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Peanut Rust: A Major Threat to Peanut Production in the Semiarid Tropics

The cultivated peanut (*Arachis hypogaea* L.), an annual legume native to South America and known as the groundnut in some areas, is grown throughout the regions of the world bounded by latitudes 40° north and 40° south. The seed contains up to 50% nondrying oil and approximately 25% protein. The oil is used mainly for cooking and for producing soaps and edible fats, and the cake remaining after oil extraction is used chiefly for animal feed. The seeds are consumed whole as boiled or roasted kernels or are processed into various confections. The haulm (hay) remaining after pod removal constitutes a valuable and nutritious animal feed.

The Food and Agriculture Organization of the United Nations estimates that in 1983, over 18.9 million ha worldwide were planted to peanuts and that 19.9 million t were harvested, with an average dried pod yield of 1,056 kg/ha. Asia was the largest producer (13.3 million t), followed by Africa (4.4 million t), North and Central America (1.6 million t), and South America (0.6 million t). Among individual countries, India produced 7.5 million t, the People's Republic of China 2.4 million t, Senegal 1.1 million t, Sudan 0.97 million t, Nigeria 0.6 million t, and the United States 0.56 million t.

Approximately 80% of world production comes from developing countries, with 67% of the total produced in the seasonally dry rain-fed areas of the semiarid tropics. In the semiarid tropics, the crop is grown almost entirely by small farmers, and yields are low, around 800–900 kg/ha, compared with approximately 2,500 kg/ha in the developed world. The major constraints are diseases, pests, and unreliable rainfall patterns (2). Few farmers can afford the cost of crop-protection chemicals and application equipment, and fewer still have the technical knowledge required for effective application of pesticides. Also, the chance of crop failure owing to inadequate rainfall discourages expensive inputs. Most cultivars grown in the semiarid tropics are susceptible to the major peanut diseases. A large number of fungus, virus, and nematode diseases of peanut have been reported, and with few exceptions, they are common in all peanut-growing regions of the semiarid tropics.

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Distribution of Peanut Rust

Peanut rust caused by *Puccinia arachidis* Speg. has increased in importance in recent years. Prior to 1969, the disease was largely confined to South and Central America, with occasional outbreaks occurring in the southernmost peanut-producing areas of the United States. The disease was also recorded in the Soviet Union in 1910, in Mauritius in 1914, and in the People's Republic of China in 1937 but did not become permanently established (1,3,11) (Fig. 1, top). In recent years, peanut rust has spread to many countries in Asia (Brunei, India, Indonesia, Japan, Korea, Malaysia, Philippines, Taiwan, and Thailand), Australasia and Oceania (Australia, Fiji, New Guinea, and Solomon Islands), and Africa (Benin, Botswana, Burkina Faso, Ethiopia, Ghana, Kenya, Malawi, Mauritius, Mozambique, Nigeria, Republic of South Africa, Senegal, Somalia, Sudan, Tanzania, Uganda, Zambia, and Zimbabwe) (3,11) (Fig. 1, bottom).

Yield Losses

Rust is now of economic importance in most peanut-growing areas of the world. Figure 2 shows a severe rust attack on a farmer's crop in southern India. Yield losses are substantial, particularly if the crop is also attacked by the two major leaf spot fungi, *Cercospora arachidicola* Hori and *Cercosporidium personatum* (Berk. & Curt.) Deighton. Because rust is commonly accompanied by these leaf spots, losses have been assessed through the use of fungicides with specific action against individual foliar diseases. This has permitted estimation of losses from various combinations of foliar diseases and from rust alone. Losses measured at two locations in Texas were 50 and 70% from rust alone. At ICRISAT in Patancheru, India, rust caused losses of over 50% (11).

The time at which rust occurs is important. In the People's Republic of China, losses when rust occurred at flowering, pegging, pre-pod-forming, and mid-pod-forming stages were estimated to be 49, 41, 31, and 18%, respectively (15). In infected plants, pods mature 2 or 3 weeks early, seeds are small, pods become detached in the soil during lifting, and the oil content is reduced. In addition, haulm yields are lowered drastically.

Symptoms and Identification of the Pathogen

Rust can be readily recognized when orange pustules (uredinia) first appear on the lower surface of the leaflet and rupture to release reddish brown urediniospores (Fig. 3A,B). In highly susceptible genotypes, the original pustules may be surrounded by secondary pustules. Later, pustules may form on the upper surface of the leaflet opposite those on the lower surface. The pustules, which develop on all aerial plant parts (Fig. 3C) except the flowers, are usually circular and range from 0.5 to 1.4 mm in diameter. In contrast with the rapid defoliation associated with leaf spots, rust-infected leaves become necrotic and dry but tend to remain attached to the plant (Fig. 3D).

Only uredinial and telial stages of the fungus are known. Urediniospores are broadly ellipsoid or obovoid, 16–22 (–24) × (21–) 23–29 (–30) μm, with brown walls 1–2.2 μm thick, finely echinulate with mostly two but occasionally three or four germ pores, nearly equatorial, and often on flattened areas (Fig. 4, right). Telia chiefly occur on the lower surface of leaves. Teliospores are oblong, obovate, ellipsoid, or ovate with a rounded to acute and thickened apex. They are constricted at the septum, smooth-walled, and light or golden yellow or chestnut brown (Fig. 4, left). Predominantly two-celled, they sometimes have three or four cells, (33–) 38–42 (–60) × (12–) 14–16 (–18) μm thick at the sides and 2.5–4.0 (–5.0) μm thick at the top, with almost hyaline apical thickening. The pedicel is thin-walled and up to 35–65 μm long. Teliospores germinate at maturity without dormancy (4).

Peanut rust occurs almost exclusively in its uredinial stage. There are a few

reports of the occurrence of teliospores, mainly from South America. Teliospores are important because karyogamy and meiosis, essential for sexual reproduction, occur during teliospore germination. As a result, basidiospores are formed and disseminated. Under favorable conditions, the basidiospores infect appropriate hosts; no such host is known for the peanut rust pathogen, however. Without knowledge of the host and of the structures produced on it, the life cycle and taxonomic relationships cannot be determined, and classification can be only tentative (J. F. Hennen, *personal communication*).

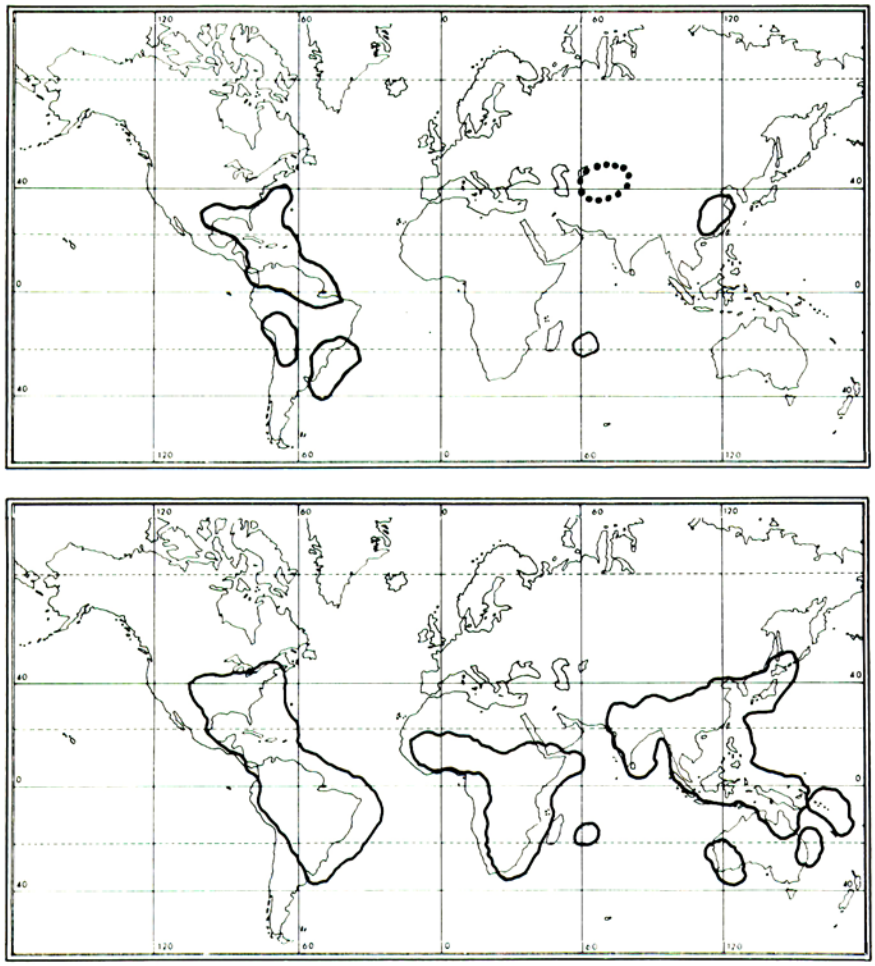

Fig. 1. Geographical distribution of *Puccinia arachidis* (top) prior to 1969 (based on Commonwealth Mycological Institute map 16, issued 30 June 1966) and (bottom) in 1983 (based on Commonwealth Mycological Institute map 160, issued 1 April 1980). (From Subrahmanyam and McDonald [11])

Fig. 2. Severe rust attack on a peanut crop in southern India.

Disease Cycle and Epidemiology

It is not known if the fungus can produce spermagonia and aecia or if any alternate host is involved in the life cycle. There is no record of any hosts other than the genus *Arachis*, so it is improbable that such hosts could be involved in perpetuating peanut rust outside their native South America (10). Local and long-distance dissemination of the pathogen may be by airborne urediniospores, by movement of infected crop debris, or by movement of pods or seeds surface-contaminated with urediniospores or infected crop debris. There is no reliable evidence of peanut rust being internally seedborne or being spread by germ plasm exchange (10).

Urediniospores are reported to be short-lived in infected crop debris. Therefore, the fungus is unlikely to survive from season to season under postharvest conditions that include a break of more than 4 weeks between crops. The practice of continuous peanut cultivation without any break (Fig. 5) appears to be an important factor in perpetuating peanut rust in India and China. The pathogen may also survive from season to season on volunteer peanut plants (10).

Plants of all ages are susceptible. The incubation period varies from 7 to 20 days, depending on host genotype and environmental conditions. An optimum temperature in the 20–30 C range, free water on the leaf surface, and high relative humidity favor infection and subsequent disease development (5). Spread of the disease within crops is facilitated by wind movement, by rain splash, and by insects. Given favorable conditions, disease spread continues throughout the season and may cause total desiccation of the foliage.

Cultural and Chemical Control Strategies

Cultural measures. Eradication of volunteer peanut plants and "groundkeepers" is important in reducing the primary sources of inoculum. Wherever possible, field management should include a break of at least 4 weeks between successive peanut crops. If cropping systems permit, sowing times should be adjusted to avoid early infection of the crop from outside sources and to avoid the environmental conditions conducive to disease buildup. Strict plant quarantine regulations should be enforced to prevent movement of pods or seeds externally contaminated with rust spores to areas where the disease is not present (10).

Chemicals. A number of chemicals developed for control of leaf spots have been tested for effectiveness against rust. Some are effective and economical when used by farmers in developed countries (8). The dust formulations (copper, sulfur, and copper plus sulfur) that were commonly used in the United States up to the 1960s controlled leaf spots well but only partially controlled rust. Sprays of Bordeaux mixture and dithiocarbamates were more effective. The structurally related systemic fungicides benomyl and carbendazim are effective against leaf spots but ineffective against rust, and in some cases, their application apparently increased the severity of rust attack. Tridemorph is effective against rust but not against leaf spots. Chlorothalonil gives good control of both rust and leaf spots (Fig. 6). It is important that any fungicide treatment applied for rust control also be effective against leaf spots (8).

Experiments on fungicidal control of rust have been conducted in a number of countries in the semiarid tropics, and large increases in yield of both pods and haulms have been obtained. Very few farmers in the semiarid tropics have adopted the practice, however, because of economic reasons.

Breeding for Resistance

Developing resistant cultivars is one of the best means of reducing crop losses from disease. It is a policy particularly well suited to small-scale farmers of the semiarid tropics who generally lack the financial resources and technical expertise required for chemical disease control. Prior to 1976, there were only a few reports of research on genetic resistance to peanut rust, but the rapid spread of rust in the early 1970s and the increasing cost of chemical control have stimulated work in this field (2,3).

Screening methods and sources of resistance. Effective screening methods have been developed for use in areas where natural disease pressure is high or where such pressure can be artificially induced. Genotypes to be screened are sown in replicated plots with rows of a highly susceptible cultivar arranged systematically throughout the trial. At ICRISAT, where rust disease is severe in the rainy season, the ratio of "infector" rows to those of test genotypes is 1:4. A higher or lower ratio may be appropriate in other localities. To enhance disease development, the plants in the infector row can be inoculated with a suspension of urediniospores (Fig. 7A) and irrigated by overhead sprinklers (Fig. 7B). Inoculation is most successful if done in the evening because strong sunlight inhibits urediniospore germination (9,11).

At ICRISAT, some 10,000 peanut germ plasm lines from different parts of the world were screened in the field for resistance to rust during 1977–1984 (Fig. 7C). Previous reports of resistance were

Fig. 3. (A) Rust pustules on lower leaf surface, (B) close-up of pustules showing masses of urediniospores, (C) rust pustules on stem, and (D) dry, necrotic rust-infected leaves.

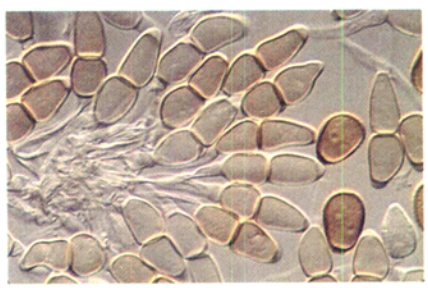

Fig. 4. (Left) Teliospores and (right) urediniospores of *Puccinia arachidis*. (Courtesy J. F. Hennen, Purdue University, West Lafayette, IN)

Fig. 5. Peanut crops at (left) pod-filling stage and (right) seedling stage in a farmer's field in Andhra Pradesh, India. Such overlapping helps to perpetuate peanut rust.

Fig. 6. Chemical control of peanut rust at ICRISAT. Plots at front and rear have been sprayed with fungicides; center plot is untreated.

confirmed and several new sources were identified (Table 1). Fourteen rust-resistant germ plasm lines have been jointly released recently by the U.S. Department of Agriculture and ICRISAT (Table 1).

The majority of rust-resistant genotypes reported so far have their origins in Peru, believed to be one of the secondary gene centers of the cultivated peanut (var. *hypogaea* and var. *fastigiata*). The resistant lines from Honduras have genes from the resistant parent, Tarapoto, which comes from Peru. The origin of the lines ICG 2716 (from Uganda) and ICG 6022 (from Sudan) is uncertain, but judging from their pod and plant characteristics, they are probably introductions from the Peruvian region. About 70% of the resistant lines listed in Table 1 are from Peru and another 20% probably originated elsewhere in South America.

Screening germ plasm for resistance to peanut rust can also be done on a limited scale in the glasshouse by measuring the components of disease resistance-incubation period, infection frequency, pustule diameter, percentage of pustules ruptured, percentage of leaf area damaged, and sporulation (Fig. 8). This technique is useful for separating resistant and susceptible genotypes but is less effective than field screening for identifying moderately resistant genotypes. A glasshouse screening method could be useful in areas where rust epidemics do not occur regularly or where other foliar diseases interfere with field screening (12,13).

Many research institutions have screened wild *Arachis* spp. for resistance to peanut rust in the field and in the laboratory, using rooted detached leaves. A large number of species were reported to be immune and some to be highly resistant (Table 2, Fig. 9) (14).

The nature of resistance. Rust resistance is not correlated with either the frequency or the size of stomata, and urediniospores germinate and germ tubes enter through stomata irrespective of whether a genotype is immune, resistant, or susceptible to rust. In immune *Arachis*

Table 1. Sources of resistance to peanut rust (*Puccinia arachidis*) available from ICRISAT (up to 1983)

Genotype	ICG no.[a]	Botanical type/variety	Seed color[b]	Country of origin
NC Ac 17090	1697	*fastigiata*	Light tan	Peru
EC 76446 (292)[c]	2716	*fastigiata*	Purple	Uganda
USA 63[c]	3527	*fastigiata*	Purple	...
PI 259747[c]	4747	*fastigiata*	Purple	Peru
Krap. St. 16[c]	4790	*fastigiata*	Purple	Argentina
NC Ac 927[c]	6022	*fastigiata*	Purple	Sudan
PI 270806[c]	6330	*fastigiata*	Purple	Zimbabwe
PI 350680[c]	6340	*fastigiata*	Purple	Honduras
NC Ac 17133-RF[c]	7013	*fastigiata*	Purple	Peru
PI 215696[c]	7881[d]	*fastigiata*	Purple	Peru
PI 314817	7882[d]	*fastigiata*	Light tan	Peru
PI 315608	7883[d]	*hypogaea*	Off-white	Israel/United States
PI 341879[c]	7884	*fastigiata*	Purple	Peru
PI 381622[c]	7885	*fastigiata*	Purple	Peru
PI 390593	7886[d]	*fastigiata*	Light tan	Peru
PI 390595[c]	7887[d]	*fastigiata*	Purple	Peru
PI 393516[c]	7888[d]	*fastigiata*	White with red blotches	Peru
PI 393517	7889[d]	*fastigiata*	Off-white	Peru
PI 393526	7890[d]	*fastigiata*	Purple	Peru
PI 393527	7891[d]	*hypogaea*	Red	Peru
PI 393527-B	7892	*hypogaea*	Red	Peru
PI 393531	7893[d]	*fastigiata*	Tan with purple stripes	Peru
PI 393641[c]	7894[d]	*fastigiata*	Light tan with purple stripes	Peru
PI 393643	7895[d]	*fastigiata*	Light tan	Peru
PI 393646	7896[d]	*fastigiata*	Purple	Peru
PI 405132[c]	7897	*fastigiata*	Purple	Peru
PI 407454	7898[d]	*fastigiata*	Tan	Ecuador
PI 414331	7899	*hypogaea*	Tan	Honduras
PI 414332	7900	*hypogaea*	Tan	Honduras

[a] ICRISAT groundnut accession number.
[b] Royal Horticultural Society color chart, London, 1966.
[c] Also resistant to late leaf spot (*Cercosporidium personatum*) at ICRISAT.
[d] Germ plasm lines jointly released by the U.S. Department of Agriculture and ICRISAT (up to 1983).

Table 2. Reaction of some wild *Arachis* species to *Puccinia arachidis*[a]

Section Species	Rust reaction
Arachis	
A. batizocoi	Immune
A. duranensis	Immune
A. spegazzinii	Immune
A. correntina	Immune
A. stenosperma	Highly resistant
A. cardenasii	Immune
A. chacoense	Immune
A. villosa	Immune
Erectoides	
A. apressipila	Immune
A. paraguariensis	Immune
Triseminale	
A. pusilla	Immune
Extranervosae	
A. villosulicarpa	Immune
Rhizomatosae	
A. hagenbeckii	Immune
A. glabrata	Immune

[a] From Subrahmanyam et al (14).

Table 3. Foliar diseases resistance varietal trial at ICRISAT, 1983 rainy season

Identity	Rust reaction[a]	Shelling (%)	Yield of dry pods (kg/ha)	
			High input[b]	Low input[c]
Rust-resistant ICRISAT lines				
ICG (CG:FDRS)-17	5.5	60	3,600	3,530
ICG (FDRS)-10	2.8	63	3,530	3,250
ICG (FDRS)-12	2.8	61	3,420	1,880
ICG 7898	3.1	56	3,310	1,710
ICG 7882	3.1	60	3,190	2,220
ICG (CG:FDRS)-18	5.9	64	3,170	3,360
ICG (FDRS)-15	3.8	58	3,140	1,920
ICG (FDRS)-4	3.0	58	3,100	2,620
ICG (FDRS)-8	3.3	59	3,080	2,350
ICG (FDRS)-2	3.3	58	3,070	1,860
ICG (FDRS)-11	3.6	65	3,010	2,560
Rust-susceptible Indian cultivars				
Robut 33-1	8.3	59	2,730	2,250
JL 24	9.0	58	2,380	1,840
J 11	9.0	62	2,110	1,720

[a] Scored on a nine-point disease scale.
[b] Phosphorus pentoxide (60 kg/ha), irrigation, insecticide spray.
[c] Phosphorus pentoxide (20 kg/ha), rain-fed, no insecticide spray.

spp., however, the fungus dies shortly after entering the substomatal cavity. Differences in resistance are associated with differences in rate and extent of mycelial development within the cavity and within leaf tissues. The rust resistance available at present in the cultivated peanut is the "slow-rusting" type, i.e., resistant genotypes have increased incubation periods, decreased infection frequency, and reduced pustule size, spore production, and spore germinability (12,13).

Stability of resistance. Stability of host resistance is an important objective for any disease-resistance breeding program and is particularly important for international programs. The stability can be checked by testing genotypes under natural disease epidemics in widely separated geographic locations. Most of the genotypes listed in Table 1 are being tested in different locations of the semiarid tropics in the International Groundnut Foliar Diseases Nursery. The results obtained so far indicate that rust resistance is stable over widely separated locations (9). There is no authenticated report of races of differing pathogenicity.

Utilization of resistance. Most of the rust-resistant germ plasm lines are unadapted and have undesirable pod and seed characters. At ICRISAT, a large-scale field hybridization program has been initiated to combine resistance with good agronomic characters (Fig. 10). More than 700 single, two-way, and three-way crosses have been made with 12 lines resistant to rust and nine lines resistant to late leaf spot. 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 by pedigree and bulk pedigree breeding methods. Backcrossing has also been adopted in a few instances to improve pod, kernel, and plant characters. Several of these resistant lines have outyielded the released susceptible Indian cultivars Robut 33-1 and JL 24 and have given a pod yield of over 4,000 kg/ha at ICRISAT (Tables 3 and 4, Fig. 11).

The early generation breeding material generated from crosses between rust-resistant peanut germ plasm lines and high-yielding but susceptible cultivars at ICRISAT has been freely distributed to breeders and pathologists in national and international programs to enable them to carry out further selection in situ under local agroclimatic conditions (6).

Cytogenetic research aimed at incorporating rust resistance from wild *Arachis* spp. into the cultivated peanut is also in progress. Wild *Arachis* spp. may have mechanisms of rust resistance that differ from those in the cultivated peanut, thus providing the possibility of combining the resistances from wild and cultivated species to give more effective and stable rust resistance in the cultivated peanut. At ICRISAT, several stable tetraploid or near-tetraploid lines have been developed from crosses between the cultivated peanut and wild species immune or highly resistant to rust (Fig. 12). Some of these derivatives have given yields significantly higher than those of

Table 4. Performance of some rust-resistant advanced breeding lines at ICRISAT, 1983 rainy season

Identity	Rust reaction[a]	Yield of dry pods (kg/ha)	Percent yield advantage over susceptible Indian cultivars	
			JL 24	Robut 33-1
(RMP 91 × DHT 200) F6-B1 (S1)	3.2	4,060	85.4	44.5
(RMP 91 × DHT 200) F6-B1 (S2)	3.0	3,730	70.3	32.7
(Robut 33-1 × PI 298115) F5-B	3.5	3,650	66.7	29.9
(NC-Fla-14 × NC Ac 17090) F9-B	3.7	3,150	37.6	18.0
(Tifspan × NC Ac 17090) F9-B	3.0	3,060	33.6	14.6
(Ah 65 × NC Ac 17090) F8-B	3.3	4,160	82.5	72.6
(NC Ac 2190 × NC Ac 17090) F8-B	3.2	4,150	82.0	72.2
(NC Ac 1107 × NC Ac 17090) F9-B	2.8	4,070	50.2	59.0
(JH 60 × PI 259747) F9-B	2.8	3,850	44.7	50.4

[a]Scored on a nine-point disease scale.

Table 5. Performance of some rust-resistant interspecific hybrid derivatives at ICRISAT, 1982 rainy season

Identity	Rust reaction[a]	Yield (kg/ha)		
		Dry pods	Haulms	Oil[b]
Rust-resistant ICRISAT lines				
CS 13	2.3	3,150	4,570	820
CS 30	2.7	2,640	6,540	760
CS 46	6.7	2,540	4,490	770
CS 11	2.7	2,520	5,620	770
CS 38	3.0	2,440	3,810	730
Rust-susceptible Indian cultivars				
TMV 2	9.0	1,240	1,630	350
Robut 33-1	9.0	1,830	1,560	510

[a]Scored on a nine-point disease scale.
[b]Estimated values.

Fig. 7. Screening of germ plasm for resistance to peanut rust at ICRISAT: (A) Inoculation of infector rows with rust, (B) irrigation by overhead sprinklers to enhance rust development, and (C) disease assessment.

Fig. 8. Glasshouse evaluation of germ plasm for resistance to rust. NC Ac 17090 (left) is resistant and TMV 2 (right) is susceptible to rust.

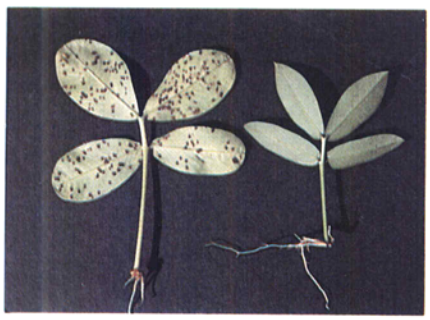

Fig. 9. Susceptible TMV 2 (left) compared with (right) wild *Arachis* sp. with immunity to peanut rust.

Fig. 10. Field hybridization combining rust resistance with elite agronomic characters at ICRISAT.

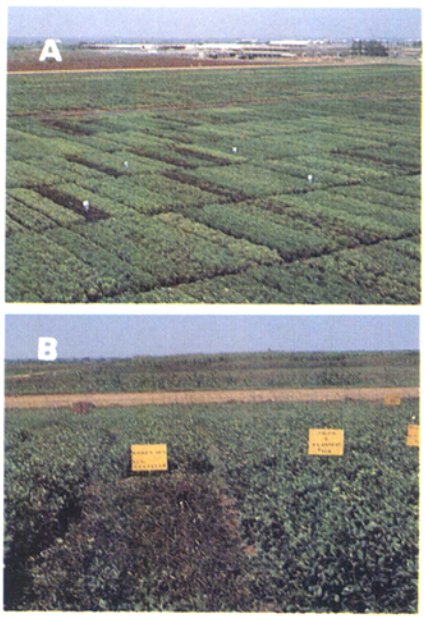

Fig. 11. (A) Aerial view and (B) close-up of yield evaluation of rust-resistant breeding lines at ICRISAT.

Fig. 12. (Left) High-yielding interspecific hybrid derivative and (right) susceptible Indian peanut cultivar.

the rust-susceptible Indian cultivars (Table 5).

Genetics of resistance. The genetic analysis of parents, F_1, F_2, BC_1, and BC_2 generations of resistant × susceptible crosses of the cultivated peanut at ICRISAT revealed that rust resistance is recessive and is predominantly controlled by additive, additive × additive, and additive × dominance gene effects. Duplicate epistasis was observed both for rust disease scores and for leaf area damage. Further studies are required to show conclusively if rust resistance is governed by two or three major genes or by many genes. In some diploid wild *Arachis* spp., however, rust resistance appears to be partially dominant, whereas it is recessive in the cultivated peanut (7). The dominant nature of the resistant genes available in wild *Arachis* spp. simplifies the backcrossing procedures in a breeding program.

Unanswered Questions

Mycologists have known about peanut rust since 1884, from infected leaf specimens collected in Paraguay. Early in this century, the disease was reported from the Soviet Union, the People's Republic of China, and Mauritius, but it did not become permanently established. In the United States, rust is not regarded as a major constraint of peanut production, despite occasional outbreaks (3,8). The disease was apparently endemic to South and Central America until 1969. Then, in the early 1970s rust was found in almost all major peanut-producing areas in Asia, Australasia, Oceania, and Africa, often reaching epidemic proportions. How do we explain this sudden spread of the disease? How were the fungal propagules carried to all these countries? Did the inoculum reach Asia and Africa via the Atlantic or Pacific? We have no satisfactory answers to these questions. Although in the past it was thought that peanut rust might have spread through exchange of germ plasm, spread of the disease to almost all peanut-producing countries outside the Americas in such a short time is very difficult to explain on this basis. Exchange of peanut germ plasm between the Americas and the rest of the world is not of recent origin, and the possibility should be slight of viable rust spores being carried on fungicide-treated kernels from pods stored for at least 40 days.

The Future

Peanut rust is a challenging problem to scientists concerned with peanut improvement programs throughout the world. A broad research effort is needed to develop rust-resistant peanut cultivars. More sources of resistance should be identified, preferably from different genetic backgrounds. Systematic exploration of peanut germ plasm in South America, especially in Peru, may be useful in obtaining more sources of rust resistance. Utilization of rust resistance from wild species would help to broaden the genetic base of resistance in cultivated peanuts.

Research on the possible occurrence of physiological races is very important for a successful disease resistance program, and efforts should be made to identify existing and future races. An understanding of the life cycle and taxonomic relationships of the rust fungus is also essential.

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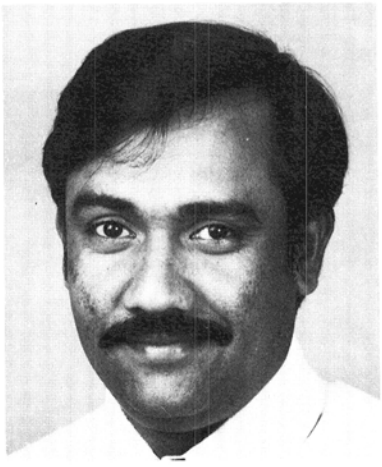

P. Subrahmanyam

Dr. Subrahmanyam is a plant pathologist in the Groundnut Improvement Program at ICRISAT. He has been working on peanut diseases since 1971, and his current research is primarily on disease resistance and epidemiology of peanut foliar diseases. He conducted the ICRISAT-USDA cooperative foliar disease nurseries and released 14 rust-resistant germ plasm lines. He participated in peanut disease surveys in India and southern and western Africa. Recently, he spent a 1-year sabbatical as a visiting research scientist at the Texas A&M University Plant Disease Research Station in Yoakum.

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Dr. Reddy is a plant breeder in the Groundnut Improvement Program at ICRISAT. He has over 14 years of experience in breeding grain legumes and was a pigeonpea breeder at ICRISAT before transferring to peanut breeding in 1982. He is concerned mainly with breeding peanuts for resistance to rust and leaf spots but also has research interests in exploiting hybrid vigor, distant hybridization, and breeding for quality and high yield.

R. W. Gibbons

Mr. Gibbons is principal groundnut breeder and leader of the Groundnut Improvement Program at ICRISAT. He worked as a peanut breeder with the Ministry of Agriculture of Northern Nigeria during 1957–1963 and as a breeder and leader of the Grain Legume Productivity Unit with the Agricultural Research Council of Central Africa and Malawi during 1963–1976. He joined ICRISAT in 1976 to set up the Groundnut Improvement Program. His research interests cover all aspects of peanut breeding and improvement with special reference to the developing world.

D. McDonald

Dr. McDonald is principal groundnut pathologist at ICRISAT. He worked on peanut diseases in Nigeria from 1957 to 1978, first with the Ministry of Agriculture of Northern Nigeria and later at Ahmadu Bello University, where he became head of the Department of Crop Protection. He joined ICRISAT in 1978. His interests cover all aspects of peanut diseases, with special emphasis on the aflatoxin problem and foliar diseases.