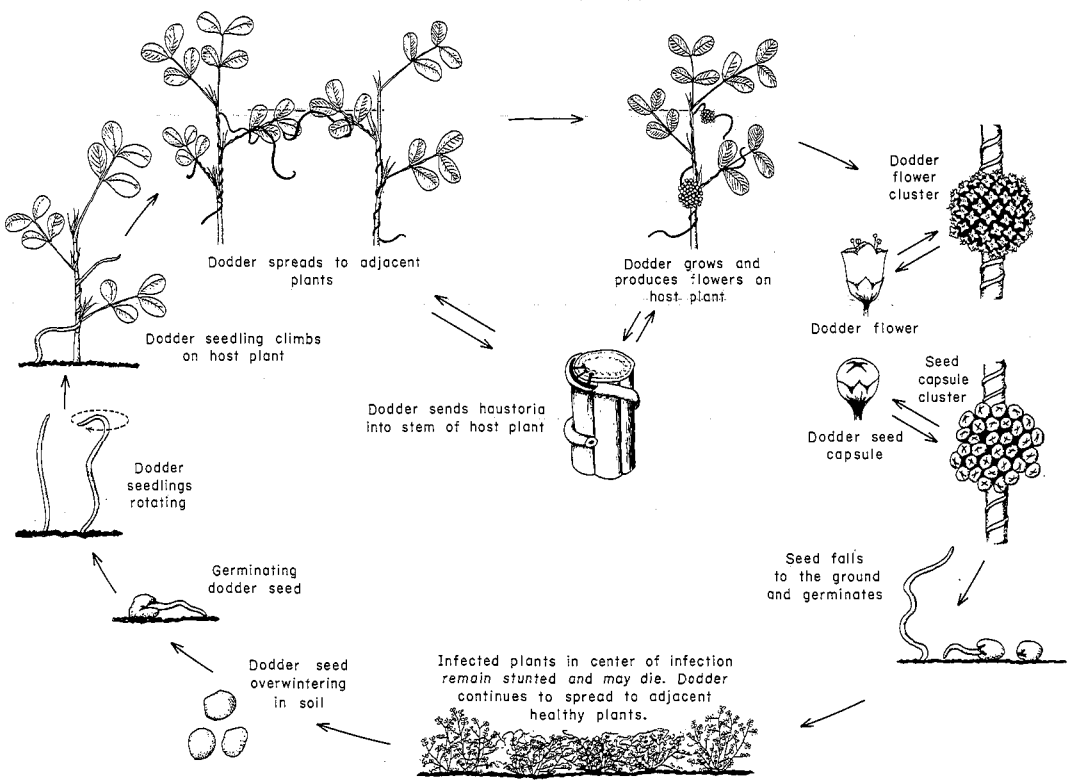

Fig. 77. Life cycle of dodder. (Modified and reprinted, by permission, from G. N. Agrios, 1978, Plant Pathology, 2nd ed., Academic Press, New York. Prepared by Nancy Browning)

ing 5 mm in length. Compact branches arise from ground level. The plant forms a large haustorium (feeding structure) with the host root, unlike *S. hermontheca.* Flowers are irregular and vary greatly in size and color but are usually creamy white, bluish, or pink.

Cuscuta campestris

Cuscuta campestris Yunck. (Convolvulaceae) (dodder) is a stem parasite that attacks a wide range of flowering plants. It is a parasite but not an important pest of peanut in the United States. *C. campestris* lacks true roots and leaves and produces a tangle of wiry branches (Plate 192) that coil around the branches of host plants and produce haustoria. The branches are orange to golden yellow and devoid of chlorophyll. Minute, bell-shaped flowers are produced in small clusters. The life cycle of *C. campestris* is outlined in Figure 77.

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(Prepared by P. Subrahmanyam)

Beneficial Organisms

Mycorrhizae

The peanut root, like roots of most other herbaceous plants, is commonly colonized by vesicular-arbuscular endomycorrhizal fungi (Figs. 78 and 79). Species of the genera *Glomus, Gigaspora, Scutellospora, Sclerocystis,* and *Entrophospora* have been reported to be naturally associated with peanuts. The association is characterized by the formation of arbuscules (haustoriumlike structures) in the roots and of chlamydospores and azygospores (Fig. 80; Plates 193 and 194) in the roots and soil. Sporocarps may also be formed in the soil.

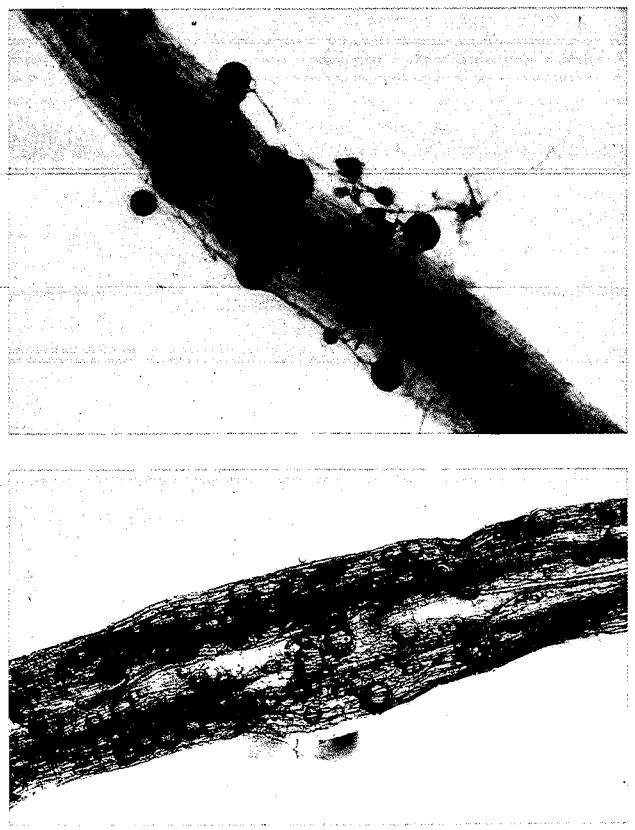

Fig. 78. Vesicular-arbuscular mycorrhizal fungi in peanut roots. (Courtesy M. Yeh)

Most research has focused on the effects of these fungi on growth of inoculated plants in the greenhouse and in sterilized soils. In general, mycorrhizal fungi have a positive effect on peanut growth. Individual species differ significantly in their effectiveness in promoting growth. Growth response may be enhanced by inoculation with mixtures of glomalean fungi and/or *Bradyrhizobium.* Vegetative growth has been enhanced by more than 300% in peanuts inoculated with various species. Some reports indicate increases in seed yield. Other reports indicate no positive response. Experiments have been conducted on the effects of metals, phosphorus, water, pesticides, and other soil microorganisms on the activity of mycorrhizal fungi associated with peanuts.

Progress in research pertaining to endomycorrhizal fungi and their effects on peanuts (and all other plants) has been hampered by the fact that the taxonomy of these glomalean fungi is little understood. Identification of species is difficult. It involves interpretation of spore color, spore size, structure and chemical reactions of cell wall layers, presence or absence of sporocarps, and morphological characteristics of the sporocarps. None of the species can be grown and maintained in pure culture in the laboratory. Cultures must therefore be grown in association with living host roots in the greenhouse and separated from the soil or other growth medium for use as

Fig. 79. Hyphae of a mycorrhizal fungus in a peanut root (note penetration site). (Courtesy D. M. Porter)

inoculum. In the future, it will be necessary to reevaluate the ecology and importance of individual species on the basis of new species concepts.

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(Prepared by R. A. Taber)

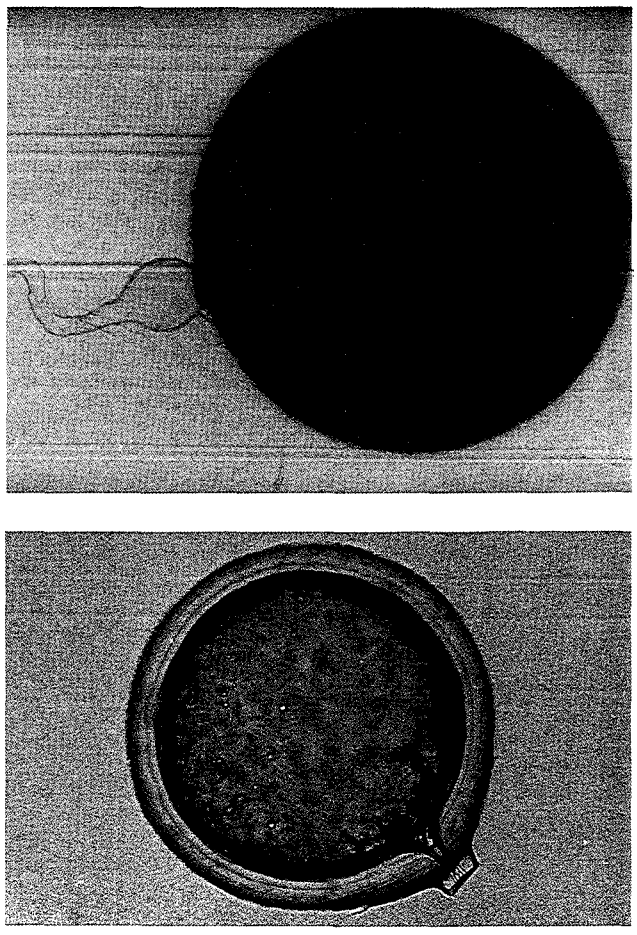

Fig. 80. Azygospore of *Gigaspora* sp. (top) and chlamydospore of *Glomus* sp. (bottom).

Rhizobia

Bacteria of the genus *Rhizobium* infect peanut and other legume roots, establishing a relationship that is generally beneficial to both the bacteria and the plant. The bacteria, which are abundant in soils where peanuts have been previously grown, can assimilate or fix atmospheric nitrogen in a symbiotic relationship with the peanut plant.

Although both symbiotic and nonsymbiotic bacterial nitrogen fixation (BNF) occur among a diverse array of prokaryotes, the former is much more important. It has been estimated that symbiotic nitrogen fixation can yield 30–300 kg of nitrogen per hectare per year, while nonsymbiotic fixation yields only 1–3 kg of nitrogen per hectare per year. This difference is caused by the high requirements for energy, varying from a minimum of 12 moles to 29 moles of ATP consumed in reducing 1 mole of N_2. Since the major source of this required energy is photosynthate, the symbiotic systems are much more efficient. It has been estimated that about 1–2 metric tons of plant carbohydrate (from photosynthesis) may be consumed annually in BNF.

It is estimated that 60 million metric tons per hectare of nitrogenous fertilizer are used globally, while annual BNF is estimated to be about 175 metric tons per year. Symbiotic nitrogen fixation by agricultural legume crops is about 50 metric tons per year. Peanuts possess a comparatively high symbiotic nitrogen-fixing capacity. Field studies with N_{15} isotope methodology showed 68–152 kg of nitrogen fixed per hectare per season. Of the total plant nitrogen, 54–90% was derived from BNF. The totals varied depending upon the *Bradyrhizobium* strain used, the peanut cultivar, and cultivar-strain interaction. The bacterial strain used, however, accounts for the larger part of the variability observed for nodulation and nitrogen fixation.

Early researchers considered all rhizobia to be a single species capable of nodulating all legumes. Extensive cross-testing on various legume hosts led to taxonomic characterization of rhizobia based on bacteria-plant cross-inoculation groups, which were defined as "groups of plants within which the root nodule organisms are mutually interchangeable." The concept of cross-inoculation groupings as taxonomic designators has gradually fallen into disrepute, although some of this philosophy is retained in the current taxonomic scheme.

Bacteria of the family Rhizobiaceae lack endospores and are normally rod shaped, aerobic, and gram negative. They are motile and have one polar or subpolar flagellum or two to six peritrichous flagella. The bacteria utilize many carbohydrates, and considerable extracellular slime may be produced during growth on carbohydrate-containing media. Best growth is usually achieved at 25–30°C on relatively simple heterotrophic media. A number of monosaccharides serve as suitable energy sources, and several organic or inorganic nitrogen sources can be used. Dinitrogen is the primary nitrogen source in symbiosis with a leguminous host. Some strains of rhizobia show a close relationship in DNA base composition with bacteria of the genus *Agrobacterium*.

Traditionally, rhizobia have been divided into two groups according to growth rate: "fast growers" and "slow growers." Fast growers are the rhizobia commonly associated with alfalfa, clover, bean, and pea, which in culture grow much faster (less than one-half the doubling time of slow growers or less than 6 hr); slow growers are exemplified by soybean and cowpea rhizobia (generation time greater than 6 hr). Most bacteria that infect peanut roots are slow growers, often with a mean generation time exceeding 12 hr. Although there is phenotypic and genotypic diversity within these major groupings and some overlap, numerous studies have demonstrated the validity of this classification.

The relative fastidiousness of the slow growers has been substantiated by recent studies. While the major biochemical

pathways seem to be similar, evidence suggests that the preferred nitrogen-fixation pathway may be different. Analyses (16S RNA) of the fast- and slow-growing rhizobia confirm that these groupings indeed represent different genetic phyla. Recent studies in which numerical taxonomy, carbohydrate metabolism, antibiotic susceptibilities, serology, DNA hybridization, RNA analysis, and DNA base ratio were used all demonstrated the validity of the fast- and slow-growing groupings.

The taxonomy of the rhizobia is in a state of transition. As more molecular information accumulates, such genetic data will, no doubt, further displace cross-inoculation approaches to classification. On the basis of the difference between the fast- and slow-growing rhizobia, the traditional rhizobia have been divided into two genera. The slow-growing strains were placed in the genus *Bradyrhizobium,* containing two species, *B. japonicum* and *B. elkanii,* which nodulate soybeans. Other bradyrhizobia occur (e.g., the peanut bradyrhizobia) but have not been classified to species or biovar levels. Until further taxa within the genus are proposed, these should be described with the appropriate host plant given in parentheses, e.g., the peanut rhizobia—*Bradyrhizobium* sp. (*Arachis*).

The fast-growing rhizobia have been placed in the genus *Rhizobium,* containing six species, *R. leguminosarum, R. meliloti, R. loti, R. galeage, R. fredii,* and *R. tropici.* Three former species, *R. phaseoli, R. trifolii,* and *R. leguminosarum,* have been combined into the species *R. leguminosarum. R. fredii* is a new species consisting of fast-growing rhizobia that effectively nodulate Chinese soybean cultivars ordinarily nodulated by *B. japonicum.*

Peanuts are able to form effective symbioses with a number of genetically diverse, slow-growing bradyrhizobia. Since many of the tropical and semitropical legumes are nodulated by bradyrhizobia, there exists a large diversity of potential symbionts, which probably accounts for the large strain variation in BNF.

Bacterial nodules arise in the junctions of lateral roots along both primary and secondary roots of the peanut. Possibly such areas provide favorable intercellular sites for rhizobial multiplication and penetration in the cortex, since no infected root hairs have been observed in peanut roots. Small indentations of the host cell wall, which may be the initial entry sites, occasionally are found. The nodule is recognizable before intracellular rhizobia can be found. Rhizobia are distributed in the nodules by cell division, and only invaded cells in the bacterial zone seem to divide. One possible consequence of such a mode of infection is that the plant has the ability to form nodules with a wide range of *Rhizobium* strains. Roots of most peanut genotypes nodulate readily with cowpea rhizobia (Plate 195). Symbiotic nitrogen fixation is a more specific property, however; and often the nodules are ineffective or inefficient.

Nodules on peanut roots are round or oval. The nodules may have a broad attachment to the root, with bacteroid tissue embedded in the root cortex so that the nodules appear hemispherical; the husk tissue may even grow around the subtending lateral root. Nodule number and development are determined by the interaction of the host and bacteria and are, furthermore, affected by competition for photosynthate among nodules on a plant, the distribution of plant hormones, and environmental factors such as moisture and temperature.

Rhizobia vary in their ability to fix nitrogen in peanut. The same strain can form effective nodules on one host plant and ineffective nodules on another of a different genotype. *Rhizobium* strains that lack the ability to form functional nodules can have an adverse effect on plant growth and may inhibit other strains with good nodulating ability. The interior of a fresh, effective nodule is dark red from the leghemoglobin that it contains, while ineffective nodules are pink, light green, or colorless.

Where legumes have never been grown or where several years have elapsed since they were grown, soils may be deficient in *Rhizobium* strains. When few bacteria are present, plants show typical nitrogen-deficiency symptoms—yellowing and low protein content of foliage (Plate 196) and low yields of pods and seed. When effective nodules are formed on young seedling roots, the plants grow rapidly and have adequate nitrogen to maintain green leaves. More than 200 kg of nitrogen per hectare can be fixed by well-nodulated peanut roots.

Environmental conditions that favor peanut plant growth also favor nodule development and nitrogen fixation. Nitrogen fixation is decreased by drought, low temperatures, and low light intensities.

When peanut is planted in fields that lack effective *Rhizobium* strains, a significant yield increase usually results from inoculation with an effective strain (Plate 196). Commercial *Rhizobium* inoculants are available and are best applied in the furrow at planting time. Once desirable strains are established in a field, it is not necessary to add new inoculum each year, since the rhizobia can survive on organic matter for several years.

When desirable strains of *Rhizobium* provide the peanut plant with adequate nitrogen, inorganic nitrogen fertilizer is of little use. Added nitrogen actually inhibits nodulation and reduces nitrogen fixation.

The infected tissue of the nodule ordinarily shows no change for some weeks or months and then eventually breaks down and disappears. Degeneration begins with tissue breakdown at the base of the nodule and progresses toward the apex until all of the nodule is destroyed. The nodule may enter the degenerative phase at an early stage if growth conditions are unsuitable for the host or if the bacteria and the host are inherently incompatible.

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(Prepared by G. H. Elkan)

Bacillus subtilis

Bacillus subtilis is well known for its ability to produce antibiotics. Since the 1950s, researchers have evaluated this bacterium for its potential as a biological control agent. During the early 1980s, researchers began to study P. Broadbent's A-13 isolate of *B. subtilis* as a possible biological seed treatment for peanuts. This isolate was compatible with most common seed-treatment fungicides, and when it was mixed with fungicides as a preplant seed treatment, improved vigor was demonstrated through earlier emergence and larger, more robust plants.

Germination studies revealed that seed treated with *B. subtilis* germinated earlier and produced longer taproots with more lateral roots than untreated seed. This response was more apparent at 20°C than at 27°C and was also more apparent on seed produced by environmentally stressed plants than on seed from unstressed plants. Since this response occurred in the

absence of seedborne pathogens (on seed treated with a fungicide) and in the absence of soil, the initial interpretation was that the bacterium must be stimulating plant growth through hormone production.

Field studies demonstrated that hormonal stimulation was one mechanism of growth enhancement of *B. subtilis*-treated plants. Yields were improved by 17% when *B. subtilis* treatments were combined with fungicides compared with fungicide alone. These data indicate that control of root-infecting fungi, such as *Rhizoctonia solani* AG-4, is a second possible mechanism for improved peanut growth. A third possible mechanism is improved nutritional status of plants resulting from enhanced nodulation by *Bradyrhizobium.* Significant increases in leaf and stem nitrogen and in boron and potassium were noted after treatment with *B. subtilis*. Vesicular mycorrhizae were unaffected.

A regional test conducted in 24 field locations in the southeastern United States with commercial seed-treatment application and planting procedures assessed yield improvements, the relationship of disease severity to yield, and the efficiency of root bacterization. All locations were effectively inoculated with *B. subtilis* at harvest, and yield improvements were related to rotational history and time of planting. The average yield increase was 7.6%. Locations that were planted to legumes in either of the previous 2 years averaged a 12% yield increase. Locations at which seed were planted early in cool soils averaged an 11.4% increase, while seed planted late in warm soils averaged a 6.8% yield increase when inoculated with *B. subtilis*. Areas with conditions that supported pathogens such as *Rhizoctonia* (poor rotations and cool, early-planted soils) showed the greatest yield improvements when *B. subtilis*-treated seed were planted.

Evaluation of mature plants grown from seed treated with *B. subtilis* strain A-13 revealed that only the upper 10 cm of the taproot was effectively colonized. Furthermore, branch roots more than 2 cm from the taproot were typically not effectively colonized. These data indicate that the yield benefits come about after colonization of only a small percentage of the total root mass.

B. subtilis forms highly stable spores, making it extremely resistant to environmental stress. Commercial preparations maintain their ability to inoculate for years. The A-13 strain of *B. subtilis* was adapted to cotton by multiple host passages and then renamed GB03. This strain is marketed under the trade name Kodiak for use on seven crops, including peanut and cotton. During 1994, less than 5% of the peanut crop was treated with this biological fungicide (and plant growth promoter), but more than 4 million acres of cotton were treated. For the 1995 season, it was projected that almost 90% of all cotton grown in the United States would be treated with this biological control agent.

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(Prepared by P. A. Backman)

Leaf-Surface Epiphytes

Although peanut foliage is host to numerous microorganisms, only a small fraction negatively interact with the peanut plant to the extent that we consider them to be pathogens. The other microbes on the leaf surface are poorly understood but do not appear to interact with the plant in a negative way. Plant pathologists have long recognized the existence of epiphytic microflora, but only recently has research been initiated to better describe and quantify this population. Interest in foliar microflora comes with a recognition of its potential application for foliar disease control. Although the influence of these epiphytes on the epidemiology of peanut diseases is largely unexplored, available evidence indicates a strong potential for using the epiphytic population for reducing disease. According to classic theory, biological control can be achieved through several mechanisms of antagonism, including competition, antibiosis, and parasitism or predation. Available evidence indicates that epiphytic populations can exhibit some of these mechanisms of biological control.

First, epiphytic microbes may reduce disease through competition by consuming nutrients or occupying infection sites. The degree of competition pathogens experience is unknown, but research is underway to characterize the microbial populations of peanut foliage. Microbial populations can reach one million or more per square centimeter (Plate 197). The epiphytic microflora of peanut leaves is quite complex and includes bacteria, yeasts, and filamentous fungi. For example, a large survey of epiphytic bacteria showed that *Methylobacterium, Curtobacterium, Rathayibacter, Bacillus, Clavibacter,* and *Aureobacterium* are the predominant bacterial genera on peanut foliage in North Carolina. The degree to which competition might reduce disease depends on the biology of the pathogen. For example, pathogens that use exogenous nutrients might be more inhibited by competition from epiphytic flora. More research is needed to fully understand the impact of microbial competition on foliar diseases.

Second, antibiosis by beneficial microorganisms may have a role in reducing disease. Field tests have been conducted with antagonistic bacteria for controlling *Cercospora arachidicola* on peanut. Foliar applications of *Bacillus thuringiensis* or *Pseudomonas cepacia* were applied at 14-day intervals during the season. Both organisms are strongly antagonistic to *C. arachidicola* in vitro. *P. cepacia* strains produce several antibiotics including pyrrolnitrin. The antagonistic mechanism of *B. thuringiensis* strain HD-1 is unknown, but *B. thuringiensis* is closely related to *B. cereus,* which does produce antifungal antibiotics. Disease control, although statistically better than that in unsprayed controls, was generally poor and was considered unacceptable compared with standard fungicide treatment. The poor survival of the biological control agents on foliage probably afforded little opportunity for antibiotic production on the leaf surface. Enzymatic mechanisms of antibiosis may hold more promise. When chitin amendments were applied to peanut foliage, chitinolytic bacteria increased from 1% of the total population to 40%. When a chitinolytic strain of *B. cereus* was applied to peanut foliage along with chitin, a 60% reduction in infection by *C. arachidicola* was observed. While it is clear that epiphytic microorganisms can reduce disease through antibiosis, more work is needed to develop the full potential of this type of control.

Third, mycoparasitism by foliar epiphytes may also play a role in reducing disease. In laboratory tests, *Verticillium lecanii* reduced infection of detached peanut leaves by *Cercosporidium personatum* and *Puccinia arachidis,* particularly when leaves were preinoculated with *V. lecanii*. In another study, *V. lecanii* and *Penicillium islandicum* were the most effective of several mycoparasites evaluated in the laboratory and field for biological control of *P. arachidis*. Direct parasitism of *C. per-*

sonatum by mycoparasites has been evaluated under field conditions.

In summary, while the epiphytic population has a clear potential for reducing disease, more research and development are needed before these epiphytes can be used as biological controls. For the present, these epiphytic organisms are perhaps best viewed as a factor that reduces the severity of foliar disease and allows greater flexibility in the use of other control measures.

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(Prepared by V. J. Elliott and H. W. Spurr, Jr.)

Dicyma pulvinata

The fungus *Dicyma pulvinata* (Berk. & M. A. Curtis) Arx (syn. *Hansfordia pulvinata* (Berk. & M. A. Curtis) S. J. Hughes) is a mycoparasite on the late leaf spot fungus, *Cercosporidium personatum*. It has not been reported on early leaf spot. It has been found in several countries, including the United States, where it was first found in Texas and later in Virginia and Florida. The fungus may almost completely cover the abaxial sporulating surface of *C. personatum*, where it penetrates and destroys the conidiophores, conidia, and subtending cells. The entire leaf spot appears gray on the abaxial surface (Plate 198). *D. pulvinata* was capable of colonizing up to 87% of the lesions under field conditions. Although not effective in preventing infection, *D. pulvinata* might have a role in reducing sporulation. Colonies on potato-dextrose agar are gray to olivaceous gray. Conidia are spherical to subspherical, hyaline to light colored, and 4–7 µm in diameter and may be minutely echinulate. Considerable experimental work, both in the laboratory and in the field, has indicated potential for biological control of late leaf spot.

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(Prepared by R. A. Taber)

Part V. Management of Peanut Diseases

Pathogenic biota, mycotoxins, insects, mites, and rodents threaten the peanut crop in the field and during storage and processing. To realize maximum profit, growers must manage peanut diseases so that losses are minimized and inputs are as low as possible.

Temperature, precipitation, soil type, elevation, endemic pathogens, and cropping systems influence the disease combination in a growing area. Thresholds for unacceptable levels of peanut disease also vary. For example, yield loss caused by late leaf spot may not occur if the level of *Cercosporidium personatum* infection is low. However, stem rot caused by *Sclerotium rolfsii* reduces yield even at low levels of infection.

Management of a particular disease may integrate regulatory measures, cultural practices, cultivar resistance, and pesticide usage. Growers need to know the effectiveness of management practices, how and when to use them, and the expected results. Safety of producers and consumers and the long-term program sustainability should also be considered. Production practices vary from labor intensive in some developing countries to nearly completely mechanized in the United States. Similarly, disease management ranges from minimal to intensive and may include practices that minimize host-pathogen contact and pathogen populations and reduce pathogen increase.

Strategies

Minimizing Host-Pathogen Contacts and Pathogen Populations

Exotic pathogens should be prevented from entering a production area. Movement of pathogens among countries or regions via seed, other plant material (living or dead), soil, or other means can have serious repercussions. Once introduced, pathogens may be very difficult or impossible to eradicate. Extensive knowledge of a pathogen and the risk it poses for production is necessary for exclusion to be effective. Exclusion is especially critical if an exotic pathogen has a wide host range or survives for long periods in the absence of peanut, if the disease caused is difficult to manage, and if resistant cultivars are not available. For example, *Cylindrocladium parasiticum* was probably introduced into the United States from Asia on a host plant other than peanut. The fungus later became established in peanut fields, and Cylindrocladium black rot now causes major losses in the United States.

Exclusion on a large scale can be accomplished by regulatory legislation. Regulations may include embargo, quarantine, and inspection. Laboratory testing of seed and imports and inspection of seed-production fields in some countries or growing areas may be necessary to prevent the introduction of exotic pathogens.

The basis of seedling disease management is high-quality, pathogen-free seed planted under conditions that favor rapid germination and emergence. These conditions include optimal timing for proper soil moisture and temperature. Most seedborne peanut pathogens can infect a wide range of plants. Seed treatments prevent long-distance spread of certain seedborne pathogenic fungi.

Exclusion on a small scale may involve site selection within a farm to avoid problem fields or sanitation of equipment and vehicles after activity in infested fields. Site selection can also be used to minimize exposure to endemic pathogens or insect vectors.

Contacts between a pathogen and peanut plants may be reduced by using cultural practices that physically remove inocula from positions of greatest damage potential and that reduce the duration of environmental conditions favoring fungal growth and pathogenesis. These include burial of host crop debris with a moldboard plow, cultivation that does not throw soil onto the plants (nondirting), crop rotation, weed control, optimal seeding rate and date, and foliar disease management.

Ideal plant populations are important for management of stem rot, Sclerotinia blight, and the tomato spotted wilt virus. Dense plant populations may increase the first two, and sparse populations may increase the third.

For pathogens that develop either early or late in the growing season, disease severity can be minimized by planting early or late, thus allowing plants to mature before environmental conditions conducive to disease prevail. Examples of diseases for which this strategy is effective are rust and Sclerotinia blight. An early planting date could be a part of peanut rust disease management in southern Texas, since early planting and early maturity allow for crop development before the arrival of infective rust spores on wind currents from the south. Late-planted fields infested with *Sclerotinia minor* are under increased risk of Sclerotinia blight during cool, wet, fall weather in some areas. Planting very early or very late in some areas may increase the incidence of the tomato spotted wilt virus.

Crop rotation with species in the grass (Poaceae) family are particularly useful in managing several endemic peanut pathogens (Fig. 81). High peanut yields are often obtained after

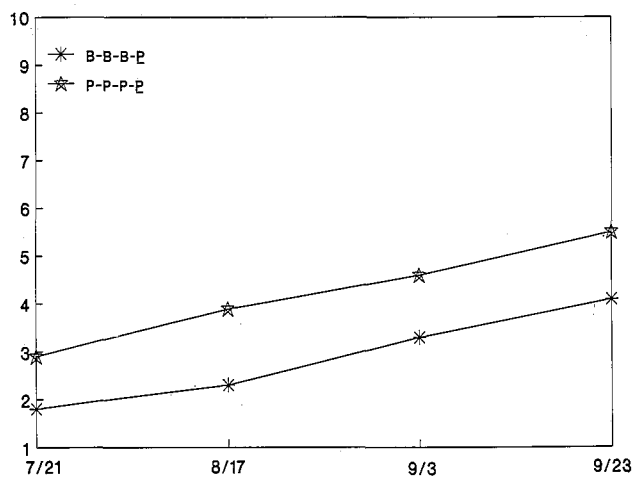

Fig. 81. Leaf spot ratings (Florida 1–10 scale) for continuous growth of peanut (P) and peanut grown after 3 years of Bahia grass (B). (Courtesy T. B. Brennemann)

rotation with Bahia grass (*Paspalum notatum*). Such increases are attributed to inoculum reduction, disease suppression, weed control, improved soil tilth, or other factors. Rotation with nongrass crops, certain nonlegumes, and even resistant peanut cultivars can also be effective in reducing inoculum levels and increasing peanut yields, even where diseases do not appear to be limiting.

Tillage and other practices that destroy crop residue can reduce overwintering inoculum of most peanut foliage- and stem-infecting fungi. Destruction of peanut crop residue is most effective as a disease-control technique during rotations with nonhosts. The majority of these pathogens survive between peanut crops on or in infested residue. Enhanced residue decay after tillage favors nonpathogenic soil microorganisms over pathogens, and pathogen survival decreases.

Reducing Pathogen Increase

Peanut producers often have options during a growing season for reducing the rate of disease increase. Rate-limiting practices include the use of protective and curative (systemic) agrochemicals, cultural practices, biological control practices, and resistant cultivars.

Fungicide seed-treatment combinations generally increase germination and emergence and reduce losses from damping-off induced by soilborne and seedborne pathogens. Seed treatments applied immediately after shelling reduce further colonization by seed fungi and pathogen spread during storage. Mechanical damage to seed, which can be particularly severe when seed have a moisture content of less than 7%, should be minimized during handling and planting. A partial list of some seed-treatment fungicides is given in Table 8.

Fungicides and nematicides applied to peanut plants and soil by ground equipment, airplane, and chemigation may increase yields through the control of pathogens of foliage and below-ground plant parts. Without good foliar disease management, pod detachment before and during harvest may be severe. Minimal fungicide drift and thorough canopy penetration are critical to the success of foliar fungicide programs. Vehicle damage from ground spray equipment can increase the incidence of several diseases. A partial list of fungicides labeled for foliar application is given in Table 9.

TABLE 8. Some Fungicides Used Alone or in Combination as Seed Protectants on Peanut[a]

Common Name	Chemical Name
Captan	*cis*-*N*-((Trichloromethyl)thio)-4-cyclohexene-1,2-dicarboximide
Carboxin	5,6-Dihydro-2-methyl-*N*-phenyl-1,4-oxythiin-3-carboxamide
PCNB	Pentachloronitrobenzene
Thiram	Tetramethylthiuram disulfide

[a] This table should not be construed as a list of recommended fungicides. Disease-management recommendations are published annually by the extension service of each peanut-producing state.

Peanut disease predictive systems help producers make fungicide-application decisions for leaf spot management. Weather information such as leaf wetness, relative humidity, and temperature can be used by growers or computer models that schedule fungicide treatments. A predictive system may call for fewer, the same, or even a greater number of fungicide applications than a calendar-based schedule.

Disease control strategies should consider fungicide effects on nontarget organisms. For example, use of chlorothalonil for leaf spot control increases incidence of Sclerotinia blight, so alternative leaf spot fungicides should be considered. Some fungicides may increase spider mite populations.

Risk of pathogen insensitivity to fungicides increases with prolonged, exclusive use of single-mode-of-action products. Risk-management strategies may involve tank mixes, prepackaged mixtures, or alternating two or more effective products with different modes of action. Partner fungicides may be systemic or protectant. The risk of developing insensitivity is further reduced by using all other available disease control strategies, including resistant cultivars, crop rotation, and cultural practices.

Use of disease-resistant cultivars that sustain competitive yield and quality is the most economical and efficient disease control method. Even with yields lower than those of susceptible cultivars, resistant cultivars may be useful in rotations on a prescriptive basis. Genetic resistance has been identified for most major peanut diseases, but acceptable cultivars with these desirable traits are in many cases not available (Table 10). There are many breeding efforts underway around the world with goals of developing cultivars with resistance to one or more peanut diseases.

Biological control agents may be applied to either the seed, plant, or soil or stimulated by cultural practices or the addition of amendments. The rationale is to favor the crop, disfavor pathogens, or stimulate plant resistance mechanisms. The bacterium *Bacillus subtilis* (Ehrenberg) Cohn is an example of an effective biological seed treatment.

Cultural practices that maximize plant health include maintaining soil pH and optimal availability of plant nutrients and water. Crop residues should be shredded in the fall and deep turned with a moldboard plow equipped with litter-covering devices near the time of planting. Tillage is a cultural practice that integrates disease control, residue and water management, physical weed control, soil compaction and salinity alleviation, and herbicide and fertilizer incorporation. Excessive herbicide or improper placement may cause root damage and predispose seedlings and plants to infection by soilborne pathogens.

Soil calcium and salinity management are necessary to reduce the pod rot complex in some growing areas. Whether the source is calcium sulfate (gypsum or landplaster) or calcium carbonate (lime or ground limestone), adequate calcium in the pegging zone during pod fill minimizes nutrition-related pod rots and is one aspect of producing high-quality seed. Calcium sulfate supplements in conjunction with deep tillage to

TABLE 9. Some Fungicides Labeled for Control of Foliar Diseases of Peanut[a]

Common Name	Chemical Name	Foliar Diseases
Basic copper sulfate	Cupric sulfate	Leaf spot
Benomyl	Methyl 1-(butylcarbamoyl)-2-benzimidazolecarbamate	Leaf spot, web blotch
Chlorothalonil	Tetrachloroisophthalonitrile	Leaf spot, rust, web blotch
Copper hydroxide	Cupric hydroxide	Leaf spot
Mancozeb	Zinc ion and manganese ethylene bisdithiocarbamate[b]	Leaf spot, web blotch
Propiconazole	1-[2-(2,4-dichlorophenyl)-4-propyl-1,3-dioxolan-2-ylmethyl]-1 *H*-1,2,4-triazole	Leaf spot
Tebuconazole	α-[2-(4-chlorophenyl)-ethyl]-α-(1,1-dimethylethyl)-1*H*-1,2,4-triazole-1-ethanol	Leaf spot, rust

[a] This table is not to be construed as a list of recommended fungicides. Disease-management recommendations are published annually by the extension service in each of the peanut-producing states.

[b] Coordination product of manganese, 16%; zinc, 2%; and ethylene bisdithiocarbamate, 62%.

break up a plow layer help to displace sodium (Na) from soil particles and facilitate its leaching out of the pod and root zone. Application of high levels of calcium sulfate suppresses Pythium pod rot in some soils.

Harvesting at optimal maturity can decrease pod rot losses. Maintaining proper storage temperature and relative humidity equilibrium will slow or preclude the growth of storage fungi, including those that produce mycotoxins.

Good disease management will pay dividends in high yields of good-quality peanuts. Disease management practices vary among growing areas, cultivars used, and the disease complex unique to each growing area. Good management is seldom simple and requires a plan that integrates wise cultivar and site selections, proper use of cultural practices, judicious chemical applications, and biological controls into a practical program (Table 10). It also involves timely planting, harvesting, and storage of the peanut crop. Disease control requires a plan based on research knowledge and extension of that knowledge to the producer.

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(Prepared by F. M. Shokes and M. C. Black)

TABLE 10. Principles of Peanut Disease Control and Relative Effectiveness of Management Practices

Disease	Control Principle[a]	Cultural Practices[b]	Crop Rotation[c]	Resistant Cultivar[c,d]	Pathogen-Free Seed[c]	Pesticides[c,e]	Harvest, Handling, and Storage[c]
Fungal diseases of foliage and upper stems							
Early and late leaf spots	I, R	P, W, WC	2	2	...	F-1	...
Rust	R	D, P, W	...	2	...	F-1	...
Web blotch	R	W	3	2	...	F-1	...
Pepper spot and leaf scorch	R	...	3	...	...	F-2	...
Scab	E, R	...	2	2	1	F-2	...
Fungal diseases of lower stems, pegs, pods, roots, and seeds							
Stem rot (southern blight, white mold)	I, R	D, P, S, T, W, WC	2	2	...	F-2	...
Sclerotinia blight	E, I, R	D, P, S, W	3	3	1	F-3, ST-2, N-3	...
Cylindrocladium black rot	E, I, R	D, S, SF	2	2	1	N-2, ST-1	...
Pythium diseases	R	S, SF, W	3	3	...	F-3, ST-3	...
Rhizoctonia diseases	I, R	D, T, W	2	3	...	ST-3, F-1	...
Charcoal rot	R	D, W	...	...	...	ST-3	...
Diplodia collar rot	I, R	W	3	RI	...	F-3	...
Black hull	I, R	T	3	3	...	F-3	...
Verticillium wilt	E, I, R	W, WC	2	RI	...	N-2	...
Botrytis blight	R	D, P, W	3	...	...	F-2	...
Aspergillus crown rot	R	D, T, W	...	RI	3	ST-3	3
Phymatotrichum root rot	I, R	pH, S, SF, T	3	...	...	...	...
Aflatoxin	I, R	D, W	3	RI	...	SI-3	1
Pod rot complex	I, R	SF, T, W	3	3	...	F-2	...
Penicillium and Rhizopus seed rots	I, R	...	...	...	...	ST-1	1
Viral diseases							
Peanut mottle	E, I	S	...	RI	1	...	...
Peanut stunt	E, I	S, WC	...	RI	1	...	...
Peanut stripe	E		...	RI	1	...	...
Peanut clump/Indian peanut clump	E	D, S	3	...	1	N-3	....
Groundnut rosette	I, R	P, WC	...	1	...	SI-1	...
Spotted wilt	R	D, P, S, WC	...	2	...	...	...
Bud necrosis	R	D, P, WC	...	2	...	...	...
Yellow spot	R	...	...	...	...	...	...
Nematode-induced diseases							...
Peanut root knot	E, I, R	S	2	RI	...	N-2	...
Northern root knot	E, I, R	S	2	RI	...	N-2	...
Javanese root knot	E, I, R	S	2	RI	...	N-2	...
Root-lesion nematodes	I	S	3	RI	...	N-2	...
Sting nematodes	I	S	3	...	...	N-1	...
Ring nematodes	I	S	3	...	...	N-2	...
Potato nematodes	E, I	S	1	...	1	...	...
Bacterial wilt	E, I, R	...	2	1	3	...	...

[a] E = exclude or avoid introduction of initial pathogen inoculum; I = reduce inoculum of an endemic pathogen; and R = reduce the rate of pathogen increase and spread.

[b] T = tillage, including crop residue management; SF = optimal soil fertility; P = plant population or density; D = seeding date; W = water management; WC = weed control; pH = adjustment of soil pH; and S = site selection.

[c] Relative effectiveness of practices: 1 = highly effective; 2 = moderately effective; and 3 = slightly effective.

[d] RI = resistance identified in plant introductions or breeding lines but not available in commercially acceptable cultivars.

[e] F = fungicide; SI = soil-applied insecticide; N = nematicide or soil sterilant; and ST = seed treatment, either fungicide or biological control bacterium.

Genetic Modification

Peanut is an important grain legume in both the semiarid tropics and warm temperate regions of the world. The species originated in the region of southern Bolivia and northern Argentina, and at least seven centers of diversity are present in South America and Africa. Several large germ plasm collections exist in North America, South America, Asia, and Africa, which together have more than 12,000 entries. Within the cultivated species are two morphologically distinct subspecies (*hypogaea* and *fastigiata*), containing two and four botanical varieties, respectively. Additionally, both genetic and cytoplasmic differences have been observed among intersubspecific hybrids. Although the germ plasm collection is large, genetic characterization of *Arachis hypogaea* is limited compared with that of other important crop species.

Inheritance of only a few morphological or agronomic traits has been determined, and fewer than 10 linkage groups have been reported. No aneuploid series is available to help associate genes with chromosomes. To date, no genetic map for the cultivated peanut has been made, nor has any gene been associated with a specific chromosome. Genetic characterization of peanut has been especially difficult because it is an allopolyploid ($2n = 4x = 40$) that arose from two distinct species with similar genomes. Hybrids originating from crosses of mutant genotypes thus result in polyploid genetic ratios that are complicated by gene duplications and interactions among genomes. At the molecular level, studies with isozymes, restriction fragment length polymorphisms (RFLPs), and randomly amplified polymorphic DNAs (RAPDs) have indicated that polymorphisms can be detected in a relatively small percentage of the genomes of the cultivated species. Thus, it has not yet been possible to construct a linkage map for the cultivated peanut.

Plant breeding efforts since the 1970s have led to steady increases in yields, the result of selection of plants in which photosynthate is preferentially partitioned into reproductive structures and metabolism. However, less progress has been made in the improvement of other traits such as resistance to diseases. In large part, this has been because of the multigenic inheritance of disease resistance and/or multiple components of resistance in the host plant. A limited number of cultivars with enhanced disease resistance have been released. These include NC 8C and its replacement, NC 10C, which have moderate resistance to Cylindrocladium black rot, caused by *Cylindrocladium parasiticum*; the virginia market type VA 81B and spanish type Tamspan-80, which are resistant to Sclerotinia blight; and the small-seeded runner market type Southern Runner, which is resistant to late leaf spot, stem rot, and the tomato spotted wilt virus. Additional breeding progress has been made to achieve resistance against the two leaf spots and rust. Particularly successful have been the efforts of the researchers at the International Crops Research Institute for the Semi-Arid Tropics (ICRISAT), who have screened the entire *A. hypogaea* germ plasm collection and conducted extensive crossing programs. Other individuals, especially in China, have attempted to create variation within the cultivated species through induced mutations. These studies have led to the release of 33 cultivars.

Unlike the cultivated species, a large amount of morphological and genetic variation at the molecular level has been documented among the wild species of *Arachis*. An RFLP linkage map was recently constructed from an *A. stenosperma* × *A. cardenasii* diploid hybrid, and if the wild species and cultivated genomes are homosequential, then these studies should have applications for analyzing *A. hypogaea*. Included among wild peanut species are accessions that exhibit extremely high levels of disease resistance (Table 11). Several species have multiple resistance to the most widespread peanut diseases. The genus has been divided into nine botanical sections with four tetraploid ($2n = 4x = 40$) and 65 diploid ($2n = 2x = 20$) species. Of these, approximately 25 will hybridize with *A. hypogaea*; but ploidy level and genomic differences result in a high degree of sterility, even when it is possible to produce F_1 progeny. Very little is known about reciprocal differences among crosses, however; and most hybrids can be obtained only when the cultivated species is used as the female parent.

Moving genes that confer disease resistance from wild to cultivated peanut species has been a primary objective of several breeding programs since the mid-1960s. Efforts have concentrated on obtaining additional germ plasm from South America, studying crossing and biosystematic relationships, investigating mechanisms of incompatibility and fertility restoration, and selecting advanced-generation tetraploid progenies with high levels of resistance. Making the initial interspecific crosses is difficult but has proven to be much easier than inducing genetic recombination between chromosomes of the wild and cultivated species. Thus, relatively few lines with high levels of disease resistance have been selected in 40-chromosome hybrid derivatives. Germ plasm releases have recently been made from interspecific peanut hybrids that confer resistance to peanut pathogens including *Cercospora arachidicola* and *Meloidogyne arenaria*. Because these lines have relatively low yields and poor agronomic quality, secondary breeding programs will be necessary to incorporate this resistance into genotypes with potential for cultivar release.

Barriers to interspecific hybridization have led to many studies of reproductive development and crossing barriers in peanut. Reproductive development in peanut is complicated by the interactions of the embryo with the geotropic growth of the

TABLE 11. Number of Accessions with High Levels of Disease Resistance in Botanical Sections of *Arachis*

Disease	Number Evaluated	Number with High Resistance[a]							
		Ara.[b]	*Cau.*	*Ere.*	*Ext.*	*Het.*	*Rhi.*	*Tri.*	Unk.
Early leaf spot	97	1	...	2	3	...	24	...	...
Late leaf spot	96	15	4	12	3	...	50	1	...
Cylindrocladium black rot	21	16	...	...	...	...	...	...	...
Web blotch	50	24	4	1	1	1	...	...	...
Rust	61	18	...	4	1	...	34	1	...
Peanut clump	38	...	...	...	...	...	...	...	1
Peanut mottle	91	4	1	4	...	1	39	...	...
Peanut ringspot	...	1	...	...	1	...	1	...	5
Peanut rosette	13	1	1	1	...	...	1	...	...
Peanut stripe	8	1	...	...	...	...	3	...	...
Peanut stunt	90	3	1	4	...	...	39	...	...
Tomato spotted wilt	42	3	...	...	...	1	...	...	...

[a] *Ara.* = *Arachis; Cau.* = *Caulorrhizae; Ere.* = *Erectoides,* including species in both sections *Erectoides* and *Procumbentes* (formally section *Erectoides* ser. *Procumbentes*); *Ext.* = *Extranervosae; Het.* = *Heteranthae; Rhi.* = *Rhizomatosae; Tri.* = *Triseminatae*; and Unk. = section unknown.

[b] Section *Arachis* species are cross-compatible with *A. hypogaea*.

peg from the stem into the soil. After fertilization, the embryo grows, becomes quiescent for a week or longer, and then reinitiates growth as the pod forms. Foremost among techniques used to overcome interspecific hybridization barriers are the use of growth regulators to enhance fertilization and promote peg elongation and embryo growth and the in vitro culture of ovules and embryos. To overcome the failure of peg growth, gibberellic acid can be applied at the base of the flower hypanthium at the time of pollination. Although this hormone effectively stimulates peg elongation, it also inhibits embryo growth. Applications of kinetin and indoleacetic acid to the peg appear to be necessary to maintain reproductive growth. Even with the addition of growth regulators, however, the embryonic tissues will not fully develop, and in vitro culture is necessary to obtain plants.

Ovule culture in peanut was first successful during the early 1970s. However, results have been highly variable, and plants have rarely been recovered. Failure of this system is attributed to the undifferentiated state (globular stage) of embryos contained within ovules. When the peg is placed into culture with the meristem left intact, it will elongate, grow geotropically, and expand into pods and may produce well-developed seed after 90 days. This technique appears to increase success rates for obtaining plants from very young embryos. If reproductive tissues abort after embryos have differentiated into a heart shape, then in vitro culture of excised embryos has been highly successful. Although several unique hybrids have been obtained through embryo culture, many of those reported in the literature also have been obtained through conventional crossing procedures.

Protoplast fusion is potentially useful as a means of broadening the genetic base in many cultivated species. The use of protoplast technologies may be forthcoming in peanut, especially if wild species can be used as source tissues to hybridize with *A. hypogaea.*

Studies with in vitro tissue culture have indicated that most peanut explants can be induced to form callus tissues. At the present time, there is no evidence of plant regeneration from undifferentiated *A. hypogaea* tissues (callus or single-cell suspensions). Thus, the state of the art for plant cell culture in peanut restricts the use of many techniques applicable for improvement of other crop species.

The use of haploid breeding procedures for *A. hypogaea* has been generally unsuccessful. Although studies on floral bud size and anther color have allowed selection of tissues at the correct developmental stage for in vitro culture of anthers or pollen, development beyond a few cell divisions has not been observed.

Several important advances have been recently made in peanut tissue culture and plant regeneration. In the most promising of these procedures, plants are regenerated from explants such as immature cotyledons or leaves or from meristematic tissues in shoot tips. Treatments such as the addition of antiviral agents or altered temperatures can be combined with these procedures to provide a source of virus-free plants and tissues.

Plant gene transfer (transformation) is a relatively new technology by which genetic elements conferring disease resistance can be transferred to crop plants. Transformation theoretically permits genes derived from virtually any source to be introduced into the crop species. The genetic base of the crop is thus greatly expanded with the possibility that novel, disease-resistant genotypes might be produced. Significant advances have been made toward development of a practical gene-transfer technology for peanut.

Of the several strategies that have been attempted for peanut transformation, microprojectile bombardment of either intact tissues or embryogenic cultures appears to be the most successful. In microprojectile bombardment, microscopic gold or tungsten particles are coated with the transforming DNA and then accelerated to high velocity in a gunlike apparatus. The high-velocity particles can penetrate intact cell walls to carry transforming DNA into the cell. DNA carried by the particles can then be integrated with the chromosomal DNA of the plant cell. Integrated genes are usually inherited stably, and the transformed plants typically express the protein encoded by the introduced gene.

An important goal of peanut transformation is to produce transgenic germ plasm sources with improved resistance to pathogens, particularly certain fungi and viruses. Enhanced resistance to fungi has been observed in plants of other species that express high levels of plant chitinase and other alien gene products.

Alien gene products also can act synergistically when they are expressed simultaneously in plant tissues, e.g., by the co-expression of chitinase and glucanase. In this case, each of the two gene products could be expected to catalyze hydrolysis of a specific component of the fungal cell wall, causing exposure of the cell membrane and thus making the fungus highly susceptible to disruption by osmotic shock.

Enhancement of virus resistance in plants may be obtained by the expression of capsid protein or other virus-encoded proteins. This effect has been demonstrated in a wide array of plant species for several major classes of viral pathogens. It is anticipated that such a strategy would also be effective in peanut.

At the present time, the technology for enhancing disease resistance through gene transfer to peanut is not yet fully developed. However, key elements are now available. The technology for enhancing resistance to viral pathogens is well developed and has been demonstrated in numerous crop species for major classes of pathogenic viruses. Strategies for enhancing resistance to fungi are less well defined; but several rational strategies for combating fungi have been advanced, and genes needed for testing them have been cloned.

In general, genetic modification and improvement of the peanut has been a steady but slow process since the 1950s. Polyploidy, little knowledge of genetic systems, few genes characterized, multiple genes conditioning traits of agronomic interest, and relatively few investigators working in peanut breeding and genetics have hindered progress. During the 1980s and into the 1990s, however, increased efforts—especially in the areas of molecular genetics, tissue culture, and gene transformation—have significantly broadened the knowledge base of peanut genetics. Applications of recent basic scientific advances should be forthcoming as previously complex and obscure procedures become routine and can then be used to solve practical problems in genetics and pathology.

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(Prepared by H. T. Stalker and A. K. Weissinger)

Disease- and Insect-Resistant Cultivars

Successful breeding and screening programs for disease, insect, and nematode resistance require close cooperation between a breeder/geneticist and pathologist, entomologist, and/or nematologist. Immunity to pathogens, insects, or nematodes has not been reported in cultivated peanut (*A. hypogaea* L.). However, many genotypes have been found with usable levels of resistance for most of the major pathogens and insects (Tables 12 and 13). Nematode resistance has been less available. In many cases, pest resistance is great enough to allow reduced levels of pesticides or to enhance the level of control currently available with pesticides. As sources of resistance are identified, they must be incorporated into an ongoing breeding program. Generally, emphasis is placed on developing resistance to those pathogens and other pests for which other control measures (cultural or chemical) are not available or are too expensive. Incorporating the many quality and agronomic characteristics needed to make a new cultivar acceptable to peanut growers and the industry requires an intensive breeding effort.

Of the fungal diseases, early leaf spot, caused by *Cercospora arachidicola* (CA); late leaf spot, caused by *Cercosporidium personatum* (CP); and rust, caused by *Puccinia arachidis,* are nearly worldwide in occurrence and frequently have great economic impact on the crop. Considerable effort has been given to identifying sources of resistance to CA, CP, and rust. All breeding programs in the United States, at ICRISAT (India and Africa), and in most other countries with peanut-breeding programs have addressed one or more of these disease problems. Most sources of resistance to the leaf spots are only partial or rate reducing. Mechanisms or components of resistance commonly reported include extended latent periods, decreased sporulation, smaller lesions, reduced necrotic area, reduced infection frequency, and reduced defoliation.

Resistance to CA, CP, and rust appear to be independently inherited. Major genes for resistance have been identified for rust but not for the leaf spots. Additive genetic factors predominate in resistance to the leaf spots, but nonadditive factors are also important. Resistance to CP seems to be more commonly available than that to CA in cultivated peanut.

Many wild *Arachis* species and their interspecific derivatives from cultivated peanut have been reported to have resistance approaching immunity for some foliar diseases, especially CP and rust. *A. diogoi*, *A. cardenasii*, and *A. stenosperma* are among *Arachis* spp. with near immunity to CP.

Late maturity and low pod yields have been associated with foliar disease resistance, especially to leaf spots. Southern Runner is a runner cultivar with moderate resistance to CP and rust that has yields and grades comparable to currently available cultivars. However, Southern Runner matures 10–20 days later than the widely grown susceptible cultivar Florunner, which may limit its adaptability to certain growing areas.

Other cultivars or germ plasm lines released from breeding programs in the United States with partial resistance to CA or CP include Altika (CA/CP), GP-NC 343 (CA), NC 3033 (CA), PI 109839 (CA), Tifrust 1 (CA/CP, rust), Tifrust 2 and 3 (CP, rust), Tifton 8 (CA/CP), and VGP 2 to VGP 7 (CA/CP).

Web blotch (*Phoma arachidicola*), a foliar disease that can be very damaging, is generally less common than CA, CP, or rust. Resistance has been identified and used successfully in some breeding programs.

Peanut roots, pegs, pods, and stems are attacked by a wide range of soilborne fungi, nematodes, and insects. Useful resistance to soilborne fungal pathogens has been identified but is generally incomplete and polygenic.

Cylindrocladium Black Rot (*Cylindrocladium parasiticum*)

Peanut breeders in Georgia, Florida, and Virginia have screened the germ plasm collection for resistance to Cylindrocladium black rot (CBR) and released 10 germ plasm lines with varying degrees of resistance. One of these lines (NC 3033) has been used in the development of two resistant virginia-type cultivars (NC 8C and NC 10C). Three spanish-type cultivars (Tamnut 74, Toalson, and Spancross) were identified as resistant after release. However, the resistant germ plasm and spanish cultivars all have relatively low yield potential.

Studies have shown that resistance in NC 3033 is probably the result of a lack of infection at inoculum densities less than 50 microsclerotia per gram of soil and tolerance to infection at greater densities. The resistance in spanish types is similar. Inoculum level, environment, and other factors can also affect disease expression.

Resistance to CBR has been attributed to additive genetic effects and is appreciably heritable. However, root and pod resistance may be inherited separately, thus complicating breeding for resistance. The potential exists for race development in *C. parasiticum.*

Sclerotinia Blight (*Sclerotinia minor*)

Screening for resistance to Sclerotinia blight in the United States has been concentrated in the Virginia-Carolina and southwestern peanut areas where Sclerotinia blight is a major problem. These efforts have resulted in the release of two virginia-type peanut cultivars (VA 81B and VA 93B) and one spanish-type cultivar (Tamspan-90) with varying degrees of partial resistance. Twelve germ plasm lines have also been released with resistance to Sclerotinia blight. Resistance varies in these lines from moderate (VGP 1 to VGP 9) to very high (Chico and TXAG 1, 2, 4, and 5).

The resistance in highly resistant lines and cultivars is thought to be largely the result of a morphological escape mechanism rather than one based on physiological characters. The growth habit in all of these genotypes is upright with an open canopy structure, which creates a microclimate less favorable for colonization and infection.

Preliminary studies have shown that the physiologic resistance in some plants may be at least partially controlled by a cytoplasmic factor. Other studies have shown low-narrow and broad-sense heritability for resistance, indicating the involvement of several genes and/or large environmental variance. Resistance to Sclerotinia blight is also often associated with early maturity. Reliable laboratory screening procedures are needed in order to definitively determine the inheritance of resistance to Sclerotinia blight.

Stem Rot (*Sclerotium rolfsii*)

Breeding programs in all major peanut-producing areas of the United States have included screening for resistance to stem rot. One germ plasm line (NC 3033) and two cultivars (NC 2 and NC 8C) with resistance to stem rot have been released. In addition, six other cultivars (Toalson, Southern Runner, Pronto, Georgia Browne, and Sunbelt Runner) have been identified since release as resistant. Recent studies have shown that resistance is not related to growth habit. While no specific genetic models for control of stem rot resistance have been reported, it has been suggested that the defense mechanism is different from that for resistance to pod rot caused by *Pythium myriotylum.*

Pod Rot (*Pythium, Rhizoctonia,* and *Fusarium* spp.)

Three breeding lines (NC 3033, TXAG 4, and TXAG 5) and two cultivars (Toalson and Tamspan-90) with resistance to pod rot have been released. Although resistance has been reported in some cultivars, the level is not high enough to prevent serious yield losses. Resistance to pod rot has been associated with more compact palisade mesophyll cells in leaf tissue; thicker, more lignified cell walls in the epicarp; and sclerenchymatous mesocarp of cell walls in the pods.

Rhizoctonia Limb Rot (*Rhizoctonia solani*)

One virginia-type cultivar (VA 81B) and one spanish-type cultivar (Toalson) were reported as resistant to Rhizoctonia limb rot after their release for resistance to other soilborne

TABLE 12. Peanut Germ Plasm Lines and Cultivars, Released and/or Registered in the United States, That Have Resistance to Diseases, Nematodes, or Insects

Type Identity	Resistance[a]	Source[b]	Year	Description (Crop Science vol.:page)
Germ plasm				
GP-NC 343	SCRW, CA	NC	1970	11:605
Chico	SB[c]	USDA-ARS, GA, VA, OK	1973	15:105
PI 337394F	AF	USDA, AL, GA	1974	15:106
PI 337409	AF	USDA, AL, GA	1974	15:106
NC 10247	LH	NC	1975	15:738-739
NC 10272	LH	NC	1975	15:738-739
NC 15729	LH	NC	1975	15:738-739
NC 15745	LH	NC	1975	15:738-739
PI 109839	CA	USDA, GA	1979	20:292
NC 3033	CBR, SR, PR, LS, CA	NC	1976	16:887
VGP 1	CBR, SB	USDA-ARS, VA	1979	20:419
CBR-R1 to R6	CBR	USDA-ARS, GA	1981	21:992-993
Tifrust 1 to 14	R, CA, CP	USDA, GA	1981	22:452-698
F 334 AB-14	CR	USDA-ARS, GA, FL	1983	23:1019-1020
GFA 1 and 2	AF	USDA, GA	1983	23:1020-1021
AR 1 to 4	AF	USDA, GA	1983	23:1021
Tifton 8	CBR, LS, SCRW, CR, CA, CP	USA-ARS, VA, GA	1984	25:203
TXAG 1 and 2	SB[c]	TX	1985	26:391
VGP 2 to 7	SB, LS, SCRW, CA, CP	USDA-ARS, VA	1986	27:1319
TXAG 4 and 5	PR, SB	TX, OK, USDA-ARS	1989	30:429
ICGV 87157	CP, TSWV, SR, LM, LH	ICRISAT	1990	32:837
ICGV 86031	T, LH, LM, BW	ICRISAT	1991	33:220
GP-NCW 1 to 4	CA, CP	NC	1992	33:1117
TXAG 6 and 7	R, CA, CP, RK	TX	1992	33:1148
VGP 9	SB, CBR	USDA-ARS, VA	1993	34:1132-1133
Cultivar				
Spancross	CBR[c]	USDA-ARS, GA, OK	1970	10:459
NC 2	SR	NC	1952	10:459-460
Tamnut 74	CBR[c]	TX, OK, GA	1974	15:603-604
NC 6	SCRW	NC	1976	17:346
Toalson	PR, CBR,[c] SR,[c] RL[c]	TX	1979	19:742
VA 81B	SB, RL[c]	USDA-ARS, VA	1982	22:1085-1086
Sunbelt Runner	AF, SR[c]	USDA-ARS, GA	1981	22:1086
NC 8C	CBR, SR	NC	1982	23:183-184
Pronto	SR[c]	USDA-ARS, OK, GA	1980	23:184
Southern Runner	LS, R, WB, SR,[c] TSWV[c]	FL	1986	27:817
NC 10C	CBR	NC	1988	31:484
Tamspan-90	SB, PR	TX, USDA-ARS	1990	31:1711
Florunner	WB[c]	FL	1969	9:850
GA 119-120	BW	USDA, GA	1954	11:313
Altika	CA, CP	FL	1972	14:339
ICGV 87-87128	TSWV	ICRISAT (India)	1988	30:959
ICGS 11	TSWV	ICRISAT (India)	1986	30:960
NC VI1	SR[c]	NC, USDA-VA	1989	31:484-485
ICGV 87141	TSWV	ICRISAT	1989	31:1096
ICG SI	TSWV	ICRISAT	1990	31:1382-1383
ICGV 87187	TSWV	ICRISAT	1990	32:278-279
ICGV 87160	R, CP, TSWV, SR, LM	ICRISAT	1990	32:1075
Sinkarzei	R, CA	ICRISAT	1989	33:212
ICGV 86590	R, CP, SR, LH, CR	ICRISAT	1991	33:357-358
Georgia Browne	SR, RL, TSWV	GA	1992	34:1125-1126
VA 93B	SB	USDA-ARS, VA	1993	34:1126

[a] SCRW = Southern corn rootworm; SB = Sclerotinia blight; CBR = Cylindrocladium black rot; SR = stem rot (white mold); PR = pod rot; LS = leaf spot; CR = Diplodia collar rot; RL = Rhizoctonia limb rot; WB = web blotch; CA = early leaf spot (*Cercospora arachidicola*); CP = late leaf spot (*Cercosporidium personatum*); R = rust (*Puccinia arachidicola*); TSWV = tomato spotted wilt virus; LM = leafminer (*Aproaerena modicella*); BW = bacterial wilt (*Pseudomonas* sp.); LH = leafhopper (*Spodoptera* sp.); T = thrips (*Thrips palmi*); RK = root-knot nematode (*Meloidogyne* spp.); and AF = *Aspergillus flavus.*

[b] NC = North Carolina Agricultural Research Service; USDA-ARS = U.S. Department of Agriculture–Agricultural Research Service; AL, GA, FL, OK, and VA = agricultural experiment stations in Alabama, Georgia, Florida, Oklahoma, and Virginia, respectively; and ICRISAT = International Crops Research Institute for the Semi-Arid Tropics.

[c] Resistance identified after release.

diseases. It has been suggested that an open canopy structure, such as that needed for Sclerotinia blight resistance, may be associated with resistance to Rhizoctonia limb rot. However, greenhouse studies have indicated that morphological barriers or active plant response may also be associated with resistance. Early maturity could be a good escape mechanism, since peanuts could be dug before environmental conditions (low temperatures) are suitable for disease development.

Diplodia Collar Rot (*Diplodia gossypina*)

One spanish-type germ plasm line (F 334 AB-14) with high levels of resistance to Diplodia collar rot has been released. A virginia-type germ plasm line (Tifton 8), unrelated to F 334 AB-14, has been released with resistance to Diplodia collar rot at low inoculum densities. While F 334 AB-14 is in the background of some cultivars being grown today, the resistance has not been transferred to these cultivars.

Black Hull (*Chalara elegans*)

The introduction of resistant cultivars successfully restored peanut production in irrigated areas of South Africa where this disease once dominated. Different sources of genetic resistance to *C. elegans* were identified, and new spanish cultivars with resistance were officially released.

Aflatoxin (*Aspergillus flavus* and *A. parasiticus*)

Screening for resistance to aflatoxin-producing fungi has been carried out in several countries by using laboratory techniques, rehydrated seed, and field-stress studies. Studies have evaluated resistance to both infection by the fungus and production of aflatoxin. PIs 337394F and 337409, GFA 1 and GFA 2, AR 1 to AR 4, and Sunbelt Runner were selected as having resistance to infection by the fungus on rehydrated seed. Selections from Senegal, 55-437 and 73-30, show field resistance under severe drought stress. Various other reports of resistance can be found in the literature listed. No cultivar with a high level of resistance to aflatoxin-producing fungi has been made commercially available in the United States.

Root Knot (*Meloidogyne arenaria* and *M. hapla*)

There has been only limited success in identifying resistance to the peanut root-knot nematode, and there are no reports of high levels of resistance in *A. hypogaea.* TXAG 6 and TXAG 7, *A. hypogaea* germ plasm lines from the Texas Agricultural Experiment Station with high levels of resistance to root knot, are derived from complex interspecific crosses with *A. cardenasii* Krap. et Greg. *nom. nud.*, *A. chacoensis* Krap. et Greg. *nom. nud.*, *A. batizocoi* Krap. et Greg., and *A. hypogaea* (Florunner). Some reports of resistance in other *Arachis* species and low to moderate levels of resistance in *A. hypogaea* have been made.

TABLE 13. Disease- and Nematode-Resistant Peanut Cultivars from Africa

Cultivar	Resistance[a]	Origin
RMP 12	CP, RV	Burkina Faso
RMP 91	CP, RV	Burkina Faso
Falcon	WB	Zimbabwe
RMP 40	RV	Burkina Faso
RGI	RV	Malawi
KH 149A	RV	Burkina Faso
RH 241D	RV	Burkina Faso
OH 243C	RV	Burkina Faso
69-101	RV	Senegal
59-426	RV	Senegal
Harts	BH	South Africa
Jasper	BH	South Africa
Kwarts	BH, PN	South Africa
Robbie	SB	South Africa
55-437	AF	Senegal
73-30	AF	Senegal

[a] CP = *Cercosporidium personatum*; RV = rosette virus; WB = web blotch (*Phoma arachidicola*); BH = black hull (*Chalara elegans*); BB = Botrytis blight (*Botrytis cinerea*); SB = Sclerotinia blight (*Sclerotinia minor*); PN = peanut pod nematode (*Ditylenchus africanus*); and AF = *Aspergillus flavus*.

Peanut Pod Nematode (*Ditylenchus africanus*)

Peanut germ plasm was screened in South Africa for resistance to *D. africanus,* and two breeding lines, US 40-1 and PI 298233, with excellent partial field resistance were found. One cultivar with partial resistance to the peanut pod nematode has been released.

Viruses

Sources of resistance have been reported in cultivated peanut to the viruses that cause groundnut rosette, peanut stunt, and tomato spotted wilt (TSWV). ICRISAT has been a primary source of germ plasm resistant to groundnut rosette (Africa) and TSWV (India), whereas breeding programs in Florida, Georgia, and Texas have had some success in developing material resistant to TSWV. The recently released variety Georgia Browne has significant resistance to TSWV.

Southern Corn Rootworm

Programs screening for resistance to southern corn rootworm have resulted in the identification of several sources with various levels of resistance. Three germ plasm lines (GP-NC 343, Tifton 8, and VGP 7) have been released. CP-NC 343 has been used to develop the partially resistant cultivar NC 6. Resistance is thought to be based on antibiosis or biochemical processes in most resistant lines identified and appears to be heritable and multigenic in nature.

Thrips and Leafhoppers

Only limited work has been done or is currently being done to search for resistance to foliage-feeding insects in peanuts. This is largely because of the ready availability of acceptable insecticides for their control and the higher priority assigned to diseases and other pests for which satisfactory controls are unavailable or limited.

Four germ plasm lines (NC 10247, NC 10272, NC 15729, and NC 15745), with resistance to the potato leafhopper, were developed from the irradiation breeding program in North Carolina and have been released. NC 10247 is considered a virginia type, while the other three are runner market types. ICRISAT has released two germ plasm lines (ICGV 87157 and ICGV 86031) and one cultivar (ICGV 86590), all spanish types, with resistance to leafhoppers. ICGV 86031 also has resistance to thrips. Inheritance studies indicate leafhopper resistance is probably controlled by a few major genes.

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(Prepared by D. W. Gorbet, T. A. Coffelt, and P. J. A. van der Merwe)

Glossary

A—acre
C—centigrade or Celsius
cm—centimeter (0.39 in.; 1 cm = 10 mm)
F—Fahrenheit
ft—foot
g—gram (453.59 g = 1 pound)
ha—hectare
hr—hour
m—meter
mt—metric ton
ml—milliliter
mm—millimeter (0.04 in.; 10 mm = 1 cm)
µg—microgram (1 µg = 10^{-6} g)
µm—micrometer (1 µm = 10^{-6} m)
nm—nanometer (1 nm = 10^{-9} m)
ppb—parts per billion
ppm—parts per million

abaxial—directed away from the stem of a plant; pertaining to the lower surface of a leaf
abiotic—pertaining to the absence of life, as diseases not caused by living organisms
abscise (n. abscission)—to cut off, separate
acervulus (pl. acervuli)—erumpent, saucerlike fungal fruiting structure bearing conidiophores, conidia, and sometimes setae
acicular—needle-shaped
acute—pertaining to symptoms that develop suddenly (as opposed to chronic)
adaxial—pertaining to the upper leaf surface
adventitious—arising or occurring sporadically from unexpected tissue origin
aecium (pl. aecia)—cuplike fruiting body of rust fungi
aerial—occurring in the air
aerobic—living only in the presence of oxygen
aflatoxin—chemical by-product from *Aspergillus flavus* and *A. parasiticus* harmful to humans and other animals
agar—gelatin from seaweed used in making laboratory culture media
alate—having wings
albino (n. albinism)—white or light-colored; having a marked deficiency in pigmentation
alternate host—another plant host for a given organism
amphigenous—growing on both sides of a leaf
anaerobic—living in the absence of oxygen
anamorphic—pertaining to the imperfect (asexual) state of a fungus
anastomosis—fusion between branches of the same or different structures (e.g., hyphae) to make a network
annule—grooved band in the cuticle of some nematodes
antagonist—an organism that limits the action of another
anterior—toward the front or head (as opposed to posterior)
anther—pollen-bearing portion of a flower
antheridium (pl. antheridia)—male sexual organ found in some fungi
anthesis—the period of the opening of a flower
antiserum—portion of blood containing antibodies
apex (adj. apical)—tip
aphicide—agent, usually a chemical, that kills or inhibits aphids
apothecium (pl. apothecia)—saucer-shaped, ascus-bearing fruiting body
appressorium (pl. appressoria)—swollen or flattened portion of a germ tube or hypha that attaches to the host during an early stage of infection
apterous—wingless
arbuscule (adj. arbuscular)—branched, haustoriumlike, intracellular development of mycorrhizal fungi
ascigerous—bearing asci
ascocarp—sexual fruiting body of an ascomycetous fungus
ascoma—a sporocarp having asci
ascomycete (adj. ascomycetous)—fungus that produces sexual spores (ascospores) within a saclike structure, the ascus
ascospore—sexual spore borne in an ascus
ascus (pl. asci)—saclike structure containing ascospores (typically eight) and borne in an ascocarp
aseptate—having no cross walls
asexual—vegetative; without sex organs, as a type of spore
attenuate (v. or adj.)—to narrow; to weaken or decrease in virulence or pathogenicity
avirulent—unable to cause disease; nonpathogenic
axenic—pertaining to a culture free of living bacteria or other organisms; pure culture
axil—the angle formed by the leaf petiole and the stem
axillary—pertaining to or placed within an axil
azygospore—spore morphologically similar to a zygospore

basidiomycete (adj. basidiomycetous)—fungus that forms sexual spores (basidiospores) on a basidium
basidiospore—sexual spore produced on a basidium
basidium (pl. basidia, adj. basidial)—short, club-shaped, haploid promycelium produced by basidiomycetous fungi
basipetal—toward the bottom
biguttulate—having two globules or vacuoles
biocide—a pesticide lethal to all living material
biotic—relating to life, as disease caused by living organisms
bitunicate—double-walled
blight—general term for sudden, severe, and extensive spotting, discoloration, wilting, or destruction of leaves, flowers, stems, or entire plants
botryose—shaped like a bunch of grapes
butyrous—butterlike

calcareous—resembling or containing calcium
calyx—outermost flower whorl; sepals, collectively
carlavirus—one of a group of related viruses, including the cowpea mild mottle virus
carpel—structural unit or units of the pistils of certain flowers
carpogenic—giving rise to fertile cells
carpophore—stalk of a fruiting body in fungi
carpospore—a diploid spore
catenate—in chains
cauda—tail
chelicera—sharply pointed appendage at the front of a sucking insect
chimera—a plant or part composed of two or more genetically different tissues
chlamydospore—thick-walled or double-walled asexual resting spore formed from hyphal cells (terminal or intercalary) or by transformation of conidial cells
chlorophyll (adj. chlorophyllous)—one of a group of green pigments found in chloroplasts and important in photosynthesis
chlorosis (adj. chlorotic)—failure of chlorophyll development, caused by disease or a nutritional disturbance (e.g., lack of iron, zinc, or magnesium); fading of green plant color to light green, yellow, or white

cilium (pl. cilia)—appendage like a flagellum, which propels cells (especially zoospores) through water
cirrus (pl. cirri)—column or tendril of mucus laden with spores
clavate, claviform—club-shaped
coalesce—to run together
coenocytic—pertaining to a multinucleate mass of protoplasm without cross walls
collar—the portion of the seedling near the surface of the ground
colonization—establishment of a pathogen within a host plant
confluent—coming together
conical—cone-shaped
conidiogenesis—method of spore formation
conidiogenous—producing and bearing conidia
conidiophore—simple or branched, fertile hypha on which conidia are produced
conidium (pl. conidia, adj. conidial)—asexual spore borne at the tip or side of a specialized hypha (conidiophore)
cornicle—hornlike projection
cortex (adj. cortical)—region of parenchyma tissue between the epidermis and phloem in stems and roots; a more or less thick outer covering
cotyledon—seed leaf
crown—region of a seed plant at which stem and root merge
cruciform—arranged in the shape of a cross
cucumovirus—one of a group of related viruses, including the peanut stunt virus
cultivar—cultivated variety
culture—artificial growth and propagation of organisms on nutrient media or living plants
cuticle—waxy, noncellular layer formed on the outer walls of plant epidermal cells; outer sheath or membrane of a nematode or plant
cylindric—of the same diameter throughout the length
cytoplasm—living protoplasm in a cell, except the nucleus

damaged kernels—damaged seed that reduces the market value of peanuts
damping-off—rapid, lethal decline of a germinating seed or seedling before or after emergence
declinous—about to fall over; drooping
defoliate—to deprive of leaves prematurely
deliquescent—dissolving, liquefying, or melting away
dematiaceous—darkly pigmented
digitate—having lobes radiating from a common center
dilution end point—stage of a serial dilution of cells or preparations at which growth or infection from a standard sample of the suspension no longer occurs
dimorphic (n. dimorphism)—having two forms
dissemination—spread of infectious material (inoculum) from diseased to healthy plants
distichous—in two lines
dolioform—barrel-shaped
dormancy (adj. dormant)—state of reduced physiologic activity

echinulate—having small, pointed spines projecting from cell walls
ectoparasite—parasite living on the exterior of its host
effuse (adj.)—stretched out, especially a filmlike growth
egg mass—group of eggs held together by a gelatinous matrix
ellipsoid—elliptic in optical section
elliptic—having the shape of a foreshortened circle
elongate—to lengthen
elytrum (pl. elytra)—one of the anterior wings on some insects that serve to protect the posterior functional wings
enation—abnormal outgrowth from the surface of a stem or leaf
endocarp—inner layer of a pericarp (fruit wall)
endoconidium (pl. endoconidia)—conidium formed inside a hypha
endoparasite—parasite living inside its host
endoplasmic reticulum—system of interconnected cytoplasmic membranes that transports materials within the cell
endosperm—nutritive tissue developed around an embryo in the ovule
endospore—an asexual spore developed within the cell
epicotyl—portion of a plant embryo or seedling above the node at which the cotyledons are attached
epidemic—general and serious outbreak of disease
epidemiology (adj. epidemiologic)—study of factors influencing the initiation, development, and spread of infectious disease
epidermis (adj. epidermal)—surface layer of cells of leaves and other plant parts
epinasty—abnormal, clawlike, downward curling of leaves
epiphyllous—on the upper surface of a leaf
equatorial pore—unthickened spot at the equator of a spore through which a germ tube may emerge
eradicate—to destroy or remove (a pest or pathogen)
erumpent—bursting or erupting through the substrate surface
extra-large kernels—seed of virginia market-type cultivars that do not pass through a 21.5/64-in. screen
exudate—liquid (ooze) excreted or discharged from diseased tissues, from roots and leaves, or by fungi
exude—to ooze or be discharged through pores

facultative parasite—organism that is normally self-dependent but is capable of being parasitic
falcate—curved like the blade of a scythe or sickle
fallow—to leave soil unseeded after plowing
fascicle (adj. fasciculate)—small group, bundle, or cluster of flowers, leaves, stems, or roots; also used with fungi
field capacity—(of water) amount of water retained by a soil after all gravitational water is drained away
filament—thin, flexible, threadlike object; anther-bearing stalk of a stamen
filamentous or **filiform**—threadlike
flagellum (pl. flagella)—hairlike, whiplike, or tinsellike appendage of a motile cell (bacterium or zoospore) that provides locomotion
fleck—minute, discolored spot in green tissue
flexuous—having turns; not rigid
focus—small area of diseased plants
foliar—pertaining to leaves
forma specialis (f. sp.)—taxonomic category reflecting incomplete knowledge of structure
fructification—general term for spore-bearing organs in both macrofungi and microfungi
fumigant—vapor-active (volatile) disinfectant used against microorganisms and other pests
fungicide—substance lethal to fungi
funiculus—stalk of the ovule
fusiform—spindlelike; narrowing toward the ends
fusoid—somewhat fusiform

gall—abnormal swelling or localized outgrowth, often roughly spherical, produced by a plant as a result of attack by a fungus, bacterium, nematode, insect, or other organism
gametangium (pl. gametangia)—cell containing gametes or nuclei that act as gametes
gamete—sex cell
geniculate—bent, like a knee
genome—set or group of chromosomes
genotype—genetic constitution of an individual or group; class or group of individuals sharing a specific genetic makeup
geocarp—fruit (e.g., peanut pod) that ripens beneath the ground
geocarposphere—soil near a pod
geotropism (adj. geotropic)—growth curvature induced by gravity
germ pore—differentiated thin area within a spore wall (especially in rust fungi) through which a germ tube can emerge
germ tube—hypha resulting from an outgrowth of the spore wall and/or cytoplasm
germinate (n. germination)—to begin growth (as of a seed, spore, sclerotium, or other reproductive body)
germling—germinating spore
girdle—to circle and cut through; to destroy vascular tissue in a canker that encircles the stem or stolon
globose—almost spherical
globule—tiny globe or ball
gnotobiotic—pertaining to growth of an organism in the absence of other organisms or in the presence of only known organisms
graft transmission—transmission of a pathogen by vegetative propagation
gram-negative, gram-positive—pertaining to bacteria that release or retain, respectively, the violet (red) dye in Gram's solution
granulous—covered with a granulelike substance
guttulate—having one or more oillike drops inside

gynophore—stalk bearing the female reproductive structures; in peanut, commonly called a peg

hamate—hooked
haulm—stem or stalks, collectively
haustorium (pl. haustoria)—specialized hyphal branch of a parasite, especially one within a living host cell, for absorbing food (often associated with rust fungi, downy and powdery mildew fungi, parasitic flowering plants, and other obligate parasites)
herbaceous—having little or no woody tissue and usually persisting for a single growing season
hilum—scar on a seed where it was attached to the funiculus
hollow heart—a boron deficiency symptom expressed as a ridged hollow between the cotyledons
holomorph—the whole organism
homothallism (adj. homothallic)—condition in which sexual reproduction can occur without the interaction of two different thalli, both sexes being present in the same mycelium
honeydew—sugary ooze or exudate, typically from aphids
host—living plant attacked by or harboring a parasite and from which the invader obtains part or all of its nourishment
host range—kinds of plants attacked by a given pathogen
hull—outer coat of a seed
hyaline—transparent or nearly so; translucent (often used in the sense of colorless)
hymenium—spore-bearing layer of a fungal fruiting body
hypanthium—enlargement of the floral receptacle
hypha (pl. hyphae, adj. hyphal)—tubular filament of a fungal thallus or mycelium; the basic structural unit of a fungus
hypocotyl—portion of a seedling below the cotyledons

icosahedral—having 20 faces, as a virus particle
immunogenic—producing immunity
imperfect state—asexual part of the life cycle of a fungus, during which asexual spores (such as conidia) or no spores are produced
in vitro—in glass, on artificial media, or in an artificial environment; outside the host
in vivo—within a living organism
inclusions—bodies suspended in the cytoplasm
indehiscent—pertaining to fruit that does not split open at maturity
infection (v. infect)—process of a pathogen entering and parasitizing a host plant
infection court—site in or on a host plant where infection can occur
infection peg—very fine hypha that is thrust through the cuticle or epidermis of a host cell
infestation (v. infest)—attack by animals, especially insects or nematodes; aggregation of inoculum or other organisms on a plant surface
inflorescence—flower or flower cluster
initial inoculum—the total population of a pathogen at the onset of an epidemic
injury—result of transitory operation of an adverse factor such as insect feeding, action of a chemical, or unfavorable environmental condition
inoculate (n. inoculation)—to place inoculum in an infection court
inoculum (pl. inocula)—pathogen or its parts capable of establishing a live colony when transferred to a favorable location
inoculum density—a measure of the number of propagules of a pathogenic organism
intercalary—between the apex and the base
intercellular—between or among cells
intercrop—to grow one crop between the rows of another crop
internode (adj. internodal)—area of stem between two nodes
interveinal—between (leaf) veins
intracellular—through or within cells
isolate (n.)—pure microbial culture separated from its natural origin
isometric—equally long

keel—the two anterior, united petals of a butterflylike legume flower
kernel—*see* seed

lamina—expanded part of a leaf
larva (pl. larvae)—juvenile; growth stage between embryo and adult
latent—present but not manifested or visible, as a symptomless infection by a pathogen
leaflet—one of the separate blades or divisions of a compound leaf
legume—simple, dry, dehiscent fruit that develops from a simple pistil and splits at maturity along two seams
lenticular—shaped like a double convex lens
lesion—wound or delimited diseased area
lignin—complex organic substance or group of substances that impregnate the cell walls of xylem vessels and certain other plant cells
local lesion—localized spot produced on a leaf upon mechanical inoculation with a virus
longevity in vitro—amount of time a virus remains infective when stored at room temperature
lygaeid—small, sucking insect

macerate—to soften by soaking; to cause disintegration of tissues by separation of cells
macroconidium—large conidium produced usually at a different period or on a different structure than microconidia
masked symptom—symptom that is absent under certain environmental conditions but that appears when the host is exposed to certain temperature and light conditions
maturation—process of becoming mature
mechanical damage—damage inflicted by equipment
mechanical transmission (or inoculation)—spread or introduction of inoculum to an infection court (especially a wound) accompanied by physical disruption of host tissues
medium (pl. media)—chemical environment providing nutrition for growth
meiosis—the process during which gametes are formed
melanized—darkened
membranous—membranelike
meristem (adj. meristematic)—formative plant tissue
mesocarp—middle coat of a pericarp
mesophyll—leaf tissue between the epidermal layers; cells containing chlorophyll
metabasidium—the part or stage of the basidium in which meiosis occurs
metula (pl. metulae)—a sporophore branch having phialides
microconidium (pl. microconidia)—the smaller conidium of a fungus that also has macroconidia
microsclerotium (pl. microsclerotia)—microscopic, dense aggregate of darkly pigmented, thick-walled hyphal cells
mildew—thin coating of mycelial growth and spores on the surfaces of infected plant parts
mitochondrion (pl. mitochondria)—various cellular organelles outside the nucleus
mitospore—uninucleate, diploid zoospores
monadelphous—pertaining to stamen filaments united into a tube or column
morphology (adj. morphologic)—form and structure of organisms
mosaic—virus disease characterized by dark and light green mottling of the foliage
motile—capable of self-propulsion by means of flagella, cilia, or amoebic movement
multinucleate—having more than one nucleus per cell
muriform—having transverse and longitudinal septa
mycelium (pl. mycelia, adj. mycelial)—mass of hyphae constituting the body (thallus) of a fungus
mycelogenic—pertaining to germination of sclerotium by production of mycelium
mycoflora—fungi characteristic of an environment
mycoparasite—fungus that attacks another fungus
mycophagous—feeding on fungi
mycoplasma—prokaryotic organism, smaller than conventional bacteria, lacking rigid cell walls and variable in shape
mycoplasmalike organism—organism with the apparent features of a mycoplasma but not proven to be a mycoplasma
mycorrhiza (pl. mycorrhizae, adj. mycorrhizal)—association between a symbiotic, nonpathogenic, or weakly pathogenic fungus and the roots of plants

necrosis (adj. necrotic)—death (of tissue), usually accompanied by discoloration
nematicide—agent, usually a chemical, that kills or inhibits nematodes

nematode—small, wormlike animal, parasitic in plants or animals or free living in soil or water
node—branch point on a stem at which leaves and buds arise
nodule (n. nodulation)—small knot or irregular, rounded lump; on leguminous plants, structures on roots that contain nitrogen-fixing bacteria
nonpedicellate—not borne on a slender stalk
nonpersistent—pertaining to viruses that are infectious within insect vectors for short periods and are transmissible without a latent period and without prior multiplication and translocation within the vector

obclavate—inversely clavate; widest near the base
obligate parasite—organism that in nature can survive only on or in living tissues
oblong—twice as long as wide and having somewhat truncate ends
obovate, obovoid—reversely egg-shaped, with the broader end uppermost
obtuse—rounded or blunt; greater than a right angle
oidium—spermatium formed on a hyphal branch; flat-ended, asexual spore formed by the breaking up of a hypha into cells; a mildew
olivaceous—olive or olive green in color
oogonium (pl. oogonia)—one-celled female sex organ of some fungi
oospore—thick-walled resting spore produced by the union of an oogonium and an antheridium
ostiole (adj. ostiolate)—pore; opening in the papilla or neck of a perithecium or pycnidium through which spores are released
ovary—enlarged basal portion of a pistil containing the ovules and developing into the fruit
ovate, ovoid—oval; egg-shaped
overwinter—to survive over the winter period
ovule—enclosed structure that, after fertilization, becomes a seed

palisade—tissue found beneath the upper epidermis of leaves composed of elongate, tubular cells arranged upright in the manner of posts in a palisade fortification
papilla (pl. papillae, adj. papillate)—small, blunt projections
paraphysis (pl. paraphyses)—sterile, upward-growing, basally attached hypha present in some kinds of fungal fruiting structures
parasite—organism that lives in or on another organism and obtains food from it
parenchyma (adj. parenchymatous)—thin-walled plant cells
pathogen (adj. pathogenic)—agent that causes disease
pathogenicity—ability to cause disease
pedicel—small stalk; stalk of an individual flower
peg—young fruit during the stalklike phase of development that comes after the union of gametes and before the enlargement of the fruit
penetration—initial invasion of a host by a pathogen
perfect state—the sexual stage in the life cycle of a fungus
pericarp—ripened and modified walls of a plant ovary (fruit)
perineal—the area between the anus and the posterior part of the external genitalia
perithecium (pl. perithecia)—flask-shaped or subglobose, thin-walled fungal fruiting body (ascocarp) containing asci and ascospores (spores are expelled or otherwise released through a pore, the ostiole, at the apex)
persistent—pertaining to viruses that are infectious within insect vectors for long periods
pest—any organism that injures plants or plant products
pesticide—a chemical used to control pests
petiole—stalk portion of a leaf
pH—measure of acidity and alkalinity (pH 7 is neutral; below pH 7 is acidic; above pH 7 is alkaline)
phaeosporous—having dark, one-celled spores
phanerogam—a seed plant or flowering plant
phialide (adj. phialidic)—end cell of a conidiophore or a conidiophore of fixed length with one or more open ends through which a basipetal succession of conidia develops
phialospore—conidium produced on a phialide
photosynthesis—manufacture of carbohydrates from carbon dioxide and water in the presence of chlorophyll(s) during which light energy is used and oxygen is released
phototropism—growth movement of plants induced by light
phragmospore—spore having two or more transverse septa
phyllotaxy—arrangement of leaves on a stem in relation to one another
phytoplasma—mycoplasmalike organism
phytotoxic (n. phytotoxicity)—harmful to plants
pigment—any coloring matter in the cells of plants or fungi
pinnate—featherlike; having parts arranged along two sides of an axis
pistil—the ovary, stigma, and style of a flower
pith—parenchymatous tissue occupying the center of the stem
plasmodium (pl. plasmodia)—naked, multinucleate mass of protoplasm moving and feeding in amoeboid fashion
pleomorphic—able to assume various shapes
plumule—bud of an ascending axis of a plant while still in the embryo
pod—fruit of a peanut plant that contains from one to six seed
pollen—male sex cells produced by anthers of flowering plants
pollination—transfer of pollen from anther to stigma or from a staminate cone to an ovulate cone
potyvirus—one of a group of related viruses
predispose—to make prone to infection and disease
primary inoculum—inoculum that initiates disease
primary root—root that develops directly from the radicle of an embryo rather than from a crown or node
primordial (n. primordia)—first in order of appearance; pertaining to the earliest stages of development
prokaryotic (n. prokaryote)—without internal membrane-bound organelles
promycelium (pl. promycelia)—in rusts and smuts, a germ tube issuing from the teliospore and bearing the basidiospores
propagule—any part of an organism capable of independent growth
pseudoparenchyma—fungal cells that resemble the parenchyma of higher plants
pseudothecium (pl. pseudothecia)—peritheciumlike fruiting body containing asci and ascospores
pulvinate—cushion-shaped
pustule—small, blisterlike elevation of epidermis formed as spores emerge
pycnidiospore—spore produced within a pycnidium
pycnidium (pl. pycnidia)—asexual, globose or flask-shaped fruiting body produced by fungi
pyramidal—pyramid-shaped
pyriform—pear-shaped

quadrifoliate—compound leaf composed of four leaflets
quarantine—legislative control of the transport of plants or plant parts to prevent spread of disease

race—subgroup or biotype within a species or variety distinguished from other races by behavior (virulence, symptom expression, or host range) but not by morphology
radiate (adj.)—spreading from or arranged around the center
radicle—part of the embryo that develops into the primary root
rasping—using a roughened surface
reniform—kidney-shaped
resistance (adj. resistant)—property of a host that prevents or impedes disease development
reticulate—having the form of a net
rhizobium nodules—galls on roots caused by *Rhizobium* spp.
rhizomorph—visible strand or cord of compacted mycelium, often dark colored
rhizosphere—microenvironment in soil immediately around plant roots
rickettsialike organism—pleomorphic or rod-shaped, nonfilterable bacterium that causes disease (many are arthropod-borne; most have not been cultured in artificial media)
RNA—ribonucleic acid
rogue—to remove and destroy by hand individual plants that are diseased, infested by insects, or otherwise undesirable
rosette—disease symptom characterized by short, bunchy growth habit resulting from shortened internodes but no comparable reduction in leaf size
rugose—wrinkled
runner plant—new plant produced asexually on a runner or stolon
russet—brownish, roughened tissue resulting from cork formation

saprophyte (adj. saprophytic)—organism that feeds on dead organic matter

scab—roughened, crustlike diseased area on the surface of a plant organ

sclerenchyma (adj. sclerenchymatous)—thick tissue made up of thick-walled plant cells

sclerotium (pl. sclerotia)—hard, frequently rounded, usually darkly pigmented resting body of a fungus composed of a mass of specialized hyphal cells (the structure may remain dormant for long periods and then germinate to produce a stroma, fruiting body, mycelium, or conidiophores when favorable conditions return)

scorch—any symptom, such as a lesion or system of lesions, that suggests the action of flame or fire on the affected part, especially on extending organs such as green leaves or petals

secondary infection—infection resulting from the spread of infectious material produced after a primary infection or from other secondary infections without an intervening inactive period

secondary root—branch from a primary root

sedimentation coefficient—measure (expressed in Svedberg units [S] and proportional to molecular weight) of the velocity with which a compound or structure is spun down during ultracentrifugation

seed—ripened ovule consisting of an embryo and stored food enclosed by a seed coat

seedborne—carried on or in a seed

self-pollination—transfer of pollen from the anthers to the stigma of the same flower

senesce (adj. senescent, n. senescence)—to decline, as with maturation, age, or disease stress

sepal—one of the modified leaves comprising a calyx

septum (pl. septa, adj. septate)—cross wall

serology (adj. serologic)—study, detection, and identification of antigens, antibodies, and their reactions

sessile—lacking a petiole, as in some leaves, or a pedicel, as in some flowers and fruits

seta (pl. setae)—bristle or bristle-shaped body, usually deep yellow or brown

setula—delicate, hairlike appendage arising from the end of a conidium

sexual spore—spore produced as a result of meiosis

shell—tissue in which seed are enclosed

sinuous—having many curves, bends, or turns

soilborne—carried on or beneath the soil surface

sorus (pl. sori)—compact fruiting structure, especially spore masses in rust and smut fungi; occasionally a group of fruiting bodies

sound mature kernels—undamaged seed large enough to ride the officially designated screen size for a specific market type of peanut

sow—to plant seed

sp. (pl. spp.)—species (a genus name followed by sp. means that the particular species is undetermined; spp. after a genus name means that several species are being referred to without being named individually)

spermagonium (pl. spermagonia)—walled structure in which spermatia are produced

spermatium (pl. spermatia)—a sex cell

sporadic—appearing at irregular intervals

sporangium (pl. sporangia)—structure in lower fungi containing asexually formed spores

spore—one- to many-celled reproductive body in fungi and lower plants

sporodochium (pl. sporodochia)—superficial, cushion-shaped, asexual fruiting body

sporulate—to produce spores

stamen (adj. staminal)—pollen-producing organ of a flower, usually consisting of a filament and an anther

stellate—pertaining to starlike extensions on spores

sterigma (pl. sterigmata)—small, usually pointed protuberance or projection that supports a spore

stigma—portion of a flower that receives pollen and on which the pollen germinates

stipe—stalk

stipule—small, leaflike appendage at the base of a leaf petiole, usually occurring in pairs

stolon—a long, slender, modified stem arising from the crown that produces new plants; runner; in fungi, hypha that grows horizontally along the surface

stoma (pl. stomata)—small pore bordered by guard cells found in the epidermis of leaves, stems, and other plant parts and through which exchange of gases occurs

striated (n. striations)—marked with delicate lines, grooves, or ridges

stroma (pl. stromata)—compact mass of mycelium (with or without host tissue) that supports fruiting bodies or in which fruiting bodies are embedded

stunt—reduction in height of a vertical axis resulting from a progressive reduction in the length of successive internodes or a decrease in their number

style—slender part of many pistils located between the stigma and the ovary and through which the pollen tube grows

stylet—stiff, slender, hollow feeding organ of plant-parasitic nematodes

stylospore—spore on a pedicel or hypha, especially a urediospore; an elongated pycnidiospore

subacute—somewhat acute

subcarbonaceous—almost like charcoal or cinders

subepidermal—occurring beneath the epidermis

subflexuous—almost flexuous

subglobose—almost globose

subhyaline—somewhat or imperfectly clear

sublethal—almost fatal or lethal

submicroscopic—smaller than can be seen with a compound microscope

subspherical—almost spherical

substrate—substance on which organisms grow

symptom—indication of disease by reaction of the host

symptomless carrier—a plant that, although infected with a pathogen (usually a virus), produces no obvious symptoms

syn.—synonym(s)

syndrome—pattern or sequence of disease development; a complex of symptoms

synergism (adj. synergistic)—concurrent parasitism of a host by two pathogens in which the symptoms or other effects are of greater magnitude than the sum of the effects of each pathogen acting alone

synnemata—a group of conidiophores cemented together and forming an elongated, spore-bearing structure

systemic—pertaining to chemicals or pathogens that spread throughout a plant rather than remaining localized

taproot—primary root that grows vertically downward and gives off smaller lateral roots

taxonomy (adj. taxonomic)—the science dealing with naming and classifying organisms

teleomorph—perfect (sexual) state of a fungus

teliospore—thick-walled resting spore that germinates to form a basidium

telium (pl. telia)—sorus that produces teliospores

testa (pl. testae)—seed coat

thermal inactivation point—lowest temperature at which heating for a limited period (usually 10 min) is sufficient to cause a virus to lose its infectivity or an enzyme its activity

tissue—group of cells, usually of similar structure, that perform the same or related functions

torulose—cylindric but having swellings at intervals

toxicity—capacity of a substance to produce injury

toxin—poison produced by a living organism

translocation—transfer or movement of foods and other products of metabolism

translucent—so clear that light may pass through

transpiration—the giving off of water vapor from living plants

truncate—ending abruptly as though the end had been cut

turgid—plump or swollen as a result of internal water pressure

unicellular—one-celled

uniloculate—containing a single cavity

urediniospore—binucleate, repeating spore of rust fungi

uredinium—fruiting body of rust fungi that produces urediniospores

variety (adj. varietal)—group of closely related plants of common origin within a species that differ from other varieties in certain minor details such as form, color, flower, and fruit

vascular—pertaining to fluid-conducting (xylem and phloem) tissues
vector—agent that transmits inoculum
vegetative—asexual; somatic
veinbanding—symptom of viral disease in which regions along veins are darker green than tissue between veins
veinclearing—disappearance of green color in or around leaf veins
vesicle (adj. vesicular)—thin, bladderlike sac or structure in which zoospores are differentiated or released; the bulbous head terminating a conidiophore of *Aspergillus*; sporelike structure formed by mycorrhizal fungi
viable—able to live; able to survive, as spores
viroplasm—cellular inclusions that are sites of synthesis of viral components and the assembly of virus particles
virulence—degree or measure of pathogenicity; relative capacity to cause disease
virulent—strongly pathogenic
volatile—evaporating rapidly
volunteer—self-set plant; plant seeded by chance

water-soaked—describing a disease symptom of plants or lesions that appear wet, dark, and usually sunken and translucent
whorl—circular arrangement of like parts
wilt—lack of freshness; drooping of leaves from lack of water (inadequate water supply or excessive transpiration); a vascular disease that interrupts the normal uptake and distribution of water

xerophyte—plant adapted to a limited water supply
xylem—water-conducting tissue in plants

yellows—a plant disease characterized by yellowing and stunting of the host plant

zonate—marked with zones; targetlike; appearing in concentric rings
zoospore—motile spore
zygospore—resting spore formed from the union of like gametangia

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