Review of disease distribution and pest status in Africa

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Introduction

Bananas are of great socioeconomic importance in moist tropical and subtropical Africa. Their all year-round fruit production ensures continuous supply of food and income to the farmer, making them a major food security crop in the region. Compared to other staples, bananas are the most economical source of carbohydrates in terms of cost per hectare, per ton and per calorie (Swennen 1984) and among the major sources of potassium, calcium and phosphorous (INIBAP 1986). They yield diverse goods from sweet fruits to staple starches as well as numerous useful secondary products, such as fibres for handicraft and wrappers. On steep slopes, they control soil erosion and conserve soil fertility. In highly populated and dry regions of Africa or semi-urban areas, banana peels and pseudostems serve as animal feeds (INIBAP 1986). In turn, these animals provide manure which is used to improve soil fertility. However, banana productivity has failed to keep pace with increasing food demand despite the steady increase in banana acreage over the past 30 years. The decline in yield, attributed to declining soil fertility, pests, diseases and socioeconomic problems, has aggravated the food deficit situation.

Diseases constitute one of the most important production constraints. The major diseases limiting banana productivity in Africa are: Panama disease (Fusarium wilt), black Sigatoka, leaf speckle, banana bunchy top virus and banana streak virus diseases. This paper reviews the distribution and pest status of these diseases in Africa and highlights key information gaps.

Panama disease (Fusarium wilt)

Distribution

The disease caused by *Fusarium oxysporum* f.sp. *cubense* (FOC) is mainly transmitted through infected planting materials. It was first recorded in Australia in 1874 but

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initial extensive studies were made by Brandes in the late 1910s (Jeger *et al.* 1995). In subsequent years, the disease was recorded in Hawai, South America, Asia and West Africa. By 1955, the disease had been recorded in most East and Central African countries (Stover 1962). The disease appears to have been present in most of these areas for quite sometime before recognition but was most likely introduced on planting materials. The disease is now widespread in Africa, and virtually occurs wherever susceptible cultivars are grown.

Three races of the pathogen have been recorded in Africa. Race 1 of the pathogen (as indicated by Gros Michel attack) is the most abundant though occasionally race 2 (indicated by Bluggoe attack) is found in isolated pockets. Race 4 of the pathogen has been recorded only in South Africa and Canary Islands. In West Africa the disease is less important because plantains, which are the dominant varieties, are resistant. However, it is likely that the pathogen may be present in spots where susceptible clones were once grown.

Studies on variability of the pathogen using vegetative compatibility testing (ability to unite and form heterokaryons) have recently been initiated. Such studies shed light on how the pathogen population changes and new races evolve. According to these studies, the pathogen populations in Africa have been assigned to the VCGs as indicated in Table 1 but the sampling has not yet covered the whole continent.

Table 1. Presence of populations (VCGs) of *Fusarium oxysporum* f.sp. *cubense* in African countries.

Country	Vegetative compatibility groups (VCG)	Source
Burundi	0124	Ploetz 1990
	0124/0125	Ploetz *et al.* 1994.
DR Congo	0125	Ploetz 1990
Kenya	01212, 0124, 0125, 0128, 01220	Kung'u *et al.* 1997.
Malawi	0124/0125	Koenig *et al.* 1997
	0124, 01214	Ploetz *et al.* 1992
Rwanda	0124	Ploetz *et al.* 1994
	0124/0125	
South Africa	0120	Ploetz 1990
Tanzania	0124	Ploetz 1990
	01212	
Uganda	01212, 01222, 0124,	Kangire 1998
	0124/0125	Ploetz 1990
	0125	Ploetz *et al.* 1994
Comoros Islands	0128	Ploetz 1990
Canary Islands	0120	Ploetz 1990

Another key gap that requires urgent attention is linking these populations to pathogenic variability. It is not clear if the identified populations differ in pathogenicity. It is important to establish the different pathogenicity groups so that screening germplasm for resistance could target all the pathotypes.

Pest status

The importance of the disease is influenced by the cultivars in use. East African Highland bananas (AAA), plantains (AAB), Cavendish and several recently developed hybrids are resistant to race 1 and 2 of the disease. Gros Michel (AAA), Apple banana (AB), Pisang awak (ABB) and several others are susceptible to the two races. All banana clones except a few recently developed hybrids (eg. FHIA-01) are susceptible to race 4 of the pathogen.

Where resistant cultivars are in use, the disease is considered of minor importance. However, where susceptible cultivars are in use complete decimation of fields (100% loss) has been reported. Farmers have turned to new cultivars or new crops in such areas. Where farmers have continued using the susceptible clones, the main reason appears to be the absence of a suitable replacement. For instance Apple banana is used to supply niche markets in Europe and fetches good money to farmers. Preventing spread of the disease as production of disease-susceptible bananas expands is a challenge in East and Central African countries.

Virgin soils appear to be free of the pathogen. Production of susceptible clones such as Gros Michel and Apple banana is still possible in such areas, provided clean planting materials are used.

Use of host plant resistance or clean planting materials are the only control measures currently being tested in various countries.

Black Sigatoka

Distribution

The disease, caused by *Mycosphaerella fijiensis*, is windborne. It was first described in Fiji in the early 1960s (Rhodes 1964) and is considered to have originated in Papua New Guinea/Solomon Islands region (Stover 1978). Subsequently, the disease spread to other banana-growing areas across the globe.

In Africa, the disease was first reported in Gabon in 1978 (Frossard 1980). It was later reported in Cameroon in 1980, Nigeria in 1986 (Mourichon and Fullerton 1990), Burundi and Rwanda in 1986, Tanzania in 1987 (Dabek and Waller 1990), and Uganda in 1989 (Tushemereirwe and Waller 1993).

Recent studies in Uganda suggested a mean minimum temperature threshold of 14-150C below which the disease fails to establish in the field (Tushemereirwe 1996). This suggests that the high elevation areas (above 1500 masl) in countries where the disease has been reported are likely to be free of the disease. Similarly the cool countries of southern Africa (high latitude countries) are likely to remain free of the disease.

Recombination of genes leading to new populations occurs easily in the black Sigatoka pathogen because it has a perfect stage. Consequently the pathogen is genetically highly diverse (Buddenhagen 1987). This diversity is reported to be highest in the South East Asia (centre of origin of the pathogen) and lowest in Africa where the pathogen

arrived recently (INIBAP 1998). There is some evidence that some of the populations are pathogenically different (Fullerton and Olsen 1995) but the pathotypes in Africa and their distributions have not been established.

Pest status

Worldwide, black Sigatoka is currently considered the most important disease of bananas and plantains (Jeger *et al.* 1995). Although the disease does not usually kill the plant, it causes heavy defoliation which severely suppresses finger filling, leading to reduced bunch weight. The East African Highland bananas (dominant in East and Central Africa) and plantains (dominant in West Africa) are all susceptible to the disease. This further increases the importance of the disease in Africa. A yield loss trial conducted in a low elevation plantain system of West Africa (Nigeria) revealed a loss of 39% in bunch weight (Mobambo *et al.* 1993).

A similar trial conducted in the mid-elevation banana systems of eastern Africa (Uganda) revealed a loss of 37% in bunch weight in the first ratoon (Tushemereirwe 1996). The two sites of the trials represented ecological extremes for the disease and it is likely that all areas in the same ecological conditions or between the two extremes would suffer similar losses.

The Ugandan study was situated at Kawanda, at 1250 masl. However, the disease can still be observed up to 1450 masl. It is not clear how much loss the disease causes in the areas where the disease tails off. For instance in the Bukoba region of Tanzania (which is above 1250 masl) the disease is dismissed as minor in importance though yield loss studies to clarify the issue have not been undertaken.

Control measures: use of host plant resistance is identified as the most suitable technology, but for some cultivars such as Highland bananas, resistant hybrids are not yet generated. Other measures being tried include the use of plant vigour to reduce disease impact and removing diseased leaves to reduce inoculum.

Banana bunchy top virus disease

For a long time this is the only virus disease that was considered important on bananas (Jeger *et al.* 1995). The disease was first reported in Fiji in 1889 (Jeger *et al.* 1995). It has since been confirmed that the disease is present in several Asian, Pacific islands and African countries. In Africa the disease has been reported in Egypt, Congo, Rwanda, Burundi and Malawi.

The virus is disseminated in infected planting materials. Within the field, it is also transmitted by the banana aphid (*Pentalonia nigronervosa*).

Pest status

In Africa, no study to establish yield loss due to the disease is reported. However, severely infected plants of highly susceptible cultivars fail to produce bunches (Jeger *et al.* 1995). The severity of infection depends on virulence of the virus strain, susceptibility of

the cultivar, and stage of infection. Tolerant cultivars and those recently infected or with an avirulent strain will have mild infection and will give some yield.

Key information gaps

a) The disease appears restricted to the Rift Valley areas/a lowland stretching in central and southern Africa. Factors restricting distribution of the disease should be established.
b) There is need to quantify the losses caused by this disease. A yield loss study carried through several cycles to account for the cumulative effect of the disease would yield useful information.
c) There is need to establish the pathogen strains and their distribution so that future germplasm screening studies can target them.

No resistant cultivar has been identified. However, varietal differences in susceptibility have been reported (Stover 1972).

Control measures in use include:

- Prevention through quarantine: there is need to prevent the virus from entering free countries or areas where the distribution is still limited to a few zones.
- Use of virus-free planting materials: this can be achieved by starting clean mother gardens using virus-indexed plants. The alternative is to identify clean plantations from which suckers should then be obtained for more plantings.
- Roguing: this involves removing (and where possible, burning) all the infected plants. This may keep the disease incidence low if done regularly.
- Varietal resistance: the search for tolerant cultivars should be intensified. If found, these would be used as replacements for the highly susceptible clones.

Banana streak virus disease (BSV)

The disease is believed to be worldwide in distribution (Lockhart and Olszewski 1993). It was first described on bananas in Côte d'Ivoire (Lassoudière 1974) but the causal organism was not identified until 1985 (Lockhart 1986).

Since then the disease has been reported in Asia, Central America and other African countries: Morocco, Nigeria, Rwanda, South Africa, Tanzania (Lockhart and Jones 1993), Uganda (Tushemereirwe *et al.* 1996), Malawi (Vuylsteke and Lockhart 1997), Guinea, Ghana, Benin, Cameroon, Kenya and Madagascar (Jones and Lockhart 1994, Diekmann and Putter 1996).

It appears that BSV has been widely distributed for many years but has always been confused with other viral diseases, particularly cucumber mosaic virus. This is supported by the fact that after the disease was identified, it was recorded in most banana-growing areas in a very short time. The origin of the disease is not known (Frison and Sharrock 1998).

Recent molecular studies have revealed that there are three forms of BSV:

a) encapsidated episomal form; this is the ordinary form of the virus with the DNA viral genome encapsidated in a protein coat;
b) unencapsidated episomal form; this is thought to be a form characterized by periodic appearance and disappearance of host symptoms;
c) integrated forms; recent conclusions suggest that there are some forms of BSV which are integrated in the banana genome. These appear to be activated by certain stresses such as tissue culture to give rise to the infectious episomal forms. BSV reported in previously indexed clones but subsequently multiplied by tissue culture, mostly likely belong to this form.

Information on the relative importance of the three forms is still lacking. New methods for detection of BSV are being developed but most national programmes have not yet acquired the capacity to use them. This has severely hampered generation of information on BSV distribution within the countries. Virus strains are believed to exist but there is no information on them yet.

Pest status

BSV appears to have been around for many years but it has never caused widespread epidemics (Frison and Sharrock 1998). However, the disease has caused significant yield loss in localized places. For instance, some fields were knocked out of production in Rakai district, Uganda (Tushemereirwe 1996).

There is no published information on yield loss due to the disease and its economic impact. However, loss for individual plants may go up to 100% depending on susceptibility of the clone, severity of the disease strain and age of infection.

The pest status of the disease appears to vary with clones though none has been found resistant. For instance in Uganda, Pisang awak (ABB) exhibited only mild infections in severely infected mixed clones at "hot-spot" locations.

Other diseases

Fungal diseases

Yellow Sigatoka

This is an airborne disease caused by *Mycosphaerella musicola*. It was first observed in Java in 1902 (Stover 1962b) and thereafter it was reported in Asia, Africa and the Americas. In Africa the disease was first reported in Uganda in 1938 and was later quickly noted in Tanzania in 1939, Cameroon 1941 and thereafter in several other African countries. The disease is now reported present in all tropical Africa. The incidence of the disease is highest in high elevation systems where black Sigatoka is absent.

Though the disease was reported as important in the Americas and Caribbeans even before arrival of black Sigatoka (Stover 1972) there is no data on its importance in Afri-

ca. It has been reported that wherever black Sigatoka has arrived, it has completely or partially displaced yellow Sigatoka (Mourichon and Fullerton 1990) within two years (Jeger *et al.* 1995) though some doubts have been expressed about this phenomenon (Jones 1990). In Uganda, the observation appears to conform to the phenomenon for susceptible cultivars but not for resistant cultivars. Yellow Sigatoka is found on the resistant Kayinja (Pisang awak = ABB) at all elevations but rarely on the infected susceptible clones (Tushemereirwe 1996). The disease is most pronounced in high elevation systems where black Sigatoka has not established. However, its pest status in such systems is yet to be determined.

Leaf speckle

This disease is caused by a windborne fungal pathogen, *Periconiella sapientumicola*.

It is reported present in almost all banana growing areas. According to Stover (1972) leaf speckle has always been considered a minor disease that affects older, mature leaves of bananas growing in humid areas. As a result, there has been little research interest in the disease, leading to absence of key information. Highland bananas appear to be susceptible to the disease (Tushemereirwe 1996). The disease heavily defoliates the bananas even in the absence of Sigatoka leaf spots.

Unfortunately, it has not been possible to determine pest status of the pathogen in the absence of other leaf spots. Such a study should be possible in an area where black Sigatoka is absent on highland bananas. In Uganda such areas have extremely low incidence of yellow Sigatoka. About 95% of defoliation is due to leaf speckle.

Matooke wilt

This disease has been reported only in Uganda where it is traced back to about 1955. Highland bananas (AAA), known to be resistant to Fusarium wilt, were found to succumb to a wilt disease in western Uganda in areas above 1330 masl (Tushemereirwe and Ploetz 1993). Initial studies had attributed the disease to *Fusarium oxysporum* f.sp. *cubense* (Ploetz *et al.* 1994) but a recent study appears to suggest this may not be the causal agent (Kangire 1998). The disease is virtually restricted to areas around the homesteads, garbage dumping sites and animal kraals. It is not clear what the impact of the disease will have as fields become less fertile and more organic materials are used. The disease will require more monitoring on top of identifying its cause.

Other minor diseases

Other minor diseases include bacterial pseudostem rot (*Pseudomonas* spp.) and bacterial corm rot (*Erwinia* sp.). The fungal diseases include cordan leaf spot (*Cordana musae*), canana leaf freckle (*Guignardia musae, Mycosphaerella musae*), deightoniella leaf spot (*Deightoniella forulosa*), banana rust (*Uromyces musae*), fruit freckle (*Phyllostictina musarum*), cigar end rot diseases (*Stachylidium theobrome, Trachysphaera fructigena, Gloeosporum musarum*), crown rot (assortment of pathogens), anthracnose (*Colletotrichum musae*) (Waller *et al.* 1991, Tushemereirwe 1996). The viral diseases

include banana mosaic (cucumber mosaic cucumovirus), banana die-back (banana die-back virus reported in Nigeria) (Diekmann and Putter 1996).

Conclusion

In conclusion, it is noted that for all the important pathogens of bananas, there is lack of information on pathogenic strains and their distribution in Africa. This information is a prerequisite for effective deployment of host plant resistance as a disease control measure. Resistant clones for use as replacements for the susceptible clones should be tested against all the strains or should not be used in areas with a strain to which they succumb. Furthermore, it is noted that there are several diseases whose economic impact is not clear. These include banana bunchy top virus and banana streak virus diseases which are reportedly important in some areas but minor in others, and banana leaf speckle of highland clones and matooke wilt which have for some time been regarded as minor but appear to be severely damaging in some locations. There is need for hard data to clarify the importance of these diseases.

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