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

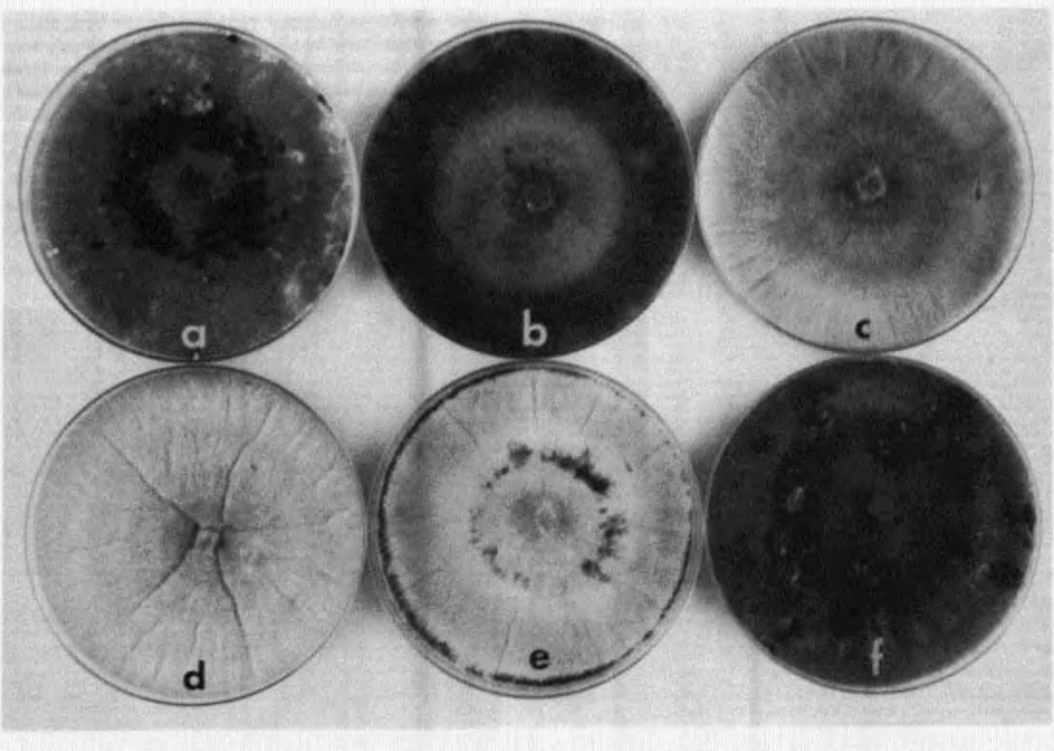

Fig. 42. **a–c,** Multinucleate anastomosis groups of *Rhizoctonia solani* and **d–f,** binucleate *Ceratobasidium* anastomosis groups of *Rhizoctonia*-like fungi from peanut. (Courtesy D. Bell)

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.

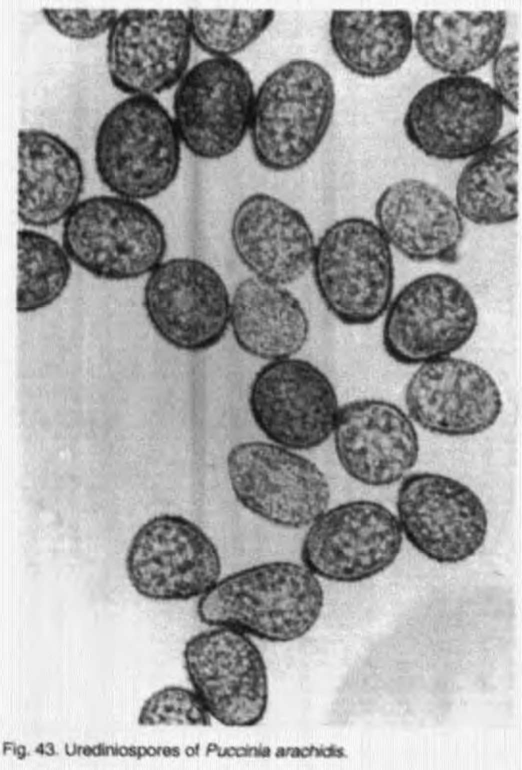

Fig. 43. Urediniospores of *Puccinia arachidis*.

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

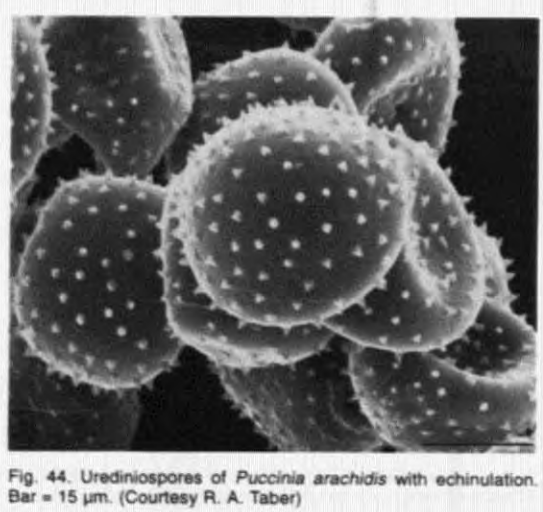

Fig. 44. Urediniospores of *Puccinia arachidis* with echinulation. Bar = 15 µm. (Courtesy R. A. Taber)

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.

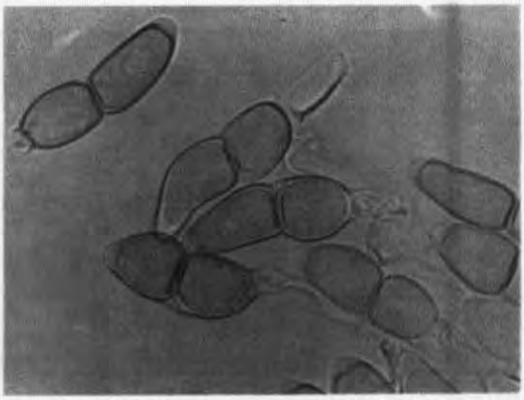

Fig. 45. Teliospores of *Puccinia arachidis*. (Courtesy J. F. Hennen)

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.