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TROPICAL PLANT DISEASES

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Preface

This book is not intended to be an encyclopedia of tropical plant diseases, but is rather intended to lead the reader to literature on identification and control of diseases of the major tropical crops. Emphasis is given to crops grown in the tropics below 1000 m altitude. Crops grown at higher altitudes in the tropics usually are not included. The book also presents treatments of a few representative tropical diseases which students in tropical countries can study rather than, or in addition to, temperate diseases. I have traveled extensively in the tropics of Latin America, Asia, and Africa during the last 30 years and have found that the books being used for both undergraduate and graduate courses in plant pathology in the tropics usually are from temperate countries. Students from tropical countries often learn more about stem rust of wheat and apple scab than they do about rice blast or Panama disease of bananas. This work is also intended to complement the many excellent textbooks of plant pathology available with in-depth discussions of selected tropical diseases.

Introductory chapters include the nature of the tropics, farming in the tropics, and the importance of tropical plant diseases. Specific crops and their important diseases are discussed next. There are probably twice as many crops grown in the tropics as in temperate areas, and my choices, treatment, and organization of tropical crops and specific diseases will not please all. Some important tropical crops such as forest crops and ornamentals are not included. Selected references follow the brief introduction to each specific crop or group of crops; thus the reader can find information on identification and control of pathogens of most tropical crops of importance. Many important references (ie. much of the literature in languages other than English) are undoubtedly missing, and this is regretted.

The specific diseases treated are intended to be useful in teaching to illustrate differences between tropical and temperate plant diseases. This is not to imply that there are principles or methods of plant pathology unique to the tropics. The same principles apply to control of tropical diseases as to control of temperate diseases, but the climates, soils, farming systems, social, political, and economic factors are often different. It is essential to have some understanding of these differences in order to be an effective plant pathologist in the tropics. The many agencies concerned with tropical plant diseases are also briefly discussed.

For the common and scientific names of tropical crops I have usually followed Purseglove (1968, 1972). For fungal names Holliday (1980) was followed with only a few exceptions. Bacterial nomenclature followed Dye et al. (1980) and Skerman et al. (1980). The original of nearly all the work cited has been seen.

Literature Cited

Dye, D. W., J. F. Bradbury, M. Goto, A. C. Hayward, R. A. Lelliot, and M. N. Schroth. 1980. International standards for naming pathovars of phytopathogenic bacteria and a list of pathovar names and pathotype strains. Rev. Plant Pathol. 59:153-168.

Holliday, P. 1980. Fungus Diseases of Tropical Crops. Cambridge Univ. Press, Cambridge. 607 pp.

Purseglove, J. W. 1968. Tropical Crops, Dicotyledons. Wiley, New York. 719 pp. 2 Vol.
Purseglove, J. W. 1972. Tropical Crops, Monocotyledons. Wiley, New York. 607 pp. 2 Vol.
Skerman, V. B. D., V. McGowan, and P. H. A. Sneath. eds. 1980. Approved lists of bacterial names. Int. J. Syst. Bacteriol. 30:225-420.

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The Tropics

The term "tropics" means many things to many people. Coconut palms gently swaying in the breeze on a graceful sweep of white sand beach overlooking a crystal clear coral lagoon is only one of many visions brought to mind by the word tropics. Geographically the tropics is the region between the Tropics of Cancer and Capricorn which are parallel to the equator at a latitude of 23°27'. Some would extend the tropics to include the area between 30°N and 30°S latitude. If we use this concept, the tropics would include sizeable portions of the continents of South America, Africa, Asia, and Australia. Most "developing" countries are within or on the border of the tropics. Uruguay, for example, is the only country of Latin America with its boundaries entirely within the temperate zone. In Africa, only Morocco and Tunisia are entirely temperate.

The tropics can also be defined in terms of mean annual temperature. For example, Gourou (1961) considers the tropics as areas which have no month with a mean temperature of less than 18°C, whereas Bates (1952) uses 21° and Wellman (1962) 22°C. Mean temperature lines, called isotherms, if drawn on a map, generally follow the geographic lines limiting the tropics with only small variations. Vegetation can also be a useful basis for climate classification. For example, Wellman (1962) suggests that the tropics might be delimited as those areas where there is year round growth of such cold sensitive plants as palms.

I grew up in South Dakota, United States, in what geographers call a "temperate" climate. Temperatures in the summer would rise to 43°C and in the winter occasionally dropped below -40°C. In the tropics such extremes do not occur. Bates (1952) suggests that the temperate zones should really be called the "intemperate zones" when comparing them to the tropics that are characterized by a relative lack of seasonality. Annual cycles in air and soil temperatures, in length of day, and in solar radiation are all small in comparison with those in temperate zones. Although in the temperate zone extremes in temperature are experienced seasonally, it is common in the tropics for extremes to be diurnal. There are considerable seasonal changes in rainfall however. Many patterns of rainfall distribution occur, such as even distribution throughout the year, a dry and a rainy season, or two wet and two dry periods. On the equator mean temperatures and day length are almost constant, but variations increase towards higher latitudes.

Altitude also affects climate in the tropics. According to Wellman (1962), mean temperatures are approximately 1°F (0.56°C) cooler for every 325 ft. (99 m) climbed, and this is equivalent to traveling about 100 miles (160 km) towards the north in a temperate country. Large areas in the tropics are mountainous, and where this is the case in Latin America most of the people live in the higher elevations. Many systems have been devised for classifying tropical regions based on altitude and climate. One of the simplest and most convenient methods for illustrating the relationship of elevation on temperature is to divide the environment into three temperature zones at the equator: hot zone - sea level to 1000 m; temperate zone - 1000-2000 m; cool zone - 2000 + m. Of course the change in elevation is gradual and the temperature of these zones will vary with latitude, prevailing wind patterns, and precipitation.

Colombia, South America, is an excellent example for illustrating the range of climatic variability occurring in the tropics. Elevations range from sea level to over 5000 m. Some of the wettest sites on the earth are found here separated only by relatively short distances from hot deserts. Snow-

capped peaks are visible from sea-level banana plantations where annual mean temperatures are 30°C. Crop production in Colombia follows the climatic zones quite well. In the hot zone (0-1000 m) the major crops produced are cacao, sugar cane, coconuts, bananas, plantains, rice, tobacco, cassava, and most of the nation's beef cattle. In the temperate zone (1000-2000 m) coffee, tea, maize, vegetables, and a variety of fruit are produced. From 2000-3000 m in the cool zone production is limited to crops commonly grown in temperate countries such as wheat, barley, potatoes, cold-climate vegetables, and dairy cattle, whereas above 3000 m alpine meadows with native or naturalized vegetation and limited potato production are found.

Rainfall is the most variable and important element of tropical climates and may vary from almost zero (e.g. the coast of Peru) to over 1000 cm of rain per year near Buenaventura, Colombia. Patterns of rainfall distribution are highly variable in the tropics, and no attempt to discuss them will be made here. Comprehensive information on tropical climates and rainfall patterns is given by Williams and Joseph (1970), Strahler (1975), Nieuwolt (1977), Webster and Nelson (1980), and Wrigley (1981). The amount of rainfall and its seasonal distribution strongly influences the type of vegetation and the agricultural potential of tropical regions within the broad temperature zones. For example, forests, grasslands, deserts, mountains, and maritime areas can all be found within a single country such as Colombia. Each zone has its own unique complex of tropical vegetation. The complexity of tropical vegetation is illustrated by the following quote from Stevens (1932):

> "The vast numbers of species in such regions (the tropics) may be imagined when one realizes that Mt. Maquiling near Manila, a mountain some 3,700 feet high and about five miles in basal diameter, is said by Dr. Merill to bear about twice as many species of woody plants as the whole United States".

Table 1 gives one of many classifications of the major climatic regions of the warm tropics and the land areas in these regions.

Soils in the tropics are poorly understood, and many misconceptions about them are found in the literature. Although vast areas are covered by highly weathered, old soils, it is safe to say that soils in the tropics are at least as diverse as those in temperate zones. Sanchez and Buol (1975) suggest that, although the basic concepts regarding temperate soils are applicable in the tropics, management of tropical soils involves different strategies than those used in temperate zones. Sanchez (1977) gives an excellent summary of current knowledge and issues regarding tropical soils.

Table 1. Land areas of major climatic regions of the tropics.[a]

Tropical climatic regions	Asia and Pacific Islands	Africa	Australia and New Zealand	South America	North America	Total
	Millions of hectares					
Rainy climates (9 1/2 to 12 months humid)	344	194	-	594	44	1,176
Humid seasonal climates (7 to 9 1/2 months humid)	271	493	9	569	51	1,393
Wet-dry climates (4 1/2 to 7 months humid)	146	637	52	97	75	1,007
Dry climates (2 to 4 1/2 months humid)	86	481	112	68	15	762
Semidesert and desert climates (less than 2 months humid)	130	301	97	18	7	553
Total	997	2,106	270	1,346	192	4,893

[a] From Chapter 8, Vol. II of the President's Science Advisory Committee Report on the World Food Problem. May, 1967. Climatic regions adapted from H. E. Landsberg, H. Lippman, K. H. Patten, and C. Troll. 1963. Die Jahreszeitenklimate der Erde. Heidelberg Akademie der Wissenschaften. E. Rodenwaldt and H. J. Jusatz. eds. Springer Verlag, Berlin.

Literature Cited

Bates, M. 1952. Where Winter Never Comes. Scribner, New York. 310 pp.

Gourou, P. 1961. The Tropical World. Wiley, New York. 159 pp.

Nieuwolt, S. 1977. Tropical Climatology. Wiley, New York. 207 pp.

Sanchez, P. A. 1977. Properties and Management of Soils in the Tropics. Wiley, New York. 618 pp.

Sanchez, P. A. and S. W. Buol. 1975. Soils of the tropics and the world food crisis. Science 188:598-603.

Stevens, F. L. 1932. Tropical plant pathology and mycology. Bull. Torrey Bot. Club 59:1-6.

Strahler, A. N. 1975. Physical Geography. Wiley, New York. 643 pp.

Webster, C. C. and P. N. Nelson. 1980. Agriculture in the Tropics. Longman, London. 640 pp.

Wellman, F. L. 1962. A few introductory features of tropical plant pathology. Phytopathology 52:928-930.

Williams, C. N. and K. T. Joseph. 1970. Climate, Soil and Crop Production in the Humid Tropics. Oxford Univ. Press, London. 177 pp.
Wrigley, G. 1981. Tropical Agriculture. The Development of Production. Longman, London. 496 pp.

Farming in the Tropics

Agricultural Systems

It is important for plant pathologists working in tropical developing countries to be well trained in their own discipline, but in addition they should have an understanding of the agricultural environment in which they are working. Often they will be working in an interdisciplinary team, and thus thorough knowledge of local agricultural systems becomes important. Many systems are similar to those found in temperate regions, but others are quite different. A quotation from G. F. Warren (1913) gives some idea of the complexity of the factors which determine the type of farming in an area. It would be difficult to improve on the statement today.

> "The chief factors that determine the type of farming in any region are: climate, soil, topography, transportation, distance to market or shipping point, market demand and supply, relation of the type to other competing types in the region, price of land, capital, labor supply, custom, insects, diseases or other pests, and personal desires of the farmer".

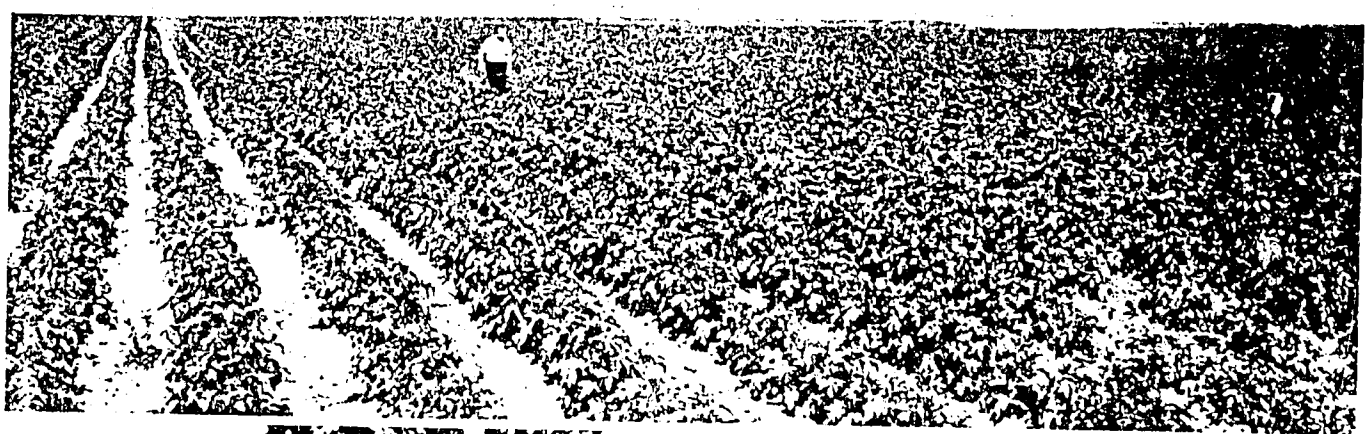


Fig. 1. Over 3000 acres of potatoes grown in a single field with modern farming techniques are seen in Idaho (above). The traditional techniques used in the harsh environment of the mountains of Peru (below) provide a striking contrast.

Much of the following information on farming systems is reproduced, with permission, from Glass and Thurston (1978), BioScience, Volume 28, copyright by the American Institute of Biological Science.

Many of the agricultural systems used in the tropics are "traditional" rather than "modern". There appears to be no satisfactory definition which separates what is commonly termed "traditional" from "modern" agriculture. Generally, but not exclusively, traditional cropping is characterized by small farms, polyculture, heterogeneous germplasm, little or no artificial fertilizers or other agriculture chemicals, minimum tillage, and often varying time periods of fallow. Modern agriculture usually involves intensive land use, monocultures, extensive use of fertilizers, pesticides, and other agricultural chemicals, uniform crop germplasm, and appropriate and timely tillage. Traditional agriculture is still practiced by a substantial proportion of the world's farmers. It has been estimated that one half of the world's population depends on a subsistence (mainly traditional) type of agriculture and that 40% of the land area of the world under cultivation is in the hands of the subsistence farmer (Wellhausen 1970). Most traditional methods of crop protection were developed empirically through centuries of trial-and-error, natural selection, and keen observation. The knowledge of traditional farmers is passed from generation to generation and is often wide and comprehensive as illustrated by Conklin's (1954) description of the agriculture of a tribe in the Philippines:

Fig. 2. Maize and climbing beans in Guatemala.

> "The knowledge displayed by shifting cultivators of soils, food plants, and cropping techniques is amazingly wide, accurate, and practical. The Hanunoo, a mountain people of Mindoro in the Philippines, know 10 basic and 30 derivative soil and mineral categories. They also understand the suitability of each for various crops as well as the effects of erosion, exposure, and over-farming. Their repertoire of 1500 useful plant types includes 430 cultigens, and they distinguish minute differences in vegetative structures".

Another insight into the vast amount of information sometimes possessed by traditional farmers is found in the description by Berlin et al. (1974) of Mayan (Tzeltal) plant classification. To quote:

Fig. 3. Mixed cropping in the highlands of Ecuador (courtesy of Roger Kirkby).

"At this time, a total of 471 mutually exclusive generic taxa have been established as legitimate Tzeltal plant groupings".

Archaeologists believe that man began crop production 8,000 to 10,000 years ago with maize, beans, and cucurbits in the Americas and with wheat, barley, lentils, and peas in the Near East (Harlan 1976). There is less certainty of the first cultivation of rice and millet in Asia, but certainly these crops have been grown for several millenia. Evidence for the origins of agriculture in the tropics is very fragmentary, but Harris (1972) presents arguments that vegiculture began in the tropics as early as or perhaps earlier than seed culture. An extensive literature on tropical agricultural systems is available: Ruthenberg (1971), Harwood (1979), McDowell and Hildebrand (1980), Shaner et al. (1982), and Beets (1982).

The term "farming systems" should be differentiated from cropping and animal systems, although the distinctions are not always sharp or universally accepted. The farming system includes the whole farm. It includes the farm household and its activities both on and off the farm, as well as the cropping and animal systems and the interactions between and within these as the farmer engages in practicing them, and his interactions with other farmers, the market, and the region in which he lives.

Many different agricultural or farming systems are found in the tropics, and innumerable variations are found of some systems. For example, although the cropping system including maize, beans, and squash was basic in the highlands of Ecuador, Kirkby et al. (1980) found over 100 distinct crop associations within a 30,000 hectare area near Riobamba. Other general systems are slash and burn, paddy rice culture, dry farming, rainfed farming, and agrisilviculture. A description of two common cropping systems in the tropics follow as illustrations.

Slash and Burn

Slash and burn agriculture is also known as shifting cultivation or swidden agriculture and is most commonly practiced today in tropical areas (Nye and Greenland 1960, Conklin 1961). In spite of pleatiful rainfall and high solar energy the lowland humid tropics are not highly productive, due in large measure to the difficulty in maintaining soil fertility and controlling weeds. Over centuries, however, traditional agriculturists evolved slash and burn

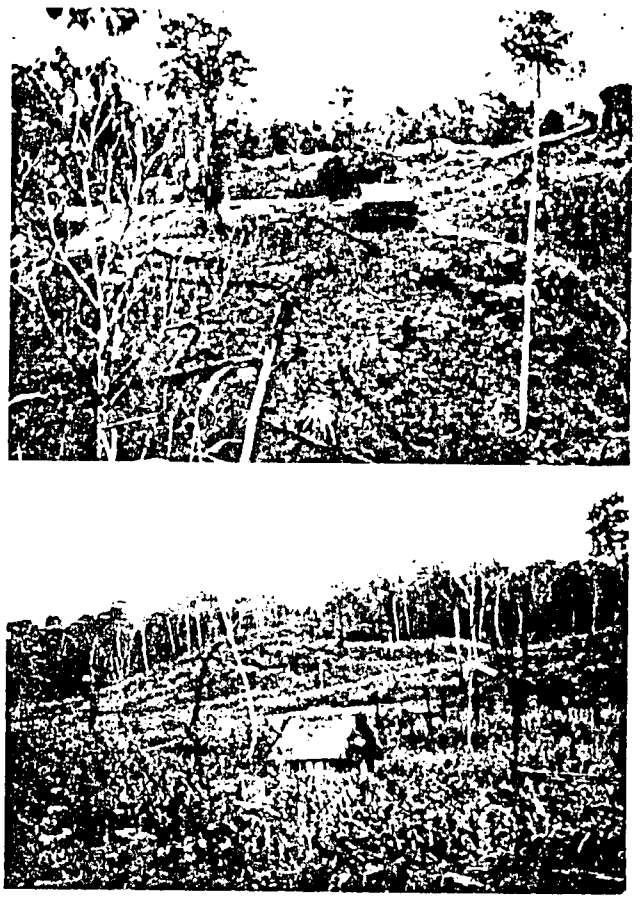


Fig. 4. Clearing the forest for agriculture in Mindanao, the Philippines (above). Land clearing by burning in Mexico (below) (Courtesy of Peter T. Ewell).

agriculture as a solution to the soil problem and a method for managing pests. Unfortunately it requires as much as 15 hectares to feed one person. According to Hauck (1974) slash and burn agriculture is the predominant method on 30% of the world's exploitable soils and supports over 250 million people.

The system usually consists of small plots which are partially cleared from the thick jungle growth. The vegetation is burned and crops are planted, generally without removing the stumps and roots. The plots are cropped for one to three years and then abandoned to native vegetation for a fallow period of up to 20 years. Slash and burn agriculture not only conserves moisture, restores organic matter and nutrients to the soil, and prevents erosion and leaching, but also controls weeds and reduces insects, nematodes, and various pathogen populations. For example, in Nigeria, nematologists of the International Institute of Tropical Agriculture have found that burning a 10 cm litter layer destroyed nematodes to a soil depth of 9 cm (IITA - 1976).

Plots under shifting cultivation mimic tropical forest ecosystems in at least two ways that influence pest problems. The great diversity of crops grown, sometimes as many as 40 growing simultaneously, provides a degree of protection because pests seldom are able to build up to destructive proportions on the isolated few plants of each species. Also the shade of some trees left standing and tall crop species such as bananas and papayas reduces the severity of weed and plant pathogen problems. However, weeds and other pest problems eventually make the cleared plots uneconomical. For example, farmers in Nigeria may spend 50% of their time in weed control (Moody 1975).

Thus burning, rotation, polycropping (plant diversity), and shading are all practices which may reduce pest losses under shifting cultivation. Furthermore, the clearing of small plots permits easy immigration of biological control agents from the surrounding natural vegetation. The slash and burn farmers also practice the selection of host resistance simply by using seed and vegetative parts from the most successful crop plants which have survived the harsh environment.

Paddy Rice Culture

This interesting agricultural system has probably existed in Asia for several thousand years. The first operation after flooding by natural rains or

irrigation is to plow under weeds, stubble, rice straw, or a green-manure crop. The wet paddies are harrowed several times at 7-10 day intervals to destroy any sprouting weeds. When properly prepared for transplanting, paddy land is well puddled and leveled, and the weeds, rice straw, and stubble are allowed to decay thoroughly Most of the rice grown by traditional farmers in paddy culture is transplanted from small seed beds planted in advance. After transplanting, rice paddies are continuously flooded to a depth of about 5 cm until shortly before harvest. On rain-fed farms, however, flooding may be intermittent, and on irrigated farms water control may be inadequate.

The weed-control components of the paddy system (plowing, harrowing, transplanting, and flooding) are obvious, but their true value may not have been recognized until recently when work was begun to develop multiple cropping systems for Southeast Asia. Rice can be grown very productively by direct seeding into unpuddled soil with flooding delayed for four weeks, but only when adequate weed control is attained either by hand or chemical weeding. Uncontrolled, weeds reduce production up to 80% (De Datta and Bernasor 1973). Hand weeding, the only method available to traditional farmers, is extremely laborious and impractical in direct-seeded rice. Thus the paddy system of submerging weeds into puddled soil, transplanting three-week-old seedlings which can compete successfully with later-germinating weeds, and flooding is a very effective weed management system for the tropics. This very laborious system of rice culture probably evolved because it was the most practical system to manage weeds.

The impact of the rice-paddy system on other pests is not as evident but has been documented to some extent. Flooding reduces the number of fungal propagules, insects, and nematodes in the soil and controls weeds which harbor rice pathogens and insects, thus reducing disease and insect damage. Rice blast (caused by Pyricularia oryzae), perhaps the most important disease of rice in the world, is much less severe on flooded paddy rice than on upland rice. Fewer hours of dew occur in paddy than upland rice, and thus P. oryzae, which requires free moisture for penetration, is less serious due to shorter infection periods.

Fig. 5. Puddling a rice paddy in preparation for planting (left). Farmers weeding by hand in a rice paddy (right).

Crop Plants

If the tropics are considered as the area between the tropics of Cancer and Capricorn, perhaps twice as many crops are grown in the tropics as in temperate zones. Almost all temperate crops can be grown in the high elevations of the tropics, but many crops can be grown only in the tropics. No listing or description will be made here, as the major tropical crops are discussed in detail elsewhere in the book. Chan (1983), Cobley and Steele (1976), Leon (1968), Ochse et al. (1961), Purseglove (1968 and 1972), and Williams (1975) give detailed descriptions of most tropical crops.

George Harrar (1961) has stated:

> "rice, wheat, maize, sorghum, the millets, rye, and barley are principal foods in cereal-producing areas, but elsewhere cassava, the sweet potato, potatoes, coconuts, and bananas are basic foods. Although over 3000 plant species have been used for food and over 300 are widely grown, only about 12 furnish nearly 90% of the world's food".

Mangelsdorf (1966) further states:

> "During his history, man has used at least 3000 species of plants for food and has cultivated at least 150 of these to the extent that they have entered into the world's commerce. The tendency through the centuries has been to use fewer and fewer species and to concentrate on the more efficient ones, those that give man the greatest return for his land and labor. Today the world's people are actually fed by about 15 species of plants. These include five cereals: rice, wheat, corn, sorghum, and barley; two sugar plants: sugar cane and sugar beet; three "root" crops: potato, sweet potato, and cassava; three legumes: the common bean, soybean, and peanut; and two so-called tree crops: the coconut and banana".

Many more of these important crop plants are grown in the tropics than in the temperate zones.

Agricultural research in tropical areas in the past was primarily on cash and plantation crops, whereas food crops -- the plants that people eat, especially in the hot, humid tropics -- were largely ignored by research workers (Thurston 1969). One group of plants ignored by researchers until recently was the starchy crops such as cassava, sweet potatoes, yams, and plantains that hardly enter into world commerce when compared to the cereals. Most can seldom be stored fresh for any appreciable time, but they are probably far more important as food in the tropics than available figures indicate.

Numerous books and references can be found on the cash and plantation crops of the tropics such as sugar cane, rubber, coffee, cacao, citrus, and bananas for fruit, but there still is a paucity of information on tropical food crops. Before World War II, most research done by colonial powers in the tropics was done on cash or plantation crops. Since World War II national research programs of many tropical nations have made important contributions towards research on tropical crops, and it is they who will ultimately have to solve their agricultural problems. In addition, other entities such as bilateral aid agencies, universities, private foundations, the UN-FAO (United Nations Food and Agricultural Organization), regional agencies such as the

Inter-American Institute of Agricultural Science of the OAS (Organization of American States), and the international research centers funded by the Consultative Group in International Agricultural Research have entered the field of research in agriculture in the tropics. Outstanding contributions which have had significant impact on alleviating food shortages have been made in research on rice, maize, wheat, and potatoes. Even with this effort, sufficient emphasis has not been focused on tropical food crops, and opportunities for research on other tropical food crops are only now beginning.

People and Governments

Although this book is about tropical plant diseases, some discussion should be made of people and governments in the tropics. These comments are aimed primarily at pathologists from temperate areas, but pathologists from the tropics often work outside of their home country and thus may also find this discussion pertinent. Whether you are working in your native country in the tropics, as a foreign "expert" to a tropical country, or never plan to leave your home in the temperate zone, some understanding of peoples and governments in the tropics is important. It is dangerous to generalize; however, most tropical countries are characterized by large numbers of the poor and very poor, usually with a small percentage (usually the highly educated and ruling class) of rich. Seldom is there a significant "middle class", but this is growing, and growing rapidly in many countries. Customs and traditions are often strange and different to people from temperate zones. It is not only important, but essential, to learn as much as possible about the culture, customs, traditions, history, and sociology of tropical peoples and countries in order to at least partially understand them. You simply can not operate effectively with the habits and customs of the temperate zone in most of the tropics. Mastering a new language and learning something of the habits, customs, traditions, history, and culture of tropical countries can be a source of personal satisfaction and edification. You can often find superior or equally efficient cultural habits and methods of doing a job and getting along with fellow workers. Frequently customs that seem to impede progress and "getting the job done" have valid reasons for existing with centuries of tradition behind them. If such customs need to be changed, you must at least understand their reason for existence before you can hope to change them.

The ability to meet your hosts in tropical countries and treat them as equals and coworkers often is more important than your scientific knowledge. Jose Nolla (1962) has pointed out that even the poorest farmer and laborer often has great pride and human dignity. The least suggestion of inferiority will be resented and may ruin all your future work. On the other hand, when treated as equals, tropical people can become the warmest of friends and loyal supporters. We of the frozen north can learn much from tropical peoples about how to live and enjoy life without losing our reputation for "getting the job done".

It is also important to study the governments of tropical countries. This is not only minimum courtesy to your hosts, but is also essential to accomplishing one's responsibilities. Often governments are the only agencies which can or should provide the funds and stimuli to accomplish the many tasks in agriculture. You should try to learn as much as possible about different political parties, systems of government, governmental agencies, and politics in general. It is often part of your job to justify your research program to governmental officials and unless you know them, their background, and the forces that make them operate the way they do your chances of increasing your

Fig. 6. San Blas Indian woman in Panama (top left). Young girl helping her father harrow in the Philippines (top right). Mexican mother and child (bottom left). Farmer and his children in Boyaca, Colombia (bottom right).

support, or even continuing it, may be diminished considerably. I am not suggesting that you (especially if you are a visitor) should enter or take sides in politics, but you must have some understanding of politics and be something of a "politician" yourself if you are to get support for your work.

Literature Cited

Beets, W. C. 1982. Multiple Cropping and Tropical Farming Systems. Westview Press, Boulder, CO. 156 pp.

Berlin, B., D. E. Breedlove, and P. H. Raven. 1974. Principles of Tzeltal Plant Classification. An Introduction to the Botanical Ethnography of a Mayan-Speaking People of Highland Chiapas. Academic Press, New York. 660 pp.

Chan, H. T. ed. 1983. Handbook of Tropical Foods. Marcel Dekker, New York, NY. 639 pp.

Cobley, L. S., and W. M. Steele. 1976. An Introduction to the Botany of Tropical Crops. Longman, London. 371 pp.

Conklin, H. C. 1954. Shifting cultivation. Ann. N.Y. Acad. Sci. 17:133-142.

Conklin, H. C. 1961. The study of shifting cultivation. Curr. Anthropol. 2(1):27-61.

De Datta, S. K., and P. C. Bernasor. 1973. Chemical weed control in broadcast-seeded flooded tropical rice. Weed Res. 13:351-354.

Glass, E. H., and H. D. Thurston. 1978. Traditional and modern crop protection in perspective. Bio Science 28:109-115.

Harlan, J. R. 1976. The plants and animals that nourish man. Sci. Am. 235(3):89-97.

Harrar, J. G. 1961. Socio-economic factors that limit needed food production and consumption. Fed. Proc. 20:381-383.

Harris, D. R. 1972. The origins of agriculture in the tropics. Am. Sci. 60:180-193.

Harwood, R. R. 1979. Small Farm Development; Understanding and Improving Farming Systems in the Humid Tropics. Westview Press, Boulder, CO. 160 pp.

Hauck, F. W. 1974. Shifting cultivation and soil conservation in Africa. FAO Soils Bull. 24:1-4.

International Institute of Tropical Agriculture. 1976. Annual Report 1975. Ibadan, Nigeria.

Kirkby, R., P. Gallegos, and T. Cornick. 1980. Metodologia para el desarrollo de technologia agricola apropiada para pequenos productores. Experiencias del Proyecto Quimiag-Penipe, Ecuador. Ministerio de Agricultura y Ganaderia, Ecuador. 38 pp.

Leon, J. 1968. Fundamentos Botanicos de los Cultivos Tropicales. Instit. Interamericano Ciencias Agricolas, Organ. Estados Am., San Jose, Costa Rica. 487 pp.

Manglesdorf, P. C. 1966. Genetic potentials for increasing yields of food crops and animals. pp. 66-71. In Prospects of the World Food Supply. Symp. Proc. Natl. Acad. Sci., Washington, D.C.

McDowell, R. E., and P. E. Hildebrand. 1980. Integrated Crop and Animal Production: Making the Most of Resources Available to Small Farms in Developing Countries. Rockefeller Found., New York. 78 pp.

Moody, K. 1975. Weeds and shifting cultivation. Pest Articles News Sum. 21:188-194.

Nolla, J. A. B. 1962. The necessarily broad view in studying tropical plant diseases. Phytopathology 52:946-957.

Nye, P. H., and D. J. Greenland. 1960. The soil under shifting cultivation. Tech. Commun. No. 51. Commonw. Bur. Soils, Harpenden. Commonw. Agric. Bur., Farnham Royal.

Ochse, J. J., M. J. Soule, Jr., M. J. Dijkman, and C. Wehlburg. 1961. Tropical and Subtropical Agriculture. MacMillian, New York. 1446 pp. 2 Vol.

Purseglove, J. W. 1968. Tropical Crops, Dicotyledons. Wiley, New York. 719 pp. 2 Vol.

Purseglove, J. W. 1972. Tropical Crops, Monocotyledons. Wiley, New York. 607 pp. 2 Vol.

Ruthenberg, H. 1971. Farming Systems in the Tropics. Clarendon Press, Oxford. 313 pp.

Shaner, W. W., P. F. Philipp, and W. R. Schmehl. 1982. Farming Systems Research and Development. Guidelines for Developing Countries. Westview Press, Boulder, CO. 414 pp.

Thurston, H. D. 1969. Tropical agriculture, a key to the world food crisis. BioScience 19:29-34.

Warren, G. F. 1913. Farm Management. Macmillan, New York. 590 pp.

Wellhausen, E. J. 1970. The urgency of accelerating production on small farms. pp. 5-9. In Myren, D. T. ed. Strategies for Increasing Agricultural Production on Small Holdings. Centro Int. Mejoramiento Maiz y Trigo, Mexico.

Williams, C. N. 1975. The Agronomy of the Major Tropical Crops. Oxford Univ. Press, Oxford. 228 pp.

The Importance of Tropical Plant Diseases

Tropical plant pathology has made great contributions to our discipline (Grant 1962, Sequeira 1962, Calpouzos 1962). For example H. Marshall Ward, a famous British plant pathologist, began studying coffee rust, caused by Hemileia vastatrix, in 1881 in Ceylon (now Sri Lanka). He found no economic control for the disease, and coffee growing was abandoned for tea and rubber. Nevertheless, the work he did on coffee rust led him to be the first to state the principle of protection (Large 1962), one of the most widely used principles of plant disease control. He stated that a fungicide had to kill a fungus before penetration, or it would not be effective. Ward's (1882) report of his research on coffee rust still makes fascinating reading today.

Plant disease organisms have often caused losses in the tropics which might be termed catastrophic. For example, H. vastatrix was introduced into Sri Lanka about 1869 and by 1890 had caused the virtual abandonment of coffee as a crop. At this time coffee was the major export of Sri Lanka. In 1970 Hemileia vastatrix was found on coffee near Bahia, Brazil. A few years after its discovery, the organism had spread into all the major coffee growing areas of Brazil. Wellman (1970) estimated that the disease may have been present in Brazil as long as five years before discovery. Much speculation has occurred on how the organism got into Brazil, but the true story will probably never be known. The organism is now found in all major coffee growing areas of Central and South America. Fungicides are needed for rust control at a great cost in time, energy, and money.

As recently as 1942 an epidemic due to the fungal pathogen Helminthosporium oryzae (brown leaf spot) was a major cause of the failure of the rice crop in West Bengal, India, and an estimated 2 million people died (Padmanabhan 1973). Many other examples could be cited, but fortunately the great majority of tropical plant diseases cannot be called catastrophic.

Sugar cane mosaic disease is probably the best known and most widely distributed disease of sugarcane. It has caused crop failures and has been highly destructive in the past. Mosaic was first found in Indonesia in 1892, but for many years no spread to other countries was reported. It was not initially recognized as a virus and was carried from Indonesia to many other sugar cane areas before its seriousness was recognized. After 1910 many reports of serious losses were made

Fig. 7. Coffee rust lesions (Hemileia vastatrix).

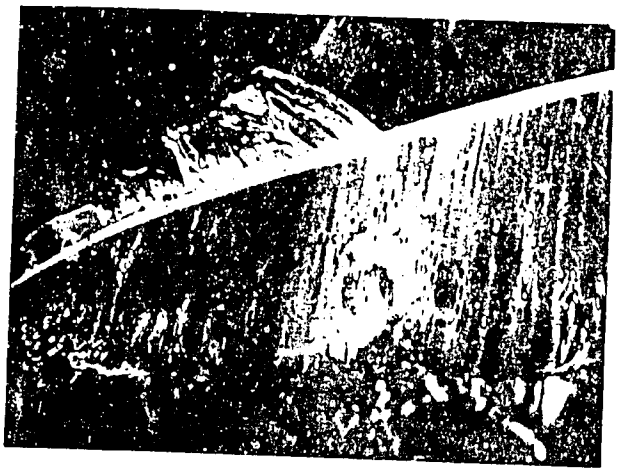


Fig. 8. Lesions on banana leaf caused by Mycosphaerella musicola (Sigatoka disease).

from the tropics. About 1916 an epidemic occurred in Puerto Rico and the industry was almost destroyed. The disease probably had a greater influence on the world sugar industry than any other disease. Losses from 30 to 80% were reported, but Edgerton (1958) doubts that all losses were due to the virus. In controlled tests he got yield losses of 8-15% with the most prevalent strains of the virus and losses of 30-40% with the severe strain. Fortunately cultivars with good resistance were found. Their introduction caused a shift in sugar cane cultivars, and control now is based almost entirely on resistant cultivars.

Another example of an extremely destructive plant disease in the tropics is fusarial wilt (Panama disease) of bananas caused by the fungus Fusarium oxysporum f. sp. cubense. Bananas and fusarial wilt became important in tropical America at the end of the nineteenth century when banana plantings were rapidly expanded. The organism caused huge financial losses and the abandonment of thousands of acres of banana land. The Gros Michel cultivar was the basis of the industry because of its superior shipping and ripening quality, but is highly susceptible to wilt. There has recently been a shift to the resistant Cavendish types such as Valery. The disease had a devastating impact on many banana producing countries. For example, bananas were first shipped from Costa Rica in 1878, and by 1913 eleven million bunches were exported. The diseases caused a rapid decline in production, and by 1931 commercial production almost ceased (Jones and Morrison 1952). Banana land in Central America represented an investment of US $1,000 to US $2,000 per acre; thus losses of abandoned land represented millions of dollars. In addition, with the introduction of banana production to some areas, the standard of living of most of the population rose from a subsistence level to one that included the educational and health-care benefits of an active economy and supported an educated middle class. Governments participated in the profits through taxes, revenues, and other benefits with increasing banana production. As production fell due to the disease, unemployment increased and national revenues decreased, contributing to an increase in political instability.

Prior to 1933 the Sigatoka disease of bananas, caused by the fungus Mycosphaerella musicola, was only found in Asia, Australia, and the Pacific Islands. After its introduction into the Americas a rapid spread with disastrous epidemics occurred beginning in 1934 (Stover 1962, Meredith 1970) threatening the existence of the banana industry in many countries. The disease is now present in all major banana growing areas of the world. Control of M. musicola with fungicides and, later, oils (Calpouzos 1966, Meredith 1970) enabled the banana industry to survive. In 1936, and for some years thereafter, the control program of the United Fruit Company was the largest private fungicide spray program in the world (Sequeira 1962).

Cacao is susceptible to many destructive pathogens. Colombia is one of the centers of origin of cacao and once was a major exporter. However, with the arrival of watery pod rot (Monilia roreri), it plus wilt (Ceratocystis fimbriata) and witches broom (Crinipellis perniciosa) caused nationwide losses from disease to increase to over 40%. Colombia now imports cacao.

Rice probably feeds more people than any other food crop in the world. Cramer (1967) estimated an overall loss of 8.9% due to rice diseases. Rice has many diseases and pests, but perhaps the most serious disease is rice blast (Pyricularia oryzae). The Japanese collect detailed data on losses due to this disease (Goto 1965), and over an eight-year period estimated average annual losses at almost 3%. There are no sound data with which to estimate losses in the tropics, although losses to blast are probably greater in tropical regions. Considering that Japan is in the temperate zone, if we estimate annual losses of rice production due to blast to be 5% of global production, this would constitute a loss of 20 million tons of paddy rice valued at over four billion U.S. dollars.

African cassava mosaic disease is probably the most destructive disease of cassava, the major food of 300-500 million people in the tropics. The viral causal agent is transmitted by Bemisia tabaci, a white fly. Much of the cassava in Africa shows disease symptoms, and Padwick (1956) estimated an 11% loss in yield due to the disease in Africa. Yield losses of 40% are common in some areas of Africa.

Losses Due to Diseases in the Tropics

The United States is one of the more highly developed countries in the world in agriculture. In the 1953 U.S. Department of Agriculture Yearbook, Wood (1953), who was associated for many years with the Plant Disease Survey organization of the U.S.D.A., wrote an article on losses due to plant disease. She estimated an annual average crop loss of about 3 billion dollars and that crop production in the United States could be increased 10 percent if plant disease could be prevented. By 1965 the estimate had increased to 4 billion dollars (U.S. Department of Agriculture). Those that have the courage to make estimates of global losses seldom break the figures down as to the type of pest. In 1975 Ennis et al. estimated a "30% annual loss in the potential worldwide production of crops, livestock and forests" due to pests. Pimentel and Perkins (1980) estimated total world food losses at about 45%. FAO officials have estimated that 5% of all food grains are lost before consumption, but that in areas such as India, South America, and Africa there is a 30% loss of food grains before harvest. The Commonwealth Agricultural Bureaux in their thirty-ninth annual report of 1968 estimated that losses from disease alone in the tropics are of the order of 10 to 13%. Padwick (1956) estimated that the percentage loss for all crops in the British colonies due to plant disease was 11.8 percent. Figures are not readily available for tropical countries, but losses might well be double those of temperate areas.

Cramer (1967) compiled the available information on crop losses throughout the world. His estimates of crop loss due to disease are as follows:

Primarily Temperate Crops	
Crop	% Loss
Wheat	9.1
Oats	9.3
Barley	7.8
Rye	3.2
Hops	8.0
Linseed	7.8
Sugar beets	10.4

Primarily Tropical Crops	
Crop	% Loss
Sugarcane	19.2
Coffee	16.6
Cocoa	20.8
Tea	15.4
Palm kernels	7.4
Copra	19.3
Natural Rubber	15.0

Cramer's data indicate that those crops grown in the tropics have the largest losses.

Paddock (1967) has pointed out that each agricultural scientist tends to give his specialty top priority. He gives figures from the U.S. Department of Agriculture to support his contention as follows:

Table 1. Average annual losses from 1951 to 1960.[a]

Crop	Disease %	Nematodes %	Insects %	Weeds %	Harvesting & storing %	Total %
Beans	17	5	20	15	?	57
Corn	12	3	12	10	8	45
Cotton	12	2	19	8	5	46
Soybeans	14	2	3	17	8	44
Wheat	14	-	6	12	5	37
Rice	7	-	4	17	5	33

[a] Adapted from Paddock (1967).

When the figures for only one type of loss are studied, they may seem reasonable, but when they are added, they look at best well padded. This situation is probably as bad or worse in the tropics. Development administrators, the public, and politicians simply do not believe the figures. Thus, the importance of serious, well planned studies of the losses caused by plant diseases in the tropics becomes obvious. If support is to be forthcoming for important problems, real proof of their importance must be made available. Main (1983) discusses the nature of crop losses and methods used in crop loss estimation.

Some of the reasons why diseases are often more serious and cause greater losses in the tropics are:

1. A lack of distinct seasons means that inoculum is present all year long for most air-borne pathogens. Organisms also pass through more generations in a year, and thus their capacity for change is greater. There are sometimes dry seasons, but often these do not stop plant growth.

2. There are relatively few countries in the tropics with seed-producing

agencies. Disease-resistant cultivars and pathogen-free seed often are not available.

3. Tropical regions often have higher rainfall and humidity than temperate zones; thus conditions are usually favorable for most plant diseases.

4. In the tropics two, three, and even four crops a year can be grown of annual crops, and with many perennial crops such as bananas or sugarcane cropping is almost continuous. These practices often permit a build-up of pathogens, especially soil-borne pathogens.

5. More pathogens occur on a given species in the tropics than in temperate zones.

Plants selected under traditional agriculture systems are able to survive and produce some yield under conditions of poor tillage, inadequate fertilization, and deficient irrigation. In addition, the wide variety of cultivars probably protects against pests. For example, before the introduction of high-yielding varieties (HYV's) from IRRI, hundreds of cultivars were grown in Indonesia, whereas now, only a few are grown. Traditional agriculture in large areas of the developing world is giving way to a modern agriculture characterized by many new inputs: high rates of fertilizer, monoculture or the widespread use of a narrow base of germplasm, improved tillage and irrigation practices, higher plant population densities, and increased pressure to produce two to three crops per year from the same land. It is paradoxical and unfortunate that any of these practices has the potential to increase disease problems. Saari and Wilcoxson (1974) attempted to analyze the impact of these practices on the performance of the HYV's of wheat in Asia and Africa and found only a limited number of examples where new inputs triggered serious losses due to diseases.

The new HYV's have not been without problems. Leaf blotch (*Septoria tritici*) of wheat was a serious problem in the Mediterranean basin following the introduction of wheat HYV's. Various rusts have also caused losses, but fortunately in the case of wheat new cultivars have rapidly been made available to reduce the losses caused by leaf blotch and the rust. The tungro epidemics of the HYV's of rice in Asia are another example (Denny 1972). The 1970 epiphytotic of southern corn leaf blight caused a loss of 10% of the USA hybrid corn crop.

The HYV's of wheat and rice involve a relatively small range of genotypes, most of which have many common genes such as those for dwarfing. New races of a pathogen or a previously unimportant disease or insect pest might have the potential in a given year, with optimal weather conditions, to cause widespread, serious losses. No one, least of all the breeders and plant protectionists of the international centers and developing countries where the high-yielding varieties are grown, would dispute this possibility. However, the breeders and plant pathologists of the international centers are cognizant of these dangers and have extensive activities to monitor changes in pest and pathogens to reduce the chances of potential disasters.

Threatening Tropical Diseases

Plant pathogens have clearly shown their ability to spread to new areas and threaten crops during the past few decades. The spread of coffee rust (*Hemileia vastatrix*) in the Americas and southern corn leaf blight (race T of

Fig. 9. Lesions caused by Helminthosporium maydis (Southern corn leaf blight) on maize.

Helminthosporium maydis), which in the 1970's caused a severe epiphytotic on maize in the United States, are outstanding examples. Information on internationally dangerous plant diseases already exists (Klinkowski 1970, O'Conner 1969, Riker 1964, Stakman and Harrar 1957, Watson 1971). Examples of threatening plant diseases are given in this book and also in the articles by Thurston (1973) and Holliday (1971).

Most of the following information on threatening diseases is reproduced, with permission, from Thurston (1973) Annual Review of Phytopathology, Volume 11, copyright by Annual Reviews Inc.

Probably twice the number of crop plants are grown in the tropics as are grown in temperate zones, and an awareness of potentially important diseases on these crops becomes increasingly difficult as information accumulates. Plant pathologists in tropical countries usually know the problems of their own country or continent well, but because of language barriers, inadequate library facilities, and lack of travel opportunities often are not cognizant of problems in other countries or continents. One tragic consequence of such lack of knowledge has frequently been the inadvertent introduction of a plant pathogen into a new area from which it easily could have been excluded. New pathogens have repeatedly spread into countries or continents which were completely unprepared for them as regards methods of eradication, resistant varieties, and other control methods. Knowledge of threatening plant diseases can give a country or region time to prepare for a new disease and thus minimize its effect on crop production. The threat of new diseases is real, and with rapid population growth, hunger, and shrinking agricultural land it is vital to expand our knowledge so as to prevent future catastrophes.

The components which make a plant disease "threatening" are numerous and diverse. To be highly threatening, a disease should be characterized by the ability to spread rapidly, cause serious losses, and be difficult to control. A thorough knowledge of the etiology and epidemiology of a pathogen is necessary for meaningful predictions on its potential for spread. Unfortunately, too often only bits and pieces of the information needed for evaluating a pathogen's potential for spread are available. Information on the ability of a pathogen to cause losses is even more difficult to find. Too often terms such as highly destructive, catastrophic, devastating, etc. are the only basis for making judgments. The potentials for disease control are usually better known, but are often controversial and backed by little field experience.

In this age of jet transportation and increasing ease and frequency of travel from continent to continent, no one would dispute the obvious fact that plant pathogens may also move intercontinentally with greater ease. Plant pathologists should stimulate greater awareness of tropical plant diseases which constitute a threat to other countries or continents if they move from their present distribution. Scientists need to remain alert for pathogens or

races of pathogens which may constitute a serious threat to crops in other areas of the world.

Sound information on which to base judgment on the potential of pathogens to cause serious losses is generally lacking. Without such information it is extremely difficult for pathologists and governments to determine which diseases are truly of potential importance. Much more research is needed to determine the ability of pathogens to spread. Serious studies of the etiology and epidemiology of many tropical pathogens are lacking. Phytopathological societies and international and national institutions need to encourage the coordinated study of these and similar pathogens on a worldwide basis. The development of gene pools for crop resistance (Leon 1974), increased exchange of information, and especially of personnel between different countries and continents is essential. Quarantines need to be strengthened on a worldwide basis. It is worth noting, however, that quarantines can stifle useful exchange of plant introductions unless knowledgeable people enforce them. A worldwide cooperative effort to monitor pathogens, perhaps including other pests, should be established.

The efforts of scientists and governments in Southeast Asia to prevent the introduction of South American leaf blight of rubber, and to be prepared for it if it does arrive, might be used as a model for dealing with a threatening plant disease

In 1963 I accompanied Dr. A. J. Riker of the University of Wisconsin, who was visiting various countries in South America, on visits to various national and international agencies in Colombia. He wanted to obtain support for a project on methods of eradicating coffee rust, and consequently he was seeking support for the project. The project, a modest US $190,000, was not funded. Had it been funded, it might have accomplished what S.E. Asian governments are now doing relative to South American leaf blight of rubber. Today many millions of dollars are being spent on coffee rust research and control by South American countries. This example illustrates that plant pathologists need sound information which will convince administrators and governments of the need to invest in research and education on threatening plant diseases before the diseases are introduced.

Post-Harvest Problems

The emphasis in this book is on pre-harvest diseases, but post-harvest diseases also take a tremendous toll in the tropics. Christensen (1979) states:

> "it has been estimated that in some of the tropical countries as much as 30% of the harvested food grains is lost in storage".

All of this loss is not due to fungi, but is also due to insects, mites and rodents. Some estimate that post-harvest losses of fruits and vegetables in the hot, humid tropics may exceed 50%, but there is little data to support this figure. Most of the agricultural reseach effort in tropical countries is towards increasing food production, but as Christensen (1979) notes, once food is produced, little effort is made to preserve it or to research the problems of post-harvest losses.

Storage fungi (primarily different species of Aspergillus) can grow in seeds at moisture contents in equilibrium with relative humidities of 68-90%. The causal agents, their products, and the conditions of temperature and moisture under which they can cause damage in storage are well known. It is tragic that the principles and practices of good storage (Christensen and

Kaufmann 1969, Christensen 1974) often are not applied in the tropics.

In addition to total spoilage, storage fungi can reduce seed germination and discolor seeds and can thus reduce quality and cause heating and mustiness which can make seed useless for food. Storage fungi also produce mycotoxins. Mycotoxins are chemical compounds produced by fungi which cause disease (mycotoxicoses) when consumed by man or animals. Aflatoxins (mycotoxins produced by Aspergillus spp.) are especially well known for their serious effects. Most of the knowledge about mycotoxins is from temperate agriculture, but they probably are far more important and damaging in the tropics than is realized.

Literature Cited

Calpouzos, L. 1962. Some contributions from the tropics to plant disease control methods. Phytopathology 52:942-945.

Calpouzos, L. 1966. Action of oil in the control of plant disease. Annu. Rev. Phytopath. 4:369-390.

Christensen, C. M. ed. 1974. Storage of Cereal Grains and Their Products. Am. Assoc. Cereal Chem., St. Paul. 549 pp.

Christensen, C. M. 1979. The effects of plant parasitic and other fungi on man. pp. 393-409. In Horsfall, J. G. and E. B. Cowling. eds. Plant Disease -- an Advanced Treatise. Vol. IV. How Pathogens Induce Disease. Academic Press, New York. 466 pp.

Christensen, C. M. and H. H. Kaufmann. 1969. Grain storage. The Role of Fungi in Quality Loss. Univ. of Minnesota Press, Minneapolis. 153 pp.

Commonw. Agric. Bur. 1968. pp. 26-32. 39th Annu. Rep. 1967-1968. London.

Cramer, H. H. 1967. Plant Protection and World Crop Production. Farbenfabriken Bayer A. G., Leverkusen. 524 pp.

Denney, E. W. 1972. Typhoons and tungro cause Philippines to import more rice. Foreign Agric. 10(5):8-12.

Edgerton, C. W. 1958. Sugarcane and its Diseases. Louisiana State Univ. Press, Baton Rouge. 301 pp.

Ennis, W. B. Jr., W. M. Dowler, and W. Klassen. 1975. Crop protection to increase food supplies. Science 188:593-598.

Goto, K. 1965. Estimating Losses from Rice Blast in Japan. pp. 195-202. In The rice blast disease. Int. Rice Res. Inst. John Hopkins Press, Baltimore. 507 pp.

Grant, T. J. 1962. The role of plant pathology in tropical plantation agriculture. Phytopathology 52:930-936.

Holliday, P. 1971. Some tropical plant pathogenic fungi of limited distribution. Rev. Plant Pathol. 50:337-348.

Jones, C. F. and P. C. Morrison. 1952. Evolution of the banana industry of Costa Rica. Econ. Geogr. 28:1-19.

Klinkowski, M. 1970. Catstrophic plant diseases. Annu. Rev. Phytopathol. 8:37-60.

Large, E. C. 1962. Advance of the Fungi. Dover Publ., New York. 488 pp.

Leon, J. (ed.). 1974. Handbook of Plant Introduction in Tropical Crops. Food Agric. Organ., Rome. 140 pp.

Main, C. E. 1983. Nature of crop losses: an overview. pp. 61-68. In Kommedahl, T. and P. H. Williams. eds. Challenging Problems in Plant Health. Am. Phytopathol. Soc., St. Paul, MN.

Meredith, D. S. 1970. Major banana diseases: past and present status. Rev. Plant Pathol. 59:539-554.

O'Conner, B. A. 1969. Exotic Plant Pests and Diseases. South Pacific Commission. Noumea, New Caldonia. ca. 400 pp.

Paddock, W. C. 1967. Phytopathology in a hungry world. Annu. Rev. Phytopathol. 5:375-390.

Padmanabhan, S. Y. 1973. The great Bengal famine. Annu. Rev. Phytopathol. 11:11-26.

Padwick, G. W. 1956. Losses caused by plant diseases in the colonies. Phytopathol. papers No. 1. Commonw. Mycol. Inst., Kew, Surrey. 60 pp.

Pimentel, D., and J. H. Perkins. eds. 1980. Pest Control: Cultural and Environmental Aspects. Am. Assoc. Adv. Sci., Washington, DC. 243 pp.

Riker, A. J. 1966. Internationally dangerous tree diseases in Latin America. J. Forest. 62:229-232.

Saari, E. E. and R. D. Wilcoxson. 1974. Plant disease situation of high-yielding dwarf wheats in Asia and Africa. Annu. Rev. Phytopathol. 12:49-68.

Sequeira, L. 1962. Special contributions from the tropics to phyto-pathological mycology. Phytopathology 52:936-942.

Stakman, E. C. and J. G. Harrar. 1957. Principles of Plant Pathology. Ronald Press, New York. 581 pp.

Stover, R. H. 1962. Intercontinental spread of banana leaf spot (Mycosphaerella musicola Leach). Trop. Agric. (Trinidad) 38:327-338.

Thurston, H. D. 1973. Threatening plant diseases. Annu. Rev. Phytopathol. 11:27-52.

U.S. Department of Agriculture. 1965. Losses in Agriculture. Agric. Handbook No. 291. Agric. Res. Serv., U.S. Dept. Agric., Washington, DC. 120 pp.

Ward, H. M. 1882. Researches on the life history of Hemileia vastatrix. J. Linn. Soc. 19:299-335.

Watson, A. J. 1971. Foreign bacterial and fungus diseases of food, forage and fiber crops: An annotated list. U.S. Dept. Agric. Handbook. 418 pp.

Wellman, F. L. 1970. The rust Hemileia vastatrix now firmly established on coffee in Brazil. Plant Dis. Rep. 54:539-541.

Wood, J. I. 1953. Three billion dollars a year, pp. 1-9 In Plant Diseases. U.S. Dept. Agric. Yearbook. Washington, DC.

RICE

Rice (Oryza sativa) is the most important staple food for more than half of the world's population and, of the cereals, was third in production after wheat and maize in 1981 (FAO 1981). About 90% of the world's rice is grown in subtropical and tropical Asia. World rice production (paddy) in 1981 was 413 million metric tons. Only a small percentage of this production enters international trade.

Although the origin of rice is not known with certainty, most authorities believe it originated in one or more areas of tropical Asia. Rice may have been grown there as early as 8000 B.C. Rice is primarily cultivated in the tropics and subtropics, but it is also important in temperate countries such as Italy, Japan, and Korea. The crop is most important in Asia, but its importance in the Americas and Africa is growing. During the thousands of years that man has cultivated rice geographical "races" have evolved. These are indica, grown in tropical areas, japonica, the short grain rice grown in all temperate areas, and javanica, restricted to parts of Indonesia and the Philippine rice terraces. Within these "races" there is tremendous diversity of cultivars. For example, Grist (1968) stated that there were thousands of cultivars grown in India, including about 600 improved cultivars. A small amount of swamp rice (O. glaberrima) is grown in Africa.

The rice plant is an erect grass that tillers readily and produces grain in a loose, drooping panicle. Plants may grow to a height of 150 cm, but improved, high yielding varieties are dwarf and grow to less than one meter. The time required after planting to obtain a crop depends on a wide variety of environmental and physiological factors. Photoperiod insensitive cultivars may yield in as little as 110 days, while photoperiod sensitive cultivars may take over eight months to produce grain, depending on when they are planted.

Rice grows under a wide range of environmental conditions in the tropics and is cultivated with a wide variety of cultural methods. Three major systems are used: upland rice, floating rice, and lowland or paddy rice. Upland (rainfed) rice is cultivated much like other small-grain cereals without flooding. Much of the rice in tropical America and Africa is upland, but it constitutes less than 10 percent of that grown in Asia. Floating rice is grown in Asia in areas subject to deep flooding as found in Thailand, Bangladesh, and Vietnam. The plant elongates rapidly in the rising water which may reach a depth of 5 meters. Harvesting is from boats or after flooding has subsided.

Fig. 10. Rice in the Philippines.

The most important system used for growing rice is the paddy or lowland rice culture system. Many variations of this system are found. The two major sub-systems are paddy rice with or without artificial water control. Generally, the first operation after flooding by natural rains or irrigation is to plow under weeds, stubble, and rice straw. The wet paddies are harrowed or puddled a few times to prepare a soft, level, and weed-free planting bed. Most of the rice grown in Asia in paddy culture is transplanted from small seed beds sown in advance. After transplanting, irrigated rice paddies are continuously flooded until shortly before harvest. On rain-fed farms, where irrigation water is provided solely by rain, flooding may be intermittent.

Weeds are a very important constraint to tropical agriculture. The paddy system of submerging weeds into puddled soil and transplanting seedlings gives them an early competitive advantage over later-germinating weeds. One of the most important advantages of flooding is that it is a very effective weed control system for the tropics.

The impact of the rice-paddy system on other pests is not as evident but has been documented to some extent. Flooding reduces the number of fungal propagules, insects, and nematodes in the soil and, by controlling weeds, which harbor rice pathogens and insects, reduces disease and insect damage. Rice blast (caused by the fungus Pyricularia oryzae) is much less severe on flooded paddy rice than on upland rice. Fewer hours of dew occur in paddy than in upland rice, and thus P. oryzae, which requires free moisture for penetration, is less serious due to shorter infection periods.

Until the advent of the high yielding cultivars produced by IRRI, rice yields in Southeast Asia averaged 1-1.5 tons/hectare compared to yields of 3-5 tons/hectare in temperate countries such as Japan, Spain, Australia and the United States (Jennings 1964). Rice is grown from sea level to 3000 meters and from the equator to northern China (53°N latitude). It grows under a wide range of soils, environments, and cultural methods, which means that resistance to many kinds of pathogens effective over a wide range of environments must be developed. Until the establishment of the International Rice Research Institute (IRRI) most of the world's research had been on improvement of rice in temperate regions. Within a few years after its establishment, IRRI's team of agricultural scientists, building on work done in Taiwan and other tropical countries, had produced two high-yielding rice varieties: IR5 and IR8. IR8 was a short-statured, photoperiod-insensitive, stiff-strawed variety with

upright leaves and could be heavily fertilized without lodging. Under some conditions it produced yields double those of old traditional cultivars under tropical conditions. The rapid and widespread utilization of IRRI's varieties in Southeast Asia triggered what journalists called the "green revolution", but disease and insect problems rapidly reduced their usefulness. Subsequent cultivars from IRRI have not improved on the yield potential of IR8, but resistance to major diseases and insects has increased production. For example, IR8 had moderate resistance to one of the five diseases and only one of three insects of major importance to rice in Asia. IR42, one of IRRI's last named varieties, had some resistance to all eight pests (Table 1). Breeding for disease and insect resistance in rice has been reviewed by Khush (1977), Jennings et al. (1979), and IRRI's program in plant pathology by Crill (1981).

Fig. 11. Rice terraces in Luzon, the Philippines.

The rapid spread and use of the high-yielding varieties of rice in Asia has been spectacular. From 49,400 hectares in the 1965/66 crop year, the area planted to the high-yielding varieties in Asia and the Neareast in 1974/75 increased to over 53 million hectares, which is 26% of the total in these areas (Dalyrmple 1976). In 1980 almost 40% of the rice produced in South and Southeast Asia was high-yielding varieties, and they were estimated to contribute 4.5 billion dollars annually to the value of rice produced in Asia (Herdt and Capule 1983). Although this adoption of new cultivars is impressive, there is concern in the international agriculture community that much valuable diversity in rice (and other crops) is being lost as traditional cultivars are being replaced. There is also concern that the widespread use of a few cultivars with common genes may increase the chance for a new race of an existing disease or a now obscure disease or insect pest to have the potential to cause widespread, serious losses.

The collection and evaluation of rice germplasm has been the basis of the plant breeding effort and one of the most valuable ways in which the Institute has served rice breeders throughout the world. The collection started in 1961 with 256 accessions and has grown to the largest and most complete in the world with over 60,000 accessions. The evaluation and utilization of this genetic resource, including distribution of breeding materials in response to requests from rice scientists around the world, constitute about 40% of IRRI's staff effort.

Diseases are serious problems in rice production throughout the world. Padwick (1950) lists 231 different fungi recorded on rice; however, all of them do not cause disease. In addition there are bacterial and viral diseases, plus

Table 1. The resistance of IR42 to pests and other adverse conditions contrasts markedly with that of IR8, released 11 years earlier.[a]

Adverse factor	IR8	IR42
Diseases		
Blast	MR[b]	R
Bacterial blight	S	MR
Grassy stunt	S	R
Tungro	S	R
Ragged stunt	S	R
Insects		
Green leafhopper	R	MR
Brown planthopper	S	R
Stem borer	MS	MR
Nutrient deficiencies		
Nitrogen	MS	R
Phosphorus	MR	MR
Zinc	S	MR
Iron	S	MR
Soil toxicities		
Salinity	MR	MR
Alkalinity	S	MR
Iron toxicity	S	MR
Boron toxicity	MR	MR
Peat soil problems	MS	MR
Drought	S	MR
Submergence	S	MR

[a] IRRI Annual Report for 1980.

[b] R = resistant, MR = moderately resistant, MS = moderately susceptible, S = susceptible.

nematodes and non-parasitic diseases. The Commonwealth Mycological Survey plant disease distribution maps give the distribution of major rice diseases worldwide. The global importance of rice diseases is difficult to estimate. Cramer (1967), after compiling data on losses from rice diseases from all over the world, calculated worldwide rice disease losses at 8.9%. According to the U.S. Department of Agriculture (1965), annual losses due to rice diseases in the United States are about 6%. Tungro virus, sheath blight, and bacterial leaf blight are the most important rice disease problems of Southeast Asia.

The two most serious bacterial diseases of rice, bacterial leaf blight (*Xanthomonas campestris* pv. *oryzae*) and bacterial leaf streak (*Xanthomonas*

campestris pv. oryzicola), are important only in Asia; however, pv. oryzae has been reported from Africa and tropical America. Bacterial leaf streak is not found in temperate rice-growing areas, but in the Asian tropics under favorable weather conditions it can cause serious losses. Bacterial leaf blight is one of the most destructive diseases of rice in Asia and is perhaps the most serious problem of rice in India. X. campestris pv. oryzae is a systemic pathogen of the xylem. Many wild grasses are hosts. Inoculum may come from weeds, plant debris in the soil, irrigation water, or infected seed. Injuries caused by high winds or typhoons greatly increase infection. "Kresek" was the name in Indonesia for an especially severe form of bacterial leaf blight. Strains of the bacterium in the tropics are often more severe than those in temperate zones. Breeding for resistance to X. campestris pv. oryzae is complicated by the occurrence of numerous strains of the pathogen. Three of IRRI's early rice cultivars, IR5, IR8, and IR24, were susceptible to bacterial blight, but IR20 and IRRI's most recent cultivars are more resistant.

There are now over 14 known rice virus or virus-like diseases, but only three of these occur outside of Asia. Most of the knowledge of these diseases is summarized in the books by Ling (1972), Ou (1972, 1973), and the proceedings of a symposium at IRRI in 1967 on virus diseases (1969). Tungro is the most important and widespread virus disease of rice in Southeast Asia. The main vector of the viruses is the leafhopper Nephotettix virescens. Varietal resistance to the tungro viruses is tested in the field and also in a system of mass screening by which large numbers of accessions can be evaluated. Cultivars resistant to the tungro viruses may be resistant to the vector or to the viruses, but both types of resistance are used in breeding. Tungro has caused severe epidemics (Barr et al. 1975). In Indonesia 30,000 to 50,000 hectares of rice were affected in the 1930's, and in Thailand in 1966 over 300,000 hectares were severely damaged (Ou 1973). In 1971 hundreds of thousands of hectares were affected by tungro in the Philippines (Ou 1973), and the disease was

Fig. 12. Sheath blight of rice (Thanatephorus cucumeris). (Courtesy American Phytopathological Society) (left). The high-yielding rice variety IR-8 susceptible to tungro virus and IR-20 resistant (middle). Symptoms of bacterial blight of rice caused by Xanthomonas campestris pv. oryzae. (Courtesy E. H. Class) (right).

especially severe on IR8 and IR5. Fortunately the resistance of IR20 was available, and IR20 and subsequently IR26 were rapidly used to replace IR8 and IR5. IR36, at present the most widely used rice cultivar in the world, has some resistance to tungro. When some resistance to tungro was achieved, the grassy stunt disease appeared. Resistance was found in a wild species of rice O. nivara and was successfully transferred to the new cultivars produced by IRRI. Ragged stunt (Ling et al. 1978) is the most recent virus disease of rice described at IRRI.

Sheath blight (Thanatephorus cucumeris) was a minor disease of rice in tropical Asia until the introduction of the high-yielding IRRI cultivars. It clearly has become more important because of the changes in the plant type and rice production systems brought about by the introduction of IRRI varieties. Crill (1981) stated "Today it probably causes more loss than any other fungus disease of rice, especially in the lowland tropics". Although 60,000 rice entries have been screened, no high levels of resistance have been found. Blast, another important rice disease, has been studied extensively over the last 50 years.

Following are selected general references on rice diseases that are useful in studying the identification and control of rice diseases.

Selected References on Rice Diseases

Atkins, J. G. and M. A. Marchetti. 1979. Rice diseases. U.S. Dept. Agric. Farmer's Bull. No. 2120. 19 pp.

International Rice Research Institute. 1969. The Virus Diseases of the Rice Plant. John Hopkins Univ. Press, Baltimore. 354 pp.

Ling, K. C. 1972. Rice Virus Diseases. Int. Rice Res. Inst., Los Banos, Philippines. 134 pp.

Mueller, K. E. 1974. Field Problems of Tropical Rice. Int. Rice Res. Inst., Los Banos, Philippines. 95 pp.

Ou, S. H. 1972. Rice Diseases. Commonw. Mycol. Inst., Kew, Surrey. 368 pp.

Ou, S. H. 1973. A Handbook of Rice Diseases in the Tropics. Int. Rice Res. Inst., Los Banos, Philippines. 58 pp.

Padwick, G. W. 1950. Manual of Rice Diseases. Commonw. Mycol. Inst., Kew, Surrey. 198 pp.

Literature Cited

Barr, B. A., C. S. Koehler and R. F. Smith. 1975. Crop losses: Rice; field losses to insects, diseases, weeds and other pests. Univ. Calif., Berkeley/U.S. Agency for Int. Dev., Pest Management and Related Environmental Protection Proj. Rep. 64 pp.

Cramer, H. H. 1967. Plant Protection and World Crop Production. Farbenfabriken. Bayer A. G., Leverkusen. 524 pp.

Crill, P. 1981. Twenty years of plant pathology at the IRRI. Plant Dis. 65:659-574.

Dalrymple, D. G. 1976. Development and spread of high-yielding varieties of wheat and rice in the less developed nations. Foreign Agric. Econ. Rep. No. 95. U.S. Dept. Agric., Econ. Res. Serv., Washington, D.C. 120 pp.

Food Agriculture Organization. 1981. FAO Production Yearbook. Vol. 35. FAO, Rome. 306 pp.

Grist, D. H. 1968. Rice. Longman, London. 548 pp.

Herdt, R. W. and C. Capule. 1983. Adoption, spread, and production impact of modern rice varieties in Asia. Int. Rice Res. Inst., Los Banos, Philippines. 54 pp.

International Rice Research Institute. 1969. The Virus Diseases of the Rice Plant. John Hopkins Univ. Press, Baltimore. 354 pp.

Jennings, P. R. 1964. Plant type as a rice breeding objective. Crop Sci. 4:13-15.

Jennings, P. R., W. R. Coffman and H. E. Kauffman. 1979. Rice improvement. Int. Rice Res. Inst., Los Banos, Philippines. 186 pp.

Khush, G. S. 1977. Disease and insect resistance in rice. Adv. Agron. 29:265-341.

Ling, K. C. 1972. Rice Virus Diseases. Int. Rice Res. Inst., Los Banos, Philippines. 134 pp.

Ling, K. C., E. R. Tiongco and V. M. Aguiero. 1978. Rice ragged stunt, a new virus disease. Plant Dis. Rep. 62:701-705.

Ou, S. H. 1972. Rice Diseases. Commonw. Mycol. Inst., Kew, Surrey. 368 pp.

Ou, S. H. 1973. A Handbook of Rice Diseases in the Tropics. Int. Rice Res. Inst., Los Banos, Philippines. 58 pp.

Padwick, G. W. 1950. Manual of Rice Diseases. Commonw. Mycol. Inst., Kew, Surrey. 198 pp.

U.S. Dept. Agric. 1965. Losses in agriculture. Agric. Handbook No. 291. U.S.D.A., Agric. Res. Serv., Washington. 120 pp.

RICE BLAST

Disease Name: Rice blast. The disease has also been called leaf blast, blast, neck blast and rotten neck.

Causal Organism: *Pyricularia oryzae* Cavara; (Dematiaceae)

Rice blast has been known for centuries. Perhaps the first written report appeared in 1637 in a Chinese Ming Dynasty book on agronomic practices in which the disease was called "rice fever" (Ou 1972). It was known in Japan by the early 1700's; however, it was not reported in Europe or the Americas until the 19th century. A disease of rice called "brusone" was reported in Italy in 1828 by Astolfi, but it is not certain that it was blast (Ou 1972). Cavara first formally described the fungus from diseased rice plants in Italy in 1891 (Padwick 1950). Fulton (1908) published the first reports of pathogenicity tests using pure cultures of the fungus *P. oryzae*.

Blast occurs in almost all rice-growing regions of the world with over 70 countries having reported its presence. A detailed distribution map may be obtained from the Commonwealth Mycological Institute.

The Italian mycologist Cavara in 1891 placed the rice blast causal agent in the genus *Pyricularia* Sacc. and gave it the specific epithet *oryzae* (Padwick 1950). Other early workers felt that the organism was better placed in the related nematode-trapping genus *Dactylaria*; however, after years of disagreement the genus *Pyricularia* has been universally accepted as correct.

There is continued controversy as to the taxonomic limits of the species causing rice blast. The first species of *Pyricularia* described as causing disease similar to blast on monocotyledonous hosts (primarily grasses) was *P. grisae*. There is disagreement whether the two species should be considered distinct. Some authors consider the species to be morphologically indistinguishable, though others feel that there are stable differences warranting retention of the two species. Although cross inoculation studies with various isolates and hosts (including rice and non-graminaceous hosts) show some host specificity, there is considerable cross pathogenicity. At present *P. oryzae* is generally used for the pathogen on rice, and *P. grisea* is used for the pathogen of other cereals and grasses. This controversy over the proper name should not be considered merely of interest to taxonomists. It may have important practical implications to breeding programs and to disease epidemiology. Ou (1972), Asuyama (1965), and Padwick (1950) gave comprehensive discussions of the literature on the classification and nomenclature of *P. oryzae*.

The perfect stage of *P. grisea* has been produced in culture by mating two strains from crabgrass (*Digitaria sanguinalis*) (Hebert 1971). The perfect stage of *P. oryzae* has been obtained by mating strains from rice with one another and/or with *Pyricularia* spp. (presumably *grisea*) from other graminaceous hosts. All crosses yielded the ascomycete *Magnaporthe grisea* (previously *Ceratosphaeria grisea*) (Hebert 1971, Yaegashi and Hebert 1976, Yaegashi and Udagawa 1978, Kato et al. 1976, Ueyama and Tsuda 1975, Kato and Yamaguchi 1982). The perfect stage has not been found in nature.

Description of Fungus

The conidiophores are pale brown, smooth and straight, or bending. The mode of branching of the conidiophores is sympodial. A single conidium develops at the conidiophore apex. As this spore matures, the conidiophore

continues growth from just below the point of attachment of the conidium and another spore is produced at the new apex after about one hour during conditions favorable to sporulation (Asuyama 1965). Conidia are usually pyriform, 2 septate (rarely 1 or 3), hyaline to pale olive, 19-27 x 8-10 μm, with a small basal appendage. Conidia usually germinate from the apical or basal cells (Ou 1972, Asuyama 1965). Detailed descriptions of the fungus are given by Padwick (1950), Asuyama (1965), and Ou (1972).

Importance

As with most plant diseases, the impact of rice blast in a given region varies over the years depending on factors such as weather, acreages planted to particular cultivars, and rice prices. Precise figures on losses due to blast throughout the world are not available, but the disease is considered of major economic importance in most rice-growing countries. Considering its wide distribution and some of the reported losses due to rice blast, it is amazing that so little information is available from tropical countries on losses due to blast. Probably the best information on losses due to P. oryzae is from temperate countries such as Japan. Even with the excellent disease forecasting and control practices used in Japan, annual average yield losses over an eight-year period (1953-1960) were estimated at 2.9 percent (Goto 1965). Much of this area had been treated with chemicals to control P. oryzae. Abeygunawardena (1966) reported the results of a systematic survey of losses due to blast in the Anuradhapura district of Sri Lanka. An average of 28% of the panicles were infected in paddy rice and 93% of the panicles in upland rice. Although the use of regression analysis, relating yield loss with incidence, is the most common method used (Mathur et al. 1964, Goto 1965, Padmanabhan 1965), it seems that yield loss estimation is greatly affected by the method of evaluation of panicle infection. Furthermore, when loss figures are given for tropical countries, it is often not clear whether losses occurred on upland or flooded rice, or whether losses are due to leaf blast or panicle blast.

Further information on losses due to P. oryzae are given by Ou (1972), Cramer (1967), and Barr et al. (1975). Ou (1972) states:

> "blast is generally considered as the principal disease of rice because of its wide distribution and its destructiveness under favorable conditions".

For illustrative purposes, if we use a figure of 5 percent as the average annual worldwide loss from rice blast, and take 5 percent of annual world production (413 million tons of paddy), losses would exceed four billion dollars (at $200/ton), to which should be added the considerable cost of chemical control. For example, in 1979 Korea imported over 350 million dollars worth of blast control fungicides (Crill et al. 1982). In addition, since susceptibility to blast is increased when nitrogen fertilizers are used, the yield reductions due to inappropriate fertilization should also be considered. Ranking diseases, plant or otherwise, is difficult since so many criteria may be used, e.g. economic loss, human suffering, or personal and cultural biases. However, given the tremendous importance of rice worldwide as a staple food, and the economic impact of blast, it seems reasonable to suggest that rice blast is one of the most important diseases in the world today.

Symptoms

All above-ground parts of the rice plant may be attacked by P. oryzae, but the symptoms on the leaves and the neck are the most valuable for identification. The spots on the leaves are spindle shaped and usually have a brownish margin and a grey center. Environmental factors and varietal resistance may affect the size, color, and shape of the lesions. Under conditions favoring the disease, spots may become large and coalesce, killing or blighting the leaves. A cultivar highly resistant to a particular isolate or population may exhibit no symptoms or only brown flecks. Probably the most severe damage occurs when the neck of the panicle is attacked. The neck is girdled by a greyish-brown lesion and the head or panicle falls over. If the attack occurs before the milk stage, no grain is formed, but in late attacks, poor quality grain may be formed. The nodes of the stem (culm) may be affected, becoming black and brittle. The fungus also causes brown spots on the spike, branches, and grain.

Fig. 13. Lesions caused by Pyricularia oryzae (rice blast).

Host Plants

Although there are numerous discrepancies in the literature, Pyricularia species have been found on a large number of grass hosts including wheat, barley, maize, and sugarcane. Some non-graminaceous hosts are also susceptible, including common ginger, Japanese wild ginger, common canna, and banana. Asuyama (1965) summarizes the host range of P. oryzae. More than 38 grass hosts have been found susceptible to P. oryzae (most by artificial inoculation). Although the fungus can attack many grasses when artificially inoculated, the role of other grasses in the disease cycle of rice blast is not clear. In nature other grasses are seldom found heavily infected by P. oryzae.

Fig. 14. Neck rot (panicle blast) of rice caused by Pyricularia oryzae

Pathogen Cycle

It may not always be appropriate to consider "primary" and "secondary" cycles of P. oryzae as there is no cold winter in the tropics, and growing rice plants and inoculum may be present all year long. Conidia have been trapped from the air every month of the year in Los Banos, Philippines, but spore populations are highest during the rainy season. Nevertheless, in some tropical rice-growing areas and in temperate countries such as Japan

and Korea, only one crop is grown, and the source of primary inoculum may be important.

The primary cycle is initiated after a period of seasonal inactivity when susceptible rice is exposed to inoculum under favorable environmental conditions. In the tropics such seasonal inactivity may be a dry season during which rice is not grown. The primary cycle may be initiated by conidia or other fungal structures which overseason.

Overseasoning structures of P. oryzae are conidia, mycelia, chlamydospores and, perhaps, the perfect stage. Mycelia has been known to survive one year in dry straw and two to four years on nodal lesions; however, moisture, temperature, and microbial activity destroy mycelia in straw. The blast fungus can also overseason with seed. Conidia can remain viable on both seed and straw, but not as long as mycelia. Several weeds are suspected of overseasoning P. oryzae, but further work is needed to clarify the role of weeds in overseasoning.

Conidia are produced on lesions only when relative humidities (R.H.) are above 89% with an optimum at 93% R.H. Sporulation of the fungus in culture or on affected nodes of culms occurs at air temperatures of 15-35°C, while the optimum for sporulation of most strains of P. oryzae in culture is generally 28°C (Hashioka 1965). Kato (1976) reports sporulation between 12° and 34°C. Conidia are released into the air only when the relative humidity exceeds 90%. The conidia are explosively released by the osmotically induced rupture of a small cell beneath the conidium (Ingold 1964). Addition of water to conidiophores will result in the release of nearly all conidia within two minutes. In general, the more water retained on the leaves, the more spores released (Ou 1972).

Spore release typically follows a diurnal pattern. Release begins around midnight, probably a result of a conidial maturation coinciding with dew deposition, and continues to daybreak, when it abruptly ceases. A second peak of spore release has been found in the tropics following afternoon showers. Conidial dispersal is primarily via wind and air currents. Although most spores travel only a relatively short distance, they have been trapped in the air at elevations of 7000 feet (Ou 1972).

The fungus can infect seedling leaves only when they are kept in near-saturated air. Between 90-92% seems to be the critical humidity for infection. Less penetration takes place when seedlings are held in saturated or very dry air. Blast susceptibility seems to be inversely related to soil moisture. Rice plants grown in dry soil have the highest number of lesions, an intermediate number of lesions are found on rice grown in moist soil, and fewer lesions occur on plants in flooded soil. Cool temperatures seem to increase susceptibility to blast, but warmer temperatures increase resistance. This helps to explain why blast is a lesser problem in flooded or irrigated rice in the tropics than it is in upland rice in the tropics and flooded or irrigated rice in the temperate regions. Also, under upland conditions, blast is more severe on susceptible varieties because of the longer dew period that is favorable for infection by P. oryzae.

Conidial germination occurs only in free water and is most abundant at 25-30°C (Hashioka 1965). Germinating conidia produce germ tubes from which appressoria develop. Appressoria are formed after 15 hours of incubation at 20-32°C and after 24 hours at 25-35°C, with an optimum temperature of 24-28°C. The appressoria form penetration pegs which enter epidermal cells through the cuticle and cell wall. Within the epidermal cell the penetration peg develops a vesicle from which hyphae emerge. These hyphae then ramify through the host

tissue both inter and intracellularly. The pathogen may also gain ingress through stomata. The period required for invasion of the host is six hours at 24°C, eight hours at 25°C and 28°C, and ten hours at 32°C (Hashioka 1965). Kahn and Libby (1958) found the minimum period necessary to initiate infection was 10 hours at 27°C, 12 hours at 21°C, and 14 hours at 18°C. Infection occurs at high relative humidities between 90-92% (Hashioka 1965).

In susceptible cultivars at the temperature ranges normally found in the field during rice blast epidemics, it is about six days before sporulation begins (Anderson et al. 1947). Ou (1972) reported that lesions are produced in 4-5 days at 26-28°C, 5-6 days at 24-25°C, and 7-9 days at 17-18°C. However, these reports did not take into account differences in latent period among different genotypes. Castano (1981), for instance, showed that a direct relationship exists between host genotype resistance and latent period; that is, the higher the degree of resistance, the longer the latent period and visa versa.

Major factors that affect infection are:

1. Soil moisture -- Soil moisture seems to be more important than the humidity of the air. Plants grown under high soil moisture conditions (flooded rice) become more resistant, while plants grown under dry conditions become more susceptible. Thus, susceptibility to blast is inversely related to soil moisture. For example, upland rice (continuously dry or only rainfed) is used to test breeding material for resistance to *P. oryzae*.

2. Soil fertility -- Many workers have shown that high nitrogen favors an increase in infection by *P. oryzae*. Excess nitrogen is used in planting blast nurseries for evaluation of susceptibility.

3. Air temperature -- Infection can occur over a wide range of temperatures, but in the tropics, since temperature variation is small, dew formation, rainfall patterns, and relative humidity are of greater importance.

4. Air moisture -- Severe blast epidemics are usually associated with moist weather. The pattern of rainfall is also important. Heavy rainfall of short duration would not be favorable to severe infection, whereas long and frequent periods of rain showers or drizzle would favor severe blast infection. Dew formation (as in upland rice) provides free water. Free water is needed for germination of conidia and very high relative humidities (90-92%) are necessary for infection.

A vast literature is available on the disease cycle and the factors that influence it. Hashioka (1965), Ou (1972) and Kato (1976) summarize much of this information. Suzuki (1975) reviewed the literature on meteorological factors in the epidemiology of rice blast.

Control

Many of the control practices useful in reducing losses to plant diseases are of only limited applicability to control of *P. oryzae*. For example, since *P. oryzae* is present in most rice-growing areas, and since it has such a wide host range, eradication and crop rotation are of little value. Although exclusion may appear to be a useless concern, one should keep in mind that the pathogen is quite variable and that virulence factors present in one population

may not be present in another geographically isolated one. It is probably worth the effort to be sure that rice material moved from one area to another be healthy -- especially in light of the many other pathogens whose distributions are currently limited to Asia or the Americas.

A degree of control may be achieved by reducing the amount of nitrogen fertilizer applied. However, this in itself may reduce yields substantially. Other cultural methods of control are: 1) early sowing and transplanting, 2) avoidance of cold water irrigation, 3) increasing the silica content of plants by the use of compost and calcium silicate (slag), and 4) altering plant populations. To date, the most effective control measures used are chemical protection and resistant varieties.

Japan probably has the most aggressive and successful chemical control program for rice blast in the world, and it is useful to briefly trace its development. The copper fungicides were first used effectively in Japan shortly after the turn of the century, and continued to be used until after the second world war. However because of the high phytotoxicity of copper, a more attractive alternative was sought. During the 1950's organomercuric fungicides rapidly replaced the copper-based compounds for blast control. These fungicides had very low phytotoxicity and gave excellent control of blast; however, they were highly toxic to mammals. Their use continued through the mid 1960's until the Japanese government banned the use of mercury-containing agricultural chemicals following some tragic and highly publicized industrial heavy-metal poisonings.

The principal sustained effort in Japanese agrochemical research for rice blast control has been toward the development of a systemic antibiotic. The first such compound, blasticidin S, was produced from Streptomyces griseochromogenes in 1961 (Fukunaya 1968). It was an improvement over the organomercury compounds for post-infection control, but was an inferior protectant. Unfortunately, because of high phytotoxicity and mammalian toxicity, blasticidin S had to be used with extreme care. Shortly after the development of blasticidin S, a new antibiotic, (kasugamycin) produced by Streptomyces kasugaensis, was discovered. Kasugamycin gave excellent control and had very low mammalian and rice toxicity (Okamoto 1972). Beginning around 1970 in areas where the antibiotics had been used intensively and exclusively for blast control, populations of P. oryzae began to show resistance to the antibiotic compounds (Uesugi 1978). However, after halting the use of the antibiotics in areas with resistant populations, the proportion of resistant types in the populations dropped to near zero. Use of antibiotics has been successfully resumed in some areas under carefully controlled conditions (Uesugi 1978).

At about the same time the antibiotics were being developed, organophosphorus fungicides were introduced to control blast in Japan. In the late 1970's reports of resistance in P. oryzae populations to these compounds were emerging. It was found that resistance to one organophosphorus fungicide did not necessarily confer resistance to other specific fungicides, and it was suggested that rotating the use of fungicides or mixing them, rather than continuously relying on a single compound, could greatly reduce the risk of developing highly resistant populations (Uesugi 1978).

Currently chemical control practices in Japan involve chemical seed and seedling bed treatment to reduce the initial inoculum level followed by fungicide or antibiotic applications during the year. Chemical control of blast can be quite costly, but is made feasible through a sophisticated extension program, disease monitoring, and a forecasting system. This system requires considerable expense on the part of both growers and government. Extension agents

advise farmers on the appropriate chemical and cultural practices to reduce losses. The last severe outbreak of blast in Japan was in 1963. Because of a severe outbreak of blast, Korea imported 350 million dollars worth of blast control chemicals in 1979 (Crill et al. 1982).

Although a model system, the applicability of the Japanese model to most tropical developing countries is extremely limited. First, most of these countries do not have the extension capabilities or infrastructre to coordinate such a program. Furthermore, the costs of chemical control alone, without the forecasting to maximize efficiency, are prohibitive to all but the largest growers in developing countries. Likewise, seed treatment would probably be of little use in the tropics where eradication of seed-borne inoculum would have little effect on the initial inoculum load. Indeed, many plant pathologists feel that chemical control of diseases of relatively low-value per hectare crops, such as rice, is impractical in the tropics. Perhaps the most feasible control measures lie in altering cultural practices and developing resistant varieties.

Fig. 15. Severe rice blast (P. oryzae) on susceptible border rows compared with resistant breeding line in a rice blast nursery.

P. oryzae is a highly variable organism and the existence of numerous physiologic races complicates breeding for resistance and has resulted in a voluminous and contradictory literature on the subject. Previously several evaluation systems were used, but in 1967 blast workers agreed on a uniform system of inoculation, differential varieties, and methods of taking notes (Atkins et al. 1967). Ling and Ou (1969) proposed a system for standardization of race numbers of P. oryzae, and the International Rice Research Institute cooperates with other entities in planting uniform blast nurseries in 50 testing stations in 22 different countries. Thus, much valuable and standardized information on the occurrence and prevalence of races of P. oryzae has been collected. More than 260 physiologic races have been reported (IRRI 1975).

Resistance to P. oryzae in rice is usually dominant and controlled by one or a few pairs of genes. At IRRI (1979) almost 100,000 lines and accessions have been tested for resistance, and no single one has been found to be completely resistant to all races. Although sources of resistance effective against many races have been found, material released still eventually succumbs to the pathogen, should conditions be suitable.

As an illustration of the discouraging degree of variation in P. oryzae, Ou and Ayad (1968) reported that conidia produced on a single lesion may give rise to 14 races and daughter conidia from monoconidial cultures also consist of many races when tested on differential varieties. Ou and Ayad (1968) suggest that:

"These findings raise the question of the adequacy of conventional methods of determining races and the use of conventional concepts of race for pathogenicity studies of this organism. Cytological and genetic information available is not adequate to explain the extreme variability of the organism, but this can be explained by heterocaryosis if the cells prove to be multinucleate".

The problem of identifying stable races of the pathogen may stem in part from a narrow view of the host range. If, as is probably the case, P. oryzae and P. grisea are indeed the same, field evaluations of resistance would be subjected to an extremely diverse and variable inoculum. With changing environment and weed populations, the pathogen population probably also changes. It is possible that in a given area the genetic composition of the rice population may have only a limited impact on the pathogen population. Furthermore, if members of the pathogen population are isolated and grown in pure culture, analyses of their virulence may reveal the presence of "unnecessary" virulence factors on rice. These virulence factors, perhaps necessary in a population which also survives on a number of graminaceous and nongraminaceous hosts, may behave in a very puzzling manner on rice under controlled conditions.

General, horizontal, or quantitatively inherited resistance to the fungus was reported in 1971 (Ou et al. 1971). Efforts to identify, characterize, and exploit this type of resistance, which should be effective across all races, have been undertaken (IRRI 1979, Castano 1981, Villareal et al. 1981, Marchetti 1983). Although cultivars with this kind of resistance will theoretically experience some losses to all pathogen populations, they should not show complete immunity to some and extreme susceptibility to others. Thus, rather than seeking very healthy individual plants in an evaluation nursery, breeders and pathologists examine the development of epidemics on populations of the plant, selecting those plants which show reduced rates of epidemic progress over time. It is not clear that this type of selection was made for horizontal resistance at IRRI. The objective of developing this type of resistance is to help to stabilize rice production by eliminating the "boom-bust" cycle of epidemics.

The 1978 epidemic of P. oryzae in Korea appears to have altered the attitude of IRRI breeders and pathologists towards horizontal resistance. Although the improved indica-japonica hybrid rice cultivars grown in Korea that suddenly became susceptible in 1978 possessed vertical (monogenic) resistance, horizontal (polygenic) resistance had been studied in Korea since 1970 (Crill et al. 1982). They defined horizontal resistance as rice varieties with disease ratings of 4 and 5 based on the International Standard Evaluation System (Goto and Kozaka 1965). According to Crill et al. (1982) in the 1978 epidemic all varieties with horizontal resistance were extremely susceptible, and "horizontal or field resistance as a long-term rice blast control measure was rejected". Rather, a blast control scheme rotating varieties with monogenic resistance and using race prediction will be used for Korea. The success of this scheme will be watched with great interest in the future.

Since the "horizontal resistance" studied in Korea (Crill et al. 1982) may have been defined qualitatively and not quantitatively, the Korean experience should not be used as a reason to discontinue the search for rice cultivars with useful levels of general or horizontal resistance. Castano (1981) discussed the difficulties of screening for horizontal resistance in the field and suggests that an approach to overcome these difficulties could be using

components analyses in the evaluation of rice germplasm to identify parents or progeny having attributes of horizontal resistance.

The production of rice cultivars with lasting resistance to P. oryzae remains one of the major challenges to plant pathologists and breeders.

Literature Cited

Abeygunawardena, D. V. W. ed. 1966. Proceedings of a symposium on research and production of rice in Ceylon. Ceylon Assoc. Adv. Sci., Colombo, Ceylon. 307 pp.

Anderson, A. L., B. W. Henry and E. C. Tullis. 1947. Factors affecting infectivity, spread, and persistence of Piricularia oryzae Cav. Phytopathology 37:94-110.

Ashuyama, H. 1965. Morphology, taxonomy, host range, and life cycle of Piricularia oryzae. pp. 9-22. In The Rice Blast Disease. Int. Rice Res. Inst., John Hopkins Press, Baltimore. 507 pp.

Atkins, J. G., A. L. Robert, C. R. Adair, K. Goto, T. Kozaka, R. Yanagida, M. Yamada and S. Matsumoto. 1967. An international set of rice varieties for differentiating races of Piricularia oryzae. Phytopathology 57:297-301.

Barr, B. A., C. S. Koehler and R. F. Smith. 1975. Crop losses: Rice; field losses to insects, diseases, weeds and other pests. Univ. Calif., Berkeley/ U.S. Agency for Int. Dev., Pest Management and Related Environmental Protec. Proj. Rep. 64 pp.

Castano, J. 1981. Identification and testing of rate-limiting resistance of rice (Oryzae sativa L.) to blast disease caused by Pyricularia oryzae Cav. Ph.D. Thesis. Pennsylvania State Univ., Univ. Park, PA. 166 pp.

Cramer, H. H. 1967. Plant Protection and World Crop Production. Farbenfabriken. Bayer, A. G., Leverkusen. 524 pp.

Crill, P., Y. S. Ham and H. M. Beachell. 1982. The rice blast disease in Korea and its control with race prediction and gene rotation. pp. 123-130. In Evolution of the gene rotation concept for rice blast control. Int. Rice Res. Inst., Los Banos, Philippines. 130 pp.

Fukunaga, K. 1968. Antibiotics as fungicides. First International Congress of Plant Pathology. Imperial College, London. (Abstr.) pp. 62.

Fulton, H. R. 1908. Diseases affecting rice in Louisiana. Louisiana Agric. Exp. Stn. Bull. 105. 28 pp.

Goto, K. 1965. Estimating losses from rice blast in Japan. pp. 195-202. In The Rice Blast Disease. Int. Rice Res. Inst. John Hopkins Press, Baltimore. 507 pp.

Goto, K. and T. Kozaka. 1965. A proposal for an international joint study for differentiation of the races of rice blast fungus. pp. 447-450. In The Rice Blast Disease Int. Rice Res. Inst., John Hopkins Press, Baltimore. 507 pp.

Hashioka, Y. 1965. Effects of environmental factors on development of causal fungus, infection, disease development, and epidemiology in rice blast disease. p p. 153-161. In The Rice Blast Disease. Int. Rice Res. Inst. John Hopkins Press, Baltimore. 507 pp.

Hebert, T. T. 1971. The perfect stage of Pyricularia grisea. Phytopathology 61:83-87.

Ingold, C. T. 1964. Possible spore discharge mechanism in Pyricularia. Trans. Brit. Mycol. Soc. 47:573-575.

International Rice Research Institute. 1975. Annual report. Los Banos, Philippines.

International Rice Research Institute. 1979. Annual report. Los Banos, Philippines.

Kahn, R. P. and J. L. Libby. 1958. The effect of environmental factors and plant age on the infection of rice by the blast fungus, Piricularia oryzae. Phytopathology 48:25-30.

Kato, H. 1976. Some topics in a disease cycle of rice blast and climatic factors. p p. 417-425. In Proc. of the Symposium on Climate and Rice. Int. Rice Res. Inst., Los Banos, Philippines.

Kato, H., T. Yamaguchi and N. Nishihara. 1976. The perfect state of Pyricularia oryzae Cav. in culture. Ann. Phytopathol. Soc. Japan 42:507-510.

Kato, H. and T. Yamaguchi. 1982. The perfect state of Pyricularia oryzae Cav. from rice plants in culture. Ann. Phytopathol. Soc. Japan 48:607-612.

Ling, K. C. and S. H. Ou. 1969. Standardization of the international race numbers of Pyricularia oryzae. Phytopathology 59:339-342.

Marchettii, M. A. 1983. Dilatory resistance to rice blast in USA rice. Phytopathology 73:645-649.

Mathur, R. S., G. K. Bajpai, L. S. Chanhan and S. C. Verma. 1964. Assessment of losses caused by paddy blast. Plant Dis. Rep. 48:711-713.

Okamoto, M. 1972. On the characteristics of Kasumin, antibiotic fungicide. Japan Pesticide Information 10:66-69.

Ou, S. H. and M. R. Ayad. 1968. Pathogenic races of Pyricularia oryzae originating from single lesions and monoconidial cultures. Phytopathology 58:179-182.

Ou, S. H., F. L. Nuque, T. T. Ebron, and V. A. Awoderu. 1971. A type of stable resistance to blast disease of rice. Phytopathology 61:703-706.

Ou, S. H. 1972. Rice Diseases. Commonw. Mycol. Inst., Kew, Surrey. 368 pp.

Padmanabhan, S. Y. 1965. Estimating losses from rice blast in India. pp. 203-221. In The Rice Blast Disease. Int. Rice Res. Inst., John Hopkins Press, Baltimore. 507 pp.

Padwick, G. W. 1950. Manual of Rice Diseases. Commonw. Mycol. Inst., Kew, Surrey. 198 pp.

Suzuki, H. 1975. Meteorological factors in the epidemiology of rice blast. Annu. Rev. Phytopathol. 13:239-256.

Ueyama, A., and M. Tsuda. 1975. Formation of the perfect state of Pyricularia sp. from some graminaceous plants (preliminary report). Trans. Mycol. Soc. Japan 16:420-422.

Uesugi, Y. 1978. Resistance of phytopathogenic fungi to fungicides. Japan Pesticide Information 35:5-9.

Villareal, R. L., R. R. Nelson, D. R. MacKenzie and W. R. Coffman. 1981. Some components of slow-blasting of rice. Phytopathology 71:608-611.

Yaegashi, H. and T. T. Hebert. 1976. Perithecial development and nuclear behaviour in Pyricularia. Phytopathology 66:122-126.

Yaegashi, H. and S. Udagawa. 1978. The taxonomical identity of the perfect state of Pyricularia grisea and its allies. Can. J. Bot. 56:180-183.

HOJA BLANCA DISEASE OF RICE

Fig. 16. Rice field 80-90 percent infected with the hoja blanca causal agent in the Tolima Valley, Colombia.

Disease Name: Through common usage the disease is known as the hoja blanca disease of rice even in non-Spanish speaking countries. "Hoja blanca" is Spanish for "white leaf".

Causal Organism: Probably a virus

Hoja blanca was first reported from Colombia in 1935 (Bernal 1939, Garces et al. 1958), although later it disappeared about 1940 and reappeared in 1957. From Colombia the disease spread to Panama (1952), Cuba (1954), Venzuela (1956), the United States (1957), Costa Rica (1958) and into other countries in Central and South America (Galvez 1969a). It was not a problem in Peru or Brazil. Malaguti et al. (1956) reported insect vectors of the hoja blanca agent or pathogen but did not identify them. Acuna et al. (1957) found the planthopper Sogatodes oryzicola to be a vector. Detailed histories of the disease are given by Galvez (1969a), Ou (1972), and Ling (1972).

The disease has been found in most of the major rice-growing areas of northern South America and the Caribbean, and also in southern United States (Galvez 1969a, Everett and Lamey 1969). Hoja blanca apparently became a serious problem early in the 1950's because of the extensive cultivation of improved U.S. cultivars such as Bluebonnet 50 and Century Patna 231. For example, these two cultivars were grown almost exclusively in Cuba in 1956 (Atkins and Adair 1957).

Herold et al. (1968), working in Venezuela, found spherical particles 42 nm in diameter in preparations from diseased leaves. Their description does not agree with those of Shikata and Galvez (1969) or Kitajima and Galvez (1973). Shikata and Galvez found numerous bundles of long, flexous particles, 8-10 nm wide and variable in length, in hoja blanca infested rice plants and the insect vector S. oryzicola. Kitajima and Galvez (1973) found similar particles in hoja blanca infected Echinochloa colonum both in the cytoplasm and nucleus. They suggest that the particles represent the causal agent of hoja blanca disease.

Importance

Apparently from 1935 (when the disease was first noted in Colombia) until the 1950's the disease was only of minor importance in Colombia. In 1958 Garces et al. reported many fields were completely lost, and I saw fields in Colombia's Tolima Valley in 1958 with 80-90% infection which represented a total loss. A 25% loss of the entire rice crop in Cuba and a 50% loss for the rice crop in Venezuela was estimated for 1956 (U.S. Dept. Agric. 1960), and many individual fields in Cuba in 1956 had a 75% loss or were not harvested (Atkins and Adair 1957).

Symptoms

Symptoms on the various hosts infected by the hoja blanca virus are similar to those on rice, although some varietal differences in symptom expression occurs. Symptoms also vary with the age of the plant when infected. In young inoculated seedlings a few small chlorotic spots are the first symptom, generally followed by long, narrow white stripes on the next leaf to emerge. In the field yellow-white stripes or entirely yellow leaves are most common, but leaves with mottling are also observed. Plants are stunted by the disease and young plants often killed. The panicles of diseased plants are usually deformed, not fully extruded from the sheath, and spikelets are generally sterile and discolored. Comprehensive descriptions of symptoms are given by Galvez (1969a), Ou (1972), and Ling (1972).

Fig. 17. Hoja blanca of rice.

Host Plants

A number of grasses growing in or near rice fields commonly show symptoms similar to those of hoja blanca. Van Hoof (1959) reported transmission from Echinochloa colonum to E. colonum using S. cubanus. Galvez et al. (1961) reported that S. orizicola transmitted the pathogen from rice to rice and from rice to E. colonum, but not from E. colonum to E. colonum or from E. colonum to rice. Galvez et al. (1961) transferred the pathogen from rice to several grasses including Digitaria sp., Leptochloa sp., wheat, barley, and oats, but not to sugarcane, maize, or sorghum. Lamey et al. (1964) transmitted the pathogen to rye, barley, oats, and wheat. Gibler et al. (1961) reported natural infection by the hoja blanca pathogen of wheat and oats. Galvez (1977) lists a number of additional grass hosts.

Vectors and Transmission

Sogatodes oryzicola (Sogata oryzicola) is the principal vector of the hoja blanca pathogen whereas Sogatodes cubanus is the major vector in grasses. According to Galvez (1969b), S. oryzicola prefers rice but S. cubanus prefers Echinochloa colonum. Collections from rice in the field predominantly yield S. oryzicola, whereas S. cubanus predominates on Echinochloa under the same conditions. S. cubanus was found to complete its life cycle on Echinochloa and Digitaria, but only rarely on rice. Populations of the vectors vary greatly and are probably influenced by many factors. In Colombia, Galvez (1967) followed populations in the field for four years and found two population peaks a year corresponding to the two annual short dry seasons.

In the field population of S. oryzicola only a small percentage (5-15%) of individuals are capable of transmitting the pathogen (Galvez 1969b). Both

adults and nymphs can transmit the pathogen. Selective breeding of active individuals (McMillan et al. 1961, Galvez 1969b) produced a population 95-100% active. McMillan et al. (1961, 1962) studied acquisition feeding time and found that after 1, 6, and 12 hours 20, 80, and 100% of the insects studied became infective. Galvez (1977) reports an acquisition period of 15 minutes, and an incubation period in the insect from 30-36 days. The incubation period in the plant depends on its age. McMillan et al. (1961, 1962) reported an incubation period of 6-14 days and Galvez et al. (1961) 5-12 days. Several investigators have shown that the pathogen can be transmitted from eggs from infective females to their progeny (Galvez 1969b). No transmission mechanically, through soil, or through seeds has been found (Galvez et al. 1960, Galvez 1967). Galvez (1969b) made a comprehensive review of the literature on transmission of the pathogen.

Control

Neither insecticide applications to control the vector nor changes in cultural practices (fertility levels, stand density, and irrigation) have shown value as control measures (Galvez et al. 1961). After serious outbreaks of the disease in Cuba from 1954 to 1956 and discovery of the disease in Florida in 1957, U.S. Department of Agriculture scientists began an extensive program of screening rice cultivars for resistance (Lamey 1969). Almost 4,000 lines were tested in Venezuela and Cuba, and it was found that most U.S. cultivars were susceptible. In general, indica types of rice were susceptible (with a few exceptions) and japonica types were resistant. Beachell and Jennings (1961) reported that resistance was conditioned by a single dominant gene, but that minor gene action might be operative in certain variety combinations. Breeders and pathologists rapidly incorporated resistance into adapted cultivars. Lamey (1969) described the extensive program of the U.S. Department of Agriculture, the testing of breeder's selections in the field, and the development of greenhouse tests for mass screening.

In Colombia about 1967 a program was also initiated to breed for resistance to the insect S. oryzicola, and today all modern Colombian rice cultivars are resistant to the insect. Hopperburn (insect damage) is no longer a problem and S. oryzicola populations remain low. However, only a few rice cultivars are resistant to the hoja blanca virus. During 1981 to 1982 another serious epidemic of hoja blanca occurred in Colombia, Venezuela, Ecuador and Caribbean islands (Personal communication - P. R. Jennings).

The cyclical nature of the disease is poorly understood. Three severe epidemics have occurred (1935 to 1940, late 1950's to 1964, and now 1981 to 1982). The work of Jennings and Pineda (1971) may suggest an explanation for the apparent cyclical nature of the disease. They found that the virus has a deleterious effect on its vector, S. oryzicola. Viruliferous insects laid fewer eggs and hatched fewer nymphs than virus-free insects. Fertility and longevity was also reduced. They concluded:

> "that viruliferous insects are less fit for propagation of the species than nonviruliferous ones, and that the HBV (hoja blanca virus) causes a disease of its insect vector."

Only 5-15% of S. oryzicola collected in the field during epidemics can transmit the virus. Thus, the deleterious effect of the virus may reduce vector populations following epidemics, and this may be an explanation for the cyclical nature of the hoja blanca disease.

Literature Cited

Acuna, J. B., L. Ramos-Ledon, and I. Lopez-Cardet. 1957. Informaciones de interes general en relacion con el arroz. Admin. Estabilizacion Arroz. Bol. 4. Cuba.

Atkins, J. G., and C. R. Adair. 1957. Recent discovery of hoja blanca, a new rice disease in Florida, and varietal resistance tests in Cuba and Venezuela. Plant Dis. Rep. 41:911-915.

Beachell, H. M., and P. R. Jennings. 1961. Mode of inheritance of hoja blanca resistance in rice. pp. 11-12, In Proc. Rice Tech. Working Group June 29 - July 1, 1960. Lafayette, La. Agric. Exp. Stations of Arkansas, California, Louisiana, Mississippi and Texas and the Agric. Res. Serv., U.S. Dept. Agric. 36 pp.

Bernal-Correa, A. 1939. La situacion patologica del arroz en el Valle de Cauca. Revista Facultad Natl. Agron., Medellin, Colombia 1:234-238.

Everett, T. R., and H. A. Lamey. 1969. Hoja blanca. pp. 361-377. In Maramorosch, K. ed. Viruses, Vectors and Vegetation. Interscience, New York. 666 pp.

Galvez E., G. E. 1967. Frequencia de Sogata orizicola Muir y S. cubana Crawf. en campos de arroz y lindrepuerco en Colombia. Agric. Trop. (Colombia) 23:384-389.

Galvez E., G. E. 1969a. Hoja blanca disease of rice. pp. 35-49. In The Virus Diseases of the Rice Plant. John Hopkins Univ. Press, Baltimore. 354 pp.

Galvez E., G. E. 1969b. Transmission of hoja blanca virus of rice. pp. 155-163. In The Virus Diseases of the Rice Plant. John Hopkins Univ. Press, Baltimore. 354 pp.

Galvez E., G. E. 1977. Rice hoja blanca virus. pp. 28-30, In Diseases, Pests and Weeds in Tropical Crops. Kranz, J., H. Schmutterer, and W. Koch. eds.. Verlag Paul Parey, Berlin.

Galvez E., G. E., P. R. Jennings, and H. D. Thurston. 1960. Transmission studies of hoja blanca of rice in Colombia. Plant Dis. Rep. 44:80-81.

Galvez E., G. E., H. D. Thurston, and P. R. Jennings. 1961. Host range and insect transmission of the hoja blanca disease of rice. Plant Dis. Rep. 45:949-953.

Garces-Orjuela, C., P. R. Jennings, and R. L. Skiles. 1958. Hoja blanca of rice and the history of the disease in Colombia. Plant Dis. Rep. 42:750-751.

Gibler, J. W., P. R. Jennings, and C. F. Krull. 1961. Natural occurrence of hoja blanca on wheat and oats. Plant Dis. Rep. 45:334.

Herold, F., G. Trujillo, and K. Munz. 1968. Virus-like particles related to hoja blanca disease of rice. Phytopathology 58:546-547.

Jennings, P. R., and A. Pineda. 1971. The effect of hoja blanca virus on its insect vector. Phytopathology 61:142-143.

Kitajima, E. W., and G. E. Galvez E. 1973. Flexous, threadlike particles in leaf cells of Echinocloa colonum infected with hoja blanca virus. Ciencia e Cultura 25:979-982.

Lamey, H. A. 1969. Varietal resistance to hoja blanca. pp. 293-311. In Int. Rice Res. Inst. 1969. The Virus Diseases of the Rice Plant. John Hopkins Univ. Press, Baltimore. 354 pp.

Lamey, H. A., W. W. McMillan, and R. D. Hendrick. 1964. Host range of the hoja blanca vector and its insect vector. Phytopathology 54:536-541.

Ling, K. C. 1972. Rice Virus Diseases. Intl. Rice Res. Inst., Los Banos, Philippines. 134 pp.

Malaguti, G., C. Diaz, and N. Angeles. 1956. La virosis "hoja blanca" del arroz. Agron. Trop. (Venezuela) 6:157-163.

McMillan, W. W., J. U. McGuire, and H. A. Lamey. 1961. Hoja blanca studies at Camaguey, Cuba. pp. 21. In Proc. Rice Tech. Working Group June 29 - July 1, 1960. Lafayette, La. Agr. Exp. Stations of Arkansas, California, Louisiana, Mississippi and Texas and the Agric. Res. Serv., U.S. Dept. Agric. 36 pp.

McMillan, W. W., J. U. McGuire, and H. A. Lamey. 1962. Hoja blanca transmission studies on rice. J. Econ. Entomol. 55:796-797.

Ou, S. H. 1972. Rice Diseases. Commonw. Mycol. Inst. Kew, Surrey. 368 pp.

Shikata, E., and G. E. Galvez E. 1969. Fine flexous threadlike particles in cells of plants and insect hosts infected with hoja blanca virus. Virology 39:635-641.

U.S. Dept. Agric. 1960. Hoja blanca, serious threat to rice crops. Agric. Res. Serv. 22-57. 14 pp.

Van Hoof, H. A. 1959. The delphacid Sogata cubana, vector of a virus of Echinocloa colonum. Tijdshr. Pl. Ziekten. 65:188-189.

MAIZE

Maize (*Zea mays*) is the second most important crop in the world in terms of total food production. World production in 1981 was 451 million tons (FAO 1981). It is the most widely distributed cereal in the tropics and is important in the Americas, Africa, and Asia. Most of the maize produced in temperate areas is used for livestock feed and industrial products. Maize produced in tropical countries is primarily for direct human consumption.

Many investigators have studied the origin of maize, and it is a fascinating story which is still unfolding. Although multiple sites of origin are postulated in both South and Central America, numerous archeological studies suggest that Mexico is the earliest site of origin. Maize has probably been utilized by man in Mexico for around 7000 years. When Europeans arrived in the Americas they found maize cultivated from Chile to Canada and from sea level on the equator to altitudes of over 3500 meters in the Andes. Today, it is probably the most widely distributed cereal in the world.

Many types of maize are found today: popcorn, flint, dent, soft or floury, sweet, and waxy. Thousands of years of cultivation in the Americas produced cultivars adapted to the numerous agroecosystems where maize is grown. Maize is the staple food for much of the population of Mexico and Central America, where it is usually consumed as a bread made from the ground kernels.

Maize grows well under warm, moist conditions, but sorghum and millet tolerate dry conditions better. Maize is grown interplanted with other crops (e.g. beans and squash) in tropical America and Africa, and as a monoculture in the North American corn belt. In the United States it is grown with sophisticated technological and chemical inputs, whereas in parts of the tropical world it is planted with a wooden digging-stick in slash and burn agriculture. These are only the extremes of the wide range of cultivation systems under which maize is grown.

Few crops have received as intensive efforts at genetic improvement as maize. Many genes on the 10 chromosomes of maize have been mapped. Maize is

Fig. 18. Stone statue of Indian grinding maize from Mexican Museum of Anthropology. (Courtesy of Peter T. Ewell)

an ideal crop for improvement, as it is cross pollinated, highly heterozygous, and is easily manipulated by breeders. The dramatic increase in maize yields in the corn belt of the United States since the introduction of hybrids in 1940 has given tremendous impetus to all crop improvement efforts throughout the world.

In cooperation with the Mexican government the Rockefeller Foundation and CIMMYT (International Center for the Improvement of Maize and Wheat) have had international programs of maize improvement since 1941. Yields in the developing countries average only 1.2 t/ha versus 4.6 t/ha in the industralized countries of Europe and North America. CIMMYT has focused on high yielding maize with wide adaptation, disease and insect resistance, higher protein quality, a shorter growing period, and shorter plants for the tropics that can be grown at higher density without lodging. Traditional tropical maize cultivars often grow 4 to 5 meters high and lodge badly. The Mexican maize improvement program first emphasized hybrids, which gave yields similar to the best in the United States. However, hybrids had only limited success because of the lack of efficient local agencies for producing hybrid seed and because of poor yields achieved under the low fertility and moisture conditions typical of small traditional farms such as those in Mexico. Similar experiences with hybrid maize occurred in Asia, Africa, and other countries of Latin America. Farmers traditionally saved seed from one crop to the next and, even if they could get it, often refused to pay higher prices for hybrid seed. Therefore, in the late 1950's, emphasis in CIMMYT's breeding program was shifted. Composites, or genetically diverse populations, were developed under programs of long-range population improvement, and these open-pollinated varieties often yielded almost as well as hybrids. In addition, their seed could be saved from one crop to the next. Improved populations adapted to the major maize growing regions of the world and designed to meet farmer requirements are now being developed in an intensive interdisciplinary program which conducts maize trials through a global network which includes over 78 countries. Varieties adapted to localized areas are selected from CIMMYT populations within various countries.

Diseases are a major problem in increasing tropical maize yields. Fortunately, excellent information is easily accessible on most maize diseases. Major worldwide problems are leaf-blighting fungi (*Helminthosporium turcicum* and *H. maydis*), rusts (*Puccinia sorghi*, *Puccinia polysora*, and *Physopella zeae*), various pathogens causing stalk and ear rots (*Pythium* spp.,

Diplodia maydis, Gibberella zeae and G. fujikuroi, Erwinia chrysanthemi pv. zeae, Macrophomina phaseolina, smuts (Ustilago maydis and Sphacelotheca reiliana), various viruses, and the downy mildews.

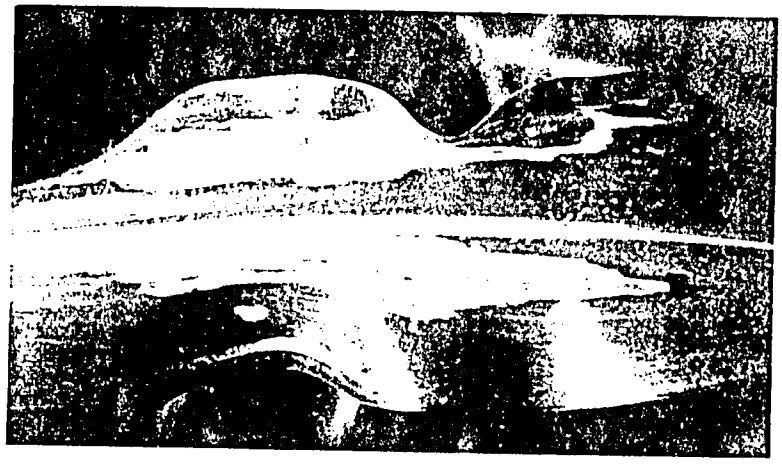


Fig. 19. Lesions on maize caused by Helminthosporium turcicum (Northern leaf blight).

The following are selected references on maize diseases that may be useful in studying their identification and control.

Selected References on Maize Diseases

De Leon, C. 1978. Maize Diseases: a Guide for Field Identification. Centro Intl. Mejoramiento Maiz y Trigo, Mexico. 93 pp.

Dickson, J. G. 1956. Diseases of Field Crops. McGraw-Hill, New York. 517 pp.

Shurtleff, M. C. ed. 1980. A Compendium of Corn Diseases. Am. Phytopathol. Soc., St. Paul, MN. 105 pp.

Ullstrup, A. J. 1974. Corn diseases in the United States and their control. Agric. Handbook No. 199, U.S. Dept. Agric., Washington, DC. 56 pp.

Literature Cited

Food Agriculture Organization. 1981. FAO Production Yearbook. Vol. 35 FAO, Rome. 306 pp.

PHILIPPINE DOWNY MILDEW OF MAIZE

Disease Name: Philippine downy mildew of maize is the common name for the disease. Seventeen different names are recorded for downy mildew in the Philippines.

Causal Organism: *Peronosclerospora philippinensis* (Weston) Shaw; (Peronosporaceae)

Downy mildews are the most serious diseases of maize in tropical Asia (Frederiksen and Renfro 1977, Williams 1983). Ten species of fungi are known to cause downy mildew on maize: *Sclerophthora macrospora*, *Sclerophthora rayssiae* var. *zeae*, *Sclerospora graminicola*, *Peronosclerospora sorghi*, *P. philippinensis*, *P. sacchari*, *P. maydis*, *P. heteropogoni*, *P. spontanea*, and *P. miscanthi*. The most destructive tropical species are *P. philippinensis*, *P. maydis* in Indonesia, *P. sacchari* in Taiwan, *P. sorghi* in Thailand, and *Sclerophthora rayssiae* var. *zeae* in India. Of the above destructive *Peronosclerospora* species, only *P. sorghi* is confirmed to occur outside of Asia in Africa and the Americas. The first report of *P. sorghi* in the Americas was in Texas and northern Mexico in 1961. Since then it has spread to 16 other southern states of the United States (Frederiksen 1980). In 1971 *P. sorghi* caused a 2-3 million dollar loss of the Texas maize and sorghum crop (R. A. Frederiksen - personal communication). Since 1976 it has spread to Central and South America, causing severe losses in Venezuela (Malaguti et al. 1977).

Although Philippine downy mildew of maize is not the most important tropical disease of the crop, potentially it may be the most destructive corn disease in Asia. More importantly, since its distribution is currently limited to Asia, there is the considerable risk that this highly destructive disease will eventually spread to other tropical and temperate maize-growing regions. At present *P. philippinensis* is found in the Philippines, India, Nepal, and Indonesia (H. A. van Hoof - personal communication). This global threat to maize production was recognized as far back as 1918 when William H. Weston, Jr. was sent to the Philippines as a pathologist for the U.S. Department of Agriculture to study this disease. In part, he was sent because of the recognized danger to corn plantings in the United States if the disease was ever introduced. His studies were the first comprehensive work done on Philippine maize downy mildews and are still an excellent source of information.

According to Weston (1920) the first downy mildew of maize in Asia was called *Peronospora maydis* by Raciborski who found it in Indonesia. Later Butler, in 1907, reported *Sclerospora* on various grass species in India, and in 1920 found it on maize, calling it *Sclerospora maydis*. Weston (1920) noted that Baker in 1916 was first to describe the disease in the Philippines. Baker and Reinking both called the Philippine downy mildew of corn *S. maydis*, but Weston (1920) found differences which promoted him to consider it a different species, *Sclerospora philippinensis*. He differentiated it from other *Sclerospora* species on corn on the basis of the size and shape of conidia. Weston (1920) first described and named the fungus *Sclerospora philippinensis* (Weston), an oomycete in the order Peronosporales and the family Peronosporaceae. Shaw (1978) changed the subgenus *Peronosclerospora* of *Sclerospora* to generic rank and thus *S. philippinensis* was transferred from *Sclerospora* to *Peronosclerospora* in 1978. Inoculation experiments have shown *P. philippinensis* (Weston 1920) to be the causal organism of Philippine downy mildew on maize, teosinte, and sorghum.

Fig. 20. Natural infection (light colored plants) by Peronosclerospora philippinensis (downy mildew of maize) in the Philippines.

Weston's (1920) description of the asexual reproductive structures of P. philippinensis was as follows:

> "Conidiophores always produced in night dew and growing out of the stomata, erect, 150 to 400 μ long, 15 to 26 μ thick, bearing a basal cell in the lower part, dichotomously branched two to four times above, branches robust, sterigmata conoid to subulate, 10 μ long, slightly curved.
>
> Conidia elongate ellipsoid, elongate ovoid, or rounded cylindrical varying in size, usually 27 to 39 μ long by 17 to 21 μ broad, hyaline, with thin episorium, minutely granular within, slightly rounded at the apex, provided with an apiculus at the base, always germinating by tube."

Oospores of P. philippinensis have never been found (Frederiksen and Renfro 1977).

Importance

The disease is sometimes extremely destructive in the Philippines. Under conditions favorable for the fungus whole fields were destroyed, and in some areas growers were forced to abandon corn production (Weston 1920). Losses of 40-60% are common in rainy seasons with disease incidence of 80-100% (Exconde and Raymundo 1974). A national survey found losses ranging from 15-40% and a total loss of 8% of the Philippine corn crop was calculated for the 1974-75 crop year. This loss was estimated to have a value of over 22 million U.S. dollars (Exconde 1976). During a January 1968 trip through Mindanao in which I saw hundreds of corn fields, the average infection was about 15-25% although one field had over 90% infection. It should be noted that in January conditions are not optimal for infection by S. philippinensis.

Payak (1975) stated that in some fields losses due to P. philippinensis may be as high as 60% in India, but other reports suggest it is not of great importance there. Splitter (1975) states that P. philippinensis is one of the most destructive maize diseases in Nepal.

Symptoms

The most striking symptom of P. philippinensis is the long, narrow chlorotic streaks on the leaves. In the morning a downy growth of conidia and conidiophores may often be seen on the underside of the leaf. Occasionally various malformations of the tassel and ear occur although these are not common. Early infection may result in stunting or even death.

Host Plants

In addition to maize, Weston (1920) also reported teosinte (Euchlaena luxurians) and sorghum (Sorghum bicolor) infected by S. philippinensis. Later "wild sugar cane", Saccharum spontaneum, and Miscanthus japonicus were found infected (Weston 1921). Exconde et al. (1968b) reported that P. philippinensis would also infect the following hosts: Avena sativa (oats), Saccharum officinarum (sugarcane), Sorghum bicolor (grain sorghum), Sorghum halapense (johnsongrass), and Sorghum propinquum.

Pathogen Cycle

Inocula which can initiate infection include conidia and possibly mycelia. Sources of conidia can be infected maize from adjacent fields, volunteer plants, and alternate weed hosts such as Saccharum spontaneum common in tropical maize growing areas. Exconde (1976) reported seed infected with mycelium and an average seed transmission of 8% in moist grain. Seedborne transmission does not appear to be a threat, as there is no evidence of transmission in dry seed.

The conditions for formation of inoculum have been studied extensively. A layer of moisture on the surface of infected plants from dew or a gentle rain and darkness are required for conidial production. Conidia are produced only at night. It takes 4-5 hours to produce conidia under these conditions (Weston 1923b, Exconde et al. 1967). Exconde (1976) reported conidial production at night with 85-95% relative humidity (RH) and a temperature of 21-26°C. Highest spore production occurred at 23-24°C at 90-94% RH for 6-8 hours. In general, temperature and RH are more important than rainfall and solar radiation in inducing sporulation on a highly susceptible maize variety (Exconde et al. 1968b). Conidial production can continue on maize plants for over two months (Weston 1923b). Weston suggested that conidia are forcibly discharged, but only 1-2 mm. Usually, all the conidia on the conidiophore are ejected at once. Apparently discharge is due to bulging out of the conidium and sterigmata until the basal septum of the conidium and the apical septum of the sterigmata bulge enough so that the conidium is shot away (Weston 1923b). Conidiophore development, sporulation, dissemination, inoculation, and penetration all take place during the night. Weston (1923b) gives the following nocturnal cycle of

Fig. 21. Symptoms of downy mildew of maize caused by Peronosclerospora philippinensis.

development on maize:

7- 8 PM - dew deposit begins
10-11 PM - surface wet, conidiophore initials appear in stomata
11-12 PM - conidiophores develop
12- 1 AM - conidia develop
1- 2 AM - discharge and dispersal begin
2- 3 AM - discharge at maximum
3- 4 AM - discharge lessens, germination of conidia
4- 5 AM - little discharge, much germination
5- 6 AM - drying of conidiophores

Duration of the above process continued for four, six, or even eight weeks in some varieties. Sorghum and sugar cane had a shorter period of conidial production but teosinte was somewhat longer. "Wild sugar cane" (Saccharum spontaneum) could maintain spore production for up to eight months. This probably helps to explain why infection takes place at all times of the year. Immense amounts of inoculum are produced under optimal conditions.

Wind is the most important agent in spore dispersal. Violent storms (typhoons) may give a sudden scattering of the disease over several miles in the path of the storm. Splashing water from dew and rain account for only limited spread of the disease. Insects probably play a minor role in dissemination.

Germination is preceded by swelling and a change in the size and shape of the conidia. Conidia germinate by producing one or more germ tubes from any part of the spore. No zoospores or secondary conidia were found. Conidia germinate readily at temperatures as low as 6.5°C even though the temperature at which they most commonly germinate is 20-24°C (Weston 1920). Dalmacio and Exconde (1969) found that conidia germinated in less than one hour after placement on maize. Exconde et al. (1967) found a temperature range of 19-20°C optimal for conidial germination. Weston (1920) found that germination took place in dew, rain water, brook water, and various dilute nutrient solutions.

Young seedlings are most susceptible, and plants become more resistant as they age. Four to six-week-old plants are almost immune. The most susceptible part of corn seedlings under controlled inoculation has been the whorl. Leaf penetration is through stomata by germ tube or hyphae. Appressoria are usually formed, but penetration without appressorial formation also occurs.

After inoculation, the first symptoms of infection are observed after three days. All organs except roots are invaded, and the fungus mycelium may be traced down through the leaf sheath to the stem where it may ramify throughout the tissue. After becoming established in the apex, the fungus survives there until the plant dies. Apical meristem infection often results in growth deformations. Hyphae are most abundant in the chlorotic areas of leaves showing symptoms but are also found in areas of the leaf with no discoloration. The tassel and ear may also be invaded. Hyphae grow intercellularly and haustoria penetrate the cell wall and enter the lumen. Penetration does not cause collapse of host cells (Dalmacio and Exconde 1969, Weston 1920).

Philippine downy mildew of maize provides a good example of how concepts of disease management and epidemiology developed under temperate conditions may not be applicable in the tropics. Current epidemiological thinking suggests that there are essentially two different ways that disease develops in the field. Monocyclic diseases are characterized as those in which the pathogen may complete only one or a very few growth cycles in one growing season.

Polycyclic diseases are caused by pathogens which complete a large number of cycles in a season and have the capacity to develop devastating epidemics in a single season, should conditions be "favorable".

Although maize can be infected by P. philippinensis throughout the growing season, generally only plants up to three weeks of age are actually infected. Thus, Philippine downy mildew might be considered a typical monocyclic disease. However, since maize is planted all year around in some areas of the Philippines (e.g. Mindanao) where the disease is highly destructive, fields differing widely in stage of development coexist in relatively small areas. Furthermore, susceptible hosts such as S. spontaneum are abundant in or near corn fields, and conidia are probably produced in large numbers whenever environmental conditions are appropriate. Thus, a pathogen (or pathogen population) with a monocyclic life cycle on a given plant, or population of plants, may have many characteristics more commonly associated with polycyclic organisms. This, of course, greatly complicates management strategies. Considerable research remains to be done to clarify the epidemiology of this and similar diseases. Bonde (1982) has recently reviewed the epidemiology of maize downy mildews.

Control

Most countries have a strict quarantine on plant material (maize, sorghum, sugar cane) from Asia, but how long this will be successful in excluding P. philippinensis from non-Asian areas is unknown. It has been shown that mycelium of P. sorghi is present in maize seed (Jones et al. 1972); the same situation also occurs with P. philippinensis (Advincula and Exconde 1975, Exconde 1976). Although oospores of P. philippinensis have never been found, Weston (1923a) was so concerned about the possibility that oospores might be present in seed that he worked out a method of treating corn seed with concentrated sulfuric acid which eliminated oospores of other downy mildew species. Seedborne transmission appears to be no threat, as there is no evidence of transmission in dry seed.

Considerable effort has been devoted to chemical control of P. philippinensis in the Philippines with a variety of fungicides and methods of application such as foliar sprays, soil, and seed treatments. Up to 1965 tests of 63 fungicides and 8 antibiotics were made (Exconde 1975). Chloroneb used as a seed treatment plus 3-4 foliar sprays showed some promise of economic control (Schultz 1971). None gave adequate, economic protection until the systemic fungicide metalaxyl was tested (Exconde and Molina 1978). Metalaxyl shows specific toxicity to fungi in the Peronosporales and completely protected four maize lines from emergence to harvest under conditions where the untreated checks had 83 to 100% infection. Later, Molina and Exconde (1981) reported that untreated seedlings had 97.6% infection, whereas metalaxyl-treated seeds were not infected regardless of rainfall frequency. Exconde (personal communication) notes that no case of resistance to metalaxyl applied as a seed dressing has been reported, although the fungicide has been used since 1979. The long term value of metalaxyl for control of P. philippinensis might not be considered high based on experience with other pathogens to selective fungicides. However, considering that it is a monocyclic disease and that plants are only infected up to three weeks of age, resistance to metalaxyl may not develop for some time.

Although relatively little research has been done on the value of cultural practices in controlling P. philippinensis, a number of such practices might reduce losses:

1. Eliminating weed hosts and volunteer corn in and near corn fields.

2. Manipulating planting dates to avoid the disease.

3. Regulating plant density, wider spacing, or interplanting maize with other crops to allow better air drainage and thus more rapid drying of plants in order to reduce periods favorable for infection.

4. Where economically feasible, removing infected plants early in the season to reduce inoculum levels.

5. Planting corn in a given area only once a year, not continuously. This would require grower cooperation or governmental intervention, but might be feasible in areas where weed hosts are not of great importance and where optimal planting dates are well established.

Considerable effort has gone into the search for host plant resistance in the Philippines since the initiation of resistance breeding in 1953 (Carangal et al. 1970, Aday 1975). Maize is not native to the Philippines, but was probably introduced there by the Spanish in the sixteenth century (Purseglove 1972). Thus, maize has probably been exposed to P. philippinensis in the field for over four centuries. Work in the Philippines has included selection and isolation of resistant lines of maize, screening of local and introduced varieties, varietal hybridization between resistant lines and high-yielding introduced germplasm, and development of composite maize populations with resistance to P. philippinensis and other good agronomic characters (Aday 1975). Lines with high levels of resistance were found among native Philippine lines, especially those from Mindanao where the disease is most severe. Resistance is probably a quantitatively inherited trait that follows an additive pattern of inheritance (Gomez et al. 1963, Francis 1967). Intensive screening of local and introduced maize germplasm indicated that Philippine germplasm was the only useful source of resistance. Maize downy mildew-resistant varieties and hybrids were developed, and the most popular is Institute of Plant Breeding Variety 1. In 1982 resistant varieties and hybrids were commercially available countrywide and were planted in almost 60% of the maize area of the Philippines (O. R. Exconde - personal communication). How stable the resistance of these lines will be with time is unknown, but since resistance is thought to be quantitatively inherited, it probably will be relatively stable. Tititarn and Exconde (1974) report differences in virulence among 7 isolates of P. philippinensis studied, but little evidence for distinct physiologic races has been found. The nature of the resistance of the Philippine resistant maize lines is still poorly understood.

Literature Cited

Aday, B. A. 1975. The Philippine program in breeding for resistance to downy mildew of maize. pp. 207-219. In Symposium on downy mildew of maize. Trop. Agric. Res. Ser. No. 8, Trop. Agric. Res. Center, Min. Agric. & For., Tokyo. 259 pp.

Advincula, B. A., and O. R. Exconde. 1975. Seed transmission of Sclerospora philippinensis Weston in maize. Philipp. Agric. 59:144-255.

Bonde, M. R. 1982. Epidemiology of downy mildew diseases of maize, sorghum and pearl millet. Trop. Pest Management 28:49-60.

Carangal, V., M. Claudio, and M. Sumayao. 1970. Breeding for resistance to maize downy mildew caused by Sclerospora philippinensis in the Philippines. Indian Phytopathol. 23:285-306.

Dalmacio, S. C., and O. R. Exconde. 1969. Penetration and infection of Sclerospora philippinensis Weston on corn. Philipp. Agric. 53:35-52.

Exconde, O. R. 1975. Chemical control of maize downy mildew. pp. 157-163. In Symposium on downy mildew of maize. Trop. Agric. Res. Ser. No. 8, Trop. Agric. Res. Center, Min. Agric. & For., Tokyo. 259 pp.

Exconde, O. R. 1976. Philippine corn downy mildew: assessment of present knowledge and future research needs. Kasetsart J. 10:94-100.

Exconde, O. R., E. A. Edralin, and B. A. Advincula. 1967. Some factors affecting formation and germination of spores of Sclerospora philippinensis. pp. 1-13. In Fourth Inter-Asian Corn Improvement Workshop, Lyallpur, West Pakistan.

Exconde, O. R., J. Q. Adversario, and B. A. Advincula. 1968a. Incidence of corn downy mildew in relation to planting date and meteorological factors. Philipp. Agric. 52:189-199.

Exconde, O. R., J. V. Elec, and B. A. Advincula. 1968b. Host range of Sclerospora philippinensis Weston in the Philippines. Philipp. Agric. 52:175-188.

Exconde, O. R., and A. D. Raymundo. 1974. Yield losses caused by Philippine corn downy mildew. Philipp. Agric. 58:115-120.

Exconde, O. R., and A. B. Molina, Jr. 1978. Ridomil (Ciba-Geigy) a seed-dressing fungicide for the control of Philippine corn downy mildew. Philipp. J. Crop Sci. 3:60-64.

Francis, C. A. 1967. Downy mildew of maize in the Philippines. M.S. Thesis. Cornell Univ. 166 pp.

Frederiksen, R. A. 1980. Sorghum downy mildew in the United States: Overview and outlook. Plant Dis. 64:903-908.

Frederiksen, R. A., and B. L. Renfro. 1977. Global status of maize downy mildew. Annu. Rev. Phytopathol. 15:249-275.

Gomez, A. A., F. A. Aquilizan, R. M. Payson, and A. G. Calub. 1963. Preliminary studies on the inheritance of the reaction of corn to downy mildew disease. Philipp. Agric. 47:113-116.

Jones, B. L., J. C. Leeper, and R. A. Frederiksen. 1972. Sclerospora sorghi in corn: its location in carpellate flowers and mature seeds. Phytopathology 62:817-819.

Malaguti, G., A. Fernandez B., and H. Nass. 1977. "Mildiu lanoso" o "punta loca" del maiz en Venezuela. Agron. Trop. (Venezuela) 27:103-129.

Molina Jr., A. B., and O. R. Exconde. 1981. Efficacy of Apron 35 SD (Metalaxyl) against Philippine corn downy mildew 1. Effects of seed-dressing methods and rainfall frequency. Philipp. Agric. 64:99-105.

Payak, M. M. 1975. Downy mildews of maize in India. pp. 13-18. In Symposium on downy mildew of maize. Trop. Agric. Res. Ser. No. 8, Trop. Agric. Res. Center, Min. Agric. and For., Tokyo, Japan. 259 pp.

Purseglove, J. W. 1972. Tropical Crops, Monocotyledons. Wiley, New York. 607 pp. 2 Vol.

Schultz, O. E. 1971. Evaluation of seed treatment with chloroneb for control of Philippine downy mildew of corn. pp. 104-107. In Seventh Inter-Asian Corn Improvement Workshop Proc., College, Laguna.

Shaw, C. G. 1978. Peronosclerospora species and other downy mildews of the Gramineae. Mycologia 70:594-604.

Splitter, M. V. 1975. Downy mildew disease in Nepal. pp. 19-20. In Symposium on downy mildew of maize. Trop. Agric. Res. Ser. No. 8, Trop. Agric. Res. Center, Min. Agric. and For., Tokyo. 259 pp.

Tititarn, S., and O. R. Exconde. 1974. Comparative virulence and gross morphology of isolates of Sclerospora philippinensis Weston on corn. Philipp. Agric. 58:90-104.

Weston, W. H., Jr. 1920. Philippine downy mildew of maize. J. Agric. Res. 19:97-122.

Weston, W. H., Jr. 1921. Another conidial Sclerospora of Philippine maize. J. Agric. Res. 20:669-684.

Weston, W. H., Jr. 1923a. A method of treating maize seed to destroy adherent spores of downy mildew. J. Agric. Res. 24:853-860.

Weston, W. H., Jr. 1923b. Production and dispersal of conidia in the Philippine Sclerosporas of maize. J. Agric. Res. 23:239-278.

Williams, R. J. 1984. Downy mildews of tropical cereals. pp. 1-103. In Ingram, D. S. and P. H. Williams. eds. Advances in Plant Pathology II. Academic Press, New York.

SORGHUM

Sorghum (*Sorghum bicolor*), although fifth in importance among the world's cereals, often produces yields in developed countries five times those of the tropics. Almost all production in developed countries is for animal feed. Nevertheless, sorghum is a major staple for the 400 million people who live in the world's semi-arid tropics and is the leading cereal in Africa. World production in 1981 was 72 million tons (FAO 1981).

The origin of sorghum was probably Ethiopia, and authorities suggest it may have been domesticated there perhaps 5000 years ago. From there it spread to West and East Africa and eventually to other tropical continents. At present, it is most important as a food crop in Africa and Asia, but is becoming increasingly important in the Americas. Sorghum requires less moisture than maize and will also tolerate poorly drained soil better than does maize. Thus, it will yield under conditions unfavorable for most cereals, and under conditions too hot and dry for maize.

Sorghum is a grass, and a wide variation in plant types and grain color have evolved through the centuries. For example, stalks may vary from 1 to 5 meters in height. For human food the seed is ground into flour and made into porridges or bread. Considerable quantities are used for making sorghum beer in Africa. The stalks are used as fuel, fodder, and building materials. In temperate areas such as the United States it is used as a fodder, for syrup production, and the grain as an animal food.

There are great opportunities for sorghum improvement, as has clearly been shown by the rapid progress made in the United States in breeding and culture. India has had an intensive breeding program for many years, and recently ICRISAT (International Crops Research Institute for Semi-Arid Tropics) has begun a worldwide program in sorghum improvement. ICRISAT is using the world germplasm collection of over 23,000 lines assembled in India. A great potential exists for improvement of sorghum by breeding for higher yield, better protein quality, resistance to pests, shorter plants, greater response to fertilizers, drought tolerance,

day-length insensitivity, and better grain quality.

Work on sorghum diseases at ICRISAT was initiated in 1974. The traditional, low-yielding sorghums in the semi-arid tropics were selected to ripen after the end of the rainy season; however, higher yielding sorghums with early maturity ripen while the rainy season is still in progress. Thus, a major problem is the fungi which reduce quantity and quality of the grain under moist conditions. Lines with resistance to grain molds have been identified and are being tested in international nurseries in Asia and Africa.

ICRISAT plant pathologists have as objectives the identification and utilization of stable resistance to the major diseases of sorghum. Diseases given priority are grain molds (a complex of several fungi), downy mildew (Peronosclerospora sorghi), stalk rots (Macrophomina phaseolina and Fusarium moniliforme), and several leaf diseases which include anthracnose (Colletotrichum graminicola) and leaf blight (Helminthosporium turcicum).

Selected references on sorghum diseases follow:

Selected References on Sorghum Diseases

Amador, J., R. W. Berry, R. A. Frederiksen, C. W. Horne, W. H. Thames, and R. W. Toler. 1971. Sorghum diseases. Bull. B-1085. Texas A&M Univ., College Station, TX. 21 pp.

Dickson, J. G. 1956. Diseases of Field Crops. McGraw-Hill, New York. 517 pp.

Doggett, H. 1970. Sorghum. Longman, London. 403 pp.

Edmunds, L. K., M. C. Futrell, and R. A. Frederiksen. 1970. Sorghum diseases. pp. 200-234. In Sorghum Production and Utilization. J. S. Wall and W. M. Ross. eds. Avi Publ., Westport, CT. 702 pp.

Edmunds, L. K., and N. Zummo. 1975. Sorghum diseases in the United States and their control. U.S. Dept. Agric. Handbook 468. U.S. Dept. Agric., Washington, DC. 47 pp.

International Crops Research Institute for the Semi-Arid Tropics. 1980. Sorghum Diseases, a World Review. Proceedings of the International Workshop on Sorghum Diseases, Texas A&M Univ. and ICRISAT, 11-15 December 1978, Hyderabad, India.

Tarr, S. A. J. 1962. Diseases of Sorghum, Sudangrass, and Broom Corn. Commonw. Mycol. Inst., Kew, Surrey. 380 pp.

Williams, R. J., R. A. Frederiksen, and J.-C. Girard. 1978. Sorghum and pearl millet disease identification handbook. Int. Crops Res. Inst. Semi-Arid Tropics Information Bull. No. 2, ICRISAT, Hyderabad, India. 88 pp.

Literature Cited

Food Agriculture Organization. 1981. FAO Production Yearbook. Vol. 35. FAO, Rome. 306 pp.

MILLETS

The term millet is used for a number of grasses with small edible seeds which do not shatter readily at maturity. The major tropical species are pearl or bulrush millet (Pennisetum typhoides) and finger millet (Eleusine coracana). Species of lesser importance are foxtail millet (Setaria italica), proso millet (Panicum miliacum), Japanese millet (Echinochloa crusgalli var. frumentacea) and browntop millet (Panicum ramosum). In the tropics, millets are fourth in importance among the cereals after maize, rice and sorghum. Annual world production in 1981 was near 30 million tons (FAO 1981).

Some millets can grow in arid areas and tolerate lower soil fertility than sorghum. Millets in temperate developed countries are used for forage and animal feed, but in the harsh conditions of the semi-arid tropics, especially in India and Africa, millets are an important staple food. The grain can be consumed as whole grain or ground into flour which is used for porridge and various flat cakes or chapattis. Beer is made from millet in Africa. The straw is valuable as forage, fuel, fencing, and thatching.

Pearl millet is probably the most important of the millets and will be considered here. It is grown primarily in India and Africa on an estimated 25 million hectares. The richest source of genetic diversity for pearl millet occurs in West Africa, and this area is probably the site of its origin. Pearl millet is undoubtedly the grain best able to grow and produce a crop in the dry, rainfed areas of the tropics with sandy, infertile soils. It is most important in the Sahel region of Africa and the arid areas of India, and will grow in areas too dry for sorghum.

ICRISAT (International Crops Research Institute for the Semi-Arid Tropics) has selected pearl millet for one of its major crop improvement programs because of its importance as a food crop in Southern Asia and Africa. Scientists at ICRISAT are evaluating the worldwide germplasm collection in an attempt to improve yields, disease and insect resistance, tolerance to drought and low fertility, protein content, and grain quality.

Improvement of yield potential in pearl millet involves the improvement of resistance to downy mildew (Sclerospora graminicola), ergot (Claviceps fusiformis), smut (Tolyposporium penicillariae), and in Africa, resistance to the

parasitic witchweed (Striga hermonthica). Diseases of potential importance include rust (Puccinia penniseti) and leaf blast (Pyricularia setariae). At ICRISAT it has been shown that resistance to all three major diseases can be rapidly increased through recurrent selection with effective screening techniques. Sources of multiple disease resistance have been developed.

Selected References on Millet Diseases

Dickson, J. G. 1956. Diseases of Field Crops. McGraw-Hill, New York. 517 pp.

Ferranis, R. 1973. Pearl Millet. C.A.B. Rev. Series No. 1. Commonw. Agric. Bur., Kew, Surrey. 70 pp.

Rachie, K. O., and J. V. Majmudar. 1980. Pearl Millet. Penn. State Univ. Press, Univ. Park, PA. 307 pp.

Ramakrishnan, T. S. 1963. Diseases of Millets. Indian Council Agric. Res., New Delhi. 152 pp.

Williams, R. J., R. A. Frederiksen, and J.-C. Girard. 1978. Sorghum and pearl millet disease identification handbook. Information Bull. No. 2. Int. Crops Res. Inst. for the Semi-Arid Tropics, Hyderbad, India. 88 pp.

Literature Cited

Food Agriculture Organization. 1981. FAO Production Yearbook. Vol. 35. FAO, Rome. 306 pp.

Fig. 22. Excellent yield of cassava in Caicedonia, Colombia. (Courtesy James M. Teri).

It has been estimated that one third of the population of tropical developing countries -- primarily the poor -- depend on root and tuber crops as staple food, with most of the production consumed directly by the producer (Goering 1979). The most important root and tuber crops in developing countries, in order of production, are: cassava, potatoes, sweet potatoes, yams, and aroids. If temperate production in developed countries is included, however, potatoes are the most important on a worldwide basis, with annual production twice that of all other edible root and tuber crops combined. Most potatoes are grown above 1000 m in the tropics and thus are not considered here. Cassava accounts for 63% of total root and tuber production in developing countries, potatoes 12%, sweet potatoes 11%, yams 11%, and aroids less than 3% (Goering 1979).

Root and tuber crops hardly enter into world commerce when compared to the cereals. Reasons include their high moisture content in comparison with the cereals and that they can seldom be stored for any appreciable time, except in the ground by delaying harvest. They are probably far more important as food in the tropics than available figures indicate. These crops have been largely ignored (with the exception of sweet potatoes which grow well in the tropics and subtropics) by research workers in tropical countries until quite recently. They have not only been neglected by researchers, but in many cases their use actually has been opposed by government officials. Social traditions influence the attitude towards root crops; for example, in Asia wealthy people eat rice whereas poor people eat root or tuber crops. In the Philippines I was told that one way to tell someone to "go to hell" is to say "go eat camotes" (sweet potatoes). During World War II, because of rice shortages, people were forced by hunger to eat crops such as sweet potatoes and cassava. They developed a dislike for them because they were forced to eat them. Properly prepared, cassava is delicious and is a food of choice for large segments of the population of Brazil, Colombia, Zaire and other tropical countries.

We should not be too surprised that these crops are not considered enthusiastically by people who have a history of thousands of years of eating cereals. As Coursey (1976) has pointed out:

"the neglect of, and opposition to the vegetatively propagated food crops, such as root crops of the tropical world, arises not entirely from rational grounds, but at least in part from culture-historical considerations".

Even a crop like the potato could not become a staple food without considerable opposition. When first introduced into Europe by the Spanish in the 16th century, potatoes were regarded as a curiosity, and it took a long time before they were considered a "good" food. For instance, their use was denounced in the Scottish pulpit on two accounts; first, potatoes were not mentioned in the Bible and were therefore not fit food for Christians and, second, they were thought to be the forbidden fruit spoken of in Genesis which caused Adam's fall (Peattie 1926). As a member of the Solanaceae, potatoes were considered to be a posionous crop like nightshade and every conceivable disease was attributed to them including leprosy and scrofula. It was declared that they would exhaust the soil, that they were an unnatural crop since potatoes produced from a seed tuber rather than true seeds, and that they grew under the ground. It was concluded that there was little doubt that potatoes were an invention of the devil.

Most of the culture of the western world relative to agricultural crops goes back some 10,000 years, and has come from the initial domestication of grasses which in time became modern grain crops. To quote Coursey (1976):

"Throughout the entire cultural history of modern Europe, therefore, grains, together with some leguminous pulses and animal products have been the major foods. The vegetable foods, the grains, and pulses, were all propagated from seed, and as a result the concept of "seed time and harvest" has entered deeply into the cultural patterns of all the civilizations that have evolved from the basis of food production originally developed in the "Neolithic revolution".

For instance, the motto of the Food and Agriculture Organization of the United Nations is Fiat Panis: "let there be bread". FAO also named its major journal "Ceres" after the classical diety of grain. Millions of Christians repeat daily "Give us this day our daily bread" in the Lord's Prayer. "Bread" means money in today's American English slang. There are many other indications of the "cereal mentality" that exists in European, North American, and in many Asian minds. Over and over again in developing countries you find statements that cassava is soil depleting, causes soil erosion, and is a "bad" crop. This type of thinking probably results, at least in part, from the "cereal mentality" bias.

The following is a partial list of some of these tropical root crops:

1. Cassava - *Manihot esculenta*
2. Sweet potatoes - *Ipomoea batatas*
3. Yam - *Dioscorea* spp.
   - Bitter Yam - *D. dumetorum*
   - Chinese Yam - *D. opposita*
   - Cush-Cush Yam - *D. trifida*
   - Greater Yam - *D. alata*
   - Intoxicating Yam - *D. hispida*
   - Lesser Yam - *D. esculenta*

Potato Yam - D. bulbifera
White Yam - D. rotundata
Yellow Yam - D. cayenensis

4. Taro (Dasheen, Eddoe) - Colocasia esculenta
5. Tania (Tanier, Yautia) - Xanthosoma sagittifolium
6. Arrachacha - Arracacia xanthorrhiza
7. Yam bean - Pachrrhizus erosus
8. Tiger nut (Chufa) - Cyperus esculentus
9. Arrowroot - Maranta arundinacea
10. East Indian Arrowroot - Tacca leontopetaloides
11. Edible canna (Queensland arrowroot) - Canna edulis
12. Potato - Solanum tuberosum
13. Ensete - Ensete ventricosum
14. Wingbean - Psophocarpus tetragonolobus

Further information can be found on tropical root crops and their diseases in the following references.

General References on Root and Tuber Crops and Their Diseases

Centre Overseas Pest Res. 1978. Pest Control in Tropical Root Crops. PANS Manual No. 4. London. 235 pp.

Cook, A. A. 1978. Diseases of Tropical and Subtropical Vegetables and Other Plants. Hafner, New York. 381 pp.

Jackson, G. V. H., and D. E. Gollifer. 1975. Disease and pest problems of taro (Colocasia esculents L. Schott) in the British Solomon Islands. Pest Articles News Sum. 21:45-53.

Kay, D. E. 1973. Root Crops. TPI Crop and Product Digest No. 2. Trop. Products Inst., London. 245 pp.

Laguna, I. G., L. G. Salazar, and J. F. Lopez. 1983. Fungal and bacterial diseases of aroids. Tech. Bull. 10, Tropical Research and Training Center, CATIE, Turrialba, Costa Rica. 30 pp.

Montaldo, A. 1972. Cultivo de Raices and Tuberculos Tropicales. Inst. Interamericano de Ciencias Agric. de la Org. Estados Am., Lima. 284 pp.

Onwuene, I. C. 1978. The Tropical Tuber Crops. Wiley, New York. 234 pp.

Parris, G. K. 1941. Diseases of taro in Hawai and their control. Univ. Hawaii Agric. Exp. Stn. Circ. 18. 29 pp.

Plucknett, D. L., R. S. de la Pena, and F. Obrero. 1970. Taro (Colocasia esculenta). Field Crop Abstracts 23:413-426.

Purseglove, J. W. 1968. Tropical Crops, Dicotyledons. Wiley, New York. 719 pp. 2 Vol.

Purseglove, J. W. 1972. Tropical Crops, Monocotyledons. Wiley, New York. 607 pp. 2 Vol.

Wang, J., and S. Higa. eds. 1983. Taro, a Review of Colocasia esculenta and its Potentials. Univ. Hawaii Press, Honolulu. 400 pp.

Literature Cited

Coursey, D. G. 1976. Some culture-historical determinants of tropical agricultural research priorities. Trop. Root and Tuber Crops Newsletter 9:4-13.

Goering, T. J. 1979. Tropical root crops and rural development. World Bank Staff Working Paper No. 324. World Bank, Washington, DC. 85 pp.

Peattie, D. C. 1926. Cargoes and Harvests. Appleton, New York. 311 pp.

CASSAVA

Cassava (Manihot esculenta) is seventh in terms of total world production of all crops and fourth in world production of all tropical crops, but few people in temperate regions recognize its importance. It is by far the most important of the tropical root crops and is estimated to be a significant source of calories for 450-500 million people in developing countries (Cock 1982). Global production in 1981 was 127 million tons produced on 14 million hectares (FAO 1981).

Cassava is a woody shrub (Euphorbiaceae), reaching 1-5 meters in height, which produces large, elongated starchy roots. It originated in the tropics of America where it has perhaps been cultivated for 4000 years (Cock 1982). Two centers of diversity have been identified, one in Mexico-Guatemala and one in northeastern Brazil. Cassava is most important in Africa and the Americas, but is becoming increasingly so in Asia. The plant was introduced into Africa in the 16th century from Brazil by the Portuguese during the slave trade, and was used as food for the slaves during the arduous ocean passages (Jones 1959). Following its introduction, the crop spread quickly across the African continent, evidently a pleasing addition to the diet.

Until the late 1960's little attention was given to cassava by agricultural scientists. This point can be illustrated with research on cassava in plant pathology. The Review of Applied Mycology (R.A.M.) is probably the best abstracting journal for information on plant diseases throughout the world. From 1945 to 1965 there were 119 references to cassava in the R.A.M. or six references per year. Carnations were on the same page and 350 references to carnations were noted during the same period or 17 references per year. In 1966 the R.A.M. had two references to cassava diseases and 17 to carnation diseases. There were 234 references to research on tobacco diseases in 1966 in the R.A.M. Mann Library of the College of Agriculture and Life Sciences, Cornell University, Ithaca, NY, is considered to be one of the largest agricultural libraries in the United States. In 1966 there were 12 books on cassava in Mann Library, 113 on carnations, and over 500 on tobacco. These figures illustrate how research (on a world-wide basis) has not given some of the important food crops in the tropics such as cassava the attention they need. I do not wish to

imply that research on plants such as carnations and tobacco is not valuable, but I do wish to make clear the point that research on some of man's basic food crops such as cassava was insignificant in comparison. Prior to 1971, it has been estimated that the total annual global expenditure for all cassava research was no more than $200,000 (Spurgeon 1975). However, lack of research on cassava is not due to lack of disease, insect, or agronomic problems.

Cassava has a wide variety of uses. However, both the roots and leaves contain cyanogenic glucosides, with a continuum from sweet to very bitter types. Sweet cassavas with low HCN may be eaten raw after peeling, but with bitter types the HCN must be removed by washing or cooking. South American Indians developed many processes to leach, ferment, and heat cassava to remove HCN. Roots may be boiled or baked, and many methods are used to prepare flour or meal from cassava. A dried or fried meal from fermented pulp called garri is produced in West Africa. Cassava starch is used for food and various industrial purposes. The leaves, which have a high protein content, are eaten as a pot-herb in some areas of Africa.

Some nutritionists have been highly critical of the use of cassava because of its low protein content, for roots of most varieties are only about 1.2% protein. Long term, heavy consumption of poorly processed cassava with inadequate levels of protein and iodine can lead to kwashiorkor, cretinism, mental retardation, and goiter. However, it is seldom by choice that people eat only cassava. It is generally supplemented with protein from plants, fish, or animals. Few foods, eaten alone, constitute an acceptable diet. Common sense dictates that cassava should be recognized and utilized for what it is, i.e. one of the most efficient crops in the tropics for producing calories (de Vries et al. 1967), and that it should be supplemented with protein from other sources in human diets. According to Cock (1982) cassava is the fourth most important source of calories for human food in the tropics after rice, maize, and sugarcane.

Cassava has a great unrealized potential for feeding tropical peoples because of its wide range of adaptability (from the hot, humid tropics to the border of temperate zones), resistance to drought, tolerance of poor soils, relative ease of cultivation, and potentially high yields. Considerable quantities of cassava are grown as a subsistence crop, often by farmers who practice slash and burn or shifting agriculture. In the tropics it can usually be planted and harvested at any season and can be left in the ground for periods up to four years which makes it useful as a security against famine (Jones 1959). Four-year-old cassava would be fibrous and not highly palatable, but still edible. Commercial yields of 30-40 t/ha are being obtained by farmers on good soils in a 7,000 hectare region near Caicedonia in Colombia. Yields of over 80 t/ha (in 12 months) have been obtained experimentally (Cock 1982). Worldwide, cassava yields are generally low (7.7 t/ha in Africa, 9.5 t/ha in Asia and 14 t/ha in Latin America). The roots must generally be used 2-3 days after harvest unless dried, in which case storage can be for long periods. The classic paper of de Vries et al. (1967) clearly demonstrates the high yield potential of cassava. They conclude that the tropical root crops have a far higher potential production than grain crops in the tropics even though they are virtually unimproved by modern agricultural technology.

The earliest cassava varieties mature in six months, but some varieties are harvested after 18 months. It grows in areas with rainfall as low as 500 mm or as high as 5000 mm. Cassava is propagated by stem cuttings generally 25-30 cm in length. It is grown within the 30° parallels and up to 2000 meters near the equator.

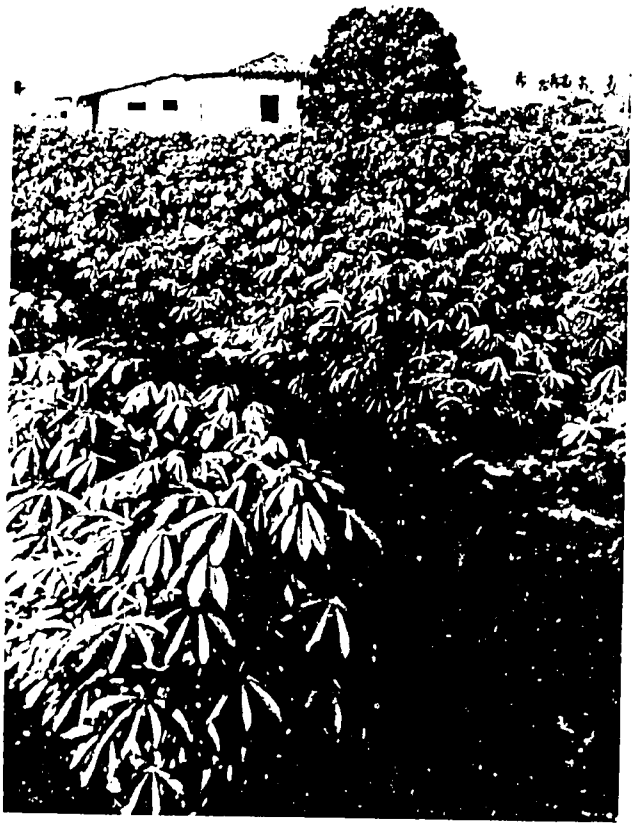


Fig. 23. Young cassava plants.

A major effort to improve cassava by breeding began in the late 1960's with the founding of CIAT (International Center for Tropical Agriculture) and IITA (International Institute for Tropical Agriculture). Both international centers have strong cassava breeding programs. Outstanding work was previously done in southern Brazil to develop high-yielding lines and in East Africa to develop varieties resistant to African cassava mosaic disease. A rich diversity of cassava germplasm is found in the Americas, and CIAT's cassava collection now consists of over 3500 clones.

Until recently, there has been a commonly held belief that cassava is a "rustic" crop and highly resistant to disease and insect attack. However, recent studies at CIAT, IITA, and national institutions show that diseases and insects seriously reduce cassava yields and are major limiting factors in growing cassava. Information on cassava diseases, when compared to that for major crops, was negligible until the early 1970's.

The most important disease of cassava in Africa is African cassava mosaic, which is transmitted by whiteflies (Bemisia spp.). Most plants in Africa are infected, and serious yield losses are common. The breeding program at IITA utilized Manihot glaziovii as a source of resistance to African cassava mosaic, with over 100,000 seedlings being screened annually for resistance. In 1972 and 1976 cassava mosaic workshops were held to assess the current status of knowledge on the disease.

Cassava bacterial blight is probably the single most important cassava disease worldwide. The pathogen can simultaneously cause leaf spotting and wilting, shoot dieback, and vascular discoloration. Lozano and Sequeira (1974) showed that the disease is caused by the bacterium Xanthomonas campestris pv. manihotis. Primary infection is most frequently initiated by infected planting material, although wind-driven rain, tools, and insects also transmit the pathogen. Studies at CIAT developed control methods for bacterial blight that gave farmers satisfactory disease control with sanitation, pruning, and disease-free propagating material. Methods of rapidly producing large quantities of disease-free planting material were developed at CIAT. Varieties of cassava resistant to X. campestris pv. manihotis have been developed both at CIAT and IITA (Hahn et al. 1980). IITA's cassava lines are tested in several African countries for resistance to African cassava mosaic and cassava bacterial blight, and in these tests, several IITA clones outyielded local standard varieties 2 to 18 times, primarily because of their disease resistance.

Other diseases being studied are the Cercospora leaf spots (Cercospora henningsii, C. caribaea, and C. viscosae), anthracnose (Colletotrichum spp.), the superelongation disease (Sphaceloma manihoticola), Phoma leaf spot and a number of serious root rots. Recent evaluation of the superelongation disease indicates that it may be the most serious threat to cassava in the Americas. The cassava germplasm collections are being screened for resistance to all of these pathogens, and promising resistance has been found. The agents causing deterioration of cassava roots before and after harvest are also studied.

At present almost no pesticides are used on cassava. CIAT's pathologists and entomologists are studying the formulation of a pest management system to keep pesticide use to a minimum on cassava by combining varietal resistance, cultural practices, biological control, insect attractants, and other methods.

Chevangeon (1956) has listed 73 species of fungi that have been found associated with cassava. In addition, bacterial diseases, several viral diseases, nematodes, and various physiological disorders are reported in the literature. Lozano and Booth (1974) and the proceedings of a cassava protection workshop (Brekelbaum et al. 1977) have summarized the available information on cassava diseases. The CIAT Documentation Center in Cali, Colombia abstracts worldwide all the literature on cassava. These published abstracts are available annually at a nominal cost.

Fig. 24. Stem cankers caused by Sphaceloma manihoticola.

The following are selected references on cassava diseases that are useful for identification and control.

Selected References on Cassava Diseases

Brekelbaum, T., A. Bellotti, and J. C. Lozano eds. 1977. Proceedings Cassava Protection Workshop. Centro Int. Agric. Trop., Cali, Colombia. 244 pp.

Chevangeon, J. 1956. Les maladies cryptogamiquies du manioc en Afrique occidentale. Encycl. Mycologique 28:1-205.

Lozano, J. C., and R. H. Booth. 1974. Diseases of cassava (Manihot esculenta Crantz). Pest Articles News Sum. 20:30-54.

Lozano, J. C., A. Bellotti, A. van Schoonhoven, R. Howeler, J. Doll, D. Howell, and T. Bates. 1976. Field Problems in Cassava. Series GE-16. Centro Int. Agric. Trop., Cali, Colombia. 126 pp.

McSorley, R., S. K. O'Hair, and J. L. Parrado. 1983. Nematodes of cassava, Manihot esculenta Crantz. Nematropica 13:261.

Literature Cited

Cock, J. H. 1982. Cassava: a basic energy source for the tropics. Science 218:755-762.

de Vries, C. A., J. D. Ferwerda, and J. Flach. 1967. Choice of food crops in relation to actual and potential production in the tropics. Neth. J. Agric. Sci. 15:241-248.

Food Agriculture Organization. 1981. FAO Production Yearbook. Vol. 35. FAO, Rome. 306 pp.

Hahn, S. K., A. K. Howland, and E. R. Terry. 1980. Correlated resistance of cassava to mosaic and bacterial blight diseases. Euphytica 29:305-311.

Jones, W. O. 1959. Manioc in Africa. Stanford Univ. Press, Stanford, CA. 315 pp.

Lozano, J. C., and L. Sequeira. 1974. Bacterial blight of cassava in Colombia: etiology. Phytopathology 64:74-82.

Spurgeon, D. 1975. Cassava: the potential is enormous. Int. Develop. Res. Centre Rept. 4(4):1-5.

AFRICAN CASSAVA MOSAIC DISEASE

Disease Names: The disease is most commonly called African cassava mosaic disease (ACMD) to distinguish it from American cassava common mosaic disease which is caused by a virus distinct from the ACMD pathogen. It also has been called cassava mosaic disease and curly leaf disease.

Causal Organism: A geminivirus

Cassava was introduced into West Africa from Brazil by the Portuguese perhaps as early as the 16th century (Jones 1959). Since cassava originated in the Americas, and the ACMD pathogen does not occur in the Americas, the virus probably originated from a native host species of the causal agent in Africa, but there is no proof for this. A mosaic disease of cassava was first reported by Warburg in 1894 in East Africa under the name Kräuselkrankheit, and transmission by whiteflies (*Bemisia* spp.) was first reported by Ghesquiere in 1932 according to Storey and Nichols (1938).

The disease is common wherever cassava is grown in Africa and the adjacent islands. It is unclear whether the disease reported from India (Menon and Raychaudhuri 1970) is the same as ACMD. Lozano and Terry (1977) suggest that ACMD is limited to Africa. At CIAT (International Center for Tropical Agriculture) in Colombia six different types of mosaic diseases of cassava have been found, some disseminated by whiteflies (C. Lozano - personal communication). The evidence indicates that a viral pathogen causes ACMD. Walter (1980), Bock (1982), and Adejare and Coutts (1982) suggest that ACMD is caused by a geminivirus and more recently Bock and Woods (1983) report successful transmission of gemini particles to cassava. Kitajima and Costa (1964) found elongated particles (rods) associated with American cassava common mosaic disease from Brazil and the cassava brown streak disease from Africa, but none were found associated with ACMD. The American cassava common mosaic virus was easily transmitted by mechanical inoculation, but the ACMD pathogen was not. Furthermore, the American cassava common mosaic pathogen has been identified and characterized as a virus (Costa and Kitajima 1972). ACMD had been reported to be caused by a virus based upon symptomatology and transmission by whiteflies, but the causal agent of ACMD has only recently been positively identified. Galvez and Kitajima (personal communication) found no virus-like particles or mycoplasma-like bodies in situ in ultrathin sections from infected leaf tissues from cassava collected in Nigeria. However, with electron microscopy observations, Plasvic-Banjac and Maramorosch (1973) found inclusions in cells of leaves infected with ACMD which they suggested might be virus particles. Subsequently, Murant et al. (1973) were unable to find any virus-like or mycoplasma-like particles that could be the causal agent of ACMD and suggested that the particles found by Plasvic-Benjac and Maramorosch (1973) were normal components of healthy plants. Bock and Guthrie (1976), using cassava plants infected with ACMD and brown streak virus, were able (by sap transmission) to infect *Nicotiana clevlandii*. From the infected *N. clevlandii* they isolated two similar paired isometric particles. The particles were serologically related, but distinct viruses. They were unable to infect cassava with either, and thus their identity was not clear.

Later Bock and Guthrie (1978) reported mechanical transmission of ACMD from cassava to cassava. This finding, plus whitefly transmission, suggested that the causal agent was a virus and not a mycoplasma. Walter (1980) and

Fig. 25. African cassava mosaic disease in Nigeria.

Adejare and Coutts (1982) isolated particles from ACMD infected material from West Africa with similar physical and chemical properties to the Kenyan material of Bock and Guthrie (1976). Although their host range was slightly different from that of the Kenyan isolate, it appears that they were probably all working with the same virus. Finally, Bock and Woods (1983) have successfully transmitted partially purified geminivirus back to cassava mechanically. Thus, the causal organism of ACMD is a geminivirus.

Importance

On a worldwide scale the most important single disease of cassava is probably ACMD. Cassava is a major food crop in Africa, and the disease affects the food supply of millions of Africans. Padwick (1956) brought together the available information on losses due to ACMD, and estimated that in Africa there is a yearly loss in yield due to the disease of 11%. High percentages of infection are common in the tropical cassava-growing areas of Africa. Beck and Chant (1958) cite authors reporting yield losses of 20 to 95% on different cassava cultivars. Bock et al. (1977) reported yield losses of 70 and 86% in two cultivars in Kenya. Jennings (1970) cites 8 authors who have studied losses due to ACMD and brown streak, and the losses ranged from 14.5 - 83%. Hahn and Howland (1972) reported that losses of 30-80% in yield are common. Terry and Hahn (1980) found an ACMD infected susceptible clone had a 69% reduction in yield 7 months after planting, whereas a resistant clone had only a 32% loss. Bock and Guthrie (1978) report an average crop loss of 70% in Kenya, although Seif (1982) reported yield reductions in Kenya of 24-75% on different clones. In Africa about 48 million tons of cassava are produced annually. Extrapolating from the above and the loss figures reported, it is obvious that the annual loss due to ACMD in Africa must be a staggering figure.

Symptoms

Numerous descriptions and illustrations of the symptoms of ACMD are given in the literature (Terry 1976, Storey and Nichols 1938, Bock and Guthrie 1977, Costa and Kitajima 1972). The symptoms are characteristic of a mosaic disease in that there is chlorosis of some areas of the leaf and not of others. Leaves are often twisted and distorted, and stunting of both leaves and the plant is common. Symptoms vary widely depending on factors such as variety, environmental factors, stage of plant growth when infected, and plant age. Symptoms may vary from a faint chlorotic mottle to a severe mosaic with severe distortion and stunting of the leaf. Terry (1976) has published a scoring system for evaluation of resistance with five severity classes.

Host Plants

Cassava (Manihot esculenta) clones vary in their degree of resistance to ACMD, but none are known to be immune. In the field only cassava and rarely Ceara rubber (Manihot glazovii) are infected.

Fig. 26. White flies on cassava leaf (vector of African common cassava mosaic). (Courtesy J. Carlos Lozano T.).

Vectors and Transmission

The causal agent of ACMD is transmitted by grafting (Storey and Nichols 1938), dodder (Dubern 1972), and whiteflies (Bemisia tabaci). Until recently, workers were unable to transmit the pathogen by mechanical means (Storey and Nichols 1938), but recently Bock and Guthrie (1978) and Bock and Woods (1983) report success. No evidence for transmission through the soil or true seed was found in the literature.

Man's dispersal of the causal agent in cuttings (pieces of stem) from diseased plants is probably of greater importance than whitefly transmission (Bock et al. 1977). Bock and Guthrie (1978), using ACMD-free plants, found a rate of reinfestation by whiteflies around 2% per year in Kenya. This means that there is a slow rate of transmission by whiteflies in the field, at least under Kenyan conditions. However, Leuschner (1978), working in Nigeria, found a close relationship between B. tabaci populations and ACMD incidence.

The studies of Storey and Nichols (1938) and Chant (1958) on whitefly transmission are still an important source of information. Bemisia tabaci is the only known vector (Leuschner 1978). Storey and Nichols (1938) found that the insect can maintain itself on mature leaves, but can inoculate successfully only immature ones. They gave precise procedures for successful inoculation of cassava with whiteflies. Chant (1958) found that whiteflies need to feed for at least 4 hours on young leaves to acquire the causal agent, and require another 4 hours to become infective. Once they became infective they could infect a healthy plant by feeding as little as 15 minutes. Adult whiteflies also could continue to infect for 48 hours after their initial feeding. A single whitefly could cause infection, although successful transmission increased when larger numbers were used. Storey and Nichols (1938) found that symptoms of mosaic appeared in new terminal growth 12-20 days after inoculation. After the virus is inoculated into immature leaves by whiteflies it does not pass out of the leaf for 8 days. Once the virus enters the stem it rapidly moves to the base of the stem and then slowly moves to side branches of the stem or to other stems arising from the same cutting. Cuttings of branches from infected stems may be free of the virus (Storey and Nichols 1938). Hahn et al. (1980b) report that B. tabaci populations are influenced by rainfall, temperature, and light intensity. As rainfall increases, whitefly populations increase. The highest vector activity was found at 27-32 C and under high light intensities.

Chant and Beck (1959) compared healthy and diseased leaves of cassava and found that the palisade tissue in chlorotic lesions is undifferentiated, there are fewer chloroplasts, and chloroplasts are abnormal in shape. The phloem in

the midrib of infected leaves is restricted to small bundles. No necrosis of sieve tubes, and no inclusion bodies were found.

Control

Strict quarantine measures should be enforced to keep ACMD out of presently uninfected areas of Latin America and Asia. It is also important to determine if the Asian cassava diseases reported in India and Indonesia are the same as ACMD. An important consideration is how to move improved lines of cassava from country to country without bringing ACMD with them. Chant (1959) studied the inactivation of the causal agent of ACMD by heat, and found that it was inactivated by growing infected cuttings at 35-39°C for periods of 28-42 days. Kaiser (1982), however, found a hot air treatment of 37°C for 42-96 days caused temporary remission of symptoms in most plants, and only 1 of 129 plants treated was free of disease. However, he was able to eradicate the ACMD virus from some tip cuttings treated at 37°C for 87-105 days. Gamborg and Kartha (1976) and Kaiser and Teemba (1979) report the successful elimination of the ACMD pathogen by culturing apical meristems. This could be a useful technique in eliminating ACMD from valuable cultivars where facilities for producing "clean" stakes for farmers are available.

Most of the emphasis in Africa has been on breeding for resistance. However, various cultural methods of control are also important. Storey (1936) reported that disease free cassava was grown in the highlands of Kenya at 914 meter elevation, and farmers in the lowlands often get their seed from these areas. Bock et al. (1977) in Kenya found that control of ACMD could be obtained by using mosaic-free cuttings, roguing infected plants, and by isolating clean plots from infected plants. They suggested that under Kenyan conditions man was a more efficient vector of ACMD through use of infected cuttings than were whiteflies. Bock and Guthrie (1982) controlled ACMD by using healthy planting material from plots established and kept free of ACMD by inspection and roguing. They found the rate of spread of ACMD into mosaic-free plots over a five-year period less than 2%. The environmental factors that influence the spread of ACMD are not well understood. Symptom expression is more severe at low temperatures. Vector activity is highest during rainy periods (Jennings, 1960).

Jennings (1976), Bock and Guthrie (1978), and Hahn et al. (1980b) have described the major effort begun in Tanzania, East Africa, in 1935 by Storey and his colleagues to breed cassava resistant to ACMD. Their first step was to assemble a world-wide collection of cassava cultivars (*M. esculenta*) and test them for resistance. Only moderate levels of resistance were found. Subsequently an attempt was made to utilize *Manihot glaziovii* (Ceara rubber) and several other *Manihot* species as a source of resistance. *M. glaziovii* gave the highest levels of resistance (Jennings 1960, 1976). It was necessary to backcross the interspecific hybrid to cassava in order to restore root quality. When these lines were tested they were found to provide adequate resistance for many coastal areas, but in two sites resistance was not adequate. Jennings (1960) found that the interspecific hybrids were not immune, but that resistance may be due to the ability of the plant to restrict the causal agent to basal parts of the plant where it does no damage. Bock and Guthrie (1978) discuss the impact of Storey's breeding program on cassava in Kenya.

Jennings (1960) found that resistant plants were often infected without showing symptoms. Plants could be induced to express symptoms if growth was checked by removing leaves. Jennings noted that the results from field trials for resistance could be influenced by environment, virus strains, soil factors,

and vector population. Trials for evaluation of resistance planted at different elevations in Kenya gave different results.

It is interesting to study the definitions Storey used relative to ACMD resistance. Storey (1936) describes "resistance" as a term "used in relation to liability to contract infection; that is, under equal conditions, a plant of high resistance contracts the disease less freely than one of low resistance". Tolerance Storey describes as "the power to avoid the ill-effects of the virus, and this is often quite independent of the plant's resistance to infection. A tolerant variety yields well though diseased". Storey believed the ideal solution for control of ACMD would be an immune variety.

Jameson (1964) in Uganda in a program looking for resistance to cassava mosaic decided to select for "resistance" rather than "tolerance". He pointed out that although "tolerant" varieties may produce an acceptable crop, they may act as reservoirs of infection and thus infect other varieties in a given region. By selecting "resistant" plants it was hoped that infection could be reduced to a level where periodic roguing could keep the crop free of disease. Jameson (1964) described the rapid virus spread in Uganda during droughts and other times of scarcity. For example, when wartime scarcity of shipping made it important to conserve local food supplies, people ate the best of the crop and used the remaining (diseased) plants for propagation. They were essentially selecting virus-infected plants for seed. Jameson also reported that in 1943-44 in the Teso District of Uganda a serious shortage of food developed due to drought and bad harvests. A survey made in the district showed that the local cassava variety (Nyumbezunga) was infected almost 100% with ACMD and that yields were less than 1/2 t/ha. Resistant varieties were introduced, and yield increased 7 fold by changing from susceptible Nyumbezunga to the resistant Aipin Valenca.

According to Hahn and Howland (1972), IITA's (International Institute of Tropical Agriculture) breeding program for resistance to ACMD began in 1971 using material from the East African program initiated by Storey and Nichols and continued by Jennings and others. The IITA program used M. glaziovii as its source of resistance to ACMD, and in 1975 over 100,000 seedlings from more than 1200 families were screened for ACMD resistance (IITA 1976). Resistance to ACMD has been successfully incorporated into high-yielding lines with good quality. Resistance appears to be inherited in a quantitative manner with additive effects (Hahn et al. 1980). Most fortuitous is the linkage of resistance to ACMD and cassava bacterial blight (X. campestris pv. manihotis) (Hahn et al. 1980a, Jennings 1978). Furthermore, the resistance to both diseases appears to be polygenic.

IITA-improved clones with resistance to ACMD and cassava bacterial blight are tested in many different locations in Africa and India which include a wide range of environmental conditions (Hahn et al. 1980b). Several IITA-improved lines give yields up to 30 t/ha (Leuschner 1978) and have outyielded local standard varieties by 2 to 18 times (IITA 1976), primarily due to their disease resistance.

Hahn et al. (1980b) report that the same number of whiteflies are found on lines resistant and susceptible to ACMD; thus it is unlikely that ACMD resistance is due to vector resistance. However, the rate of infection differs among lines, and they conclude:

> "rate of resistance to CMD appears to be resistance to vector infection and to spread of the pathogen within the plant".

Storey and Nichols (1938) describe mild and severe strains of the virus distinguishable on the basis of symptom severity. Terry and Jennings (1976) discuss the literature on "variants" of ACMD and present a proposal to categorize them. Referring to the IITA clones Leuschner (1978) suggests that:

> "The chances of resistance breaking down due to new disease strains is not very high as we are dealing with multigenic horizontal resistance, but this aspect should not be overlooked".

The most rational control of ACMD would be a combination of host plant resistance (where available in locally adapted and accepted varieties) with cultural methods of control as suggested by Bock et al. (1977) and Bock and Guthrie (1978). They suggested the selection and use of disease-free planting material, strict roguing of infected plants, and isolating healthy plantings from diseased. The epidemiology of the disease needs further study, since the recommendations of Bock et al. (1977) for Kenya might not be appropriate for West Africa because of differences in environmental factors.

Literature Cited

Adejare, G. O., and R. H. A. Coutts. 1982. The isolation and characterization of a virus from Nigerian cassava plants affected by the cassava mosaic disease and attempted transmission of the disease. Phytopathol. Z. 103: 198-210.

Beck, B. D. A., and S. R. Chant. 1958. A preliminary investigation on the effect of mosaic virus on Manihot utilissima Phol. in Nigeria. Trop. Agric. (Trinidad) 35:59-64.

Bock, K. R. 1982. Geminivirus diseases on tropical crops. Plant Dis. 66:266-270.

Bock, K. R., and E. J. Guthrie. 1976. Recent advances in research on cassava viruses in East Africa. pp. 11-16. In Nestle, B. L. ed., African Cassava Mosaic. E. Afr. For. Res. Organ. and Int. Develop. Res. Centre. 071-e, Ottawa, Canada. 48 pp.

Bock, K. R., and E. J. Guthrie. 1977. African mosaic disease in Kenya. pp. 41-44. In Brekelbaum, T., A. Bellotti and J. C. Lozano. eds. Proc. Cassava Protection Workshop. IDRC Series CE-14. Centro Int. Agric. Trop., Cali, Colombia. 244 pp.

Bock, K. R., E. J. Guthrie, and A. A. Seif. 1977. Field control of cassava mosaic in coast province, Kenya. pp. 160-163. In Cock, J., R. MacIntyre and M. Graham. eds. Proc. 4th Symp. Int. Soc. for Trop. Root Crops. Centro Int. Agric. Trop. and Development Res. Centre, Ottawa, IDRC 080e. 277 pp.

Bock, K. R., and E. J. Guthrie. 1978. Transmission of African cassava mosaic by mechanical inoculation. Plant Dis. Rep. 62:580-581.

Bock, K. R., and E. J. Guthrie. 1982. Control of cassava mosaic disease in Kenya. Trop. Pest Management 28:219-222.

Bock, K. R., and R. D. Woods. 1983. Etiology of African cassava mosaic disease. Plant Dis. 67:994-995.

Chant, S. R. 1958. Studies on the transmission of cassava mosaic virus by Bemisia spp. (Aleyrodidae). Ann. Appl. Biol. 46:210-215.

Chant, S. R. 1959. A note on the inactivation of mosaic virus in cassava (Manihot utilissima Pohl.) by heat treatment. Emp. J. Exp. Agric. 27:55-58.

Chant, S. R., and B. D. A. Beck. 1959. The effect of cassava mosaic virus on the anatomy of cassava leaves. Trop. Agric. (Trinidad) 36:231-236.

Costa, A. S., and E. W. Kitajima. 1972. Cassava common mosaic virus. Descriptions of Plant Viruses, Commonw. Mycol. Inst., No. 90. Kew, Surrey.

Dubern, J. 1972. A contribution to the study of African cassava mosaic disease. pp. 13-17. In Proceedings Cassava Mosaic Workshop. Int. Inst. Trop. Agric. Ibadan, Nigeria. 48 pp.

Gamborg, O. L., and K. K. Kartha. 1976. In vitro techniques in the control of cassava mosaic virus. pp. 30-35. In B. L. Nestel ed. African Cassava Mosaic. E. Afr. Agric. For. Organ. and Int. Develop. Res. Centre. 071-e. Ottawa. 48 pp.

Hahn, S. K., and A. K. Howland. 1972. Breeding for resistance to cassava mosaic. pp. 37-39. In Proceedings Cassava Mosaic Workshop. Int. Inst. Trop. Agric., Ibadan, Nigeria.

Hahn, S. K., A. K. Howland, and E. R. Terry. 1980a. Correlated resistance of cassava to mosaic and bacterial blight diseases. Euphytica 29:305-311.

Hahn, S. K., E. R. Terry, and K. Leuschner. 1980b. Breeding cassava for resistance to cassava mosaic disease. Euphytica 29:673-683.

International Institute of Tropical Agriculture. 1976. 1975 Annual Report, Ibadan, Nigeria. 219 pp.

Jameson, J. D. 1964. Cassava mosaic disease in Uganda. E. Afr. Agric. and For. J. 29:208-213.

Jennings, D. L. 1960. Observations on virus diseases of cassava in resistant and susceptible varieties. I. Mosaic diseases. Emp. J. Exp. Agric. 28:23-34.

Jennings, D. L. 1970. Cassava in East Africa, pp. 64-65. In Tropical Root and Tuber Crops Tomorrow. Vol. 1. Proc. 2nd Int. Symposium on Tropical Root and Tuber Crops. Univ. of Hawaii, Honolulu. 171 pp.

Jennings, D. L. 1976. Breeding for resistance to African cassava mosaic disease: progress and prospects, pp. 39-44. In B. L. Nestel. ed. African Cassava Mosaic. E. Afr. Agric. For. Organ. & Int. Develop. Res. Centre. 48 pp.

Jennings, D. L. 1978. Inheritance of linked resistances to African cassava mosaic and bacterial blight diseases, pp. 45-50 In Brekelbaum, T., A. Bellotti, and J. C. Lozano. eds. Proc. Cassava Protection Workshop. Series CE14. Centro Int. Agric. Trop., Cali, Colombia. 244 pp.

Jones, W. O. 1959. Manioc in Africa. Stanford Univ. Press, Stanford, CA. 315 pp.

Kaiser, W. J. 1982. Heat therapy of cassava infected with African cassava mosaic disease. Plant Dis. 66:475-477.

Kaiser, W. J., and L. Teemba. 1979. Use of tissue culture and thermotherapy to free East African cassava cultivars of African cassava mosaic and cassava brown streak diseases. Plant Dis. Rep. 63:780-784.

Kitajima, E. W., and A. S. Costa. 1964. Elongated particles found associated with cassava brown streak. E. Afr. Agric. For. J. 30:28-30.

Leuschner, K. 1978. Whiteflies: biology and transmission of African mosaic disease. pp. 51-58. In Brekelbaum, T., A. Bellottii, and J. C. Lozano. eds. Proc. Cassava Protection Workshop. Int. Develop. Res. Centre Ser. CE-14. Centro Int. Agric. Trop., Cali, Colombia. 244 pp.

Lozano, J. C., and E. R. Terry. 1977. Cassava diseases and their control. pp. 156-160. In Cock, J., R. MacIntyre and M. Graham. eds. Proc. 4th Symp. Intl. Soc. Tropical Root Crops. Centro Int. Agric. Trop. and Int. Develop. Res. Centre, Ottawa. 277 pp. IDRC 080e.

Menon, M. R., and S. P. Raychaudhuri. 1970. Cucumber: A herbaceous host of cassava mosaic virus. Plant Dis. Rep. 54:34-35.

Murant, A. F., I. M. Roberts, and D. L. Jennings. 1973. Electron microscopy of cassava leaves with African cassava mosaic. 3rd Int. Symp. Trop. Root Crops. Int. Inst. Trop. Agric., Ibadan, Nigeria.

Padwick, G. W. 1956. Losses caused by plant diseases in the colonies. Phytopathol. Papers No. 1. Commonw. Mycol. Inst., Kew, Surrey. 60 pp.

Plavsic-Banjac, B., and K. Maramorosch. 1973. Cassava mosaic virus. Phytopathology 63:206. (Abstr.)

Seif, A. A. 1982. Effect of cassava mosaic virus on yield of cassava. Plant Dis. 66:661-662.

Storey, H. H. 1936. Virus diseases of East African plants: VI. A progress report on the disease of cassava. E. Afr. Agric. J. 2:34-39.

Storey, H. H., and R. F. W. Nichols. 1938. Studies of the mosaic diseases of cassava. Ann. Appl. Biol. 25:790-806.

Terry, E. R. 1976. Description and evaluation of cassava mosaic disease in Africa. pp. 53-54. In: Terry, E. R. and R. MacIntyre. eds. Proc. Int. Exchange and Testing of Cassava Germplasm in Africa. Int. Develop. Res. Centre. and Int. Inst. Trop. Agric., Ibadan, Nigeria. 59 pp.

Terry, E. R., and S. K. Hahn. 1980. The effect of cassava mosaic disease on growth and yield of a local and an improved variety of cassava. Trop. Pest Management 26:34-37.

Walter, B. 1980. Isolation and purification of a virus transmitted from mosaic-diseased cassava in the Ivory Coast. Plant Dis. 64:1040-1042.

SWEET POTATOES

Sweet potatoes (Ipomoea batatas) are one of the world's major food crops with a global production in 1981 of 146 million metric tons (FAO 1981). Worldwide, only wheat, maize, rice, potatoes, and barley outproduce them. Sweet potatoes are grown extensively in the tropics, but although 15 million hectares are grown in the world, only 2.8 million are grown in developing countries (Cummings 1976). A member of the Convolvulaceae family, I. batatas, is a perennial vine, but is treated as an annual when cultivated. Because of its highly perishable nature, most sweet potatoes are consumed in the country where they are produced, and very few enter international trade. There is probably more information available on sweet potatoes than on any of the other tropical root and tuber crops.

Sweet potatoes originated in tropical America, but were also found growing in Polynesia and New Zealand by European explorers. Considerable controversy exists as to whether they originated in Polynesia or were introduced there from South America. One theory suggests that they were carried on rafts from Peru to Polynesia as Heyerdahl (1963) showed was feasible with his voyage to Tahiti from Peru. Purseglove (1968) believes that sweet potato capsules (which float in water and are almost impervious to water) were carried by ocean currents from South America to Polynesia. Yen (1974) has made an exhaustive study of these questions, but came to no definite conclusions. Yen's book is a fascinating account of the cultural uses and ethnobotany of the sweet potato in the Pacific. I. batatas was taken to Spain by Columbus in 1492 and since that time has spread rapidly throughout the world.

The sweet potato is generally grown in temperate areas in highly intensive, high-input systems of agriculture, whereas in tropical countries it is more commonly a subsistence crop. I. batatas is usually planted on ridges or mounds. In the tropics apical stem cuttings, 30-45 cm long, are usually used for propagation. Harvesting is after 3-8 months (depending on the variety grown), and yields range up to 20 t/ha in temperate countries, but yields of 7-13 t/ha are common, in most tropical countries (Villareal 1982).

I. batatas grows in a wide variety of soils in tropical to warm temperate areas from 32° S to 40° N (Purseglove 1968, Kay 1973) and need a frost-free period of 4-5 months. In most tropical countries sweet potatoes are primarily grown for their edible roots which are boiled, baked, fried, and candied. Root use as a livestock feed or for industrial extraction or preparation of starch, glucose, syrup, and alcohol is important in some countries. The vines also can be fed to livestock as fodder. The tender top leaves are commonly used as a pot-herb in the Philippines, Indonesia, and some African countries. Sweet potato varieties vary greatly in many characters such as skin and flesh color, root size and shape, starch, sugar and vitamin A content, maturity, depth of rooting, and resistance to diseases and insects. Numerous programs for improvement through breeding are found worldwide (Harmon et al. 1970, Fujise 1970, AVRDC 1979, IITA 1978).

Diseases are of great importance to sweet potato growers. Cook (1953) estimated that in the United States 20-40% of the sweet potato crop is destroyed in the field, in storage, or in transit to market. Important sweet potato diseases have been reviewed by Martin (1970), Morrison (1970), Nielsen (1970), and Terry (1981). Important virus or virus-like diseases are numerous including internal cork, feathery mottle, leaf spot, mosaic, witches broom (mycoplasma), mild mottle, and others, but feathery mottle and mild mottle are the only characterized viruses. Considerable work on the etiology of sweet potato

virus diseases is needed. Fungus diseases of major importance worldwide are stem rot (Fusarium oxysporum f. sp. batatas), black rot (Ceratocystis fimbriata), scurf (Monilochaetes infuscans), and foot rot (Plenodomus destruens). Many other fungi attack sweet potatoes, especially in storage (Moyer 1982). Soil rot or pox is a destructive disease in the United States caused by the soil-borne actinomycete Streptomyces ipomoea. Nematodes are also serious pathogens of I. batatas (Morrison 1970).

Selected References on Sweet Potato Diseases

Arene, O. B., and A. O. Nwankiti. 1978. Sweet potato diseases in Nigeria. Pest Articles News Sum. 24(3):294-305.

Centre for Overseas Pest Res. 1978. Pest Control in Tropical Root Crops. PANS Manual No. 4. London. 235 pp.

Edmond, J. B., and G. R. Ammerman. 1971. Sweet Potatoes: Production, Processing, Marketing. Avi, Westport, CT. 334 pp.

Harter, L. L., and J. L. Weimer. 1929. A monographic study of sweet-potato diseases and their control. U.S. Dept. Agric. Tech. Bull. No. 99. 118 pp.

Hildebrand, E. M. 1959. Sweet potato diseases. Farmer's Bull. No. 1059. U.S. Dept. Agric., Washington, DC. 28 pp.

Martin, W. J. 1970. Virus diseases. pp. 49-55. In Hernandez, T. P. ed. Thirty years of cooperative sweet potato research, 1939-1969. Southern Coop. Serv. Bull. No. 159, Louisiana State Univ., Baton Rouge.

Morrison, L. S. 1970. Nematode diseases. pp. 56-59. In Hernandez, T. P. ed., Thirty years of cooperative sweet potato research, 1939-1969. Southern Coop. Serv. Bull. No. 159, Louisiana State Univ., Baton Rouge.

Moyer, J. W. 1982. Postharvest disease management for sweet potatoes. pp. 177-184. In Villareal, R. L. and T. D. Griggs. eds. Sweet Potato. Asian Vegetable Res. Develop. Center, Shanhua, Taiwan. 481 pp.

Nielsen, L. W. 1970. Fungus and miscellaneous diseases. pp. 60-71. In Hernandez, T. P. ed. Thirty years of cooperative sweet potato research, 1939-1969. Southern Coop. Serv. Bull. No. 159, Louisiana State Univ., Baton Rouge.

Terry, E. R. 1981. A review of major pests and diseases of sweet potato with special reference to plant exchange and introduction. Afr. J. Plant Protec. 1:117-125.

Steinbauer, C. E., and L. J. Kushman. 1971. Sweet potato culture and diseases. U.S. Dept. Agric., Agric. Res. Serv., Agric. Handbook No. 388. 74 pp.

Walker, J. C. 1952. Diseases of Vegetable Crops. McGraw-Hill, New York. 529 pp.

Literature Cited

Asian Vegetable Research Development Center. 1979. Progress report for 1979. AVRDC, Shanhua, Taiwan. 107 pp.

Cook, A. A. 1978. Diseases of Tropical and Subtropical Vegetables and Other Plants. Hafner, New York. 381 pp.

Cook, H. T. 1953. The fungi that cause rot in sweet potatoes, pp. 444-447. In Plant Diseases. U.S. Dept. Agric. Yearbook. Washington, DC.

Cummings, R. W. 1976. Food Crops in Low-Income Countries: the State of Present and Expected Agricultural Research and Technology. Rockefeller Foundation, New York. 103 pp.

Food Agriculture Organization. 1981. FAO Production Yearbook. Vol. 35. FAO, Rome. 306 pp.

Fujise, K. 1970. Sweet potato and its breeding efficacy in Japan. pp. 19-21. In Plucknett, D. L. ed. Proc. 2nd Int. Symp. Trop. Root and Tuber Crops. Univ. of Hawaii, Honolulu.

Harmon, S. A., H. L. Hammett, T. Hernandez, and D. T. Pope. 1970. Progress in the breeding and development of new varieties. pp. 8-17. In Hernandez, T. P. ed. Thirty years of cooperative sweet potato research, 1939-1969. Southern Coop. Serv. Bull. No. 159, Louisiana State Univ., Baton Rouge.

Heyerdahl, T. 1963. Prehistoric voyages as agencies for Melanesian and South American plant and animal dispersals to Polynesia. pp. 23-26. In Barran. ed. Plants and Migrations of Pacific Peoples. Bishop Museum Press, Honolulu.

International Institute Tropical Agriculture. 1978. 1978 Annual Report. Ibadan, Nigeria. 129 pp.

Kay, D. E. 1973. Root Crops. Trop. Prod. Inst., London. 245 pp.

Purseglove, J. W. 1968. Tropical Crops, Dicotyledons. Wiley, New York. 719 pp. 2 Vol.

Villareal, R. L. 1982. Sweet potato in the tropics - progress and problems. pp. 1-15, In Villareal, R. L. and T. D. Griggs. eds. Sweet potato. Asian Vegetable Res. Develop. Center, Shanhua, Taiwan. 481 pp.

Yen, D. E. 1974. The Sweet Potato and Oceania: an Essay in Ethnobotany. Bishop Museum Press, Honolulu. 389 pp.

YAMS

Yams are an important food crop for millions of small farmers in the tropics. The term "yam" is widely misused, and here will only refer to plants of the genus Dioscorea. For example, sweet potatoes (Ipomoea batatas) are often called yams in the United States, and some aroids such as Colocasia esculenta are called cocoyams. The genus Dioscorea has perhaps 650 species, but only a few are important as food crops. Nine species of major importance (Kay 1973) are listed here:

1. D. alata - Greater yam
2. D. bulbifera - Potato yam
3. D. cayenensis - Yellow yam
4. D. dumetorum - Bitter yam
5. D. esculenta - Lesser yam
6. D. hispida - Intoxicating yam
7. D. opposita - Chinese yam
8. D. rotundata - White yam
9. D. trifida - Cush - Cush yam

The best source of information on yams is the fascinating book by Coursey (1967) who lists over 70 species of major economic importance. It is most difficult to evaluate the relative importance of yams as a food since few enter trade channels. Purseglove (1972) estimates a global production of 20 million tons and notes that West Africa produces two-thirds of the world's yams. They are a staple food for millions of small farmers in the tropics, but also play an important role in their culture, especially in West Africa. Coursey (1967) states:

> "In West Africa, especially, the ceremonies associated with the yam harvest are often the major social-cum-religious occasions of the year, while in parts of this area quite complicated religious cults centre on the yams."

Considering the number of edible species, an understanding of their centers of origin is necesarily complicated, and for detailed information Coursey (1967) and Purseglove (1972) should be consulted. Four distinct centers of origin are suggested by Coursey (1967); the Indo-Chinese peninsula, Southern China, West Africa, and the Caribbean. Yams are mentioned in Chinese documents about 2000 B.C. (Coursey 1967). Most of the different major species of yams have spread throughout the tropics. For example D. alata, which probably originated in Southeast Asia, is now the preferred species in most parts of the world and is popular in Africa and the Caribbean (Purseglove 1972).

Yams are dioecious perennial vines, but are usually grown as annuals. Most varieties are propagated vegetatively by means of "setts" (small tubers or

pieces of tuber). Yam culture is labor intensive, and in Africa yams are most often grown by small, subsistence farmers, often in a slash and burn system of agriculture. Yams are usually planted in carefully prepared mounds or ridges, and a variety of cultural practices are used for growing the many different species. It is necessary to provide support for some species of yams; thus stakes or other forms of support are used. Yams can be stored for several months. In Africa they are often tied to a shaded frame which allows good ventilation (Purseglove 1972 and Coursey 1967).

Fig. 27. Yams in market -- Ghana. (Courtesy Conrad Bonsi)

The major yam producing area in the world is West Africa, followed by Southeast Asia, the Pacific islands, and the Caribbean region. D. alata is the most important species grown. Although yields are variable and depend on the variety and growth conditions, they average about 10 t/ha which is generally less than those of sweet potatoes. The primary use of yams is for food. According to Purseglove (1972), in the yam zone of West Africa daily consumption per person is 0.5-1.0 kg. Yams are baked, boiled, and fried, although in West Africa they are also eaten as "fufu", a stiff glutinous dough prepared by pounding yams in a wooden mortar. Unlike many root crops, yams contain about 6% or more crude protein on a dry weight basis. Consumption of yams in West Africa has declined, even though they are still the preferred carbohydrate. They are expensive and time consuming to grow and to prepare for food, and thus competition from cassava and sweet potatoes has caused a decline in their importance in recent years.

Several wild species of yams contain steroidal sapogenins, and in the past were grown commercially in the Americas as a source of compounds to synthesize cortisone, sex hormones, and oral contraceptives (Martin 1970). Synthetic production of such compounds have made Dioscorea species less important sources in recent years.

Little work has been done on yam improvement until recently. Coursey and Martin (1970) suggest that since yams have been "virtually untouched by plant breeders" substantial improvement should be possible with only limited effort. Martin (1977) made a worldwide collection of varieties of the principal species in Puerto Rico. The IITA (International Institute of Tropical Agriculture) in Ibadan, Nigeria has a program of yam improvement by breeding, and their effort is primarily concentrated on D. rotundata, D. alata and D. dumetorum (IITA 1980).

Because of the general neglect of yams until quite recently, and because of the many species grown, little information on yam pathology is available in comparison to that for other important crops. The PANS (1978) manual on pest control in tropical root crops is one of the best sources of information on yam diseases. As major diseases they list anthracnose (Glomerella cingulata), Cercospora leaf spots (several spp. of Cercospora), leaf spot and neck rot caused by Corticium rolfsii, web blight (Thanatephorus cucumeris), and leaf spots caused by Pestalotiopsis cruenta, Colletotrichum cruenta, and Phyllosticta spp. A number of fungi cause tuber rots of which the most important

is Botryodiplodia theobromae. Many viruses have been reported to attack yams (Centre for Overseas Pest Research 1978, Miege and Lyonga 1982, Nwankiti and Arene 1978), but much more work is needed on virus etiology. Nematodes also cause serious problems on yams (Bridge 1982).

Fig. 28. Yam storage in Nigeria.

References on yam diseases follow:

Selected References on Yam Diseases

Bridge, J. 1982. Nematodes of yams. pp. 253-264. In Miege, J. and S. N. Lyonga. eds. 1982. Yams: Ignames. Clarendon Press, Oxford. 411 pp.

Centre for Overseas Pest Res. 1978. Pest Control in Tropical Root Crops. PANS Manual No. 4., London. 235 pp.

Coursey, D. G. 1967. Yams. Longman, London. 230 pp.

Coursey, D. G. 1967. Yam storage: A review of yam storage practices and of information on storage losses. J. Stored Prod. Res. 2:229-244.

Miege, J., and S. N. Lyonga. eds. 1982. Yams: Ignames. Clarendon Press, Oxford. 411 pp.

Noon, R. A. 1978. Storage and market diseases of yams. Trop. Sci. 20:177-188.

Nwankiti, A. D., and O. I. Arene. 1978. Diseases of yam in Nigeria. Pest Articles News Sum. 24(4):486-494.

Literature Cited

Coursey, D. G., and F. W. Martin. 1970. The past and future of the yams as crop plants. pp. 87-90. In Plucknett, D. L. ed. Proc. 2nd Int. Symp. on Trop. Root and Tuber Crops. Univ. of Hawaii, Honolulu.

International Institute of Tropical Agriculture. 1980. Annual report for 1979. Ibadan, Nigeria. 152 pp.

Kay, D. E. 1973. Root Crops. Trop. Prod. Inst., London. 245 pp.

Martin, F. W. 1970. The current status of the sapogenin-bearing yams. pp. 99-101. In Plucknett, D. L. ed. Proc. 2nd Int. Symp. on Trop. Root and Tuber Crops. Univ. of Hawaii, Honolulu.

Martin, F. W. 1977. Selected yam varieties for the tropics. pp. 44-49. In Cock, J., R. MacIntyre, and M. Graham. eds. Proc. 4th Symp. Int. Soc. Trop. Root Crops. Int. Develop. Res. Centre-080e and Centro Int. Agric. Trop., Cali Colombia.

Purseglove, J. W. 1972. Tropical Crops, Monocotyledons. Wiley, New York. 607 pp. 2 Vol.

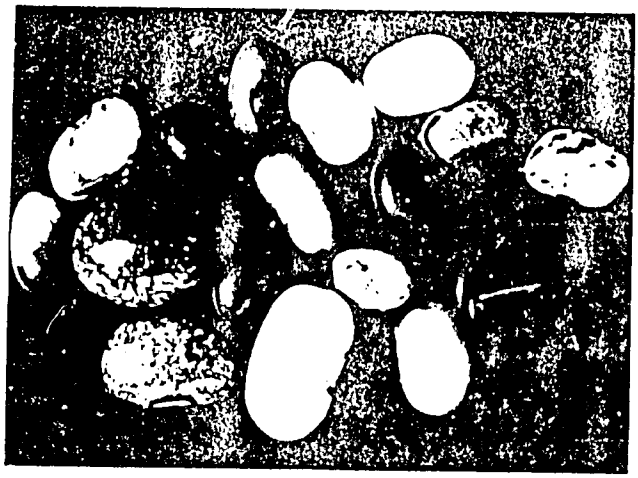


Fig. 29. Diverse types of beans grown by farmer on 1 1/2 hectare farm near Puebla, Mexico.

A recent book on legumes of world economic importance (Duke 1981) listed nearly 150 economically important species of legumes, and a U.S. National Academy of Sciences study (1979) described almost 200 species. The number of edible grain legumes important in the tropics is much less. Several important grain legumes are "cool weather" crops, primarily grown in higher elevations in the tropics, and thus will not be considered here. For example, chick peas (Cicer arietinum), dry peas (Pisum sp.), lentils (Lens esculenta), and broad beans (Vicia faba) are important "cool weather" legumes in developing countries.

Grain legumes of major importance in the lowland tropics which will be discussed are groundnuts, soybeans, cowpeas, pigeon peas, and mung beans. Common beans, although primarily a "cool weather" crop, will also be discussed as considerable acreages are grown in the lowland tropics. This is by no means a complete list, and many other edible legumes play an important role in agriculture in tropical developing countries. The excellent review by Rachie and Roberts (1974), and the books by Smartt (1976), Kay (1979), Duke (1981), and Purseglove (1968) describe these crops in detail.

A major problem in tropical developing countries is an unbalanced diet often low in protein. The chronic protein malnutrition problem is considered by many to be second only to the total world food supply problem, and it is estimated that 50% of the world's children do not receive adequate protein nutrition (Altschul, 1967). Protein "quality" is also important. Certain amino acids are necessary but frequently their level is deficient in major food sources, especially cereals. Grain legumes contain between 20 and 40% protein, and thus their use in tropical developing countries should be encouraged.

On a global basis, according to Rachie and Roberts (1974):

> "Cereals contribute two-thirds of all plant proteins consumed directly; grain legumes 18.5%; and other sources (roots, tubers, nuts, fruits, and vegetables), 13.5%".

Grain legumes can play an increasingly important role in providing improved nutrition throughout the tropics where inadequate nutrition is most serious. The nitrogen contribution to the plant by nitrogen-fixing legumes is also important with the increasing cost of fertilizer in the tropics. For information on diseases of the many food grain legumes (especially those not discussed here) the books "Pathology of tropical food legumes" (Allen 1983), "Pest control in tropical grain legumes" (Centre Overseas Pest Research 1981) and "Tropical diseases of legumes" (Bird and Maramorosch 1975) are useful.

Literature Cited

Allen, D. J. 1983. Pathology of Tropical Food Legumes. Wiley, New York. 488 pp.

Altschul, A. M. 1967. Food proteins: new sources from seeds. Science 158:221-226.

Bird, J., and K. Maramorosch. eds. 1975. Tropical Diseases of Legumes. Academic Press, New York. 171 pp.

Centre Overseas Pest Res. 1981. Pest Control in Tropical Grain Legumes. London. 206 pp.

Duke, J. A. 1981. Handbook of Legumes of World Economic Importance. Plenum, New York. 345 pp.

Kay, D. E. 1979. Crops and Product Digest No. 3 - Food Legumes. Trop. Prod. Inst., London. 435 pp.

National Academy of Sciences. 1979. Tropical Legumes: Resources for the Future. Natl. Acad. Sci., Washington, DC. 331 pp.

Purseglove, J. W. 1968. Tropical Crops. Dicotyledons. Wiley, New York. 719 pp. 2 Vol.

Rachie, K. O., and L. M. Roberts. 1974. Grain legumes of the lowland tropics. Adv. Agron. 26:1-132.

Smartt, J. 1976. Tropical Pulses. Longman, London. 348 pp.

COMMON BEAN

The common bean (*Phaseolus vulgaris*) is probably the most widely cultivated grain legume. Beans are high in protein (20-25% of dry weight), and thus are important in tropical diets based primarily on cereals and other starchy crops. Worldwide, annual production has been about 7 million tons of dry beans.

P. vulgaris originated in South and Central America where it was an important crop when European explorers arrived. Latin America currently accounts for about one-third of the total world bean production. Beans are extremely important to middle and low income families in tropical Latin America and Africa where they provide a large part of the dietary protein. The pods of some beans are eaten as a vegetable (snap beans), and the dried seed is used in a wide variety of dishes.

Most beans are grown under rainfed conditions in the tropics, usually in more or less traditional cropping systems under a wide range of management practices. Planting methods, spacing, etc. vary greatly, and often beans are interplanted with maize, squash, or other crops. Van Rheenen et al. (1981) concluded, from the results of a study on the effect of growing beans together with maize, that:

> "a kind of cultural control of the major bean diseases in Kenya is effected by growing beans in association with maize."

Most bean production is limited to cooler regions, but with increasing population pressure in highland mountainous areas many people are migrating from high elevations to the lowland tropics and taking beans with them. They usually encounter serious disease and pest problems in growing them under lowland conditions. Bean yields in the tropics (around 600 kg/ha) are low compared to average yields of over 1400 kg/ha in the United States (Schwartz and Galvez 1980).

The largest collection of *P. vulgaris* germplasm is in CIAT (International Center for Tropical Agriculture), Colombia. In 1981 they had 30,000 accessions of *Phaseolus* beans and 27,000 were *P. vulgaris*. In beans, tremendous variation occurs for many characters such as grain color, plant growth habit (dwarf or bush plants versus climbing or pole types), types of snap beans, and others. Consumers are typically very particular regarding these characteristics. Preferences may vary widely over relatively short distances, greatly complicating the work of those involved in developing improved varieties.

Diseases are major limiting factors of bean production, especially in tropical areas where a large number of pathogens attack them. Planting disease-free or chemically treated seeds can reduce or eliminate some diseases. A major objective of many national bean breeding programs and CIAT's international program is the development of disease-resistant cultivars.

Important fungal pathogens of beans in the tropics are rust (*Uromyces appendiculatus*), anthracnose (*Colletotrichum lindemuthianum*), angular leaf spot (*Isariopsis griseola*), web blight (*Thanatephorus cucumeris*), white mold (*Sclerotinia sclerotiorum*), and root rots caused by several species of fungi. Many additional fungal pathogens cause diseases of lesser importance (Schwartz and Galvez 1980). Bacterial diseases of importance include common bacterial blight (*Xanthomonas campestris* pv. *phaseoli*) and halo blight (*Pseudomonas syringae* pv. *phaseolicola*). A variety of diseases caused by viruses and mycoplasma-like pathogens are important in the tropics. Among the most important are bean common mosaic virus and the whitefly-transmitted bean golden

mosaic virus. Root knot (Meloidogyne spp.) and lesion (Pratylenchus spp.) nematodes cause serious losses on beans in the tropics.

Excellent information on the identification and control of bean diseases is available. The CIAT documentation center in Cali, Colombia, abstracts worldwide all the literature on beans. These published abstracts are available annually at a nominal cost.

Selected References on Bean Diseases

Allen, D. J. 1983. Pathology of Tropical Food Legumes. Wiley, New York. 488 pp.

Chupp, C., and A. F. Sherf. 1960. Vegetable Diseases and Their Control. Ronald Press, NY. 693 pp.

Schwartz, H. F., G. E. Galvez E., A. van Schoonhoven, R. H. Howeler, P. H. Graham, and C. Flor. 1978. Field Problems of Beans in Latin America. Centro Int. Agric. Trop., Cali, Colombia. 136 pp.

Schwartz, H. F., and G. E. Galvez. eds. 1980. Bean Production Problems: Disease, Insect, Soil and Climatic Constraints of Phaseolus vulgaris. Centro Int. Agric. Trop., Cali, Colombia. 424 pp.

Walker, J. C. 1952. Diseases of Vegetable Crops. McGraw-Hill, New York. pp. 529.

Zaumeyer, W. J., and H. R. Thomas. 1957. A monographic study of bean diseases and methods for their control. Tech. Bull. No. 868. U.S. Dept. of Agric., Washington, DC. 255 pp.

Literature Cited

Van Rheenen, H. A., O. E. Hasselbach, and S. G. S. Muigai. 1981. The effect of growing beans together with maize on the incidence of bean diseases and pests. Neth. J. Plant Pathol. 87:193-199.

WEB BLIGHT OF BEANS

Disease Name: Web blight of beans.

Causal Organism: Thanatephorus cucumeris (Frank) Donk; (Tulasnellaceae)
Imperfect state - Rhizoctonia solani Kühn

Fig. 30. Web blight of beans caused by Thanatephorus cucumeris.

In the humid lowlands of the tropics, web blight is possibly the single most destructive disease of beans (Phaseolus vulgaris). In temperate areas T. cucumeris generally causes damping-off and stem canker-type diseases and a pod rot of beans, whereas in the humid tropics, in addition, it causes a severe aerial blight disease of beans called web blight. Kühn in Germany in 1858 was the first to describe diseases caused by R. solani on potatoes. Web blight was reported for the first time on figs in Florida (Matz 1917) and later on beans in 1921 in Puerto Rico. Weber (1939) reported web blight of beans from Florida and gave a detailed description of the disease. T. cucumeris has a very wide host range and can attack most cultivated crops in their early growth stages. The fungus is found all over the world. Web blight has been reported from every country of tropical Latin America and the Caribbean, plus Africa and Asia. It probably exists in all tropical countries where beans are grown.

Historically T. cucumeris has been more commonly known as Rhizoctonia solani (the imperfect state). The most recent synonyms of T. cucumeris are Corticium solani (Prill. and Delacr.) Bourd. and Galz and Pellicullaria filamentosa (Pat.) Rogers. The complicated taxonomy and nomenclature of the fungus are discussed by Talbot (1970).

Importance

Web blight is one of the main constraints to bean production in the hot, humid tropical areas of Latin America. Beans are traditionally grown in cooler, temperate areas in Latin America, but because of population pressures, farmers migrate from high to low-altitude areas and often take beans with them. Little can be offered regarding control measures for web blight.

In warm and humid tropics T. cucumeris can cause rapid defoliation and sometimes complete crop failure of beans. In 1980, an epidemic of web blight occurred in the Guanacaste region in the northern part of Costa Rica, resulting in a 90% reduction in bean yields (Galindo 1982). As with many tropical diseases precise information on losses is difficult to find, but it is characterized as severe in Mexico (Crispin and Gallegos 1963), Costa Rica (Echandi 1965), and elsewhere in Latin America (Schwartz and Galvez 1980).

Symptoms

Initial symptoms of web blight of beans are small water-soaked spots on the foliage. The spots are lighter in color than the surrounding unaffected tissues and later become tan-brown with a dark border. They may enlarge and coalesce to cover the entire leaf surface, giving the plant a scalded appearance. Under moist, warm conditions, the fungal hyphae grow from the infected leaf areas to other plant parts, binding them together with a mat of mycelial strands, and thus explaining the name of the disease, "web blight". Early infection of young pods appears as light-tan, irregular-shaped specks which often enlarge and coalesce, destroying the pod. Older pods continue to develop after being attacked, and the seed within the pod may become infected.

Many small, brown sclerotia also occur abundantly on bean tissue affected by web blight. Weber (1939) noted the ease with which small, lightweight sclerotia were detached and disseminated by the wind and other agents from infected to healthy plants. Echandi (1965) and Galindo (1982) observed lesions caused by basidiospores of T. cucumeris (small, circular, reddish-brown necrotic spots) on beans planted in the lowland regions of Costa Rica.

Host Plants

T. cucumeris has an extremely wide host range (Parmeter 1970), attacking as many as 200 plant species (Schwartz and Galvez 1980). The web blight disease is reported to occur not only on beans, but on a large number of other crops as diverse as figs and pigeon peas. In addition to crop plants the fungus can also infect a number of weed species (Weber 1939).

Pathogen Cycle

The main sources of inocula which can initiate infection are mycelial fragments and sclerotia. Basidiospores can also cause infection (Echandi 1965). Bean seed infected at a rate of 1.5% with T. cucumeris has been reported from Costa Rica (Castro 1970, cited by Galindo 1982). Thus seed might introduce the pathogen into a new area or serve as a source of primary inoculum. A study by Galindo et al. (1983b) found sclerotia and mycelia free in soil or in the form of colonized debris to be the main source of inoculum in Costa Rica. Inoculation of beans occurred mainly by splashing of rain drops containing infested soil. Large numbers of small sclerotia were produced on rain-splashed soil and debris adhering to bean tissues and on detached tissues on the soil surface. These sclerotia provide new sources of inoculum which again can be splashed onto beans. Weber (1939) suggested that sclerotia may also be disseminated by wind.

In the study by Galindo (1982, 1983b) infections caused by basidiospores were observed as previously reported by Echandi (1965). However, the lesions observed were not numerous, remained restricted in size, and apparently caused little damage. Studies by Galindo et al. (1983a, 1983b) on control of web blight by mulching indicated that in Costa Rica basidiospores played a minor role in disease spread during the period of their study.

Little is known about the precise environmental conditions favoring inoculation. Web blight occurs in hot, humid areas of the tropics and subtropics but almost never in temperate areas. This is not due to different strains of the fungus. Galindo et al. (1981) studied 33 isolates of R. solani from New York and found that several produced as severe aerial infections (web blight) as did web blight isolates from Colombia. Tests were made at 25°C with continuous misting in a growth chamber.

Anastomosis among isolates of R. solani has been used by Parmeter et al.

(1969) as a reliable method of grouping isolates. Four anastomosis groups are recognized by Parmeter et al. (1969), but more are known (Anderson 1982). Galindo et al. (1981) found that all the six aerial isolates obtained from Colombia (isolated from web blight lesions) were in anastomosis group (AG) 1. All isolates made in Costa Rica were pathogenic, but virulence was highly variable. Of 73 Costa Rican isolates 26 were in AG group 1 and 38 in AG-2 (Galindo et al. 1982).

Control

Work in Colombia has shown that a number of fungicides can control the pathogen (Schwartz and Galvez 1980). Four applications of a systemic fungicide gave yields of 1 ton/ha compared to no yield from the unsprayed check. However, chemicals are not used in Latin America for control of web blight primarily because of their high cost.

There are differences among bean cultivars in their degree of resistance to T. cucumeris, but none have been found with high levels of resistance or immunity (Oyekan et al. 1976, Schwartz and Galvez 1980). Plant architecture appears important; the upper foliage of climbing beans escapes infection, since inoculum in splashing rain cannot reach it. Schwartz and Galvez (1980) suggest that upright plant architecture, an open canopy, and wide plant spacing may contribute toward control.

Experiments by Galindo et al. (1983a) in Costa Rica showed that mulching was highly effective and superior to chemical treatments for web blight control. Mulching with rice husks in a layer 2.5 cm thick greatly reduced splashing of inoculum and disease severity. Galindo (1982) visited small farms in the region to observe production practices of the farmers and to determine whether they employed any specific practices to control web blight. He found that throughout the region a common production practice called "frijol tapado" was used.

"Frijol tapado" means "covered beans". This practice consists of broadcasting bean seeds into carefully selected weeds, then cutting the weeds with a machete so the broadcast bean seeds are covered with a mulch of weeds. An indeterminate type of bean, between a bush and a climbing bean, is used. It grows through the mulch and eventually covers it. This combination of mulch and bean plants effectively prevents weed growth and appears to conserve soil moisture. In addition, the mulch prevents soil splashing, which was found in this study to be the most important source of inoculum causing web blight. Thus the disease is effectively controlled by limited-resource farmers who use the cultural practice of frijol tapado, even in areas where climate is optimal for web blight development as shown by its high incidence in the experimental area and adjacent fields planted under clean cultivation.

Frijol tapado fields are generally planted in hilly areas. Farmers select hills that receive the full effect of sunlight early in the morning, thereby reducing the periods of high humidity that favor infection by R. solani. The fields selected are generally occupied by broadleaf weeds and certain grasses that will not regrow after they are cut. Thus the weeds do not compete with the beans for light, nutrients, or moisture. The farmers have other practices aimed at reducing damage caused by web blight. When beans are planted in rows, for example, the direction of the rows follows the path of the sunlight or the direction of the predominant winds in the region, conditions known by farmers to reduce humidity within the crop canopy.

The frijol tapado system was compared with another mulch system (2.5-cm thick layer of rice husks, a cheap by-product commonly found in the area) using

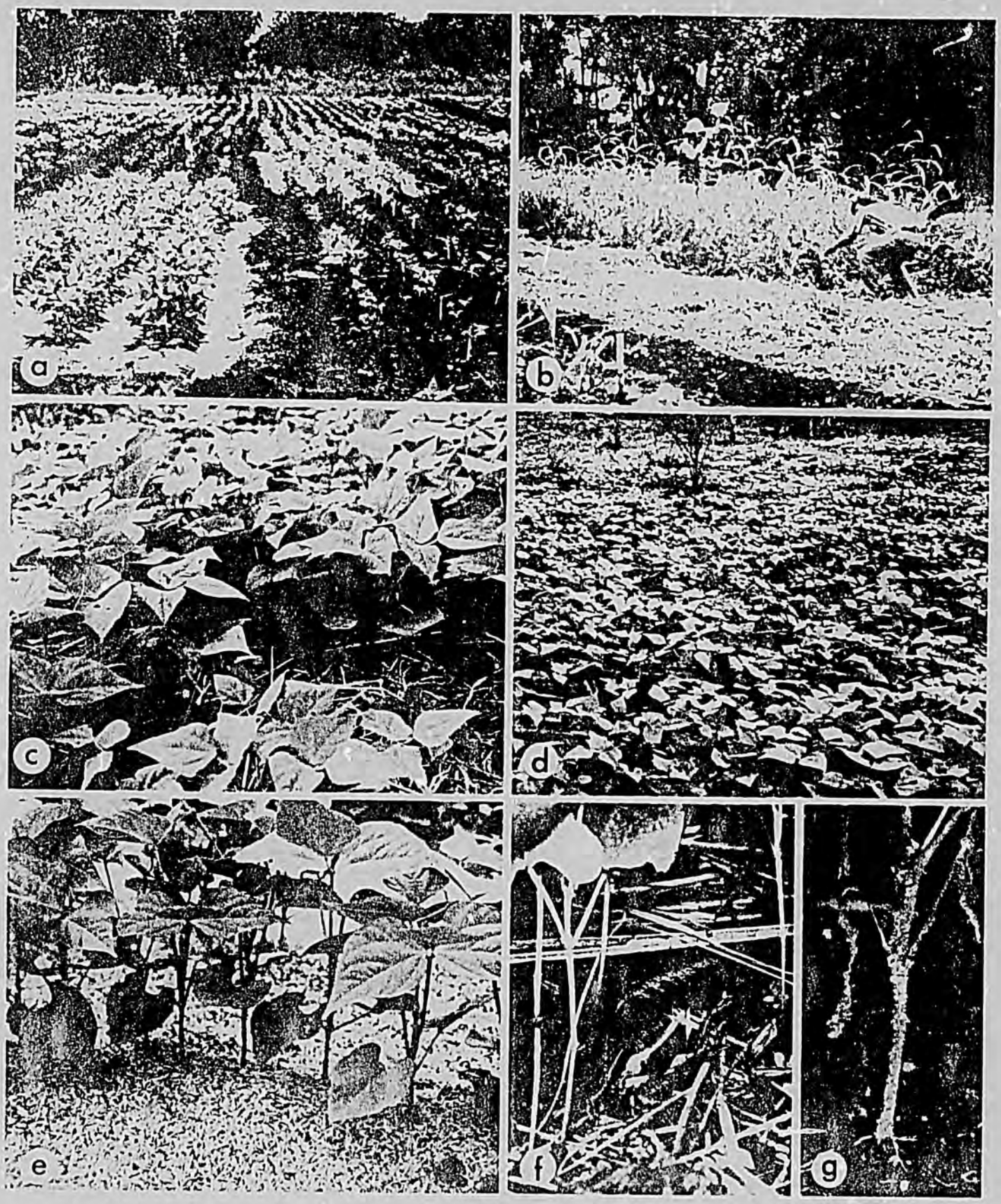


Fig. 31. Web blight (WB) of beans. a, Effective control of WB provided by mulching with rice husks (left foreground) compared to severe WB in the nonmulched check (right foreground). b, Cutting weeds with a machete to prepare "frijol tapado" planting. c, Close-up of the mulch layer in the frijol tapado. d, Frijol tapado field 3 weeks after planting. e and f, Close-up of plants mulched with rice husks or frijol tapado debris. g, Rain-splashed soil on WB infected plant.

both web blight susceptible and tolerant bean cultivars. The rice husks and frijol tapado were equally effective in avoiding splashing of infested soil and in controlling web blight. Both frijol tapado and mulch with rice husks gave better control of web blight than the fungicide PCNB (pentachloronitrobenzene).

PCNB is a highly effective chemical against R. solani and can be applied as a soil or foliar treatment. Bean yields of both tolerant and susceptible varieties were increased significantly by mulching with rice husks or by frijol tapado (Table 1).

In the absence of web blight, the yields in fields under the frijol tapado system are generally lower than in those planted in drilled rows with clean cultivation. For this reason, some agronomists in Latin America oppose continuation of the frijol tapado system; however, on many small farms in Costa Rica, about 85 percent of the beans currently produced are grown by the frijol tapado system. Small farmers persist in using the system because of its low risk, its small investment in labor (primarily to cut weeds), and because there is always some yield even when prolonged periods of rain produce conditions which allow T. cucumeris to destroy bean yields under the clean cultivation system.

Galindo et al. (1983a) showed that the frijol tapado system has some important advantages; however, some modifications are needed if bean yields are to be increased and the benefits of reducing web blight incidence and severity maintained. One possible change would be to drill rather than broadcast the seeds and, when appropriate, to use more fertilizer supplement. Drilling might be done after the weeds are cut. The practice might be feasible in some of the small holdings in which frijol tapado is used, and would be similar to the no-till or minimum-tillage systems used increasingly in modern agriculture.

Table 1. Effect of mulch treatments on bean yield of two cultivars planted in two fields in Costa Rica in 1980.[a]

Mulch Treatment	Bean seed yield (kg/ha)			
	Experimental field		Commercial field	
	Porillo 70	Mexico 27	Porillo 70	Mexico 27
None (clean cultivation)	0	0	273	217
Frijol tapado	-	-	637	534
Rice husks	655	587	835	679

[a] From Galindo et al. 1983a.

Data obtained in Galindo's investigation have helped deemphasize research aimed at looking for foliar application of fungicides to control web blight. The information collected suggests that control measures have to be directed toward reducing the level of inoculum in the soil and avoiding the splashing of the inoculum on bean tissues. These results will also help improve the screening procedures used to evaluate the reaction of bean varieties and breeding lines for resistance to the web blight fungus.

Literature Cited

Anderson, N. A. 1982. The genetics and pathogenicity of Rhizoctonia solani. Annu. Rev. Phytopathol. 20:329-347.

Crispin, A., and C. C. Gallegos. 1963. Web blight -- a severe disease of beans and soybeans in Mexico. Plant Dis. Rep. 47:1010-1011.

Echandi, E. 1965. Basidiospore infection by Pellicularia filamentosa (= Corticum microsclerotia), the incitant of web blight of common bean. Phytopathology 55:698-699.

Galindo, J. J., G. S. Abawi, and H. D. Thurston. 1981. Variability among isolates of Rhizoctonia solani associated with snap bean hypocotyls and soils in New York. Plant Dis. 66:390-394.

Galindo, J. J. 1982. Epidemiology and control of web blight of beans in Costa Rica. Ph.D. Thesis. Cornell Univ., Ithaca, N.Y. 141 pp.

Galindo, J. J., G. S. Abawi, H. D. Thurston, and G. Galvez. 1983a. Effect of mulching on web blight of beans in Costa Rica. Phytopathology 73:610-615.

Galindo, J. J., G. S. Abawi, H. D. Thurston, and G. Galvez. 1983b. Source of inoculum and development of web blight in Costa Rica. Plant Dis. 67:1016-1021.

Galindo, J. J., G. S. Abawi, H. D. Thurston, and G. Galvez. 1982. Characterization of Thanatephorus cucumeris isolates causing web blight of beans in Costa Rica. Turrialba 32:447-455.

Matz, J. 1917. A Rhizoctonia of the fig. Phytopathology 7:110-118.

Oyekan, P. O., F. T. Onesirosan, and R. J. Williams. 1976. Screening for resistance in cowpea to web blight. Trop. Grain Legume Bull. 3:6-8.

Parmeter, Jr., J. R. ed. 1970. Rhizoctonia solani: biology and pathology. Univ. California Press, Berkeley. 255 pp.

Parmeter, J. R., R. T. Sherwood, and W. D. Platt. 1969. Anastomosis groupings among isolates of Thanatephorus cucumeris. Phytopathology 59:1270-1278.

Schwartz, H. F., and G. E. Galvez, eds. 1980. Bean production problems: disease, insect, soil, and climatic constraints of Phaseolus vulgaris. Centro Int. Agric. Trop. Cali, Colombia. 434 pp.

Talbot, P. H. B. 1970. Taxonomy and nomenclature of the perfect state of Rhizoctonia solani. In J. R. Parmeter Jr. ed. Rhizoctonia solani: biology and pathology. Univ. California Press, Berkeley, 255 pp.

Weber, G. F. 1939. Web-blight, a disease of beans caused by Corticum microsclerotia. Phytopathology 29:559-575.

SOYBEANS

The soybean (Glycine max) is an annual legume and one of the world's major sources of oil and protein. Although most production (66% in 1980) is in temperate areas, there is also considerable production in tropical countries such as Indonesia and Thailand. Brazil has recently become a major producer of soybeans, although in temperate areas. In 1981 88 million tons were produced on about 50 million hectares worldwide (FAO 1981). They are an important food and oil crop in many areas of Asia. Soybeans likely originated in eastern Asia, probably China, where they have been cultivated for thousands of years. They are currently grown from the equator north and south to 52° latitude, and are often grown in the same areas as maize, requiring similar climatic and soil conditions. Propagation is by seed.

Soybeans are consumed in innumerable ways. Seeds may be eaten green before maturity, and the dried seeds are eaten whole, sprouted, ground into a flour, or processed into soymilk or tofu. Seeds may be fermented to produce a high protein product as in China and Indonesia, or a dark sauce widely used in Asian cooking. The oil is edible, but also has many industrial uses. Soybean meal after oil extraction is used as a high protein animal feed.

Sinclair (1982) states that over 100 pathogens affect soybeans of which 35 are economically important. Important diseases in the tropics are soybean rust (Phakopsora pachyrhizi), anthracnose (Colletotrichum dematium f. sp. truncata), Rhizoctonia disease (root rot, web blight), pod and stem blight (Diaporthe phaseolorum var. sojae - imperfect stage Phomopsis sojae), downy mildew (Peronospora manshurica), Phomopsis seed decay, frogeye leafspot (Cercospora sojina), bacterial pustule (Xanthomonas campestris pv. phaseoli), bacterial blight (Pseudomonas syringae pv. glycinea), and numerous virus diseases.

Fig. 32. Soybeans.

Numerous collections of soybean germplasm are found throughout the world. The AVRDC (Asian Vegetable Research and Development Center) in Taiwan has a collection of over

10,000 accessions. AVRDC is a major center of tropical soybean improvement as is INTSOY (International Soybean Program of the University of Illinois).

Selected References on Soybean Diseases

Allen, D. J. 1983. Pathology of Tropical Food Legumes. Wiley, New York. 488 pp.

Caldwell, B. E. ed. 1973. Soybeans: Improvement, Production and Uses. Am. Soc. Agron., Madison, WI. 681 pp.

Ford, R. E., and J. B. Sinclair. eds. 1977. Rust of Soybean - the Problem and Research Needs. Univ. Illinois, Champaign-Urbana Col. Agric. INTSOY Series No. 12.

Sinclair, J. B. 1977. Infectious soyabean diseases of world importance. Pest Articles News Sum. 23:49-57.

Sinclair, J. B. 1982. Compendium of Soybean Diseases. Am. Phytopathol. Soc., St. Paul, MN. 104 pp.

Sinclair, J. B., and O. D. Dhingra. 1975. An Annotated Bibliography of Soybean Diseases 1882-1974. Univ. Illinois Champaign-Urbana, Champaign, IL. 280 pp.

Literature Cited

Food Agriculture Organization. 1981. FAO Production Yearbook. Vol. 35. FAO, Rome. 306 pp.

GROUNDNUTS

Groundnuts or peanuts (Arachis hypogaea) in terms of total production (19 million tons in 1981 - unshelled) are by far the most important tropical grain legume (FAO 1981). However, most peanut production goes for processing into oil and the byproduct peanut cake which is used for animal feed. Groundnuts are not as important for human food as are some other major tropical grain legumes; however in some areas of Africa and Asia they constitute an important part of the diet.

Two main subspecies (hypogaea and fastigiata) are described (Rachie and Roberts 1974), and these are further divided into Virginia and Spanish-Valencia types. Peanuts originated in South America, probably in Brazil or Bolivia. From there they were carried to other areas of the Americas, Africa, and Asia by the Spanish and Portugese. Most are grown 40°N and S of the equator. They are particularly well suited to semi-arid tropical regions.

Commercial production of peanuts is from seed. Good rainfall distribution is necessary during the growing season, but dry conditions are important for harvesting. The "nuts" are borne underground. After flower pollination the meristem at the base of the ovary grows to form a peg bearing the ovary at its tip. The peg elongates, growing downward, and places the ovary a few inches below the soil surface. Development of the fruit, or elongated pod, takes place completely below the ground surface.

Over 10,000 cultivated peanut accessions are known in various collections around the world. Breeding programs have attempted to increase yield, oil content, drought resistance, and resistance to diseases and insects. ICRISAT (International Crops Research Institute for the Semi-Arid Tropics) in Hyderbad, India, has an extensive groundnut improvement program.

Important pathogens of peanuts in the tropics (Rachie and Roberts 1974) are Cercospora leaf spots (C. arachidicola and Cercosporidium personatum), rust (Puccinia arachidis), Sclerotium rolfsii, the rosette virus, peanut mottle virus, and Aspergillus flavus. Fusarium spp. (F. solani and F. oxysporum) are also important pathogens of peanuts, causing pod rots in the tropics. The rosette disease is perhaps the most serious problem in Africa south of the Sahara, and the virus is aphid transmitted. The fungus Aspergillus flavus (Diener and Davis 1977) attacks seed before and after harvest causing considerable post-harvest loss. These losses are aggravated by the production by the fungus of aflatoxins, which are highly toxic to livestock and humans.

Selected References on Peanut Diseases

Allen, D. J. 1983. Pathology of Tropical Food Legumes. Wiley, New York. 488 pp.

Diener, U. L., and N. D. Davis. 1977. Aflatoxin formation in peanuts by Aspergillus flavus. Alabama Agric. Exp. Stn. Bull. 493:1-49.

Feakin, S. D. ed. 1973. Pest Control in Groundnuts. PANS Manual No. 2. Centre for Overseas Pest Res., London. 197 pp.

Garren, K. H., and C. R. Jackson. 1973. Peanut diseases. pp. 424-494. In American Peanut Research and Education Association. Stone Printing Co., Roanoke, Virginia. 684 pp.

International Crops Research Institute for the Semi-Arid Tropics. 1980. Proceedings of the international workshop on groundnuts, 13-17 October, 1980. Patancheru, A. P., India.

Jackson, C. R., and D. K. Bell. 1969. Diseases of peanut (groundnut) caused by fungi. Univ. of Ga., Col. Agric. Exp. Res. Bul. 56.
MacDonald, D. 1969. Groundnut pod diseases. Rev. Appl. Mycol. 48:465-474.
Porter, D. M., D. H. Smith, and R. Rodriguez-Kabana. 1984. Compendium of peanut diseases. Am. Phytopathol. Soc., St. Paul, MN. 73 pp.

Literature Cited

Food Agriculture Organization. 1981. FAO Production Yearbook. Vol. 35. FAO, Rome. 306 pp.
Rachie, K. O., and L. M. Roberts. 1974. Grain legumes of the lowland tropics. Adv. Agron. 26:1-132.

Fig. 33. Healthy cowpeas (Vigna unguiculata) in Nigeria. (Courtesy R. J. Williams)

Cowpeas or black-eyed peas (Vigna unguiculata) are the most important food grain legume in Africa, and are also grown extensively in the Americas and Southeast Asia. World production in 1975 was estimated at over one million tons (Kay 1979). Cowpeas contain high protein levels and the highest percentage of methionine, an essential amino acid, of all the food legumes. Cowpeas are believed to have originated in West Africa where many wild types are still found. Three types of V. unguiculata are recognized: unguiculata (cowpea), cylindrica (catjang), and sesquipedalis (yard-long bean). The latter two types are grown primarily in Asia.

Cowpeas are eaten as immature pods and seeds and as dried seed (which is sometimes ground into a meal). The leaves are also cooked as a pot-herb in some areas. They are grown primarily under rainfed conditions, most often interplanted with other crops such as maize and sorghum. Cowpeas will tolerate drier and warmer conditions than Phaseolus vulgaris and are often grown on poor soils, especially in West Africa.

Little effort had gone into cowpea improvement by breeding until the early 1970's. A global germplasm collection of more than 10,000 accessions has been assembled at IITA (International Institute for Tropical Agriculture) in Ibadan, Nigeria. Average yields in Africa are estimated at about 400 kg/ha, but as yields of about 1500 kg/ha are obtained in the United States a great potential exists for cowpea improvement (Singh 1980). Diseases and insects are major constraints to increased productivity in West Africa. Work at IITA has identified lines with multiple disease resistance and high yield (Williams 1977).

Diseases are most severe in the forest region of West Africa and less so in the Savanah regions where most cowpeas are grown. Williams (1975) and Singh and Allen (1980) describe the most important diseases of cowpeas.

Two virus diseases are most important: cowpea yellow mosaic virus and cowpea aphid-borne mosaic virus. Fungi of importance are Cercospora leaf spots (Cercospora canescens and C. cruenta), anthracnose (Colletotrichum lindemuthianum), rust (Uromyces appendiculatus), web blight (Thanatephorus cucumeris), Corynespora leaf spot (Corynespora cassiicola), and Pythium stem rot (Pythium aphanidermatum). Bacterial pustule (Xanthomonas sp.) and bacterial blight (Xanthomonas campestris pv. vignicola) are also important.

Selected References on Cowpea Diseases

Allen, D. J. 1983. Pathology of Tropical Food Legumes. Wiley, New York. 488 pp.

Singh, S. R., and D. J. Allen. 1979. Cowpea Pests and Diseases. Manual Series No. 2, Int. Instit. Trop. Agric., Ibadan. 113 pp.

Singh, S. R., and D. J. Allen. 1980. Pests, diseases, resistance and protection in cowpeas. pp. 419-443. In Advances in Legume Science. Summerfield, R. J. and A. H. Bunting. eds. HM Stationary Office, London.

Williams, R. J. 1975. Diseases of cowpea (Vigna unguiculata (L.) Walp.) in Nigeria. Pest Articles News Sum. 21:253-267.

Williams, R. J. 1977. Identification of multiple disease resistance in cowpea. Trop. Agric. (Trinidad) 54:53-59.

Literature Cited

Kay, D. E. 1979. Crops and Product Digest No. 3 - Food Legumes. Trop. Products Inst., London. 435 pp.

Singh, S. R. 1980. Biology of cowpea pests and potential for host plant resistance. pp. 398-421. In Biology and breeding for resistance to arthropods and pathogens in agricultural plants. M. K. Harris. ed. Texas A&M Univ. Bull. MP-1451.

PIGEONPEAS

Pigeonpeas (Cajanus cajan), a major grain legume in South Asia, are especially important in India which produces over 90% of the world's crop. They are also grown in the tropics of Africa and the Americas. Global production was estimated at about 2 million tons (Kay 1979). Both India and East Africa have been suggested as possible centers of origin for pigeonpeas. They may have been cultivated in Egypt around 2,000 B.C., as seed has been found in Egyptian tombs dated near this time.

Seeds are used whole or split in cooking and also are processed into flour. Green seeds, leaves, and pods are eaten as a vegetable. The plant is a perennial shrub with woody stems and branches. It is sometimes used as an animal fodder, and the dried stalks are useful as firewood. Pigeonpeas are usually grown under rainfed conditions, and among grain legumes they are one of the most resistant to drought and heat. They are widely adapted to a variety of lowland tropical soil and climate conditions. Often they are interplanted with other crops such as maize, sorghum, and millet.

Many cultivars are grown in the tropics, and a collection of over 5,000 accessions was made in India before ICRISAT (International Crops Research Institute for the Semi-Arid Tropics) began its program. ICRISAT's breeding program has as major objectives increased yield and incorporation of disease and insect resistance. Diseases of major importance are wilt (Fusarium udum), Phytophthora blight (Phytophthora drechsleri f. sp. cajani), sterility mosaic, witches broom, and rust (Uredo cajani). Fusarium wilt is probably the most serious disease of pigeonpea worldwide and is a limiting factor in some areas of India and East Africa.

Selected References on Pigeonpea Diseases

Allen, D. J. 1983. Pathology of Tropical Food Legumes. Wiley, New York. 488 pp.

Kannaiyan, J., O. K. Ribeiro, D. C. Erwin, and Y. L. Nene. 1980. Phytophthora blight of pigeonpea in India. Mycologica 72:169-181.

Kannaiyan, J., Y. L. Nene, T. N. Raju, and V. K. Sheila. 1981. Screening for resistance to Phytophthora blight of pigeonpea. Plant Dis. 65:61-62.

Nene, Y. L., and M. V. Reddy. 1976. Screening for resistance to sterility mosaic of pigeonpea. Plant Dis. Rep. 60:1034-1036.

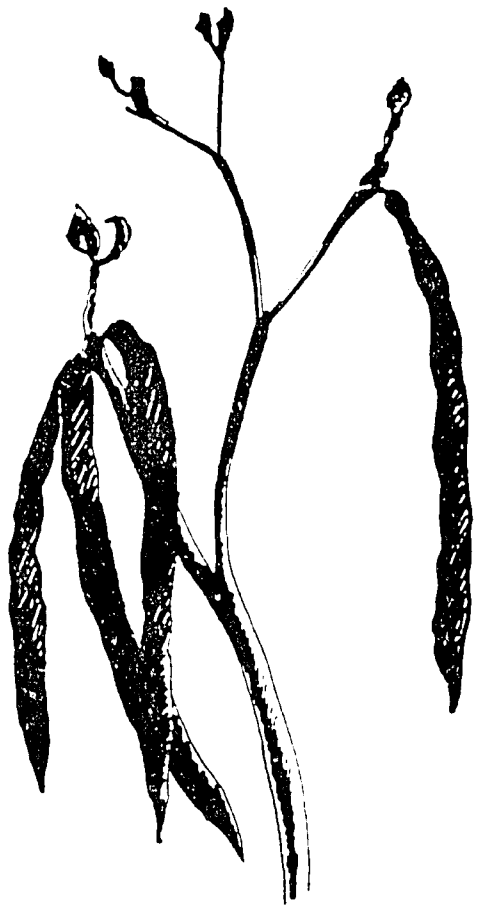
Nene, Y. L., Kannaiyan, J., and Reddy, M. V. 1981. Pigeonpea diseases: resistance-screening techniques. Information Bull. No. 9, Int. Crops Res. Inst. Semi-Arid Tropics, Patancheru, A.P., India.

Nene, Y. L. ed. 1981. Proceedings International Workshop on Pigeonpeas. Vol. 1. Int. Crops Res. Inst. Semi-Arid Tropics, Patancheru, A.P., India. 508 pp.

Literature Cited

Kay, D. E. 1979. Crop and Product Digest No. 3 - Food Legumes. Trop. Products Inst., London. 435 pp.

MUNGBEANS



Mungbeans (Vigna radiata) are also known as green gram, mungo, and urdbean or black gram (V. mungo) depending on seed color. They are especially important in Southeast Asia, but some are also grown in Africa and the Americas. World production in 1979 was estimated to be 1.4 million tons from 3.5 million hectares. Two-thirds of this production was in India. Mungbeans are usually grown under rainfed conditions, often following rice in Asia. Some cultivars can tolerate drought and some highly saline soils. They are often intercropped and grow at low to intermediate elevations in the tropics.

The seeds of the highly nutrious mungbean are consumed directly in a variety of dishes, or are ground into flour. The pods are also eaten as a vegetable. Beansprouts are most commonly prepared from mungbeans for use in Indian and Chinese dishes. Yields are low as mungbeans are usually grown as a low input crop. Mungbeans have received relatively little attention until recently, considering their potential for the lowland tropics. AVRDC (Asian Vegetable Research and Development Center) has a program on mungbeans and has assembled a worldwide collection of over 5000 accessions. Considerable research on mungbeans has also been reported from India.

Like most legumes, there are a large number of pathogens which seriously affect mungbeans. The most important of these in the tropics are fungi such as Uromyces appendiculatus (rust), Erysiphe polygoni (powdery mildew), Cercospora canescens and C. cruenta (leaf spots), bacteria such as Pseudomonas syringae pv. phaseolicola (halo blight), and several viruses of which yellow mosaic appears most important. Major objectives of the AVRDC program are the development of widely adapted, higher-yielding mungbean lines with multiple pest resistance, uniform maturity, and improved seed quality, as well as the development of appropriate management technology.

Selected References on Mungbean Diseases

Cowell, R. ed. 1978. 1st International Mungbean Symposium. Asian Vegetable Res. and Develop. Center, Shanhua, Taiwan. 262 pp.

Nene, Y. L. 1972. A Survey of Viral Diseases of Pulse Crops in Uttar Pradesh. Univ. Press, Pantnagar, India. 191 pp.

Bananas and Plantains

The most important tropical fruit is the banana (Musa sp.). All of the bananas that reach world markets are grown in the tropics. Many people living in the temperate zones do not realize that bananas for fruit constitute only part of the bananas grown. Simmonds (1966) notes:

> "Roughly half of the bananas of the world are eaten raw and ripe and half are eaten cooked".

Cooking bananas or plantains are produced by millions of small farmers throughout the tropics. About 90% of the bananas consumed in Africa are plantains. Referring to plantains Simmonds (1966) states:

> "the biggest producer of fruit (plantains) for local consumption is Uganda, with a domestic crop amounting to some 15% of the total world production, roughly equal in amount to the entire trade of the fruit".

In 1981 world production of bananas (not including plantains) was about 40 million tons (FAO 1981). More bananas are consumed daily, raw or cooked, than perhaps any other fruit in the world. The number one fresh fruit in the United States in terms of total consumption is bananas (USDA 1975).

Bananas originated in the humid lowland tropics of Southeast Asia. Since their introduction into the American tropics, bananas and plantains are grown in almost all tropical regions of the world, primarily in the region between 30°N and 30°S latitude. With cassava, plantains constitute one of the most important foods for low income families living in the hot, humid, lowland tropics of Asia, Africa, and the Americas.

The taxonomy of the many species or subspecies of Musa is somewhat confused and different systems are found in the literature. Edible bananas probably originated from Musa acuminata and M. balbisiana. Most are triploid, although a few are diploid or tetraploids. Simmonds (1966) has classified edible bananas based on ploidy and on the basis of a scoring system using 15 plant characters showing the contributions of the two species M. acuminata and M. balbisiana to their origin. Most bananas in the Gros Michel and Cavandish groups are classified AAA, signifying that they are triploid with each

chromosome set coming from M. acuminata. It is this type that predominates in world banana trade. Most plantains are AAB, with one set of chromosomes from M. balbisiana. In addition to producing a more starchy fruit, these plants tend to be hardier, more drought-resistant, and resistant to some pathogens which can devastate AAA plantings.

Plantains have a variety of uses. They are boiled, baked, steamed, fried, made into a porridge, delicious plantain chips, and beer. Consumption in Uganda by the Buganda is said to reach 4-4.5 kg per person per day (Purseglove, 1972). Gourou (1961) states that in Uganda 61% are used for baking, 31% for making beer, 7% for grilling, and 0.5% for eating raw. The bracts from male banana flowers are used as a vegetable in some parts of Asia.

The banana is a large perennial herb consisting of an underground stem, or corm, a pseudostem, and a terminal crown of leaves. Shoots or suckers arise from the corm and give rise to new plants. Since edible varieties produce no seeds, they are propagated vegetatively from "sword suckers" or pieces of the corm. They have a high demand for moisture, and commercial bananas are often irrigated.

In terms of both quantity and quality, research on bananas for fresh fruit compares favorably to that on any tropical crop. Although some of the research is applicable to plantains, little emphasis is currently placed on this crop by any research organization. The plantain is one tropical crop that is not yet included in the program of any international center, but one that certainly deserves inclusion.

Several hundred cultivars or clones of bananas are known, and there are undoubtedly more undiscovered cultivars in Asia. The United Fruit Company made extensive collections in Southeast Asia, and in 1972 their collection in La Lima, Honduras included 575 accessions (Rowe and Richardson 1975). Several germplasm collections of edible Musa are found in Asia and tropical America. Until the 1960's Gros Michel was the major cultivar exported to temperate markets, but it was highly susceptible to Panama disease (Fusarium oxysporum f. sp. cubense) and has now been replaced by the Cavendish cultivars such as Valery which are resistant. Banana breeding programs in Trinidad and Jamaica, plus those of private companies, have had limited success (Rowe and Richardson 1975). Problems of ploidy complicate banana breeding in a crop whose fruit can produce no seed if it is to be commercially acceptable.

Historically, diseases have been major limiting factors in banana production. Worldwide, the Sigatoka leaf spots are the most important disease problem of bananas because of their destructiveness, wide distribution, and the high cost of fungicidal control. Three related pathogens cause Sigatoka leaf spots: Mycosphaerella musicola causes Sigatoka disease, M. fijiensis black leaf streak, and M. fijiensis var. difformis causes black Sigatoka (Stover 1980). Sigatoka is the name of a valley in Fiji where the Sigatoka disease became important about 1912, but M. musicola was first reported in Java in 1902. Sigatoka disease has been reviewed by Meredith (1970) and Stover (1972). Its introduction and rapid spread in the Americas starting about 1933 caused disastrous epidemics which threatened the existence of the banana industry in many countries until control measures were devised. Control of M. musicola (imperfect stage Cercospora musae) with fungicides and later oils (Meredith 1970, Calpouzos 1966) enable the industry to survive. After 1936 and for some years thereafter, the control program of the United Fruit Company was the largest private fungicide program in the world (Sequeira 1962).

Rhodes (1964) reported a new disease found in 1963 in Fiji which he called black leaf streak. Although the disease was similar to Sigatoka, it was

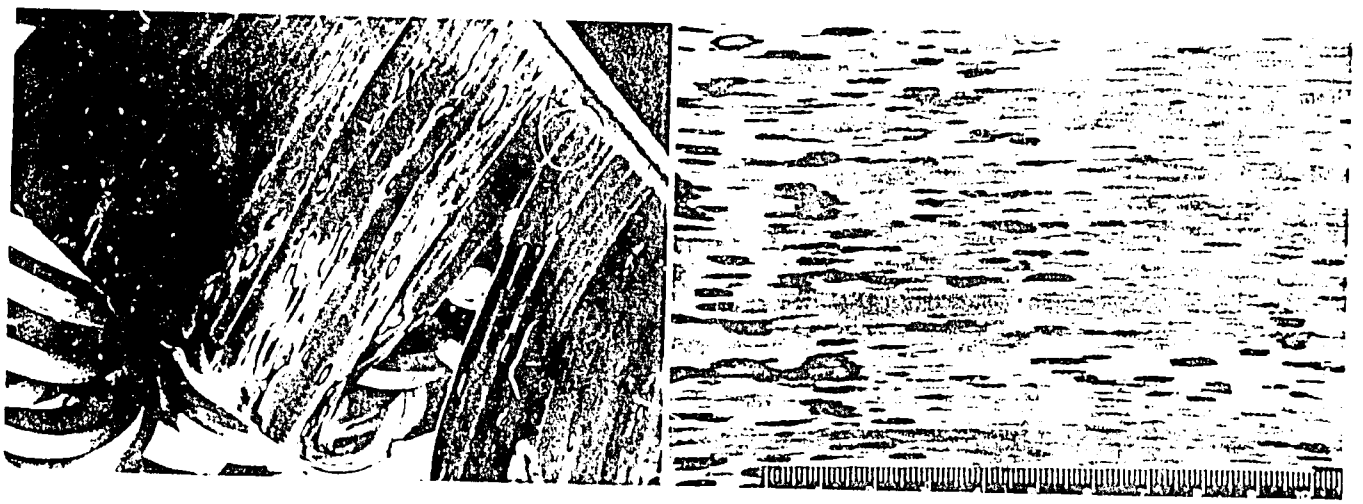


Fig. 34. Lesions on banana leaf caused by Mycosphaerella musicola (Sigatoka disease) (left). Black Sigatoka streaks and mature spots (Mycosphaerella fijiensis var. difformis (right). (Courtesy R. H. Stover)

somewhat more severe, and spraying methods which had been effective against Sigatoka were less effective against black leaf streak.

Black Sigatoka was discovered in Honduras in 1972 and is the most aggressive and destructive of the Sigatoka leaf spots. It has spread rapidly throughout Central America and will probably double costs of spraying (Stover 1980). Control of Sigatoka was primarily with fungicides and oils. According to Stover (1972) Bordeaux mixture was used from 1934-1958, oil-in-water emulsions with dithiocarbamate fungicides from 1958-1973, and since 1970 oil-in-water emulsions with systemic fungicides such as benzimidazoles have been important. Since strains of M. fijiensis var. difformis resistant to benomyl have evolved, use of chlorothalonil and the dithiocarbamates has increased. Bananas for fruit can be protected in the Americas at an increased cost, but the plantain plantings of many small growers will probably no longer be economic, since they seldom can afford fungicides and spray equipment. Resistant plantain cultivars such as Pelepita and Saba, and research on the value of cultural practices such as wider spacing, better water and air drainage, good weed control, and destruction of diseased leaves on disease severity, may help small farmers control black Sigatoka.

Fusarial wilt of bananas (Fusarium oxysporum f. sp. cubense) caused great economic losses in the American tropics on Gros Michel until the resistant Cavendish cultivars were introduced. Moko disease (race 2 of Pseudomonas solanacearum) has also caused serious losses in the American tropics.

Bunchy top is the most destructive virus disease of bananas. The disease is assumed to be caused by a virus, because the causal agent is transmitted by the banana aphid Pentalonia nigronervosa. The virus has never been purified. The disease was first described in Fiji in 1890. When infected bananas were introduced from Fiji into New South Wales, Australia in 1913, the virus devastated the young banana industry there, and over 2000 hectares were abandoned (Magee 1927). Economic control was achieved in Australia by regular inspections, destruction of diseased plants, and planting virus-free corms. In countries where such strict phytosanitary controls are not used the disease continues to spread and cause serious losses. Bunchy top is found in several countries of Asia and Africa, in Australia, and in some Pacific Island. Vakili

(1969) reported the disease in Vietnam in 1968, and suggested that two strains (abaca and banana) were present there. Most spread is by P. nigronervosa, but diseased corms also spread the pathogen. Great care should be taken to prevent the introduction of this destructive disease into the Americas and other areas where it is not found.

Nematode root rot caused by Radopholus similis is the most important banana disease caused by a nematode. There are many other important diseases of bananas. Excellent current information on control and identification of banana diseases is available, and most of it is also applicable to plantains.

Selected References on Banana and Plantain Diseases

Feakin, S. D. ed. 1972. Pest Control in Bananas. PANS Manual No. 1. Centre for Overseas Pest Res., London. 128 pp.

Simmonds, N. W. 1966. Bananas. Longman, London. 512 pp.

Stover, R. H. 1972. Banana, Plantain and Abaca Diseases. Commonw. Mycol. Inst., Kew, Surrey. 316 pp.

Wardlaw, C. W. 1972. Banana Diseases Including Plantains and Abaca. Longman, London. 878 pp.

Literature Cited

Food Agriculture Organization. 1981. FAO Production Yearbook. Vol. 35. FAO, Rome. 306 pp.

Calpouzos, L. 1966. Action of oil on the control of plant diseases. Annu. Rev. Phytopathol. 4:369-390.

Gourou, P. 1961. The Tropical World. Wiley, New York. 159 pp.

Magee, C. J. 1927. Investigation on the bunchy top disease of the banana. Aust. Counc. Sci. Ind. Res. Bull. 30:1-64.

Meredith, D. S. 1970. Major banana diseases: past and present status. Rev. Plant Pathol. 49:539-554.

Purseglove, J. W. 1972. Tropical Crops: Monocotyledons. Wiley, New York. 607 pp. 2 Vol.

Rhodes, P. L. 1964. A new banana disease in Fiji. Commonw. Phytopathol. News 10:38-41.

Rowe, P. R., and D. L. Richardson. 1975. Breeding bananas for disease resistance, fruit quality, and yield. Trop. Agric. Res. Serv. Bull. No. 2. La Lima, Honduras. 41 pp.

Sequeira, L. 1962. Special contributions from the tropics to phytopathological mycology. Phytopathology 52:936-942.

Stover, R. H. 1980. Sigatoka leaf spots of bananas and plantains. Plant Dis. 64:750-756.

U.S. Dept. Agric., Econ. Res. Serv. 1975. Fruit situation. TSF-195. 45 pp.

Vakili, N. G. 1969. Bunchy top disease of bananas in the central highlands of South Vietnam. Plant Dis. Rep. 53:634-638.

FUSARIAL WILT OF BANANAS

Causal Agent: *Fusarium oxysporum* f. sp. *cubense* (E. F. Smith) Snyder & Hansen; (Moniliaceae)

Disease Name: Fusarial wilt of bananas is the common name for the disease, but it is also known as Panama disease, *Fusarium* vascular wilt, "mal de Panama", and banana wilt.

Fusarial wilt once ranked among the most devastating tropical diseases. It is one of the most thoroughly studied tropical diseases, and the literature has been exhaustively reviewed (Wardlaw 1972, Stover 1962, 1972, Simmonds 1966, Meredith 1970). The history of the disease was tied closely to the susceptible Gros Michel banana variety, which is now seldom grown and survives in quantity only in Ecuador. Fusarial wilt is of minor importance in areas where cultivars other than Gros Michel are grown, except in southern Taiwan, certain soils in the Canary Islands, and South Africa. In these areas fusarial wilt has caused serious losses on Cavendish varieties (Personal communication - R. H. Stover). The disease has been most important on Gros Michel in the Caribbean and Central America, and of minor importance in Africa, Asia, and the Pacific islands.

Stover (1962) summarized the possible origins of fusarial wilt. It probably originated in Asia and was introduced into the Americas with new varieties. Silk, a susceptible cultivar which was known to be affected with wilt in Australia in 1876, was extensively planted in the Caribbean before Gros Michel was introduced in the early 1800's. Gros Michel plantings expanded onto lands infested by diseased Silk. Planting of infected Gros Michel disseminated the fungus between 1890 and 1910 into many new areas, before the damage the pathogen could cause was realized. Wilt was causing heavy losses by 1914, and through 1960 was the most damaging disease of Gros Michel bananas in the Americas. Since then, resistant Cavendish varieties have replaced Gros Michel, and the disease is no longer of major importance to commercial bananas. Unfortunately, race 2 of the fungus and an insect-transmitted strain of *Pseudomonas solanacearum* have been attacking Bluggoe plantains, one of the most important plantain varieties and sources of food in the tropics of Latin America. Stover and Richardson (1968) described the variety Pelipita that is resistant to the two diseases and is similar to Bluggoe plantains.

The pathogen is present in almost all countries where susceptible banana varieties are grown. The Commonwealth Mycological Institute distribution maps of plant disease and Stover (1962, 1972) give detailed information on distribution of the pathogen. Erwin F. Smith (1910) was the first to isolate the causal organism from Cuban material. He called the fungus *Fusarium cubense*, although he did not see the disease in the field. Brandes (1919) first proved conclusively that *Fusarium cubense* was the cause of banana wilt. With the work of Snyder and Hansen in 1940 on *Fusarium* classification the name was changed to *Fusarium oxysporum* f. sp. *cubense* from the old system of Wollenweber. Stover (1962, 1972) presents a complete discussion of the taxonomy and nomenclature of the pathogen.

Microconidia, macroconidia, and chlamydospores are produced by *F. oxysporum* f. sp. *cubense*. To determine whether a *Fusarium* attacking banana is *F. oxysporum* identification should be made using a key such as those of Toussoun and Nelson (1976) or Booth (1971). A pathogenicity test using bananas is necessary to determine whether the *F. oxysporum* is forma specialis *cubense* (Stover 1972).

Importance

Fusarial wilt of bananas is similar to late blight of potatoes in Ireland in respect to the profound influence it has had on the history, economic well being, and political stability of a region. Aspects of this fascinating story are given by Carefoot and Sprott (1967), McCann (1976), Stover (1962), Wardlaw (1972), and May and Plaza (1958). Fusarial wilt had a devastating effect on the economy of many Caribbean countries and was a major cause of shifting land utilization. Losses caused by wilt are illustrated by figures given by Jones and Morrison(1952). In 1923 Costa Rica exported over 11 million bunches, but in 1941 only 1.4 million bunches. The Panama disease has caused the abandonment of thousands of acres of land which, with improvements and installations, represented investments of $1,000-$2,000 per acre. Stover (1972) states:

> "Over a period of 50 years 100,000 acres of bananas were destroyed or abandoned in Central and South America because of fusarial wilt".

The economic losses due to abandoned plantings, buildings, villages, potential earnings never realized, and costs of maintaining stability in regions devastated by unemployment would be many millions of dollars.

Symptoms

As with many diseases, symptoms of fusarial wilt may vary with plant age, environmental factors, variety, infection with other organisms, and strains of the pathogen. For example, the Santa Marta strain of the pathogen from Colombia often causes wilting without the yellowing characteristic of the disease (Stover 1962). The internal symptoms of the disease can easily be confused with those of the moko disease caused by P. solanacearum. Thus, for a definite diagnosis careful observation of external and internal symptoms should be combined with isolation of the pathogen.

The first external symptom is usually a yellow color in the lowest or oldest leaf petioles. Subsequently, leaves progressively become yellow and wilt, eventually withering and turning brown. Wilting is in succession from the oldest leaf until only the innermost leaf in the psuedostem remains green. The corm may not die for some time, and suckers arising from it seldom become diseased when young, unlike moko disease where suckers become necrotic and blacken. Internal symptoms are seen when an infested corm or pseudostem is cut revealing discolored, light yellow to dark brown vascular strands. The first vascular discoloration is seen in the outer leaf sheaths of the pseudostem with the innermost showing discoloration last.

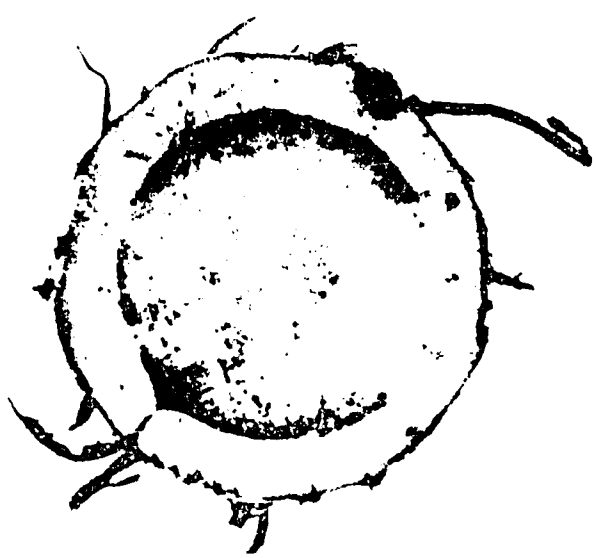


Fig. 35. Panama disease (Fusarium oxysporum f. sp. cubense) in a young banana corm. (Courtesy R. H. Stover).

Host Plants

In nature, outside of *Musa* spp., *Heliconia caribaea* is the only host systemically infected (Waite 1963). Various species of *Heliconia* (Musaceae) are common plants in the jungles of tropical America. Waite (1963) isolated *F. oxysporum* f. sp. *cubense* from *Heliconia caribaea* occurring in the jungle, but it was not highly pathogenic to banana. Isolates from *Heliconia* in areas where "Gros Michel" had been grown were pathogenic to bananas. It appears that some species of *Heliconia* are natural hosts of *F. oxysporum* f. sp. *cubense*, but race 3 of *Fusarium oxysporum* f. sp. *cubense*, which attacks *Heliconia* in virgin jungle, may have originated independently from the isolates that attack banana.

The fungus can invade the roots of various weeds and grasses found associated with bananas, but according to Stover (1972), the pathogen is saprophytic or only slightly parasitic on them. The ability to survive in grasses and weeds may account for the persistence of the fungus in soil without bananas.

Pathogen Cycle

Inoculum of *F. oxysporum* f. sp. *cubense* consists of macroconidia, microconidia, chlamydospores, and mycelia. The fungus survives primarily in the soil where it can exist as chlamydospores for several years. Populations of the pathogen decline naturally in soil, so that 2-3 years after susceptible material is removed, only very low populations are found, but this remaining inoculum is sufficient to cause infection. The most important source of inoculum is planting material (corms or suckers). The fungus can also be transported by man, wind, soil, and infested plant debris. Flood and irrigation water can transport the fungus, and wind can probably carry infested dust and trash a few miles. Any movement of infested soil or plant debris can disseminate the organism.

Chlamydospore germination is stimulated by mechanical wounding of banana roots. Sequeira et al. (1958) noted that mechanical wounding of roots produced a nutritional stimulus to germination and growth of spores. Amino acids, including glutamine, were exuded from wounds. Germination of spores was inhibited on intact, unwounded roots. Stover (1962) has summarized the many studies on spore germination and infection.

Healthy, actively growing main roots are not infected. Almost all infections occur in lateral or branch rootlets, and corms are not infected. Mechanical wounding increases the probability of infection, but is not essential for infection to take place. A response to wounding may be as much nutritional as opening a channel to the xylem (Stover 1962). Several species of nematodes are parasites of banana roots, and studies have been made of the relationship between nematodes and wilt incidence, but Stover (1962) concluded that much more research is necessary before it can be stated that nematodes influence wilt incidence.

There are few tropical plant diseases in which the host's response to infection has been so thoroughly studied (Wardlaw 1961, Stover 1962, 1972, Beckman et al. 1962). Once within the xylem of the root, the fungus is confined to this tissue until an advanced stage of the disease. The fungus stimulates formation of tyloses in some cases and the total collapse and compression of vessels in others. There is also a profuse secretion of gel. Comparing susceptible Gros Michel and resistant Lacatan bananas, Beckman et al. (1962) found growth and sporulation of *F. oxysporum* f. sp. *cubense* similar in both varieties, thus ruling out a biochemical inhibition of the fungus. They concluded that resistance is due to:

"physical occlusion of the plant vascular system which prevents distribution of Fusarium spores within the host."

Perforation plates and vessel endings can trap spores, and gel and tyloses can cause occlusions. Other defense mechanisms are also active in resistant varieties (Stover 1972). Eventually, the fungus grows out of the vascular system into the parenchyma and sporulates profusely, producing conidia and chlamydospores, which are returned to the soil when the plant dies or decays.

In their efforts to control fusarial wilt an immense research effort was made by commercial banana companies to study factors which might have an effect on the disease. Soil pH, soil texture, soil moisture, rotations, soil fertility, soil microorganisms, inoculum potential, and the effects of climate and rainfall on disease severity were among the factors studied. Stover (1962) reviewed this research in his monograph.

Control

Since the disease is present in almost all areas of the world where bananas are grown, exclusion by quarantines is of limited value. Nevertheless, where new plantings are contemplated every effort should be made to obtain disease free "seed". Quarantines were once extensively used to prevent the introduction of Panama disease, but results were not outstanding (Wardlaw 1972 and Stover 1962). A recent report from Taiwan indicates that a race of F. oxysporum f. sp. cubense can attack Cavendish varieties grown there. It is important to exclude this race from other banana-growing areas. Stover (1972) suggested that the susceptibility of Dwarf Cavendish in Taiwan was due to soil conditions and not a new race of the pathogen; however, subsequent studies (Sun et al. 1978) indicate that a new race, designated race 4, is involved.

Various chemicals were used in attempts to eliminate F. oxysporum f. cubense from soil. Some were effective in laboratory or pot-scale experiments, but none were effective in controlling the disease economically under field conditions (Stover 1962).

Dunlap (cited by Stover 1962) in Honduras was the first to successfully use flood fallow for the control of banana wilt. The use of the system increased rapidly from 1945 to 1955 in Honduras and Panama. By 1956 about 15,000 acres in Honduras and 10,000 in Panama had been flood fallowed and planted. Periods of 3-18 months were tested for the period of flooding, but it was found that 6 months was as good as 12 or 18 months. Later work indicated that if the lake was drained and plowed after 3-4 months flooding, and then flooded again for 1-2 months better, control (4-5 years) was obtained (Stover 1962) However, with rising costs of labor, engineering, and equipment the use of flood fallowing became uneconomic. Nevertheless, it did help in keeping large areas in production until resistant varieties could be planted. It can only be used in areas where water is available and level land is suitable for the construction of dykes, lakes, and water retention. Two to five feet of water was needed for flood fallow to be effective. Flood fallowing also apparently destroys the nematode Radopholus similis (Wehunt and Holdeman 1959).

The most effective method of controlling fusarial wilt of bananas is through the use of resistant varieties. The existence of high levels of resistance to F. oxysporum f. sp. cubense in Cavendish varieties has been known for decades, and breeding had been underway since the 1920's to find a substitute for Gros Michel. However, it was believed by those controlling the commercial banana industry in the Americas that Cavendish varieties could not be substituted for Gros Michel. Gros Michel was of excellent quality, highly

resistant to bruising, well adapted to a range of soil conditions, and could be delivered on the stem to markets thousands of miles from where it was grown. It was not plant pathologists, but the post-war emergence of Ecuador as a mass producer of cheap fruit and the conversion to boxing fusarial wilt-resistant Cavendish varieties in Central America that resolved the problem of Panama disease.

Methods were devised for harvesting single bunches of Cavendish varieties such as Valery, treating them with chemicals, packaging them in plastic, boxing them in the plantation where they were grown, and rapidly transporting them to markets. The U.S. market quickly adapted to different handling methods required for Cavendish varieties shipped in cartons, contrasted to Gros Michel shipped as stems. Essentially, a variety change was made and accepted in the United States without a public realization.

The edible varieties of banana are seedless and, as most of them are highly sterile, breeding bananas for resistance to F. oxysporum f. sp. cubense, combined with acceptable agronomic and quality factors, has been most difficult, expensive, and time consuming. Efforts to breed for resistance began in the early 1920's in Jamaica, and Larter (1947), Stover (1962, 1972), Simmonds (1966), and Rowe and Richardson (1975) describe these efforts in detail. Larter (1947) stated that resistance is inherited as a dominant character and is relatively easy to incorporate into seedlings from a resistant male crossed to Gros Michel. Fifty percent or more of the progeny from such crosses were resistant. There was no rapid method for evaluating resistance in these studies, and a year was required to tell whether a seedling was resistant. Later, Vakili (1965) described a rapid method of inoculating seedlings in a program of screening for resistance. Since three races of the pathogen (races 1, 2, and 4) have been identified as attacking both dessert and cooking bananas, testing of potential new varieties must be extensive.

Literature Cited

Beckman, C. H., S. Halmos, and M. E. Mace. 1962. The interaction of host, pathogen and soil temperature in relation to susceptibility to Fusarium wilt of bananas. Phytopathology 52:134-140.

Booth, C. 1971. The Genus Fusarium. Commonw. Mycol. Inst., Kew, Surrey. 237 pp.

Brandes, E. W. 1919. Banana wilt. Phytopathology 9:339-389.

Carefoot, G. L., and E. R. Sprott. 1967. Famine on the Wind. Rand McNally, Chicago. 231 pp.

Jones, C. F., and P. C. Morrison. 1952. Evolution of the banana industry in Costa Rica. Econ. Geogr. 28:1-19.

Larter, L. N. H. 1947. Report on banana breeding. Dept. Agric. Jamaica Bull. 34. 24 pp.

May, S., and G. Plaza. 1958. The United Fruit Company in Latin America. Natl. Planning Assoc., Washington, DC. 263 pp.

McCann, T. P. 1976. An American Company: the Tragedy of United Fruit. Crown, New York. 244 pp.

Meredith, D. S. 1970. Major banana diseases: past and present status. Rev. Plant Pathol. 49:539-554.

Rowe, P. R., and D. L. Richardson. 1975. Breeding bananas for disease resistance, fruit quality, and yield. Trop. Agric. Res. Serv. Bull. No. 2. La Lima, Honduras. 41 pp.

Sequeira, L., T. A. Steeves, M. W. Steeves, and J. M. Riedhart. 1958. Role of root injury in Panama disease infections. Nature 182:309-311.
Simmonds, N. W. 1966. Bananas. Longman, London. 512 pp.
Smith, E. F. 1910. A Cuban banana disease. Science 31:754-755.
Snyder, W. C., and H. N. Hansen. 1940. The species concept in Fusarium. Amer. J. Botany 27:64-67.
Stover, R. H. 1962. Fusarial Wilt (Panama disease) of Bananas and Other Musa Species. Paper No. 4. Commonw. Mycol. Inst., Kew, Surrey. 117 pp.
Stover, R. H. 1972. Banana, Plantain and Abaca Diseases. Commonw. Mycol. Inst., Kew, Surrey. 316 pp.
Stover, R. H., and D. L. Richardson. 1968. "Pelipita", an ABB Bluggoe-type plantain resistant to bacterial and fusarial wilts. Plant Dis. Rep. 52:901-904.
Sun, E. J., H. J. Su, and W. H. Ko. 1978. Identification of Fusarium oxysporum f. sp. cubense race 4 from soil or host tissue by cultural characters. Phytopathology 68:1672-1673.
Toussoun, T. A., and P. E. Nelson. 1976. A pictorial guide to the identification of Fusarium species according to the taxonomic system of Snyder and Hansen. Penn. State Univ. Press, University Park, PA. 43 pp.
Valkili, N. G. 1965. Fusarium wilt resistance in seedlings and mature plants of Musa species. Phytopathology 55:135-140.
Waite, B. H. 1963. Wilt of Heliconia spp. caused by Fusarium oxysporum f. cubense Race 3. Trop. Agric. (Trinidad) 40:299-305.
Wardlaw, C. W. 1972. Banana Diseases Including Plantains and Abaca. Wiley, New York. 878 pp.
Wehunt, E. J., and Q. L. Holdeman. 1959. Nematode problems of the banana plants. Proc. Soil Crop Sci. Soc. Florida 19:436-442.

MOKO DISEASE OF BANANAS AND PLANTAINS

Disease Name: Moko disease, bacterial wilt disease

Causal Organism: *Pseudomonas solanacearum* E.F.S.; Race 2. (Pseudomonadaceae)

P. solanacearum is one of the most fascinating of all plant pathogens, especially to those with tropical interests. It is necessary to differentiate between the Moko disease of bananas (caused by race 2) and the bacterial wilt disease in general which is known to attack over 300 different plant species, including many food or fiber crops of great economic importance. Erwin F. Smith (1896) was the first to adequately describe bacterial wilt disease and its causal agent. His studies were done with tomatoes.

The bacterial wilt disease was observed about the same time on many different hosts in widely separated parts of the world in the late ninteenth century. It is strange that it was not recognized as a serious problem before this time, as it is difficult to believe it began almost simultaneously in such widely separated areas as Europe (1884), the United States (1890), Indonesia (1892), Japan (1881), and India (1892) (Kelman 1953). Probably the earliest record of bacterial wilt on bananas (Moko disease) was by Schomburgk in 1840 (Martyn 1934), but the first study was on bananas and plantains in 1901 by Rorer (1911). He studied the disease in Trinidad, and reported that it almost eliminated the Moko plantain about 1890. Moko disease has subsequently been found in many of the tropical and sub-tropical areas of the Americas where bananas are grown.

Rorer's (1911) description of the disease in Trinidad is worth study. The "Moko" plantain was a hardy and well adapted variety which was widely used in Trinidad as a shade tree for cacao plantations. These cooking bananas were important to the people of the island and were consumed baked, boiled, fried, or pounded. About 20 years before Rorer's visit an epidemic had practically eliminated the "Moko" variety. When Rorer was there, the disease was also attacking other banana varieties including those in the Cavendish group. He also found surviving plants of the "Moko" plantain. His descriptions of the disease leave no doubt that he was describing the Moko disease caused by *P. solanacearum*. Forsyth (1967) states that the banana industry in Trinidad was destroyed by the Moko disease.

Moko disease is caused by race 2 of *P. solanacearum*. Thus, although the bacterial species is found worldwide, Moko disease is restricted to the Americas and the Philippines where it was introduced from Central America (Rillo 1979). Buddenhagen (1961) and

Fig. 36. Effect of *Pseudomonas solanacearum* (Moko disease) on raceme of plantain.

the CMI plant disease distribution maps give comprehensive information on the distribution of the disease. Race 1 does not normally attack commercial bananas; symptoms caused by race 1, if they do occur, are not identical to the Moko disease induced by race 2 (Buddenhagen 1961, 1968, Zehr 1970). Infection of bananas by P. solanacearum had been reported before the 1960's occasionally in localized areas in Africa, Asia, and the Pacific Islands, but the validity of these reports as being of Moko disease has been questioned by Buddenhagen (1961, 1968) either because the symptoms described were not typical of the Moko disease, or because the pathogenicity of the bacterium was not properly established. Although Chattopadhyay and Mukhopadhyay (1968) reported Moko disease in India, it probably was not caused by race 2 of P. solanacearum. The Moko disease still has a restricted distribution in tropical America, but the insect-transmitted strain is still spreading and has been found in four new Central American countries since 1961 (Stover 1972). The disease also is moving into the Amazon basin from Peru (French and Sequeira 1970).

Erwin F. Smith in 1896 was the first investigator to give an adequate description of P. solanacearum and to demonstrate its pathogenicity (Kelman 1953), although others had probably been working with the same organism. Rorer (1911) described the Moko disease on bananas and plantains in Trinidad and proposed the name Bacillus musae for the causal agent. He noted similarities to his organism and Bacillus solanacearum (P. solanacearum) but was unable to infect solanaceous hosts, so he considered his bacterium a distinct species. Ashby (1926) showed that B. musae was sufficiently similar to P. solanacearum, attacking solanaceous plants and bananas, to be classified as P. solanacearum. Kelman (1953) discussed the history and taxonomy of the organism in detail. Hayward (1964) gives additional information on the characteristics of P. solanacearum in culture.

Buddenhagen, Sequeira, and Kelman (1962) have described 3 races of the pathogen. Buddenhagen and Kelman (1964) in a review article discuss the problems of race and strain separation. Three major races are designated on the basis of hosts attacked:

> "race 1, affecting solanaceous and other plants; race 2, affecting bananas and Heliconias; and race 3 affecting potato. Within each race there are numerous known and unknown pathotypes differing in each area".

Buddenhagen and Elasser (1962) and Buddenhagen and Kelman (1964) describe a highly virulent strain of race 2 that oozes more readily from diseased banana tissue and that is primarily insect-transmitted. The strain is designated as SFR (small, fluidal, round), and, on the basis of colony characteristics, it can be differentiated from the common banana (race 2) strain on Kelman's TTC medium. Stover (1972), based on work by Buddenhagen, lists four major strains of race 2 designated D, B, SFR and H.

Hayward (1964, 1976) classified 185 isolates of P. solanacearum into four major biochemical types, called "biotypes". His biotypes do not, in general, agree with the "races". Therefore they are not biotypes in an ecological or epidemiological sense. Hayward's basis for separation was on carbohydrate utilization in culture.

The Pathogen in Culture

Isolations are made by streaking dilute bacterial suspensions on Kelman's TTC (triphenyl tetrazolium chloride) medium (Kelman 1954). P. solanacearum can

be identified on this medium by its morphological characteristics when examined with a binocular microscope using obliquely transmitted reflected light. The bacterium rapidly produces mutants in culture, and pathogenic colonies are easily differentiated on TTC medium from non-pathogenic or weakly pathogenic mutants (Husain and Kelman 1958).

Since the symptoms of Moko are so easily confused with those of the fusarial wilt disease (Fusarium oxysporum f. sp. cubense), it is important to isolate the causal agent if doubt exists regarding identification. The race causing disease in bananas can be distinguished on the basis of morphological characters on TTC media from P. solanacearum races attacking other hosts (Buddenhagen and Kelman 1964). In addition, the insect transmitted strain from bananas can be differentiated morphologically from the common banana strain on TTC (Buddenhagen and Elasser 1962).

Importance

Moko disease caused almost total destruction of the "Moko" plantain in Trinidad in the late 19th Century (Rorer 1911), and serious losses as early as 1840 in Guyana (Buddenhagen 1958). A highly virulent insect-disseminated strain (Buddenhagen and Elasser 1962) caused an epidemic that devasted plantains in Central America in the 1960s with a loss of millions of plants. Another insect-disseminated strain has spread rapidly in Colombia (Lozano et al. 1969) and more recently into the Amazon jungle of Peru (French and Sequeira 1970), where the disease threatens a major food source for thousands of people. Buddenhagen (1968) estimated that the disease eliminated susceptible plantains (the Moko or Bluggoe cultivar) from thousands of square miles in Latin America. However, after the first sweep of the epidemic, survivors became reestablished (personal communication - I. W. Buddenhagen). The bacterium can be spread rapidly by man through mechanical means, but the insect-disseminated strain of P. solanacearum can spread much more rapidly.

The disease is a major problem of commercial bananas, not so much for the direct losses it causes, but for the expensive preventive measures used to keep it under control (Stover 1972). The Moko plantain is grown primarily by small farmers in Latin America and the Caribbean where it is, or has been, a major source of food. Losses have been highest on plantings made by small farmers.

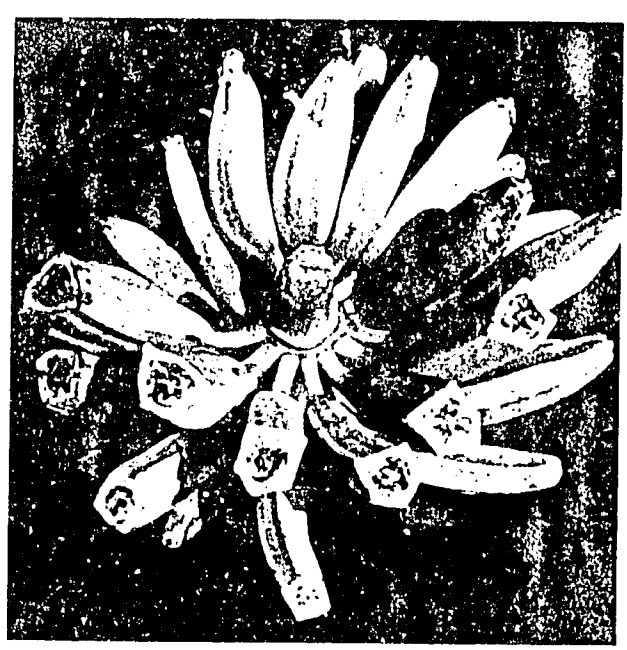


Fig. 37. Symptoms of moko caused by Pseudomonas solanacearum on plantains.

Symptoms

Symptoms of Moko disease may easily be confused with those of fusarial wilt, so care should be taken before a positive identification is made. Symptoms produced when the bacterium enters the plant by mechanical wounding of cut suckers are different than those produced when the bacterium is transmitted to the male

inflorescence. When insect-transmitted infection occurs, frequently no external symptoms are noticed until fruit is produced, although the male flower bud on the variety Bluggoe may blacken and shrivel. The fruit often becomes yellow prematurely, and when cut internally, is firm but has a brown dry rot. Eventually the entire raceme may rot and turn black. Vascular discoloration ranging from light to dark brown is usually found throughout the plant. Bacteria often ooze from cut surfaces. Young plants may turn partially yellow and wilt, whereas young regrowth suckers have split and blackened leaf sheaths. Sequeira (1958), Buddenhagen (1961), and Stover (1972) give detailed descriptions of symptoms.

Host Plants

Pseudomonas solanacearum attacks more than 200 different species of plants (Buddenhagen and Kelman 1964). Common economic hosts are banana, plantain, tobacco, potato, tomato, pepper, eggplant, and peanut. In addition, many ornamentals and weeds are infected. Kelman (1953) gives a comprehensive summary of the host range of *P. solanacearum*. Buddenhagen, Sequeira, and Kelman (1962) have separated the isolates of *P. solanacearum* into 3 races on the basis of host range.

> "Race 1 affects tobacco, tomato, many solanaceous and other weeds, and certain diploid bananas. Race 2 causes bacterial wilt of triploid bananas (moko disease), Heliconia, or both. Five subraces or groups of races 1 and 2 can be distinguished. Race 3 affects potatoes and tomatoes, but is not highly virulent on other solanaceous crops."

Nearly all banana and plantain varieties are susceptible to race 2 if inoculated, but some more than others. Several species of *Heliconia* (a commonly occurring weed in the jungle related to the banana) have been found to be naturally infected in virgin jungle (Sequeira and Averre 1961) in Costa Rica. Race 2 of *P. solanacearum* was consistently isolated from diseased *Heliconia* and was able to attack bananas. Buddenhagen (1960), Belalcazar et al. (1968), and Berg (1971) report additional weed hosts of race 2. Much more work is needed to clarify the role of other suscepts in the epidemiology of the Moko disease. Race 1 of *P. solanacearum* is often present in banana plantations and may invade the roots of bananas. It seldom causes disease, however, because normal defense reactions keep it from establishing itself (Buddenhagen 1965).

Pathogen Cycle

Inoculation of bananas occurs whenever the bacterium comes into contact with the vascular elements of the root system, is introduced through wounds, or is carried to the vascular system of the inflorescence by insects. Detailed studies of the mode of entry of this pathogen into banana and plantain in the field are not available. Kelman (1953) summarizes much of the knowledge to date with other suscepts. Wounding and high levels of inoculum increase chances for root infection. Under field conditions infection of stems or foliage without handling has not been observed.

Root-to-root infection occurs with tomatoes (Kelman and Sequeira 1965). Some investigators have noticed a correlation between high wilt incidence in bananas and nematode infestation (Buddenhagen and Kelman 1964), but this needs confirmation. *P. solanacearum* can enter the roots of bananas, but this method of entry is not as common as in other hosts, and wounds are probably necessary

(Sequeira 1958). Wounds made with the tools used in cultural operations are the main entry for the bacterium into commercial bananas (Sequeira 1958). In banana and plantain plantings such cultural operations as removing leaves and suckers, harvesting fruit, and removing flower buds cause wounds and may transmit the bacterium. Sucker pruning is probably most important. Root-to-root spread also occurs with bananas. Transmission also occurs by use of infected seed pieces.

In 1962 Buddenhagen and Elasser showed that insects were an important means of dissemination in Bluggoe plantain. Bacteria become accessible for transmission by breaking through the peduncle, emerging through open surfaces on the inflorescence, through cracks in diseased fruit, and at surfaces of cut suckers (Buddenhagen and Kelman 1964). The source of bacteria for insect transmission is from the male part of the banana inflorescence which may ooze bacteria for as long as three months. When the bracts of the male flower abscise, bacteria ooze from diseased peduncles and from bases of bracts in milky white droplets. Buddenhagen and Elasser (1962) state:

> "Large numbers of bees, wasps, and other insects normally frequent banana flowers and the moist surface on the peduncle. Fifty species of insects have been collected from inflorescences from one location, and up to 100 bees and wasps per hour have been counted frequenting a single inflorescence".

Five percent of the insects were carrying the bacterium as determined by direct isolation techniques. Bagging experiments showed the disease did not occur if insects were excluded from the inflorescence.

Kelman (1953) and Buddenhagen and Kelman (1964) have discussed at length the work on the mechanism of wilt induction and the role of growth substances after infection. Beckman et al. (1962) and Buddenhagen and Kelman (1964) have studied wilting due to P. solanacearum. As regards wilting, Buddenhagen and Kelman (1964) state:

> "Movement of water in vessels declines as viscosity of vessel fluid increases because of formation of extracellular slime (polysaccharides) surrounding masses of bacterial cells. Secondary causes of reducing water movement include tylose formation and vessel collapse, but their role has not been clearly defined. Other factors, including the action of cellulolytic enzymes and possibly pectic enzymes, may be involved but are not considered to play an essential role in wilting per se."

Sequeira and Kelman (1962) found that levels of indole-3-acetic acid (IAA) increased greatly in susceptible bananas when infected. Synthesis of IAA and ethylene (Freebairn and Buddenhagen 1964) may, even at low concentrations, provide the bacterium with the means of creating favorable conditions for development in host tissue during the period of invasion (Buddenhagen and Kelman 1964). Subsequent to infection, the vascular system of succulent plants is degraded. In young tomato or potato plants infected by P. solanacearum, cells in the xylem, phloem, and later in the adjacent pith and cortex are macerated. Hussain and Kelman (1958) have shown that pectin-methylesterase and polygalacturonase degrade the pectic substances of the middle lamella. Degradation of cellulose in the cell walls appears due to cellulase (Kelman and Cowling 1964).

Little is known of the effect of environment on the severity of Moko disease in the hot tropics since inoculation and infection take place in the roots, through mechanically induced wounds, or with transmission by insects to the male inflorescence. Low temperatures will slow or reduce disease development. High soil moisture probably favors the disease by increasing the survival and spread of the bacterium in the soil and increasing the amount of infection.

Control

Since the Moko disease does not occur in many continents and countries which are important producers of bananas, every effort should be made by governments and growers to prevent its introduction by appropriate quarantines. Unfortunately, the spread of the disease by insects cannot be halted at political boundaries, and the disease (at least in tropical America) will probably eventually spread to all important banana and plantain growing areas. Governments in Africa and Asia should be especially careful to protect themselves from the insect-transmitted strains of the bacterium. Tissue-cultured banana plantlets are now available and have been used to start plantings in new areas. They should be safe for intercontinental shipments when properly monitored.

Almost all of the literature has indicated that P. solanacearum can survive in the soil in the absence of host plants (Kelman 1953). Sequeira, however, has shown that bacterial wilt of bananas can be effectively controlled in infested soils fallowed for 24 months. Weed fallow was as effective as rotation with tropical kudzu. Infections after 18 months were only fractions of a percent. These studies (Sequeira 1962) show that survival in soil of the banana strain is relatively poor. Buddenhagen (1965) discusses some details of the survival of race 2 of P. solanacearum in soil. It is possible that the rather erratic results obtained by some investigators regarding survival of the bacterium in soil were due to failure to completely eliminate susceptible weed hosts in their survival experiments. A completely clean fallow is necessary for effective elimination of the bacterium from soil.

Sanitation is the principal control method for Moko disease. The monthly pruning of suckers with machetes is (with the exception of insect and seed transmission) the principal means of the rapid spread of the bacterium. Buddenhagen and Sequeira (1958) showed that formaldehyde was the most effective chemical for use in sterilizing machetes and between pruning suckers. Other more economical chemical disinfectants are now used. A thorough tool disinfestation program will give almost complete control of the disease in horn plantains (AAB) which are not vulnerable to insect borne transmission of P. solanacearum.

Control of the disease in the Bluggoe (ABB) plantain can be obtained with a combination of sanitation and removal of the male inflorescence (Buddenhagen and Elasser 1962, Lozano et al. 1967). If the male inflorescence is broken off by hand before the male flowers and bracts absciss or are cut off by a machete properly disinfested with formaldehyde or other disinfectant, excellent control can be obtained. The male part of the inflorescence serves no useful function. In an experiment made in the Tolima Valley of Colombia, 328 racemes were harvested from 300 plants in plots where sanitation and male inflorescence removal was practiced (10 months after inoculation of 2 plants in the center of the plots), and only 46 racemes were collected from the plots where no control was practiced. This is an 85% reduction of yield. The cost of the treatment was about one dollar per hectare (Lozano et al. 1967).

No resistance to P. solanacearum is known among edible banana varieties, but a single resistant diploid has been found (Buddenhagen and Kelman 1964). Stover and Richardson (1968) report a bluggoe-type plantain, 'Pelipita', resistant to bacterial and fusarial wilts. They suggest that it might replace the bluggoe-type plantain in areas of Central and South America where the SFR strain of P. solanacearum is destructive. Pelipita is resistant because it retains the male flowers, and thus no infection sites are produced.

An integrated control which includes sanitation, removal of the male inflorescence, and resistant varieties is successfully applied in the coffee growing regions of Colombia. Similar control measures are used in the north coast of Colombia, but additionally, infected plants and surrounding neighbors are killed with herbicides to eliminate foci of infection.

Literature Cited

Ashby, S. F. 1926. A bacterial wilt disease of bananas in Trinidad caused by Bacterium solanacearum E. F. Smith. Trop. Agric. (Trinidad). 3:127-129.

Beckman, C. H., W. A. Brun, and I. W. Buddenhagen. 1962. Water relations in banana plants infected with Pseudomonas solanacearum. Phytopathology 52:1144-1148.

Belalcazar, S., G. Uribe, and H. D. Thurston. 1968. Reconocimiento de hospedantes a Pseudomonas solanacearum E. F. Sm., en Colombia. Fitotecnia Latinoamericana 5:89-99.

Berg, L. A. 1971. Weed hosts of the SFR strain of Pseudomonas solanacearum, causal organism of bacterial wilt of bananas. Phytopathology 61:1314-1315.

Buddenhagen, I. W. 1960. Strains of Pseudomonas solanacearum in indigenous hosts in banana plantations of Costa Rica, and their relationship to bacterial wilt of bananas. Phytopathology 50:660-664.

Buddenhagen, I. W. 1961. Bacterial wilt of bananas: history and known distribution. Trop. Agric. (Trinidad) 38:107-121.

Buddenhagen, I. W. 1965. The relation of plant-pathogenic bacteria to the soil. pp. 269-284. In Baker, K. F. and W. C. Snyder. eds. Ecology of Soil-Borne Plant Pathogens. Univ. of California Press, Berkeley, CA. 571 pp.

Buddenhagen, I. W. 1968. Banana diseases in the Pacific area. Food Agric. Organ. Plant Protec. Bull. 16(2):17-31.

Buddenhagen, I. W., and L. Sequeira. 1958. Disinfectants and tool disinfection for prevention and spread of bacterial wilt of bananas. Plant Dis. Rep. 42:1399-1404.

Buddenhagen, I. W., and T. A Elasser. 1962. An insect -- spread bacterial wilt epiphytotic of Bluggoe banana. Nature 194:164-165.

Buddenhagen, I. W., L. Sequeira, and A. Kelman. 1962. Designation of races in Pseudomonas solanacearum. Phytopathology 52:726. (Abstr.).

Buddenhagen, I. W., and A. Kelman. 1964. Biological and physiological aspects of bacterial wilt caused by Pseudomonas solanacearum. Annu. Rev. Phytopathol. 2:203-230.

Chattopadhyay, S. B., and N. Mukhopadhyay. 1968. Moko disease of banana: a new record. Food Agric. Organ. Plant Protec. Bull. 16:52.

Forsyth, W. G. C. 1967. Farming systems: plantations or small landholders. pp. 126-129. In Turk, K. L. and L. V. Crowder. eds. Rural development in tropical Latin America. NY State Coll. Agric. Cornell Univ., Ithaca, NY.

Freebairn, H. T., and I. W. Buddenhagen. 1964-65. Ethylene production by Pseudomonas solanacearum. Nature 202:313-314.

French, E. R., and L. Sequeira. 1970. Strains of Pseudomonas solanacearum from Central and South America: a comparative study. Phytopathology 60:506-512.

Hayward, A. C. 1964. Characteristics of Pseudomonas solanacearum. J. Appl. Bacteriol. 27:265-277.

Hayward, A. C. 1976. Systematics and relationships of Pseudomonas solanacearum. pp. 6-21. In Proceedings of the first international planning conference and workshop on the ecology and control of bacterial wilt caused by Pseudomonas solanacearum. Sequeira, L. and A. Kelman. eds. North Carolina State Univ., Raleigh.

Husain, A., and A. Kelman. 1958. Relation of slime production to mechanism of wilting and pathogenicity of Pseudomonas solanacearum. Phytopathology 48:155-165.

Kelman, A. 1953. The bacterial wilt caused by Pseudomonas solanacearum. North Carolina Agric. Exp. Stn. Tech. Bull. 99. 194 pp.

Kelman, A. 1954. The relationship of pathogenicity in Pseudomonas solanacearum to colony appearance on a tetrazolium medium. Phytopathology 44:693-695.

Kelman, A., and L. Sequeira. 1965. Root-to-root spread of Pseudomonas solanacearum. Phytopathology 55:304-309.

Kelman, A., and E. B. Cowling. 1965. Cellulase of Pseudomonas solanacearum in relation to pathogenesis. Phytopathology 55:148-155.

Lozano T., J. C., H. D. Thurston, G. E. Galvez E. 1969. Control del "Moko" del platano y banano causado por la bacteria Pseudomonas solanacearum. Agric. Trop. (Colombia) 25:315-324.

Martyn, E. B. 1934. A note on plantain and banana diseases in British Guiana with special references to wilt. Agric. J. Brit. Guiana 5:120-183.

Rillo, A. R. 1979. Bacterial wilt of banana in the Philippines. Food Agric. Organ. Plant Prot. Bull. 27:105-108.

Rorer, J. B. 1911. A bacterial disease of bananas and plantains. Phytopathology 1:45-49.

Sequeira, L. 1958. Bacterial wilt of bananas: dissemination of the pathogen and control of the disease. Phytopathology 48:64-69.

Sequeira, L., and C. W. Averre III. 1961. Distribution and pathogenicity of strains of Pseudomonas solanacearum from virgin soils in Costa Rica. Plant Dis. Rep. 45:435-440.

Sequeira, L. 1962. Control of bacterial wilt of bananas by crop rotation and fallowing. Trop. Agric. (Trinidad). 39:211-217.

Sequeira, L., and A. Kelman. 1962. The accumulation of growth substances in plants infected by Pseudomonas solanacearum. Phytopathology 52:439-448.

Smith, E. F. 1896. A bacterial disease of the tomato, eggplant and Irish potato (Bacillus solanacearum nov. sp.). U.S. Dept. Agric., Div. Vegetable Physiol. Pathol., Bull. 12:1-28.

Stover, R. H., and D. L. Richardson. 1968. 'Pelipita', an ABB Bluggoe-type plantain resistant to bacterial and fusarial wilts. Plant Dis. Rep. 52:901-903.

Stover, R. H. 1972. Banana Diseases. Commonw. Mycol. Inst., Kew, Surrey, England. 316 pp.

Zehr, E. I. 1970. Isolation of Pseudomonas solanacearum from abaca and banana in the Philippines. Plant Dis. Rep. 54:516-520.

Beverage Crops

Coffee, cocoa, and tea are the major non-alcoholic beverages of the world. All might be characterized as "cash" or plantation crops, but they are essential to the economies of many tropical countries and often are grown primarily by small-scale farmers. Fruit juices are also important beverages.

COFFEE

Coffee (Coffea sp.) is grown almost exclusively in tropical countries, and is the major export crop of many tropical Latin American countries such as Brazil, Colombia, Mexico, and Costa Rica. Brazil is the largest producer with about half of the world's crop. Coffee is growing in importance as an export crop in Africa, and several Asian countries are also important producers. World production of coffee in 1981 was 5.9 million tons (FAO 1981).

About 60 species of Coffea are found, although the taxonomy of the genus is confusing. Arabica coffee (C. arabica) is the most important species and originated in the highlands of Ethiopia, where it is still found growing wild. C. canephora (robusta coffee) is the next most important species, and a small amount of C. liberica is also grown commercially. The major use of coffee is as a delicious, stimulating drink. Coffee consumption as a beverage probably began before the 15th century in Arabia and gradually spread from there to Europe and Asia. Europeans carried plants to the Americas, Asia, and Africa for cultivation. The majority of the coffee grown in the world today may be descended from material thought to have been taken from Yemen in the late 17th century. Progeny of this material was taken to Indonesia and back to Europe from where it was distributed to the Americas (Purseglove 1968). There probably is little genetic variability in C. arabica plantings worldwide.

The genetic uniformity of coffee has important implications for both plant pathologists and breeders, since the potential for epidemics in plantations of genetically uniform crops is high. When this uniformity extends over a nation or a continent epidemics can be serious, as in the case of coffee rust. From the breeding standpoint there is probably little exploitable diversity among available cultivars, especially for disease resistance. The best sources of resistance will probably be found in the Ethiopian center of origin.

C. arabica favors an average temperature of 24°C and is grown between latitudes of 26°N and S and altitudes of 1200-2200 m. C. canephora requires higher average temperatures and more rain. Robusta coffee can be grown between 10°N and S at low altitudes. Coffee is generally grown from seed in nurseries,

and transplanted to the field. Vegetative propagation by cuttings can also be used for important cultivars. Arabica coffee is an evergreen shrub or tree which may grow to 5 meters without pruning. The first crop generally is harvested 3-4 years after planting, and under ideal conditions trees may bear for half a century or more. Although most of the world's coffee is planted without shade, many areas still cultivate coffee with shade. Coffee without shade demands a higher level of management.

The coffee fruit is a berry (botanically a drupe), with the only usable portion being the hard seed. Two processing procedures are used. In dry processing, the picked berries are sun dried, and subsequently the seeds are mechanically extracted from the dry pulp and parchment. In the wet process the seeds are squeezed out of the fruit and the mucilagenous coating removed by fermentation in water for one or two days. The seeds are dried and hulled before transport.

Fig. 38. Coffee pickers in Costa Rica.

Coffee rust is undoubtedly the most serious disease of Arabica coffee. Two species of fungi cause rust: Hemileia vastatrix and H. coffeicola (found only in Africa). Anthracnose (Colletotrichum coffeanum) also causes considerable losses. A specialized form of the fungus causes coffee berry disease in East Africa which has been highly destructive. American leaf spot of coffee (Mycena citricolor) occurs only in the Americas. Wellman (1961) cites losses of 75% in some areas, and states that Costa Rica once had a 20% annual loss due to M. citricolor. Improved coffee culture and the use of eradicant sprays have reduced losses in recent decades. Cercospora coffeicola causes brown eye spot on leaves which leads to defoliation and fruit decay. The disease is more severe on trees

Fig. 39. Coffee bush in Mexico.

grown in the sun than those in the shade.

Selected References on Coffee Diseases

Coffee Research Foundation. 1977. An Atlas of Coffee Pests and Diseases. Coffee Board of Kenya, Ruiru, Kenya. 146 pp.

Cook, A. A. 1978. Diseases of Tropical and Subtropical Vegetables and Other Plants. Hafner, New York. 381 pp.

Wellman, F. L. 1961. Coffee: Botany, Cultivation and Utilization. Interscience, New York. 482 pp.

Literature Cited

Food Agriculture Organization. 1981. FAO Production Yearbook. Vol. 35. FAO, Rome. 306 pp.

Purseglove, J. W. 1968. Tropical Crops, Dicotyledons. Wiley, New York. 719 pp. 2 Vol.

COFFEE RUST

Disease Name: Coffee rust is the most common name of the disease. Other names have been the oriental leaf disease, orange rust, common rust, oriental rust, and coffee leaf disease.

Causal Organism: *Hemileia vastatrix* Berk. and Br.; (Pucciniaceae)

The history of the coffee rust disease is well documented and one of the most fascinating in tropical plant pathology. Large (1940), and more recently Carefoot and Sprott (1967), have written popular histories of the disease. Cramer (1957), Wellman (1961), Scheiber (1972, 1975), and Waller (1982) also discuss the history of the disease. Coffee rust was first noted in 1861 on wild coffee near Lake Victoria in East Africa (Scheiber 1972). It was next found in Sri Lanka in 1869 and a century later caused great consternation in the Americas when it was found in January 1970 near Itabuna, Brazil. The fungus had been found in the Americas (Puerto Rico) once before in 1903, but was eradicated (Wellman 1961). *H. vastatrix* was also successfully eradicated from Papua, New Guinea in 1965 (Holliday 1980).

H. Marshall Ward was sent as a young man to Sri Lanka to work on coffee rust. He spent two years there, and his studies of *H. vastatrix* (Ward 1882) still make interesting reading today. Although he was unable to make recommendations that could save the industry, he was the first plant pathologist to state clearly the principle of protection in chemical control, ie. that the chemical must be present on leaves and kill the fungus before it has time to penetrate the leaf or the chemical will be ineffective (Horsfall 1945).

Although discovered in 1869, by 1890 nearly 90% of the area planted to coffee was abandoned and Sri Lanka gradually changed to growing tea (Cramer 1957). The disease was subsequently found in other countries of Asia. In the Philippines 35% of the crop was lost to *Hemileia* in 1891, and later coffee growing was almost abandoned there due to rust. In Indonesia, although some coffee production was maintained by planting robusta coffee, most of the coffee plantations were replanted to rubber trees. From available evidence, it appears that coffee rust probably originated in Ethiopia where coffee is indigenous (Wellman 1961).

H. vastatrix is widely distributed in Asia, Africa, and the Pacific Islands. It was found in Brazil in 1970, rapidly spread to adjacent countries in South America, and is now found in Argentina, Bolivia, Colombia, Ecuador, Paraguay, and Peru. In 1976 it was found in Nicaragua, and since then has spread to Mexico and Central America.

Berkely and Broome were the first to describe the fungus. Ward (1882) gave conclusive proof of the pathogenicity of *H. vastatrix* in some of the earliest scientific investigations ever made in the tropics. The fungus belongs to the order Uredinales and the family Pucciniaceae. Many races of *H. vastatrix* have been described to date on the basis of their reaction on differential hosts of *C. arabica* and certain *Coffea* species (Coffee Rust Research Center 1971). Much of the early work on physiological specialization was done in Oeiras, Portugal, by D'Olivera at the Coffee Rust Research Center (D'Olivera 1965). Resistance to *H. vastatrix* is found both in *C. arabica* and in other *Coffea* species. A second rust of coffee, sometimes known as grey rust, is caused by *H. coffeicola*. Although now restricted to Africa, it should be emphasized that no *C. arabica* clone exists with resistance to *H. coffeicola*. Rayner (1960b), Nutman and Roberts (1970), Rodrigues et al. (1975),

and Eskes (1983) discuss physiological specialization of H. vastatrix. Ten years after the introduction of rust into Brazil, 10 races were found in coffee breeding plots near Sao Paulo, Brazil (Eskes and Toma-Braghini 1981). Eskes (1982) states that all the C. arabica grown in Central and South America is susceptible to race II of H. vastatrix.

The urediospores produced by H. vastatrix are kidney-shaped, echinulate on their convex face, and smooth on the concave face. Kranz (1977) reports urediospore size as 28-36 x 18-28 m. Spores are hyaline with orange inclusions. Laundon and Waterson (1964) and Holliday (1980) give detailed descriptions of the sori, urediospores, and teliospores of the fungus.

Importance

Large (1940) in his book "The Advance of the Fungi" considers coffee rust to be one of the seven major pests or diseases of the last century. The disease essentially wiped out a considerable coffee industry in Sri Lanka, Indonesia, and the Philippines. In most areas where coffee is grown in the tropics today, it is necessary to spray with fungicides to control the disease. The immense cost of spraying must be considered a direct loss due to the disease.

> "The introduction of Hemileia vastatrix into the Western Hemisphere could have very serious repercussions, especially on the economies of Brazil, Colombia, and the Central American republics. Even if the rust did not cause the devastation it did in Sri Lanka its introduction and establishment would probably force growers to spray, and this additional cost might mean ruin to many thousands of coffee growers".

I wrote the above paragraph for an earlier version of this book in 1968. Unfortunately, as predicted, many marginal growers in Brazil and Central America have abandoned growing coffee since rust became established. Monaco (1977) has estimated that yield losses in Brazil would be 30% if no control measures were used.

Coffee is a major source of foreign exchange for many Central and South American countries. For example, in 1981 coffee represented 60% of the value of Colombia's exports (Colombia Today 1981). Coffee rust, recently found in Colombia, may cause serious economic and political problems. Scheiber (1972) analyzes the possible economic impact of H. vastatrix on the Americas and, to illustrate the importance of coffee to Latin America, quotes from a Rockefeller Report of 1970.

> "It has been calculated that a fall of one cent per pound in the coffee price, signifies a loss of US $55 million in foreign exchange for the 14 countries that produce coffee in the Western Hemisphere".

Symptoms

The most obvious symptoms of H. vastatrix are the spots with a powdery appearance and a yellow to orange color found on the underside of the leaves. Lesions may first appear as small yellow-colored spots which change color to bright orange. Rust sori are characterized as "oil spots" on the upper leaf surface. A yellow-green diffuse halo may surround the lesions. Eventually the lesions may become necrotic, turning grey or brown. Extensive defoliation can

Fig. 40. Coffee rust lesions (Hemileia vastatrix).

weaken and eventually kill coffee plants. In contrast, H. coffeicola does not produce distinct spots, but rather the spores completely cover the lower surface of the leaf.

Host Plants

There are many Coffea species and almost all are susceptible, but differences in degree of susceptibility are found. Gardenia spp. are also susceptible. No alternate host for an aecial stage has been found for H. vastatrix.

Pathogen Cycle

Since the alternate host (if one exists) of H. vastatrix has not been found, and since the disease exists in the tropics where coffee has green foliage all year round, the fungus has only one continuing cycle carried on by the urediospore stage. Teliospores and sporidia are produced, but apparently play no role in the life cycle of the fungus.

The urediospores produced on coffee leaves constitute the inoculum which can be dispersed by wind, splashing water, and insects. Much of the early work done on the biology of H. vastatrix was done in Kenya. Some workers (Nutman 1959, Nutman et al. 1960, 1970, Bock 1962) suggested that rain splash was most important in spore dispersal and that wind played little role in dispersal. Others (Bowden et al. 1971, Rayner 1960a, 1961b, and Becker et al. 1975) believe wind is most important in dispersal and spread. Some believe the fungus could have been blown long distances, as from Africa to Brazil (Bowden et al. 1971). Waller (1982) suggests that it may have been introduced into Brazil on diseased or contaminated material. Martinez et al. (1975) found urediospores in the air up to 1,000 m above coffee plantations in Brazil. The rapid spread of H. vastatrix after its discovery in Brazil strongly suggests that wind dissemination of urediospores is important. Spore numbers and infection are related to rainfall and increase greatly after heavy rainfalls. Becker et al. (1975) described a diurnal cycle of spore liberation. According to Kranz (1977) young leaves are more susceptible than older ones.

Several studies have been made (Nutman and Roberts 1970, Rayner 1961a, Harr and Guggenheim 1978) on germination of urediospores. They require free water for germination and have a bimodal temperature requirement for germination with one peak at 21°C and another about 25°C. Spores germinate best in the dark or at low light intensities. Appressoria are produced and penetration is through stomata found on lower leaf surfaces. After leaf penetration, the length of the incubation period depends on environmental conditions. An incubation period of about five weeks was reported for Kenya, but sporulation two

weeks after penetration has been reported (Rayner 1960a). After infection the optimal temperature for development of H. vastatrix is between 21-25°C. Little development takes place below 15°C or above 30°C. Waller (1982) recently reviewed the epidemiology of coffee rust.

Control

The fungus has been successfully eradicated from Puerto Rico and more recently Papua, New Guinea. Wellman (1961) states that a horticulturist, O. W. Barrett, destroyed all diseased material when the fungus was introduced into Puerto Rico in 1903. H. vastatrix was also eradicated from Papua, New Guinea, in 1965 (Holliday 1980). A serious attempt was made to eradicate H. vastatrix from Nicaragua after its introduction there in 1976, but the attempt failed as increasing political instability and violence in Nicaragua disrupted the eradication efforts.

In 1963, I accompanied Dr. A. J. Riker of the University of Wisconsin, who was visiting various countries in South America, on visits to various national and international agencies in Colombia. He was seeking support for a project on methods of eradicating coffee rust. The project, a modest $190,000, was not funded. Had it been funded, it might have prevented the introduction of H. vastatrix into Brazil and accomplished what Asian governments are now doing relative to preventing the introduction of South American leaf blight of rubber into Asia. Today, many millions of dollars are being spent on coffee rust research and control by South American countries. This example illustrates the need for sound information which will convince administrators and governments of the need to invest in research and education on threatening plant diseases before such diseases are introduced.

In countries where H. vastatrix is established, one of the major control methods is the use of fungicides. Wellman (1955) cited yield increases of 50 to 100% through the use of chemical control for coffee rust. Marshall Ward worked on chemical control of coffee rust and was the first to point out the necessity of protecting the foliage before the fungus penetrated. There is a voluminous literature on chemical control of coffee rust (Wellman 1955, Rayner 1960c, Mayne 1971, Chaves 1972, Wybou and Stripecke 1980, Waller 1982). Bordeaux mixture and later fixed coppers were used for many years in Asia and Africa for coffee rust control. The main fungicides used in Brazil are copper fungicides (Wybou and Stripecke 1980), with fixed coppers, primarily copper oxychloride, being preferred. Many organic fungicides have been tested, but in general their performance has been inferior to that of the copper fungicides. Some of the systemic fungicides appear highly promising for control (Wybou and Stripecke 1980). In 1982 recommended fungicides for control of H. vastatrix in Kenya were 50% copper fungicides, fentin hydroxide, dithianon, pyracarbolid and triadimefon (Kenya Coffee 1982).

A tragic aspect of the appearance of H. vastatrix in the Americas has been its effect on small growers. Large, modern coffee producers can control the disease, but small growers often simply cannot afford the costs of spraying. Small farmers in Latin America have often gone out of production after coffee rust has been introduced (Horsley 1977). Wellman and Echandi (1981) estimate that the cost of spraying adds 10-20% to total production costs.

Arabica coffee is the most widely cultivated coffee species, (65% of total world area), but according to Rodrigues (1975) no selection of C. arabica is completely resistant (vertical resistance) to the known races of H. vastatrix. Work in Portugal with a large collection of coffee germ plasm has shown at least 30 physiologic races of H. vastatrix (Rodrigues et al. 1975, Eskes

1983). Considerable resistance is found in the natural hybrid between C. arabica and C. canephora "Hibrido de Timor" and in species with inferior quality such as C. canephora and C. liberica. The cultivar Catimor, resulting from a cross by D'Oliveira between Hibrido de Timor and Caturra, is well adapted to Latin American coffee areas and is highly resistant to H. vastatrix. There is considerable interest in horizontal resistance to H. vastatrix, and tests with collections made in Ethiopia indicate the presence of horizontal resistance (Rodriques et al. 1975). Horizontal resistance is also being studied in Brazil (Ribeiro et al. 1981, Eskes and Toma-Braghini 1981, Eskes 1982). Breeding and selection for resistance to H. vastatrix has been done in India, Portugal, Brazil, and Kenya (Firman and Hanger 1963, Narasimhaswamy 1961, Rayner 1960b, D'Olivera 1965). There appear to be 5 simple and dominant genes controlling resistance to rust in C. arabica (Rodrigues et al. 1975). The extensive literature on breeding for resistance to rust has been reviewed by Rodrigues et al. (1975).

An integrated pest management program for coffee rust should use all possible means of control. In addition to chemical control and the use of resistant varieties, growers can have an appreciable effect on rust severity by various cultural practices such as proper spacing or removal of plants to facilitate air movement, pruning and thinning to permit better spray coverage and to allow plants to dry quickly after dew or rain, and adequate fertilization to promote vigorous plants and leaf replacement.

Literature Cited

Becker, S., S. K. Mulinge, and J. Kranz. 1975. Evidence that uredospores of Hemileia vastatrix Berk. and Br. are wind-borne. Phytopathol. Z. 82:359-360.

Bock, K. R. 1962. Dispersal of uredospores of Hemileia vastatrix under field conditions. Trans. Brit. Mycol. Soc. 45:63-74.

Bowden, J., P. H. Gregory, and C. G. Johnson. 1971. Possible wind transport of coffee leaf rust across the Atlantic Ocean. Nature 229:500-501.

Carefoot, G. L., and E. R. Sprott. 1967. Famine on the Wind. Rand McNally, Chicago. 229 pp.

Chaves, G. M. 1972. Control quimico de la roya del cafe en Brazil. pp. 39-51, In Simposio sobre la roya del cafe (Hemileia vastatrix Berk. & Br.). Inst. Interamericano Ciencias Agrarias. Publ. Misc. No. 126. IICA Zona Norte, Mexico. 64 pp.

Coffee Rust Research Center. 1971. Report for 1971. Oeiras, Portugal.

Colombia Today. 1981. The Colombian economy 1981-1982. 16(11):1-6.

Cramer, P. J. S. 1957. A review of literature on coffee research in Indonesia. Misc. Publ. No. 15. Inter-American Inst. Agric. Sci. Turrialba, Costa Rica. 262 pp.

D'Olivera, B. 1965. Progress report 1960-1965. Coffee Rust Res. Center, Oeiras, Portugal. 144 pp.

Eskes, A. B., and M. Toma-Braghini. 1981. Assessment methods for resistance to coffee leaf rust (Hemileia vastatrix Berk. & Br.). Food Agric. Organ. Plant Prot. Bull. 29:56-66.

Eskes, A. B. 1982. The use of leaf disk inoculations in assessing resistance to coffee leaf rust (Hemileia vastatrix). Neth. J. Plant Pathol. 88:121-141.

Eskes, A. B. 1983. Qualitative and quantitative variation in pathogenicity of races of coffee leaf rust (Hemileia vastatrix) detected in the state of Sao Paulo, Brazil. Neth. J. Plant Pathol. 89:31-46.

Firman, I. D., and B. F. Hanger. 1963. Resistance to coffee leaf rust in Kenya. Kenya Coffee 5:49-54.

Harr, J., and R. Guggenheim. 1978. Contributions to the biology of Hemileia vastatrix I. SEM-investigations on germination and infection of Hemileia vastatrix on leaf surfaces of Coffea arabica. Phytopathol. Z. 92:70-75.

Holliday, P. 1980. Fungus Diseases of Tropical Crops. Cambridge Univ. Press, Cambridge. 607 pp.

Horsfall, J. G. 1945. Fungicides and Their Action. Chronica Botanica, Waltham, MA. 239 pp.

Horsley, B. 1977. Central America gears to fight coffee rust. Foreign Agric. June 27, 1977-:11-14.

Kenya Coffee. 1982. Control of coffee berry disease and leaf rust in 1982. 47:9-14.

Kranz, J. 1977. Hemileia vastatrix Bark. and Br. pp. 158-159. In Diseases, Pests and Weeds in Tropical Crops. Kranz, J., H. Schmutterer and W. Koch. eds. Verlag Paul Parey, Berlin. 666 pp.

Large, E. C. 1940. The Advance of the Fungi. Dover, New York. 488 pp.

Laundon, G. F., and J. M. Waterson. 1964. Hemileia vastatrix. Commonwealth Mycological Institute Descriptions of Pathogen Fungi and Bacteria No. 1. Commonw. Mycol. Inst., Kew, Surrey.

Martinez, J. A., D. A. Palazzo, M. Karazawa, M. V. M. Monteiro and N. R. N. Reu. 1975. Presenca de esporos de Hemileia vastatrix Berk. and Br., agente causal de ferrugem do cafeiro, em diferentes altitudes nas principais areas cafeeiras dos estados de Sao Paulo e Parana (Brasil). Biologico 41:77-88.

Mayne, W. W. 1971. Control of coffee leaf disease in southern India. World Crops 23:206-207.

Monaco, L. C. 1977. Consequences of the introduction of coffee leaf rust into Brazil. Ann. N.Y. Acad. Sci. 287:57-71.

Narasimhaswamy, R. L. 1961. Coffee leaf disease (Hemileia) in India. Coffee 3:33-39.

Nutman, F. J. 1959. Evidence that the spores of coffee leaf rust are not dispersed by wind. Kenya Coffee 24:451-453.

Nutman, F. J., F. M. Roberts, and K. R. Bock. 1960. Methods of uredospore dispersal of the coffee leaf rust fungus, Hemileia vastatrix. Trans. Brit. Mycol. Soc. 43:509-515.

Nutman, F. J., and F. M. Roberts. 1970. Coffee leaf rust. Pest Articles News Sum. 16:606-624.

Rayner, R. W. 1960a. Rust disease of coffee 1-Nature of the disease. World Crops 12:187-190.

Rayner, R. W. 1960b. Rust disease of coffee 3-Resistance. World Crops 12:261-264.

Rayner, R. W. 1960c. Rust diseases of coffee 4-Control by fungicides. World Crops 12:309-312.

Rayner, R. W. 1961a. Germination and penetration studies on coffee rust (Hemileia vastatrix B. et Br.) Ann. Appl. Biol. 49:497-505.

Rayner, R. W. 1961b. Spore liberation and dispersal of coffee rust Hemileia vastatrix B. & Br. Nature 191:725.

Ribeiro, I. J. A., A. B. Filho, P. C. T. Carvalho. 1981. Avaliacao da resistencia horizontal a Hemileia vastatrix Berk. & Br. em cultivares de Coffea arabia em condicoes naturais de epidemia. Summa Phytopathol. 7:80-95.

Rodrigues Jr., C. J. 1975. Resistance del cafe a las royas. pp. 52-64. In Simposio sobre la roya del cafe (Hemileia vastatrix Berk. & Br.). Inst. Interamericano Ciencias Agrarias. Publ. Misc. No. 126. IICA, Zona Norte, Mexico. 64 pp.

Rodrigues, Jr., C. J. A., A. J. Bettencourt, and L. Rijo. 1975. Races of the pathogen and resistance to coffee rust. Annu. Rev. Phytopathol. 13:49-70.

Scheiber, E. 1972. Economic impact of coffee rust in Latin America. Annu. Rev. Phytopathol. 10:491-510.

Schieber, E. 1975. Present status of coffee rust in South America. Annu. Rev. Phytopathol. 13:375-382.

Waller, J. M. 1972. Coffee rust in Latin America. Pest Articles News Summaries 18:402-408.

Waller, J. M. 1982. Coffee rust - epidemiology and control. Plant Prot. 1:385-404.

Ward, H. M. 1882. Researches on the life history of Hemileia vastatrix. J. Linn. Soc. 19:299-335.

Wellman, F. L. 1955. Am. Chem. Soc. Advances in Chemistry Studies. No. 13, Coffee diseases, insects, and weeds controlled by chemicals. pp. 43-63. In Pesticides in Tropical Agriculture. Am. Chem. Soc., Washington, DC.

Wellman, F. L. 1961. Coffee: Botany, Cultivation, and Utilization. Interscience, New York. 488 p.

Wellman, F. L., and E. Echandi. 1981. The coffee rust situation in Latin America in 1980. Phytopathology 71:968-971.

Wybou, A., and W. Stripecke. 1980. Control of coffee rust (Hemileia vastatrix Berk. et Br.) with Bayleton in Brazil. Bayer Pflanzenschutz - Nachrichten 33:123-151.

CACAO

Cacao (*Theobroma cacao*) is a small tree still found in the forests of the head waters of the Amazon, basin. Europeans' first encounter with cacao, or chocolate obtained from cacao, occurred when Hernando Cortez, the Spanish conqueror of Mexico, was served the drink "chocolatl" in golden goblets in the court of the Emperor Montezuma. With its introduction to Europe it was found that by adding sugar and condiments chocolate made a delicious drink. The discovery of a method of extracting the fat from chocolate and the invention of milk chocolate greatly increased the use and demand for cacao. Today chocolate is popular as a beverage, as candy, and in many desserts. The largest consumers of chocolate are developed countries, but the major producers are tropical countries in Africa and the Americas. World cacao production was 1.7 million tons on 4.7 million hectares in 1981 (FAO 1981).

Three main types of *T. cacao* are found: criollo, forastero, and trinitario. Over 90 percent of the world's cacao production is of forastero origin. Most cacao is grown in the tropics in countries between 15°N and S latitude with abundant rainfall. Plants from seed are usually used to establish plantings, but cuttings can also be used although their use is more expensive. Almost all cacao is grown under shade. Plantings are usually established at densities ranging from 500-2000 trees per hectare.

The flowers and fruits (pods) of cacao develop directly from the branches and trunks of the tree. Normally 35-40 seeds (called "beans") are produced in a pod. When beans are removed from the ripe pods a mucilagenous material remains attached to them. This is removed by fermenting the beans in large wooden boxes for several days. During fermentation, when temperatures often rise to over 45°C, the embryo is killed and the chocolate flavor begins to develop in the cotyledons. Dried beans are roasted and then cracked to remove the shell and germ. The remaining "nibs" are processed to produce cocoa powder and cocoa butter.

Diseases are serious limiting factors in cacao production. The single

most serious and widely distributed disease of cacao worldwide is probably black pod rot caused by Phytophthora spp. It is found in almost all cacao producing countries, and worldwide losses have been estimated at 10% (Padwick 1956). Individual growers may occasionally lose their entire crop. P. palmivora was thought to be the only causal agent until recently. Presently, several Phytophthora species (P. palmivora, P. capsici, P. megakarya, and P. citrophthora), are considered to cause black pod rot. P. palmivora has a wide host range in the tropics. Chee (1969) lists 138 hosts of P. palmivora including many crops of economic importance in the tropics. All plant parts may be infected including the roots, stems, flowers, pods, and leaves. Extensive epidemiological studies of P. palmivora have been made (Gregory 1974, Gregory and Maddison 1981, Thorold 1975). Splashing rains appears to be the most important means of fungal dissemination. Cultural and sanitation practices similar to those for control of Monilia roreri are also useful against Phytophthora spp. Removal of weeds, good drainage, proper pruning, wide spacing of plants, and frequent harvest of pods (to assure removal of those infected) are recommended. Fungicides, especially coppers, have been widely used for control. Cultivars with different levels of resistance to Phytophthora spp. are known and should be used in disease control programs, but the complex of Phytophthora species causing black pod rot may reduce the long-term value of resistance and breeding efforts.

Witches' broom disease of cacao is caused by the basidiomycete Crinipellis perniciosa. It is considered to be one of the most destructive diseases of cacao, often causing losses of 50-80% of the potential harvest. The disease caused the temporary abandonment of cacao cultivation in Surinam and Guyana in the 1920's (Baker and Holiday 1957) and threatened the industry in Ecuador. Witches' broom disease was first reported in Surinam in 1895, but is now found in several countries of northern South America, and Panama, Trinidad, and Grenada. It is widely distributed in the Amazon jungle on wild Theobroma species. The fungus infects meristematic tissue resulting in the production of characteristic fan-shaped hypertrophied vegetative shoots. Buds, fruit, and flower pads can be infected. Infected brooms produce basidiocarps (mushrooms) which produce basidiospores at night that are wind dispersed. Races of the pathogen may exist, as highly resistant material developed in Trinidad was less resistant in Ecuador (Holliday 1980). Destroying infected brooms several times per year provides some control, but in countries where aggressive races are not found resistant cultivars give effective control. Comprehensive information on the disease is found in Baker and Holliday (1957), Holliday (1980), Coto Royo (1983), and Thorold (1975). The major cacao growing areas of the world are not yet infected, and every effort should be made to restrict C. perniciosa to its present distribution.

Monilia pod rot (Monilia roreri), another serious disease, is found in South and Central America. "Mal de Machete" or wilt is caused by Ceratocystis fimbriata. It is similar in many respects to other tree diseases caused by Ceratocystis species and can be transmitted by insects, but mechanical transmission is more important.

Cacao swollen shoot disease is the most destructive of the several viruses attacking cacao (Brunt and Kenten 1971). The disease is caused by a complex of virus strains grouped under the name cacao swollen shoot virus (CSSV). The disease was first found in Ghana in 1936, and is now found in four other West African countries. Devastating losses with widespread economic and political effects occurred in Ghana. Over 140 million trees have been removed in eradication programs for CSSV in Ghana (Brunt and Kenten 1971). CSSV is not found

in the Americas where cacao originated. It undoubtedly originated in trees occurring in native West African forests, and these now serve as reservoirs of infection. Several species of mealybugs transmit CSSV. Biological and chemical control of mealybugs for CSSV control has not been ecouraging. Eradication of diseased trees has been the major control method; however, since removal of diseased trees is extremely expensive and not entirely successful, perhaps due to reservoirs of infection in forest trees, disease resistance appears to be the most promising long-term control. All cacao lines tested have been infected, but lines with high levels of tolerance to CSSV have been found. These are being utilized in breeding, and eventually tolerant lines may replace the susceptible one now grown (Kenten and Legg 1971). CSSV is a serious threat to cacao production in the Americas and Africa, and every effort should be made to prevent its introduction.

Selected References on Cacao Diseases

Brunt, A. A., and R. H. Kenten. 1971. Viruses infecting cacao. Rev. Plant Pathol. 50:591-602.

Desrosiers, R. 1977. Cacao Handbook. Am. Cocoa Res. Inst. McLean, VA. 22 pp.

Gregory, P. H. ed. 1974. Phytophthora Disease of Cacao. Longman, London. 348 pp.

Imle, E. P. 1966. Plant material distribution and quarantine measures for cocoa. Food Agric. Organ. Plant Protection Bull. 14:134-140.

Thorold, C. A. 1975. Diseases of Cocoa. Clarendon Press, Oxford. 423 pp.

Thresh, J. M., and T. W. Tinsley. 1959. The viruses of cacao. W. Afr. Cocoa Res. Inst. Tech. Bull. 7. 32 pp.

Urquhart, D. H. 1962. Cocoa. Longman, London. 293 pp.

Wood, G. A. R. 1975. Cocoa. Longman, New York. 292 pp.

Literature Cited

Baker, R. E. D., and Holliday, P. 1957. Witches' Broom Disease of Cacao (Marasmius perniciosus Stahel). Phytopathol. Paper No. 2. Commonw. Mycol. Inst., Kew, Surrey. 42 pp.

Chee, K. H. 1969. Hosts of Phytophthora palmivora. Rev. Appl. Mycol. 48:337-344.

Coto Royo, L., and C. Villegas. eds. 1983. Escoba de bruja del cacao (Marasmius perniciosus): bibliographia parcialmente anotada. Centro Interam. de Doc. e Inform. Agric., Turrialba, Costa Rica. 136 pp.

Food Agriculture Organization. 1981. FAO Production Yearbook. Vol. 35. FAO, Rome. 306 pp.

Gregory, P. H., and A. C. Maddison. eds. 1981. Epidemiology of Phytophthora on cocoa in Nigeria. Phytopathol. Paper No. 25., Commonw. Mycol. Inst., Kew Surrey. 188 pp.

Holliday, P. 1980. Fungus Diseases of Tropical Crops. Cambridge Univ. Press, Cambridge. 607 pp.

Kenten, R. H., and J. T. Legg. 1971. Varietal resistance of cacoa to swollen shoot disease in West Africa. Food Agric. Organ. Plant Protection Bull. 19:1-12.

Padwick, G. W. 1956. Losses Caused by Plant Diseases in the Colonies. Phytopathol. Paper No. 1., Commonw. Mycol. Inst., Kew, Surrey. 60 pp.

MONILIA POD ROT OF CACAO

Disease Name: The disease is also called Moniliasis or watery pod rot and recently the name frosty pod rot was proposed by Evans et al. (1978).

Causal Organism: Monilia roreri Cif. and Par.; (Moniliaceae)
Synonym: Moniliophthora roreri Evans, Staplers, Sampson, and Benny.

Monilia roreri is an extremely destructive pathogen which has caused serious losses in South America and has a demonstrated capacity to spread to other cacao-growing regions. In Colombia the disease has been called in Spanish "moniliasis", "ceniza", "polvillo", pringue", "mal paludico" and "pudricion acuosa". In Ecuador it has been called "pudricion acuosa", "enfermedad de Quevedo", "helada", and "la mancha". The disease was first found in 1914 near the city of Quevedo, Ecuador, and was once known as the "Quevedo disease". From Ecuador M. roreri has spread to neighboring Colombia, Brazil, and Peru, subsequently to Venezuela, Panama, and most recently to Costa Rica (Enriquez and Suarez 1978). It constitutes a considerable threat to the cacao industry in Central America and other regions of tropical America.

Rorer in 1918 was the first to describe the fungus (Ampuero 1967). R. E. Smith identified it as a species of the genus Monilia (Barros 1982). Later Cifferi and Parodi (1933) named the fungus Monilia roreri. Recently Evans et al. (1978) redescribed the fungus. They found dolipore septa in the mycelium which indicates that it is a basidiomycete and therefore proposed a new genus - Moniliophthora - for the fungus. This work needs confirmation by scientists in Latin America before it will be generally accepted.

Conidia are hyaline, formed in basipetally maturing chains, globose to subglobose (6.5-) 8-15 (-25) µm diameter or ellipsoid (8-20 x 5-14 µm) Evans et al. 1978). Detailed descriptions of the fungus are given by Holliday (1970, 1980), Thorold (1975) and Evans et al. (1978). The perfect state is unknown.

Importance

Monilia pod rot has caused serious losses in Ecuador and Colombia. In Colombia the disease caused yield losses of 30-40% (Barros 1966), and losses reached 90% in some areas (Orellana 1954). Barros (1982) estimated an average annual loss of 30% equivalent to 20 million dollars (USA) in Colombia due to M. roreri. Ampuero (1967) estimated that 15-80% of the pods in Ecuador and Colombia are lost due to the fungus. Many cacao plantations have been abandoned because of M. roreri in Colombia and Ecuador. Sanchez (1982) estimated that 60% of the harvest was lost in Costa Rica in 1980 due to M. roreri.

Symptoms

The only part of the plant that M. roreri is reported to attack is the pods, although Evans et al. (1977) reported circumstantial evidence that pod cushions or the stem may be infected. Young pods are most susceptible. The first symptoms are small, discolored spots on pods which later turn brown, often with a yellow halo. An internal discoloration is evident when these young pods are cut. The brown spots enlarge and may coalesce to cover all or part of the pod. Later, lesions become covered with a cottony layer of mycelium on which profuse sporulation consisting of cream, [illegible], tan, or brown colored conidia occurs. Swellings are often found on [illegible] pods. The interior

of the pod is usually watery, and its contents are destroyed. Since the symptoms are similar to those produced by Phytophthora spp. (black pod rot) and Crinipellis perniciosa (witches broom), microscopic examination of the spores is best for a positive identification.

Host Plants: M. roreri attacks species of the genera Theobroma and Herrania (Evans et al. 1978).

Pathogen Cycle

The only inocula known to initiate infection are the conidia. Conidia are produced on diseased pods remaining on trees, or may come from harvested or discarded pods which are often left in the cacao plantings. The fungus survives as conidia on pods between wet and dry seasons. Moist weather increases conidial production.

Conidia are disseminated by wind, water, and insects. Desrosiers et al. (1955) and Villacreses et al. (1963) reported flower infection. They also reported that young pods are most susceptible. Evans et al. (1978) and Desrosiers and Suarez (1974) state that pods can be penetrated at any stage of development. Franco (1958) found that infection was increased when "stink bugs" (Mecistorhinus tripterus) were placed on the infection court with spores of M. roreri. The increase in infection was attributed to puncturing of the young pods. Sepulveda (1955) earlier had similar results in his experiments. However, the pathogen can penetrate the cuticle of pods directly and cause infection without prior injury (Desrosiers and Suarez 1974, Ampuero 1977), although Naundorf (1954) found that injury facilitated infection.

Fig. 41. Healthy cacao pod (left) and one infected (right) with Monilia roreri (Monilia pod rot).

Desrosiers (1955) showed a close correlation between high rainfall and a high incidence of frosty pod rot in studies made during a four year period in Ecuador. There is little information on the conditions necessary for conidial germination (Naundorf 1954), but Monilia species usually need free water for spore germination. Lopez (1954) found that spores germinated better at 22°C than at 35°C at 80% RH.

Evans et al. (1978) found that, after penetration by conidia, the mycelium grows intercellularly between the cortical parenchyma cells. They described the hyphae as swollen, convoluted, and lumpy. Obvious symptoms appear 45-90 days after penetration as dark-brown coalescing lesions; however, this may be preceded by signs of premature ripening. Little information is available on the effect of temperature and moisture on infection. In general, most disease development occurs in warm, moist areas, but since these conditions are also optimum for cacao culture, little can be done by growing cacao in drier or cooler areas. Infected pods are found at all times of the year; thus infection

occurs all year long in a tropical climate if moisture is available.

Control

Since M. roreri is restricted to a few tropical countries, every effort should be made to exclude it from countries where it does not now exist. Plant quarantines can be very important in excluding the disease. Imle (1966) discusses the danger of introducing M. roreri into other countries and discusses precautions which should be observed to prevent its spread. He suggests that seed or budwood are probably the safest material for distribution. Conceivably (no evidence is available) mycelia of M. roreri might be present in seed or budwood so great care should be taken, and a reliable source of material investigated, before importing material from any country where M. roreri exists. Conidia can probably be easily eliminated from seed by surface sterilization. Entire pods should never be distributed.

Partial control of M. roreri can be achieved by certain cultural or sanitation methods. Hardy (1961) recommended harvesting pods at frequent intervals, destroying infected pods that are found during harvest, processing the pods at a distance from the plantations, and not leaving mounds of processed pods within the plantation. Barros (1966) studied the value of various cultural methods in a cacao planting which (at the initiation of the experiment) had 30% of the pods infected by M. roreri. He divided the planting into two parts. In one the farmer's normal practices were continued (monthly harvests and three weedings a year), and in the other the practices were as follows:

1. Elimination of suckers
2. Two prunings a year
3. Wound treatment with chemicals
4. Monthly harvests
5. Removal of diseased pods at each harvest
6. Improved drainage
7. Three weedings per year

By the above treatments better water drainage and air circulation was achieved, and environmental conditions conducive to disease development were reduced. Sources of inoculum were also reduced. Pods discarded after harvest were removed from the plantation. The amount of M. roreri was reduced from 30% infection initially to 17% infection the first year to 9% in the second year of the experiment. A yield increase of 462 kilograms of dry cacao per hectare was achieved (equivalent to a 44% increase in yield) using the above methods.

Numerous studies on chemical control of M. roreri with many different fungicides have been made (Delgado 1963, Ampuero 1967, Jorgensen, 1970). Most of the results have been economically disappointing, and to date it is not considered economic to spray most cacao plantings. Ampuero (1977) states that zineb, maneb, and copper compounds are effective if applied every 2-4 weeks. The difficulties of effectively spraying trees with rapidly developing pods in areas with an annual rainfall of 2000 mm are considerable.

Some information is available on resistance of M. roreri. Imle (1966) stated that workers in Ecuador reported yield losses among 24 cacao clones of 1.7 to 47 percent. Ampuero (1967) reports several clones showed low incidence of the disease under field conditions and in the field when artificially inoculated. At Turrialba, Costa Rica, where a large cacao collection has been maintained for several decades, clones CC-210, CC-266, EET-48, and EET-59 showed tolerance or resistance to M. roreri. In Pichilingue, Ecuador clone

EET-233 had a low incidence of infection (G. A. Enriquez - personal communication). Much more work needs to be done on finding resistance to M. roreri.

Other cacao-producing countries in the Americas and other continents should take every precaution to prevent the spread or introduction of this highly destructive pathogen into their areas.

Literature Cited

Ampuero, C. E. 1967. Monilia pod rot of cocoa. Cocoa Grower's Bull. 9:15-18.

Ampuero, C. E. 1977. Monilia roreri. pp. 219-220, In J. Kranz, H. Schmutterer, W. Koch. eds. Diseases, Pests and Weeds in Tropical Crops. Verlag Paul Parey, Berlin. 666 pp.

Barros, N. O. 1966. Valor de las practicas culturales como metodo para reducir la incidencia de Monilia en plantaciones de cacao. Agric. Trop. (Colombia) 22:605-612.

Barros, O. 1982. Historia de la moniliasis y sus repercusiones en los paises productores de cacao en sur America. In Enriquez, G. A. ed. La Moniliasis de Cacao. Centro Agron. Trop. Investigacion y Insenanza, Turrialba, Costa Rica. 93 pp.

Ciferri, R., and E. Parodi. 1933. Descrizione del fungo che cause la "Moniliasis" del Cacao. Phytopathol. Z. 6:539-542.

Delgado, J. C. 1963. Efecto de diversas dosis de oxido cuproso y zineb aplicados a bajo volumen en el control de la Monilia en el cacao. Turrialba 13:129-131.

Desrosiers, R., and C. Suarez. 1974. Monilia pod rot of cacao. pp. 273-282. In Gregory, P. H. ed. Phytophthora Disease of Cocoa. Longmans, London. 384 pp.

Desrosiers, R., A. von Buchwald, and C. Bolanos. 1955. Effect of rainfall on the incidence of Monilia pod rot of cacao in Ecuador. Food Agric. Organ. Plant Prot. Bull. 3:161-164.

Enriquez, G., and C. Suarez C. 1978. Monilia disease of cocoa in Costa Rica. Turrialba 28:339-340.

Evans, H. C., D. F. Edwards, and M. Rodriguez. 1977. Research on cocoa diseases in Ecuador: past and present. Pest Articles New Sum. 23:68-80.

Evans, H. C., R. A. Stalpers, R. A. Samson, and G. L. Benny. 1978. On the taxonomy of Monilia roreri, an important pathogen of Theobroma cacao in South America. Can. J. Bot. 56:2528-2532.

Franco, T. 1958. Transmission de la Moniliasis del cacao por el Mecistorhinus tripterus F. In Septima Conf. Interamericana de Cacao, Palmira, Colombia. 606 pp.

Hardy, F. 1961. Manual de Cacao. Inst. Interamericano de Ciencias Agricolas, Turrialba, Costa Rica. 439 pp.

Holliday, P. 1970. Monilia roreri. C.M.I. Descriptions of Pathogenic Fungi and Bacteria. No. 226. Commonw. Mycol. Inst., Kew, Surrey.

Holliday, P. 1980. Fungus Diseases of Tropical Crops. Cambridge Univ. Press, Cambridge. 607 pp.

Imle, E. P. 1966. Plant material distribution and quarantine measures for cocoa. Food Agric. Organ. Plant Prot. Bull. 14:134-140.

Jorgensen, H. 1970. Monilia pod rot in Ecuador. Cacao 15(4):4-13. Inst. Interamericano Ciencias Agricolas, Turrialba, Costa Rica.

Lopez, F. R. 1954. Fisiologia de la germinacion de esporas de Monilia sp. Cacao en Colombia 3:183-207.

Naundorf, G. 1954. Contribuciones al problema de la moniliasis en cacao. Cacao en Colombia 3:35-61.

Orellana, R. G. 1954. Cacao diseases in Venezuela, Colombia, Ecuador and Trinidad. Food Agric. Organ. Plant Prot. Bull. 2:49-52.

Sanchez L., J. A. 1982. Reaccion de cultivares de cacao a la inoculacion artificial con Monilia roreri. M.S. Thesis. Univ. Costa Rica, Ciudad Universitaria. 55 pp.

Sepulveda, L. R. 1955. Biologia del Mecistorhinus tripterus F. (Hem. Pentatomidae) y su posible influencia en la transmission de la Moniliasis del cacao. Cacao en Colombia 4:15-42.

Thorold, C. A. 1975. Diseases of Cocoa. Clarendon Press, Oxford. 423 pp.

Villacreses, G. B., J. C. Delgado, K. D. Doak. 1963. Methods of inoculation and factors favorable for the infection of cacao by Monilia roreri. Phytopathology 53:26.

TEA

The most widely used beverage in the world is tea (Camellia sinensis). The continent of Asia is the largest producer followed by Africa and South America. Unlike coffee, much of the Asian production is consumed locally, although tea is a major export of countries such as India and Sri Lanka who are the world's largest producers. World production in 1981 was 1.9 million tons (FAO 1981). Tea originated in Southeast Asia, probably about 2700 B.C., and has been grown commercially in China since 800 A.D. (Personal communication - C. S. Venkata Ram).

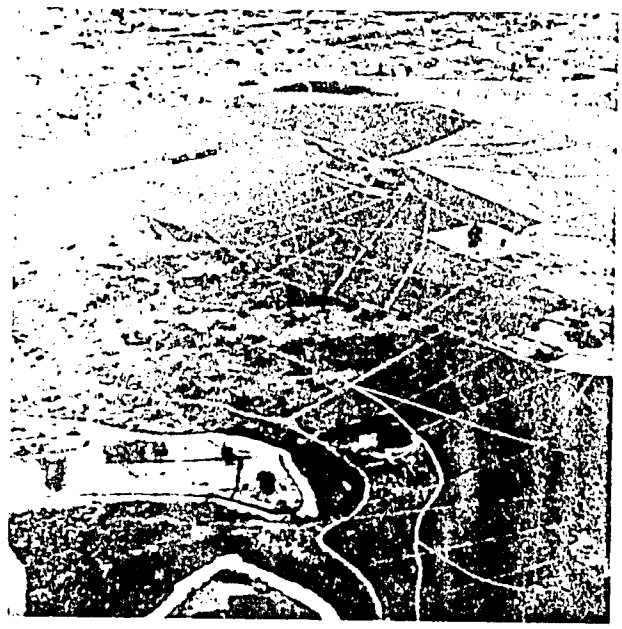


Fig. 42. Tea plantings near Lake Victoria in Kenya.

There are countless varieties of tea which are classified into two main groups (Purseglove 1968): China teas (C. sinensis var. sinensis) and Assam teas (C. sinensis var. assamica). The young leaves of tea are still almost all picked by hand, usually every 7-10 days. After harvest, leaves for black tea are withered, fermented, dried, and graded for sale. Green tea is processed similarly, but the leaves are steamed soon after harvest to inactivate enzymes and prevent fermentation.

The tea plant is a small evergreen shrub grown primarily in the tropics or subtropics on acidic soils. It grows at elevations ranging from 100 to 2500 m, in regions with over 125 cm of rainfall that are not affected by severe frost. Numerous systems of pruning are used, generally to produce a low, spreading bush for ease of harvest. Plants are usually grown from seed in nurseries and transplanted to the field, but cuttings are now planted on a large scale. Shade was once used extensively, but tea in full sunlight is now more common as yields are higher and blister blight (Exobasidium vexans) is less serious. Tea is grown as a monoculture in plantations which may remain in production for more than 50 years.

Tea diseases can cause tremendous losses. The most serious disease of tea is blister blight, caused by the fungus Exobasidium vexans. The disease has been known in Northeast India since 1855, but it was not until 1946 that it spread to Southern India. Blister blight is now endemic in all the major tea-producing areas of Asia, but is not found in Africa or the Americas. A loss of 36 million pounds of tea per year over a five-year period was estimated for Southern India until control by copper sprays was developed. The disease is most serious at high elevations under cool, moist conditions. Infection by the fungus produces characteristic blisters which become white with basidiospores. E. vexans attacks the foliage and, since young foliage is the economic product harvested, the disease is especially destructive. Both protectant and systemic fungicides have been used for control. Resistant varieties have not

been of value. Considerable information has been accumulated on the epidemiology of E. vexans. Using spore counts and sunshine records, disease incidence can be predicted three weeks in advance with considerable accuracy, and thus considerable savings in fungicide applications can be made. A simple, wheel-like device which enables growers to determine disease incidence has been devised (Kerr and Rodrigo 1967).

Tea is subject to attack by a number of root-rotting organisms: Phellinus noxius, Rosellinea arcuata, Poria hypobrunnea, Armillariella mellea, and others. Red rust is caused by an alga (Cephaleuros parasiticus) which attacks both the stems and leaves. Pink disease (Corticum salmonicolor) may be serious under moist conditions, and wood rot (Hypoxylon serpens) is another important stem disease. Grey blight (Pestalotiopsis theae), brown blight (Colletotrichum camelliae), and thread blight or black rot (Corticium invisium, C. theae, and Thanatephorus cucumeris) are other leaf diseases of tea. Chen and Chen (1982) recently reviewed the diseases of tea and their control in the People's Republic of China.

Selected References on Tea Diseases

Chen, T. M., and S. F. Chen. 1982. Diseases of tea and their control in the People's Republic of China. Plant Dis. 66:961-965.

Eden, T. 1976. Tea. Longman, London. 236 pp.

Hainsworth, E. 1952. Tea Pests and Diseases and Their Control. Heffner, Cambridge. 130 pp.

Kerr, A., and W. R. F. Rodrigo. 1967. Epidemiology of tea blister blight (Exobasidium vexans). IV. Disease forecasting. Trans. Brit. Mycol. Soc. 50:609-614.

Mulder, D. 1963. Recent developments in the study of tea diseases and pests in Ceylon. Food Agric. Organ. Plant Prot. Bull. 11:121-128.

Petch, T. 1923. The Diseases of the Tea Bush. Macmillan, London. 220 pp.

Venkata Ram, C. S. 1975. Blister blight of tea. Adv. Mycol. Plant Pathol., New Delhi. pp. 211-221.

Literature Cited

Purseglove, J. W. 1968. Tropical Crops, Dicotyledons. Wiley, New York. 719 pp. 2 Vol.

Food Agriculture Organization. 1981. FAO Production Yearbook. Vol. 35. FAO, Rome. 306 pp.

To simply list all of the tropical fruits would be a considerable undertaking. Anyone who has lived and traveled for a few years in the tropics soon realizes the difficulty of even learning the names of all of the tropical fruits. Bananas (discussed elsewhere) and citrus fruits are the most important and are often found growing in back yard gardens in the tropics. Statistics on production of tropical fruits are not too reliable, except as they relate to world trade, since considerable quantities are consumed locally and never enter world markets.

Fruit is a difficult term to define since in the botanical sense almost all higher plants produce fruit. The term fruit is generally restricted to those that are fleshy, can be eaten raw, and that have a high sugar content, but there are exceptions to this, i.e., avocados, breadfruit, and lemons. Many are a source rich in vitamins, especially vitamin C.

Fig. 43. Papaya planting in Puerto Rico.

Citrus fruits such as oranges, grapefruits, limes, lemons, tangerines, and citrons are widely grown. They are more important in subtropical than in tropical areas, although the potential for citrus production in the tropics is great. Most people living in temperate zones are familar with tropical fruit crops which enter world markets such as bananas, pineapples, dates, figs, and avocados. There still remain innumerable tropical fruits such as the mango, papaya, guava, breadfruit, cherimoya, soursop, sugar apple, durian, granadilla, naranjilla, caruba, and many others (Samson 1980). Space does not permit a discussion of each tropical fruit. Rather, general and selected references for the most important tropical fruits are given.

The term nut is also difficult to define. For example Menninger (1977) defines nuts as the hard-shelled fruit or seed of which the kernel is eaten. However, Webster's dictionary (Guralnik and Friend 1968) define nuts as dry, one-seeded fruit of trees or bushes consisting of a kernel in a hard shell or "any hard-shelled fruit that will keep more or less indefinitely". Tropical fruits and nuts are often considered together (Cook 1975, Cobley and Steele 1976 and Samson 1980). Coconuts and peanuts are important tropical nuts, but are discussed elsewhere in this book. There are many other nuts of importance in the tropics such as Brazil nuts, the cashew, macadamia nuts and cola nuts. Menninger (1977), Samson (1980), and Kennard and Winters (1960) list and describe the many tropical nuts.

Fig. 44. Avocados.

Literature Cited

Cobley, L. S., and W. M. Steele. 1976. An Introduction to the Botany of Tropical Crops. Longman, London. 371 pp.

Guralnik, D. B., and J. H. Friend. eds. 1968. Webster's New World Dictionary of the American Language. World Publ. Co., Cleveland, OH. 1724 pp.

General References on Tropical Fruits and Nuts and Their Diseases

Cook, A. A. 1975. Diseases of Tropical and Subtropical Fruits and Nuts. Hafner Press, New York. 317 pp.

Holtzmann, O. V., and W. Y. J. Yee. 1965. Control of diseases of fruits and nuts in Hawaii. Univ. Hawaii Coop. Ext. Serv. Circ. 408. 20 pp.

Jaynes, R. A. ed. 1969. Handbook of North American Nut Trees. Northern Nut Growers Assoc., Knoxville, TN. 421 pp.

Kennard, W. C., and H. F. Winters. 1960. Some fruits and nuts for the tropics. Misc. Publ. No. 801. U.S. Dept. Agric., Agric. Res. Serv., Washington, DC. 132 pp.

Kranz, J., H. Schmutterer, and W. Koch. eds. 1977. Diseases, Pests, and Weeds in Tropical Crops. Verlag Paul Parey, Berlin. 666 pp.

Menninger, E. A. 1977. Edible Nuts of the World. Horticultural Books, Stuart, FL. 175 pp.

Samson, J. A. 1980. Tropical Fruits. Longman, London. 250 pp.

Weber, G. F. 1973. Bacterial and Fungal Diseases of Plants in the Tropics. Univ. Florida Press, Gainesville. 673 pp.

Selected References on Avocado Diseases

Cook, A. A. 1975. Diseases of Tropical and Subtropical Fruits and Nuts. Hafner Press, New York. 317 pp.

Ruehle, G. D. 1963. The Florida avocado industry. Florida Agric. Exp. Stn. Bull. 602. 100 pp.

Stevens, H. E., and R. B. Piper. 1941. Avocado diseases in Florida. U.S. Dept. Agric. Circ. 582. 45 pp.

Weber, G. F. 1973. Bacterial and Fungal Diseases of Plants in the Tropics. Univ. of Florida Press, Gainesville. 673 pp.
Zentmyer, G. A. 1953. Diseases of the avocado. pp. 875-881. In Plant Diseases. U.S. Dept. Agr. Yearbook, Washington, DC.
Zentmyer, G. A. 1961. Avocado diseases in the Americas. Ceiba 9:61-79.
Zentmyer, G. A., A. O. Paulus, C. D. Gustafson, J. M. Wallace, and R. M. Burns. 1965. Avocado diseases. Calif. Agric. Exp. Stn. Circ. 534. 11 pp.
Zentmyer, G. A., and H. Ohr. 1978. Avocado root rot. Calif. Agric. Exp. Stn. Circ. 511.

Selected References on Citrus

Chapot, H., and V. L. Delucchi. 1964. Maladies, Troubles et Ravageurs des Agrumes an Maroc. Institute National de la Recherche Agronomique, Rabat. 339 pp.
Fawcett, H. S. 1936. Citrus Diseases and Their Control. McGraw-Hill, New York. 656 pp.
Klotz, L. J. 1973. Color Handbook of Citrus Diseases. Univ. California, Riverside. 122 pp.
Knorr, L. C. 1973. Citrus Diseases and Disorders. Univ. Florida Press, Gainesville. 163 pp.
Reuther, W., E. C. Calavan, and G. E. Carman. eds. 1978. The Citrus Industry. Vol. IV. Crop Protection. Univ. California, Berkeley. 362 pp.
Smoot, J. J., L. G. Houck, and H. B. Johnson. 1971. Market Diseases of citrus and other Subtropical Fruits. Agric. Handbook No. 398. U.S. Dept. Agric., Agric. Res. Serv., Washington, DC. 115 pp.
Proceedings of the International Organization of Citrus Virologists.
Note: The citrus virologists have a conference every three years and publish the proceedings in book form with a different editor for each volume. The series started in 1959 and contains virtually all of the general information pertinent to citrus virology from all over the world.

Selected References on Mango Diseases

Cook, A. A. 1975. Diseases of Tropical and Subtropical Fruits and Nuts. Hafner Press, New York. 317 pp.
De Laroussilhe, F. 1980. Le Manguier. G.-P. Maisonneuve and Larose, Paris. 312 pp.
Ruehle, G. D., and R. B. Ledin. 1955. Mango growing in Florida. Florida Agric. Exp. Stn. Bull. 574. 90 pp.
Sauco, V. G., A. C. Hernandez, J. Hernandez, and F. P. Padron. 1982. Revision Bibliografica de las Plagas y Enfermedades del Mango. Comm. 16. Instit. Nacional Investigaciones Agrarias, Madrid. 95 pp.
Singh, L. B. 1960. The Mango. Interscience, New York. 438 pp.

Selected References on Papaya Diseases

Cook, A. A. 1972. Virus diseases of papaya. Florida Exp. Stn. Tech. Bull. 750. 19 pp.
Cook, A. A. 1975. Diseases of Tropical and Subtropical Fruits and Nuts. Hafner Press, New York. 317 pp.

Hine, R. B., O. V. Holtzmann, and R. D. Raabe. 1965. Diseases of papaya (Carica papaya L.) in Hawaii. Hawaii Agric. Exp. Stn. Bull. No. 136. 26 pp.
Hunter, J. E., and I. W. Buddenhagen. 1972. Incidence, epidemiology and control of fruit disease of papaya in Hawaii. Trop. Agric. (Trinidad). 49:61-71.
Univ. of Hawaii. 1970. Papayas in Hawaii. Univ. Hawaii - Coop. Ext. Serv. Circ. 436. 57 pp.

Selected References on Pineapple Diseases

Collins, J. L. 1960. The Pineapple. Interscience, New York. 294 pp.
Cook, A. A. 1975. Diseases of Tropical and Subtropical Fruits and Nuts. Hafner Press, New York. 317 pp.
Guerout, R. 1975. Nematodes of pineapple: a review. Pest Articles News Sum. 21:123-140.

Fig. 45. Pineapple.

CITRUS CANKER

Disease Name: Citrus canker. The disease is also called bacterial canker and canker in the literature.

Causal Organism: *Xanthomonas campestris* pv. *citri* (Haase 1915) Dye 1978; (Pseudomonadaceae)

Clara H. Haase (1915) was the first to isolate the organism, describe it, and prove pathogenicity. She called it *Pseudomonas citri*. The bacterium was later classified by Dowson (1949) as *Xanthomonas citri* (Haase) Dowson. Following the most recent classification scheme of the bacteria, the pathogen should be called *X. campestris* pv. *citri* (Haase 1915) Dye 1978. The bacterium is a Gram-negative rod, motile with a single polar flagellum. Dimensions are 1.5-2.0 x 0.5-0.75 µm. Dye and Lelliott (1974) describe tests needed for identification of *Xanthomonas* spp.

Fawcett and Jenkins (1933) found herbarium specimens identical with citrus canker which had been collected in Northwest India probably between 1827 and 1831 and others which had been collected in Java (Indonesia) between 1842 and 1844. Thus, it was postulated that the disease probably originated in the Indo-Malay region of Asia and spread to other Asian countries during the last century.

The eradication of citrus canker is one of the few examples of successful disease eradication which plant pathologists can cite. *X. campestris* pv. *citri* was first found in the United States about 1910. Figures given by Knorr et al. (1957) state that 257,745 grove trees and over 3 million nursery trees were destroyed in Florida on 414 different properties. No infections have been found in Florida since 1926. State, federal, and private funds of $2.5 million were spent in the eradication campaign. To eradicate the disease, infected trees were burned, quarantines were established on properties which had the disease, and careful sanitation methods were used by all inspectors or others who came into contact with infected material (Fawcett 1936). Dopson (1964) and Sinclair (1968) give details of the eradication of citrus canker in other Gulf coast states of the United States. The last citrus canker was found in the United States in Texas in 1943. *X. campestris* pv. *citri* has also been eradicated from South Africa, Australia, and New Zealand (Laville 1977).

The disease has been found in many of the tropical and subtropical countries of Asia and the Pacific islands and is also found in a number of countries of Africa and South America, although it has been eradicated from several of the countries where it once occurred. Commonwealth Mycological Institute Map No. 11 gives its distribution in 1978. A new strain was recently found in Mexico (Namekata and de Olivera 1972).

Importance

The organism attacks most citrus species grown commercially, causing unsightly lesions on the fruit, fruit drop, and defoliation. The loss due to the disease in Florida was estimated at $6,500,000 between 1914 and 1931 (Knorr et al. 1957). The disease is of worldwide importance and many millions have been spent in eradication campaigns in addition to direct disease losses.

Symptoms

Any aboveground part of a tree may be affected but leaves, twigs, and fruits most commonly show symptoms. On the leaves the spots are at first

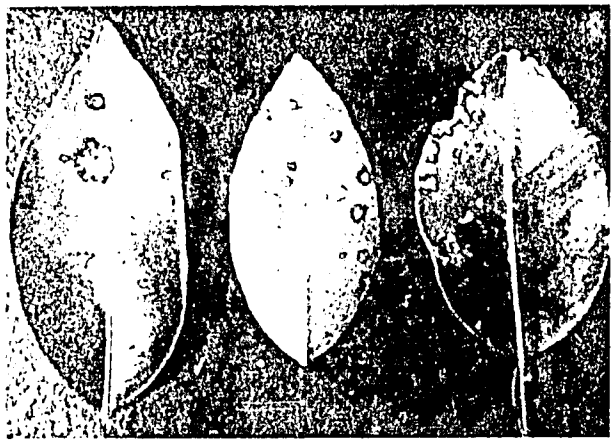


Fig. 46. Lesions caused by *Xanthomonas campestris* pv. *citri* (citrus canker).

white, changing to gray or tan, and finally the spongy lesion ruptures to expose a typical crater-like formation. The lesions have a oily margin or yellowish halo. The size of the lesion varies with the citrus species attacked, but may be as large as 12 mm in diameter on grapefruit. On fruit the lesions are spongy, raised, have oily margins, and often erupt to show the typical crater-like appearance. Fawcett (1936), Klotz (1973), and Knorr et al. (1957) give detailed descriptions of disease symptoms.

Host Plants

In general, grapefruit is the most susceptible of the citrus fruits, but in addition (in descending order of susceptibility) trifoliate orange, limes, sweet oranges, lemons, and Satsuma mandarins are attacked. Tangerines and citrons are most resistant (Klotz 1973 and Fawcett 1936). The order of susceptibility may vary with the strain of the bacterium causing disease.

Pathogen Cycle

The bacteria may survive environmental conditions unfavorable for infection on the plant in lesions on leaves, twigs, and stems. Longest survival is in cankers on woody tissue. Bacteria are also reported (Goto et al. 1975b) to survive in plant debris and for short periods in the soil. Goto et al. (1975a) also report survival in a grass host (*Zoysia japonica*) found in citrus plantings in Japan. Bacteria are exuded from lesions when wet and are disseminated by rain or dew onto other parts of the plant. Wind, man, and possibly insects aid in dissemination. Wind is especially important. Insects may cause injuries which increase disease by producing avenues of entrance.

Peltier (1920) found 20-30°C optimal for both growth and infection. *X. campestris* pv. *citri* penetrates through stomata, lenticels, and wounds. The activities of the organism once it is established within the host are poorly understood. Koizumi (1976) found multiplication of *X. campestris* pv. *citri* in intercellular spaces at 6-25°C, but not at 40°C. Hypertrophy of parenchyma cells and subsequent abundant bacterial multiplication were observed.

X. campestris pv. *citri* may remain inactive in cankers on older woody tissue for long periods. This seems to be the most important means of overseasoning, although the organism also may survive to some extent on plant debris and in the soil. Optimal conditions for infection occur when rapidly growing tissue is covered with free moisture for 20 minutes or more, and temperatures are between 20-30°C (Peltier 1920).

Control

The successful eradication of citrus canker from several countries is one of the few examples of successful eradication of a plant pathogen. Exclusion by means of rigid quarantines is the best means of control where the disease does not exist, or after it has been eradicated. Thousands of plants or plant

parts have been intercepted by quarantine agents of the United States, California, and [illegible] disease was eradicated in the United States. For example X. campestris pv. citri was intercepted 378 times by United States quarantine agents [illegible] material coming from 24 countries of Asia, Africa, and South America [illegible] 1978 through September, 1979 (U.S.D.A. 1981). This is a clear [illegible] the need for strict quarantines in countries or regions where [illegible] not exist.

In countries [illegible] disease is established, and where eradication measures would [illegible], resistant cultivars or species can reduce disease losses. As [illegible] mentioned, most citrus species are susceptible, but tangerines, [illegible], pummelo, and citrons are commercially resistant. The Tahiti lime is resistant, and sour orange and sweet orange cultivars are available which [illegible] with little loss from canker. In Japan when the moderately resistant [illegible] Satsuma mandarin) is grown, no additional control measures are needed [illegible] 1978).

Citrus leaves [illegible] more resistant with age. Stall et al. (1982) reported that, although resistance in leaves to the bacterium was previously believed to be determined by stomatal structure, they found a positive correlation between inoculum density and numbers of lesions developing in infiltrated mesophyll. Resistance of the mesophyll to infection increased as leaves matured.

Two major strains of X. campestris pv. citri are known and designated as strain A (native to Asia and the one eradicated from Florida) and strain B which is found in Argentina, Paraguay, and Uruguay (Stall and Seymour 1983). Strain B is most serious on lemons and is less damaging to sweet and sour orange, lime, citron, and grapefruit. Recently Namekata and de Oliveria (1972) described a third or biologically distinct strain of the bacterium which mainly affects Mexican lime.

There is an extensive literature on chemical control of X. campestris pv. citri. Copper compounds such as Bordeaux mixture, copper sulfate, various fixed coppers, and [illegible]dium arsenite, organic mecury, and various organic compounds have been used alone or in combination for control. Antibiotics such as streptomycin and [illegible]romycetin also are used (Laville 1977). Applications should be generally timed to the growth stage of leaves and fruit. In Japan copper compounds [illegible] streptomycin are applied seven times per year on susceptible cultivars ([illegible] 1978). In Japan, in addition to resistant cultivars and chemical control, pruning, wind fences (to reduce wind velocity and thus prevent spread), and [illegible]rasting are also used.

Literature Cited

Commonwealth Mycological Institute. 1978. Distribution maps of plant disease No. 11, ed. [illegible]. Commonw. Agric. Bur., Kew, Surrey.

Dopson Jr., R. W. 1964. The eradication of citrus canker. Plant Dis. Rep. 48:30-31.

Dowson, W. J. 1939. Manual of Bacterial Plant Diseases. Adam & Charles Black, London. [illegible] pp.

Dye, D. W., and [illegible] Lelliott. 1974. Genus II. Xanthomonas. Dowson 1939, 187. pp. [illegible]. In Bergey's Manuel of Determinative Bacteriology, 8th ed. R. E. [illegible] and N. E. Gibbons. eds. Williams & Wilkins, Baltimore, MD. 1246 pp.

Fawcett, H. S., and A. E. Jenkins. 1933. Records of citrus canker from herbarium specimens of the genus citrus in England and the United States. Phytopathology 23:820-824.

Fawcett, H. S. 1936. Citrus Diseases and Their Control. McGraw-Hill, New York. 656 pp.

Goto, M., K. Ohta, and N. Okabe. 1975a. Studies on saprophytic survival of Xanthomonas citri (Hasse) Dowson. 1. Detection of the bacterium from a grass (Zoysia japonica). Ann. Phytopathol. Soc. Japan 41:9-14.

Goto, M., K. Ohta, and N. Okabe. 1975b. Studies on saprophytic survival of Xanthomonas citri (Hasse) Dowson. 2. Longevity and survival density of the bacterium on artificially infested weeds, plant residues and soils. Ann. Phytopathol. Soc. Japan 41:141-147.

Haase, C. H. 1915. Pseudomonas citri, the cause of citrus canker. J. Agric. Res. 4:97-100.

Klotz, L. J. 1973. Color Handbook of Citrus Diseases. Univ. California, Riverside. 122 pp.

Knorr, L. C., R. F. Suit, and E. P. DuCharme. 1957. Handbook of citrus diseases in Florida. Florida Agric. Exp. Stn. Bull. 587. 157 pp.

Koizumi, M. 1976. Behavior of Xanthomonas citri (Hasse) Dowson in the infection process. I. Multiplication of the bacteria and histological changes following needle-prick inoculation. Ann. Phytopathol. Soc. Japan 42:407-416.

Kuhara, S. 1978. Present epidemic status and control of the citrus canker disease (Xanthomonas citri (Hasse) Dowson) in Japan. Rev. Plant Protec. Res. 11:132-142.

Laville, E. 1977. Xanthomonas citri (Hasse) Dowson pp. 62-64, In Kranz, J., H. Schmutterer, and W. Koch. eds. Diseases, Pests and Weeds in Tropical Crops. Verlag Paul Parey, Berlin. 666 pp.

Namekata, T., and A. R. de Oliveria. 1972. Comparative serological studies between Xanthomonas citri and a bacterium causing canker on Mexican lime. pp. 151-152 In Maas Geesteranus, H. P. ed. Proc. 3rd Intl. Conf. on Plant Pathogenic Bacteria. Centre Agric. Publ. and Documentation, Wageningen.

Peltier, G. L. 1920. Influence of temperature and humidity on the growth of Pseudomonas citri and its host plants and on infection and development of the disease. J. Agric. Res. 20:447-506.

Sinclair, J. B. 1968. Eradication of citrus canker in Louisiana. Plant Dis. Rep. 52:667-670.

Stall, R. E., G. M. Marco, and B. I. Canteros de Echenique. 1982. Importance of mesophyll in mature-leaf resistance to cancrosis of citrus. Phytopathology 72:1097-1100.

Stall, R. E., and C. P. Seymour. 1983. Canker, a threat to citrus in the Gulf-Coast states. Plant Dis. 67:581-585.

U.S.D.A. 1981. List of intercepted plant pests. Animal and Plant Health Inspection Serv., U.S. Dept. Agric., Hyattsville, MD. 171 pp.

Sugar cane (various *Saccharum* spp.) provides 65% of the world's sugar. Global production of centrifugal cane sugar was over 64 million tons produced on 13 million hectares in 1981, but significant quantities of non-centrifugal sugar were also produced. It is an important cash crop in many tropical countries. Sugar cane originated in the tropics and subtropics of Asia. The most important product of the cane is sucrose. Important byproducts of sugar refining are molasses, rum, industrial alcohol, cane syrup, numerous chemical derivatives, and bagasse, the fibrous residue left over after extraction. Bagasse and molasses may be used as supplemental animal feed. Sugar cane chewing is common in the tropics, and special soft varieties are planted near homes for this purpose. Some species are important locally as construction material for houses, and may be grown commercially for this purpose.

Controversy exists on the taxonomy of Saccharum. Purseglove (1972) lists the following species: *S. spontaneum*, *S. robustum*, *S. officinarum*, *S. barberi*, and *S. sinense*. *S. officinarum*, called "noble cane," has thick stems unlike the thin stems of *S. barberi* and *S. sinense*. Captain Bligh of "Mutiny on the Bounty" fame transported varieties of noble canes from Tahiti to Jamaica to introduce it for the first time in the Americas. A vast number of sugarcane clones are found in the tropics.

Sugar cane is propagated by pieces of stalk, usually with two or three nodes with buds. These seed-pieces are placed in a furrow and covered with soil to germinate. Sugar cane is a perennial grass and the first crop, depending on climate and soil conditions, may be harvested after 9 months or several years. After harvesting, again depending on local conditions, two to four additional crops, called "ratoons", may be harvested, although up to 20 ratoon

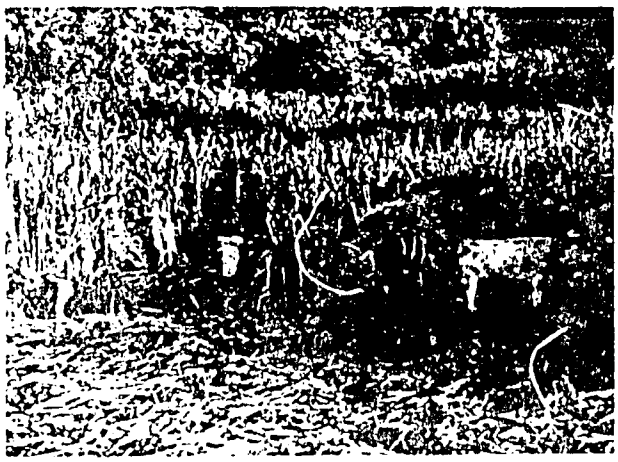


Fig. 47. Cutting sugar cane by hand in Puerto Rico.

crops have been recorded. It is often the only crop grown and may be planted on the same land for centuries. Sugar cane is usually grown as a monoculture but under a wide variety of soil and climate conditions in the tropics. Sugar cane grows between 35°N and S latitude.

The situation regarding large-scale commercial production of sugar cane is very similar to that of banana. Because of the tremendous infrastructure required for cane transportation and processing, very large areas are continuously cropped to cane. This insures that the factories will continuously operate to near capacity. Cane and bananas are usually grown on the best land, and management tends to be concentrated in the hands of a very few. The question of devoting the best lands of a nation to production of export crops is a very difficult one to address. Many of these countries are net food importers. However, it is undeniable that a country requires foreign currency to import necessary items, other than food, such as petroleum, medicine and other technology. Often, these export crops are the only feasible means available at this time of earning currency. Deducting the cost of food imports from sugar earnings may still leave a sizeable foreign exchange surplus.

Diseases have caused immense losses to the sugar industry in the form of abandonment of land and factories, migration of peoples, and severe financial losses or failures to many growers. Because of the risk of great losses should cane production in a region become permanently impossible, and because of the relatively low value of the crop on a per hectare basis, large-scale chemical treatments are generally impractical. Cane growers must rely on a variety of methods for disease and pest control. These include careful control of planting material quality to avoid planting virus-infected stems and chemical treatment of material prior to planting to prevent losses due to rot. Many sugar cane companies have among the world's best biological control programs to reduce losses due to insects. However, the most important disease control measure is through varietal resistance.

Considerable success has been achieved in sugar cane improvement by breeding especially through interspecific hybridization. Severe disease epidemics were recorded on Mauritius and Renunion as early as 1840. Later, sugar cane mosaic, which first appeared in Indonesia about 1890, was carried throughout the world with the noble canes before its seriousness was appreciated. It almost destroyed the industry in Louisiana in the 1920's and caused serious losses in many other sugar-growing areas. Control was obtained with resistant canes. In 1887, the Dutch scientist Soltwedel, working in Indonesia, succeeded in growing seed for the first time from tassels (sugar cane infloresence). Resistance to sugar cane mosaic was later achieved by breeding, and since that time, new varieties have repeatedly saved the industry as new diseases have appeared. Large germplasm collections are found in the United States, India, and Brazil. Sugar cane improvement is generally well financed by governments and commercial interests.

Hughes (1978) recently reviewed the worldwide importance of sugar cane diseases. At present, he considers ratoon stunting disease, leaf scald (Xanthomonas albilineans), and smut (Ustilago scitaminea) the most important among the 85 parasitic diseases of cane known. Other important sugar cane diseases listed by Hughes (1978) are red stripe (Pseudomonas rubrilineans), eye spot (Helminthosporium sacchari), pineapple disease (Ceratocystis paradoxa), red rot (Glomerella tucumanensis), Fiji disease (virus), and mosaic (virus).

Selected References on Sugar Cane Diseases

Abbot, E. V. 1953. Sugarcane and its diseases. pp. 526-535. In Plant Diseases. U.S. Dept. Agric. Yearbook, Washington, DC.

Edgerton, C. W. 1958. Sugar Cane and its Diseases. Louisiana State Univ. Press, Baton Rouge, LA. 301 pp.

Hughes, C. G., E. V. Abbott, and C. A. Wismer. 1964. Sugar-Cane Diseases of the World. Vol. 2. Elsevier, New York. 354 pp.

Martin, J. P., E. V. Abbott, and C. G. Hughes. 1961. Sugar-Cane Diseases of the World. Vol. 1. Elsevier, New York. 542 pp.

Literature Cited

Hughes, C. G. 1978. Diseases of sugarcane - a review. Pest Articles News Sum. 24:143-159.

Purseglove, J. W. 1972. Tropical Crops, Monocotyledons. Wiley, New York. 607 pp. 2 Vol.

Fig. 48. Sugar cane mosaic.

RATOON STUNTING DISEASE OF SUGAR CANE

Disease Name: Ratoon stunting disease of sugar cane, stunting disease.

Causal Organism: *Clavibacter xyli* subsp. *xyli*.

"Ratoon stunting disease is undoubtedly the most important disease problem with the world cane industry" stated Hughes (1978) in a recent article. Ratoon stunting disease was first suspected to be a distinct disease in Australia in the 1944-1945 crop in Queensland, Australia and was most severe on the then new variety Q 28 (Hughes and Steindl 1955). Since that time the pathogen has been found all over the world and has been a problem almost everywhere cane is grown. The disease causes few distinct symptoms and thus was not easily recognized until spread had already taken place. The Commonwealth Mycological Institute's maps of plant disease give its distribution. The disease is found in almost all sugar cane growing areas of the world.

For many years the ratoon stunting disease was thought to be caused by a virus as the causal organism had many virus characteristics. Work by Gillaspie et al. (1973), Teakle et al. (1973) and Maramorosch et al. (1973) indicated that a bacterium of the coryneform group might be the causal agent. Subsequently, the causal agent has been cultivated on artificial media by Davis et al. (1980) and Liao and Chen (1981). In culture the bacterium is a rod (0.25-0.35 x 2-5 μm), gram-positive, non-motile, lacking endospores, aerobic, and of clublike or V-form morphology (Liao and Chen 1981). Gillaspie et al. (1981) referring to the work of Davis et al. (1980) state that their work "proves that the RSD-associated bacterium is the causal agent of the disease". Similar bacteria from Australia, Brazil, Japan, South Africa, and the U.S.A. were closely related serologically (Gillaspie et al. 1979). Davis et al. (1984) recently named the new bacterium *Clavibacter xyli* subsp. *xyli* in the new genus *Clavibacter*.

Importance

The pathogen is easily transmitted mechanically, and since all cane for planting is cut, it spreads very rapidly and in a few years infection levels become very high. Yields, especially of ratoon crops, are reduced considerably, and it may become necessary to change varieties. In Australia, where the problem was first shown to be a disease, the susceptible Q 28 variety, which reached a harvest of over 800,000 tons in 1948, soon became of minor importance because of the disease (Hughes and Steindl 1955). Edgerton (1958) believes that many of the shifts in varieties throughout the sugar cane growing areas of the world may be due in great part to ratoon stunting disease in the leading varieties.

In Louisiana Forbes et al. (1960) reported an increase in yield of 33 percent for the plant cane and 86 percent for the first ratoon with elimination of the disease. In Colombia, Galvez and Thurston (1961) reported yield losses of 70% in the variety EPC-33833 for the plant cane. Steib (1972), also in Lousiana, reported losses between 13-24% due to ratoon stunting disease on different varieties. In Taiwan losses of 20% were reported (Steindl 1961).

Symptoms

Ratoon stunting does not have well defined external symptoms. Stunting, though present in many varieties, is absent in others. A general "unthriftiness" and chlorosis may be present but can be caused by so many factors, i.e.

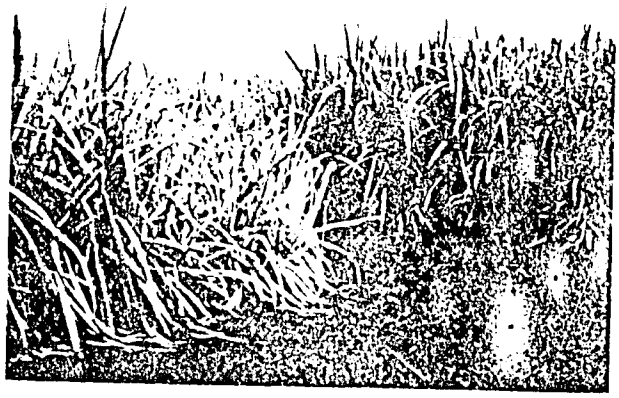


Fig. 49. Right - Cane treated with hot water for ratoon stunting disease control. Left - Non-treated cane. Palmira, Colombia.

poor cultural practices, lack of moisture or fertilizer, that diagnosis is difficult. Diseased cane may be especially susceptible to lack of moisture, and in some varieties the diseased cane will wilt more readily than healthy cane. Internal discoloration is generally associated with ratoon stunting. In mature diseased cane of many varieties there is a reddish discoloration of the vascular system at the level of the nodes. In very young cane of some varieties the nodes are pink. Color may vary from orange to reddish brown. The color symptom is quite variable, and one must know what colors are found in healthy material. There may be symptomless carriers of the disease, and other factors can cause vascular discoloration in sugar cane; thus, finding reddish discoloration of the vascular system is not a positive diagnosis of the disease. Positive diagnosis of ratoon stunting disease is now often based on the presence of the coryneform bacterium utilizing phase microscopy (Teakle et al. 1973, Richardson 1978) or by inoculation of a sensitive indicator plant such as elephant grass (Matsuoka 1972) or cane variety Q 28.

Host Plants

The host range has been investigated, primarily with the hope of finding a plant that would show diagnostic symptoms, but also to determine the role of other plants which might act as a source of the bacterium. A number of grass hosts have been found including maize, sorghum, sweet sudan grass, and a number of grass weeds commonly found in cane fields. Elephant grass (*Pennisetum purpureum*) was described by Matsuoka (1972) as a useful indicator plant in which symptoms appear 20-30 days after inoculation. The role of alternate hosts in the epidemiology of the disease is not clear.

Pathogen Cycle

The bacterium is found in all parts of the sugar cane plant and is transmitted easily by mechanical means. Juice from diseased plants at dilutions of 1/25,000 is infectious (Steindl 1961). Cutting knives used for preparing cuttings or sets (seeds) and cutter planting machines readily transmit the bacterium. Sixty consecutive plants have been infected with a cutter planter (Steindl 1961). Machines used in harvesting operations also transmit the pathogen. Man is the primary agent in spreading the bacterium; however rats have been shown to transmit the pathogen by chewing (Wehlburg 1956). The pathogen does not survive in soil; there is no evidence of transmission through true seed; and no insect vector is known.

Control

Infected cane can be treated either by hot water (2 hours at 50°C), aerated stem (4 hours at 53°C), or hot air (8 hours at 58°C) (Damann and Benda

1983). Heat treatment ovens and tanks are commercially manufactured for treating cane. The hot air treatment causes less loss in seed germination than the hot water treatment. After treatment, seed should be treated with fungicides to prevent attack by fungi in the soil. Cane which is free from the disease after being heat treated should be kept free in the field from volunteer cane which might carry the disease. All equipment, knives, planters, harvesters, etc., should be sterilized before use in treated cane. Intelligent use of sanitation can maintain stocks free of the bacterium indefinitely. Hot water, aerated steam, and hot air treatments are not necessarily 100% effective. Hot air treatment was not as effective as hot water according to South African workers (Bailey et al. 1978).

Resistant varieties are important in reducing losses from ratoon stunting, and varieties vary considerably in their resistance. Teakle et al. (1975) suggest that three types of resistance to ratoon stunting occur. First, "tolerant" clones which, although infected, do not suffer serious yield losses. Second, "field resistant" clones which may escape infection in the field but are seriously affected when artificially inoculated. Thirdly, Wismer (1971) reported a clone (H60 - 6909) that was inoculated, but failed to transmit the disease to indicator clones after repeated inoculations. This clone appeared to be "immune", but recently several investigators have observed that H60-6909 will support growth of low numbers of the bacterium. Relatively little work appears to have been done on breeding for resistance to ratoon stunting disease. Teakle et al. (1975), by studying the flow of water and India ink through the vascular system of clones of known resistance to ratoon stunting disease, concluded that vascular anatomy may be a factor in resistance to ratoon stunting. Low numbers of large, continuous vessels passing through nodes were clearly correlated with resistance. Koike et al. (1982) found that yield effects of ratoon stunting disease in the field were related to the numbers of the causal bacterium in the stalk juice.

Literature Cited

Bailey, R. A., G. R. Bechet, and S. R. Richardson. 1978. Ratoon stunting disease in imported sugarcane varieties after hot air treatment. Sugarcane Pathologist's Newsletter 21:16.

Damann Jr., K. E., and G. T. A. Benda. 1983. Evaluation of commercial heat-treatment methods for control of ratoon stunting disease of sugarcane. Plant Dis. 67:966-967.

Davis, M. J., A. G. Gillaspie Jr., R. W. Harris, and R. H. Lawson. 1980. Ratoon stunting disease of sugarcane: isolation of the causal organism. Science 210:1365-1367.

Davis, M. J., A. G. Gillaspie Jr., A. K. Vidaver, and R. W. Harris. 1984. Clavibacter: a new genus containing some phytopathogenic coryneform bacteria, including Clavibacter xyli subsp. xyli sp. nov., subsp. nov. and Clavibacter xyli subsp. cynodontis subsp. nov., pathogens that cause ratoon stunting disease of sugarcane and bermudagrass stunting disease. Int. J. Syst. Bacteriol. 34:107-117.

Edgerton, C. W. 1958. Sugarcane and its Diseases. Louisiana State Univ. Press, Baton Rouge. 301 pp.

Forbes, I. L., R. J. Steib, and S. J. P. Chilton. 1960. Ratoon stunting disease of sugar cane. Louisiana State Univ. and Agric. and Mech. Coll., Bull. No. 532. 23 pp.

Galvez, G. E., and H. D. Thurston. 1961. Ratoon stunting disease of sugar cane in Colombia. Plant Dis. Rep. 45:954-956.

Gillaspie, A. G. Jr., R. E. Davis, and J. F. Worley. 1973. Diagnosis of ratoon stunting disease based on the presence of a specific microorganism. Plant Dis. Rep. 57:987-990.

Gillaspie, A. G. Jr., R. W. Harris, and D. S. Teakle. 1979. Serological comparison of RSD-associated bacteria from five sugarcane growing areas of the world. Sugarcane Pathologist's Newsletter 23:23-24.

Gillaspie, A. G. Jr., M. J. Davis, R. W. Harris, and R. H. Lawson. 1981. Cultivation of the causal agent of ratoon stunting disease of sugarcane. Sugarcane Pathologist's Newsletter 26:6.

Hughes, C. G., and D. R. L. Steindl. 1955. Ratoon stunting of sugar cane. Tech. Comm. No. 2, Bur. Sugar Exp. Stn., Brisbane, Australia. 54 pp.

Hughes, C. G. 1978. Diseases of sugarcane -- a review. Pest Articles News Sum. 24:143-159.

Koike, H., A. G. Gillaspie, Jr., and G. T. A. Benda. 1982. Cane yield response to ratoon stunting disease. Int. Sugar J. 84:131-133.

Liao, C. H., and T. A. Chen. 1981. Isolation, culture, and pathogenicity to Sudan grass of a Corynebacterium associated with ratoon stunting of sugarcane and with Bermuda grass. Phytopathology 71:1303-1306.

Maramorosch, K., B. Plasvic-Banjac, J. Bird, and L. J. Liu. 1973. Electron microscopy of the ratoon stunted sugarcane: microorganisms in xylem. Phytopathol. Z. 77:270-273.

Matsuoka, S. 1972. Ratoon stunting disease diagnosis with elephant-grass as an indicator plant. Sugarcane Pathologist's Newsletter 8:10-11.

Richardson, S. R. 1978. An improved method of xylem-sap extraction using positive pressure for the rapid diagnosis of ratoon stunting disease. Sugarcane Pathologist's Newsletter 21:17-18.

Steib, R. J. 1972. Results of studies relating to the mosaic - RSD disease complex of sugarcane in Louisiana. Sugarcane Pathologist's Newsletter 8:8-9.

Steindl, D. R. L. 1961. Ratoon stunting disease. pp. 432-459, In Martin, J. P., E. V. Abbot, and C. G. Hughes. Sugar Cane Diseases of the World. Vol. 1. Elsevier, New York. 542 pp.

Teakle, D. S., P. M. Smith, and D. R. L. Steindl. 1973. Association of a small coryneform bacterium with the ratoon stunting disease of sugar cane. Aust. J. Agric. Res. 24:869-874.

Teakle, D. S., P. M. Smith, and D. R. L. Steindl. 1975. Ratoon stunting disease of sugarcane: possible correlation of resistance with vascular anatomy. Phytopathology 65:138-140.

Wehlburg, C. 1956. Ratoon stunting disease in Cuba. Sugar 51:27-29.

Wismer, C. A. 1971. A sugarcane clone apparently immune to RSD. Sugarcane Pathologist's Newsletter 6:46.

Vegetables

Little reliable information is available on the importance of tropical vegetables. A few such as tomatoes, Chinese cabbage, eggplant, peppers, sweet potatoes, onions, and various curcurbits are widely grown. Many are grown and consumed locally, but it is difficult to judge their relative importance as few reliable statistics are compiled on tropical vegetable production. Considerable confusion exists in defining what a vegetable is, and innumerable different classifications of plants into categories as vegetables and horticultural crops are found throughout the world (Grubben 1977). Hundreds of crops are considered vegetables depending on the definition and classification used. For example, the leaves of innumerable species of "weeds" are used as pot-herbs in Latin America, Asia, and Africa (Martin and Ruberte 1975, Oomen and Grubben 1978), and some would consider these vegetables.

Many vegetables are important sources of vitamins, minerals, and proteins and could play a much more important role than they do at present in supplementing tropical diets which are primarily carbohydrate. Grubben (1977) estimates that over half of the vegetables grown in developing countries (primarily tropical) are for home consumption, but that in developed countries only 13% are for home consumption. In addition to improving nutrition, especially among low income groups, many vegetables are labor intensive and often in short supply, and thus are suitable for increasing small farmer income.

The Asian Vegetable Research and Development Center (AVRDC) has estimated that yields of vegetables in the tropics are about one-third of those of the same crops in temperate countries. The AVRDC is located in the Republic of China (Taiwan) near Tainan City. Funding, construction of facilities, and initial research began in 1972. AVRDC is primarily supported by the Republic of China (Taiwan) and the governments of the United States, Thailand, the Philippines, Japan, Korea, and the Federal Republic of Germany. In Southeast

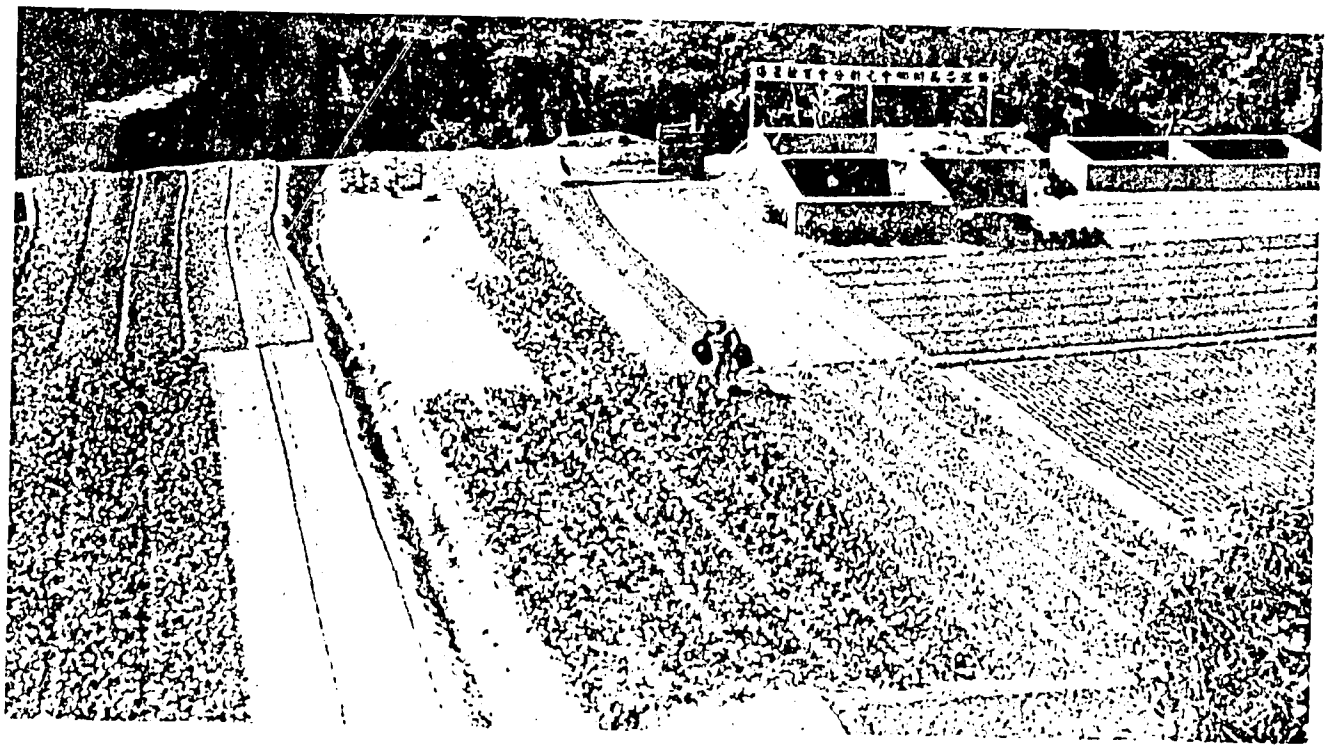


Fig. 50. Vegetable growing in Hong Kong.

Asia, vegetables, which are sources rich in vitamins, minerals, and protein, are needed to supplement rice which is the major source of calories in diets. AVRDC has selected five vegetables on which to concentrate - mung bean, soybean, tomato, Chinese cabbage, and sweet potato. Many nations in the tropics have programs of vegetable improvement (Grubben 1977).

Much of the vegetable seed planted in the tropics is imported from temperate countries. Often, varieties are grown that have been developed for temperate conditions and they are lacking in adaptation to tropical environments. In addition, seed of inferior quality with poor germination, and susceptible to local diseases, often is all that is available in the tropics.

Diseases are major limiting factors in vegetable production in the tropics. For example, tomatoes are among the most widely grown vegetables in the tropics (Villareal 1980). The bacterium Pseudomonas solanacearum (causal agent of bacterial wilt) is the most serious pathogen in tomato production below 1000 meters in many tropical countries throughout the world. Resistance to P. solanacearum has been found but is not highly effective at high temperatures. The tomato grows well in the tropics, and its strong market demand and high vitamin content make it important in improving nutrition and for improving small farmer income. Resistance to P. solanacearum in tomatoes that would be effective in the hot, humid areas of the lowland tropics would constitute an important contribution to the economies and nutrition of tropical countries. Great losses occur after harvest as most vegetables are highly perishable, and methods of preservation such as refrigeration, canning, drying, and pickling often are not available or economical in many tropical countries. A number of general references on tropical vegetables and their diseases are available.

Selected References on Tropical Diseases of Vegetables

Chupp, C., and A. F. Sherf. 1960. Vegetable Diseases and Their Control. Ronald Press, New York. 693 pp.

Cook, A. A. 1978. Diseases of Tropical and Subtropical Vegetables and Other Plants. Hafner Press, New York. 381 pp.

Herklots, G. A. C. 1972. Vegetables in South-East Asia. George Allen and Unwin, London. 525 pp.

MacNab, A. A., A. F. Sherf, and J. K. Springer. 1983. Identifying Diseases of Vegetables. Pennsylvania State Univ., Univ. Park, PA. 62 pp.

Messiaen, C.-M., and R. Lafon. 1970. Les Maladies des Plantes Maraicheres. Inst. Natl. de la Recherche Agronomique, Paris. 441 pp.

Mortenson, E., and E. T. Bullard. 1964. Handbook of Tropical and Subtropical Horticulture. Agency Int. Development, Dept. of State, Washington, DC. 186 pp.

Tindall, H. D. 1983. Vegetables in the tropics. Avi, Westport, CT. 533 pp.

Walker, J. C. 1952. Diseases of Vegetable Crops. McGraw-Hill, New York. 529 pp.

Weber, G. 1973. Bacterial and Fungal Diseases of Plants in the Tropics. Univ. Florida Press, Gainesville, FL. 673 pp.

Winters, H. F., and G. W. Miskimen. 1967. Vegetable Gardening in the Caribbean Area. Agric. Handbook No. 323. U.S. Dept. Agric., Agric. Res. Serv., Washington, DC. 114 pp.

Literature Cited

Grubben, G. J. H. 1977. Tropical Vegetables and Their Genetic Resources. Int. Board Plant Genet. Resources, Rome. 197 pp.

Martin, F. W., and R. M. Ruberte. 1975. Edible Leaves of the Tropics. Mayaguez Inst. Trop. Agric., Mayaguez, Puerto Rico. 235 pp.

Oomen, H. A. P. C., and G. J. H. Grubben. 1978. Tropical Leaf Vegetables in Human Nutrition. Royal Trop. Inst., Amsterdam. 140 pp.

Villareal, R. L. 1980. Tomatoes in the Tropics. Westview Press, Boulder, CO. 174 pp.

Oil Crops

Both edible and nonedible oils can be obtained from a wide variety of tropical crops. Soybeans and peanuts are major sources of oils for human consumption and industrial purposes, but have been discussed in this book under grain legumes. Maize, cotton, and cocoa are other important sources of oil and are also discussed elsewhere. Safflower, tung, castor oil plants, and lemon grass are only a few of the many other tropical oil crops.

COCONUTS

Coconuts (Cocos nucifera) were the major worldwide source of edible oil until soybean oil recently became more important. Worldwide, five million tons of copra were produced in 1981 (FAO 1981). The coconut is considered to be one of the most valuable plants in the tropics, not just for the oil it produces, but for its many other uses. They are important to the economy of many tropical countries such as the Philippines, whose major export is coconuts.

Coconuts probably originated in Asia, but this is still a subject of controversy, and some evidence exists for an origin along the Pacific coasts of Colombia and Ecuador. The first European explorers found them in all areas of the tropics except for the Atlantic coasts of America and Africa. Coconuts appear to have been distributed by man, but possibly the floating nuts were carried to new areas by ocean currents.

The coconut palm is typically a tall, graceful, unbranched tree which can reach a height of 20-30 meters. As coconuts are not propagated vegetatively, but rather by seed, populations may be quite heterozygous. Two major types are tall palms and dwarf palms, which grow to only 10 m. Tall cultivars do not begin to produce until they are 6-7 years old, but dwarf varieties may begin production after only three years. The widely distributed "Malayan Dwarf" cultivar is becoming increasingly important because of its resistance to the lethal yellowing disease.

The fruits or nuts are large (20-30 cm in diameter) with a thick, fibrous mesocarp, and weigh up to one kilogram. A hard shell, or endocarp, envelops the seed which has a large, white, meaty endosperm. This endosperm is high in oil and completely surrounds the internal cavity filled with coconut water. A mature tree will produce 35-70 nuts per year. Coconuts may live and produce

for a century. As they are salt-tolerant, they are commonly found near the ocean, but also can be grown inland.

The majority of the international coconut trade is in copra, the dried endosperm. Copra contains roughly 60-70% oil of which over 60% is extractable. This oil is widely used in the production of soap, cosmetics, margarine, and processed foods. In addition to these industrial applications of the oil, innumerable uses of coconuts are found in tropical areas, especially on the Pacific islands. They are an important source of food both fresh and dried, the water in unripe fruits is a delicious beverage, and coconut milk (made by squeezing the endosperm) is used in cooking. The oil is edible and locally used for soap, cooking, and as a body ointment. Shells are often burned for fuel, and various containers and other articles are made from them. The fiber in the husks is made into rugs, mats, rope, and twine. Coconut palm wine is made in many countries. The trunk is used in construction, and the leaves are used as house thatching and for woven articles. Few of man's plants have such a wide variety of uses.

Fig. 51. Maize and coconuts in the tropics of the Philippines.

Considering the importance of coconuts relatively little is known about their diseases. Some important diseases are bud rot (Phytophthora palmivora), lethal bole rot (Marasmiellus cocophilus), red ring (the nematode Rhadinaphilanchus cocophilus), cadang-cadang (a viroid suspected), and lethal yellowing (mycoplasma). Many coconut diseases are of unknown etiology (Marasmorosch 1964).

Lethal yellowing of coconut palms is a serious threat to coconuts throughout the world. The disease is confined to the Americas and Africa at present. For some time it caused the loss of 100,000 coconuts per year in Jamaica (Plavsic-Banjac et al. 1972). Mycoplasma-like bodies have been found associated with the disease and that evidence, plus remission of symptoms in trees treated with tetracycline antibiotics (McCoy 1972), suggest a mycoplasmal etiology. It is probable that the causal agent is transmitted by an insect vector, but none has been found to date. Control measures such as detection and eradication of diseased palms are not entirely effective, as disease spread continues. Tetracycline therapy, although economic for high value palms in Florida, is too expensive for most tropical countries. The use of resistant coconut cultivars, such as Malayan Dwarf, appears to be the best control measure.

Cadang-cadang disease of coconuts is only found in the Philippines. First reported in 1937 (Ocfemia 1937), it has destroyed millions of trees since that time in the limited area of the Philippines where it was found. The disease moves very slowly, and a tree may not die from cadang-cadang for 5-10 years after first symptom appearance. The causal agent of cadang-cadang disease probably is a viroid (Mohamed and Imperial 1984).

Selected References on Coconut Diseases

Briton-Jones, H. R. 1940. The Diseases of the Coconut Palm. Bailliere, Tindall and Cox, London. 176 pp.

Central Plantation Crops Research Institute. 1976. Coconut diseases of uncertain etiology. Tech. Bull. No. 1. Central Plantation Crops Res. Inst., Kerala, India. 30 pp.

Child, R. 1974. Coconuts. Longman, London. 335 pp.

Del Rosario, M. S. E. 1967. Coconut diseases. Tech. Bull. 20. Coll. Agric., Univ. Philippines, College, Laguna, Philippines. 41 pp.

Maramorosch, K. 1964. A Survey of Coconut Diseases of Unknown Etiology. Food Agric. Organ., United Nations, Rome. 39 pp.

Menon, K. P. V., and K. M. Pandalai. 1958. The Coconut Palm. Indian Central Coconut Comm. Ernakulum, India. 384 pp.

Thampan, P. K. 1981. Handbook on Coconut Palm. Oxford and IBH, New Delhi. 311 pp.

Literature Cited

Food Agriculture Organization. 1981. FAO production yearbook. Vol. 35, FAO, Rome. 306 pp.

McCoy, R. E. 1972. Remission of lethal yellowing in coconut palm treated with tetracycline antibiotics. Plant Dis. Rep. 56:1019-1021.

Mohamed, N. A., and J. S. Imperial. 1984. Detection and concentration of coconut cadang-cadang viroid in coconut leaf extracts. Phytopathology 74:165-169.

Ocfemia, G. O. 1937. The probable nature of cadang-cadang disease of coconuts. Phillip. Agric. 26:338-340.

Plavsic-Banjac, B., P. Hunt, and K. Maramorosch. 1972. Mycoplasmalike bodies associated with lethal yellowing disease of coconut palms. Phytopathology 62:298-299.

RED RING DISEASE OF COCONUT

Disease Name: Red ring disease of coconut is the most common name. Older names were root disease and fever.

Causal Organism: *Rhadinaphelenchus cocophilus* (Cobb) Goodey; (Aphelenchoididae)

The disease was first described in Trinidad in 1905 (Dean 1979), but the causal agent was not found until Nowell (1919, 1920) showed that "nematode worms" caused the disease. The disease is found in several islands of the Caribbean and in Central and South America, where it causes serious losses (Dean 1979). Nolla and Valiela (1976) state that the disease is possibly the greatest menace to coconut production in the Americas.

Cobb (1919) first described the nematode which causes red ring of coconuts as *Aphelenchus cocophilus*. Synonyms have included *Aphelenchoides cocophilus* and *Chitinoaphelenchus cocophilus*. Goodey (1960) renamed the nematode *Rhadinaphelenchus cocophilus* (Cobb) Goodey. *R. cocophilus* is in the family *Aphelenchoididae*. For a complete description of the pathogen Dean (1979) should be consulted.

Economic Importance

Since coconuts do not begin to produce for 5-6 years after planting, and may continue to produce for many decades, the loss of a coconut palm is a serious matter to individual growers. Fenwick (1956) reported losses of 20-25% of the trees in plantings in Trinidad, and 60% losses were described by Nowell (1919). Dean (1979) summarized loss data from the Americas and reported losses from 1 to 80% in 20 references from different countries. He noted that it often is difficult to determine whether a palm has died due to *R. cocophilus* or to insect damage. Considering the extensive literature on the red ring disease, and the strong adjectives used to describe its importance, it is puzzling that relatively little data are found on its economic importance.

Symptoms

The most characteristic and striking symptom of the disease is the orange-red or reddish-brown ring (in cross section) found 2.5 cm or more into the stem. The position, size, and color of the ring may vary somewhat. Detailed descriptions of symptoms are given by Corbett (1959), Fenwick (1969), and Dean (1979). In young infected palms a yellowing of the lowest leaves from the tip backwards is common. As the disease progresses the leaves turn brown, wilt, and eventually die. The entire plant may die 3-4 months after the first symptoms are noted. A rot of the terminal bud is common. Identification of *R. cocophilus* from diseased tissues is necessary for positive identification, although, according to Corbett (1959), the red ring does not appear without *R. cocophilus* being present.

Host Plants

Coconut (*Cocos nucifera*) and oil palm (*Elaeis guineensis*) are the only economically important hosts infected in nature, but inoculation experiments have shown that other wild and cultivated palms are also susceptible (Blair 1969, Dean 1979).

Pathogen Cycle

R. cocophilus can complete its life cycle within 9-10 days which is

considered a short period for a plant parasitic nematode. The eggs, larvae, and mature forms of R. cocophilus can serve as inoculum. Mature nematodes have been found in the coconut weevil (Rhynchophorus palmarum), various tissues of the coconut such as stem, petiole, and roots, and soil in, on, and adjacent to infected coconuts. Sources of inoculum are infected palms. Large numbers of the larvae of R. cocophilus are found in the discolored stem and petiole tissues, whereas adults are normally present only on the edge of the discolored ring. Some nematodes are also found in diseased root tissues (Fenwick 1968). The coconut weevil (R. palmarum) appears to be the major means by which nematodes are disseminated from palm to palm. However, Fenwick (1968) found that R. cocophilus can penetrate all types of unwounded coconut tissue, so other means of dissemination of the pathogen probably occur.

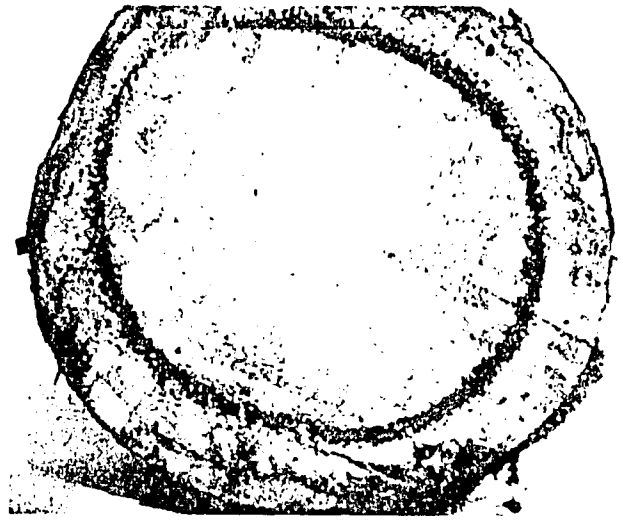


Fig. 52. Vascular discoloration caused by Radinaphelenchus cocophilus, causal agent of red ring of coconuts. (Courtesy of J. E. Edmunds).

Some evidence exists that infection may occur through the roots. When the nematode is found in roots, healthy palms adjacent to diseased palms later become infected (Fenwick 1960, 1968). Furthermore, when pieces of infected tissue are deposited on the ground near healthy palms, many healthy palms later become infected (Fenwick 1968). However, Blair (1969) doubts that roots are an important site of primary infestation, since R. cocophilus cannot survive in the soil more than 2-3 days.

All forms of the nematode are found in the discolored tissue of the crown (Blair 1964). The coconut weevil (R. palmarum) lays its eggs in the tissue of the crown. After hatching, the larvae of R. palmarum feed on the infected tissue and later spin cocoons in which to pupate in the infected tissue. Nematodes can survive pupation in the insect. R. cocophilus has been found in the intestinal tract, mouth parts, body surface, and feces of R. palmarum (Fenwick 1969, Hagley 1962, 1963). About 90% of the weevils in an infested coconut palm plantation have been reported to carry R. cocophilus (Blair 1969, Hagley 1963). In addition to being a vector, R. palmarum may carry pieces of infected tissue from coconut to coconut and distribute the nematode in this manner. Nematodes are reported to live 10 days in the digestive tract of the weevil and 2-6 days on the body surface or on bits of infested tissue (Hagley 1963). Larvae of R. palmarum prefer feeding on diseased tissue and ingest large quantities of nematodes. Maggenti (1981) suggests that an individual larval insect can consume 2.5×10^6 nematode larvae during its development, and that the nematode persists in the weevil throughout its metamorphosis to the adult insect. When R. palmarum oviposits its eggs, the nematode is also injected into the tissue of the palm.

In addition to R. palmarum, many other insects have occasionally been implicated in the dissemination of R. cocophilus (Dean 1979). More than 300

insects are reported associated with coconut palms, and many animals such as rats, birds, bats, etc., can occasionally transfer nematodes or infested material from one palm to another. Dean (1979) lists other possible factors associated with transmission. Nematodes probably are occasionally transmitted by agricultural implements, by soil movement, coconut seedlings, in irrigation or splashing water, and by man.

After penetration and infection, nematodes migrate to the vascular system where they multiply rapidly. The life-cycle can be completed in 9-10 days. R. cocophilus is found both intracellularly and intercellulary. In the discolored ring, over 11,000 nematodes per gram of tissue have been recovered (Blair and Darling 1968).

Control

R. cocophilus is a most difficult organism to control. Dean (1979) points out that Nowell in his early papers (1918 and 1919) recommended isolation and destruction of diseased palms and trapping of the insect transmitter, and that over 60 years later those are still the best recommendations which can be given to growers.

Since the disease is not present in Africa or Asia, great care should be taken to prevent its introduction. By using nematode-free planting material local spread can also often be prevented. A major recommendation to growers is inspection and chemical elimination of diseased palms. If palms are simply cut or a chemical is used which does not kill both the nematode and its insect vector (R. palmarum), the palm may be a source of further infection. Arsenical compounds were most effective in killing both pathogen and vector (Blair 1969 and Majaraj 1964). Treating palms and the soil around palms with a nematicide was not effective in controlling the nematode (Blair 1969).

Cracks in the leaf axil appear to be the most important area for nematode penetration; thus, sealing of these cracks physically or protecting the area with a nematicide were also studied as possible controls. No appropriate material was found to physically seal the cracks (Blair 1969), but granular Nemafos (diethyl O-2-pyrazinyl phosphorothioate) was effective in killing R. cocophilus and also acted as an insect attractant (Blair 1964). Unfortunately, its use could not be recommended because of its high mammalian toxicity. Hagley (1963) attempted control by applying nematicides to the crown of the tree, and by baiting and trapping with attractants. Since the nematode can cause infection via roots, and debris from trees can contain nematodes, it has been recommended that herbicides be used for weeding in coconut plantings rather than mechanical implements which could spread the nematode (Blair and Darling 1968). Biological control has been suggested both for the nematode and the vector, but is not yet sufficiently effective for utilization in a control program (Dean 1979).

Although programs for breeding varieties resistant to R. cocophilus have not been established, differences in resistance or tolerance in coconut varieties to R. cocophilus do exist, and a long-term breeding program might be most productive (Dean 1979).

For effective control of R. cocophilus an integrated program using cultural, chemical, biological, and host resistance methods should be designed for each location. Every effort should be made to prevent the introduction of R. cocophilus to other tropical areas.

References

Blair, G. P. 1964. Red-ring disease of the coconut palm. J. Agric. Soc. Trinidad and Tobago 64:31-49.

Blair, G. P. 1969. The problem of control of red ring disease. pp. 99-108. In Peachy, J. E. ed. Nematodes of tropical crops. Tech. Commun. No. 40 Commonw. Bur. Helminthol., St. Albans. 355 pp.

Blair, G. P., and H. M. Darling. 1968. Red ring of the coconut palm, inoculation studies and histopathology. Nematologica 14:395-403.

Cobb, N. A. 1919. A newly discovered nematode (Aphelenchus cocophilus n. sp.) connected with a serious disease of the coconut palm. West Indian Bull. 17:203-210.

Corbett, M. K. 1959. Diseases of the coconut palm. III. Red ring. Pricipes 3:83-86.

Dean, C. G. 1979. Red ring disease of Cocus nucifera L. caused by Radinaphelenchus cocophilus (Cobb, 1919) Goodey, 1960. An annotated bibliography and review. Commonw. Agric. Bur., Farnham Royal, Bucks. 70 pp.

Fenwick, D. W. 1956. Preliminary investigations into the red ring disease of coconut. J. Agric. Soc. Trinidad and Tobago 56:253-275.

Fenwick, D. W. 1960. Presence of Aphelenchoides cocophilus in roots of Cocos nucifera, the coconut palm. Nature 185 (4708):259-260.

Fenwick, D. W. 1968. Red-ring disease of the coconut palm. pp. 38-48. In Smart, G. C. and V. G. Perry. eds. Tropical Nematology. Univ. Florida Press, Gainesville. 153 pp.

Fenwick, D. W. 1969. Red ring disease of the coconut palm. pp. 89-98. In Peachy, J. E. ed. Nematodes of tropical crops. Tech. Communication No. 40 Commonw. Bur. Helminthol. St. Albans. 355 pp.

Goodey, J. B. 1960. Rhadinaphelenchus cocophilus (Cobb, 1919) n. comb., the nematode associated with red ring disease of coconut. Nematologica 5:98-102.

Hagley, E. A. C. 1962. The palm weevil, Rhychophorus palmarum L., a probable vector of red ring disease of coconuts. Nature (193(4814):499.

Hagley, E. A. C. 1963. The role of the palm weevil, Rhychophorus palmarum, as a vector of red ring disease of coconuts. I. Results of preliminary investigations. J. Econ. Entomol. 56:375-380.

Maggenti, A. 1981. General Nematology. Springer-Verlag, New York. 372 pp.

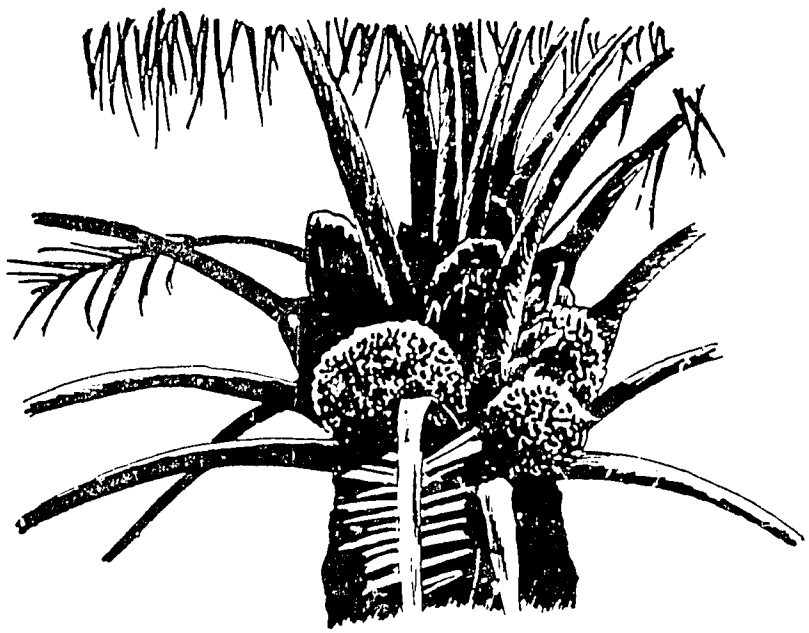
Majaraj, S. 1964. The use of arsenic on coconut trees suffering from red ring disease. J. Agric. Sc. Trinidad and Tobago. 64:59-66.

Nolla, J. A. B., and M V. Fernandez Valiela. 1976. Contributions to the history of plant pathology in South America, Central America, and Mexico. Annu. Rev. Phytopathol. 14:11-29.

Nowell, W. 1919. The red ring or root disease of coconut palms. West Indian Bull. 17:189-202.

Nowell, W. 1920. The red ring disease of coconut palms, infection experiments. West Indian Bull. 18:74-76.

OIL PALM



The oil palm (Elaeis guineensis) can produce more oil per unit area than any other plant. It is of major importance to the economies of Malaysia, Indonesia, and several West African countries, and is increasingly important in tropical Latin America. World production of oil in 1981 was 5.3 million tons (FAO 1981).

The oil palm originated in the rain forests of West Africa, and today is cultivated throughout the tropics. A large tree (up to 30 m tall), it produces numerous small fruits in large bunches in the leaf axils. These bunches may weigh up to 90 kg. Oil palms are classified either based on fruit structure or on the color of the exocarp, which may be red or black (Purseglove 1972). Since oil palms are monecious and cross-pollinated, they are highly heterozygous. They are propagated by seeds, but treatment of seeds by removal of the pulp, drying, and subsequent heat treatment is usually needed for good germination. They are primarily cultivated in lowland, humid tropics (10°N-S) in areas with high rainfall. Palms may produce for 50-60 years.

Two kinds of oil, palm oil and palm kernel oil, are obtained. Palm oil is obtained from the mesocarp (fleshy covering of the seed) and palm kernel oil from the kernel or endosperm. The oil is important to the nutrition of millions in Africa who use it for cooking. Palm oil and palm kernel oil are also used in margarine and soap production. Palm wine is made by tapping the terminal bud or male flower for sap. The leaves are used in West Africa for thatching, fencing, and mulching.

Increasing oil production has been a major objective of breeding programs. Oil palm improvement has been centered primarily in Cameroon, Nigeria, Ivory Coast, Zaire, and Malaysia. In recent decades the center of oil palm production has shifted from West Africa to Asia. Oil palm plantations are also becoming more important in Latin America.

A large number of diseases attack both seedlings and adult palms. Diseases appear to have become more important in the last few decades with increased plantings of large acreages. In Southeast Asia the most important pathogen is Ganoderma spp. in old and replanted areas causing basal stem rot. In Africa Ceratocystis paradoxa (dry basal rot) and Fusarium oxysporum f. sp. elaeidis, causing vascular wilt, have been especially serious. Sudden wilt (marchitez sorpresiva in Spanish) caused great losses in Colombia and has been reported in other South American and Caribbean countries. A protozoan flagellate of the Trypanosomatidae, probably Phytomonas staheli, has consistently

been found associated with the disease, but proof is lacking that it is the causal agent (Van Slobbe et al. 1978, McCoy and Martinez-Lopez 1982). Cercospora elaeidis causes an important nursery leaf spot in Africa, but has not been reported in Asia or the Americas.

Fig. 53. Oil palm plantation in Ecuador.

Selected References on Oil Palm Diseases

Aderungboye, F. O. 1977. Diseases of the oil palm. Pest Articles News Sum. 23:305-326.

Corley, R. H. V., J. J. Hardon, and B. J. Wood. eds. 1976. Oil Palm Research. Elsevier, Amsterdam. 532 pp.

Hartley, C. W. S. 1977. The Oil Palm. Longman, London. 806 pp.

Surre, C., and R. Ziller. 1963. Le Palmier a Huile. G.-P. Maisonneuve and Larose, Paris. 243 pp.

Turner, P. D., and R. A. Gillbanks. 1974. Oil Palm Cultivation and Management. Yau Seng Press, Kuala Lumpur. 672 pp.

Turner, P. D. 1981. Oil Palm Diseases and Disorders. Oxford Univ. Press, Oxford. 280 pp.

Literature Cited

Food Agriculture Organization. 1981. FAO Production Yearbook. Vol. 35. FAO, Rome. 306 pp.

McCoy, R. E., and G. Martinez-Lopez. 1982. Phytomonas staheli associated with coconut and oil palm diseases in Colombia. Plant Dis. 66:675-677.

Purseglove, J. W. 1972. Tropical Crops, Monocotyledons. Wiley, New York. 607 pp. 2 Vol.

Van Slobbe, W. G., M. V. Parthasarathy, and J. A. J. Hesen. 1978. Hartrot or fatal wilt of palms II. Oil palm (Elaeis guineensis) and other palms. Principes 22:15-25.

SESAME

Sesame (Sesamum indicum) is an erect, annual plant, about 1-2 m high whose seeds produce a high quality cooking oil. The crop is grown in the tropics as well as in warm temperate regions (40 N-S latitudes). Three to four months are needed for it to reach maturity at temperatures ranging from 21 to 30°C; at temperatures lower than 18°C the growing period is much longer, up to 6 to 8 months. Sesame is thought to have originated in Northeast Africa (Ethiopia), or in the Middle East (Afghanistan/Iran). It has been grown in India and China perhaps as early as 2000 B.C. World production in 1981 was 1.95 million tons (FAO 1981), and major producers of sesame are India, China, Burma, Sudan, and Mexico.

To obtain good yields of sesame, up to 800 kg/ha, at least 500 mm rainfall are required during the growing season. Heavy rainfall and high humidity are detrimental, especially during flowering, and a dry period for ripening is required. The crop prefers light, permeable soils. In desert areas where irrigation is used, yields of 2000 kg/ha can be obtained. Sesame is grown from seed in rows, often in raised beds. Many cultivars are found in the countries where it is important. Shattering is a problem, and breeders are trying to develop non-shattering cultivars.

Important diseases of sesame (Weiss 1971) are leaf spots (Pseudomonas syringae pv. sesami, Alternaria sesami, Cercospora spp.), wilt (Fusarium oxysporum f. sp. sesami), and charcoal rot (Macrophomina phaseolina). The most serious virus disease is the phyllody disease which results in sterility. Phyllody is important in India and Burma.

Selected References on Sesame Diseases

Cook, A. A. 1981. Diseases of Tropical and Subtropical Field, Fiber and Oil Plants. MacMillan, New York. 450 pp.

El Bardi, T. A. 1972. Sesame. Trop. Abstr. 27:153-160.

Food Agriculture Organization. 1981. Sesame:Status and Improvement. FAO, Rome. 198 pp.

U.S. Department of Agriculture. 1958. Sesame production. Farmer's Bull. No. 2119. Washington, DC. 12 pp.

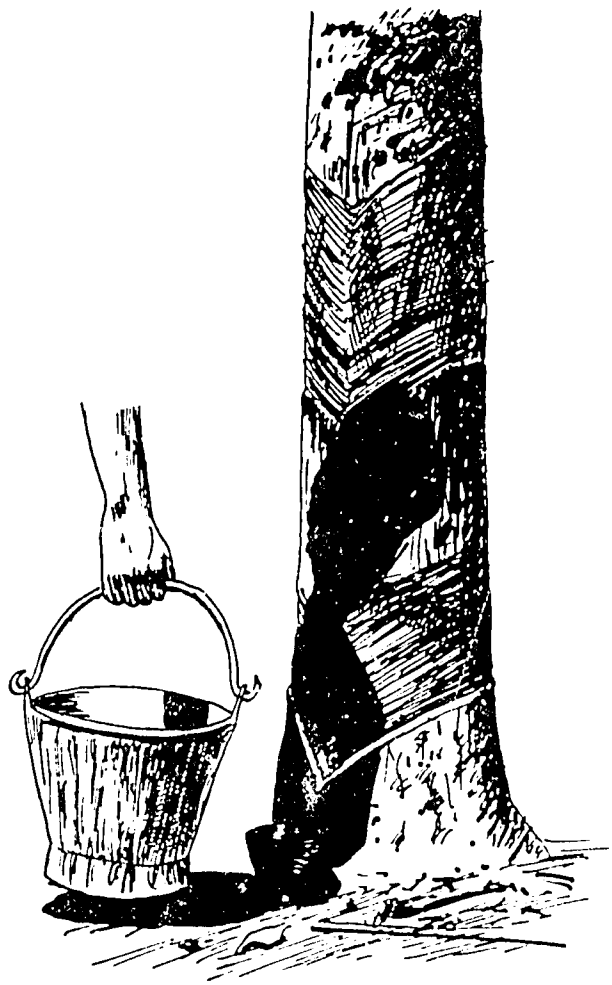
Weiss, E. A. 1971. Castor, sesame and safflower. Leonard Hill, London. 901 pp.

Literature Cited

Food Agriculture Organization. 1981. FAO Production Yearbook. Vol. 35. FAO, Rome. 306 pp.

Rubber

Almost all of the world's rubber is produced from *Hevea brasiliensis*. Other species of *Hevea*, and plants of *Castillia elastica*, *Ficus elastica*, *Manihot glaziovii*, *Funtumia elastica*, plus many others, have also been used to produce rubber. Although *H. brasiliensis* originated in the Amazon basin, over 90% of *Hevea* rubber is now grown in Asia, with Malaysia being the largest producer. Africa produces a small amount, and Brazil, where *H. brasiliensis* originated, produces only around 1% of world production. In 1981 world rubber production was 3.8 million tons (FAO 1981).



Like many members of the family Euphorbiaceae, *Hevea* produces a thick white latex beneath the bark. Latex is about 30% rubber (polyisoprene). The trees are large (sometimes exceeding 40 m in height in the wild), natural components of Amazonian tropical rain forests. They grow best under the hot, humid, high rainfall environments found within 15° of the equator near sea-level. In the Amazon forest *Hevea* grows as widely scattered individuals, with only a few trees per hectare (Imle 1978).

The process of extracting the rubber, called tapping, involves cutting into the bark to sever the latex ducts. As the latex flows from the wounds on the "tapping panel", as the area cut is called, it is collected. Unless great care is taken, tapping can be damaging to a tree and even kill it. *Hevea* rubber was destructively tapped in scattered areas throughout the Amazon jungle in the 19th century. Ridley, in 1889, developed a nondestructive form of tapping which not only allowed a tree to be tapped for decades, but actually stimulated the production of more latex (Purseglove 1968). Latex is diluted with water prior to processing, and the rubber coagulated by the addition of acetic and/or formic acid. The coagulated rubber is then rolled to form sheets and cured in smoke houses.

Until 1900, practically all of the world's rubber came from Brazil and Peru. Increased rubber usage in the late 1870's stimulated H. A. Wickam, an English botanist, to collect seeds in Brazil; these were shipped to the Royal Botanical Gardens at Kew, England, and the germinated and growing seedlings were then sent to Sri Lanka and Singapore. These seedlings were the source of clones for most subsequent Asian rubber plantings.

Fig. 54. Rubber tapping.

Thousands of different products are made from rubber, and it is now essential in an industralized world. Synthetic rubber was industralized in the United States on a large scale during World War II when Asian supplies were cut off. Nevertheless, synthetic rubber cannot replace Hevea rubber for some uses, and demand for natural rubber remains high.

Seeds are planted directly or in nurseries prior to transplanting. Hevea is commonly bud grafted in Asia to produce a plant with a high-yielding tapping panel on a vigorous rootstock.

Diseases are often the limiting factor in rubber production, not only in the Americas, but also in Asia. South American leaf blight (Microcyclus ulei) is the most destructive disease of rubber in the American tropics and has made the development of plantation culture in the Western hemisphere almost impossible. A number of root rots of the trunks and branches are important in rubber. White root disease (Rigidoporus lignosus) is serious in Asia. Armillariella mellea is damaging in West Africa. Red root rot (Ganoderma philippi) is also a serious disease in Indonesia and Sumatra. Black stripe and leaf blight (Phytophthora palmivora), pink disease (Corticium salmonicolor), and moldy rot (Ceratocystis fimbriata) are other important rubber diseases. Powdery mildew of rubber (Oidium heveae) is common in Africa and Asia and can cause considerable damage. Great progress has been made in rubber improvement in recent decades, especially at the Rubber Research Institute of Malaysia. Extremely high yielding cultivars have been developed.

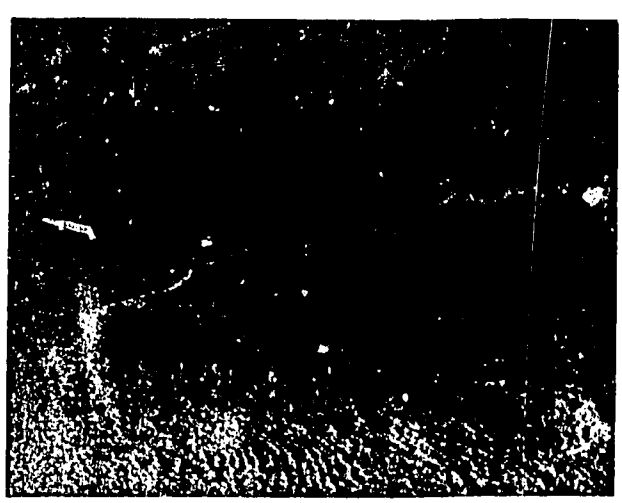


Fig. 55. Rubber and oil palm plantings in Malaysia.

Selected References on Rubber Diseases

Imle, E. P. 1978. Hevea rubber-past and future. Econ. Bot. 32:264-277.
Polhamus, L. G. 1962. Rubber: Botany, Cultivation and Utilization. Interscience, New York. 449 pp.
Rao, B. S. 1975. Maladies of Hevea in Malaysia. Rubber Res. Inst. of Malaysia, Kuala Lumpur. 108 pp.
Wastie, R. L. 1975. Disease of rubber and their control. Pest Articles News Sum. 21:268-288.

Literature Cited

Food Agriculture Organization. 1981. FAO Production Yearbook. Vol. 35. FAO, Rome. 306 pp.
Purseglove, J. W. 1968. Tropical Crops, Dicotyledons. Wiley, New York. 719 pp. 2 Vol.

SOUTH AMERICAN LEAF BLIGHT OF RUBBER

Disease Names: South American leaf blight, South American leaf disease.

Causal Organism: *Microcyclus ulei* (P. Henn.) Arx.; (Dothidaceae)
Synonym - *Dothidella ulei* P. Henn.

The rubber tree (*Hevea brasiliensis*) is a native of South American tropical rainforests. *Microcyclus ulei*, which causes South American leaf blight of rubber, is the most destructive disease of rubber in the American tropics and has made the development of plantation culture of rubber in the Americas almost impossible.

Before 1900 practically all of the world's rubber came from destructive tapping of scattered wild trees in Brazilian and Peruvian forests. In 1876 H. A. Wickam, an English botanist, collected seed of *H. brasiliensis* near the Tapajoz river in Brazil. The seeds were shipped to the Royal Botanical Gardens at Kew, England, germinated, and growing seedlings were shipped to Sri Lanka and Singapore (Holliday 1970). These seedlings were the source of clones for most of the subsequent Asian rubber plantings. By germinating the seeds in England, Wickam probably prevented the introduction of *M. ulei* into Asia. However, since relatively few seeds were taken from Brazil, the genetic diversity of Asian rubber plantings is extremely limited.

Most of the rubber in Brazil was tapped from trees growing in their natural habitat in the jungle, and *M. ulei* was hardly considered a problem. Hennings first described the fungus in 1904, but the disease did not become a serious problem until attempts were made, early in the 20th Century, to grow rubber in large plantations in South America. Plantations established in Trinidad and the British and Dutch Guianas (Guyana and Surinam) were a total failure because of *M. ulei* (Rands 1945, Holliday 1970). The Ford Motor Co. planted 3200 ha in Fordlandia, Brazil, in an early attempt to exploit the potential of the Amazon Basin. By 1933 the disease had destroyed a quarter of the Ford rubber planting. Similar failures occurred in Panama, Costa Rica, and Colombia (Holliday 1970). Hilton (1955) has reviewed the literature on the damage by *M. ulei* in tropical America.

Stahel (1917) made an intensive investigation of the disease in Dutch Guiana (Surinam), but was unable to find practical methods for controlling it (cited by Rands 1945). In 1940, just before World War II, a hemisphere-wide project to increase rubber production was started involving a cooperative effort of the United States, most tropical American countries, and several commercial concerns. Control of South American leaf blight was a principal, though unrealized, objective of this cooperative effort. At present the disease is confined to South and Central America and Trinidad from 18°N in Mexico to 24°S in Brazil (Holliday 1970).

Hennings (1904) first reported the fungus on leaves of *Hevea brasiliensis* collected in Brazil, and named the perfect stage *Dothidella ulei*. The pathogen has since been reclassified and is now known as *Microcyclus ulei*, an ascomycete in the Dothidaceae. Langford (1945) was the first to isolate *M. ulei* and give proof of its pathogenicity. The imperfect stage (conidial) is a Hyphomycete *Fusicladium macrosporum* Kuypert. Holliday (1970, 1977, 1980) described the conidia as hyaline changing to greyish, one to two-celled, ellipsoidal, acrogenous, 23-63 X 5-10 µm, with the proximal cell broader with a thickened truncate end, and a distinct twist. Ascospores are hyaline, two-celled, ellipsoidal, and 12-20 X 2-5 µm. Conidia are produced before the perithecial stage is formed.

Importance

The failure to control *M. ulei* has prevented the establishment of *Hevea* rubber plantings on any extensive scale in the tropics of America. In addition, rubber-growing countries in Asia have spent considerable sums to develop eradication procedures should the pathogen be introduced there.

Symptoms

The young leaves are most seriously affected, although older leaves, stems, twigs, and other plant parts are also attacked by the fungus. The lesions are first translucent olive-green changing to light gray on young leaves. Young leaves later become black and eventually may drop. The undersurface of the spots becomes grayish-white and velvety from the production of conidia. Light infection results in a shot-hole effect. The perithecia of *M. ulei* are found mainly on the underside of the older leaves. Successive defoliation results in stem necrosis and eventually the tree dies (Petch 1921, Rands 1924, and Holliday 1970, 1980).

Host Plants

M. ulei infects *H. brasiliensis* and eight other wild *Hevea* spp. Holliday (1970), Rands (1945), and Langford (1945) discuss host-range in detail.

Pathogen Cycle

Inoculum consists of conidia and ascospores. Initially sporulation is heavy on infected leaves, but 2 to 3 months later it becomes sparse and eventually no more conidia are produced. Conidia are produced about a week after infection and are wind and water disseminated. According to Holliday (1980), on dry days most conidia are found in the air at mid-morning with less dispersal at other times of the day. Rocha and Vasoncelos (1978) found the most conidia between 7 AM and 2 PM with a peak at 12 PM. Rainfall causes temporary large increases in the number of conidia produced. Perithecia are found on 2-4 month-old leaves, and mature ascospores can be found in perithecia (Langford 1945). Ascospores are produced 8-9 weeks after infection, and the largest number are found in the air near dawn on dry days. Since ascospores do not tolerate direct sun, effective dispersal probably is limited to night and overcast days. Again, rainfall increases ascospore numbers and changes discharge times (Holliday 1980).

Young leaves up to 10 days old are more susceptible to infection than older leaves which become resistant after about 16 days. The appearance of new leaves on rubber trees in "flushes" explains frequent disease escapes and reports of apparent disease resistance which later breaks down. As older trees may have many "flushes" of new foliage, while a young tree may have only one flush of leaves at any given time, young trees often escape infection or are only lightly infected until they are 5-6 years old (Langford 1945).

The optimum temperature for germination and germ tube elongation of both conidia and ascospores is 24-28°C (Langford 1945). Water, in the form of dew or rain for about 8 hours, is necessary for germination, the formation of an appressorium, infection hypha, and penetration. Such conditions are frequent in most tropical areas where rubber is grown (Langford 1945). Stahel (cited by Rands 1924) states that penetration is direct and through the leaf cuticle. Little is known about the incubation or infection processes after penetration. Lesions appear 5-8 days after penetration.

Wherever rubber is grown in the tropics the temperature is probably ideal for the development of the fungus. Moisture is the critical factor. Enough

moisture in the form of fog, rain, or dew must be present for almost 8 hours for infection to take place. Long dry seasons can reduce the amount of infection considerably (Holliday 1970). Langford (1945) postulated that ascospores probably account for a small proportion of infections, but that they might play an important role in airborne dissemination over long distances, or in hybridization of the fungus to produce new races.

Control

South American leaf blight does not occur outside of the Western Hemisphere; therefore, every effort should be made to exclude it from other continents by quarantine. Rubber-growing countries in Asia have set up strict quarantine measures to prevent the introduction of M. ulei (Berg 1970, Rao 1973, Thurston 1973). Malaysia has distributed a well-illustrated color brochure in three languages on the disease to alert rubber growers of the danger from M. ulei. In addition, Malaysia has worked out a comprehensive plan for a campaign of eradication in case the disease is ever introduced (Holliday 1970) and has scientists working in the Americas on resistance to M. ulei. Aerial spraying to defoliate rubber was investigated in order to eradicate M. ulei (Hutchinson 1958). Clones in Asia are highly susceptible to the disease, and for this reason Asian nations are prepared to eradicate the disease at any cost, since environmental conditions there (especially in Malaysia) are considered favorable for the disease. The approach of Malaysia to the dangers of South American leaf blight can serve as a model to other countries faced with similar problems. Although it is hindsight, it is interesting to speculate on what might have been the history of coffee rust in the Americas if similar preparations had been made.

One of the best means of control for M. ulei is budding or grafting clones with high foliage resistance on root stocks of highly susceptible, but high-yielding clones. In order to do this, the highly susceptible clones must be grown in nurseries before budding. It is usually necessary to use chemical control of M. ulei in nurseries. Experiments were made over a period of many years to determine the best fungicides for control in the nursery; initially Bordeaux mixture and various fixed coppers were used, but later the organics such as zineb were found to give better control (Wellman 1955). Crown budding was an old technique first used with rubber in Indonesia to reduce wind damage on certain clones. Later it was used in Asia to protect against powdery mildew (Oidium heveae). Budding is done when seedlings have reached a height of about six feet. The seedlings which will serve as the high latex-yielding trunk have to be sprayed to control M. ulei until the graft is successfully established and the resistant foliage of the top can take over.

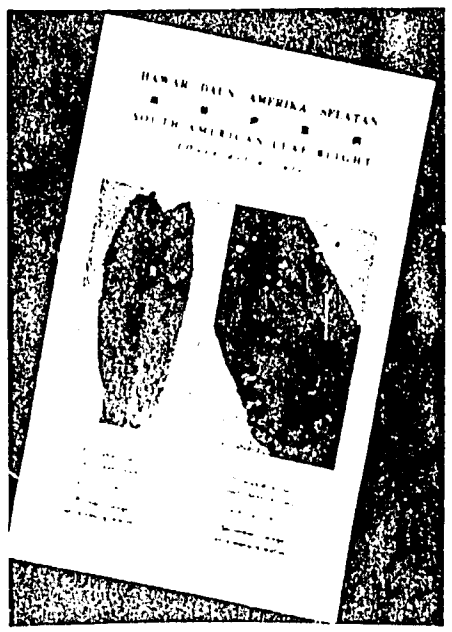


Fig. 56. Bulletin published by the Rubber Research Institute of Malaysia in 1966 to alert growers to the danger of South American leaf blight.

Double budded trees consist of three parts: 1) an unselected seedling root stock, 2) a high-yielding stem budded onto the root stock, and 3) a vigorous, highly resistant crown (Rands 1945).

South American leaf blight illustrates the dangers of growing a formerly wild plant in a monoculture. In the native habitat of Hevea in the Amazon jungle there are only a few Hevea trees per hectare (Imle 1978). Many of the native rubber trees are low-yielding and somewhat tolerant of the disease; thus, their mortality is not as high as those of selected high-yielding trees grown as a monoculture. In addition, each tree is screened from the other rubber trees by the foliage of trees of other genera, which serve as barriers to windborne spores. The disease did little damage during the time when rubber was collected from the jungle (Langford 1945). Higher yielding Hevea trees grown in large plantations were more susceptible and had more exposure to attacks by M. ulei. The disease thus became a serious problem and destroyed thousands of acres of rubber in Latin America. Breeding for disease resistance has been a difficult task because of the linkage of susceptibility to high yield and, conversely, resistance to low yield.

A breeding program has been under way for many years and considerable Hevea material with resistance has been found. Attempts have been made to combine high yield and vigor with resistance to M. ulei and other diseases and pests. One of the best sources of resistance has been clones of H. benthamiana. When crossed with H. brasiliensis it has given clones of good resistance not only to blight, but to other diseases as well. Langford (1945), Ochse et al. (1961), and Holliday (1970) discuss rubber-breeding programs, and Goncalves (1968) and Imle (1978) give the current status of breeding for resistance to M. ulei. Physiologic races of M. ulei complicate breeding. There is considerable interest in finding horizontal or multiple-gene resistance to M. ulei as true immunity may never be found.

Literature Cited

Berg, G. H. 1970. Plant quarantine measures against South American leaf blight. Food Agric. Organ. Plant Protec. Bull. 18:1-7.

Goncalves, J. R. C. 1968. The resistance of Fx and IAN rubber clones to leaf disease in Brazil. Trop. Agric. (Trinidad) 45:331-336.

Hennings, P. 1904. Ueber die auf Hevea - arten bisher beobacheten parasitischen pilze. Notizbl. Bot. Gart. Mus. Berlin 4:133-138.

Hilton, R. N. 1955. South American leaf blight. A review of the literature relating to its depredations in South America, its threat to the Far East and the methods available for its control. J. Rubber Res. Inst. Malaya 14:287-337.

Holliday, P. 1970. South American leaf blight (Microcyclus ulei) of Hevea brasiliensis. Phytopathol. Paper No. 12. Commonw. Mycol. Inst., Kew Surrey. 31 pp.

Holliday, P. 1977. Microcyclus ulei. In Diseases, Pests and Weeds in Tropical Crops. Kranz, J., H. Schmutterer, and W. Koch. eds. Verlag Paul Parey, Berlin. 666 pp.

Holliday, P. 1980. Fungus Diseases of Tropical Crops. Cambridge Univ. Press, Cambridge. 607 pp.

Hutchinson, F. W. 1958. Defoliation of Hevea brasiliensis by aerial spraying. J. Rubber Res. Inst. Malaya 15:241-274.

Imle, E. P. 1978. Hevea rubber-past and future. Econ. Bot. 32:264-277.

Langford, M. H. 1945. South American leaf blight of hevea rubber trees. U.S. Dept. of Agric. Tech. Bull. 882. 31 pp.

Ochse, J. J., M. J. Soule, Jr., M. J. Dijkman, and C. Wehlburg. 1961. Tropical and Subtropical Agriculture Vol. 1-2. MacMillan, New York. 1446 pp.

Petch, T. 1921. The Diseases and Pests of the Rubber Tree. MacMillan, London. 278 pp.

Rands, R. D. 1924. South American leaf disease of para rubber. U.S. Dept. Agric., Dept. Bull. 1286. 18 pp.

Rands, R. D. 1945. Hevea rubber culture in Latin America, problems and procedures. pp. 183-199. In Plants and Plant Science in Latin America. F. Verdoon. ed. Chronica Botanica, Waltham, MA. 381 pp.

Rao, B. S. 1973. Potential threat of South American leaf blight to the plantation rubber industry in Southeast Asia and the Pacific region. Food Agric. Organ. Plant Protec. Bull. 21:107-113.

Rocha, H. M., and A. P. Vasconcelos. 1978. Epidemiology of the South American leaf blight of rubber in the region of Itubera, Bahia, Brazil. Turrialba 28:325-329.

Stahel, G. 1917. De Zuid--Amerikansche Hevea bladziekte veroorzaakt door Melanopsammopsis ulei nov. gen. (Dothidella ulei P. Hennings). Suriname Dept. Landb. Bull. 34. 111 pp.

Thurston, H. D. 1973. Threatening plant diseases. Annu. Rev. Phytopathol. 11:27-52.

Weilman, F. L. 1955. Control of rubber disease by chemicals. pp. 31-42. In Pesticides in Tropical Agriculture. No. 13. Am. Chem. Soc., Adv. Chem. Ser. Washington, DC.

Fiber Crops

Fiber crops produce fibers that can be used to manufacture cloth, cordage, and a host of other articles. Cotton (*Gossypium* sp.) is the most important worldwide. With the exception of flax, most fiber crops are grown primarily in tropical or subtropical areas. Major tropical fiber plants are jute (*Corchorus* spp.), hemp (*Cannabis sativa*), kenaf (*Hibiscus cannabinus*), ramie (*Bachmeria nivea*), kapok (*Ceiba pentandra*), abaca (*Musa textilis*), sisal (*Agave sisalana*), henequen (*Agave fourcroydes*), cabuya (*Furcraea cabuya*), and Sansevieria (*Sansevieria* spp.). Kirkby (1963), Lock (1969), Ochse et al. (1961), Berger (1969), and Purseglove (1968) discuss fiber plants in detail.

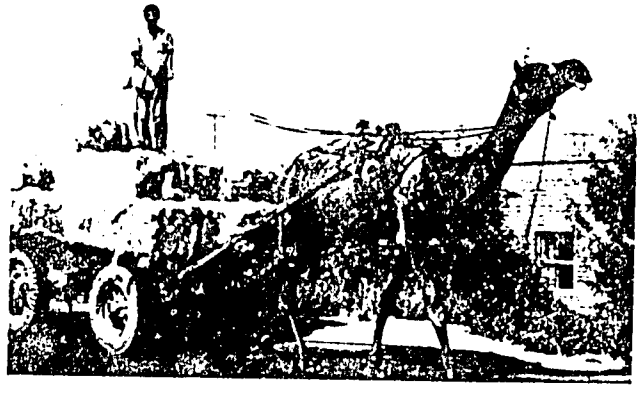


Fig. 57. Camel pulling wagon-load of cotton in Pakistan.

Selected References on Fiber Crops and Their Diseases

Berger, J. 1969. The World's Major Fiber Crops: Their Cultivation and Manuring. Centre d'Etude l'Azote, Zurich. 294 pp.

Cook, A. A. 1981. Diseases of Tropical and Subtropical Field, Fiber, and Oil Plants. MacMillan, New York. 450 pp.

Dempsey, J. M. 1975. Fiber Crops. Univ. Florida Press, Gainesville. 475 pp.

Kirby, R. H. 1963. Vegetable Fibers. Interscience, New York. 464 pp.

Kranz, J., H. Schmutterer, and W. Koch. eds. 1977. Diseases, Pests and Weeds in Tropical Crops. Verlag Paul Parey, Berlin. 666 pp.

Lock, G. W. 1969. Sisal. Longman, New York. 365 pp.

Ochse, J. J., M. T. Soule Jr., M. J. Dijkman, and C. Wehlburg. 1961. Tropical and Subtropical Agriculture. Vol. 1-2. MacMillan, New York. 1446 pp.

Purseglove, J. W. 1968. Tropical Crops: Dicotyledons. Wiley, New York. 719 pp. 2 Vol.

Stover, R. H. 1972. Banana, Plantain and Abaca Diseases. Commonw. Mycol. Inst., Kew, Surrey. 316 pp.

COTTON

Cotton (Gossypium spp.) is grown in most tropical and subtropical countries. It is one of the most ancient crops grown by man, as specimens have been found in Pakistan dated 3000 B.C. and in Peru dated 2400 B.C. (Purseglove 1968). It is the most important fiber plant in the world. World production of lint in 1981 was 15.3 million tons (FAO 1981). The American continents are the largest producers followed by Asia and Africa. The United States is the largest single producer, but cotton is extremely important to the economies of many developing nations.

There are several classifications of cotton species. The four main species are upland cotton (Gossypium hirsutum), American-Egyptian cotton (G. barbadense), and Asiatic cottons (G. arboreum and G. herbaceum). Cotton had multiple sites of origin in America, Africa, and Asia, and controversy on the origin of the many species continues.

The major use of cotton lint is for the production of a variety of fabrics and related products. The seeds are used to produce a high quality edible oil. The cotton seed cake or press cake remaining after oil extraction is used as an animal feed, as it has a high protein content. Cotton is a small shrub or tree which produces a fruit or "boll" containing seeds bearing the cotton lint. It is grown under a wide variety of soil and climatic conditions, both rainfed and with irrigation.

Among the most important cotton diseases are two wilts (caused by Verticillium dahliae and Fusarium oxysporum f. sp. vasinfectum), bacterial blight caused by Xanthomonas campestris pv. malvacearum, rust (Puccinia cacabata), and leaf curl disease caused by a white fly transmitted virus. Boll rots are caused by many species of fungi and bacteria. For example, boll rots caused by the fungus Nematospora sp. and the bacterium Erwinia aroideae are important in many tropical areas. Most boll rot is found after boll injury by insects.

Selected References on Cotton Diseases

Ebbels, D. L. 1976. Diseases of upland cotton in Africa. Rev. Plant Pathol. 55:747-763.

Lagiere, R. 1966. Le Cotonnier. G.-P. Maisonneuve and Larose, Paris. 306 pp.

Prentice, A. N. 1972. Cotton. Longman, London. 282 pp.

Ripper, W. E., and L. George. 1965. Cotton Pests of the Sudan. Blackwell Sci. Publ., Oxford. 345 pp.

Tarr, S. A. J. 1964. Virus diseases of cotton. Misc. Publ. 18. Commonw. Mycol. Inst., Kew, Surrey. 26 pp.

Watkins, G. M. ed. 1981. Compendium of Cotton Diseases. Amer. Phytopathol. Soc., St. Paul. 87 pp.

Literature Cited

Food Agriculture Organization. 1981. FAO Production Yearbook. Vol. 35. FAO, Rome. 306 pp.

Purseglove, J. W. 1968. Tropical Crops, Dicotyledons. Wiley, New York. 719 pp. 2 Vol.

Forages

Extensive areas of natural grasslands are found in tropical and sub-tropical areas. For example, about 30 percent of the native vegetation in Latin America is made up of grasslands. Large tropical land areas are sub-marginal for crop production, but as pastures they can be utilized for animal production.

There are about 10,000 species of grass known to exist in the world, and 40 of these account for more than 98 percent of those used in sown pastures. Many others are important in "natural grasslands". Just to list all of the important tropical and sub-tropical grasses is a lengthy task. Most tropical grasses originated in Africa, some in the Americas, and a few in Asia. This may be due to grasses evolving with the numerous wild grazing animals in Africa, which are less common in America and Asia.

Legumes are important in pastures and as fodder, especially in improving the protein quality of pastures. Legumes can also fix nitrogen through symbiosis with Rhizobium bacteria, and thus add nitrogen to the soil. Immense areas of acid infertile soils (oxisols and ultisols) occur in South America (the Llanos of Colombia and Venezuela and the Campo Cerrado of Brazil) on which cattle productivity is very low due to severe malnutrition and resulting diseases. Low quality pastures and mineral imbalances are the major reasons for these animal problems. These areas constitute 40-50% of the total land resource in tropical America. The Tropical Pasture Program of CIAT (International Center for Tropical Agriculture) in Colombia is attempting to develop forages adapted for production on these infertile soils. In 1981 their germplasm collection of grasses and legumes included over 7,000 accessions.

Information on diseases of tropical grasses and forages is scarce and not found in an easily accessible form. General references on these crops and their diseases follow.

Fig. 58. Dr. J. J. Castaño in elephant grass plot.

Selected References on Tropical Forages and Their Diseases

Bogodan, A. V. 1977. Tropical Pasture and Fodder Plants. Longman, London. 475 pp.

Couch, H. B. 1973. Diseases of Turfgrass. Krieger, Huntington, NY. 348 pp.

Crowder, L. V., and H. R. Chheda. 1982. Tropical Grassland Husbandry. Longman, London. 562 pp.

Endo, R. M. 1972. The turfgrass community as an environment for the development of facultative fungal parasites. pp. 170-202. In Younger, V. B. and C. M. McKell. eds. The Biology and Utilization of Grasses. Academic Press, New York.

Humphreys, L. R. 1978. Tropical Pastures and Fodder Crops. Longman, London. 135 pp.

National Academy Sciences. 1979. Tropical Legumes: Resources for the Future. NAS, Washington, DC. 331 pp.

Sampson, K., and J. H. Western. 1954. Diseases of British Grasses and Herbage Legumes. University Press, Cambridge. 118 pp.

Skerman, P. J. 1977. Tropical Forage Legumes. Food Agric. Organ., Rome. 609 pp.

Smiley, R. W. 1983. Compendium of Turfgrass Diseases. Am. Phytopathol. Soc., St. Paul, MN. 104 pp.

Sprague, R. 1950. Diseases of Cereals and Grasses in North America. Ronald Press, New York. 538 pp.

Spices

Fig. 59. Pepper vines (Piper nigrum) in the Philippines.

Spices are somewhat difficult to define as they are classified by use and not botanically. Webster's dictionary (1968) defines spices as:

> "Any of several vegetable substances, as clove, cinnamon, nutmeg, pepper, etc. used to season food: spices are usually dried for use and have distinctive flavors and aromas".

Spices may be the leaves, bark, roots, seed, or fruit of a wide variety of plants. Although most spices are grown in tropical or subtropical areas, they are used throughout the world. Pepper, vanilla, cloves, nutmeg, mace, oregano, cinnamon, cassia, ginger, and allspice are important, but dozens of other spices enter world commerce. The United State's annual imports of spices are valued at over 100 million dollars. Black pepper (Piper nigrum) is the world's most important spice in terms of usage and value in trade. A worldwide limiting factor in black pepper production is foot rot caused by Phytophthora palmivora (Holliday and Mowat 1963).

World exploration was greatly stimulated by the search for spices, and it was the profitable spice trade that interested the Europeans in sea routes to the Asian tropics where many spices originated. This fascinating story is given by Parry (1969), Rosengarten (1973), and Purseglove et al. (1981).

Selected References on Spice Diseases

Chattopadhyay, S. B. 1967. Diseases of Plants Yielding Drugs, Dyes, and Spices. Indian Council of Agric. Res., New Delhi. 100 pp.

Cook, A. A. 1978. Diseases of Tropical and Subtropical Vegetables and Other Plants. Hafner, New York. 381 pp.

Holliday, P., and W. P. Mowat. 1963. Foot rot of Piper nigrum L. (Phytophthora palmivora). Phytopath. Paper 5. Commonw. Mycol. Inst. Kew, Surrey. 62 pp.

Purseglove, J. W., E. G. Brown, C. L. Green, and S. R. J. Robbins. 1981. Spices. Longman, London. 813 pp. 2 Vol.

Weber, G. 1973. Bacterial and Fungal Diseases of Plants in the Tropics. Univ. of Florida Press, Gainesville. 673 pp.

Literature Cited

Guralnik, D. B., and J. H. Friend. eds. 1968. Webster's New World Dictionary of the American Language. World Publ. Co., Cleveland, OH. 1724 pp.

Parry, J. W. 1969. Spices. Vol. 1. Chemical Publ. Co., New York. 235 pp.

Rosengarten Jr., F. 1973. The Book of Spices. Pyramid, New York. 475 pp.

Tobacco

Tobacco (Nicotiana tabacum) is an important crop in many tropical developing countries, and much of the crop is exported, thus earning valuable income for farmers and governments. In the United States tobacco production is worth approximately three billion dollars annually (U.S.D.A. 1982). World production of tobacco leaves in 1981 was 5.3 million tons (FAO 1981). Despite well known health hazards, tobacco, considering the nature of man, will continue to be important in any foreseeable future.

Akehurst (1981) reported October 11, 1492, as the date when Europeans first recorded the use of tobacco among the Arawak Indians of the Caribbean. Although its use was widespread among American Indians, it wasn't until the early 1600's that it was grown as a commercial crop in North America and exported to Europe. At present, it is the most widely grown non-food plant in the world (Akehurst 1981).

There are about 50 species of Nicotiana, but N. tabacum is the only species of economic value. N. rustica is cultivated but is of minor importance. Tobacco is significant in the economies of many tropical countries such as Brazil, China, Cuba, India, Indonesia, Philippines, and Zimbabwe. It is grown from 60°N to 45°S latitudes in a wide range of climates (Akehurst 1981). Until recently, most attempts at genetic improvement of tobacco have been directed towards obtaining disease resistance.

The tobacco leaf is the valuable part of the plant; therefore, any disease which causes even slight damage to the leaf may considerably reduce the value of the crop. Cramer (1967) has estimated worldwide losses to tobacco diseases at about 18% of the annual value of the crop. Worldwide, the root-knot nematodes (Meloidogyne spp.) are the major disease problem. Acting alone nematodes can cause great losses. However, plants attacked by nematodes are more susceptible to other pathogens. The combined effect of such disease complexes may be disastrous. Other important diseases are those caused by bacteria such as Granville wilt (Pseudomonas solanacearum), and wildfire (Pseudomonas syringae pv. tabaci). Other serious diseases caused by fungi include blue mold (Peronospora hyoscyami f. sp. tabacina), black shank (Phytophthora nicotianae var. nicotianae), brown spot (Alternaria alternata), black root rot (Thielaviopsis basicola), and frog eye (Cercospora nicotianae). A few of the many virus problems of tobacco are tobacco mosaic, cucumber mosaic, vein-banding, and tomato spotted wilt. Non-parasitic leaf spots caused by phytotoxicants, principally ozone, in the air are becoming more prevalent as air pollution increases.

Selected References on Tobacco Diseases

Akehurst, B. C. 1981. Tobacco. Longmans, London. 764 pp.

Gayed, S. K. 1978. Tobacco Diseases. Pub. 1641. Agric. Canada, Ottawa. 56 pp.

Lucas, G. B. 1975. Diseases of Tobacco. Biol. Consulting Associates, Raleigh, NC. 621 pp.

Literature Cited

Cramer, H. H. 1967. Plant Protection and World Crop Production. Farbenfabriken. Bayer A. G., Leverkusen. 524 pp.

Food Agriculture Organization. 1981. FAO Production Yearbook. Vol. 35. FAO, Rome. 306 pp.
United States Agricultural Marketing Service. 1982. U.S. Dept. Agric. Stat. Bull. (685):1-37.

Drug Crops

Drugs include substances used as or in medicines and also narcotic or habit-forming substances. For example, several wild species of yams (Dioscorea sp.) contain steroidal sapogenins, and were grown in tropical America as a source of compounds to synthesize cortisone, sex hormones, and oral birth control drugs (Martin 1970). Today, synthetic production of these compounds has made Dioscorea a less important source. Chinchona (Chinchona spp.) is the tree from whose bark quinine was obtained (Taylor 1945). It was a standard remedy for malaria for over three centuries. Synthetic drugs for malaria have largely replaced quinine, but with the recent development of resistance to synthetic drugs, quinine may again become important. Hundreds of other tropical plants are also used for medicines.

Man has also used many plants and plant products for their narcotic effects. Among the best known are the opium poppy (Papaver somniferum), coca (Erythroxylon coca), and marihuana (Cannabis sativa). (Emboden 1972, Joyce and Curry 1970). Tobacco and the plants used to produce alcohol could be added to the list.

Little information is found on diseases of drug plants except in scattered references.

Selected References on Diseases of Drug Crops

Barloy, J., and J. Pelhate. 1962. Premières observations phytopathologiques relatives aux cultures de chanvre en anjou. Annales Epiphyties 13:117-149.

Chattopadhay, S. B. 1967. Diseases of Plants Yielding Drugs, Dyes, and Spices. Indian Council of Agric. Res., New Delhi. 100 pp.

Cook, A. A. 1978. Diseases of Tropical and Subtropical Vegetables and Other Plants. Hafner, New York. 381 pp.

Schmitt, C. G., and B. Lipscomb. 1975. Pathogens of Selected Members of the Papaveraceae -- an Annotated Bibliography. U.S. Dept. Agric., Agric. Res. Serv., ARS-NE-62. 186 pp.

Literature Cited

Emboden, W. 1972. Narcotic Plants. Studio Vista, London. 168 pp.

Joyce, C. R. B., and S. H. Curry. eds. 1970. The Botany and Chemistry of Cannabis. J. & A. Churchill, London. 217 pp.

Martin, F. W. 1970. The current status of the sapogenin-bearing yams. pp. 99-101. In Plucknett, D. L. ed. Proc. 2nd Int. Symp. Trop. Root and Tuber Crops. Univ. of Hawaii, Honolulu.

Taylor, N. 1945. Cinchona in Java: the Story of Quinine. Greenberg, New York. 87 pp.

General Publications on Tropical Diseases

Cook, A. A. 1981. Diseases of Tropical and Subtropical Field, Fiber and Oil Plants. Macmillan, New York. 450 pp.

Cook, A. A. 1978. Diseases of Tropical and Subtropical Vegetables and Other Plants. Hafner, New York. 381 pp.

Cook, A. A. 1975. Diseases of Tropical Fruits and Nuts. Hafner, New York. 317 pp.

Fröhlich, G., and W. Rodewald. 1970. Pests and Diseases of Tropical Crops and Their Control. Pergamon Press, London. 371 pp.

Graham, K. M. 1971. Plant Diseases of Fiji. Min. of Overseas Development. Overseas Res. Publ. No. 17. H. M. Stationery Office. London. 251 pp.

Hill, D. S., and J. M. Waller. 1982. Pests and Diseases of Tropical Crops. Vol. 1. Principles and Methods of Control. Longman, London. 175 pp.

Hoiliday, P. 1980. Fungus Diseases of Tropical Crops. Cambridge Univ. Press, Cambridge. 607 pp.

Kranz, J., H. Schmutterer, and W. Koch. eds. 1977. Diseases, Pests and Weeds in Tropical Crops. Verlag Paul Parey, Berlin. 666 pp.

O'Conner, B. A. 1969. Exotic Plant Pests and Diseases. South Pacific Commission, Noumea, New Caledonia. 460 pp.

Peachey, J. E. ed. 1969. Nematodes of Tropical Crops. Commmonw. Agric. Bur., Farnham Royal, Bucks. 355 pp.

Roger, L. 1951, 1953, 1954. Phytopathologie des Pay Chauds. Paul Lechevalier, Paris. 3154 pp. Vol. 1-3.

Roman, J. 1978. Fitonematologia Tropical. Est. Exp. Agric. Univ. Puerto Rico, Rio Piedras. 256 pp.

Smart, G. C., and V. G. Perry. eds. 1968. Tropical Nematology. Univ. Florida Press, Gainesville. 153 pp.

Weber, G. F. 1973. Bacterial and Fungal Diseases of Plants in the Tropics. Univ. Florida Press, Gainesville. 673 pp.

Webster, J. M. ed. 1972. Economic Nematology. Academic Press, New York. 563 pp.

Wellman, F. L. 1972. Tropical American Plant Diseases. Scarecrow Press, Metuchen, NJ. 989 pp.

Wellman, F. L. 1977. Dictionary of Tropical American Crops and Their Diseases. Scarecrow Press, Metuchen, NJ. 495 pp.

Agencies Concerned With Tropical Plant Diseases

It is a challenge just to learn the names and acronyms of all of the innumerable agencies concerned with tropical plant diseases. Any of the hundreds of institutions involved in agriculture in tropical developing countries may have a valid concern with plant diseases at some time. The 1983 "Acronym List of International Organizations Related to Agriculture, Economic Development, and Pest Management" published by the Consortium for International Crop Protection (CICP) (1981) listed over 500 different organizations. Other references to international agencies which may have a concern with tropical plant diseases are Chiarappa (1970), Thurston (1977), Kriesberg (1977), Warnken et al. (1978), International Agriculture Development Service (1979), and Zentmyer et al. (1983). CICP (1981) classified the organizations as 1) national, state and local 2) regional and international 3) bilateral and multilateral technical and economic assistance 4) education, research and reference. Zentmyer et al. (1983) listed the several kinds of agencies engaged in scientific and agricultural development as 1) governmental organizations 2) inter-governmental organizations 3) international agriculture research centers 4) universities 5) private institutions and private industry 6) scientific societies 7) foundations. Many other listings or classifications could be made.

Before World War II most of the research on tropical plant diseases was done by the various colonial powers. They took responsibility for most aspects of plant disease control and established a number of excellent experiment stations, some of which still exist today. Especially during the later half of the nineteenth century, botanists visiting the tropics collected fungi extensively and sent most of their specimens to Europe for identification. They called attention to diseases of tropical crops and generated interest in such problems. In addition, a number of private corporations or planters' associations supported research on cash or plantation crops such as bananas, sugarcane, rubber, coffee, and tea. Most research in the colonial era was on cash and plantation crops and not on the tropical food crops.

After World War II the disappearance of the colonial powers, especially in tropical Asia and Africa, created a vacuum in agricultural research that many outside agencies stepped in to fill. Examples of such agencies were FAO (Food and Agriculture Organization of the United Nations), US-AID (United States Agency for International Development), and many universities and private foundations. Today, as previously noted, a bewildering number of agencies are concerned with tropical plant diseases.

National Organizations

Most tropical countries have organizations concerned with controlling plant diseases. Ultimately, these national organizations must solve the problems of increasing food production and improving equity of resource distribution in tropical countries. Bilateral and international aid agencies supported by wealthy countries can help, but can never and should never try to solve problems without the close involvement of local organizations.

It will be useful to compare the entities responsible for plant disease control in a country like the USA with those in tropical developing countries. Although not a complete listing, in the USA the following entities have responsibility:

1) Individuals: growers, farmers, custom sprayers, greenhouse operators, disease control consultants, etc.

2) Federal and state agencies: extension, quarantines and eradication, seed certification, parks, national forests, U.S. Department of Agriculture, universities.

3) Private institutes: private universities, Boyce Thompson Institute, etc.

4) Private industry: commercial producers of fungicides and nematicides, agricultural machinery manufacturers, seed producers, etc.

Before making comparisons it should be noted that tropical countries are highly variable in regard to their capacity to control plant diseases. For example, some countries have no trained plant pathologists, and others have large numbers of pathologists with Ph.D.s. Some tropical countries are as advanced in certain sectors of their agriculture (usually cash and plantation crops) as any temperate country in Europe or North America. Thus, there are exceptions to all of the following generalizations.

In some of the least developed tropical countries, except for individual farmers, few or none of the agencies responsible for plant disease control in the USA are found. 1) Of the various agencies engaged in research, extension, and education, those in extension usually receive the least support and have the least prestige. This is most unfortunate, since there probably is a greater need for technology transfer or effective extension of existing information to farmers in most tropical countries than there is for additional research. Extension is seldom linked administratively with research or education and may even be openly antagonistic to them because of competion for funds and influence. Often extensionists are poorly trained, operate with few resources, and are given impossible responsibilities. Extension specialists (ie. extension plant pathologists) are not common. Duplication of effort in extension is common, since often many agencies are engaged in extension rather than one, centralized, well supported organization. 2) Quarantine and eradication agencies often have poorly trained personnel, inadequate facilities, and lack sufficient human and physical resources to do their assigned tasks properly. Borders are often impossible to guard, and smuggling may be common. Nevertheless, quarantines have been effective in some tropical countries and need to be strengthened in most. It should be remembered, however, that quarantines can prevent useful plant introductions unless knowledgeable people enforce them. 3) National research organizations vary widely in their effectiveness, organization, and human and physical resources. Many work primarily on cash and plantation crops important to the national economy, and not on the food crops important to the majority of the population. In many countries budgets are small even though agriculture may be the major sector of the economy. Lack of continuity is common. For example, we had 18 different Ministers of Agriculture during the 11 years I worked in Colombia, South America. Most changed the top personnel in the Ministry when they took office, thus destroying program continuity. Researchers are often expected to work on a variety of problems as the need arises (ie. "put out fires"), rather than working on long term, carefully planned, basic problems of food production. Fortunately, there are a growing number of countries where this is not the case and excellent, long term research programs are found.

Research in the tropics seldom has adequate government support and thus lacks the fine laboratories, greenhouses, and field facilities that scientists take for granted in most temperate countries. Budgets are often completely inadequate and, in addition, in far too many cases a highly trained plant

pathologist returns to the tropics to find few or no facilities and no trained help. Duplication of effort in reseach is common. 4) Universities seldom do more than teach. Many professors do not teach full time because of poor salaries. Often universities have no effective links with either research or extension, and professors are not rewarded for research. 5) Few private, indigenous institutes are found in the tropics. 6) Private industry (such as the producers of pesticides, agricultural machinery, and seed producers) is highly active in the tropics. Many have their own experiment stations in the tropics where they can test their own products. Most firms are foreign (European, North American, or Japanese). Private industry can play an important role in extension as farmers are almost completely dependent on outside help for information on new fungicides and nematicides and their proper use.

Bilateral Aid Organizations

Most of the wealthy developed countries have governmental organizations which provide a variety of assistance in agricultural development to tropical developing countries. This assistance frequently involves some aspect of plant disease control. For example, in 1982 the Agency for International Development (AID) of the United States State Department requested 1.9 billion dollars for AID development assistance out of a total of 8.1 billion requested for all foreign and economic assistance (McPherson 1981). To keep this sum in perspective, it might be noted that in 1978 30.9 billion dollars were spent for alcohol and 17.9 billion for tobacco products in the United States according to the U.S. Department of Commerce (Report of the Presidential Commission on World Hunger 1980). AID has frequently used U.S. universities and the U.S. Department of Agriculture in its overseas assistance activities. A number of plant pathologists have had long and productive careers with AID. Although the USA has the largest assistance program in the world in terms of dollars spent, most other developed countries spend a greater share of their wealth for assistance than does the USA.

Other bilateral aid organizations include the United Kingdom's Commonwealth Agricultural Bureaux (CAB) and the Overseas Development Administration. The Commonwealth Mycological Institute (CMI) of the CAB provides an important information service for plant pathologists in tropical countries in addition to publishing the Review of Applied Plant Pathology. France has been active in tropical agriculture through its assistance activities coordinated by GERDAT (Le Groupement d'Estudes et de Recherches pour le Developpement de l'Agronomie Tropicale) and ORSTOM (Office de le Recherche Scientifique et Technique Outre-Mer). The Federal Republic of Germany provides assistance to the tropics through the German Agency for Technical Cooperation or GTZ (Deutsche Gesellschaft für Technische Zusammenarbeit). The Netherlands government through the International Agency for Technical Assistance (DGIS) supplies financial support and expertise to a number of tropical developing countries. For agricultural projects backstopping and guidance is provided by the International Agricultural Center (IAC) of the Ministry of Agriculture. The Royal Tropical Institute of the Netherlands supervises many assistance projects in the tropics and publishes Tropical Abstracts. Other countries with assistance programs include Australia, Belgium, Denmark, East Germany, Japan, Norway, Peoples Republic of China, the Republic of China, Spain, Sweden, Switzerland, and the USSR.

Multilateral Organizations

The most important and well known intergovernmental organization concerned with improving agriculture in the tropics is FAO (Food and Agriculture Organization of the United Nations) with headquarters in Rome, Italy. FAO was established in 1945. Chiarappa (1980) recently summarized its activities in controlling plant diseases. Many plant pathologists have been involved in FAO activities in the last several decades.

The OAS (Organization of American States) established IICA (Inter-American Institute of Agricultural Cooperation) in 1942. IICA is a regional organization concerned with agriculture in the Americas. CATIE (Centro Agronomico Tropical de Investigacion y Ensenanza) was begun in 1973 in Turrialba, Costa Rica, in cooperation with IICA and other agencies and governments of the Americas. CATIE emphasizes helping small farmers of the American tropics improve their agriculture.

The South Pacific Commission, founded in 1947, includes several Pacific states plus the governments of Australia, France, New Zealand, the United Kingdom, and the United States. In addition to improving the South Pacific's economic welfare, it includes a regional plant protection service.

University Programs

Many universities, especially those of North America and Europe, have been deeply involved in agricultural projects in tropical countries. Most projects included primarily training and institution building. Plant pathologists have been active in many of these projects. A few projects go back to before World War II, such as the N.Y. State College of Agriculture of Cornell University's project begun in 1924 with the University of China in Nanking. To list all of the university projects in the last few decades would be impossible. An example of the involvement of plant pathologists in an overseas university project and the typical problems encountered is given by French and Apple's (1974) description of the North Carolina State University project in Peru.

Private Industry

Because of the excessive numbers it is impossible even to list all of the commercial organizations involved with plant diseases in the tropics. Major groups include pesticide manufacturers, private organizations involved with cash and plantation crops, seed-producing companies, and manufacturers of pesticide application equipment. Most companies producing fungicides and nematicides are European, North American, or Japanese. Many of these companies have major research and extension activities related to tropical plant diseases. Some seed-producing companies, especially those producing hybrid maize seed, are active in a few tropical countries. The manufacturers of pesticide application equipment often have representatives in the tropics.

Private organizations have supported research on all of the major tropical cash and plantation crops. The larger rubber manufacturing companies had research programs which contributed to rubber production. Research on bananas was supported for decades by companies such as United Fruit and Standard Fruit Company. Although the banana companies now generally do not own their own plantations, research on banana production problems continues. Researchers for these companies made outstanding contributions towards the control of banana diseases. The American Cocoa Research Institute (ACRI), supported by private chocolate manufacturers, supported research, training, and extension activities in the tropics. Similar groups in Europe work with researchers in Africa and Asia and cooperate with ACRI. Private companies had their own pineapple

programs, and in addition supported research activities such as those of the Pineapple Research Institute in Hawaii. Private groups interested in sugarcane support priva e and public sugar cane research and also finance the outstanding publication "The Sugar Cane Pathologists Newsletter". The coffee industry in many parts of the world has supported excellent research programs. For example, the coffee interests in Kenya started an independent research station around 1940, initially financed by the Coffee Board of Kenya and later jointly with the government.

International Agriculture Research Centers

Most of the international agriculture research centers (IARC's) are supported by the Consultative Group on International Agricultural Research (CGIAR), but a few are not. The CGIAR supported IARC's developed from the Rockefeller Foundation's agricultural programs in Mexico, Colombia, Chile, and India. Led by plant pathologist J. George Harrar, a cooperative agricultural program was begun in Mexico in 1943 by the Rockefeller Foundation and the Mexican government (Stakman et al. 1967). The success of these programs (Wellhausen 1976), and the experience gained led to the subsequent establishment of the International Rice Research Institute (IRRI) and later CIMMYT (Centro Internacional de Mejoramiento de Maiz y Trigo) by the Ford and Rockefeller Foundations. The Mexican wheat program, led by plant pathologist Norman E. Borlaug (Dalrymple 1975), developed germplasm which led to the dramatic increase in wheat production in Asia in the 1960's which became known as the green revolution. Most of this accomplishment occurred before the IARC's were in operation.

After the establishment of IRRI in 1960 and CIMMYT in 1966, the Ford and Rockefeller Foundations established IITA (International Institute of Tropical Agriculture) in Nigeria in 1968 and CIAT (Centro Internacional de Agricultura Tropical) in Colombia in 1969. The outstanding success of the high-yielding varieties (HYV's) of wheat and later rice developed at IRRI prompted the establishment of additional IARC's. These additional IARC's were beyond the financial means of the Ford and Rockefeller Foundations. In 1971 a consortium of potential donors was organized to coordinate funding and establish additional centers. The consortium is called the Consultative Group on International Agricultural Research (CGIAR) and is sponsored by the World Bank, FAO, and the United Nations Development Programme (CGIAR 1980). Thirty-five countries, international and regional agencies, and private foundations are among the donors.

The budgets of the IARC's are modest, considering the scope of their activities and their global responsibilities. The 1984 budget for the CGIAR supported IARC's was about 180 million dollars. This figure may appear large, but when compared with the United States National Science Foundation's 1983 budget of almost one billion dollars or the National Institute of Health's 1983 budget of over two billion (Norman 1983) their funding might even be considered meager. Another comparison can be made with the three billion dollars invested in agricultural research in the United States in 1982 by the states, the federal government, and the private sector according to "Research 1984", distributed by the Experiment Station Committee on Organization and Policy and the Cooperative State Research Service. Private sector funding was about 1.6 billion dollars whereas public sector funding was near 1.4 billion.

Thurston (1977) has described the activities in plant pathology of the IARC's. Table 1 lists the centers and their activities. In addition to the IARC's funded by CGIAR, there are a few others not funded by CGIAR with

activities concerned with tropical plant diseases. These are AVRDC (Asian Vegetable Research and Development Center) in Taiwan, Republic of China, CATIE (Centro Agronomico Tropical de Investigation y Ensenanza) in Turrialba, Costa Rica, and INTSOY (International Soybean Program) of the University of Illinois.

Table 1. The global network of international agriculture research centers concerned with plant pathology.

Center	Crop and research emphasis	Location	Date of Establishment
Asian Vegetable Research and Development Center (AVRDC)	Small farmer vegetable production, especially of tomato, Chinese cabbage, sweet potato, soybean, and mung bean	Shanhua, Taiwan, Republic of China	1972
Centro Agronomico Tropical de Investigacion y Ensenanza (CATIE)	Latin American Tropics, small farmer systems, polycropping	Turrialba, Costa Rica	1973
Centro International de Agricultura Tropical (CIAT)	Tropics of the Western Hemisphere; cassava, beans, tropical pasture crops, and rice	Cali, Colombia	1967
Centro Internacional de Papa (CIP)	Potatoes for high-altitude temperate and lower tropical regions	Lima, Peru	1971
Centro Internacional de Mejoramiento de Maiz y Trigo (CIMMYT)	Maize, wheat, barley and triticale improvement worldwide	El Bataan, Mexico	1966
International Board for Plant Genetic Resources (IBPGR)	Collection, conservation, documentation, and use of plant germplasm	Rome, Italy	1974
International Center for Agricultural Research in the Dry Areas (ICARDA)	Rainfed agriculture in the arid and semi-arid regions of North Africa and West Asia. Barley, lentils, faba bean, wheat, chickpea, and forage improvement	Aleppo, Syria	1976

Table 1 (continued)

Center	Crop and research emphasis	Location	Date of Establishment
International Crops Research Institute for the Semi-Arid tropics (ICRISAT)	Agriculture in the semi-arid tropics. Sorghum, pearl millet, chickpea, pigeonpea, and groundnut improvement	Andhra Pradesh, India	1972
International Institute of Tropical Agriculture (IITA)	Humid and subhumid tropics; traditional farming systems, cassava, yam, sweet potato, rice, maize, soybean, lima bean, winged bean, and pigeonpea improvement	Ibadan, Nigeria	1967
International Rice Research Institute (IRRI)	Rice improvement and related farming systems	Los Banos, Philippines	1960
International Service for National Agricultural Research (ISNAR)	To provide assistance to national programs in planning and executing research and development	The Hague, Netherlands	1980
International Soybean Program (INTSOY)	Soybean improvement worldwide	Urbana, Illinois, United States	1973

Literature Cited

Chiarappa, L. 1970. Phytopathological organizations of the world. Annu. Rev. Phytopathol. 8:419-440.

Chiarappa, L. 1980. The role of FAO in research and control of plant diseases. Plant Dis. 64:362-367.

Consortium for International Plant Protection. 1981. Acronym List of International Organizations Related to Agriculture, Economic Development, and Pest Management. Pest Management and Related Environ. Protec. Proj., Berkeley, CA. 124 pp.

Consultative Group on International Agricultural Research. 1980. Consultative Group on International Agricultural Research. CGIAR, Washington, D.C. 50 pp.

Dalrymple, D. G. 1976. Development and spread of high-yielding varieties of wheat and rice in the less developed nations. Foreign Agric. Econ. Report no. 95. U.S. Dept. Agric. Econ. Res. Serv., Washington, DC. 120 pp.

French, E. R., and J. L. Apple. 1974. The commodity in-depth projects of North Carolina State University in Peru: a model bilateral aid programme. FAO Plant Protec. Bull. 22:42-47.

International Agric. Development Service. 1979. Agricultural Assistance Sources. Intl. Agric. Develop. Service, New York. 222 pp.

Kriesberg, M. 1977. International organizations and agricultural development. U.S. Dept. Agric. Foreign Agric. Econ. Report No. 131. 135 pp.

McPherson, M. P. 1981. America: continuing a proud tradition of helping others. Agenda 4(4):25-28.

Norman, C. 1983. Reagan's budget boosts basic research. Science 219: 747-748.

Report of the Presidential Commission on World Hunger. 1980. Overcoming World Hunger: the Challenge Ahead. U.S. Gov't. Printing Office, Washington, D.C. 29 pp.

Stakman, E. C., R. Bradfield, and P. C. Mangelsdorf. 1967. Campaigns against Hunger. Belknap Press, Cambridge, MA. 328 pp.

Thurston, H. D. 1977. International crop development centers: a pathologist's perspective. Annu Rev. Phytopathol. 15:223-247.

Warnken, P. F., R. M. Ragsdale, and C. M. Flaim. 1978. International Agriculture: Challenging Opportunities. Univ. Missouri Bull. M 107. 166 pp.

Wellhausen, E. J. 1976. The agriculture of Mexico. Sci. Am. 235(3):128-150.

Zentmyer, C. A., H. D. Thurston, and J. S. Niederhauser. 1983. International cooperation in agricultural research: the basis for feeding more people and feeding people more. In Kommedahl, T. and P. H. Williams. eds. Challenging Problems in Plant Health. Amer. Phytopath. Soc., St. Paul. 533 pp.

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