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Shoot and pod diseases of cowpea induced by fungi and bacteria

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Abstract
This paper presents a global perspective on bacterial and fungal pathogens that directly infect cowpea foliage, stems, and pods. A brief outline is presented of the main symptoms, distribution, economic importance, epidemiology, and control of bacterial blight/pustule and of 11 fungal diseases: anthracnose, Ascochyta blight, black leaf spot (= leaf smut), brown blotch, brown rust, Cercospora and Pseudocercospora leaf spots, powdery mildew, Pythium soft stem rot, Septoria leaf spot, Sphaceloma scab, and web blight. Minor diseases are listed in a Table.

Introduction

This paper will focus, as the title indicates, on shoot and pod diseases of cowpea induced by fungi and bacteria. It will thus exclude bacterial and fungal diseases incited by soilborne pathogens, i.e., those which naturally infect the plant only through its underground parts, even if they induce major symptoms in any of the aerial parts of the cowpea plant. Other papers in this volume cover nematodes and other soilborne pathogens (Florini 1997; Roberts et al. 1997), the parasitic weeds *Striga* and *Alectra* (Singh and Emechebe 1997; Lane et al. 1997), and virus diseases (Hampton et al. 1997; Huguenot et al. 1997). Taken together, these papers bring us up to date and supplement information contained in an earlier volume (Aggarwal 1985; Caveness and Ogunfowora 1985; Emechebe and Shoyinka 1985; Lin and Rios 1985; Mew et al. 1985; Patel 1985; and Thottappilly and Rossel 1985) on the global range of cowpea diseases and pathogens.

Major bacterial diseases

Bacterial blight and bacterial pustule. Bacterial blight (induced by *Xanthomonas campestris* pv. *vignicola* [Burkholder] Dye) is probably the most widespread disease of cowpea, having been reported from all regions of the world in which cowpea is cultivated. By contrast, bacterial pustule has a more restricted distribution; until the recent report of its occurrence in Nepal by Dahal et al. (1992), it was considered to be limited to Africa (Patel 1981). There is still some controversy about the species of *Xanthomonas* that induces bacterial pustule. Based on differences in pathogenic behavior of the bacterial blight and the pustule pathogens, Patel and Jindal (1982) suggested that the pustule pathogen should

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be regarded as a distinct pathovar of *X. campestris*, namely *X. campestris* pv. *vignae-unguiculatae*. Emechebe and Shoyinka (1985) speculated that it could be a strain of the bacterial blight pathogen, *X. campestris* pv. *vignicola*, and preliminary characterization of 120 isolates from pustule or blight symptoms support their point of view (K. Wydra, personal communication, IITA, Cotonou, Benin). Pathogenic variability has been reported for both pathogens; Patel (1981) reported the existence of three races of the bacterial pustule pathogen, while Prakash and Shivashanker (1982) suggested that the race of the bacterial blight pathogen prevalent in India differs from that prevalent in Nigeria.

The pathogens of both diseases are seed transmitted, while secondary spread occurs by wind-driven rain (Preston 1949; COPR 1981). Insects have also been implicated in secondary spread of the bacterial blight pathogen (Kaiser and Vakili 1978). Both diseases cause premature leaf fall and water-soaked dots on the undersurface of leaves (Williams 1976). Unlike bacterial pustule, bacterial blight induces large, irregular foliar lesions with yellow margins (Patel 1982), stem cankers, and both preemergence and postemergence seedling mortality (Kishun 1989).

Total crop loss in susceptible varieties may result from seedling cankers or severe cankers of peduncles and floral cushions on older plants. Kishun (1989) working in India—where bacterial blight is considered the most destructive among all cowpea diseases (Prakash and Shivashanker 1982)—reported grain yield losses of 2.7–92.2%, depending on the susceptibility of the variety.

Apart from the work of Ekpo (1978, quoted by Allen 1983), who reported yield losses due to bacterial pustule of 1.8% and 26.6% in resistant and susceptible varieties, respectively, the only other attempt to quantify losses caused by bacterial pustule was that of Omotunde (1987) at Ibadan, Nigeria. He reported 76.8% and 2.3% losses in grain yields of susceptible (TVx 301) and resistant (TVu 43) lines, respectively.

The influence of some cultural practices on the severity of bacterial blight has received relatively little attention. Rao and Hiremath (1985) in India showed that disease severity was increased by N and P applications, but was decreased by the applications of moderate levels of K and Mo, and high doses of Ca and Mg. In Kenya, Ouko and Buruchara (1987) showed the contrasting effects of cropping system on the incidence and severity of bacterial blight in cowpea grown in the long or the short rainy season. At 40 days after inoculation during short seasons, disease incidence was 62.5% in a cowpea/maize intercrop, compared to 75% in a sole crop of cowpea and 92.3% in a cowpea-maize relay crop. By contrast, in long seasons, blight incidence was 68.7% in a cowpea-maize relay crop and 100% in both sole cropped cowpea and a cowpea/maize intercrop. In a sowing date trial in India, Kishun and Chand (1989) showed that damage by bacterial blight was lower in an early-sown crop than in a later-sown crop, and that the disease intensified with an increase in plant populations.

Emechebe and Shoyinka (1985) suggested that the incidence and severity of both diseases would decrease if farmers sowed only pathogen-free seeds. Soni and Thind (1991) showed that it was easy to obtain pathogen-free seeds from healthy pods. The effectiveness of this control measure can be enhanced by seed treatment with an antibiotic or a mixture of an antibiotic and a fungicide, such as streptocycline (100 µg/ml) plus captan (2000 µg/ml) (Jindal and Thind 1990). Suitable rotations of three consecutive cowpea-free growing seasons should also be effective against these host-specific xanthomonads.

Host plant resistance is the most viable option for the control of cowpea bacterial blight and pustule (Emechebe and Shoyinka 1985). Singh (1994) has listed many advanced breeding lines that are resistant to bacterial blight and are being used in breeding work.

Minor bacterial diseases

In their review of cowpea diseases in Latin America, Lin and Rios (1985) listed bacterial blight and two minor bacterial diseases, namely bacterial wilt (*Pseudomonas syringae* pv. *solanacearum*) and halo blight (*P. syringae* pv. *tabaci*). While halo blight was found in two states in Brazil, bacterial wilt was reported only in an irrigated area. Both diseases were thought to be of no economic importance in cowpea production in Brazil.

According to Patel (1985), cowpea in the USA is affected by two bacterial diseases: bacterial blight and bacterial leaf spot induced by *Pseudomonas syringae* pv. *syringae*. Although leaf spot has been reported from several states and the pathogen has an extensive host range, the disease is considered to be economically unimportant in the USA (Patel 1985). The same disease was recently reported in Romania (Severin and Stancescu 1990). Bacterial leaf spot has not been reported under natural conditions in Africa. In the rainforest zone of Nigeria, Oluwadare and Umechuruba (1991) recorded the effect of antibiotics on the isolation of *P. syringae* pv. *syringae* from cowpea seeds, but their report did not indicate whether or not the bacterium induced leaf spot in cowpea in the field.

Major fungal diseases

The major fungal diseases of cowpea are discussed below in the alphabetical order of their common names.

Anthracnose. Until recently, the pathogen of cowpea anthracnose was regarded as a form of *Colletotrichum lindemuthianum* [Sacc. and Magn.] Briosi and Cav., the pathogen of anthracnose on *Phaseolus* beans. However, Bailey and associates (Bailey et al. 1990; O'Connell et al. 1992; Pain et al. 1992) have raised important questions about the taxonomic status of the cowpea anthracnose pathogen. On the basis of the molecular, morphological, and antigenic differences that exist between the anthracnose pathogens of cowpea and *Phaseolus* beans, it was suggested that the cowpea anthracnose pathogen should be regarded as a species that is distinct from *C. lindemuthianum*, probably a form of *C. gloeosporioides*.

Typical anthracnose lesions (tan to brown, sunken and lenticular) on susceptible varieties enlarge rapidly and coalesce to girdle stems, peduncles, and petioles. Profuse sporulation occurs. In contrast, lesions on resistant varieties are tiny, necrotic flecks or lenticular, shiny reddish-brown lesions; the fungus does not sporulate on such lesions (Williams 1975a; Emechebe and Shoyinka 1985).

The pathogen is seed transmitted (Emechebe and McDonald 1979) and Qureshi et al. (1985) suggested that it was introduced into Pakistan from Nigeria on infected seed. Prasanna (1985) found 2–88% infected seeds in seed samples from India and showed that the germination decreased with an increase in seed infection, which resulted in seed rot and seedling mortality. Infected seed is one source of primary inoculum (Prasanna 1985), as is infected trash (Onesirosan and Sagay 1975; COPR 1981). Secondary spread is by rain splash, air currents, and contact with man and animals (COPR 1981).

Anthracnose causes economic losses in tropical regions of Africa, Latin America, and Asia where conditions are wet and humid for the main part of the growing season (Dhiman et al. 1989; Latunde-Dada 1990). In the rainforest zone of Nigeria, yield losses of up to 50% occurred in susceptible varieties in the early 1970s (Williams 1973) but anthracnose is now less important following the use of resistant commercial varieties, e.g., TVx 3236. A grain yield loss of about 43% was reported in India by Sohi and Rawal (1984), but they also found high levels of resistance in many cultivars (Sohi and Rawal 1983).

Although cowpea varieties resistant to anthracnose are readily available (Singh 1994), the pathogen is highly variable and the occurrence of five putative races has been reported on breeding lines evaluated in various parts of Nigeria (Emechebe 1986). Consequently, other control measures are usually combined with the growing of resistant varieties, such as sowing seed obtained from anthracnose-free multiplication fields. While foliar application of fungicides by low-input farmers is probably not economical, some foliar fungicides, such as benomyl and carbendazim have reduced losses from > 40% to < 5% (Sohi and Rawal 1984). However, strains of the pathogen resistant to several of the most effective fungicides (e.g., carbendazim and thiophanate-methyl) have been detected in India (Naik and Anilkumar 1991).

Ascochyta blight. Emechebe and Shoyinka (1985) listed Ascochyta blight (*Ascochyta phaseolorum* Sacc.) among the major cowpea diseases in Africa. In Latin America, it occurs more frequently in the hot, rainy season than in the dry season (Lin and Rios 1985). Kannaiyan et al. (1987) also reported that the disease is severe only in the wet season in Zambia. We did not find reports of the disease in the USA.

Ascochyta blight causes severe defoliation and lesions on stems and pods, which may result in death. Severe epidemics occur mostly at medium elevations (500–1200 m); thus, screening of germplasm lines is in progress in Plateau state of Nigeria. Primary inoculum comes from infected seed and plant debris, while secondary spread is by rain splash, air currents, and wind-driven moisture.

There are few recent reports on Ascochyta blight. Price and Cishahayo (1986) suggested that the same species attacked *Phaseolus* bean and soybean in Rwanda. In Brazil, Rios et al. (1986) showed that the cropping system affected the number of leaf lesions and necrotic leaf area but not the lesion diameter, which was a good indicator of level of susceptibility. They also found that applying benomyl to foliage, burning crop residue, or incorporating crop residue into the soil did not influence Ascochyta blight development.

Black leaf spot or leaf smut. The taxonomy of the cowpea black leaf spot pathogen is still controversial. While pathologists in Latin America regard the pathogen as a true smut (Basidiomycotina), *Entyloma vignae*, because chlamydospores germinate to produce promycelia and sporidia (Prabhu and Albuquerque 1982), pathologists in Africa and India (Allen 1983) consider the pathogen to be *Protomycopsis phaseoli*, a Hemiascomycete, because a spore-filled vesicle is produced while chlamydospores germinate (Haware and Pavgi 1976). The symptoms from samples collected in parts of Africa and in Brazil were found to be identical in all respects (Allen 1983). Thus, black leaf spot and leaf smut are regarded as synonymous, pending further taxonomic work.

A good account of the symptoms has been provided by Singh and Allen (1979). The disease occurs widely in tropical Africa, Central America, Brazil, India, and Nepal (Vakili 1978; Allen 1983; Rios 1988). In Nigeria, we have observed the disease in various agroecological zones, from the rainforest to the Sudan savanna. The disease appears early in the season: typically, sooty black leaf spots usually remain confined to lower leaves in the canopy, except on susceptible varieties, where the spots may be seen on upper leaves. Cowpea smut is one of the most important diseases of cowpea in the north and northeast of Brazil, causing up to 40% loss in grain yield there (Lin and Rios 1985).

Some *Protomycopsis* spp. survive as chlamydospores in infected plant debris on the soil surface for at least 2 years, but lose viability if the debris is incorporated into the soil (Pavgi and Haware 1969). Thus, control measures include destroying crop residue, deep plowing, or crop rotation. Several cultivars (including one of the most popular Nigerian cultivars, Ife Brown) are resistant to the pathogen in Brazil (Lin and Rios 1985). In Nigeria, some varieties found to be resistant in Brazil, including Ife Brown, were moderately susceptible under natural and augmented inoculum pressure; many varieties were resistant, but only IT88S-584-1 had no symptoms in replicated trials in Kano and Ibadan in 1995 (T.O. Adejumo, T. Ikotun, and D.A. Florini, 1995, unpublished data, IITA and University of Ibadan, Ibadan, Nigeria).

Brown blotch. Brown blotch, first described in 1981 by Emechebe (1981), is induced by two species of *Colletotrichum: C. capsici* [Syd.] Butler and Bisby and *C. truncatum* [Schw.] Andrus and Moore. Results of surveys conducted from 1984 to 1986 showed that > 90% of brown blotch specimens were infected by *C. capsici*, although mixed infections on the same plant part were observed (A.M. Emechebe, 1986, unpublished data, IAR, Zaria, Nigeria). All plant parts above soil level show symptoms of the disease, which include one or more of the following: seeds failing to germinate, seedlings damping off, stems or branches girdling, flowers aborting, immature pods mummifying, and/or pods and leaves showing lesions.

The pathogen infects all parts of the seed (Alabi 1981), and it survives the dry season in seed (Emechebe 1981) and in infected debris (Okpala 1981); secondary inoculum is disseminated by rain splash, wind-driven rain, and air currents. The optimum temperature for radial growth and sporulation in artificial culture is 25 °C (Alabi and Emechebe 1992). Seedlings aged 1–2 weeks at the time of artificial inoculation were more severely affected by brown blotch than those inoculated at 3–6 weeks of age (Alabi 1994). The incubation period on all aerial plant parts was 2–3 days, regardless of age of plant at inoculation; by contrast, the latent period varied from 5 days (on the petiole) to 16 days (on the stem) (Alabi 1994).

Emechebe (1986) described eight possible races of *C. capsici,* after studying the qualitative interactions between 120 Nigerian isolates of *C. capsici* and different cowpea lines. Four races occurred mostly in the Guinea and Sudan savanna ecologies, while the other four were obtained from the rainforest zone. The most virulent races attacked both TVx 3236 and IT82D-716, which are known for their high levels of resistance to brown blotch.

In West and Central Africa, brown blotch is particularly important in the rainforest zone, the southern Guinea savanna, and the southern part of the northern Guinea savanna.

In the northern Guinea savanna of Nigeria, yield loss due to brown blotch was 46% (Alabi 1994), but it can reach 75% in very wet years in the same area (Emechebe and Shoyinka 1985). Infected plants produce tiny and wrinkled seeds that are unmarketable. Equally important is the reduction in stand establishment from 88% (for healthy seeds) to 24% (for seeds infected by *C. capsici*) (Emechebe 1981).

There is little information about the importance of brown blotch in Asia. Although Ravi and Anilkumar (1991) indicated that they obtained a virulent culture of *C. truncatum* (used in their fungicide resistance study) from cowpea cultivar C157, they did not indicate the importance of the fungus in cowpea production in India. Earlier, Prasanna (1985) merely noted that *C. capsici* is seedborne in cowpea without indicating if the fungus induced any disease in cowpea in the field.

The tactics used for the control of anthracnose outlined above also apply to brown blotch. In addition, seed treatment with benomyl or carbendazim has been shown to be a viable option for the peasant farmer in the West African northern Guinea savanna (Emechebe et al. 1994). By contrast, although foliar-applied fungicidal sprays are effective under field conditions (Alabi and Emechebe 1992), the technology may not be economically feasible for the low-input farmer.

Brown rust. The exact name of the cowpea rust fungus has been a subject of controversy among plant pathologists. The one point of agreement is that it is a species of *Uromyces*. Many authors (Emechebe and Shoyinka 1985; Lin and Rios 1985; Patel 1985) regard it as *U. appendiculatus* [Pers. ex Pers.] Unger, while others (Chandrashekar et al. 1989) consider it as *U. phaseoli* var. *vignae*. Detailed studies by Heath and associates (Kim et al. 1985; Elmhirst and Heath 1989) have provided strong support for the designation of the cowpea rust fungus as a separate species, namely *U. vignae* Barclay. In their subsequent histopathological studies, they have consistently referred to the rust pathogen as *U. vignae* (Chen and Heath 1990; Heath 1990) and more recent authors, such as Xu and Mendgen (1991), have adopted this nomenclature.

The main symptoms of brown rust are slightly raised brown or black pustules on the leaves (COPR 1981). When leaves of young plants are covered by pustules, wilting may occur during periods of acute soil moisture deficit. Leaves on heavily infected older plants dry up and fall prematurely. Dissemination of the uredospores may be through contact with people, animals, and farm implements, but the main agents are wind and, to a much lesser extent, insects (COPR 1981). The pathogen survives the period between crops as teliospores in infected crop residue.

Cowpea rust can be regarded as a major cowpea disease in the rainforest and southern Guinea savanna zones of West Africa and in medium-elevation areas of East Africa (Emechebe and Shoyinka 1985). Moderate to high intensities of rust occur as well in the northern Guinea savanna of Burkina Faso (Konate and Ouedraogo 1988). Quantitative estimates of crop losses caused by brown rust are rare, but we have observed severe epidemics in the Jos plateau and the rainforest zone of Nigeria, causing premature defoliation and even crop failure. Similarly, Mariga et al. (1985) reported that cowpea rust occasionally causes epidemics of economic importance in Zimbabwe.

Although Patel (1985) and Lin and Rios (1985) indicated that cowpea rust is not economically important in the USA and Latin America, Stoffella et al. (1990) have shown

that brown rust is one of the two most important fungal diseases of cowpea at Fort Pierce, Florida, USA.

The only economically viable option for the control of brown rust of cowpea, apart from crop sanitation, is the growing of resistant varieties; many commercial varieties are resistant to the disease (Patel 1985; Singh 1994).

Cercospora and Pseudocercospora leaf spots. Cercospora leaf spot is induced by *Cercospora canescens*, while Pseudocercospora leaf spot is induced by *Pseudocercospora (Mycosphaerella) cruenta*, formerly *C. cruenta* (Emechebe and Shoyinka 1985). Pseudocercospora leaf spot appears as chlorotic spots on the upper leaf surface, which gradually become necrotic, with profuse masses of conidiophores and spores, appearing as downy gray to black mats on the lower leaf surface (Emechebe and Shoyinka 1985; Lin and Rios 1985; Patel 1985). Severely affected plants defoliate prematurely. Cercospora leaf spot is characterized by mostly circular, cherry red lesions. Coalescence of leaf spots results in generalized yellowing of the leaf and subsequent defoliation of severely infected plants.

Both pathogens survive the no-crop season on infected crop residue and in infected seed (Williams 1975b; Patel 1985). Sporulation is favored by humid weather, warm temperatures, and dense plant populations. Spores are dispersed by wind and rain splash. Yield losses of 18–42% have been recorded for these leaf spots in Nigeria (Williams 1975a) and the USA (Schneider 1973).

Since 1985, very little work has been done on the two diseases. Kannaiyan et al. (1987) reported that "Cercospora" leaf spots (*C. canescens* and *P. cruenta*) are severe in the wet season in Zambia and that none of the 336 cowpea entries screened was resistant to the diseases. Similarly, Zhang and Huang (1990) listed Pseudocercospora leaf spot as one of the important diseases of cowpea in China. In Zimbabwe, however, Mariga et al. (1985) did not consider Cercospora and Pseudocercospora leaf spots to be economically important. Hartmans (1988) and Emechebe (1988) reported that *P. cruenta* has become more prevalent in the Nigerian Sudan savanna, although its effect on cowpea production in this zone is yet to be determined.

Powdery mildew. Cowpea powdery mildew is induced by the oïdial phase (*Oidium* spp.) of *Erysiphe polygoni* DC and *Sphaerotheca fuliginea*. *E. polygoni* is prevalent in all cowpea growing regions, but *S. fuliginea* has been reported only from India (Jhooty et al. 1985).

The diagnostic sign of this disease is copious, white, powdery fungal growth, mainly consisting of oïdia, the repeating spores of the fungus, on the upper leaf surface. Chlorotic and then brown patches appear first on the undersurface of the leaf, and they later become distinct on the upper leaf surface. Severely mildewed leaflets fall, resulting in partial or complete defoliation of the plant.

E. polygoni has a broad host range of more than 500 species of higher plants, both annuals and perennials, especially in the family Leguminosae (Ainsworth 1971). The fungus probably perpetuates itself on these hosts from one season to another as conidia; ascospores have not been detected in the tropics. Disease development in Latin America and Zambia was favored by wet weather (Lin and Rios 1985; Kannaiyan et al. 1987). By contrast, in the Sudan savanna zone of Nigeria, we observed moderate damage by powdery

mildew during the dry period at the end of the rainfed season and greater severity in irrigated, dry-season cowpea than in rainfed cowpea of the same variety. The disease is also destructive under hot, dry conditions in the screenhouse. In India, the disease increases rapidly during the dry and cool season (Mew et al. 1985). Since there are several races of the pathogen (Lin and Rios 1985), it is reasonable to expect the differences in the above reports. Indeed, Rodríguez and Meléndez (1984) have suggested that there is a new race capable of attacking cowpea under high relative humidity and heavy rains in Puerto Rico.

Cowpea powdery mildew is important in Zambia (Kannaiyan et al. 1987), Zimbabwe (Mariga et al. 1985), Florida, USA (Stoffella et al. 1990), Puerto Rico, and other cowpea-producing countries of Latin America (Rodríguez and Meléndez 1984; Lin and Rios 1985). The disease is so important in India that fungicidal sprays have been recommended for its control (Singh and Anilkumar 1986). However, we found no estimates of yield losses due to powdery mildew in cowpea.

Two control methods have received the greatest attention: growing resistant varieties and application of fungicides. Lin and Rios (1985) noted that resistant cultivars exist in Latin America but their use is limited by the occurrence of races, presumably with matching virulence genes. In India, both highly resistant and partially resistant lines have been identified (Raju and Anilkumar 1990, 1991). In Zambia, Kannaiyan et al. (1987) found no line to be resistant out of 140 entries, although two of them were moderately resistant (scoring 2–3 on a rating scale of 1–9). Fungicides have been evaluated as seed, soil, or foliar treatments for the control of cowpea powdery mildew. Singh and Anilkumar (1986) concluded that effective protection of cowpea was obtained by seed treatment with carbendazim, followed by one foliar-applied spray of triadimefon. In Puerto Rico, Rodríguez and Meléndez (1984) obtained very effective control of powdery mildew with dinocap in the dry season but not in the rainy season. Biweekly application of 0.26 kg/ha of benomyl also protected cowpea from infection by *E. polygoni.*

Pythium soft stem rot. Soft stem rot of cowpea, induced by *Pythium aphanidermatum*, is a mature plant disease that is distinct from seedling damping-off induced by the same fungus. The disease appears to be important only in warm, humid tropical conditions such as those of the rainforest, the southern part of the southern Guinea savanna of West and Central Africa (Onuorah 1973), and the humid, subtropical zones of India (Verma and Mishra 1989). We have also observed damaging levels of the disease in the northern Guinea savanna of Nigeria during long periods of very wet weather. Pythium soft stem rot caused crop loss of 11% under rainforest conditions in Ibadan, Nigeria (Onuorah 1973) but the disease is unimportant in Brazil (Lin and Rios 1985).

The characteristic symptom of Pythium soft stem rot is a gray-green, water-soaked rot that completely girdles the stem and kills the plant. The slimy stem base is covered by abundant growth of white, cottony mycelium during periods of high humidity. The pathogen is soilborne, surviving for many years in the soil in the form of perennating oospores; in addition, it has a broad host range of > 100 higher plant species. It has not been established whether the seedling disease and soft stem rot are induced by the same strain(s) of *P. aphanidermatum.*

Control of Pythium soft stem rot is difficult. However, Emechebe and Shoyinka (1985) have suggested that the infection rate can be reduced in moderate plant populations, since

the disease is enhanced by high plant populations. Application of some fungicides, such as benomyl, which are effective against other diseases of cowpea, can increase the severity of Pythium stem rot (Williams and Ayanaba 1975). However, Ogundana (1986) showed that some fungicides (e.g., thiram and fentin acetate) better controlled the disease when used as a seed treatment than as a soil drench.

Septoria leaf spot. Septoria leaf spot of cowpea is induced by three species of *Septoria*, namely *S. vignae*, *S. vignicola*, and *S. kozopolzanskii* (Emechebe and Shoyinka 1985). The most prevalent and most economically important across Africa is *S. vignae*, with reports of *S. vignicola* in East Africa and of *S. kozopolzanskii* in Zimbabwe (Mariga et al. 1985). By contrast, *S. vignicola* has been consistently reported as the pathogen of the disease in India (Rawal and Sohi 1981, 1984, 1986), while *S. vignae* is a minor pathogen in Nicaragua (Lin and Rios 1985).

The disease is characterized by red or reddish-brown leaf spots, which are regular to irregular and 2–4 mm wide, with the lesions on both surfaces of the leaf being essentially identical. The lesions coalesce to give the leaf a freckled appearance. Severe spotting results in generalized chlorosis and premature defoliation.

The pathogen is seed transmitted (Emechebe and McDonald 1979) and survives the dry season on infected seed as well as on infected leaf tissue lying on the soil (Tarfa 1986). We observed that secondary spread is by rain splash, wind-driven moisture, air currents, and contact with man, animals, and farm implements. Severe epidemics of the disease occur in the Guinea savanna zone of West Africa (Emechebe 1988; Konate and Ouedraogo 1988). At Zaria, Nigeria, no consistent relationship was found between disease severity and sowing date, although in 1 of 2 years, the crop sown in mid-July sustained more disease than crops sown in early August (Tarfa 1986).

Tarfa (1986) showed that grain yield losses due to *S. vignae* varied in the Nigerian northern Guinea savanna from 56.5% in 1984 to 42.5% in 1985. In India, Rawal and Sohi (1984) reported that the infection of cowpea at one week of age by *S. vignicola* reduced green pod yield by about 65%.

Although Septoria leaf spot causes high yield losses in susceptible cowpea in both India and Africa, it can be effectively controlled. The most economic and effective method is growing resistant varieties and such varieties are available (Singh 1994). The problem is that some of the most popular varieties grown in the northern Guinea savanna are susceptible to Septoria leaf spot. The incidence of leaf spot in such varieties can be reduced by using pathogen-free seeds, which can be further protected by seed treatment with benomyl or carbendazim (Emechebe et al. 1994). Foliar sprays with the same chemicals are also effective (Tarfa 1986). Similarly, foliar application of benomyl or carbendazim gives effective control of *S. vignicola* in India (Rawal and Sohi 1986).

Sphaceloma scab. Scab, induced by the Sphaceloma (conidial) stage of *Elsinoe phaseoli* Jenkins, produces characteristic lesions that are oblong to elongate, dark brown, buff, or white on stems, peduncles, and petioles. Lesions may coalesce. Heavy stem scabbing of a young plant results in severe stunting. An infected young leaf has a puckered lamina with white spots; the centre of old lesions frequently falls out to produce shot holes. Pod lesions, varying from a few to up to 200 per pod, are ovoid, with dark brown borders which become

black as chlamydospores form; heavily scabbed young pods abort or remain attached to the plant as mummified black masses. Heavy scabbing of the flowering axis either completely prevents flower formation or causes flower and pod abortion (Emechebe 1980).

The longevity of survival is probably mediated by chlamydospores produced on pod and stem tissues. The role of the ascospores in the epidemiology of the disease in the tropics is not known. Infected seed and plant material provide primary inocula (Donli 1983; Lin and Rios 1985; Emechebe 1988), while the subsequent dispersal of secondary conidial inoculum is by rain splash and wind-driven moisture (Emechebe and Shoyinka 1985).

Sphaceloma scab is probably the most important disease of cowpea wherever it occurs in both the northern and the southern Guinea savanna zones of West and Central Africa (Emechebe and Shoyinka 1985). Under conditions conducive for disease development (i.e., moderate temperatures of about 23–28 °C, 3 or more consecutive days of wet weather, and consequent high relative humidity) (Emechebe 1980) in the northern Guinea savanna of Nigeria, we have observed grain yield losses of 70% in Zaria in 1989 and 1990 (Mungo et al. 1995) and complete crop loss in susceptible varieties in Kachia. The disease is also one of the most destructive diseases of cowpea in Central America, Suriname, and Brazil (Lin and Rios 1985). We are not aware of any reports of the occurrence of Sphaceloma scab in India or the USA; in the latter, however, a different scab, induced by *Cladosporium vignae*, occurs (Table 1).

There are several options for the control of Sphaceloma scab. Much success has been achieved through deployment of resistance genes both in Latin America (Lin and Rios 1985) and in Africa (Singh 1994). However, TVx 3236, which is resistant to scab in Nigeria, is susceptible in Burkina Faso (Konate and Ouedraogo 1988), suggesting the existence of at least two races of the pathogen in West Africa. Good control of the disease has been achieved through fungicidal seed treatment (Emechebe et al. 1994) and foliar-applied fungicides (Mungo et al. 1995). Crop rotation and sanitation might be viable options for the control of a highly specialized pathogen like *E. phaseoli.* Preliminary results suggest that rotation does not affect scab incidence although scab symptoms were less severe in fields where cowpea followed another crop in rotation than in those where cowpea followed cowpea (C. Mungo, unpublished data, IAR, Zaria, Nigeria). Further study of the effectiveness of these measures is needed.

Web blight. Cowpea web blight is induced by an aerial type of *Rhizoctonia solani* (teliomorph = *Thanatephorus cucumeris*), the pathogenicity and biology of which are distinct from those of the strains that induce root rots and seedling diseases. Whereas the strains of *R. solani* that induce the latter diseases are strongly soilborne, the web blight strain, as suggested by Onesirosan (1977), has only a transient association with the soil.

Web blight symptoms range from small, circular brown spots to large irregular lesions with zonate banding, surrounded by water-soaked borders (Allen 1983). Under humid conditions, heavy blighting and premature defoliation occur, with affected leaves often bound together by webs of fungal hyphae (Singh and Allen 1979). The affected aerial parts of the plant may be covered with sclerotia, which resemble a dark coarse sand deposit.

The fungus has a broad host range (Lin and Rios 1985) and survives on infected crop debris (mostly as sclerotia), weed hosts, and seed (Onesirosan and Sagay 1975; Emechebe

Table 1. Occurrence of minor fungal shoot and pod diseases of cowpea in the major regions of the world.

Common name	Pathogen	Regions	Reference
Alternaria leaf spot	*Alternaria* sp.	Southern Africa	Maramba (1983); Mariga et al. (1985)
Aristatoma white leaf spot	*Aristatoma guttulosum* Sutton; *A. oeconomicum* (Ellis and Tracy) Tehon	West Africa; USA	Emechebe and Shoyinka (1985); Patel (1985)
Basal stem rust	*Aecidium* sp.	West Africa	Emechebe and Shoyinka (1985)
Chaetoseptoria leaf spot	*Chaetoseptoria wellmanii* Tehon	USA; Central America	Patel (1985); Singh and Allen (1979)
Choanephora pod rot (lamb's tail pod rot)	*Choanephora cucurbitarum* (Berk. and Rav.) Thaxt. *C. infundibulifera* (Currey) Sacc.	West Africa; India; Brazil; USA	Singh and Allen (1979); Bashir et al. (1985); Patel (1985); Toler and Duke (1965)
Cladosporium scab	*Cladosporium vignae*	USA; Southern Africa	Patel (1985); Mariga et al. (1985)
Corynespora target leaf spot	*Corynespora cassiicola* (Berk. and Curt.) Wei	West Africa; Central America	Emechebe and Shoyinka (1985); Lin and Rios (1985)
Dactuliophora zonate leaf spot	*Dactuliophora tarrii* Leakey	West, Central, East and Southern Africa; India	Emechebe and Shoyinka (1985); Chandrashekariah and Hiremath (1982)
Leptosphaerulina leaf spot	*Leptosphaerulina vignae* Tehon and Stout	USA	Patel (1985)
Myrothecium leaf spot	*Myrothecium roridum* Tode ex Fries; *M. graminum*	India	Singh and Shukla (1986); Mahrishi (1986)
Phyllosticta leaf spot	*Phyllosticta* spp.	Southern Africa	Mariga et al. (1985)
Pink rust	*Phakopsora pachyrhizi* Syd.	West Africa	Emechebe and Shoyinka (1985)
Red stem canker	*Phytophthora cactorum* (Lebert and Cohn) Schroet.	USA	Patel (1985)
Diaporthe stem rot	*Diaporthe phaseolorum* (Cook and Ellis) Sacc.	USA	Patel (1985)
Yellow blister (false rust)	*Synchytrium dolichi* (Cooke) Gaum.	East and Southern Africa	Emechebe and Shoyinka (1985); Kannaiyan et al. (1987)

and McDonald 1979). The only data on crop losses caused by web blight are those of Oyekan (1979), who reported losses of 28–40% in southwestern Nigeria. However, the pathogen can cause complete destruction of the leaf canopy during periods of heavy rain with long periods of overcast skies. We have observed further aggravation of the disease in portions of fields that contain stagnant water for 24 hours or more. In Latin America and India, the disease is destructive in hot, humid regions (Lin and Rios 1985; Verma and Mishra 1989).

Very little research effort has been devoted to developing a practicable control strategy against web blight. However, since the disease is favored by dense planting, a moderate plant population could reduce disease severity, as could any practice that ensures good drainage of the field. Latunde-Dada (1991) has demonstrated the potential use of a foliar-applied spore suspension of *Trichoderma koningii* as a biocontrol agent against the web blight pathogen. The level of disease control and the yield increase compared favorably with those obtained with a foliar fungicide spray.

Minor fungal diseases

Table 1 lists the minor fungal diseases of cowpea based on previous reviews (Emechebe and Shoyinka 1985; Lin and Rios 1985; Patel 1985), as well as on some new references. Although yellow blister (false rust) induced by *Synchytrium dolichi* is reported to be severe on rainfed cowpea in Zambia (Kannaiyan et al. 1987) and causes localized epidemics at medium elevations in Uganda, there are no published reports of crop losses caused by the disease. As Zambia and Uganda are, as yet, minor cowpea producers, yellow blister is listed with the minor diseases.

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