# 1

# Symptomatology and Economic Importance

*Jean-Luc Renard*

The term disease applied to a plant expresses an abnormal state of the whole or a part of the plant due to the influence of the environment. Parasitic diseases, in contrast to physiological diseases which are caused by physical factors (especially soil and climate), are caused by the aggression of an organism which is both parasite as well as pathogen. This change, which may be temporary, permanent or fatal depending on the case, has the potential of becoming contagious and developing into an epidemic. This phenomenon is the result of direct contamination, gradually getting closer and closer, or through the mediation of a vector of the pathogen. Fungi, bacteria, phytoplasmas, trypanosomas, viruses, viroids and nematodes are responsible for causing the diseases presented in this work.

The tropical environment, with its hot and humid seasons, which may be more or less prolonged depending on the regions, is favourable for the development of parasites, especially fungi and bacteria. These organisms go into the resting stage in various resistant forms (chlamydospores, oospores, cysts, etc.) during dry periods. When the conditions become favourable again, the parasite resumes its activity and sporulates profusely. These spores are easily disseminated by wind and rain at a time when the plant is also extremely susceptible to infection (liquid water on the surface of the leaves, open stomata, etc.). In contrast to annual crops which are absent during a part of the year, perennial crops remain standing and constitute a permanent storage host of the parasites, which serve as the primary inoculum when infection recommences with the onset of rains, Regions without a real dry season are particularly favourable for the development of pathogens.

Symptoms of parasitic diseases are of various kinds: necrotic spots, rots, wilts, blights, etc. They depend on the type of parasitic activity and the part that is infected. Disease incidence on the production is variable. It depends on the part that is affected, intensity of the symptoms and the pathogenic ability

of the causal agent organism. Death of the tree is the ultimate stage in the evolution of symptoms. It may be sudden or result from a slow and general decay. In both cases the damage caused to the productive capital is irreparable. Infection of fruits results in low yields and often changes the quality of the produce. Diseases of leaves and branches reduce the vigour of the plant and affect the yield. In some cases, repeated infections on the leaves weaken the plant and ultimately lead to its death.

Thus, the heavy investment made by the agriculturist by planting a tree crop, whichever it may be, could be quickly compromised or even wiped out.

This is why a planter should be specially vigilant with respect to the health of the crop, so that he can do everything possible to arrest the development of an epidemic at the right time. Moreover, he should ensure that the plant material selected by him has, as much as possible, all the characteristics for resistance to a disease present in the region under consideration.

The first part of this chapter will be devoted to the description of the various symptoms caused by pathogenic organisms on tropical tree crops. The consequences of the disease on the vegetative growth of the plant and on its production will be indicated in function of the symptoms expressed. Because of the diversity of the pathogens as well as of the concerned crops and for a coherent presentation, the affected plant parts are presented in the same order throughout this chapter.

## GENERAL DECAY

These diseases inevitably lead to the death of the plant more or less quickly. All kinds of pathogens may be responsible for general decay.

### Diseases caused by fungi

In the quasi-totality of cases, the pathogenic organism grows in the soil. It attacks the plant through the roots or is transported through the vessels which get blocked; nevertheless, it can still be transported to different parts of the plant.

#### Rot diseases

The causal pathogens have similar life-cycles and attack the roots and then the trunk of a large variety of trees.

##### White root rot of rubber tree

The name of this disease is derived from the white mycelium filaments or rhizomorphs which the fungus, *Rigidoporus lignosus* (Klotzsch) Imaz (Basidiomycetes, Polyporaceae), commonly called *Fomes*, produces in the form of a network on the roots and collar of rubber trees. It is possible to see this on an

infected tree by clearing the roots and collar of the soil covering them. When the parasite is already well established in the woody part of the roots and base of the trunk, the rubber tree shows yellowing and shrivelling of the leaves (photo 1). The leaves turn reddish-brown before falling off. These symptoms are accompanied by unseasonal flowering. Sometimes the tree gives out new leaves which are smaller and lighter in colour than the leaves of a healthy tree. Defoliation is followed by a gradual wilting of the branches and death of the tree. Fruiting bodies of *Fomes* can be seen at the base of the trunk before the shrivelling of leaves and wilting of branches (photo 2).

These fructifications are in the form of semi-circular brackets, yellow to orange on the upper side and lighter in colour on the lower side. At this stage, the taproot and base of the trunk are partially or completely rotten and the tree falls down with the slightest wind.

*Fomes* is a root parasite on a large number of forest tree species. As soon as the forest is felled, the conditions become favourable for the growth of *Fomes* which invades the stumps of the felled trees. These constitute the primary sources of infection which infect the roots of the rubber tree. Infections by *Fomes* appear from the end of the first year of the plantation. The disease then gradually spreads from a diseased tree by the transmission of *Fomes* from infected roots to healthy roots. Once again, primary sources could be formed when the roots come into contact with infected stumps of forest trees.

Mortality increases every year, attaining a maximum towards the sixth year (6% annual mortality). A regression in the disease rate is then observed and the evolution of the disease becomes stabilised at about 1-2% mortality per year. The development of disease centres creates a clearing in the plantations, as a result of which the trees are easily broken by wind leading to considerable losses (Tran Van Canh, 1996) At the age of about twenty years, some infested plantations have no more than 200 trees per hectare, i.e., less than 50% of the trees planted initially.

### Crack rot of rubber

Attacks by *Armillaria* on rubber are manifested by the flow of latex through deep cracks in the bark on the collar and base of the trunk (photo 3). The cortical epidermis becomes grey and dry and shows shallow cracks. Superficial scraping reveals black spots due to necrosis of the primary epidermal cork layers. The bark is reddish brown in colour and encloses a white mycelial mass intermixed with filaments of coagulated latex (photo 4). A white mycelial mass is also seen on the cortical layer.

When the infection is at an advanced stage, the foliage becomes yellow and takes on a reddish copper-red tinge. Defoliation follows and the branches become dry, eventually leading to the death of the tree (Guyot, 1997).

The disease is caused by *Armillaria heimii* (Basidiomycetes, Agaricales) which grows on the roots and taproot of the tree. The tree reacts to the infection by producing a mantle of irregular black rubbery substance that

grows around the roots, amalgamated with the mycelium of the parasite. It is possible to detect the early stage of the disease by removing the soil covering the roots at the base of the tree (Michels, 1990; Petit-Renaud, 1991).

The economic impact of *Armillaria* on rubber cultivation is known only in the commercial plantations in Gabon, where the sanitary situation is regularly supervised. The annual infection rate between 3 and 5 years does not exceed 2.5% of the trees present and the mortality rate is only 2.1 trees per hectare per year. This represents just 10-30% of the total mortality due to rots, the most destructive causal organism being *Rigidoporus lignosus* (Guyot, 1997). In village plantations this rate seems to be higher but no precise data is available.

## Basal rot of oil palm

Rot disease in oil palm is caused by a Basidiomycete fungus, *Ganoderma lucidum* (Leyss. ex Fr.) Karst. Depending on the advancing stage of the parasite at the base of the trunk, the palm exhibits a slackening in growth which is expressed by an accumulation of unopened leaves and sometimes the petioles of the lower still green leaves break, thus forming a kind of 'skirt' around the trunk. This disease can be recognised as soon as brown carpophores appear at the base of the trunk (photo 5). In the young stage, these fructifications are characterised by white protuberances on the surface of the trunk and evolve into brown brackets. The upper surface of these brackets is shiny and more or less dark, while the lower surface is white to whitish with a porous aspect. At this stage, the interior of the trunk is invaded by the mycelium of the parasite; the tissues have a more or less spongy consistency and become whitish or yellowish in colour.

The disease is found mainly in oil palm plantations in Indonesia and Malaysia. It also devastates plantations in Africa, the Democratic Republic of Congo, Cameroon and Nigeria. Symptoms have been observed in plantations established in forest clearings. The palms suffer considerable damage in the second and third generations. Under these conditions, the first manifestation of the disease could take place from the second year after replanting and the damage becomes devastating from the age of 10 years, sometimes with 50% of the palms infected or dead. An infected tree can survive and continue to yield for two to three years after the appearance of the first carpophores. However, it eventually falls down when the entire base of the trunk is invaded by the parasite. Umar Akbar *et al.* (1971) have reported the influence of mineral nutrition and plant material on the behaviour of oil palm faced with the presence of *Ganoderma*.

Besides field observations and their cartographic representation, the occurrence of *Ganoderma* can be estimated by remote sensing. By calibrating radiometric measurements, in liaison with agronomic parameters from the field and appropriate interpretation of Spot images, a satisfactory relationship can be shown between the percentage of dead trees and leaf area index

(LAI) and reflection measurements expressed as vegetation index (Naert *et al.*, 1990).

By eliminating as much dead wood (stumps, large roots) as possible before planting, preventive control against this disease is largely inspired by that which is practised in rubber plantations.

### Rots on coffee, cocoa and tea

Several rots affect the roots of coffee, cocoa and tea plants.

Decaying symptoms on coffee plants, due to rot caused by *Clitocybe (Armillaria) elegans* Heim, are characterised by cracks in the collar and base of the trunk accompanied by longitudinal splitting of the roots. Mycelial fan-shaped patches develop under the bark and the cracks in the wood are invaded by thick xylostroma which are responsible for the splits observed. The fruiting bodies of the fungus arise mainly from the xylostroma at the base of diseased trees (photo 6). Shade trees such as *Albizzia malaccocarpa* are also infected by *C. elegans* (Muller, 1959).

On cocoa trees, white rot *(Leptoporus lignosus* or *Fomes)* has been observed in the Ivory Coast, whereas *Armillaria* attacks are seen in the northern (Blaha, 1982) and central (Blaha, 1989) parts of Gabon. Trees with infected taproots collapse suddenly; others show the characteristic radial fissures in the collar region preceding a general wilting of the foliage (photos 7 & 8).

As in all afflictions of this type, these attacks lead first to a fall in yield and then to the death of the sick trees. This results in the loss of the productive potential and investment.

A number of authors have reported the presence of rot disease on coffee plants in Africa. Blaha (1978) has proposed the measures to be adopted to reduce the incidence of rot on coffee cultivated at high elevations in Cameroon, viz., regular disinfection of the disease centres.

The polyphagic nature of these fungi complicates their control: before planting, it is suggested that a crop called "cleansing" crop be cultivated for several years, for example, a herbaceous species capable of completely decomposing the woody debris. It is also recommended that coffee and cocoa should not be planted after a rubber crop, nor in association with cassava.

Rot diseases are also common on tea (Bonheure, 1988). *Armillaria mellea* (Vahl) Pat. causes radial and longitudinal cracks near the collar and base of trunks, which are responsible for splitting of the wood and hence the name "collar crack" given to this disease.

White mycelial forming fan-shaped patches develop between the bark and wood and on the surface of the roots. The fungus is propagated mainly through the black rhizomorphs which can spread in the soil.

*Rosellinia arcuata* (Petch) can be recognized by the presence of a mycelial veil on the surface of the collar. The tea plant reacts by excessive growth of the bark forming a cushion-like structure and produces a large number of adventitious roots.

Other parasitic fungi such as *Phellinus lamaensis* (Murr) Heim, *Poria hypolateritia* (Berk), *Ustulina deusta* (Fr. Petrak), *Sphaerolstilbe repens* (B. Br.), are among the organisms responsible for root diseases of tea plants.

Phthiriasis or false rot of coffee, which presents itself in the form of a thick tube of crusty mycelium enveloping the collar, should not be confused with the "true" rot infections described earlier. This disease, which is linked to the development of bugs, is treated in the chapter on insect vectors.

## Rots of tropical forest trees

Teak (*Tectona grandis*) is one of the most susceptible species to rot diseases. Attacks by *Rigidoporus lignosus* observed in teak plantations in the Ivory Coast are difficult to detect. The diseased trees usually fall down following a gust of wind or after a clearing done to accelerate the growth of the still standing trees (Mallet *et al.*, 1985). It appears that the tree, whose taproot is seriously infected by *R. lignosus*, strengthens its anchorage by developing lateral roots, which enables it to conserve its foliage. Nevertheless, when the infection of the root system becomes generalised and despite the protection provided by neighbouring trees against wind, the tree ultimately dies standing. Prior to this stage, a careful observation will help to detect the presence of rhizomorphs as whitish fan-shaped patches under the bark in the collar region. The first symptoms of attack by *Armillaria* and *Phellinus* on one or several large roots (photos 9 & 10), are expressed by a yellowing and premature but progressive leaf fall. *A. heimii* and *P. noxius* (Corner) G.H. Cunn. are the species most commonly implicated in basal rots. *Phellinus* can be recognized by the presence of cylindrical blackish rhizomorphs in contact with the roots. In the case of *Armillaria*, the yellow to whitish rhizomorphs, which look like a spider's web, grow on the roots. *Phellinus* (and sometimes *Armillaria*) forms a blackish and granular crust under the bark of the roots; on the other hand, *R. lignosus* is characterised by whitish fan-shaped patches. These parasites cause the wood and roots to rot. The rots which are spongy in a humid environment and fibrous in drier conditions (Lanier *et al.*, 1976), disturb and completely block the water and nutrient uptake of the tree.

In the forest, rots develop on the roots and collar of trees and gradually destroy the root, bark and wood. Disease symptoms vary depending on the parasite involved and the plant species infected.

Under natural conditions and in forest plantations, the first signs of rot infection are not easy to spot because the crowns are at a great height and overlap one another. Moreover, in moist forests the trees lose their leaves and replace them throughout the year. When the parasites spread to all the roots and go up the trunk, the bark cracks and peels off the trunk. At this stage there is a generalised leaf fall, followed by drying up of the branches. The tree dies one to three years after the onset of the attack (Arbonnier, 1995). Diagnosis is not always easy and other factors should always be taken into consideration (*Phytophthora* infection, influence of drought and root asphyxiation, attacks

by xylophagous insects) to identify the cause of decay with a certain degree of certainty. Baiting with sticks of tender wood (*Hevea, Gmelina...*) embedded near the foot of diseased trees or girdling the collar of trees with a mantle of grass facilitates identification of the rots implicated in the decay (Samasserou *et al.*, 1981).

The economic impact of rot infections on tropical forest trees is generally considered to be low, considering the mode of exploitation practised. Management of plantations involves the removal (during their growth and hence spaced over time) of a majority of the trees planted as they are growing. For example, in a teak plantation in the Ivory Coast, the trees are planted with a density of 2000 plants per hectare and by the end of twenty to thirty-five years they are gradually brought to a final density of 100 to 125 stems per hectare. If the infection takes place at a young age, the contaminated trees are cut down and integrated in the quota of trees that have to be felled during the clearing operation following the date of observation of the attacks. If the attacks take place at a time when the trees are almost mature, the loss is generally less than 10%.

Treatment of the damages caused in plantations involves the implementation of large-scale and costly measures in terms of labour (necessary for drawing up inventories to localise and quantify the attacks, estimate the quantity of fungicides required...), materials (tractors with tanks and trailers) and chemicals. Treatments against rots are therefore rarely undertaken because the expenses are generally higher than the amount of losses.

In natural forests, the distribution of commercial and exploitable trees is from one to a few trees per hectare. They are very rarely accessible except on foot. In a forest of several thousand hectares, the loss of a few trees is generally considered to be negligible and the difficulties in accessing them make it almost impossible to treat them.

## Systemic diseases

### Vascular wilt of oil palm caused by *F. oxysporum* f. sp. *elaedis*

Fusarium wilt of oil palm is the most serious disease of this crop in West and Central Africa. The causal organism is *F. oxysporum* f. sp. *elaedis* (Schlecht) Toovey, which enters through the roots and then invades the vascular system.

Expression of the disease varies depending on several factors, including the age of the palm, the place it occupies in the crop cycle, stage of infection and degree of susceptibility of the tree itself (Renard and Franqueville, 1989). Three categories of symptoms can be distinguished schematically.

At a young age, fusarium wilt is expressed by a yellowing, often unilateral and then browning and drying of the middle leaf of the crown (photo 11). This symptom spreads to the neighbouring leaves and then to the lower leaves. Death may occur by a general drying up of the plant two months after the appearance of the first symptom. However, we sometimes come across trees

of resistant progenies with partial or even total remission of symptoms, the initial symptom of yellowing remaining localised without further evolution. A section of the trunk shows brown vascular fibres which are characteristic of the disease, whether or not there is remission (photo 12).

On bearing palms, the typical (or extreme) symptom of the disease is characterised by the yellowing of a middle leaf, followed by the sudden drying of the middle and lower leaves and breaking of the rachis at about one-third distance from the petiole (photo 13). This drying may be generalised or may affect only a section of the palm. Death of the tree may occur three to four months after the appearance of the first symptoms.

In case of remission, the dry lower leaves fall, the tree gives out two, three or four spear leaves which open very slowly. The trunk becomes narrow and pointed and looks like a pencil (photo 14). A few bunches may be produced sporadically. The tree often dies many years after the appearance of the symptoms when the occasion presents itself, for example, when there is a drought. These palms always contain brown fibres which are evidence of the presence of the parasite. Besides, observations have shown that there are palms which seem to be perfectly healthy while they actually harbour the parasite.

Disease incidence varies depending on the region. Sandy soils are favourable for the development of vascular wilt, but the disease is also found in soils of volcanic regions, for example in Cameroon.

In the first generation, vascular wilt is a disease of adult plants in savannas as well as in forests. The first symptoms appear only on palms which are 6 or 7 years old. On the other hand, in replantations, vascular wilt appears from the first year itself on susceptible progenies. It has a tendency to become stabilised in the fourth or fifth year, when it begins to develop by spreading. These phenomena are due to differences in the infective potential in these two situations.

In the Ivory Coast, for a long time it was thought that the incidence of vascular wilt of palm on a savanna antecedent was higher than that observed on a forest antecedent (Renard and Quillec, 1984a). Franqueville (1991) observed that large infection centres developed in old forest zones, the percentage of infected palms increasing from 2 to 20 from 1984 to 1987. Initially confined to Africa, fusarium wilt is now found highly localised and without economic incidence in Latin America. The Asian continent remains unscathed by the disease.

It is difficult to assess the loss in yield caused by vascular wilt considering the compensation phenomenon observed in a healthy palm standing near a dead tree. Nevertheless, Renard and Ravise (1986) have shown that these losses are in the order of 0.9% by weight of fresh **fruit bunches** for 1% of the palms showing external symptoms of fusariosis. The fall in yield may reach 30% in 15 to 20 years.

This assessment was specified recently by Renard *et al.* (1993) thanks to a

study which took into account the different kinds of symptoms, whether or not they were expressed, as the trunks of healthy looking palms had been bored in order to look for the eventual presence of brown fibres in them . Two categories of plant material, C1001 and C1401 (the former more tolerant than the latter), reproducing known crosses and representatives of a widely distributed material, were analysed. Figure 1 shows that in the case of the C1001 category, the yield of palms with remission of symptoms (R) and of trees infected but without symptoms (f) is the same as that of healthy palms (H). The yield of palms, showing symptoms (F), is significantly lower. In the case of Category C1401, palms whose symptoms have disappeared (R) and those showing symptoms (F) have a significantly lower yield than healthy trees (S) and trees with latent infection (f), the yield in the last two being more or less equivalent. The phenomenon of remission of symptoms thus varies in function of the tolerance of the category under consideration, especially in proportion to the duration of the expression. For category C1001, the evolution of the yield of trees where the symptoms were only temporary is traced in Fig. 2. These yields are expressed in relation to that of healthy palms, which thus constitute the base 100 of the results. Class R1 represents palms which showed symptoms in only one survey, R2 in two successive surveys and R3 in three surveys. It is quite evident that the fall in production is proportional to the duration of the manifestation of symptoms and that this effect is more evident in susceptible than in resistant material.

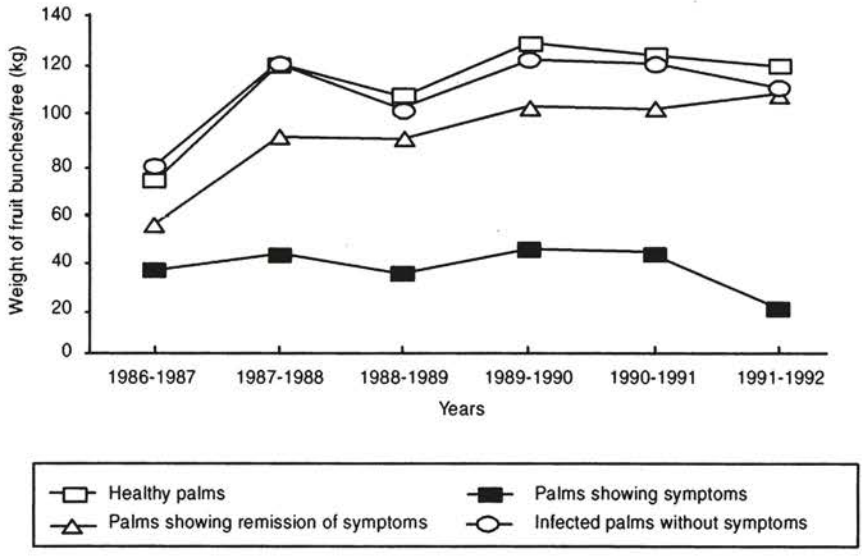


**Fig. 1.** Incidence of fusarium wilt on the yield of oil palm. Category C1001 (from Renard *et al.*, 1993).

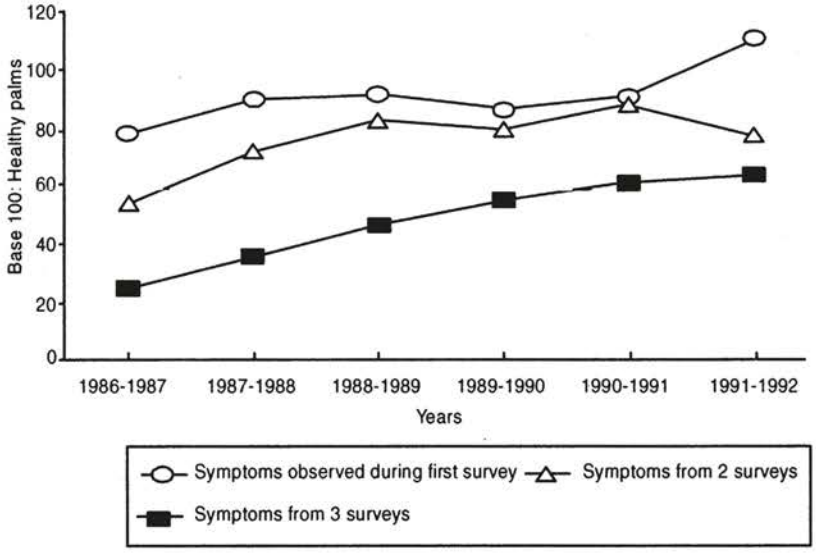


**Fig. 2.** Incidence of the intensity of fusarium wilt on the yield of oil palm. Category C1001 (from Renard *et al.*, 1993).

The bayoud of date palm, caused by *Fusarium oxysporum* f. sp. *albedinis*, shows several similarities with the vascular wilt of oil palms. This disease, for which inoculation tests on young seedlings were performed with success (Laville, 1962), is coming increasingly closer to oasis (Laville and Lossois, 1963).

## Tracheomycosis of coffee

Tracheomycosis of coffee (carbuncullariosis), caused by *Fusarium xylarioides* (Stey.) Gordon or *Gibberella xylarioides* (Steyaert) Heim et Saccas, is characterised by a sudden wilting of the plant due to the invasion of the vascular vessels by the pathogen.This decline may be limited to a part of the plant before becoming generalised and ultimately leading to its death. The presence of black to purplish-brown bands on the central column when the bark is removed and in the bark itself, is evidence of the fruiting bodies of the fungus. These fructifications are in the form of small balls (perithecia) which are first brown and then turn black , thus enabling a positive identification of tracheomycosis in the field.

This disease on coffee plants appeared for the first time in the 20s on *Coffea excelsa* in the Central African Republic where it may have at first been confused with a rot disease. In 1949, the disease affected *Coffea neo-arnoldinia* in the same country (Saccas, 1951). In 1970, the disease was reported on *Coffea arabica* in Ethiopia.

During the same period a coffee disease appeared on *Coffea canephora* in the Ivory Coast, mainly affecting two local varieties, Kouilou Bandama and

Kouilou Touba, as well as *Coffea abeokutae*, Indénié, and led to the destruction of tens of thousands of hectares. Similarly, in the Democratic Republic of Congo (ex-Belgian Congo), a similar disease was discovered on *Coffea robusta* of Ineac causing the death of 30-40% of the plants. Lastly, in Cameroon, in the 50s the disease affected *Coffea excelsa* in the eastern part of the country.

Curiously, the disease disappeared almost completely during the years 1960-65. This disappearance is certainly not very surprising because of the change in the plant material used during the same period, when Robusta replaced the other varieties. However, this conversion does not explain all because tracheomycosis reappeared in the Democratic Republic of Congo in the 80s and in East Africa in the 90s. Should we not therefore think that aging of the plantations and poor cultivating conditions are among the most important factors for the development of the disease? We know that the parasite makes its entry through injuries caused on the aerial parts of the plant or on roots, and several authors acknowledge the role played by environmental factors in the spread of the disease. Considering the behaviour observed in plantations, there is no doubt that selection of resistant coffee varieties is the method best adapted for controlling tracheomycosis. Behaviour tests, through artificial inoculations, have helped to bring out differences in varietal susceptibility (Saccas, 1956; Meiffren, 1961). A more intensive search in this domain should also take into account pathogen variability, given the diversity of the species and varieties affected in the different regions.

### Citrus decline caused by *Ceratocystis fimbriata*

A serious decay caused by *Ceratocystis fimbriata* Ellis et Halst. has been observed since three or four years in citrus orchards in the coffee growing zone in Colombia (Mourichon, 1994). The spread of this disease is rather disturbing as it has infected an average of 10% of the citrus in the affected zone. This disease is also found on lime trees in Cuba. It is favoured by the severe pruning of these trees done without disinfecting the wounds or the pruning tools.

Tahiti lime and orange trees are particularly affected. The very first symptom is a yellowing followed by a more or less severe wilting of the foliage which is often localised in parts.

A transversal section of the trunk or low branches reveals a necrosis of variable colour in the woody tissues of the central column (photo 15). A longitudinal section shows that the necrosis can be continuous from the base of the trunk above the graft line up to the upper part of the tree. The necrosis spreads centrifugally, the whole looking like a flame with a characteristic black colour, especially on lime, or orange coloured with serrated edge lesions. These lesions may be visible on the trunk. Symptoms of foliar decay correspond to intensive internal necrosis. Microscopic examination of woody tissue taken from just behind the growing edge revealed a large number of chlamydospores characteristic of *C. fimbriata*. On the other hand, very few

mycelial elements and endospores were observed. The conidial stage of the fungus, corresponding to the genus *Chalara,* can be easily obtained as a pure culture on a nutritive medium.

### Ecofungal wilt of cocoa

In 1985, a wilting of cocoa branches was observed in Uganda. This dieback was first attributed to *Botryodiplodia theobromae* Pat. (Snowden, 1920). The high incidence of this disease in plantations later planted with material of the Amelonado type and hybirds of the Upper Amazonian type, enabled researchers (Leakey, 1965) to identify the causal agent of this wilt as *Verticillium dahliae* Kleb.

The symptom, characterised by the hanging position of leaves in the process of wilting, which is moreover known in cases of extreme drought, is the first manifestation of the disease. The edges of the lamina and veins become brown and this colour quickly spreads throughout the leaves. As the foliage withers, a generalised yellowing of the lamina may in some cases be a transitory symptom before the wilt. The symptoms begin at the tips of small branches and then spread to all the branches. The dry leaves remain attached to the branches for a long time before falling off.

These external symptoms correspond to light brown elongated aligned spots on the twigs and branches. They are accompanied by distinct thin black lines located in the extension of the spots or next to them. The brown spots are also found in the trunk but are rare, almost non-existent, in the roots. Generally speaking, the concentration of internal symptoms increases from the roots towards the tips of branches.

These elements were determinant for reserving the name branch dieback for this disease of cocoa (Trocmé, 1972). Although artificial infections have demonstrated the pathogenic character of *Verticillium dahliae,* Trocmé has shown that the manifestation of symptoms is greatly dependent on environmental factors. In Uganda, the absence of shade in particular predisposes the cocoa plant to drying of branches and hence it is also known as ecofungal wilt of cocoa, the name given by this author for the disease. According to him, adequate attention was not paid to the special climatic conditions in Uganda for developing the cultivation of cocoa crops, which would have required adaptation of cultural techniques.

## Decays caused by *Phytophthora*

### Avocado wilt

Several species of *Phytophthora* and more particularly *P. cinnamomi* Rands, cause considerable damage in avocado orchards. These diseases are found in almost all the cultivated areas and none of the varieties, grafted or used as rootstocks, can be considered to be totally resistant. Two kinds of symptoms are distinguished, depending on the site of attack by the parasite.

The development of *P. cinnamomi* on roots results in the formation of small leaves that are greenish-yellow and often withered (photo 16). These leaves ultimately fall off and are not replaced. The foliage becomes very sparse and the tips of branchlets are bare and dry. The infected trees sometimes have a large number of small fruits. These symptoms are due to the growth of *P. cinnamomi* on the radicles and medium size roots. These roots become dark brown in colour while the cortex is destroyed and crumbles (Assas M'Billaut, 1978): the roots are not renewed, the tree can no longer nourish itself and it withers and dries up. The development of *P. cinnamomi* at the base of the trunk is manifested by large water-soaked spots on the bark, irregular in shape and dark brown in colour. A light brown, slightly gummy liquid oozes from the cracks in the bark (Brun, 1975). When the infected bark is removed, we can observe a browning of the underlying wood, which is generally more extensive than can be supposed from the external necrosis. The branches corresponding to the necrotic zone of the trunk dry up. The tree dies when the necrosis has girdled the whole trunk (photo 17). *P. cinnamomi* dominates these cankers, but *P. cactorum* (Lebert and Cohn) Schröter and *P. citrophthora* (Sm. and Sm.) Leonian may also be isolated.

## Citrus gummosis

*Phytophthora* attacks almost all the parts of the citrus plant and at all stages too. In seeed-beds, *P. nicotianae* var. *parasitica* (Dastur) Waterh. causes damping off disease which is, however, not a big constraint (Boccas and Laville, 1978; Laville, 1984).

*Phytophthora* species are capable of infecting the elongation zone of *Citrus* and *Poncirus* roots and in susceptible varieties the parasite establishes itself in the cortical zone where it induces necrosis. Root decay causes yellowing of leaves which fall off. The tree which cannot regenerate its root system is not nourished adequately and hence it vegetates and eventually dies. This fatal outcome is more rapid if the parasite reaches the large roots.

The large roots are attacked following contamination of the fibrous roots. These insidious attacks on the root system may be common (for example, in Corsica) even with grafted varieties of root stocks (sour orange, citrange) that are generally considered to be resistant (Laville, 1974).

*Phytophthora* infections are most common at the base of the trunks (photo 18) and on low branches. Different species of *Phytophthora* induce necrosis and cankers of variable intensity depending on the citrus variety and scion-rootstock combination.

The first symptoms appear on the bark in the form of irregular, water-soaked dark brown spots about the size of a small coin. The bark then splits and exudes an amber-brown gum, more or less abundant depending on the variety attacked and the vegetative state of the tree. It must be stated that this gummy water soluble exudate of pectic origin cannot be easily observed when rainfall is heavy and is therefore difficult to record. While the bark

dries up, the cambium remains moist and takes on a light brown stain, a colouring which also develops in the underlying wood. The necrosis spreads upwards and encompasses the trunk. At this stage the foliage begins to yellow in parts, the tree flowers out of season and bears small, immature fruits. The tree dries in proportion to the spread of the canker, the leaves fall in their turn and the fruits remain on the tree for a longer time but ultimately fall down as well. The rate of development of the canker varies depending on the climate and the physiological state of the tree. In Corsica, spring and autumn are favourable periods for the rapid development of cankers, the low winter temperatures limiting the development of these cankers as do high temperatures.

## Bud rot of coconut

In coconut trees, *Phytophthora* rots are manifested on the meristems and nuts (Quillec and Renard, 1984). Only meristem rot will be examined in this section. The first symptom is the withering and drying up of the spear leaf and loss of turgidity of the first leaf, followed or accompanied by a slight yellowing of a central leaf (about the 3-6 position) of the crown (photo 19). The yellowing becomes generalised in the upper leaves and then gradually the leaves become brown from the tip to the base and finally dry up completely, break and hang down between the petioles of older leaves which are still green. This evolution is the fate of all the leaves from the middle to the periphery of the crown. It reaches the oldest leaves by the end of a year. All that is left of the tree is the pollard trunk. The coconut bunches continue to grow as long as the leaves supporting them are green. The persistence of green leaves and bunches can be explained by the fact that the roots of the coconut tree are still healthy and continue to nourish it. Dissection of a coconut tree showing the first symptoms reveals advanced internal rot in the young growing leaves (photo 20). At this stage the rot has completely invaded the merismatic zone and the base of the spear and the young leaves. The infected tissues become purple to pinkish in colour. The rot spreads downwards in the central part of the trunk, softening the tissues which become yellow with a brown edge, doubled by a salmon pink margin at their border with healthy tissues. When the rot is in an advanced stage, the decomposed tissues become creamy and give off a foul smell. Often, particularly in the case of *P. katsurae* Katsura et Uchida, small oily looking rot centres evolving towards a brown colour develop inside the petioles of old leaves, these symptoms becoming visible only after cleaving the petiole longitudinally. There may be variations in the colour of the infected tissues, as well as in the rate of development of the symptoms, depending, on the cultivars or the species of *Phytophthora* involved, but the stages in the evolution of symptoms remain the same.

Symptoms caused by *Phytophthora* have been reported in a number of countries in the intertropical zone. Renewed outbreaks of this disease seem to

have occurred during the 70s. In 1977, a large centre of bud rot appeared in the Ivory Coast (Quillec *et al.*, 1984). Within a few years 50% of the West African Tall palms were dead in this centre.

In Indonesia, the disease which was initially confined to the local Tall cultivars and on the whole without any major economic impact, appeared in the 80s in new plantations planted with Nias Yellow Dwarf × West African Tall hybrids. The disease can cause the mortality of 40% of the trees within a few years (Thevenin *et al.*, 1994). The disease is known in the Philippines and the Caribbean (Renard, 1992) where more than 25% of the trees could die in less than 15 years.

## Diseases transmitted by insect vectors

By stinging the plant, insects are capable of transmitting a large number of organisms: virus, phytoplasma, trypanosoma and bacteria. In the chapter on insect vectors, it will be seen that insects can be carriers of other types of pathogens as well, viz., fungi and nematodes.

### Phytoplasma diseases

Only diseases affecting oil palm and coconut are dealt here. However, citrus plants can also be affected by this type of pathogen (see the table in the annex for diseases not treated in this book).

#### Lethal yellowing of coconut

All lethal yellowing type diseases, whether in America or in Africa, begin by a premature fall of mature and immature nuts. The leaflets at the tips of the lower leaves become yellow at the same time or immediately after the nut fall. The yellowing spreads to the whole leaf and then gradually extends to the increasingly younger leaves (photo 21). From the onset of yellowing, there appears a browning of the rachillas of ready to open or newly opened inflorescences (photo 22). The flowers, both male and female, also exhibit a more or less deep brown or grey-brown to black colour. The initial stages of lethal yellowing are similar to those of hartrot. In the final stage, when the oldest leaves have fallen, all that remains is a bouquet of yellow erect leaves (photo 23) which are shorter than the leaves of healthy trees. Secondary infections cause a rot in the upper part of the stem which sways and then falls down. All that is left of the coconut tree is an upright pollard trunk.

Lethal yellowing of coconut was recorded for the first time in the Cayman Islands in 1830. It then spread to the Caribbean (Haiti and Jamaica) and then to Florida and in the 80s to the Yucatan peninsula in Mexico. In was reported in the Honduras in 1996 by Ashburner *et al.* (1996). In Africa, it appeared in Togo in 1930 where it is called Kaïncopé disease (Bachy and Hoestra, 1958) and in south-east Ghana it is called St. Paul wilt. It is known as Kribi disease in Cameroon (Dollet *et al.*, 1977), Akwa disease in Nigeria

and fatal yellowing or lethal disease in Tanzania. In the early 60s, the disease appeared in the Cape Three Point region in Ghana, after having almost disappeared in an endemic manner from the region of Cape St. Paul and Togo (Mariau *et al.*, 1996). In less than thirty years, it is estimated that a million coconut trees were destroyed by this disease in Ghana—all around Cape St. Paul on the Ghanaian coast. Lethal yellowing is transmitted by insects and is capable of completely annihilating a coconut plantation in one or two years (photo 24).

## Blast of oil palm and coconut

Blast is the most important disease in oil palm nurseries in Africa. Although etiological studies have still not been able to demonstrate the role of an insect vector *(Recilia mica)*, it was observed that in the absence of shade, 80% of the trees could die of this disease (Bachy, 1958). The term blast, designated to characterise this malady, expresses the rapidity with which the symptoms evolve and lead to the death of the plant. The first symptom is blackening of the base of the spear leaf. At this stage, the spear leaf can be easily pulled out. Almost simultaneously the lower leaves of the plant begin to fade, quickly followed by browning of the leaf blade. This symptom evolves within one or two weeks, from the lower to the upper leaves, leading to a generalised wilting of the plant (photo 25). With the appearance of the first symptoms the roots exhibit cortical rot. A longitudinal section of the pseudo-bulb shows a brownish-yellow colouring. Remission of symptoms has been observed. However, when it occurs it is recommended not to plant these apparently cured palms (Franqueville *et al.*, 1991). Older plants are more tolerant than young nursery seedlings. Very exceptionally, cases of blast have been reported in the field in the year following planting. Blast may also affect coconut plants in nurseries. The symptoms are similar to those observed on oil palms (Quillec *et al.*,1978).

## Trypanosoma diseases of oil palm (marchitez), coconut (hartrot) and coffee

Marchitez of oil palm and hartrot of coconut are two similar diseases which were of great concern during different periods: from the end of the nineteenth century on coconut in Surinam and in the 60s on oil palm when this crop began to be cultivated in countries such as Colombia and Peru. They are known only in Latin America. These two diseases with a common syndrome are associated with the presence of an intraphloem flagellate protozoan, recognized and identified well after the damage caused by them was described for the first time. The ascending wilt of the foliage characterises these two diseases. In the case of marchitez, the first symptom of the disease is a browning of the tips of the terminal leaflets of the lower leaves (photo 26). At this stage the roots may look normal and healthy. All the leaflets of the first affected leaf turn brown, and the colour then quickly spreads to all the

leaves, from the bottom to the top. As the browning becomes generalised, the leaves become proportionally dry and turn ash grey in colour (photo 27). Rot may set in more or less quickly in the spear leaf depending on the environmental conditions. The young developing inflorescences get aborted and the bunches rot and dry up. At this stage the cortex of the primary and secondary roots become brown and the roots rot. It takes about three to six weeks from the appearance of the first symptom to the death of the palm (Lopez *et al.*,1975).

The evolution of hartrot of coconut is quite comparable to that of marchitez. The difference lies in the colour of the foliage. The first symptom is a yellow to light brown colouring of the leaflets depending on the cultivars (photo 28). Yellowing often precedes browning which becomes quickly generalised throughout the foliage. These foliar symptoms are accompanied by a premature fall of immature nuts and browning of the rachillas of the ready to open inflorescences (Renard, 1989; photo 29). The interior of the female flowers becomes brown and the male flowers are easily detached from the spikelets (photo 30). As in the case of oil palm, the roots become rotten. The tree generally dies about three to five weeks after the appearance of the first symptoms.

These symptoms are associated with the presence of an intraphloem flagellate protozoan (Dollet and Lopez, 1978). It is possible to confirm the presence of these protozoans as soon as the first symptoms appear by taking a drop obtained by pressing the still young root tissues of the oil palm or the peduncle of a ready to open coconut inflorescence and examining it under a light microscope.

These diseases are found in the northern part of South America, Peru and the State of Bahia in Brazil, up to Costa Rica. Hartrot has been known in Surinam since the end of the nineteenth century. Later it was also identified (and called cedros wilt) in Trinidad, where 15,000 coconut trees died within three years. It has caused sporadic destruction in small farming plantations in Latin America, particularly in Colombia, Brazil and French Guyana. It has devastated vast areas in commercial coconut plantations within a period of a few months.

In the case of oil palm, especially in some large commercial plantations, hundreds of hectares were infected by marchitez and thousands of hectares were, and still are, threatened. Treatment with insecticides along with regular maintenance of the plantations and their access helps to limit the damage to some extent. In the absence of preventive measures, the two diseases pose a serious threat for oil palm and coconut cultivation in Latin America.

A decaying disease of coffee has also been attributed to *Phytomonas* in Surinam and Guyana (Stahel, 1931). It must be noted that the symptoms are found in very humid, poorly drained zones where asphyxiation phenomena may superimpose a weak parasite or may favour it.

## Virus diseases

### Foliar decay of coconut

Coconut foliar decay is manifested by the appearance of diffuse orange-yellow spots and/or brown necrotic spots, depending on the cultivars (photo 31) on the leaflets of the upper third of the crown, from leaves 7-11 beginning from the unopened leaf (Calvez *et al.*, 1980). The yellowing on these leaves becomes generalised at the same time as the appearance of lateral necrosis on the petioles, symptoms which are very typical of this disease (photo 32). These leaves become dry and then fall off, leaving a gap between a tuft of yellowing leaves above and still green leaves below, giving the crown and X appearance. When the lower leaves have fallen, all that remain are 6 to 10 scraggy yellowish leaves, similar to the last stage of lethal yellowing disease caused by phytoplasmas.

The rot in the petiole of the middle leaves is perpetuated in the corresponding inflorescences, which become brown and necrotic. The inflorescences situated at the base of the young scraggy leaves remain healthy and it is this feature which distinguishes coconut foliar decay from lethal yellowing. The root system is normal until a very advanced stage of the disease. Susceptible cultivars (Malaysian Red Dwarf) remain for at least twelve months before dying. Remission is possible in some cultivars such as Rennell Tall (Calvez *et al.*, 1980). With the manifestation of the disease, the coconut tree stops yielding. The yield becomes normal only several years after it is completely cured. This disease is known only in Vanuatu (New Hebrides).

### Citrus tristeza virus (ctv)

Tristeza disease of citrus is a very destructive virus disease in the majority of the citrus growing regions of the world. It has destroyed more than 25 million trees in South America and 3 million in California. Only some regions in Mediterranean Africa, Saharan Africa and Central America are still free of this disease, although recent data show that it is advancing in the Caribbean arc through the vector (Aubert *et al.*, 1992).

The disease is expressed by a rapid and generalised wilting (or decline) of trees grafted on Seville orange, accompanied by necrosis of the phloem tissues along the graft line and anomalous cambial growth causing invagination of the bark into the wood, a symptom called stem pitting (photo 33). This stem pitting reduces the vigour of the tree considerably, leading to a reduction in the size of the fruits as well as a fall in the yield in the case of lime and pomelo.

### Swollen shoot disease of cocoa

Swollen shoot disease of cocoa is a virus disease known since 1992 in Ghana. It is characterised by the appearance of mosaic symptoms on the leaves, followed by swellings on the stems. When the swelling takes place at the tips of branches, it looks like a club and the apical meristem becomes non-

functional (photos 34 & 35). Wilting may follow and in the most serious cases the tree dies. The virus is transmitted by several species of bugs.

The most consistent sign of the disease is leaf mosaic. Several kinds of symptoms have been described by Partiot *et al.* (1978) in Togo. Form Agou 1, which is similar to the one found in Ghana, is the most typical of the disease. It exhibits a 'feathery mosaic' along the veins and swelling of the shoots and roots. The pods produced are small and rounded. An attenuated form has been identified; in this case, the mosaic symptom is not pronounced and the shoots are not swollen. The yield of the tree is not affected by this weak form. The third kind of symptom is characterised by a slight defoliation and a few swollen shoots. The yield is not affected by these symptoms.

Variations of these symptoms are found on leaves. Depending on the case, they are characterised by intervenal mottling, without swellings, or by a stippled mosaic which begins along the veins and then spreads throughout the lamina. Swelling of shoots was not observed in this case. Totally, about thirty forms have been described. The disease causes wilting of the leaves and branches and a general and gradual weakening of the plant with a fall in its productivity. The ultimate stage of the disease is the death of the tree.

Swollen shoot disturbs the formation of cocoa seeds, which is responsible for the fall in production. Thresh (1958) considers swollen shoot to be one of the most important factors limiting the production of cocoa trees and one of the plant diseases responsible for the most serious economic losses in the world.

The virus is also found on several plant species growing near cocoa trees: *Cola chlamydantha, C. gigantea, Erythropsis barteri, Sterculia chinopetala, S. tragacantha, Adansonia barteri, Bombax brunuopense* and *Ceiba pentendra,* as well as a few herbaceous species such as *Corchorus* sp.

This disease was described in its serious form in Africa: in Sierra Leone (Attafuah *et al.*, 1963), Ivory Coast (Alibert, 1946), Nigeria (Murray, 1945) and Togo Partiot *et al.*, 1978). In Ghana it was responsible for the uprooting of 163 million trees (Legg, 1979) and continues to be the objective of a new eradication programme. Curiously, in the Ivory Coast where two virus strains had been identified (as Kongodia and Sankadiokro forms), the disease has remained localised in these two sites for more than fifty years. Faced with such situations, are we not right in questioning the interest of eradication campaigns?

### Bacterial diseases: Huanglongbin or citrus greening

*Huanglongbin* or greening is a bacterial disease on citrus plants and is transmitted by insects.

The symptoms of this disease are similar to symptoms of mineral deficiency (photo 36). The disease can be identified with certainty only when the various sequences of the pathological stages following infection are taken into consideration. Greening is characterised by three kinds of abnormalities: chlorosis of leaves, disturbed phenology and abnormal fruits (Aubert, 1988).

Foliar chlorosis assumes varied aspects. In the island of Réunion, the most common symptom is the appearance of green spots on a yellow background which could be confused with zinc or iron deficiency. A second symptom which is often observed is that of small, pale yellow, spoon-shaped leaves, which is also similar to zinc deficiency. A third symptom corresponds to a mottling of the lamina, similar to that caused by manganese deficiency. Some old leaves may become thick and coriaceous and a network of protuberant veins may appear on their upper surface, sometimes with traces of suberisation resembling boron deficiency.

In contrast to deficiencies which induce general yellowing, chlorosis caused by greening appear in parts and are irreversible.

A disturbed phenology is expressed by the fall of chlorotic leaves, interspersed with vegetative growth and out of season flowering. The most chlorotic branches eventually dry up (photo 37). The fruits are asymmetrical and do not become coloured on maturity. The columella is deformed and the seeds are aborted. The juice and sugar content is low, making the fruits commercially unviable.

Besides these directly visible symptoms, anatomical disorders are also seen in the sieve elements, with the formation of secondary phloem, thickening of the cell walls and deposition of lignin in the sclerenchyma. All the disturbances in the phloem are particularly traumatic for the plant, as much for the leaves as for the reproductive organs, branches and roots. All these contribute more or less quickly to a progressive wilting and ultimately the death of the plant.

Greening is a serious problem in Asia and is also responsible for considerable destruction in sub-Saharan Africa.

In China greening, which is commonly called cgd (citrus green disease), caused the death of about 12 million trees in the 50s, mainly because of the divulgation of contaminated material. In 1960 and 1970, in Indonesia where the disease is known as cvpd (citrus vein phloem degeneration), the loss was estimated to be 3 million trees, to which should be added the plants infected in the nursery (Tirtawidjaja, 1980). The best commercial variety of Keprok tangerines has disappeared almost completely in Java. In the Philippines it is estimated that 5 million trees have been affected by greening (locally called leaf mottle disease) since the early 70s, reducing the area planted by 60%. In Africa, greening is responsible for the mortality of several million trees and the disease, which was initially localised in South Africa, has now spread to East Africa, up to Ethiopia, Cameroon and Madagascar (Aubert, 1992).

## Diseases of unknown etiology

### Dry bud rot of oil palm and coconut

Dry bud rot (or heart rot) of oil palm and coconut, which had remained undiscovered for a long time, spread with the generalisation of nurseries

without shading following the results of researches conducted on blast disease. It is also found during the initial years of plantations, especially in areas where a vegetation dominated by Gramineae has become established in place of a homogenous cover of *Pueraria*. The etiological agent of the disease is not known but we know that it is transmitted by two insects.

The appearance of small yellow and white spots on the spear or first leaf is the most typical expression of the disease (photo 38). Sometimes this symptom is localised on the lower part of the leaflets of the still white spear leaf, in which case the spots are brown. At an advanced stage the base of the spear leaf is completely brown. These symptoms are accompanied by a pronounced stunting of the young leaves. Lenticular or elongated brown zones develop on the petioles of the middle leaves. Sometimes there is a variation of these symptoms. It is manifested by the appearance of a large number of small white spots on the young leaves and a reduction in their size. In this case, there is no rotting of the spear leaf nor of the bud, nor is the pseudo-bulb coloured. The plants remain stunted and sometimes remission of symptoms may be observed. In the nursery the symptoms on oil palm and coconut are almost identical. In the case of coconut the pseudo-bulb is brown in colour with a corky aspect (Renard *et al.*, 1975), while purple is the dominant colour in the oil palm (Renard and Quillec, 1984b; photo 39). In the case of coconut these symptoms are the same in plantations, whereas in the case of oil palm the most characteristic symptom in plantations is expressed by fairly deep yellowing of the young leaves.

The leaflets of the spear leaf are scattered with round, oily looking spots. The most typical symptom of the disease is still the wine purple colour which appears in the trunk in the first year of planting. All these symptoms gradually lead to the death of the tree, in the case of both oil palm and coconut: the leaves becomes yellow and then dry up, from the youngest to the oldest. The root system, which remained healthy for a long time, begins to rot from the tips of the roots, which in their turn become brown and dry up.

The damage caused is varied. The diseased plants are either scattered or grouped together depending on the environment (weeds, forest border, topography). The disease rate is about 2-3% on the average, but can go up to 10-25% of the plants in restricted areas or even more in certain parts of oil palm plantations.

## Ring spot disease of oil palm

Symptoms of this disease are allied to those of dry bud rot of coconut because they also appear on the spear and young leaves. The disease begins with a slight chlorosis of the young leaves, concomitant with the appearance of a large number of elongated or ring-shaped spots (Dzido *et al.*, 1978) that are lighter in colour than the rest of the leaf blade (photo 40). These initial symptoms are followed by a generalised and intense yellowing of the young leaves and then of the older leaves. At this stage rot sets in the spear leaf and

spreads towards the meristem. The lower leaves become brown, the fruit bunches and roots also rot and the plant ultimately dies. In the trunk, a large number of necroses can be observed in the vascular bundles. From the onset of the first symptoms to the death of the tree it takes about three months.

The damage caused by this disease is mainly during the initial years of the plantation and is generally not very serious. However, some small plantations have been devastated to the tune of 95%. Ring spot symptom has been reported only in Latin America, especially in Ecuador and Peru, and to a lesser extent in Colombia and Brazil.

## Viroid diseases

It is believed that some of these diseases, like that of coconut, are transmitted by insects, but no formal demonstration has been possible until now.

### Coconut cadang-cadang disease

Coconut cadang-cadang is a debilitating disease of coconut palms, which gradually leads to the death of the tree. It gets its name from gadan-gadan, which means dying in a Philippine dialect. Appearing in the late 20s in the island of Juan Miguel in the Philippines, this disease has killed about 30 million coconut trees since its appearance (Bigornia, 1977). Luzon province, situated in the north of the Philippine archipelago, is the only region affected by cadang-cadang. Mindanao island, the major copra-producing region in the south, is free of it.

Coconut trees infected by cadang-cadang can be recognized by the reduced number of leaves and bronze-yellow colouring of the foliage (photo 41). This colour is due to the presence of a multitude of small yellow, more or less circular, translucent spots which look oily and olivaceous when viewed against light (photo 42). This stage, which is already advanced, is due to an extremely slow evolution of the following process. The first stage of the disease corresponds to the appearance of more or less round nuts with brown equatorial scarifications. The first small yellow spots are scattered on the leaf. The next stage is characterised by the persistence of stipules at the base of the rachis of the leaves, as well as reduced production of nuts as the inflorescences begin to rot. The leaves given out are short and the large number of yellow spots gives the crown a chlorotic appearance. In the last stage, nut production ceases, the tree is fully chlorotic and the apical part of the trunk is narrowed. The plant stops producing flowers. It may take about ten years (from 8 to 17 years) from the appearance of the first symptoms to the death of the tree (Zelazny *et al.*, 1982). For a long time the disease was considered to be a malady of old coconut trees. We now know, by inoculating a viroid, that young coconut trees can show slight symptoms eighteen months after inoculation.

This disease may also attack oil palms. The symptoms are manifested by

a cessation of flower production, reduced emission of leaves and the leaves given out becoming smaller and smaller and chlorotic. Orange coloured spots develop on the entire foliage (Randles *et al.*, 1980).

### Exocortis and cachexia of citrus

Exocortis and cachexia are two diseases of citrus plants in which the causal organisms are transmitted through grafts and mechanically through the sap transported on the tools. Transmission of these diseases by insect vectors has not been reported. Expression of the symptoms is favoured by high temperatures.

Exocortis is manifested on *Poncirus trifoliata* rootstock and on most of its citrange hybrids, as well as on some lime trees (Rangpur, sweet lime) and certain citron clones. Scaling and cracking appear on the rootstock about four years after grafting of infected material (photo 43). The tree thus infected is more or less stunted and its production is reduced (Vogel and Bové, 1986).

Species susceptible to cachexia are mainly *Citrus macrophylla*, Mandarin-orange trees and their hybirds (tangelo, tangor). Small more or less deep pits appear in the wood above the graft line. At the same level small spots can be observed on the inner side of the bark, which are extensions going deep into the wood. This symptom is called stem pitting by Anglo-Saxons. Gummy impregnations are visible in the thickness of the bark (photo 44). Highly infected trees remain underdeveloped, become chlorotic and may perish (Bové, 1993).

A number of citrus varieties are tolerant to one or the other of these diseases and the symptoms are manifested when plant material from these healthy carriers is grafted on a susceptible rootstock. The disease can be prevented by using scions free of these viroids.

## Diseases caused by nematodes

### Coffee decline

Nematode pathogens of coffee belong to two genera, *Meloidogyne* (gall-causing nematodes) and *Pratylenchus* (nematodes causing lesions). These two nematodes are found throughout the coffee-producing region in Latin America causing heavy losses (Campos *et al.*, 1990). Despite its seriousness, the problem of coffee decline caused by nematodes was taken up in Central America only in the 60s (Schieber and Sosa, 1960; Salas and Echandi, 1961).

Nematodes of the genus *Pratylenchus* grow on the roots of coffee plants, inducing a separation of the cortex from the central column of the root. These nematodes are capable of leaving the roots if the conditions become unfavourable to go and colonise new roots (photo 45). They grow at the expense of the cortical parenchyma cells causing large lesions that are often accentuated by secondary pathogens, especially fungi. These infections are expressed by a general chlorosis of the foliage of the coffee plant, followed in

severe cases by a progressive wilting of the primary branches which could lead to the death of the plant within a few years (photo 46). The symptoms evolve slowly, growth of the plant slows down and the yield is reduced. Any treatment to cure the disease is then impossible. According to a survey conducted by Anacafé in Guatemala, the loss in production was estimated to be 20% in some coffee growing regions. A special study (Villain *et al.*, 1996) showed that the losses cumulated over several seasons could exceed 75%.

Nematodes of the genus *Meloidogyne* are sedentary endoparasites which attach themselves to the pericycle of the root. The plant tissues react to the infection by producing galls (photo 47). These galls may invade the entire root system. Severe symptoms are similar to those described for nematodes of the genus *Pratylenchus*. However, the symptomatology and damage caused are varied depending on the species involved. The species, *M. exigua* Goeldi, induces large galls without destroying the roots. This species is not very pathogenic on adult coffee plants. On the other hand, it can cause severe symptoms on young plants or when the soil is not very fertile and also under drought conditions (Campos *et al.*, 1990).

*M. arabicida* n. sp. (Lopez and Salazar, 1989), a species described recently in the Juan Vinas region in Costa Rica, is associated with a very serious wilt, corchiosis. This disease is manifested by very large corky galls on the taproot as well as on large roots, leading to the death of the plant within two to three years. Fungi belonging to the genera *Fusarium, Cylindrocladium* and *Phialophora* are associated with this decline (Calderon-Vega, 1989).

### Red ring of oil palm and coconut

This disease, caused by the nematode *Rhadinaphelenchus cocophilus* (Cobb), affects oil palm as well as coconut trees in Latin America and the Caribbean. Despite some small differences, the general symptoms of this disease are similar on these two plants. They are manifested by a slowing down of the growth of the tree, visible in the shrinking of young leaves which become light green to yellowish in colour. This discoloration spreads throughout the foliage with the yellowing becoming more intense concomitant with wilting of the middle leaves. This wilting then gradually spreads throughout the crown, from the top towards the bottom. We often observe a rotting of bunches of oil palm and nut fall on coconut.

In very typical cases, a section of the trunk shows a pink to light brown coloured ring in coconut (photo 48) and brownish-grey in oil palm (photo 49). Depending on the stage of the disease, this ring is more or less complete and its location in the trunk is variable. It may be continuous from the base to the top of the trunk or may be present only at the top or only at the bottom of the trunk. Grey or brown necrotic spots are sometimes visible at the base of the petioles of young leaves (photo 50).

At an advanced stage of the disease, the infected tissues of the trunk rot, giving rise to a large cavity. As long as there is no rot at the top of the trunk,

the palm tree does not die and gives out a succession of very short leaves making the tree totally unproductive. Total remission of the disease is unknown and death always ensues after a fairly long period.

Disease incidence varies depending on the regions. Some plantations may be badly affected, as observed for example in Venezuela where 70% of the palms died of red ring disease at the age of 15 years (Mariau, 1978). In Bahia State in Brazil, several plantations were severely affected with a mean annual disease rate of 1.5 to 3.4%, and some plots could be eliminated within a year (Renard, 1985).

## Disease of unknown origin: heart rot of oil palm

Among the oil palm diseases present in Latin America, the one commonly called heart rot is the most devastating and constitutes a limiting factor for the cultivation of oil palm in several regions (Mariau *et al.*, 1992).

The first symptom of the disease is the chlorosis of young leaves (leaves 4, 5 and 6, starting from the spear leaf), quickly followed by browning and necrosis of the tips of leaflets (photo 51). Spotting, or yellow mottling, is also common on these leaflets. Asymmetrical rots appear on the leaflets and spear leaves. Often a wet deteriorating rot of the tissues is observed at the base of the spear leaves, which advances towards the terminal bud (photo 52). Transverse necrotic cracks appear on the inner side of the petioles and rachis of young leaves (photo 53). These symptoms generally develop rapidly which causes the central part of the crown to wither and sway. The remaining leaves yellow slowly, from the tip towards the base and from the middle leaves to the basal ones. The slow progress of symptoms is due to the absence of severe rot and momentary emission of small atrophied leave. In almost all cases, death of the palm is inevitable, from a few months to eighteen months after the appearance of the first symptoms.

The hanging production on the plant is not directly and immediately affected and matures in most cases. On the epidemiological front, this disease evolves slowly, even very slowly, with an arithmetical progression of 0.1 to 1.3 per year during the first few years following planting. Suddenly, the progression becomes geometrical (Fig 3) with the formation of large infection centres, this progression being more or less related to environmental factors such as soil and climate depending on the region. However that may be, the destruction is always heavy and spectacular.

In the early 70s, a 2000-hectare plantation in the region of Turbo in Colombia was completely destroyed in a few years. Similar symptoms were observed in small palm plantations on the Pacific coast. A disease showing some features in common with the heart rot disease of palm was described in the Llanos region in Colombia by research workers of that country (Nieto Paez, 1993). A fungal hypothesis was advanced to explain this type of heart rot for which a large number of more or less long lasting cures are available.

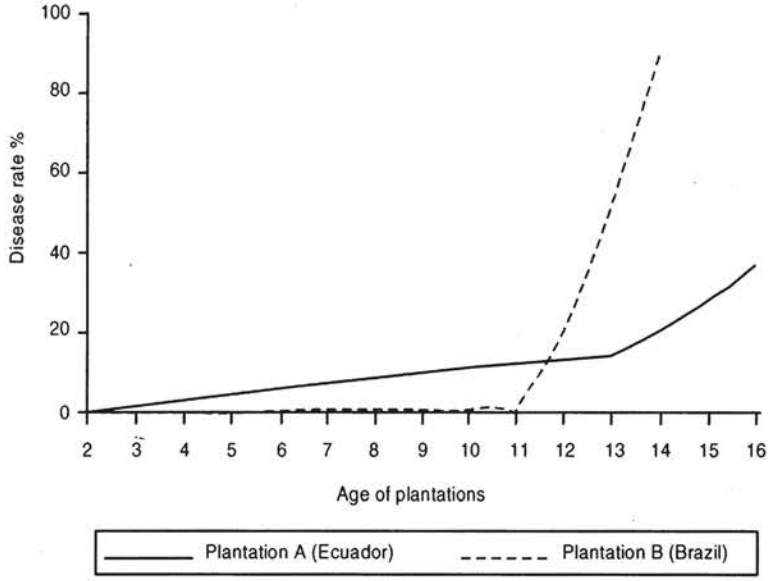


**Fig. 3** Evolution of bud rot disease (from Mariau *et al.*, 1992).

In Ecuador, plantations situated in the Amazonian part of the country were seriously affected and in the end appeared to be highly endangered. Plantations situated on the Pacific side were less affected and the disease does not assume epidemic proportions as in the Amazonian region. In Brazil, an oil palm plantation in the Belem region was also destroyed by heart rot at the same time as a plantation in the Téfé region, several hundred kilometres west of Manaus. In Surinam, the disease was manifested when the palms were only four years old in the largest plantation in the country (1700 hectares). Disease centres have reduced the area planted by more than 500 hectares in six years and almost completely destroyed it in the following years. In Panama, a plantation of *Elaeis guineensis* was also destroyed.

## DISEASES OF LEAVES, BRANCHES AND TRUNK

All foliar diseases are related to the development of fungi. The plant becomes weak as a result of such attacks and may even die in the most serious cases. The causal organisms of diseases of the trunk and branches are fungi and bacteria.

### Diseases caused by fungi

Fungal diseases that affect leaves cause them to fall or render them incapable of photosynthesis. Other fungal diseases attack the trunks and branches.

## South American leaf blight of rubber

Generally called Salb (South American leaf blight), this South American leaf disease is caused by *Microcyclus ulei* (P. Henn.) Von Arx. Repeated infections of this fungus induce successive defoliation (photo 54) accompanied by a decaying of the tips of branches and in extreme cases may lead to tree mortality. Symptoms caused by the asexual form (conidia) of the parasite vary depending on the age of the leaf at the time of infection. When the leaves are in the reddish-brown stage, i.e., four to nine days after their formation, dark grey lesions (bearers of conidia) make their appearance deforming the lamina and this is followed by leaf fall. Generally, slightly older leaves (ten to fifteen days) do not fall and laminal deformation is not severe. Lesions are formed on the lower surface and may enlarge up to 2 cm in diameter. Conidia are produced on these lesions which are grey to olive green with a downy aspect (photo 55). On the upper surface of the leaflet, each lesion gives rise to a chlorotic translucent spot. With the hardening of the leaf, the lesions lose their velvety aspect and become brown in colour. The centre of the lesion may become dry and even fall off (Chee and Holliday, 1986; photo 56). At an advanced stage of the infection (one month), black pycnidia can be observed on the upper surface of the leaves. They increase in size and number, forming black masses arranged in small circles a few mm in diameter (photo 57). Normally the leaf does not fall at this stage; it becomes rough to the touch. By the time the leaf reaches maturity (three months after its sprouting), the stromatic masses have become large and black; perithecia have replaced the pycnidia. They are situated at the margins of the necrotic spots, almost always on the upper surface of the leaf, giving it a sooty appearance.

This succession of the three stages of the disease may also take place on the petioles, green shoots, inflorescences and green fruits. The first symptom is a small swelling on which the conidia are produced. The infected petioles and stems become deformed, curve and may roll into a spiral. The lesions become corky and may disappear. The injured tissues become hypertrophied. The flowers may also be infected (Rivano, 1992).

Since the beginning of the century South American leaf blight, which is strictly confined to Latin America, has been the main factor hindering the development of rubber cultivation in this part of the world. Between 1908 and 1948, the disease was found wherever rubber was cultivated, irrespective of the size of the plantation—whether a plantation of a few thousand hectares or a small bud wood garden of one hectare.

*M. ulei* is present in Colombia, Bolivia, Ecuador, Peru, Venezuela, the Honduras, Guatemala and even in Mexico, which extends its distribution area up to 18°N.

In the south, the disease has spread to Brazil, first in Bahia state (in 1930) and later up to the state of Sao Paulo state (in 1960), soon after the establishment of the first plantations at latitudes quite uncommon for rubber, i.e., 24°S (Holliday, 1970).

*M. ulei* has been detected in Trinidad and Haiti, but is not found in Guadeloupe. This island has a quarantine station for rubber plant material transiting from America towards Asia and Africa (see the chapter on Healthy plant material and certification). Figure 4 shows the distribution of *M. ulei* as given by Holliday (1970) and Chee and Holiday (1986).

Rubber companies had to abandon their projects for establishing rubber plantations in Brazil, Surinam and Panama following very severe attacks by *M. ulei*.

### Leaf anthracnose of hevea

Although leaf anthracnose of hevea rubber is present in all the continents, it is rampant mainly in Central Africa and Asia. It is caused by a fungus *Colletotrichum gloeosporoides* Penz., whose sexual form *Glomerella cingulata* (Ston.) Spauld and Schr. is almost never found on the rubber plant.

On very young leaves the disease is manifested by small red spots with a black border on the major part of the leaf blade. The tip of the leaf becomes soft, black and curled up. At this stage the leaf falls with the slightest gust of wind. On older leaves the symptoms appear at the edge of the lamina which may fall off (photo 58). The remaining part of the leaf shows large necroses around which the lamina is deformed. The parasite sporulates in the necrotic

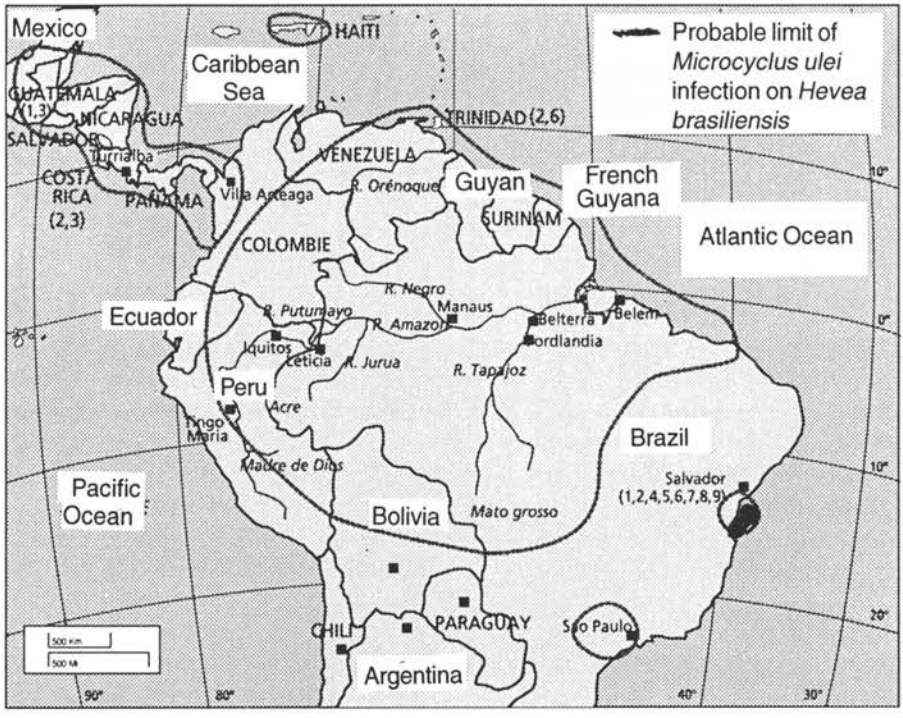


**Fig. 4.** Distribution of *Microcyclus ulei* (from Holliday, 1970).

areas by producing orange acervuli. Symptoms of anthracnose are also observed on the green branches as necrosis from which latex exudes. On the petioles of leaves and floral parts the symptoms are limited to small black spots. Sometimes the fruits are aborted and dry up and become areas of prolific spore production (photo 59).

The fall of young leaves and deforming necrotic spots on adult leaves lead to a reduced leaf surface and in some cases to the death (or dieback) of the terminal branches. It is difficult to make an accurate estimate of the economic loss but it is acknowledged that severe and repeated attacks slow down the growth and tire the plant due to repeated emission of new leaves (Peries, 1966; Senechal, 1986). In a plantation in Gabon planted mostly with susceptible clones, foliar density diminished by half in a span of six years (Guyot, pers. comm.).

This general weakening of the plant opens the door to other parasites such as *Botryodiplodia theobromae.* By allowing in more light and favouring the growth of grasses, it imposes frequent and prolonged maintenance of the plantations.

### *Corynespora* leaf spot of rubber

Hevea leaves are susceptible to *Corynespora cassiicola* Berk and Curt. for about the first four weeks after the sprouting of leaves. The parasite causes brown foliar lesions which enlarge in an irregular manner. A browning of the veins characterises most of the lesions, giving rise to the expression 'fish-bone' symptoms (Breton *et al.*, 1997; Chee, 1988) (photo 60). The infected leaves gradually become yellow and then fall off. As in the case of *Colletotrichum,* repeated attacks by *Corynespora* weakens the tree. Differences in susceptibility to the parasite have been observed. This foliar disease is of great concern in Africa and also in Asia where its spread is favoured by the presence of susceptible clones. Genetic control through tolerant plant material is possible but it should take into account the extreme variability in the pathogenicity of *Corynespora cassiicola* (Breton *et al.*, 1997).

### Helminthosporiosis of coconut

Helminthosporiosis of coconut, caused by *Heliminthosporium halodes* (Dresch.), is characterised by the presence of brown, usually elongated foliar spots, which are initially isolated and then become coalescent (Quillec and Renard, 1975; photo 61). The parasite sporulates on the lesions on the lower surface of the leaves, giving the symptom a dark brown and velvety appearance. These fragile spores are washed off by rain water and so well dispersed by wind that on older symptoms it is difficult and even impossible to identify the parasite when hyperparasites have invaded the necrotic tissues. The more the parasite spreads on the leaf blade, the more the centre of the spot becomes necrotic and grey and breaks up, the leaflet and the leaf assuming a laciniate aspect.

Reduction in the leaf surface, which may be considerable in susceptible cultivars, slows down the growth of plants in the nursery as well as in the field. The coconut plants thus look stunted. Coconut trees of Polynesian origin and certain coconut populations from Indonesia are susceptible to helminthosporiosis. Nevertheless it was possible to identify Polynesian cultivars that are more tolerant than others, which could be used for producing disease tolerant Dwarf × Tall or Tall × Tall hybrids tolerant to the disease. The West African Tall is particularly tolerant to helminthosporiosis and exhibits a hypersensitive reaction towards the parasite. In the case of susceptible material, standard fungal treatments are necessary in the nursery; chlorothalonil (Daconil) is particularly effective.

### Coconut leaf diseases in Brazil

The warty disease of coconut, known by the Brazilian name of *lixa pequena* (small rasps), is caused by a fungus called *Phyllachora torrendiella* (Bat.) nov. comb. It is first manifested by the presence of black, slightly prominent stromata structures on the upper surface of the leaf, rachis and midrib of the leaves. These stromata become increasingly prominent on leaves of 8-10 whorls starting from the summit of the crown; they are made up of spherical, black, rugose perithecia with a dome opening through an ostiole ornamented with radial stripes (Irho, 1989). In case of severe infections, the leaflets dry prematurely, first on both sides of the midrib of the leaflets and then the necrosis spreads gradually leading to a generalised wilting of the leaf (photo 62). The middle and lower leaves lose about one-third of their assimilating surface.

Foliar damage is often aggravated by the intervention of *Botryosphaeria cocogena* n. sp., a parasite which generally grows on lesions caused by *Phyllachora torrendiella* (Subileau, 1993). Light brown necrotic areas showing a darker zonation are characteristic symptoms of *B. cocogena* (photo 63). When these necroses reach the midrib of the leaf, they lyse the tissues and a brown gummy exudate oozes out. This lysis accelerates the wilting of leaves. When primary infection begins at the tip of the midrib, wilting of the leaflets progresses from the tip towards the base of the leaf, giving the symptom a V shape (photo 64). A gummy exudate is seen at the point of insertion of the leaflets. Both the symptoms, which are often seen on the same leaf, are called *queima das folhas*, the Brazilian name for 'leaf burn' or coconut leaf blight (Warwick *et al.*, 1991). The combined action of *lixa pequena* and *queima das folhas* lead to a generalised wilting of the leaves situated below leaf no. 12. Weighed down by the fruits, the fragile leaves collapse at the point of insertion on the trunk. The peduncle of the fruit bunch breaks and is often accompanied by the fall of immature nuts, leading to loss in production which may reduce the potential yield of the coconut tree by 20-40%. These diseases are present in varying degrees in all the coconut-growing regions in Brazil. They have also been reported in French Guyana.

In Brazil, especially in the state of Para, a fungal hyperparasite, *Septofusidium elegantulum*, invades the perithecia of *P. torrendiella*, thereby interrupting the life cycle of the parasite and contributing naturally to control of the incidence of *lixa*.

## Cercosporiosis

### *Cercospora* disease of oil palm

Cercosporiosis of oil palm is a common disease in Central and West Africa. It is a foliar disease that is manifested in prenurseries and nurseries and on young palms in plantations.

Cercosporiosis caused by *Cercospora elaeidis* Steyaert is expressed by the appearance of yellow spots marked by a brown point in the middle. These spots spread, become brown and surrounded by a yellowish halo. They later assume a brown mottled aspect, scattered on the leaflets of the palm frond. A fine brownish down may cover the spots on the lower side of the leaflets. The spots spread and coalesce; the leaf blade becomes yellowish-brown, then brown and begins to dry from the tip towards the base and from the edge of the leaflet towards the midrib (photo 65). The leaf becomes grey and brittle (Renard and Quillec, 1979). The disease is propagated by conidia, which are disseminated by wind and rain. Under favourable conditions (especially when there is dew) the conidia germinate, the germinating tubes entering the leaf through the stomata. The incubation period is about 25 days (Renard and Quillec, 1977).

In the nursery, the assimilating surface of the leaf may be reduced by half and the global mass of the plant by 25-30% (Quillec and Renard, 1977). In the field, the disease is responsible for a reduction of 10% of the average number of leaves given out, 15% of the length of leaf no. 4 and 25% of the girth of the collar. Flowering is delayed in diseased plants and female inflorescences are less abundant than on healthy plants. However, we do not have precise estimates of the influence of cercosporiosis on the production of bunches.

### Cercospora disease of citrus

Once called *Cercospora angolensis*, the fungus responsible for cercosporosis of citrus was recently described under the name *Phaeoramularia angolensis*. The sexual stage of the fungus is not known and there is no information available to help resolve its variability. This disease is not specific to leaves and also develops on fruits (Brun, 1972; Kuate *et al.*, 1994). The first symptoms on the leaf appear as small discoloured specks. These small lesions become bigger and are concomitantly surrounded by a yellow halo (photo 66). The spherical lesions enlarge to a diameter of 3-4 mm and then their centre dries up and the necrotic tissues break, leaving a hole in the lamina. The infected leaves fall prematurely. The damage is severe especially in the cultivation zones of medium and high elevations. Although not negligible, the damage to leaves is less than the damage to fruits (photo 67). The disease is found in Angola,

where it was described for the first time in 1952 (De Carvalho and Mendes, 1953), and in a number of African countries and also in Yemen.

## Coffee rust

### Orange rust

Orange rust of coffee, caused by *Hemileia vastatrix* Berk. et Br., which actually affects only *Coffea arabica,* is characterised by the appearance of small discoloured spots on the lower surface of the leaf (photo 68).

In one to two weeks these spots are covered by uredospores from the centre to the periphery, forming a powdery orange-coloured mass (photo 69). These spots grow bigger, coalesce and form large lesions on the leaf blade and become necrotic in the middle as they age.

Photosynthetic activity is reduced and leaf fall follows. Massive defoliation leads to wilting of the branches. If defoliation is successive, the coffee plant loses its vigour and may eventually die. Several important works have been carried out by CIRAD research workers in several coffee-growing countries in Africa, Latin America, South-East Asia and Papua New Guinea. Rust attacks are more severe at medium elevations than at high elevations. Other factors such as rainfall, structure of the plantation, berry load, alternation in production from one year to another, govern the development of the epidemic and impact of the disease on the yield (Avelino *et al.*, 1991; see chapter on Rational chemical control and cultural techniques). Moreover, studies under controlled conditions (Berry *et al.*, 1987; André *et al.*, 1989) have shown that strong light favours the development of rust and intensifies its influence on the physiology of the coffee plant in terms of susceptibility of the plant and lower gas exchange between the plant and environment. This is why rust disease has repercussions on the yield of the coffee plant. Although it can be said that very severe attacks result in nil or almost nil harvests, we do not have accurate data to enable us to correctly predict the damage based on the intensity of attacks observed in the field. Recent researches conducted in the Honduras have attempted to forecast the damage by integrating the influence of all the biotic and abiotic factors comprising the environment of the coffee plant.

The disease originated in Africa but appeared for the first time in Ceylon (Sri Lanka) during the years 1860-1870 and later in Java in 1876, resulting in the almost total disappearance of Arabica in these regions. From Sri Lanka the disease quickly spread to all the coffee-growing regions in Asia. It was reported in Kenya in 1890 and is getting closer and closer to East Africa. It was detected in West Africa in 1960 and then in Brazil in 1970, from where it spread to South and Central America within a few years. It varies in intensity in all these countries and is often less serious than what was feared before it arrived. At present there is no economically important coffee production zone that has been spared by the rust.

## Mealy rust

Mealy rust of coffee caused by *Hemeleia coffeicola* Maublanc et Roger is characterised by the presence of large lesions on the lower surface of the leaf on which the yellow-coloured fructifications of the parasite are found scattered. In contrast to orange rust symptoms, there is no discolouration of the lamina preceding the appearance of the spores. This is why mealy rust is not as easily detectable as orange rust. However one should not minimise the gravity of its incidence where it exists, even if defoliation is not heavy and is delayed. Studies conducted in Cameroon have shown that it disrupts photosynthesis and assimilation of phosphorus (Muller, 1980). Heavy losses in production following severe attacks of mealy rust have been reported in Cameroon.

In contrast to orange rust which is now widespread in all the coffee-growing countries, mealy rust is confined to a few countries in Africa. The disease was reported for the first time in Cameroon, on Arabica in 1929 and then on Robusta (Muller, 1954). Saccas (1951) reported it in the Central African Republic on Excelsa and Nana coffee plants. It is found mainly in Central Africa (Cameroon, Central African Republic, Nigeria, São Tomé and Angola), but has also been reported on Robusta in Togo and on wild *Coffea* in the Ivory Coast. Humidity favours its development but in contrast to orange rust, mealy rust is found at high as well as low elevations. This absence of ecological specialisation poses a threat for coffee cultivation throughout the world.

### American foliar disease of coffee leaves

The American disease of coffee caused by *Mycena citricolor* (Berk. et Curt.) Sacc. was detected for the first time in Colombia in 1880. It is now found in several countries of Latin America at high elevations. The parasite induces different kinds of lesions on the leaves. The disease can be recognized by the large number of round, light to dark brown coloured spots on the leaf blade (photo 70). The tissues of these spots dry and fall off leaving holes in the leaves and eventually nothing is left of them. Other kinds of spots which are reddish in colour and angular are also found. This variability in symptoms could be due to genetic variability. Other parts of the coffee plant such as stems, branches and fruits may also get infected (photo 71).

*M. citricolor,* a Basidiomycetes of the Order Agaricales (Dennis, 1950), produces two kinds of fruiting bodies: gemmae, which are produced by vegetative propagation and basidiocarps, resulting from reproduction. The gemmae are lemon yellow in colour and made up of a mycelial pedicel with a globular head at its tip (which is itself mycelial) giving them a pin-head like appearance. They are 5-10 mm high and a few mm in diameter and constitute the infective component of the fungus. The pin-head (or large spore) gets detached on maturity. Basidiocarps are less common. They are found mostly on lesions of fallen leaves and are more than 5 mm high.

The disease is found in old plantations which have never been pruned and in shaded coffee plantations. *M. citricolor* has a large number of hosts (more than 500). Among them, there are many self-propagating fruit trees such as orange and cocoa. Avelino *et al.* (1995) reported that the disease is capable of causing considerable damage in coffee plantations, especially above elevations of 1200 m in countries near the equator: firstly, a direct loss due to the fall of fruits at the end of the period of physiological fall considered as normal and secondly, an indirect loss induced by defoliation which affects the production of the following year, i.e., a total loss of 36% of coffee berries in two years.

As its name indicates, the disease is found only in the American continent and in the Caribbean. In Costa Rica, growing coffee without shade has helped to reduce the damage caused by this malady. However, in the last few years American coffee disease has once again become very serious in Costa Rica and in some parts of Guatemala where the plantations are without shade.

### Black stripe disease of hevea

The black stripe symptoms on hevea are caused by the growth of *Phytophthora palmivora* on the tapping panels. The disease begins with the appearance of cankerous changes on the bark accompanied by exudation of latex above the tapping cut (photo 72). The black stripes appear a few days after the beginning of the infection. The bark peels off from the wood and the intervening space gets filled with latex. This latex ferments without coagulating and oozes out as a thick brown foul-smelling liquid. If the bark is removed at this place a series of thin black parallel lines can be observed extending across the tapping panel above the notch. These black lines extend up to the wood in the form of blackish scales. From the point of infection, the infection spreads downwards to areas which have not yet been tapped and upwards in the young regenerated bark (Delabarre and Serier, 1995).

This disease is found in all the rubber-growing regions. The abundance of infection points on the same tapping panel destroys vast areas of regenerating bark, disrupts the normal functions of the tree and reduces the yield and quality of the latex. In the absence of preventive measures, the disease completely destroys the tapping panel resulting in the loss of the tree for exploitation.

### Witches' broom disease of cocoa

The organism responsible for causing witches' broom disease of cocoa is a Basidiomycetes fungus called *Crinipellis perniciosa* (Stahel) Singer. The characteristic feature of this pathogen is that it attacks the vegetative tips of the plant, especially the shoot primordia, flower buds and cambial layers. The pathogen induces anomalous growth and hypertrophy of the tissues, giving the characteristic witches' broom appearance (photo 73). It also infects the young developing cocoa fruits and pods, sometimes resulting in heavy loss of

pods (Thorold, 1975). A broom may be composed of a large number of infected shoots with short internodes and bear malformed leaves.

The parasite may also produce swollen lesions on the stems, without forming brooms, which are comparable to cankers. The infection causes a hypertrophy of the cushion flowers which give out vegetative shoots and abnormal star-shaped flowers, or star blooms, the whole looking like a cushion broom (photo 74). These infected floral cushion flowers produce abnormal young fruits that look like strawberries, or *chirimoyas,* which do not contain any beans and degenerate very quickly.

The spectacular nature of the infections on stems and formation of brooms have sometimes masked the intensity of infection on the young fruits and pods, which suffer the most. The symptoms on the pods are often confused by growers with those produced by *Phytophthora* or *Moniliophthora,* thus diminishing the actual impact of witches' broom disease (Evans, 1991). The infected pods are poorly developed and do not mature resulting in the total loss of yield.

This disease was reported for the first time in Surinam in 1880. It was responsible for the abandonment of cocoa cultivation in this country as well as in Guyana (South America). It is now the main limiting factor for cocoa production in many South American countries. Very little precise data is available to estimate the losses caused by witches' broom disease. Nevertheless an infection rate of 68% of pods reported from Trinidad gives an idea of the magnitude of the problem (Baker and Holliday, 1957). The death of branches and banchlets due to the disease also intervenes in the productive potential of the tree.

Losses of 80% of the pods were reported in Ecuador in the 50s and 60s. In the state of Rondonia in Brazil, high-yielding clones suffered 90% infection of the pods (Evans, 1991).

Witches' broom disease made its appearance in the late 80s in Bahia State in Brazil, the traditional cocoa-growing region of the country, causing extensive damage. The production fell from 300,000 to 180,000 tons of cocoa in 1997, leading to the abandonment of some plantations. Witches' broom disease is therefore a very serious threat for cocoa cultivation in Latin America, for which only varietal breeding can provide a satisfactory solution.

### Stem bleeding of coconut

The first sign of stem bleeding is manifested by a blackish, more or less extensive spot on the stem. A blackish-brown liquid oozes out from the middle of this spot, blackening the stem over a length which may exceed 50 cm (photo 75). These irregular trails correspond to a light brown to black rotting of the stem tissues. This rot may affect a large portion of the stem as much in depth as in height. On aging the rot becomes fibrous and the cavity formed sometimes contains a clear liquid under pressure which spurts out in a jet if, for example, the bark is cut with a knife. In case of extreme dryness,

we often observe the drooping leaves hanging along the trunk and premature nut fall. In extreme cases of extensive internal infection, the stem may break. In most cases, the internal rot dries up and the infected walls get cicatrised, which is a prelude to healing. However this internal cavity weakens the stem of the coconut tree. Variations of this symptom have been observed. They are seen as swellings on the stem containing brown gummy and granular concretions. This kind of symptom was considered to be the dormant symptom of stem bleeding.

In Indonesia, two mineral nutrition experiments demonstrated the important role of chlorine deficiency in the appearance of stem bleeding symptoms (Renard *et al.*, 1984), especially during periods of extreme drought. A good supply of chlorine to the coconut tree is an effective approach for controlling stem bleeding and dryness. Lastly, stem bleeding, which is often associated with the presence of *Thielaviopsis paradoxa* (de Seynes), appears more like a physiological disorder, favouring the establishment of weak parasites, rather than being a disease per se.

## Bacterial diseases

### Black spot disease of mango

Although the symptoms on leaves and fruits are by far the most commonly observed symptoms, *Xanthomonas* sp. *mangiferaeindicae* may cause lesions on all the aerial parts of the mango tree. The first visual signs on the leaves are small angular oily-looking spots measuring 1-3 mm and delimited by the veins. They then turn black and appear in relief on both sides of the leaves (photo 76). Most often young lesions have an oily-looking margin and are surrounded by a chlorotic halo. Old foliar lesions are brown and then become ash grey.

*X.* sp. *mangiferaeindicae* does not cause tree mortality but reduces the yield of susceptible cultivars, sometimes drastically, due to very heavy defoliation. Disease incidence is acute on these cultivars in all regions with concomitant hot and humid periods. The global distribution of mango black spot disease is given in Fig. 5. The disease was first observed on herbarium specimens collected from India in 1880 (Pruvost, 1989; Pruvost and Manicom, 1993; Pruvost *et al.*, 1995).

### Bacterial canker of citrus

Symptoms of bacterial canker caused by *Xanthomonas axonopodis* pv. *citri* have been observed on leaves (photo 77), stems (photo 78), spines and fruits. Their morphology is quite similar on all these parts. The lesions become visible a few days after infection during the dry season. They are seen as water-soaked spots which evolve into small, white, slightly raised specks. The lesions then grow radially and become beige in colour with a corky texture and are

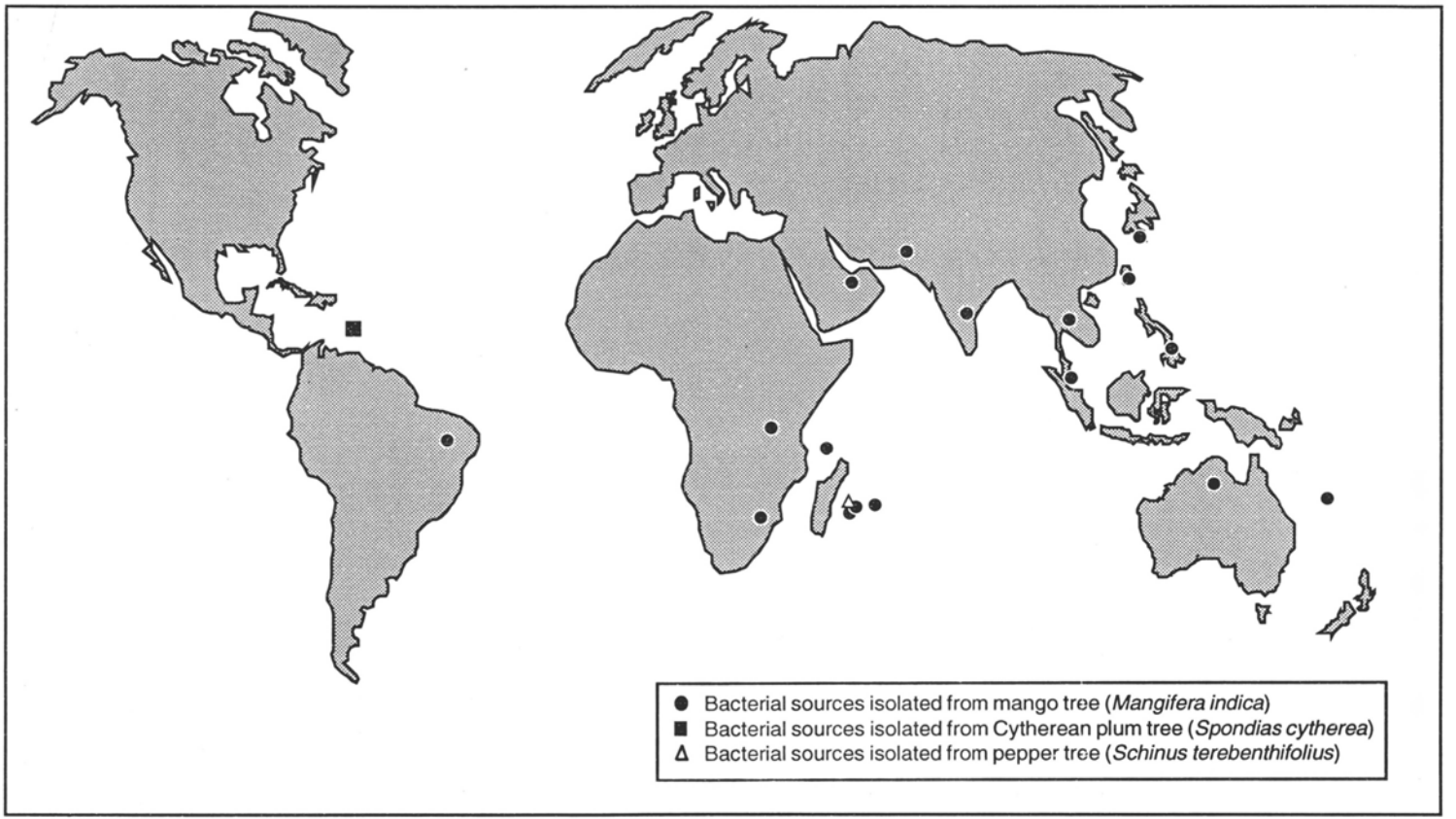


**Fig. 5.** Global distribution of *Xanthomonas* diseases on Anacardiaceae.

slightly raised (hypertrophy, hyperplasia). Young lesions are most often bordered by an oily looking margin. A chlorotic halo is usually visible around these lesions. Old lesions may be 1 to 1.5 cm in diameter. The symptomatology is quite characteristic and there is not much risk of committing mistakes in the visual diagnosis (Vernière, 1992).

*Xanthomonas axonopodis* pv. *citri* does not kill the trees but it can cause fairly severe defoliation in susceptible hosts. Generally speaking, the incidence of citrus bacterial canker on these citrus cultivars is high in all regions where the hot and humid seasons are concurrent. The potential gravity of this disease has led some countries (United States and member countries of the European Union) to impose strict quarantine regulations (Vernière, 1992). The global distribution of bacterial canker is given in Fig. 6. This disease was first observed on herbarium specimens collected in India in 1830. It is most probably of Asian origin.

## FRUIT DISEASES

Although these diseases do not affect the life of the tree, the yield may be partly or almost completely destroyed. Most of these infections are caused by fungi.

### *Phytophthora* rots

BROWN POD ROT OF COCOA

Brown rot of cocoa pods is curiously called black pod rot by English speaking authors, which may lead to a confusion with the black rot of cocoa pods. It is caused by several species of *Phytophthora*, but for a long time it was believed to be only one species, viz., *P. palmivora* (Butler) Butler. Many other species are also responsible for brown rot. *P. citrophthora* is found in the Ivory Coast, *P. capsici* is abundant in Brazil, *P. megakarya* (Brasier et Griffin) is found only in Central Africa (Cameroon, Gabon, São Tomé and equatorial Ghana) and West Africa (Nigeria, Togo and Ghana). *P. megasperma*, which is confined to Venezuela, causes a soft rot. Brown rot is one of the most serious diseases of cocoa because of its economic impact, the fruits alone getting destroyed whatever may be the stage of their development (photo 79). It is also one of the most spectacular because of its sudden and inevitable appearance with rainfall. The infection generally begins at the distal or peduncular end of the fruits. A necrotic spot with a translucent halo delimits the infected zone which becomes brown more or less rapidly depending on the varieties (Blaha and Lotodé, 1976). The beans are affected even before the fruit rots fully, which takes about 3 to 10 days. The sporocysts which are produced on the necroses are powdery and greasy to the touch. They liberate the zoospores which

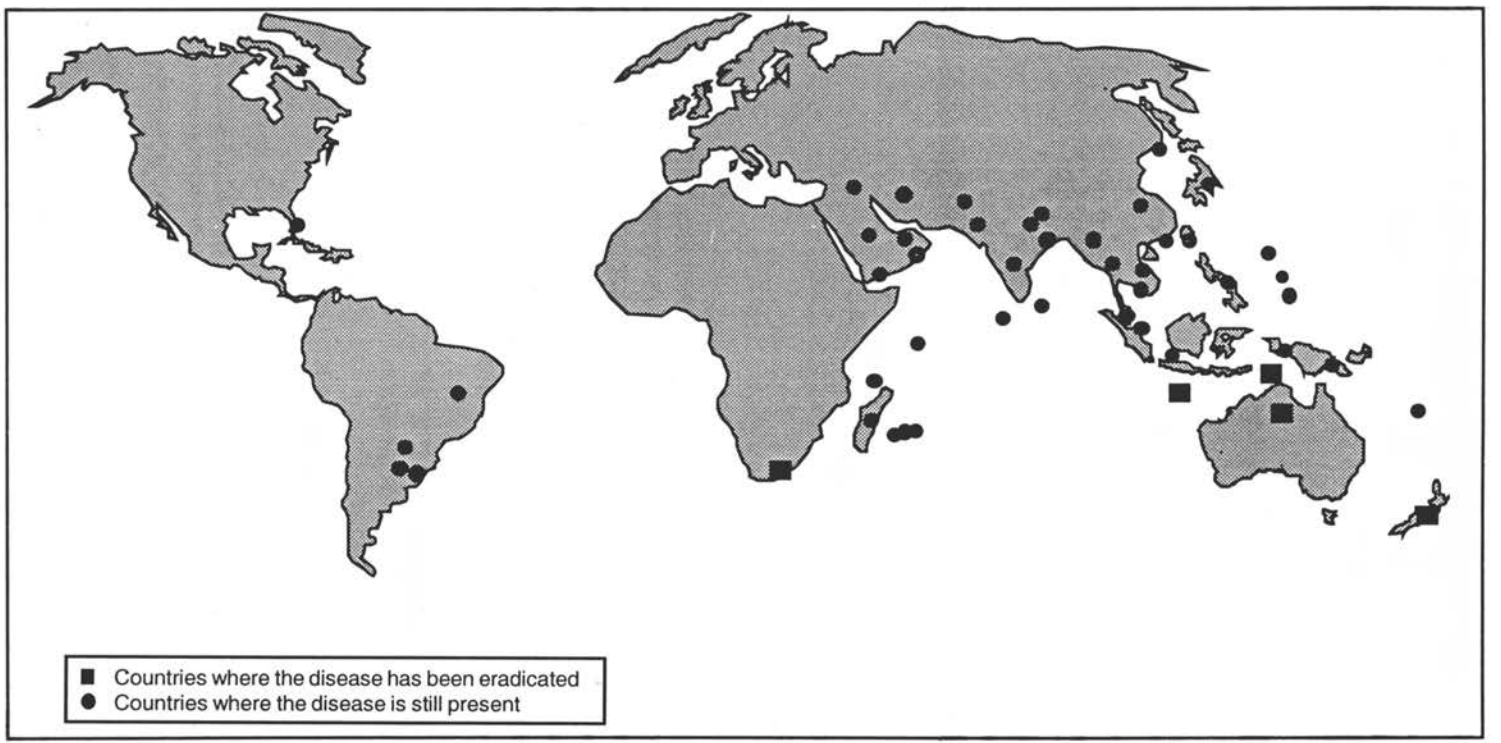


Fig. 6. Global distribution of Asian citrus canker.

infect the neighbouring fruits. The intensity of autoinfection by pod to pod contamination is related to the density of fruits on the trunk. Cauliflory makes the pods on the trunk more vulnerable to infection than those on fronds where the different spatial position may delay the epidemic kinetics by one to two weeks (Costa Deus Lima *et al.*, 1994) .

Although the symptoms of infection induced by the different species conform globally to the description just given, they can nevertheless be distinguished. For example, the infection front is a regular line in the case of *P. palmivora* while it is very sinuous with isolated diseased areas in front of the general infection line in the case of *P. megakarya* (photo 80). The habit of citing brown pod rot as a pathological entity may be eventually changed in order to attribute a specific disease to each of the species involved.

At the global level, the losses may be as high as about 30% of pod production. They may vary from one country to another: 15-20% in West Africa, Ivory Coast and Ghana; 30-75% in Nigeria; more than 50%, even 80-95% in cocoa farms in Cameroon and *P. megakarya* dominated areas in Gabon and equatorial Guinea (Jouve and Milly, 1990). In Latin America, the losses could be equally severe on the economic scale: 20-30% in Brazil, 20% in Venezuela, 20-40% in Mexico (Ortiz-Garcia *et al.*,1994). In South-East Asia (Malaysia and Indonesia) and the South Pacific (Papua New Guinea), the steep increase in the area under cocoa cultivation during the last fifteen years and counter-selections have now made the impact of *Phytophthora* diseases much more perceptible (Blaha, 1996).

### *Phytophthora* rot of coconut

*Phytophthora* rot of coconut is characterised by mottling which is light brown in the middle, yellow at the periphery and oily in appearance (photo 81). The infection generally begins around the floral parts or in the equatorial part of the coconut, particularly in the region of contact between the nuts. On the surface the rot extends towards the apex of the nuts and in depth towards the shell. The infected husk turns form brown to black. The rot may enter through the germinating pores of the coconut, killing the embryo and lysing the albumen. At a more or less advanced stage of the infection the nuts fall down and their kernel cannot be used for preparing copra.

Nut rot is independent of bud rot and, depending on the place and cultivars, the two kinds of rot may or may not coexist on the same tree. It may cause the premature fall of 20 to 25% of the immature nuts in some village plantations of Yellow Dwarf × West African Tall hybirds in the Ivory Coast (Franqueville and Renard, 1989). In some seed-gardens of Yellow Dwarf coconut in Indonesia, 50% nut fall has been recorded (Thévenin *et al.*, 1995).

## Black pod rot of cocoa

Black rot of cocoa pods is generally attributed to *Botryodiplodia theobromae* Pat.

Necrosis caused by *B. theobromae*, which affects the cortex of the fruits (photo 79) and then the pulp and beans gives rise to a characteristic soft rot and hence the term soft pod rot often used to designate this rot. In the final stage, the pod is completely covered by a black powder which looks like soot and so there is no room for confusion with any other kind of rot. Considered a parasite of wounds or a weak parasite (Blaha, 1989), the fungus generally causes very little damage to the pods. On other parts this fungus may be found associated with other microorganisms such as *Calonectria rigidiuscula*, which it accompanies in the descending trachaeomycosis of cocoa capable of causing the death of the tree. This fungus enters through wounds caused by bug bites.

## Moniliosis of cocoa pods

The symptoms of moniliosis are observed mainly on the pods. The first signs of the disease are manifested by the appearance of oily specks. Deformation of pods (photo 82) follows the intracellular growth of the parasite fungus (*Moniliophthora roreri* Cif. et Par.), which stimulates cambial activity. Brownish-black irregular lesions then appear and quickly invade the entire surface of the pods. A white mycelium grows on the surface, followed by a profuse production of spores which form a thick creamish to greyish-brown coloured felt. The internal tissues become disorganized. A wet rot develops and the cocoa beans cannot be used (Thévenin and Trocmé, 1996; photo 83). The perfect stage of this fungus is not known. Reproduction is by means of conidia produced in basipetal chains from the mycelium. *M. roreri* is a semi-biotrophic fungus because its life cycle passes through two phases: one is the germination of conidia at the time of intercellular invasion of the pods, and the other is the necrotic phase when the fungus invades the cells. One of the important characteristics of the disease is the very long incubation period, which is 3 to 6 weeks depending on the age of the fruit. The long incubation period of the spores and their transport over long distances are the important elements which govern the formulation of strategies for controlling this disease.

The disease was reported for the first time in Ecuador in 1914 (Desrosiers *et al.*, 1955) and was responsible for a steep fall in production in the 20s (Petithuguenin and Roche, 1995). The disease then spread to Peru and later to Colombia and Venezuela. In 1956 it appeared in Panama (Orellana, 1956) and in 1978 in Costa Rica (Enriquez and Suarez, 1978). For a long time confined to the west of the Andean range, the disease was reported in the zone east of Peru in 1988 (Hernandez *et al.*, 1990). Losses are always heavy and may affect 90% of the harvest. An average of 15 to 50% is common in many countries (Evans, 1986).

## Anthracnosis of fruits

### Anthracnosis of mango, avocado and papaya fruits

Anthracnose of mango, avocado and papaya fruits is the term commonly used to describe the disease caused by *Colletotrichum gloeosporioides* (Penzig) Sacc., a parasite common to these three fruits. In many regions this infection is considered to be one of the most important limiting factors affecting the quality of the fruits and making them either unsuitable for export or unmarketable when they reach the distribution circuits (Mourichon, 1987).

Although the characteristic symptoms of this disease appear only at a late stage in the development of the fruits and generally after harvesting, the contamination may have taken place at the time of fruit setting and during the months following it. This infection takes place by means of conidia issuing from cankers on stems or from foliar necroses and transported by rain water (droplets, streaming). The conidia germinate on the surface of the fruits and produce a special penetrating structure called the appressorium. The fungus then stops growing at this stage and undergoes a latent period which may last several months, depending on whether the variety is of the early or late type.

### Anthracnosis of Arabica coffee berries (coffee berry disease)

*Colletotrichum kahawae* sp. nov. (Waller *et al.*, 1993) is responsible for causing anthracnose of Arabica coffee berries. The disease is manifested on fruits at all the stages of their development, but only the symptoms on young fruits enable an accurate diagnosis. Two kinds of lesions may be observed.

Lesions called 'active', are brown and slightly shallow, which coalesce and give rise to a wet rot of the pulp and seeds and ultimately fruit fall (photo 84). When atmospheric humidity is high, pink coloured acervuli of the parasite appear in a concentric manner on the surface of the spots and liberate the conidia which are the dispersal organs of the disease.

Lesions of the scab type generally increase in number during the prematuration period. These spots are slightly shallow with irregular edges, light brown in colour and have a slightly corky aspect. They do not have a serious effect on the yield nor on the quality of the seeds.

The damage caused by *C. kahawae* may be confused in South America, especially in Brazil, with the blackening of berries resulting from the anthracnose of leaves and branches (dieback) caused by another *Colletotrichum* sp.

Anthracnose of coffee berries, or coffee berry disease, is strictly restricted to *Coffea arabica* and is specific to fruits. When infection is severe the leaves and branches remain free of lesions. This particular feature should be considered as one of the important elements for a correct diagnosis (Muller, 1980). The disease, which was discovered in north-west Kenya in 1922 (MacDonald, 1926), gradually spread to eastern Kenya in 1939 and then to most countries in East and Central Africa: to the Democratic Republic of Congo in 1937, Cameroon in 1955, Rwanda in 1957, Uganda in 1959 and Tanzania in 1964.

The disease was reported in Ethiopia only in 1971 and in Malawi, Zimbabwe and Zambia in 1985. This disease confined to the African continent and only to the coffee-growing regions at high altitudes, affects only a small part of the producing regions at the global level. However, it is a formidable scourge which can destroy more than 80% of the production when conditions are favourable for the parasite (Aubin *et al.*, 1993). This is why active research is being continued as it still represents a serious potential threat for Arabica-producing countries outside Africa. Very often the presence of black berries borne on wilted branches lead inexperienced observers to think that it is this disease whereas it is only dieback.

The coincidence of climatic factors favourable for the development of the parasite with the optimal susceptible phases of the fruit determine the gravity of the disease (Berry *et al.*, 1991), as well as the strategy for the application of phytosanitary treatments.

## Bacterial diseases of mango and citrus fruits

Bacterial diseases found on the leaves also develop on the fruits.

In the case of black spot of mango caused by *Xanthomonas* sp. *mangiferaeindicae*, the first symptoms on the fruits appear in the form of small water soaked lesions concentrated around lenticels or wounds. They then become black with a crater-like appearance (photo 85). They are most often 8 to 10 mm in diameter but can attain 15 mm. The lesions extend to a depth of about 8 to 10 mm inside the flesh. Very often a highly infectious gummy substance is exuded, causing secondary infection along its flow (Pruvost *et al.*, 1995). This disease induces a heavy fall of young fruits and even if they mature it is impossible to market them.

Bacterial citrus canker, caused by *Xanthomonas axonopodis* pv. *citri*, induces small lesions on the fruits (photo 86), which are quite similar to those observed on the leaves. Only the superficial tissues of the fruits are affected. This disease greatly depreciates the external quality of the fruit. In susceptible cultivars the yield may also be considerably reduced due to the fall of unripe fruits.

# CONCLUSION

The study of plant diseases has been presented first by an analysis and detailed descriptions of the symptoms, which are summarised in Table 1, followed by the identification of the causal organism.

This observation phase accompanies the development of the disease, over time (on the plant) and space (in the plantation or in a cultivated or natural population of the species). Furthermore, the incidence of the parasite on the

**Table 1.** Major diseases of tropical tree crops and their symptoms

| Plants | Roots, collar | Trunk, stems, branches | Leaves | Fruits |
|---|---|---|---|---|
| Citrus | Gummosis (*Phytophthora*) | Gummosis<br>Tristeza (virus) Stem pitting<br>Decay (*Ceratocystis*) | Greening (bacteria)<br>Tristeza (wilt)<br>Wilt (*Ceratocystis*) | Greening (bacteria)<br>Bacterial canker (*Xanthomonas*) |
| Fruit trees | Avocado wilt<br>(*Phytophthora*) | | Avocado wilt (secondary)<br><br>Black spot of mango (bacteria) | Anthracnose of fruits<br>(*Colletotrichum*)<br>Black spot of mango |
| Oil palm | Marchitez<br>(trypanosomatids)<br>Basal rot (*Ganoderma*) | <br>Heart rot (pathogen?)<br>Fusarium wilt (*Fusarium*)<br>(in section)<br>Red ring (nematodes) | Marchitez (secondary)<br>Ring spot<br>Fusarium wilt (secondary effect)<br>Cercosporiosis | Marchitez (secondary) |
| Coconut | Hartrot (trypanosoma) | <br>Heart rot (*Phytophthora*)<br>Dry bud rot (virus?)<br>Red ring (nematode) | Hartrot (secondary)<br>Helminthosporiosis<br>Dry bud rot<br>Lethal yellowing (phytoplasma)<br>Cadang-cadang (viroid) | Hartrot (fall)<br>*Phytophthora*<br><br>Lethal yellowing (nut fall) |
| Coffee | Wilt (*Armillaria*)<br>Wilt (nematodes) | Trachaeomycosis (*Fusarium*)<br>(visible in section) | Trachaeomycosis (secondary effect)<br>Decay (secondary effect)<br>Orange rust (*Hemileia*)<br>American disease (*Mycena*) | Berry anthracnose<br>(*Colletotrichum*) |
| Cocoa | | Witches' broom<br>(*Crinipellis*) (*shoot tips*) | Swollen shoot (virus) | Brown pod rot (*Phytophthora*)<br>Moniliosis (*Moniliophthora*) |
| Hevea | Fomes (*Rigidoporus*)<br>Armillaria (*Armillaria*) | Black stripe (*Phytophthora*) | South American leaf blight<br>(*Microcyclus*)<br>Anthracnose (*Colletotrichum*)<br>*Corynespora* | |

vegetative growth has also been noticed, as also the consequences on the production in order to assess the losses in yield.

During the last fifty years a lot of information has been acquired regarding diseases of tropical tree crops. Considerable progress has been made in combating these diseases, through the selection of tolerant or resistant varieties, agronomic methods and chemical control strategies.

Despite the spectacular results, the spread of these diseases remains a reality. The parasites can overcome resistance factors learnedly incorporated into new varieties, can adapt to the new chemical molecules developed and reach new territories and new continents till then untouched by the disease. Diseases, hitherto unreported, have appeared and the etiology of some of them is still not known.

This general situation is partly due to the extraordinary development of agriculture and its intensification in the tropical zone. It is also the consequence of the formidable power of almost all the causal organisms to adapt thanks to their extreme genetic diversity. Finally, the plant and its pathogen carry on a relentless battle in the centre of which the plant pathologist and other specialists have a preponderant place. At present descriptive studies are increasingly giving place, out of necessity, to extensive researches on the structure of pathogen populations, their variability and host-parasite relationship. The second part of this work presents the knowledge acquired in these domains by CIRAD during the last twenty years with respect to the most important pathogens of the tree crops studied.

By giving preference to integrated control methods, researches on the causal organisms help to envisage the development of tolerant varieties adapted to different ecological situations. With the development of in vitro culture and new genetic techniques used for obtaining resistant varieties, the agriculturist will have a wide array of control methods at his disposal in the third millennium.

## REFERENCES

Alibert, H. 1946. Note préliminaire sur une nouvelle maladie du cacaoyer, le *swollen shoot*. L'Agronomie Tropicale **1:** 34-43.

André, M., Berry, D., Ducamp, M., Bieysse, D., Massimino, J. and Muller, R.A. 1989. Innovations récentes en matière d'étude de la physiologie du caféier. In: XIII[e] colloque scientifique international sur le café, Paipa, Colombie, 29 juin-3 juillet. Asic, Paris, France, pp. 600-613.

Arbonnier, M. 1995. Evaluation de l'état sanitaire des plantations forestières de Guyane. Rapport de mission du 6 au 13/10/1995, CIRAD-forêt, 23 pp.

Ashburner, R., Cordova, I. and Oropeza, M.C. 1996. First report of coconut lethal yellowing disease in Honduras. Plant disease **80 (8):** 960.

Assas M'Billaut, G. 1978. La pourriture brune des racines d'avocatier. Etude histologique de la pénétration et de l'infection des racines par *Phytophthora cinnamomi*. Fruits **2:** 107-115.

Attafuah, A., Blencowe, J.W. and Brunt, A.A. 1963. *Swollen shoot* disease of cocoa in Sierra Leone. Trop. Agric. Trin. **40:** 229-232.

Aubert, B. 1988. Le *greening*, une maladie infectieuse des agrumes. Stratégie de lutte développée à l'île de la Réunion. Circonstances épidémiologiques en Afrique/Asie et modalités d'intervention. Irfa-CIRAD édit. Setco Paris, France. 185 pp.

Aubert, B. 1992. Citrus greening disease, a serious limiting factor for citriculture in Asia and Africa. Proc. Int. Soc. Citriculture **2:** 817-820.

Aubert, B., Etienne, J., Cottin, R., Leclant, F., Cao Van P., Vuillaume C., Jarmillo, C. and Barreau G. 1992. Citrus tristeza disease, a new threat for the Caribbean basin. Report of a survey to Colombia, Dominican Republic, Guadeloupe, Martinique and Trinidad. Fruits **36 (1):** 1-12.

Aubin, V., Berry, D., Cilas, C. and Mouen Bedimo, J. 1993. Anthracnose des baies du caféier arabica au Cameroun: estimation du délai de chute des baies malades. Association scientifique internationale du café. *In:* XVI[e] colloque scientifique international sur le café, Montpellier, France, 6-11 juin 1993: 807-811.

Avelino, J., Toledo, J.C. and Medina, B. 1995. Desarrollo del ojo de gallo (*Mycena citricolor*) en una finca del Norte de Guatemala y evaluación de los daños provocados por esta enfermedad. Simposio sobre caficultura latino americana, Costa Rica, lica, 1993, XVI.

Avelino, J., Muller, R.A., Cilas, C. and Velasco, H.P. 1991. Développement et comportement de la rouille orangée de caféier (*Hemileia vastatrix* Berk. et Br.) dans des plantations en cours de modernisation, plantées de variétés naines, dans le sud-est du Mexique. Café, Cacao, thé **35 (1):** 21-37.

Bachy, A. 1958. Le *blast* des pépinières du palmier à huile. Observations et moyens de lutte. Oléagineux **13 (8-9):** 653-660.

Bachy, A. and Hoestra, H. 1958. Contribution à l'étude de la maladie de Kaincopé du cocotier, au Togo. Oléagineux **13 (10):** 721-729.

Baker, R.E.D. and Holliday, P. 1957. Witches' broom disease of cocoa. Phytopathological paper n° 2. Commonwealth Mycological Institute, Kew, UK.

Berry, D., Bieysse, D. Andre, M. and Muller, R.A. 1987. Photosynthèse et photorespiration du caféier (*Coffea arabica* L.): effets d'une attaque de rouille (*Hemileia vastatrix* Berk et Br). *In:* XII[e] colloque scientifique international sur ie café, Montreux, Suisse, 29 juin-3 juillet. Asic, Paris, France, pp. 600-613.

Berry, D., Nankam, C., Mouen Bedimo, J. and Aubin, V. 1991. L'anthracnose des baies du caféier arabica (Cbd, *coffee berry disease*) au Cameroun: épidémiologie et lutte chimique, Asic, 1991. *In:* XIV[e] colloque, San Francisco, USA, pp. 499-509.

Bigornia, A.E. 1977. Evaluation and trends of researches on the coconut cadang-cadang disease. Philippines journal of coconut studies **2:** 5-34.

Blaha, G. 1978. *Clitocybe* (Armillaria) *elegans* Heim. Un grave pourridié dus caféier arabica au Cameroun. Café, cacao, thé **22 (3):** 203-216.

Blaha, G. 1982. Mission sur la pourriture brune des cabosses du cacaoyer au Gabon. Prospections et identifications; proposition pour un programme d'étude et d'amélioration. Rapport de mission (15 novembre-5 décembre), 75 pp.

Blaha, G. 1989. Etat phytosanitaire de la plantation cacaoyère de Njolé à Koulamoutou (province de l'Ogoué-Solo). Rapport de mission au Gabon auprès de la Socagab, 19-30 mai, 68 pp.

Blaha, G. 1996. PNG's experience on diseases of cocoa: management through knowledge of use of chemical treatments and resistant plant material. *In:* XIIth International cocoa research conference, Salvador, Bahia, Brazil.

Blaha, G. and Lotodé, R. 1976. Un critère primordial de sélection du cacaoyer au Cameroun. La résistance à la pourriture brune des cabosses (*Phytophthora palmivora*), variations de réactions à la maladie en liaison avec les données écologiques et l'état physiologique des fruits. Café, cacao, thé, **20 (2):** 97-116.

Boccas, B. and Laville, E. 1978. Les maladies à Phytophthora des agrumes. Setco, Paris, France, 162 pp.

Bonheure, D. 1998. Le théier. Le technicien d'agriculture tropicale. Maisonneuve et Laroes, Paris, France, 160 pp.

Bové, J-M., 1995. Virus and virus like disease of Citrus in the near East regions. FAO Rome, Italy, 518 pp.

Breton, F., Garcia, D., Sanier, C., Eschbach, J.-M. and D'Auzac, J. 1997. L'interaction entre *Corynespora cassiicola* et *Hevea brasiliensis*. Plantations, recherche, développement **4 (5):** 322-335.

Brun, J. 1972. La cercosporiose des agrumes provoquée par *Cercospora angolensis*. Fruits **27 (7-8):** 539-541.

Brun, J. 1975. Le chancre de l'avocatier provoqué par *Phytophthora cinnamomi* Rands. Fruits **5:** 339-344.

Calderon-Vega, M. 1989. Reacciones de diferentes genótipos de café a *Meloidogyne arabicida* López y Salazar, gama de hupendantes y hongos fitopatógenos asociados. Tesis M.Sc., universidad de Costa Rica. Catie, Turrialba, Costa Rica, 62 pp.

Calvez, C., Renard, J-L. and Marty, G. 1980. La tolérance du cocotier hybride Local × Rennell à la maladie des Nouvelles Hébrides. Oléagineux **35 (10):** 443-449.

Campos, V.P., Sivapalan, P. and Gnanapragasam, N.C. 1990. Nématode parasites of coffee, cocoa and tea. *In:* Plant parasitic nematodes in subtropical and tropical agriculture. M., Luc, R.A. Sikora and J. Bridge (eds). CAB international, Wallingford, UK, pp. 387-430.

Chee, K.H. 1998. *Corynespora* Leaf Spot. Planter's Bulletin **194:** 3-7.

Chee, K.H. and Holliday, P. 1986. South American leaf blight of hevea rubber 10, 50 pp. Irrdb Monographs n° 13. Kuala Lumpur, Malaysia, Irrdb.

Costa Deus Lima, A.M., Cilas, C. and Blaha, G. 1994. Etude épidémiologique de la pourriture brune des cabosses et évolution des dégâts de *Bathycoelia* spp. dans les cacaoyères de São Tomé. *In:* XIth International Cocoa Research Conference, Yamoussoukro, Côte d'Ivoire, pp. 111-116.

De Carvalho, T. and Mendes, O. 1953. Una nova especie de Cercopora em *Citrus sinensis* Osbeck. Boi. Soc. Broteriana, Ser. **2 (27):** 201-202.

Delabarre, M.C. and Serier, J-B. 1995. L'hévéa. Collection Le technicien de l'agriculture tropicale. Maisonneuve et Larose, Paris, France, 238 pp.

Dennis, R.W.G. 1950. An earlier name for *Omphalia flavida* Maub. et Rangel. Kew Bulletin **3:** 434.

Desrosiers, R., Buchwald, A. and Bolanos, C. 1995. Influence des précipitations sur l'incidence en Equateur de la moniliose des cabosses du cacaoyer. Bull. Phytosanit. FAO **3 (11):** 161-164.

Dollet, M., Giannotti, J., Renard, J-L. and Ghosh. 1997. Etude d'un jaunissement létal des cocotiers au Cameroun: la maladie de Kribi. Observations d'organismes de type mycoplasmes. Olégineux **32 (7):** 317-322.

Dollet, M. and Lopez, G. 1978. Etude sur l'association de protozoaires flagellés à la *marchitez sorpresiva* du palmier à huile en Amérique du Sud. Oléagineux **33 (5):** 209-217.

Dzido, J-L., Genty, P. and Ollagnier, M. 1978. Les principales maladies du palmier à huile en Equateur. Oléagineux **33 (2):** 55-63.

Enriquez, C.G.A. and Suarez, C.C. 1978. *Monitia*, disease of cocoa in Costa Rica. Turrialba **28 (4) :** 339-340.

Evans, H.C. 1986. A resassessment of *Moniliophthora* (Monilia) pod rot of cocoa. Cocoa Growers' Bulletin **37:** 34-43.

Evans, H.C. 1991. Witches' broom disease, a case study. Cocoa Growers' Bulletin **32:** 5-19.

Franqueville, H. de. 1991. Antécédent savane ou antécédent forét: influence des isolats du pathogène sur le comportement des lignées de palmier à huile vis-à-vis de la fusariose. Oléagineux **46 (5):** 179-186.

Franqueville, H. de and Renard, J-L. 1989. Intérét du phoséthyl-Al dans la lutte contre le *Phytophthora* du cocotier. Modalités d'application. Oléagineux **44 (7):** 351-358.

Franqueville, H., de Renard, J-L., Philippe, R. and Mariau, D. 1991. Le *blast* du palmier à huile et perspectives en vue de l'amélioration de la méthode de lutte. Oléagineux **46 (6):** 223-231.

Guyot, J. 1997. *Armillaria* spp., pourridié de l'hévéa en Afrique centrale: le cas du Gabon. Plantations, recherche, développement **4 (6):** 408-419.

Hernandez, T.T.A., Aranzazu, F., Arevalo, G.E. and Rios, R.R. 1990. La moniliasis del cacao en Perú. Agrotropica **2 (1):** 56-58.

Holliday, P. 1970. South American leaf blight (*Microcyclus ulei*) of *Hevea brasiliensis*. Kew, Surrey, UK. Phytopathological papers nº 12. Commonwealth Mycological Institute, 30 pp.

IRHO. 1989. Les maladies foliaires du cocotier au Brésil. Oléagineux. Rapport d´activités IRHO **44 (4):** 117-118.

Jouve, P. and Milly, H. de. 1990. Compétitivité du cacao africain Analyse du marché mondial et des principaux producteurs. Rapport d´étude du ministère de la coopération et du développement, 279 pp.

Kuate, J., Bell Mangua, Foure, E. and Rey, J-Y. 1994. Symptômes de la cercosporiose des agrumes due à *Phaeoramularia angolensis*. Fruits **49 (1):** 31-36.

Lanier, L., Joly, P., Bondoux, P. and Bellemère, A. 1976. Mycologie et pathologie forestières. Masson, Paris, France, tomes 1 et 2, 487 pp, 478 pp.

Laville, E. 1962. Infestation expérimentale des jeunes plantules de palmier-dattier par *Fusarium oxysporum* var. *albedinis*. Fruits **17 (2):** 88-90.

Laville, E. 1974. Les maladies à *Phytophthora* des agrumes en Corse. Fruits **4:** 297-301.

Laville, E. 1984. La gommose à *Phytophthora* des clémentiniers en Corse. Arboriculture fruitière **2:** 32-35.

Laville, E. and Lossois, P. 1963. Méthode de Van der Plank et mode de propagation du bayoud. Fruits **18:** 249-253.

Leakey, C.L.A. 1965. Sudden death disease of cocoa in Uganda, associated with *Verticillium dahliae* Kleb. EAAFRO Journal, **31 (1).**

Legg, J.T. 1979. The cocoa swollen shoot research project at the Cocoa Research Institute, Tafo, Ghana. General report 1969-1978. Overseas Develop. Admin. London, UK.

Lopez, G., Genty, P. and Ollagnier, M. 1975. Contrôle préventif de la *marchitez sorpresiva* de l´*Elaeis guineensis* en Amérique latine. Oléagineux **30 (6):** 243-250.

Lopez, R. and Salazar, L. 1989. *Meloidogyne arabicida* sp. n. (Nemata: Heteroderidae) nativo de Costa Rica: un nuevo y severo patógeno del cafeto. Turrialba (CR) **39 (3):** 313-323.

Mac Donald, J. 1926. Fungoid disease in Kenya colony. Colony and Protectorate of Kenya, Department of Agriculture 3, 17 pp.

Mallet, B., Geiger, J-P., Nandris, D., Nicole, M. and Renard J-L. and Tran Van Canh. 1985. Les champignons, agents de pourridiés en Afrique de l´Ouest. Sonderdruck aus European Journal of Forest Pathology **15 (5-6):** 263-268.

Mariau, D. 1978. Informe de visita de la plantación Esperanza (Venezuela). Doc. IRHO nº 1435, 18 pp.

Mariau, D., Dery, S.K., Sangare, A., N´cho Yavo, P. and Philippe, R. 1996. Le jaunissement mortel du cocotier au Ghana et tolérance du matériel végétal. Plantations, recherche, développement **3 (2):** 105-112.

Mariau, D., Lande Van de, H.L., Renard, J-L., Dollet, M., Rocha de Souza, L., Rios, R., Orellana, F. and Corrado F. 1992. Les maladies de type pourriture du coeur sur le palmier à huile en Amérique latine. Symptomatologie, épidémiologie, incidence. Oléagineux **47 (11):** 605-618.

Meiffren, M. 1961. Contribution aux recherches sur la trachéomycose du caféier en Côte d´Ivoire. Café, cacao, thé **5 (1):** 28-37.

Michels, T. 1990. Contribution à l´étude des pourridiés à Armillaire dans les plantations d´hévéa au Gabon. Rapport de stage. Institut supérieur technique d´outré-mer, 61 pp.

Mourichon, X. 1987. L´anthracnose à *Colletotrichum gloeosporioides* des manguiers, avocatiers et papayers. Quelques données sur la biologie et le contrôle de la maladie. Document technique nº 52, RA 87, CIRAD-IRFA, 6 pp.

Mourichon, X. 1994. Serious citrus dieback in Colombia caused by *Ceratocystis fimbriata*. Fruits **49 (5-6):** 415-416.

Muller, R.A. 1954. *Hemileia coffeicola* Maublanc et Roger, sur *Coffea robusta*. Rapport non publié du centre de recherche agronomique du Cameroun.

Muller, R.A. 1959. Le dépérissement des pisquins, arbres d'ombrage du caféier arabica au Cameroun (régions Bamoun et Bamiléké). L'Agronomie Tropicale **14 (1):** 13-17.

Muller, R.A. 1980. Contribution à la connaissance de la phytomycénose constituée par *Coffea arabica* L., *Colletotrichum coffeanum* Noach (Sensu Hindorf), *Hemileia vastatrix* B.et Br., *Hemileia coffeicola* Maublanc et Roger. IFCC, Bull., **15:** 174.

Murray, D.F. 1945. First progress report of the Nigerian cocoa survey, April 1944-March 1945.

Naert, B., Gal, R., Lubis, A.U. and Suwandi, Olivin J. 1990. Première évaluation des possibilités d'exploitation de la télédétection spatiale pour l'étude du fonctionnement d'une plantation de palmiers à huile à Nord Sumatra. Oléagineux **45 (5):** 201-214.

Nieto Paez, L.E. 1993, Pruebas de patogenicidad con *Fusarium solani* y *Thielaviopsis* sp. en palma de aceite *Elaeis guineensis* Jacq. Palmas **14 (2):** 47-52.

Orellana, R.G. 1956. La moniliose des cabosses et autres maladies du cacaoyer dans la partie orientale du Panamá. Bull. Phytosanit. FAO **4 (11):** 170-171.

Ortiz-Garcia, F.C., Hérail, C. and Blaha, G. 1994. Utilisation des isozymes en tant que marqueurs pour l'identification spécifique des *Phytophthora* responsables de la pourriture des cabosses dans les pays producteurs de cacao. *In:* XIth International Cocoa Research Conference, Yamoussoukro, Côte d'Ivoire, pp. 135-143.

Partiot, M., Djeikpor, E.K., Amefia, Y.K. and Bakar, K.A. 1978. Le *swollen shoot* du cacaoyer au Togo. Inventaire préliminaire et première estimation des pertes causées par la maladie. Café, cacao, thé, **22 (3):** 217-228.

Peries, O.S. 1996. The economics of disease control in hevea. Bull. Rubb. Res. Inst. Ceylon, pp. 1-27.

Petithuguenin, P. and Roche, G. 1995 Equateur: la filière cacao, bilan et perspectives. Plantations, recherche, développement **2 (4):** 15-26.

Petit-Renaud, D. 1991. Contribution à l'étude du pourridié de l'hévéa (*Hevea brasiliensis*), causé par *Armillaria heimii* au Gabon. Rapport de stage, Ecole nationale supérieure agronomique de Toulouse, France, 38 pp.

Pruvost, O. 1989. La maladie des taches noires de la mangue (*Xanthomonas campestris* pv. *mangiferaeindicae*). Etude bactériologique, biologique, épidémiologique et mise au point des bases d'un système de lutte intégrée dans les conditions de l'île de la Réunion. Thèse de doctorat. Université Paris Sud, France, 280 pp.

Pruvost, O. and Manicom, B.O. 1993. *Xanthomonas campestris* pv. *mangiferaeindicae*, cause of bacterial black spot of mangoes: 91-95. *In: Xanthomonas*, J.G. Swings, E.L. Civerolo. (eds). Chapman and Hall, London, UK, 399 pp.

Pruvost, O., Couteau, A., Luisetti, J. and Vernière, C. 1995. Biologie et épidémiologie de l'agent de la maladie des taches noires de la mangue. Fruits **50:** 183-189.

Quillec, G. and Renard, J-L. 1975. L'helminthosporiose du cocotier. Etudes préliminaires., Oléagineux **30 (5):** 209-213.

Quillec, G. and Renard, J-L., 1977. Protection contre la cercosporiose du palmier à huile. Conseil de l'IRHO n° 175. Oléagineux **32 (8-9):** 363-365.

Quillec, G. and Renard, J-L. 1984. La pourriture à *Phytophthora* du cocotier. Conseil de l'IRHO 242. Oléagineux **39 (3):** 143-147.

Quillec, G., Renard, J-L and Guesquiere, H. 1984. Le *Phytophthora heveae* du cocotier: son rôle dans la pourriture du coeur et dans la chute des noix. Oléagineux **34 (10):** 477-485.

Quillec, G., Morin, J.P., Renard, J-L. and Mariau, D. 1978. Les maladies du cocotier dans le jeune âge; causes, méthodes de lutte. Oléagineux **33:** 495-500.

Randles, J.W., Boccardo, G. and Imperial, J.S. 1980. Detection of the *cadang-cadang* associated RNA in African oil palm and buripalm. Phytopathology **70 (3):** 185-189.

Renard, J-L. 1985. Etude de maladies du palmier à huile au Brésil. Doc. IRHO n° 1955, 37 pp.

Renard, J-L., 1989. *Hartrot* du cocotier. Caractérisation et moyens de lutte. Conseil de l'IRHO 302. Oléagineux **44 (10):** 475-484.

Renard, J-L. 1992. Introduction to coconut *Phytophthora* diseases in coconut *Phytophthora*. Workshop Proc., October 1992. CIRAD, Manado Indonesian, 180 pp.

Renard, J-L. and Quillec, G. 1977. Lutte contre la cercosporiose du palmier à huile. I. En pépinière. Oléagineux **32 (2):** 43-50.

Renard, J-L. and Quillec, G. 1979. Les maladies et anomalies du palmier à huile en pépinière. Conseil de l'LRHO 195. Oléagineux **34 (7):** 331-337.

Renard, J-L. and Quillec, G. 1984a. Les maladies graves du palmier à huile en Afrique et en Amérique du Sud. Oléagineux **39 (2):** 57-67.

Renard, J-L. and Quillec, G. 1984b. La pourriture sèche du coeur du palmier à huile. Conseil de l'LRHO 248. Oléagineux **39 (10):** 471-476.

Renard, J-L. and Ravise, A. 1986. La fusariose du palmier à huile. Phytoma **374:** 44-46.

Renard, J-L. and Franqueville, H. de. 1989. La fusariose du palmier à huile. Conseil de l'LRHO n° 300. Oléagineux **44 (7):** 341-349.

Renard, J-L., Quillec, G. and Arnaud, F. 1975. Une nouvelle maladie du cocotier en pépinière. Symptômes, moyens de lutte. Oléagineux **30 (3):** 109-112.

Renard, J-L., Brahmana, D. and Rognon, F. 1984. Comportement du cocotier hybride Nain laune × Grand Ouest African vis-à-vis du *Stem bleeding* en Indonésie. Mise en évidence du rôle du chlore dans l'expression des symptômes. Oléagineux **39 (6):** 311-319.

Renard, J-L., Franqueville, H. de, Diabate, S. and Ouvrier, M. 1993. Study of the impact of vascular wilt on FFB production in oil palm. Oléagineux **48 (12):** 495-504.

Rivano, F. 1992. La maladie sud-américaine des feuilles de l'hévéa. Etude, en conditions naturelles et contrôlées, des composantes de la résistance partielle des l'hévéa à *Microcyclus ulei* (P. Henn.) V. Arx. Thèse de doctorat, université Paris XI, Orsay, France, 260 pp.

Saccas, A.M. 1951. La trachéomycose (carbuncullariose) de *Coffea excelsa, neo-arnoldiana* et robusta en Oubangui-Chari. L'Agronomie Tropicale 6 (9-10).

Saccas, A.M. 1956. Recherches expérimentales sur la trachéomycose des caféiers en Oubangui-Chari. L'Agronomie Tropicale **11 (1).**

Salas, L.A. and Echandi, E. 1961. Parasitic nematodes in coffee plantations of Costa Rica. Coffee **3:** 6-9.

Samassekou, S., Mallet, B. and Nicole, M. 1981. Présence de pourridié sur *Cedrela odoratae* Côte d'Ivoire, 14 pp.

Schieber, E., Sosa, O., 1960. Nematodes on coffee in Guatemala. Plant Disease Reporter **44:** 722-723.

Sénéchal, Y. 1986. L'anthracnose de l'hévéa, étude épidémiologique et recherche de moyens de lutte. Thèse de docteur ingénieur, université Paris XI, France.

Snowden, J.D. 1920. Annual Report. Department of Agriculture, Uganda.

Stahel, G. 1931. Zurkenntnis der Siebz Ihrenkrankheit (Phloemnekrose des kaffee-banmes in Surinam). I mikroskopische untersuchungen und Infektions versuche. J. Phytopathology **4 (1):** 65-82.

Subileau, C. 1993. Systematique et biologie du complexe parasitaire constitué de *Phyllachora torrendiella* (Bat.) nov. comb. et de *Botryosphaeria cocogena* nov. sp., agents fongiques du dessèchement foliaire de cocotier au Brésil. Thèse de doctorat, université Paris VI, France, 121 pp.

Thévenin, J-M., Motulo, H.F.J., Kharie, S., Mangindaan, H.F. and Warokka, J.S. 1994. Epidemiological studies on *Phytophthora* diseases of coconut in North Sulawesi province, Indonesia. In: Coconut *Phytophthora* Workshop, Manado, Indonesia, 23-30 octobre 1992. CIRAD-Cp, Montpellier, France, pp. 21-25.

Thorold, C.A. 1975. Diseases of Cocoa. Charenden Press, Oxford, UK. 423 pp.

Thévenin, J-M. and Trocmé, O. 1996. La moniliose du cacaoyer. Plantations, recherche, développement **3 (6):** 397-406.

Thévenin, J-M., Motulo, H.F.J., Kharie, S. and Renard, J-L. 1995. Lutte chimique contre la pourriture du coeur à *Phytophthora* du cocotier en Indonésie. Plantations, recherche, développement **2 (6):** 41-50.

Tirtawidjaja, S. 1980. Citrus virus research in Indonesia. Proc. VIIIth International Conference Org. Citrus. Virol., pp. 129-132.

Tran Van Canh. 1996. Lutte contre le pourridié blanc (*Fomes*) de l'hévéa. Plantations, recherche, développement **3 (4):** 245-250.

Tresh, T.M. 1958. The control of cocoa *swollen shoot* disease in West Africa. A review of the present situation. Tech. Bull. Wacri **4:** 1-36.

Trocmé, O. 1972. Contribution à l'étude d'une maladie du cacaoyer en Ouganda: le dessèchement écofongique des branches. (A contribution to the study of a cocoa tree disease in Uganda, the eco-fungal wilt of the branches). Café, cacao, thé **16 (3):** 219-235.

Umar Akbar, Kusnadi, M. and Ollagnier, M. 1971. Influence de la nature du matériel végétal et de la nutrition minérale sur la pourriture sèche du tronc du palmier à huile due à *Ganoderma*. Oléagineux **26 (8-9):** 527-534.

Vernière, C. 1992. Le chancre bactérien des agrumes (*Xanthomonas campestris* pv. citri): étude épidémiologique et écologique dans le cadre de l'île de la Réunion. Thèse de doctorat, université Paris Sud. Paris, France, 140 pp.

Villain, L., Sarah, J-L., Decazy, B., Molina, A. and Sierra, S. 1996. Evaluation of grafting on *Coffea canephora* var. *robusta* and chemical treatment for control of *Pratylenchus* sp. in *C. arabica* cropping systems. Proc. IIIrd International Nematology Congress. Gosier, Guadeloupe, 7-12 July 1996.

Vogel, R. and Bové, J-M. 1986. Influence de l'exocortis sur la croissance et la production du clémentier greffé sur 39 lignées, de *Poncirus trifoliata*. Fruits **41:** 639-647.

Waller, J-M., Bridge, P.M., Black, R. and Hakiza, G. 1993. Characterization of the *coffee berry disease* pathogens, *Colletotrichum kahawae* sp. nov. Mycological Research **97 (8):** 989-994.

Warwick, D.R.N., Bezerra, A.P.T. and Renard, J-L. 1991. Reaction of coconut hybirds to leaf blight (*Botryodiplodia theobromae* Pat.), field observations. Oléagineux **46 (3):** 100-108.

Zelazny, B., Randles, J.W., Boccardo, G. and Imperial, J.S. 1982. The viroid nature of the *cadang-cadang* disease of coconut palm. Scientia Filipines **2:** 45-63.