Chapter 13
Bacterial, Fungal and Nematode Diseases

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Introduction

Cassava is affected by a wide range of diseases caused by viruses, bacteria, fungi and nematodes (Table 13.1). With the exception of some of the virus diseases (see Chapter 12) and bacterial blight, most of the other diseases have been regarded as of minor or local importance. This may not always be the case and this perception may be at least partly because cassava in many countries is a subsistence crop, where yields are limited more by other major factors, such as low soil fertility and moisture stress, than by plant diseases. At present, there is often too little information on losses caused by these diseases to draw any conclusions about their importance. There is considerable variation among cassava cultivars in susceptibility to the 'minor' diseases, and where susceptible cultivars are grown and conditions favour disease development, losses can be substantial. This is particularly true of the root rot diseases, where cassava is grown in soil with a high water table, or, on land newly cleared from forest or bush. Brown leaf spot is common almost everywhere that cassava is grown, but usually regarded as unimportant, yet the defoliation it causes may have a significant effect on yield, where cassava is grown intensively for commercial production.

The diseases considered of economic importance vary to some extent between countries and between continents. Cassava mosaic virus disease occurs wherever the crop is grown in Africa but is absent from South America. Bacterial blight occurs in Africa, South America and Asia. In both Africa and South America the next most important group of diseases are the root rots. In Nigeria, Cameroon and Benin the pathogens causing root diseases of economic importance are *Sclerotium rolfsii*, *Botryodiplodia theobromae*, *Fomes lignosus*, *Rosellinia necatrix*, *Rhizoctonia solani*, *Phytophthora* spp. and *Fusarium* spp. (Arene *et al*., 1990; Afouda and Wydra, 1996). In Brazil, *Phytophthora* is probably the most important root pathogen but in some areas *Fusarium* spp. are also a problem (Lozano, 1991).

Bacterial Diseases

Cassava bacterial blight

Causal organism, distribution and importance

Cassava bacterial blight (CBB) is the most important bacterial disease of cassava and in Africa, it is second to cassava mosaic virus disease as a

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Table 13.1. Pathogens associated with cassava diseases worldwide.

Pathogen	Disease	Selected references
Fungi and bacteria		
Agrobacterium tumefaciens	Stem gall	CIAT (1978)
Armillariella mellea (*Armillaria mellea*)	Dry root rot	Makambila and Koumouno (1994); Mwenje *et al.* (1998)
Botryodiplodia theobromae	Stem rot, root rot	Lozano and Booth (1976); Afouda and Wydra (1996, 1997)
Cercospora caribaea	White leaf spot	Chevaugeon (1956); Lozano and Booth (1976)
Cercospora henningsii	Brown leaf spot	Lozano and Booth (1976); Ayesu-Offei and Antwi-Boasiako (1996)
Cercospora vicosae	Diffuse leaf spot	Lozano and Booth (1976)
Colletotrichum gloeosporioides f.sp. *manihotis*	Anthracnose	Lozano *et al.* (1981); Ikotun and Hahn (1994); Fokunang *et al.* (1997)
Cochliobolus lunatus	Stem rot	Msikita *et al.* (1997a)
Elsinoe brasiliensis	Superelongation disease	Lorenzo *et al.* (1981)
Erisyphe manihotis	Ash disease (powdery mildew)	Ferdinando *et al.* (1968); Lozano and Booth (1976)
Erwinia carotovora subsp. *carotovora*	Soft rot of stems and roots	Lozano and Bellotti (1979); Daniel *et al.* (1981)
Fomes lignosus	White thread	Lozano and Booth (1976)
Fusarium moniliforme	Wet root rot	Osai and Ikotun (1993); Msikita *et al.* (1996)
Fusarium oxysporum	Wet root rot	Afouda and Wydra (1996, 1997); Msikita *et al.* (1996)
Fusarium semitectum	Wet root rot	Afouda and Wydra (1996)
Fusarium solani	Wet root rot	Msikita *et al.* (1997b)
Leptoporus lignosus	Root rot	Makambila and Koumouno (1994)
Phaeolus manihotis	Root rot	Makambila and Koumouno (1994)
Phoma sp.	Ring leaf spot	Ferdinando *et al.* (1968); Lozano *et al.* (1981)
Phytophthora drechsleri	Root rot	Lima *et al.* (1995); Poltronieri *et al.* (1997)
Ralstonia solanacearum	Sudden wilt	Nishiyama *et al.* (1980); Machmud (1986)
Rhizoctonia solani	Root rot	Afouda *et al.* (1995)
Rosellinia necatrix	Dry root rot	Lozano and Booth (1976)
Sclerotium rolfsii (*Corticium rolfsii*)	Root/stem rot	Martin (1970); Osai and Ikotun (1993); Afouda and Wydra (1997)
Scytalidium lignicola	Black rot	Laranjeira *et al.* (1994)
Sphaerostilbe repens	Stem/root rot	Osai and Ikotun (1993); Makambila and Koumouno (1994); Afouda *et al.* (1995)
Uromyces sp.	Rust	Normanha (1970); Santos *et al.* (1988)
Xanthomonas campestris pv. *manihotis*	Bacterial blight	Boher and Verdier (1994); Laberry and Lozano (1992)
Xanthomonas campestris pv. *cassavae*	Angular leaf spot	Onyango and Mukunya (1982); Janse and Defranq (1988)
Nematodes		
Helicotylenchus erythrinae	Spiral nematode	McSorley *et al.* (1983)
Helicotylenchus dihystera	Spiral nematode	Caveness (1980)
Meloidogyne arenaria	Root-knot	Tanaka *et al.* (1979)
Meloidogyne hapla	Root-knot	Tanaka *et al.* (1979)
Meloidogyne incognita	Root-knot	Caveness (1982); Bridge *et al.* (1991); Crozzoli and Hidalgo (1992)
Meloidogyne javanica	Root-knot	Hogger (1971); Caveness (1980); Jatala and Bridge (1990)
Pratylenchus brachyurus	Lesion nematode	Charchar and Huang (1981); McSorley *et al.* (1983); Coyne (1994)
Rotylenchulus reniformis	Reniform nematode	Caveness (1980); Jatala and Bridge (1990)
Scutellonema bradys	Yam nematode	Caveness (1980)

cause of yield loss (Centro Internacional de Agricultura Tropical; CIAT, 1996). The causal agent of CBB is *Xanthomonas campestris* pv. *manihotis* (Berthet and Bondar, 1915) Dye 1978 (*Xcm*), proposed by Vauterin *et al.* (1995) to be reclassified as *Xanthomonas axonopodis* pv. *manihotis*. The bacterium can induce symptoms on cassava (*Manihot esculenta*) and related, wild *Manihot* species (*Manihot apii*, *Manihot glaziovii* and *Manihot palmata*), and after artificial inoculation, on (poinsettia) *Euphorbia pulcherrima* and *Pedilanthus tithymaloides* (Dedal *et al.*, 1980). Epiphytic survival for up to 1 month was demonstrated on several weed species occurring in cassava fields in Latin America and Africa (Marcano and Trujillo, 1984; Fanou *et al.*, 1998). *Xcm* is a Gram-negative rod and has a whitish to cream colony colour which is atypical for xanthomonads. Information on cassava bacterial blight was reviewed by Laberry and Lozano (1992) and by Boher and Verdier (1994).

Xcm originated in Latin America, where bacterial blight has been known since the early 1900s and the disease spread to the cassava growing regions of Africa and Asia during the 1970s (Bradbury, 1986; Boher and Verdier, 1994; Wydra and Msikita, 1998). CBB caused severe epidemics in Africa when it was first introduced, but the disease remains of minor importance in Asia. Severe infection, causing losses of up to 75% (Ohunyon and Ogio-Okirika, 1979), were reported in the 1970s from some areas in Ghana (Aklé and Gnouhoué, 1979) and Benin (Korang-Amoakoh and Oduro, 1979) and from Togo, Cameroon, Congo, Central African Republic, Nigeria, as well as from East Africa – Tanzania, Rwanda, Burundi and Kenya (Hahn and Williams, 1973; Terry and MacIntyre, 1976; Persley and Terry, 1977; Terry, 1977; Maraite and Perreaux, 1979). In Democratic Republic of Congo, CBB led in some areas to total loss of yield and availability of planting material, causing widespread famine. After the introduction of quarantine measures in some countries (e.g. Ghana) and the identification and planting of resistant varieties, the disease was contained. Further surveys were rarely conducted until the late 1990s (see below), so the current situation in respect of the distribution and severity of CBB remains unclear in several countries.

Surveys in 1994/1995 revealed a widespread occurrence of CBB in West Africa. Epidemics occurred in the transition forest and in the moist and dry savannah zones, where the disease occurred respectively at 50, 60 and 42% of sites at a mean disease incidence (% plants infected) of 16, 34 and 16%. CBB occurred only sporadically in the rainforest zone (Wydra and Msikita, 1998). Average disease severity of infected plants was high, with scores between 2.4 (savannah zones) and 3.1 (transition forest) on a scale of 1 to 5. Observations from Benin revealed a disease incidence of 19% in the transition forest zone, 77% in the Southern Guinea (moist) savannah, 35% in the Northern Guinea (dry) savannah and 84% in the Sudan savannah, with a high percentage of plants scored in classes 4 and 5 (Wydra and Verdier, unpublished). This level of severity indicates severe systemic infection which causes losses in root and leaf yield, and in planting material for the subsequent season. Most strains of the pathogen collected from all five ecozones were highly virulent (Wydra *et al.*, 2001c).

Strains of *Xcm* were collected from cassava growing in different edaphoclimatic zones and the population structure was evaluated by analysis of DNA polymorphisms and virulence variation. The population from South America was grouped into five clusters (ribotypes), each largely composed of strains from the same edaphoclimatic zone. Strains varied in virulence but this was unrelated to origin and was not correlated with DNA polymorphisms (Restrepo and Verdier, 1997). By contrast, the African population was very uniform, all strains belonging to one of the five ribotypes identified in South America, reflecting its more recent introduction in Africa (Verdier *et al.*, 1993, 1997). Care must be taken therefore, when introducing planting material from South America to Africa (see below).

Importance

Yield losses to CBB in the major cassava production zone, the humid lowlands of Africa, are estimated at 3.2 million t with 60% of the area affected. For Africa as a whole, it is estimated that up to 7.5 million t are lost annually due to CBB (CIAT, 1996). Comparing losses in five ecozones of West Africa, 13–50% loss of root yield was recorded with the highest losses in the dry savannah zone (Wydra *et al.*, 2001a). Crop

losses of 75% and of 90–100% were reported from Nigeria (Ohunyon and Ogio-Okirika, 1979) and Uganda (Otim-Nape, 1980). Strong genotype–environment interactions occurred and losses varied with variety, ecozone and year (Wydra *et al.*, 2001a,b). Disease epidemics and yield losses vary from year to year but may cause the same percentage loss, averaged over a number of years as has been reported for diseases with a steady, more predictable effect on yield. The unpredictability of disease outbreaks and of associated losses makes the disease a major risk for cassava production by subsistence farmers. Due to vascular infection, 30% of cuttings taken from an infected plantation may be lost in the first season of planting into a previously clean field. The losses can reach 80% by the third year of production (Restrepo and Verdier, 1997).

Symptoms and management

CBB symptoms appear with the first rains after the dry season and reach their maximum during the peak of the rainy season. Infected plants show typical water-soaked, angular leaf spots (Fig. 13.1), leaf blight and leaf fall, and systemic symptoms, resulting in the formation of cankers on the stems. In severe cases, CBB causes shoot die-back (Fig. 13.2), showing the typical 'candle stick' symptom. The pathogen invades the plant systemically, entering the stem and the seeds, but often producing no symptoms initially. The bacterium may then survive for a considerable period within the seed (Lozano *et al.*, 1989). Symptoms of CBB were described in detail by Maraite (1993).

Integrated control strategies comprise of improved cultural methods, crop sanitation, resistant cultivars and quarantine measures to prevent introduction of highly aggressive strains to areas with low or no infection (Wydra *et al.*, 1998; Fanou, 1999b; Wydra and Rudolph, 1999). A PCR-based diagnostic test has been developed which is sensitive down to the level of 10×2^2 colony forming units ml^{-1}, in cassava stem and leaf tissue (Verdier *et al.*, 1998).

Crop rotation to completely break the life cycle of the pathogen, and burying or burning infected debris, can provide some control, especially under dry conditions, when the pathogen may survive in crop residues for up to 5 months (Fanou *et al.*, 1998). Other control measures which may contribute to control of CBB, are weeding and avoiding bush fallow around cassava fields, control of grasshoppers – vectors of the disease (Fanou *et al.*, 1998; Fanou, 1999a) – mixed cropping associating cassava with maize to suppress the disease (Fanou, 1999b), avoiding the peak time of the epidemic by shifting planting date towards the end of the rainy season (International Institute of Tropical Agriculture; IITA, 1998), and application of potash fertilizer to reduce disease severity (Odurukwe and Arene, 1980).

Fig. 13.1. Cassava bacterial blight (*Xanthomonas campestris* pv. *manihotis*) – leaf symptoms showing typical water-soaked, angular spots.

Most cassava cultivars planted in Africa are sensitive to *Xcm* (Boher and Verdier, 1994). Several lines derived from inter-specific crosses between *M. glaziovii* and *M. esculenta* display good resistance to bacterial blight (Hahn, 1978; Hahn *et al.*, 1980). Varieties with considerable resistance to CBB now exist in the germplasm collection of IITA (Hahn, 1979; Wydra *et al.*, 1998; Fanou, 1999b). Nevertheless, varieties selected for stable resistance in various environments tend to lose some of their resistance over time, probably due to natural selection for more aggressive strains of the pathogen (Verdier *et al.*, 1994; Wydra *et al.*, 1998). Periodic reselection for resistance is advisable, following inoculation with freshly collected, virulent strains of the pathogen.

Screening for resistance can be performed by observing symptom development in the field under strong disease pressure over several crop cycles. This takes account of inoculum remaining in the stem (Boher and Verdier, 1994). Less time-consuming screening methods have been developed. Inoculation of young shoots on cuttings enables selection to be done in 2 months (Pacambaba, 1987; Boher and Agbobli, 1992; Wydra *et al.*, 2001c). The resistant response to *Xcm* has been associated with physical and chemical barriers in the host which restrict bacterial growth within the phloem (Boher *et al.*, 1996). An *in vitro* method was reported by Flood *et al.* (1995) which could identify resistance to *Xcm* in embryonic cell suspensions of the host, based on more rapid electrolyte leakage from cells of the susceptible lines.

Sanitary measures and quarantine precautions

During the cropping season, removal of infected leaves can significantly reduce disease severity (Fanou, 1999b). However, this is labour-intensive and is not a practical option for small-scale farmers. Planting material should be selected from fields free of CBB symptoms when inspected at the end of the preceding rainy season. New plantations should not be established near old cassava fields. Movement of man and animals in cassava fields, especially after rain, and use of contaminated tools contribute to the dissemination of the pathogen (Lozano, 1986).

For international germplasm exchange, the risk of introducing new strains of *Xcm* to Africa are greatest with seeds and this should generally be avoided. However, seeds can be disinfected by heat-treatment (Persley, 1979; Lozano *et al.*, 1989; Frison and Feliu, 1991; Fanou, 1999b) and tested for contamination using semi-selective media (Fessehaie *et al.*, 1999) or serological methods (Fessehaie, 1997; Fessehaie *et al.*, 1997). Vegetative propagating material should be exchanged by meristem cultures originating from heat-treated shoot tips of healthy plants. The FAO/IBPGR technical guidelines for the safe movement of cassava germplasm provide details on exchange of germplasm (Frison and Feliu, 1991).

Fig. 13.2. Cassava bacterial blight (*Xanthomonas campestris* pv. *manihotis*) – shoot dieback.

Other bacterial diseases

Angular leaf spot

Angular leaf spot (or bacterial necrosis) of cassava induced by *X. campestris* pv. *cassavae* Wiehe & Dowson (*Xcc*) is reported only from East Africa (Onyango and Mukunya, 1982) and Southern Africa, with one unconfirmed exception from Niger (Janse and Defrancq, 1988). Typical symptoms are angular leaf spots which develop less rapidly than leaf spots induced by *Xcm*, and leaf blight does not develop. Bright yellow exudates from infected leaf tissue during periods of high humidity are characteristic of this disease (Maraite and Perreaux, 1979). In severe cases bacterial necrosis can lead to defoliation of the plant, although the disease does not become systemic and the pathogen invades only the cortex of stems, not the vascular tissues (Maraite and Weyns, 1979). In contrast to *Xcm* with whitish colonies, *Xcc* colonies have a yellow colour which is typical for xanthomonads. Pathological, physiological and biochemical characteristics were described in detail by Maraite and Weyns (1979) and Van den Mooter *et al.* (1987). The disease occurs mainly on poor soils and after rainstorms injure the plants (Mostade and Butare, 1979). Fertilization is reported to retard disease development (Butare and Banyangabose, 1982).

Soft rot of stems and roots

Erwinia carotovora ssp. *carotovora* (*Ecc*) has been reported from Latin America, causing internal rotting of stems and branches, dark lesions and external cankers, necrosis of roots, wilting of young shoots and tip dieback, associated with injuries by insects, among them the cassava fruitfly (*Anastrepha* spp.; Lozano and Bellotti, 1978; Hernandez *et al.*, 1986). In the Democratic Republic of Congo and the Central African Republic, soft rot of harvested roots due to *Ecc* was observed (Daniel *et al.*, 1981). Infection causes root yield loss and loss of planting material (Cock, 1978). Planting uninfested, healthy cuttings of varieties resistant to the fruitfly and use of insecticides is recommended to control the disease (Lozano and Bellotti, 1978; Guevara *et al.*, 1992).

Sudden wilt, leaf drop and linear discoloration of stems and roots, and soft rot of roots were attributed to *Ralstonia solanacearum* [*Pseudomonas solanacearum*] in India and Indonesia (Nishiyama *et al.*, 1980; Machmud, 1986), whereas Lozano (1979) does not consider cassava as a host of *R. solanacearum*. *Agrobacterium tumefaciens* has been reported to cause stem gall on cassava (CIAT, 1978).

Fungal Diseases of the Leaf

Brown leaf spot

Causal organism, distribution and importance

Several *Cercospora* spp. and related fungi induce leaf spots on cassava but the most important of these is brown leaf spot (BLS) caused by *Cercosporidium henningsii* Allesch. [syn. *Cercospora henningsii*, *Cercospora manihotis*] (Powell, 1972). The fungus has a relatively wide host range, affecting in addition to *M. esculenta*, *M. glaziovi* (ceara rubber), *Manihot piauhynsis* and by inoculation, sweet potato (*Ipomea batatas*; Golato, 1963; Powell, 1968, 1972; Golato and Meossi, 1971). Lozano and Booth (1976) provide a detailed description of pathogen morphology. Another disease known in Brazil as brown large spot (diffuse spot) is caused by *Cercospora vicosae* (see below).

BLS is of worldwide distribution and occurs in most cassava fields in the lower canopy of crops more than 5 months old. It is favoured by high temperature and humidity. The optimum conditions for spore production were reported to be free water on the leaf surface and a temperature of 25–32°C (Ayesu-Offei and Antwi-Boasiako, 1996). Extensive surveys conducted in several West African countries showed that BLS was widely distributed with site incidence varying between 78% (wet savannah) and 98% (transition forest) and incidence within sites of 41% and 85%, respectively. However, average disease severity score was low (Wydra and Msikita, 1998).

The importance of BLS may be underestimated because it is often confined to the lower-canopy leaves, but it can cause leaf chlorosis and extensive defoliation. The effect of this defoliation, particularly when infection is followed by a period of drought-stress is difficult to quantify. It

has been reported that in Africa, in areas of high rainfall, the disease can cause yield losses on individual plants of up to 20% (Théberge, 1985). The effect on yield of a single leaf spot pathogen is often difficult to assess as they commonly occur as a leaf spot complex. In Brazil, the combination of *C. henningsii* and *C. vicosae* can cause up to 30% yield loss (Takatsu *et al*., 1990).

Symptoms and management

The leaf symptoms on cassava are visible on both sides of the lamina but are more pronounced on the upper surface, where the spots are a uniform brown with a distinct darker border (Fig. 13.3). During humid conditions, the presence of conidia gives the underside of the spots a greyish appearance. Spots are generally 3–12 cm in diameter and roughly circular but becoming more irregular as they expand. On some cultivars there may be a yellow halo around the spots and as the disease progresses leaves turn yellow, dry and eventually fall (Lozano and Booth, 1976). The older leaves are more susceptible to infection than younger leaves, but in highly susceptible cultivars, leaves in the upper canopy can also be infected (Chevaugeon, 1956).

BLS can be managed by wider spacing to reduce humidity and by the use of copper fungicides (Golato and Meossi, 1966, 1971). Disease management measures are rarely required in practice but where the crop is grown in high rainfall areas, the use of less susceptible cultivars is advised. Differences in susceptibility between cultivars has been reported from Africa and in the extensive germplasm collection at CIAT, Colombia (Lozano and Booth, 1976).

White leaf spot

Causal organism, distribution and importance

Cercospora caribaea Chupp & Ciferri has a worldwide distribution but white leaf spot (WLS) which it causes, is less common than BLS and is favoured by cooler conditions. A survey which included several West African countries showed WLS to be widely distributed in all the ecozones, although severity was generally low. The percentage of plants with symptoms ranged from only 1% in the dry savannah to 62% in the rainforest zone (Wydra and Msikita, 1998). Sporulation occurs readily on the surface of the lesions during humid weather and the conidia are distributed by wind and rain (Lozano and Booth, 1976). Little is known about the effect of the disease on the cassava plant but it generally appears to be negligible. There does, however, seem to be some differences between cultivars in susceptibility and considerable defoliation can occur in the most susceptible cultivars (Chevaugeon, 1956).

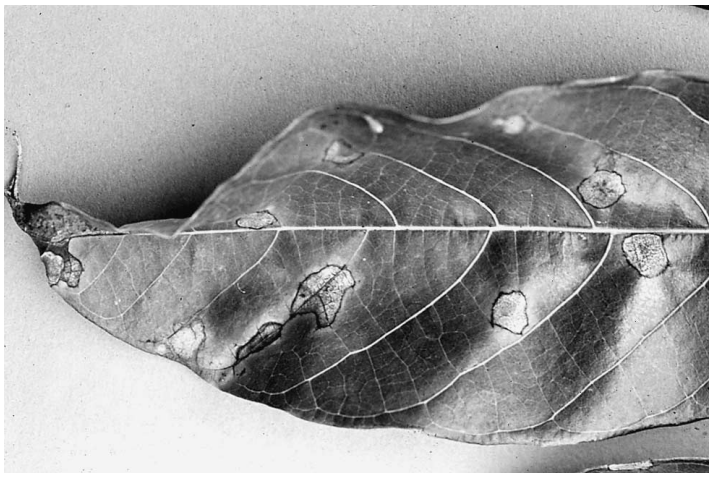

Fig. 13.3. Brown leaf spot (*Cercosporidium henningsii*).

Symptoms and management

First symptoms of the disease are circular chlorotic areas on the upper lamina which later develop small (1.5–2 mm) circular white lesions in the centre (Fig. 13.4) which are also visible on the underside of the leaf. The spots enlarge to 3–5 mm and may have a purple border, retaining the chlorotic halo (Lozano *et al*., 1981; Théberge, 1985). Sporulation occurs mainly on the underside and conidia are elongated and tapering, typical of *Cercospora* spp.

Diffuse leaf spot

Causal organism, distribution and importance

Cercospora vicosae Muller and Chupp, causes diffuse leaf spot (DLS). Again of worldwide distribution, it is more prevalent in the warmer and wetter areas where BLS is also common, particularly in Brazil and Colombia (Lozano and Booth, 1976) and in West Africa (Théberge, 1985). Wydra and Msikita (1998) refer to *Cercospora* leaf spot and their surveys in West Africa showed it to be one of the most widely occurring leaf spot diseases on cassava. Site incidences varied from 63% in the dry savannah zone to 100% in the mountain zone. Plant incidence was 97% in the mountain zone compared to 79% in the lowland rainforest zone which is surprising in view of other reports that the disease is more prevalent in warm, wet areas. The disease can cause severe defoliation in the more susceptible cultivars but this tends to occur in plants more than 6–9 months old and yield losses are believed to be slight.

Symptoms

The symptoms are quite distinct from the other two leaf spots described above, being large and diffuse without definite borders. Each spot may cover more than one-fifth of the leaf lobe (Fig. 13.5). They are uniformly brown on the upper side but on the underside, the centre of the spot takes on a greyish appearance, when sporulation is induced by humid weather (Lozano and Booth, 1976).

Ring leaf spot

Causal organism, distribution and importance

The exact aetiology of ring leaf spot (RLS) is unknown. A number of fungi originally identified as *Phyllosticta* spp. have been associated with the leaf lesions. The more generally accepted taxonomy for these fungi is now *Phoma* spp. (Lozano and Booth, 1976). RLS is more common in Latin America (Viegas, 1943; CIAT, 1972) than elsewhere, but has been reported

Fig. 13.4. White leaf spot (*Cercospora caribaea*).

also from India (Ferdinando *et al.*, 1968) and Africa (Vincens, 1915). There do not seem to be any more recent reports from Africa and the disease is not mentioned in the IITA field guide to pests and diseases of root and tuber crops in Africa (Théberge, 1985). The disease is favoured by temperatures below 22°C and is more common at high altitudes or in lowland areas during prolonged periods of cool, wet weather. In Latin America the disease is known to cause defoliation of the more susceptible cultivars and can, under optimum conditions for disease development, attack the young shoot to cause dieback and even in severe cases, death of the plant. Considerable losses are attributed to the disease where cassava is grown in cooler areas (Lozano *et al.*, 1981).

Symptoms and management

RLS is characterized by large brown spots visible on both sides of the lamina that are roughly circular, 1–3 cm in diameter and commonly found at the edges of the leaf lobes or associated with the midribs. On the upper surface, lesions bear a pattern of concentric rings due to the production of pycnidia. These may be washed off older lesions by rain, so that the spots resemble those of diffuse leaf spot. No control measures are known but there may be differences in susceptibility between cultivars as the disease usually affects the younger leaves but has also been observed in the lower canopy in some cultivars.

Ash disease

Causal organism, distribution and spread

The causal organism of ash disease is a powdery mildew fungus, *Oidium manihotis* Henn. and the sexual stage has been described as *Erisyphe manihotis* (Ferdinando *et al.*, 1968). Although this disease is widespread and of common occurrence (Lozano and Booth, 1976), the lesions are superficial and it is not considered to be damaging. The disease is usually observed during the dry season in warmer cassava growing areas.

Symptoms

The first symptoms of the disease are seen as small white patches of mycelium on the upper leaf surface. Beneath the mycelium the cells are killed to form diffuse yellow patches within which pale brown angular water-soaked spots develop and become necrotic. Mature, fully expanded leaves appear to be the most susceptible.

Fungal Diseases of the Stem

Anthracnose

Causal organism, distribution and importance

The causal organism of anthracnose on cassava is described as *Glomerella manihotis* Chev.

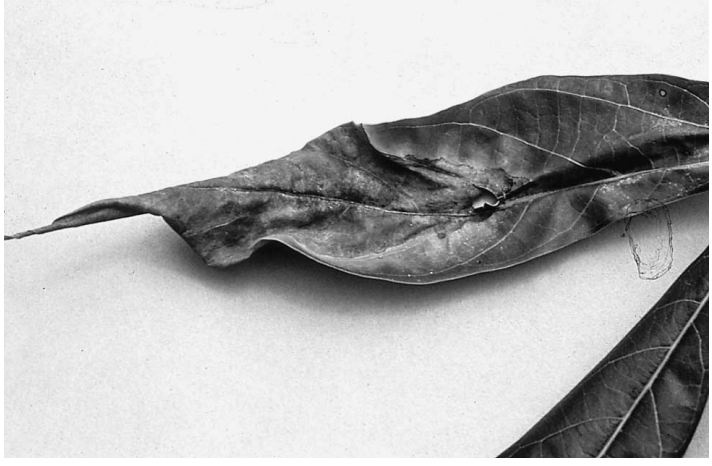

Fig. 13.5. Diffuse leaf spot (*Cercospora vicosae*).

However, the *Colletotrichum* state [*Colletotrichum gloeosporioides* f. sp. *manihotis* Henn. (Penz.) Sacc] is more commonly referred to than the perfect state. The disease occurs worldwide but is more common in the wetter areas of Latin America (CIAT, 1972; Lozano *et al*., 1981) and West Africa (Chevaugeon, 1950; Affran, 1968; IITA, 1972; Akonumbo and Ngeve, 1998). Disease surveys in West Africa showed disease incidence and severity to be greatest in cassava growing in the lowland rain forest, where 64% of all plants examined showed symptoms and the wet savannah zones, decreasing towards the drier ecozones (Wydra and Msikita, 1998). Although the disease is less conspicuous in the dry season, anthracnose causes dieback and affected plants have fewer leaves than unaffected plants. During surveys, extensive areas were observed where cassava crops had been defoliated by anthracnose (Wydra and Msikita, 1998). However, one of the main effects of the disease in Africa is in reducing the quality of stem cuttings as planting material (Théberge, 1985).

Symptoms and management

In Latin America anthracnose symptoms are usually found on the leaves but in Africa, the disease mainly affects the stem. Leaf spots are about 10 mm in diameter and, as with brown spot, they are produced at the base of the leaves. In Latin America, stems may also be attacked, causing a wilt of young plants and cankers on older ones. New leaves produced at the beginning of the rains are reported to be most susceptible (Irvine, 1969). In Africa, the stem canker is the most important symptom but the fungus also causes twig and fruit cankers as well as leaf spots and shoot-tip dieback. Tip dieback was reported to be a common symptom in Ghana (Moses *et al*., 1996). The disease begins on the stems as slightly depressed oval lesions, 1–1.5 cm in diameter and a patch of greenish tissue in the centre of the lesion soon turns dark brown (Fig. 13.6). On older stems, raised fibrous lesions develop into deep cankers, causing brittleness of the stems.

Fokunang *et al*. (1997) have reported that the pathogen is transmitted in cassava seed in Nigeria with up to 40% incidence in some genotypes. Spore dispersal is mainly by wind and water but in Africa, a sap-sucking insect (*Pseudotheraptus devastans* Dist.) is believed to facilitate infection with the fungus by stem puncturing (Boher *et al*., 1983). In the Republic of Congo, anthracnose on cassava stems is associated with the feeding punctures (Makambila, 1994). The fungus then invades the necrotic tissue and infection develops under conditions of high relative humidity. Some cassava cultivars are more sensitive to attack by the insect than others.

There appears to be some variability in the reaction of some cultivars to anthracnose. Screening experiments in Nigeria identified several cultivars in which the onset of first symptoms was delayed until after tuberization had started and as a result, yield was little affected (Ikotun and Hahn, 1994). The parameters considered to be most useful in differentiating the less susceptible cultivars were number of cankers per plant, size of cankers

Fig. 13.6. Anthracnose (*Colletotrichum gloeosporioides* f. sp. *manihotis*) – raised lesions and cankers on cassava stem.

and distance from soil level to the first stem canker.

Glomerella stem rot

This is mainly a problem in stored cassava cuttings and is caused by *Colletotrichum* sp. which may be the same as that causing anthracnose and has been identified as being within the broad grouping of *Glomerella cingulata* (Stonem.) Spauld. Schrenk.

This problem seems to be more common in Latin America than elsewhere (Lozano and Booth, 1976) and cuttings may be predisposed to this infection if they are taken during wet periods. The rot appears at the cut end of the stem and spreads throughout the cutting during storage. The vascular strands become blackened and dark-coloured blisters which contain the perithecia appear on the bark.

Botryodiplodia stem rot

A similar stem rot of stored cuttings to that described above is caused by *Botryodiplodia theobromae* Pat. Symptoms are similar except that the blisters on the bark contain pycnidia instead of perithecia. This disease is frequently found in Nigeria, Benin and Cameroon (Afouda and Wydra, 1996, 1997).

Rust

Rust is caused on cassava by *Uromyces* sp. and has been reported from Brazil and Colombia (Normanha, 1970). Although six rust species have been reported on cassava in different parts of the world (Lozano *et al.*, 1981), rust does not seem to be common in Africa or elsewhere. The orange to reddish brown pustules are most often seen on the green stems but also on the petioles and veins on the underside of the leaf. Occasionally the pustules are surrounded by a yellow halo. Affected leaves may be distorted but the disease is not generally considered of economic importance. Of 485 genotypes screened in Brazil, 139 were highly resistant and 122 showed some resistance (Santos *et al.*, 1988).

Superelongation disease

Causal organism, distribution and importance

Superelongation disease was first reported during the 1970s and appears to be confined to parts of Colombia (Lozano, 1972; Lozano and Booth, 1976). The causal organism was thought to be a species of *Taphrina* or *Sphaceloma*, but is now reported to be *Elsinoe brasiliensis*. The disease is reported to cause considerable losses in plantations where susceptible cultivars are grown (Lozano *et al.*, 1981). Symptoms are more common in the wet season than at other times, when the infection spreads rapidly as spores are distributed by wind and rain. High humidity is required for spore germination.

Symptoms and management

The disease is observed in the field as abnormal elongation of the internodes on young stems which appear thin and weak. Infected plants are taller than surrounding healthy ones. Young shoots, leaves and petioles become distorted and bear eye-shaped cankers appear along the midribs and veins. White, irregular spots may occur on the leaf lamina. There may also be dieback and defoliation. The disease is believed to be spread through planting of infected cuttings so it is recommended in endemic areas to select planting material from disease-free plants and to treat cuttings with captafol solution (3000 p.p.m.; Lozano *et al.*, 1981). Field observations suggest that some cultivars may be resistant (Lozano and Booth, 1976). Cultivars with some resistance to superelongation disease have been identified by CIAT in Colombia. Resistance was associated with thickened cuticles and Bonilla *et al.* (1992) suggested that this could be used to identify resistant genotypes.

Other stem diseases

Cochliobolus lunatus was reported to be the causal organism of a stem disease which was severe on some cultivars in south Ghana and southeast Nigeria. Disease incidence per field ranged from 0 to 80% (Msikita *et al.*, 1997a). A fungal complex was associated with a rot of

mini-stems grown in polythene bags in Nigeria, consisting of *B. theobromae*, *Corticium rolfsii* and *Sphaerostilbe repens* (Osai and Ikotun, 1993).

Fungal Diseases of the Root

Phytophthora root rot

Causal organism, distribution and importance

A number of *Phytophthora* species have been associated with soft rots of cassava roots and these often occur with a number of other soil-borne fungi, particularly *Pythium* spp. and *Fusarium* spp. *Phytophthora drechsleri* Tucker has been reported from Latin America and *Phytophthora erythropseptica* Pethybr. from Africa. Soft rot in general is a worldwide problem and cool wet conditions and root damage predispose the tuberous roots to infection. In areas close to drainage ditches or in poorly drained soils, losses can be up to 80% (Théberge, 1985). In Brazil the pathogens associated with soft rot were identified as *Phytophthora drechsleri*, *Phytophthora richardii*, *Phytophthora parasitica* and *Pythium scleroteichum* (Poltronieri *et al.*, 1997).

Symptoms and management

Young roots initially show water-soaked patches which later turn brown and the feeder roots die. As the rot progresses, the starch-bearing tissue disintegrates and the affected roots have a pungent odour. Root dysfunction causes dieback of the terminal shoots and leads to sudden wilting in advanced stages of root decay.

Cultivars differing in resistance to *Phytophthora* have been identified in Brazil (Lima *et al.*, 1995). Differences in susceptibility were identified using stem inoculation but not using root inoculation. A greenhouse inoculation method was developed based on inoculation of the stem of 40 day-old plants with a plug of mycelial culture of the fungus and their evaluation 7 days later. Only three of 96 cultivars screened were considered to be resistant but a further 23 were moderately resistant.

White thread disease

Causal organism, distribution and importance

White thread is caused by *Fomes lignosus* (Klot.) Bres (*Rigidoporus lignosus* Johansen and Ryv.). Lozano and Booth (1976) state that this is the most widespread and serious root disease of cassava in Africa but that it is less common in Latin America. However, the disease is not described in the IITA field guide to pests and diseases of root crops in Africa (Théberge, 1985). The disease tends to be a problem only in cassava planted immediately after forest clearance or near virgin forest. The fungus survives on dead tree roots and can grow through the soil to attack cassava plants. *F. lignosus* has a wide host range among woody species growing in the humid tropics.

Symptoms

The disease can be recognized by the white mycelial mat under the bark of the roots and by the characteristic white cotton-like mycelial threads on the exterior of the root up to the stem base. Internal tissues exhibit a dry rot which is associated with a smell of rotting wood. Occasionally young plants are affected causing sudden wilting (Lozano and Booth, 1976).

Sclerotium root rot

This root rot is caused by *Sclerotium rolfsii* Sacc., which is common in tropical soils, causing root, crown and stem rot diseases of diverse crops. In the humid tropics, particularly West Africa, *Sclerotium* rot is among the most common diseases of cassava roots (Afouda *et al.*, 1995), generally affecting older plants. In Latin America, it is reported to affect young cuttings and as a surface coating on mature tuberous roots (Martin, 1970; CIAT, 1972). The disease can be identified by the white mycelium on affected roots which sometimes penetrates the epidermis to cause necrosis and occasionally root rot, but this usually occurs in combination with other pathogens. Spherical brown sclerotia may be visible associated with the mycelium on roots close to the soil surface during moist weather.

Dry root rot

Causal organism, distribution and importance

Dry root rot in cassava may be caused by *Rosellinia necatrix* (Hartig) Berl. or *Armillariella mellea* (Vahl.) Pat. (*Armillaria mellea* (Vahl) Fr.), or by both fungi together. Both of these pathogens have been recorded on cassava in different parts of the world, mainly where the crop is growing in moist soils high in organic matter. Both pathogens have a wide host range among woody perennials. Like white thread disease, the dry root rot fungi normally attack cassava planted after forest clearance and can completely destroy the roots of affected plants. However, incidence is normally low and the disease is not regarded as a serious problem. It can be managed by planting annual crops after forest clearance before planting cassava. In the Republic of Congo, *A. mellea* and *Armillaria heimii* were identified, as members of the dry root rot complex in the Republic of Congo. In farms cleared from the bush within the previous 3 years, about half the farms were affected by root rot caused by *A. heimii* but the incidence increased with increasing age of the farms and was 69% in the 2–3-year-old group (Makambila and Koumouno, 1986). Both fungi produce rhizomorphs which appear as thickened mycelial strands on the outside of the roots. They are at first white later turning black. Infected roots are discoloured and exude a watery liquid when squeezed. Rhizomorphs penetrate into the infected tissues. Above-ground plants wilt but do not shed their leaves, eventually desiccating to assume a scorched appearance (Théberge, 1985). Although *Armillaria* has a wide host range, there is evidence for some degree of host specialization. Mwenge *et al.* (1998) showed that in Zimbabwe, where *Armillaria* isolates have been separated into three distinct groups, only isolate from groups I and III were pathogenic to cassava.

Other root rot fungi

Fusarium moniliforme Sheldon, *Fusarium oxysporum* Schlecht and *Fusarium semitectum* were reported from Benin, Nigeria and Cameroon as cause of root, stem and storage rot (Osai and Ikotun 1993; Afouda and Wydra, 1996; Msikita *et al.*, 1996). *F. moniliforme* and *F. oxysporum* were isolated from 44–55% of rotted roots and crowns and from discoloured chips and were re-isolated from cassava plants showing symptoms of wilting and necrosis, 6–10 days after inoculation (Msikita *et al.*, 1996). Lozano (1992) lists *Fusarium solani* (Mart.) Sacc. and *F. oxysporum* Schlecht as contributing to root rots in Colombia. *F. solani* is associated with wet root rots also in Africa where an *in vitro* system has been developed to screen for resistance (Msikita *et al.*, 1997b). Root rot incidence in the humid forest area of the Republic of Congo was 30% in 20-month-old cassava and the pathogens identified were *Armillaria* spp., *S. repens* B. and Br., *F. lignosus* and *Phaeolous manihotis* Heim (Makambila, 1994; Makambila and Koumouno, 1994). In Brazil, a disease known as black rot, affecting roots and shoots has been attributed to *Scytalidium lignicola* Pesante (Laranjeira *et al.*, 1994).

Nematodes

Numerous nematode species have been associated with cassava roots and several of these multiply on cassava roots to reach high populations. Extensive lists of these nematodes have been produced by Hogger (1971), Caveness (1980) and McSorley *et al.* (1983) but there is little evidence that they have a significant effect on yield. Jatala and Bridge (1990) consider that the nematodes with the greatest effect on cassava production are two species of root-knot nematode (*Meloidogyne* spp.), the lesion nematode (*Pratylenchus brachyurus* Filipjev and Strekhoven), the spiral nematodes (*Helicotylenchus* spp.) and the reniform nematode (*Rotylenchulus reniformis* Lindford & Oleveira). In addition, *Scutellonema* spp., particularly *Scutellonema bradys*, are commonly associated with cassava roots in large numbers, although this can also be said for a wide range of crops growing in tropical sandy loam soils. Nevertheless, *Scutellonema* spp. may decrease yields and contribute to postharvest deterioration as they do in other root and tuber crops.

Root-knot nematodes

The most widely reported parasitic nematodes on cassava are the root-knot nematodes (RKN), which occur in Latin America (da Ponte *et al.*, 1980; Crozzoli and Hidalgo, 1992), the USA (McSorley *et al.*, 1983), West Africa (Caveness 1979, 1982; Sikora *et al.*, 1988), East Africa (Saka, 1982; Bridge *et al.*, 1991; Van den Oever and Mangane, 1992) and the Pacific (Bridge, 1988).

The most important species are *Meloidogyne incognita* (Kofoid & White) Chitwood and *Meloidogyne javanica* (Treub.) Chitwood. Galls are produced on cassava roots but there appears to be a wide range of susceptibility to galling between cultivars, ranging from immunity to high susceptibility (da Ponte *et al.*, 1980; Nwauzor and Nwanko, 1989). However, there is no information on the relationship between susceptibility to galling and yield loss. The most widespread severe galling due to RKN was reported from Uganda (Bridge *et al.*, 1991), where 94% of 88 fields examined were affected and 17% of the damaged roots were in the severe category (Coyne and Namaganda, 1994).

Direct effects of *Meloidogyne* spp. on root yield have been difficult to demonstrate. In Uganda yields were found to be consistently lower in fields with greater root-knot damage and for two of the more susceptible cultivars Bukalasa 11 and TMS 30337, yields were decreased by 24–38% (Coyne, 1994). In Nigeria, RKN is regarded as an important pest, causing galls exceeding 1 cm diameter on susceptible cultivars. In severe attacks, the feeder root system is greatly reduced, causing stunting and decrease in stem diameter to which root yield losses of 17–50% have been attributed (Théberge, 1985). RKN has other indirect effects on cassava production. Severe galling causes reductions in plant height and weight which decreases the quality and quantity of planting material (Gapsin, 1980, 1981; Caveness, 1981, 1982). The greatest effect of the nematode may be on the storability of the harvested roots. Caveness (1982) reported postharvest losses of up to 87% due to rapid root deterioration under severe nematode attack.

The evidence presently available indicates that damage caused by root-knot nematodes does not warrant control measures. Studies in Uganda, however, show that losses can be substantial if nematode populations build-up and highly susceptible cultivars are grown. Although yield increases have been obtained in Latin America when nematodes have been controlled by soil fumigation (da Ponte and Franco, 1981), such measures are inappropriate and would rarely provide an economic return and root-knot is best managed by the use of less susceptible cultivars and crop rotation to avoid excessive nematode population increases.

Lesion nematodes

The lesion nematode *Pratylenchus brachyurus* is the second most important parasitic nematode species affecting cassava and occurs on the crop in many parts of the world including the USA (McSorley *et al.*, 1983), East Africa (Coyne, 1994) and Latin America (Charchar and Huang, 1981). Greenhouse tests conducted in Brazil, showed an eightfold increase in populations of *P. brachyurus* after 3 months and a gradual decline in production over several years was attributed to the nematode (Charchar and Huang, 1981). McSorley *et al.* (1983) reported yield increases after nematicidal treatment of fields where cassava was affected by a nematode complex consisting of *P. brachyurus* and *Helicotylenchus erythrinae* (Zimmerman) Golden. If necessary, control can be achieved with resistant cultivars as it has been shown that there is considerable variability between cassava cultivars in susceptibility to *P. brachyurus* (Corbett, 1976).

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