

Bud rot and other major diseases of coconut, a potential threat to oil palm

Dollet Michel¹, Hubert de Franqueville² Michel Ducamp¹

¹CIRAD, TA A-98/F, Campus International de Baillarguet, 34398 Montpellier Cedex 5

²PalmElit - Parc Agropolis Bat.14 - 2214 Bd de la Lironde, 34980 Montferrier sur Lez

INTRODUCTION

Since the last quarter of the 20th century, it has had to be accepted that any pathogen can move from one continent to another, in a very short time, and affect any place on the planet. The best-known examples are animal and/or human pathogens. Reference will briefly be made to them as they are very concrete examples of the current epidemic context. However, this also applies for the spread of plant diseases that we have been witness to in the last thirty years. Not only do pathogens travel over long distances, they also “jump hosts”, be it in the Animal Kingdom or the Plant Kingdom. Climate changes may be conducive to such events. Using these data, we shall attempt to examine the possible risks of seeing pathogens of the coconut palm, *Cocos nucifera*, or even of other plants, attacking the oil palm, *Elaeis guineensis*.

DISPERSAL/PROPAGATION OF ANIMAL AND HUMAN PATHOGENS

Sars

The first example involves SARS (*Severe Acute Respiratory Syndrome*) which was identified in humans in China at the end of 2002. At the beginning of 2003, a hotel in Hong Kong very close to the original focus was the starting point for an epidemic that affected Vietnam, Singapore and even Toronto in Canada within a few weeks. Two key factors were conducive to that epidemic: (i) the hotel, a confined meeting place for people of different geographical origins, (ii) aircraft, which can convey any pathogen, or its vector, from one continent to another in under 24 hours. It has been shown that various animals, such as the Civet cat found on food markets in China can carry the virus. Very similar viruses have also been found in bats displaying no pathology. There has therefore been a major host jump from these animals to humans.

Avian Influenza

The avian influenza virus, H5N1, a poultry virus originating from Asia, has been a spectre since 2004. In this case too, it has been demonstrated that all kinds of exchanges, be it trade or tourism, and means of travel – road, rail, air – were more to blame for spreading the virus than the movements of migratory wild birds. The H5N1 virus is a chicken virus. Ducks can be healthy carriers. But it also passes to swine and more rarely to humans. Host-jumping is therefore perfectly possible. Moreover, a Dutch research team recently showed that the virus can acquire the capacity for airborne transmission between mammals.

West Nile Virus (WNV)

As its name suggests, WNV was first identified in Africa in the West Nile province of Uganda in 1937. This virus mostly infects birds, but can also be fatal for horses and it now exists on every continent. It has become a serious health problem in the USA where over 1,200 human cases were recorded in Texas in the first half of 2012.

Aspergillosis

Aspergillus niger causes infections in onion seedlings and common postharvest diseases. This fungus also causes a crown rot disease commonly resulting in seedling blight in peanuts. However, *A. niger* and other *Aspergillus* species can cause human diseases such as otomycosis or aspergillosis, a severe lung disease.

DISPERSAL/PROPAGATION OF PLANT PATHOGENS OR PESTS

The xylem bacterium *Xylella fastidiosa*

In the 1880s, Pierce Disease wiped out commercial viticulture in the Los Angeles Basin of California. This disease was caused by the bacterium *Xylella fastidiosa*, restricted to the xylem vessels. Over the last 30 years, diseases caused by *X. fastidiosa* have emerged throughout the American continent. Introduction of exotic strains of this bacterium resulted in new host-pathogen combinations, and new diseases appeared, such as almond leaf scorch, citrus variegated chlorosis, coffee leaf scorch, elm leaf scorch, oleander leaf scorch, etc., all causing environmental and economic damage.

HuangLongBing (HLB) or Citrus Greening

HLB, caused by the phloem-restricted bacterium *Candidatus Liberibacter asiaticus* (Las) originated in China in the 1940s. In the 1970s, HLB spread to Saudi Arabia. In 2004, the bacterium started causing damage on the American continent in Brazil (2004) and Florida (2005) then on several Caribbean islands and in Central America. In Florida, the Las virus was apparently imported by accident via a scion brought back by an inhabitant of southern Florida returning from a holiday in China. In 2012 in California a lemon/pomelo tree in a neighbourhood of Los Angeles County was found infected by HLB. A graft of pomelo — a symbol of prosperity in several Asian countries — was the likely source of this case. The budwood, was probably passed freely among amateurs who loved to garden and experiment with hybridization.

Paysandisia archon

Paysandisia archon is a butterfly (Lepidoptera, Castniidae) native to Argentina. It was accidentally introduced into Europe by palm imports from South America, taken from the wild. Its known hosts in Argentina are *Butia yatay*, *Syagrus romanzoffiana* and *Trithrinax campestris*. The insect is now found in Portugal, Spain, France, Italy, Greece, Turkey and even the United Kingdom, destroying thousands of palms such as *Chamaerops humilis*, *Phoenix canariensis*, *P. dactylifera*, *P. reclinata*, *Livistona chinensis*, *Trachycarpus fortunei*, etc.

BUD ROT OF COCONUT/*PHYTOPHTHORA*

The genus *Phytophthora* as severe plant pathogen

About 100 species of the genus *Phytophthora* de Barry 1875 (Kingdom Stramenopila, phylum Oomycota, Class Oomycetes, Order Peronosporales, Family Peronosporaceae) have been described. Several species are the causal agent of plant diseases. The most famous case of *Phytophthora* damage is potato blight, caused by *Phytophthora infestans*, responsible for the great Irish famine in the 1840s resulting in 1 million people dying and a million more emigrating from Ireland. At least 55 species are rife in the tropics.

The species *P. ramorum* alone can infect over 150 species of plants in 60 different genera, and the same plant can be infected by several species of *Phytophthora* (*P. ramorum*, *P. psychrophilia*, *P. europaea*, *P. uliginosa*, *P. nemorosa*, *P. quercina* in oaks). *Phytophthora sojae* is responsible for soya bean root and stem rot, leading to longstanding problems, and other species cause severe damage to cactus, eucalyptus, vegetables, fruits, pistachio, cocoa, Cucurbitaceae, strawberries etc.

In recent years, *Phytophthora* became a new major threat to different crops such as citrus in Florida, and in Italy olive trees and vegetables grown in greenhouses are steadily suffering from *Phytophthora* attacks.

Palm diseases and *Phytophthora* species

Bud rot of coconut. (Plate 1)

Bud rot of coconut is a lethal disease. The first symptom is wilting of the spear leaf and often leaf No.1. Tilting of the spear leaves while all other leaves stay green and healthy is the most typical symptom. The dissection of a palm at this stage reveals a foul-smelling internal rot above the meristematic zone. The rot is surrounded by a yellow/brown border. Central rot of the stem may be found in palms with the collapse of the spear leaves. Cross and transversal sections of the rachis of the leaves sometimes reveal light brown speckles varying in width. Generally the roots are not affected and look like healthy roots. In Côte d'Ivoire, affected palms died slowly in 6 to 8 months and sometimes much later (Quillec *et al.*, 1984).

A coconut disease with bud rot symptoms was first reported on Grand Cayman Island in 1834 (Tucker 1926, in Menon and Pandalai 1958). In the 1920s "budrot diseases" were identified in Jamaica, Puerto Rico, Africa, Peninsular Malaysia and the Philippines (Menon and Pandalai, 1958). Later, it was also reported in India, Sri Lanka, Central America, the West Indies, Fiji, Vanuatu etc. *Phytophthora* bud rot of coconut was only slight up to the sixties, but serious outbreaks were recorded in the Philippines, in Laguna province, between 1961 and 1967 when 25,000 coconut palms became infected (Celino, 1970). In the late 1970s/early 1980s, outbreaks of bud rot occurred for the first time in Côte d'Ivoire and in Indonesia (North Sumatra, North and Central Sulawesi) (Quillec *et al.* 1984; de Franqueville and Renard, 1989; Thevenin *et al.* 1994).

Phytophthora palmivora

Several palm species are susceptible to *P. palmivora* (table 1). It is impossible to really know in 2012 which species of *Phytophthora* were involved in the 1920s even if *P. palmivora* is generally cited. At that time, the losses due to *Phytophthora* were generally low: usually only sporadic, but sometimes a few per cent per year, mainly in the rainy season – 2.5% loss per month in Jamaica after 1 or more

months of heavy rainfall on flat lands (Nowel 1923; 1925). *P. palmivora* has been isolated from diseased coconuts in Jamaica, the Dominican Republic, Indonesia, the Philippines, India, Ghana, French Polynesia and Pacific Islands.

On oil palm, rot diseases are frequently suspected of being caused by fungi. As early as 1928, Reinking—quoted by Richardson (1955)—set out to identify microorganisms associated with bud rot in Panama. He discovered the existence of bacteria, *Fusarium moniliforme* and a possible *Phytophthora*. At that time, Reinking considered that the latter was probably the causal agent of the disease, by analogy with coconut bud rot, which had been widely described in the Caribbean. Seven years later, in central Africa, Ghesquière (1935) reported the association between *Phytophthora palmivora*, *Bacillus coli* and bud rot of oil palm in the Belgian Congo. *Thielaviopsis paradoxa* has been mentioned as capable of invading oil palms affected by *Phytophthora*. It has to be remembered that isolation of *Phytophthora* from palms affected by bud rot is not very easy because secondary invasions very quickly appear. The fungus *Thielaviopsis paradoxa* is very common in the very fast secondary invaders, but also *Fusarium* sp., *Penicillium* sp., *Cylindrocladium* sp. and *Cephalosporium* sp. Bacteria are also very abundant; most of them are Enterobacteriaceae (such as *Erwinia* species and *Enterobacter* sp.). More recently, *Phytophthora palmivora* was found associated with bud rot of oil palm (Pudricion del cogollo or “PC”) in Colombia and its pathogenicity was established (Sanchez *et al.* 1999; Torres *et al.* 2010; Álvarez *et al.* 2011; Martínez *et al.* 2011).

Other *Phytophthora* species in palms

At the end of the 1970s, in Côte d’Ivoire, bud rot of coconut suddenly became a serious problem in some small farmers’ plantations. *P. katsurae* (previously mis-identified as *P. heveae*) was isolated from diseased palms (Quillec *et al.* 1984). In Hawaii also, large numbers of coconuts have been lost because of *P. katsurae*. In Jamaica, the species *P. palmivora* and *P. katsurae* both exist on coconut (Steer and Coates-Beckford 1990). In Indonesia in the 1980s bud rot caused severe damage to several coconut plantations. *P. palmivora*, *P. nicotianae* and *P. aracaе* were isolated. (Bennet *et al.* 1985; 1986; Brahma and Desmier de Chenon 1992; Thevenin 1992).

P. nicotianae causes seedling blight of *Dyopsis lutescens* and stem rot of *Washingtonia filifera* (Elliot *et al.* 2004). *P. parasitica* has been observed in Costa Rica (Thevenin, 1992). *P. aracaе* was reported to cause relatively minor root rot on *Chamaedorea* spp. (Elliot *et al.* 2004). *P. palmivora* may be associated with *P. nicotianae* var. *parasitica* in Sri Lanka (Child 1974).

Nutfall

In Côte d’Ivoire, *P. katsurae* is also associated with a symptom of unripe nut fall without bud rot. Large 8 to 10-month-old green nuts are the most susceptible. The youngest nuts are generally not affected. (Quillec *et al.* 1984). The two symptoms—bud rot and nutfall—are generally independent but they can sometimes be associated.

In Southeast Asia and the Caribbean, early nutfall cases have also been attributed to *P. palmivora* (Bennet *et al.*, 1986).

Variability of *Phytophthora* species.

In Côte d’Ivoire, measurement of the enzyme activities of different isolates of *P. katsurae*, correlated to pathogen aggressiveness on the plant, showed that some isolates were less aggressive than others (Yao *et al.* 1990). Characterization by isoenzymes led to different isolates of *Phytophthora palmivora* being distinguished in coconut (Concebido-Manohar and Blaha 1992; Ortiz Garcia and Blaha 1992;

Blaaha 1994). This means that, as for other pathogens, there is variability in the different *Phytophthora* species, with consequences for their aggressiveness. This variability is probably continuously evolving as oospores of the fungus are formed when two different mating types of the organism, called A1 and A2, are present. This sexual phase is a potential problem, as it produces genetically different offspring, with new characteristics/properties and possibilities of overcoming any host resistance.

P. palmivora, an etiological agent of other plant diseases

P. palmivora, previously mis-identified as *P. parasitica* Dast., is one of the most important causes of papaya diseases. *Phytophthora* on fruit, stem, and root rots of papaya was first reported in the Philippines in 1916. The disease also occurs in Malaysia, Sri Lanka, Hawaii, Australia, Brazil, Spain and Taiwan.

Black pod of cocoa is caused by several species of *Phytophthora*: *P. megakarya*, *P. capsici*, *P. citrophthora*, and *P. palmivora* (Brasier and Griffin 1979, Tsao and Alizadeh 1988; Babacauh 1980). Each year an average 20% of the world's cocoa crop is lost due to *P. palmivora*, the causal agent of black pod rot. In some areas, such as American Samoa, cocoa cannot be grown economically because of black pod.

Durian trees also suffer from *P. palmivora* (Lim and Chan 1986; Navaratnam 1966; Tai 1970).

This may mean that the origin of *P. palmivora* is not the palm but another crop suffering, or not, from this fungus. It is frequent in plant diseases that the name of the pathogen is given accordingly to the first host in which it was discovered. For instance *Peanut clump virus* is in fact a virus of graminaceous plants (Dollet et al. 1976; Doucet et al. 1989).

Potential danger for the oil palm

Given the diversity of the different species, be they pathogenic or not, their intra-specific variability, their highly extended host range and their sexual reproduction, it is clear that *Phytophthora* are potential threats to all crops. The recent discoveries of *P. palmivora* on olive trees in Italy is an example. It should be noted that the *Phytophthora* species isolated from coconut in Côte d'Ivoire is *P. katsurae*. It was first isolated from chestnut in Japan and has also been reported in Taiwan, and Korea. Another important factor for a possible sudden invasion by *Phytophthora* is the long conservation of the fungus. Chlamydospores are produced by the mycelium of some isolates; they germinate under certain conditions and form sporangia. They are able to live in soil and dead plants during times when host plants are absent. Chlamydospores and oospores are therefore important survival structures. It cannot therefore be ruled out that, one day, a pathological syndrome associated with a *Phytophthora* species may emerge somewhere, on any plant, including oil palm

Table 1.

Palm species on which *Phytophthora* has been isolated (From Ridings 1972; Elliot et al. 2004)

Leaf, bud and crown rots	<i>Areca catechu</i> , <i>Borassus flabellifer</i> , <i>Butia capitata</i> , <i>Carpentaria acuminata</i> , <i>Chamaedora elegans</i> , <i>Dypsis lutescens</i> , <i>Howea belmoreana</i> , <i>Howea fosteriana</i> , <i>Livistona otundifolia</i> , <i>Phoenix canariensis</i> ,
---------------------------------	---

Rhopalostylis baueri, *Roystonea regia*, *Sabal causiarum* sp., *Syagrus romanzoffiana*, *Washingtonia filifera*.

Root rots: *Chamaedora hybrids*, *Dypsis decaryi*, *Howea forsteriana*, *Phoenix robelenii*

PORROCA/LITTLE LEAF

Porroca disease, or little leaf disease, has been known since the very beginning of the 1950s in Northwest Colombia (Mesa-Bernal 1951, Ferrand 1959). A very similar disease was observed in Trinidad in 1964 (Maramorosch 1964). At the end of the 1990s, in Panama, an outbreak of Porroca worried the OIRSA (Organismo Internacional Regional de Sanidad Agropecuaria) and a group of scientists from Europe visited the affected regions. (Lovisol O., personal communication). The disease spread rapidly between 1998 and 2006. In some locations the number of coconut palms increased 18-fold in 2 years (Gilbert and Parker 2008). Although the total number of cases only amounted to 0.6% of the coconut palms in that place, percentages of 2.52% have been observed in other places. In the 1990s a disease with identical symptoms appeared in a small plantation in northern French Guiana. In the following 3 years the syndrome could be observed in several places over a linear distance of around 160 km (Dollet M., Julia J-F., Perthuis B. unpublished data).

The first visible characteristic symptom is very severe stunting of new leaves, leading in less than 1 to 2 years to a tuft of very short, chlorotic/yellow leaves. In the most severe cases, after 2/3 years the palm has only half a dozen short, erect leaves with short leaflets. The diameter of the stem becomes smaller and takes the form of a pencil point. The youngest leaves are sometimes so deformed that only a stunted curled rachis remains (Plate 2). Remission cases are not rare, but it takes at least 3 to 4 years before the palm produces new bunches of nuts. However, the palm may have a relapse. Some affected coconut palms die. In one plantation of Dwarf varieties in Guiana it was possible to study the syndrome of little leaf as the palms were less than 4 metres high. Most of the time, prior to leaf shortening, there was rotting of several leaflets of the spear leaf, and sometimes of leaves 1 and 2.

The etiology of Porroca remains a mystery. In the discussion of their article, Gilbert and Parker (2008) wrote that a phytoplasma of the group 16SrXII could be detected. But these data have never been published. Our electronic microscopy investigations of the little leaf syndrome in Guiana did not reveal any phytoplasmas or bacteria in the sieve tubes of the phloem, and PCR analyses did not detect any phytoplasma sequence (Dollet M., Fabre S. and Perthuis B. unpublished data).

Little leaf or Porroca symptoms have been observed in Colombia on other palm species such as *Bactris gasipaes* (peach palm) and *Roystonea regia* (royal palm) (Esquivel 2002). In Guiana we have seen wild *Mauritia flexuosa* (Morange palm) with little leaves. But the most puzzling phenomenon concerns little leaf symptoms in oil palm. In the 1980s-1990s, several cases of oil palms of every age – between 2 and 15 years old- were seen with crowns of small, erect leaves in several countries of Latin America. There was variability in the detailed symptoms but the final result was the same. (Dollet M. unpublished data). In Amazonian Ecuador some little leaf symptoms were called “bloqueo” (block, meaning that growth had stopped). Sometimes some variations were called “pudricion del cogollo” (bud rot). The etiology of “bloqueo” remains unexplained, and it is impossible

to say whether it is the same disease occurring in these different palm species. Some nematodes, sometimes found at the base of the young hidden inflorescences, were suspected of being the cause of the symptom, but their pathogenicity has not been proved. Oil palms can remain a long time with these little leaves (more than three years). Sometimes they slowly recover, but they are usually removed from commercial plantations.

CADANG-CADANG AND TINANGAJA.

Much has been written about coconut cadang- cadang (CCC) and the cadang- cadang viroid (CCCVd). The disease, which is so far confined to the Philippines, destroyed more than 7,900,000 coconut palms between 1951 and 1957 in the Bicol region of Southern Luzon, i.e. 45% of the coconut palms in the region. Some plantations lost 90% of their palms (Bigornia 1977). San Miguel Island lost 99% of all its coconuts in less than 40 years. Doubtless, CCC is probably one of the most destructive diseases of coconut, if not the most destructive. In the 1980s the estimation of losses due to CCC stood at around 500,000 coconut palms per year. Nothing is known about its natural way of spreading.

CCC is associated with a viroid, very small RNA molecules consisting of four forms of 246, 247, 296 and 297 nucleotides (Haseloff et al. 1982). The pathogenicity of the purified viroid has been demonstrated (Randles, Boccardo et al. 1977; Imperial, Rodriguez et al. 1981; Mohamed, Bautista et al. 1985)

A large range of symptoms has been observed with CCC, but the disease cannot be unequivocally diagnosed on the basis of symptoms during a survey. One of the symptoms is the presence of olivaceous or water-soaked spots on the leaflets giving a bronze yellow colour to the lower two-thirds of the crown. Another characteristic is the persistent attachment of the stipules to the base of the rachis of the leaves. There is a gradual reduction in flower and nut emission. The leaves of infected coconut leaves become increasingly small. CCC is a very slow debilitating disease. It has been estimated that the life span for an adult tall variety of coconut could be around 15 to 17 years. At the final stage, the crown is greatly reduced with short leaves, and without nuts or inflorescences. Symptoms that are more or less similar to those of CCC occur in the Philippines on buri palm (*Corypha elata*). In the Philippines too, this viroid has been detected in African oil palm (*Eleais guineensis*) showing broken pinnae, a small number of short leaves and some orange spotting (Randles, Boccardo et al. 1980). However, there is no report of any major losses of oil palms because of these symptoms, and nothing like for CCC. The footstool palm or ananaw (*Livistonia – Saribus-rotundifolia*) may also be a host of the CCC pathogen (Bigornia 1977).

The purified viroid was successfully inoculated in betel palm (*Areca catechu*), date palm (*Phoenix dactylifera*), royal palm (*Roystonea regia*), manila palm (*Veitchia merillii*) and golden cane palm (*Chrysalidocarpus lutescens*) (Imperial, Bautista et al. 1985).

Mechanical transmission of the purified viroid has sometimes led to the appearance of severe symptoms of brooming in 12% of inoculated coconut seedlings (Rodriguez and Randles 1993). These severe symptoms were shown to be associated with three types of CCCVd mutations.

In Guam, another coconut disease called Tinangaja and reported as early as 1917, is also associated with a viroid – CtiVd - 64% homologous with CCCVd (Boccardo et al. 1985; Keese et al.1988). The

disease primarily affects coconut palms over 25 years old. Chlorotic spots, similar to those observed in CCC, although not considered as a diagnostic symptom, occur on the leaflets. One symptom seems to characterize Tinangaja: mummified nuts with no kernel inside (Boccardo *et al.* 1985). The affected palms die soon after nut production ceases.

Variants of CCCVd and/or molecules related to CCCVd (MR-CCCVd) were detected in coconut palms in Thailand, Malaysia, western Indonesia, Sri Lanka and the Solomon islands, where cadang-cadang disease has never been reported (Hanold and Randles 1989; Vadamalai *et al.* 2006; Randles *et al.* 2009; Vadamalai *et al.* 2009). Some of these molecules were detected in palms with orange spotting, but also in palms without that leaf symptom. This symptom seems not to be specific to MR-CCCVd.

Indeed, orange spotting can be the result of different causes. For instance, the mite *Retracus elaeis* Keifer causes orange spotting on oil palms in Latin America. In a production study it was shown that 50% losses were recorded on affected palms (Genty and Reyes 1977; Genty 1981). Potassium is a very important nutrient for oil palm. Deficiencies induce orange spotting followed by “confluent orange spotting” (Turner 1981a; von Uexküll H. R., Fairhurst T.H. 1999). In the event of severe potassium deficiency, heavy spotting gives the older leaves a bronze appearance and culminates in terminal and marginal necrosis of the leaflets and premature desiccation of the fronds. This deficiency can be controlled by applying potassium, but the results are more significant when this is done preventively (after routine leaf analysis) or at the very beginning of the symptoms. Another form of orange spotting has a genetic origin (Ford and Leyritz, 1968; Gascon and Meunier, 1979; Tuner 1981b). This orange spotting is characteristic of the progenies of certain crosses or selfed parents.

Lastly, it should be remembered that some molecules with similar sizes to CCCVd exist in the oil palm. However, the behaviour of these molecules in thermogradient electrophoresis suggests that they resemble double stranded RNAs and are different from viroid structures. (Dollet *et al.* 1994). These double stranded RNA have been found in wild *Elaeis guineensis* populations from Cameroon that had never left that country and were showing no sign of orange spotting, or any disorder. The same type of double stranded RNA has been found in diseased as well as healthy oil palms in Brazil (Beuther *et al.* 1992).

Interestingly, MR-CCCVd has been identified in other monocot crops such as ginger and *Alpinia* sp. (Zingiberaceae) that were apparently completely healthy (Hanold and Randles 1991).

To conclude, CCCVd has been responsible for a devastating disease of coconut palms in the Philippines, and CtiVd was the cause of the Tinangaja syndrome of coconut in Guam. Molecules with around 90% homology with CCCVd have been found in oil palms in the Philippines, the Solomon Islands and Malaysia, but no epidemic disease was associated with the presence of these molecules. However, a very small mutation in viroids can lead to a severe strain, or conversely to a non-pathogenic strain. For instance, this has been very well documented in the case of the Potato Spindle Tuber Viroid (Owens *et al.* 1995; Wassenegger *et al.* 1996).

LIXA GRANDE/ LIXA PEQUENA /QUEIMA DAS FOLHAS (Plate 3)

The fungus *Sphaerodothis acrocomiae* (ex. *Coccostroma palmicola*) is the etiological agent of a leafspot disease, *Lixa grande* (*large rasp*). This fungus is responsible for drying out of the lower leaves of coconuts in some Brazilian plantations (Marto Pinto Viana, 2004).

Lixa pequena (*small rasp*), also a leafspot disease, occurs in Brazil and in French Guiana. It has been known in Brazil since the 1940s (Batista-Chaves 1948). The fungus *Phyllachora torrendiella* (ex. *Catacauma torrediella*) is responsible for this disease. In the event of severe attacks, the leaflets completely dry out, leading to the complete wilt of the lower and middle leaves. The palms lose around one third of their assimilating leaf area and the yield drops. In Sergipe state, this fungus has developed over *S. acrocomiae* (Subileau, 1993).

The main problem with *C. torrendiella* is that it opens the door to another fungus, generally growing on its lesions, *Botryosphaeria cocogena*, inducing the disease locally called *queima das folhas* or leaf blight. This disease was reported for the first time in Sergipe in 1975 and invaded other states in the following years up to Para state in 1987 (Franco 1975; Souza Filho *et al.* 1979; Subileau 1993). In 1990, the disease reached French Guiana (Dollet, unpublished). Leaf blight induces a generalized wilting of the lower and middle leaves, and immature nutfall resulting in 20 to 40% production losses (Warwick *et al.* 1991). Most of the fungicides tested are often ineffectual. In the worst cases, as we found in French Guiana, the coconut palm may die. So far, *B. cocogena* seems to be specific to *Cocos nucifera*. *P. torrendiella* and *B. cocogena* seem to be specific to coconut so far. However, leaf blight is an emerging disease and, because of its damage, it needs to be carefully monitored.

STEM BLEEDING

Stem bleeding is recognized by reddish/dark–brown spots and sap flow down the stem. The colour changes over time, becoming darker and turning black. These reddish/brown streaks correspond to a yellow to reddish colour of the internal tissues of the stem. Stem cavities appear. The rot may affect a large part of the stem, as much in depth as in height. The leaves droop and hang down the stem and the nuts fall. Natural remission can occur, but the stem may break (Plate 4)

Under adverse conditions, such as a long dry period, with ground-based drip irrigation, all the fronds starting with the lower fronds turn brown and dry out from their tips. There then only remains a tuft of a dozen or so young fronds and the meristematic zone rots. As the browning rises, the nuts start to exhibit brown rot around their point of attachment. Under such conditions (perhaps also due to a combined infection with *Lixa grande*?) 50% losses were recorded in some plots of Malayan Green Dwarf plantations in Sergipe over the 2004-2007 period. Palm death occurred between 4 months and a year after the first symptoms (Warwick, personal communication).

The etiology of stem bleeding is quite controversial. Several fungi are often isolated from different species of palms (*Coconut*, *Phoenix*, *Borassus*, *Bactris*, *Raphys* etc.) affected by different types of symptoms including stem bleeding: they have been referred to as “Thielavopsis diseases” (Elliot *et al.* 2004). There are at least two causal agents. One is *Ceratocystis paradoxa* (Dade) C. Moreau (synonym = *Ceratocystis paradoxa* Dade); the anamorph is *Thielaviopsis paradoxa* (de Seyn.) Höhn. (synonym = *Chalara paradoxa* (de Seyn.) Sacc.). Another species is *Ceratocystis radicola* (Bliss) C.

Moreau (synonym = *Ceratostomella radicola* Bliss.); the anamorph is *Thielaviopsis punctulata* (Hennebert).

Seedlings of *Elaeis guineensis* are on the list of susceptible species for “*Thielaviopsis* diseases” (Elliot et al. 2004). The disease is called black rot.

HARTROT/MARCHITEZ SORPRESIVA (Plate 5)

Hartrot (Dutch name for bud-rot) of coconut has been known in Surinam – Latin America- since the end of the 19th century. Bud rot is only the final stage of the disease. The first symptom is a yellowing of the lower leaves and the total fall of both mature and immature nuts. The yellowing progresses towards the youngest leaves, the inflorescences become necrotized and the palm dies 6 to 8 months later. The hartrot syndrome is identical to the Lethal Yellowing Type Syndromes (LYTS) associated with phytoplasmas. A small plantation affected by hartrot can be wiped out in a few years. (Parthasaraty *et al.* 1978; Dollet 1984; Louise *et al.* 1986).

Marchitez sorpresiva of the oil palm also appeared for the first time in Surinam, in 1919. However, it was only in the 1960s-1970s that this wilt became a factor limiting oil palm development in Latin America - Colombia, Ecuador and Peru. (Lopez *et al.* 1975; Dollet 1984). An affected palm may die in less than two months after the first symptoms. The syndrome is a fast-rising drying out of the leaves.

Both diseases, hartrot and marchitez, are associated with the presence of phloem-restricted trypanosomatids (*Phytomonas* sp.) transmitted by pentatomid bugs (Heteroptera) of the genus *Lincus* (Perthuis *et al.* 1985; Louise *et al.* 1986).

Trypanosomatids also attack other palm species such as *Bactris gasipaes*, *Chrysalidocarpus lutescens*, *Bentickia nicobarica*, *Roystonea regia*, *Maximiliana maripa*, *Caryota mitis*.

In Surinam too, a disease of *Coffea liberica*, called coffee phloem necrosis, raged during the first half of the 20th century (Stahel, 1954). Trypanosomatids morphologically identical to those of coconut or oil palm were specifically associated with that pathological syndrome.

In the 1990s, very severe losses were observed on the island of Grenada (southeastern Caribbean Sea) in the fields of *Alpinia purpurata* (red ginger), a Zingiberaceae cultivated for its brightly coloured red bracts. Several plantations of half to one hectare were destroyed in two years by a yellow wilt associated with phloem-restricted trypanosomatids. These parasites were *in vitro* cultured and compared using several molecular markers to those of hartrot and marchitez. They were identical to the *Phytomonas* associated with marchitez sorpresiva of oil palms from Venezuela, Colombia or Ecuador (Dollet *et al.* 2001). It therefore seems that the host range of pathogenic trypanosomatids can be quite large.

RED RING

Red ring is a common problem in coconut throughout Latin America from Brazil up to Mexico and through the southern Caribbean (Trinidad and Tobago (Griffith, 1987). The nematode

Bursaphelenchus cocophilus (Cobb) Baujard 1989 (ex. *Rhadinaphelenchus cocophilus* (Cobb 1919) J.B. Goodey 1960 is responsible for the disease. The nematode is propagated by the insect *Rhynchophorus palmarum* (Coleoptera, Curculionidae), the “palm weevil”, but other beetles such as *Dynamis borassi* and *Metamasius hemipterus* are also reported as vectors of the red ring nematode (Giblin-Davis *et al.* 2003)

This nematode infects over 17 species, but disease severity and symptoms are variable. In addition to coconut, the most economically important species are the African oil palm, *Elaeis guineensis* and the date palm, *Phoenix dactylifera*. The royal palm (*Roystonea oleracea*), gru-gru palm (*Acrocomia aculeata*), the moriche (*Mauritia flexuosa*), and cucurite palm (*Maximiliana maripa*) can be infected artificially.

The symptoms vary with palm species and age, cultivar, and environmental conditions. In coconut, red ring symptoms include premature nut fall, withering of inflorescences, and yellowing/browning of the leaves. This yellowing can be confused with LYTS symptoms. Several of the yellow/brown leaves often break close to the petiole and remain hanging down the stem. However, the only typical symptom is a 2-5 cm wide “red ring” inside the stem, which can be seen in cross-section (Plate 6). In fact the colour of the ring can vary according to the stage of the disease, the variety or species of palm, etc. It may be dark-cream, brown, rust or brick red. Irregular-shaped and thin brown/purplish-blue rings are common in oil palm. Coconut palms between 3 and 10 years of age die within 2 months (Griffith 1987). There is no red ring disease without a “red ring” (or brown or purple ring) inside the stem as it has sometimes been claimed in the past. In oil palm, symptoms involve progressive yellowing and death of the lower leaves, which break and hang down. An internal ring that is not continuous throughout the stem is common. In old coconut and oil palms too, a “chronic form” exists under the symptom of “little leaves”. The two palm species begin producing increasingly short leaves, giving the crown an appearance of Porroca symptoms, or “bloqueo” (see above), but in the case of Porroca or bloqueo there are no *B. cocophilus* nematodes and no red ring. (Chinchilla, 1992). New inflorescences are aborted and the palms become unproductive.

CONCLUSION

Rare are the pathogens restricted to only one plant species. Several, such as *Xylella fastidiosa*, have a wide range of hosts. According to Hopkins and Purcell (2002), the known list of *Xylella* hosts is probably limited more by a researcher’s limitations in searching for natural or experimental hosts than by the bacterium’s actual host range. Viruses sometimes have an even wider host range. Around 1,200 species in over 100 families of monocots and dicots, including many vegetables, ornamentals and woody and semi-woody plants are hosts of the cucumber mosaic virus!

It is not rare to see one pathogen affecting both coconut and oil palm, like the red ring nematode and the trypanosomatids associated with hartrot and marchitez, the cadang cadang viroid, the fungus *Ganoderma* associated with basal stem rot, and *Pestalotiopsis palmarum* (Elliott *et al.* 2004) .

There are cases for which coconut is a host but not oil palm and vice versa. The best example is the coconut Lethal Yellowing Type Syndrome occurring in the Caribbean. In this region five subgroups (A, B, D, E, and F) of the phytoplasma group 16SrDNA-IV can attack more than 30 different species of

palms but *E. guinenensis* is not on the list of susceptible species. One reason could be there are few oil palms in this region, even though there are some quite large oil palm plantations in Honduras. However, in West Africa and East Africa where LYTS of coconut associated with phytoplasma from other groups occur, oil palm plantations are abundant and often very close to coconut plantations, side by side, as in Ghana. However, the oil palms have never been affected by any LYTS (Dollet *et al.*, 2009; McCoy *et al.* 1983).

However, there is no definitive status about host ranges. For instance, *Washingtonia robusta*, which is not on the list of susceptible species of the “historic” LYTS that scourged Florida in the 60s-70s – probably the subgroup 16SrDNA-IV-A, the one present today in almost all the Caribbean area – is now affected by a phytoplasma of the same group but another subgroup (F). (Harrison *et al.* 2008). The same thing happened to *Sabal palmetto* and *Phoenix roebelinii* in Florida, which have started being attacked by sub-group D of group IV in the last 4 years. (Harrison *et al.* 2009; Jeyaprakash *et al.* 2011).

Fungi such as the genus *Phytophthora* also have a wide host range and host switches happen. So it seems that for oil palm, as for any other perennial crop, there is no “risk zero”. It has to be managed as far as possible through a Pest Risk Analysis, in a similar way to what has been done by the FAO for the coconut palm, in order to avoid completely banning germplasm movements.

Acknowledgments.

The authors thank Michel Ducamp (CIRAD, UMR BGPI) for helpful discussion and recent information on *Phytophthora* species.

References

Álvarez E., Marroquín-Guzman M., Mejía J.F., Pardo J.M. 2011. Early detection of *Phytophthora palmivora* in oil palm, using real-time polymerase chain reaction (QPCR) and molecular beacon probes. *Phytopathology*. 101: S253.

Babacauh K.D. 1980. Structure et dynamique des populations de *Phytophthora sp.* parasite du cacaoyer (*Theobroma cacao* L). Ph.D. thesis, Université of Paris-Sud, Orsay, France.

Bennett C.P.A., Roboth O., Sitepu G., Lolong A. 1986. Pathogenicity of *Phytophthora palmivora* (Butler) Butler causing premature nutfall disease of coconut (*Cocos nucifera* L.). *Indonesian Journal of Crop Science*.2: 59-70

Beuther E., Wiese, U., Lukacs, N., Van Slobbe W. G., Riesner D. 1992. Fatal yellowing of oil palms: search for viroids and double-stranded RNA. *Journal of Phytopathology*.136: 297-311

Bigornia A.E. 1977. Evaluation and trends of research on the coconut cadang-cadang disease. *The Philippine Journal of coconut studies*. 11: 5-12.

Blahe G, Hall G., Warokka J.S, Concibido E., Ortiz-Garcia C. 1994. *Phytophthora* isolates from coconut plantations in Indonesia and Ivory Coast: characterization and identification by morphology and isozyme analysis. *Mycological Research* 98: 1379–1389

Boccardo G., Beaver R. G., Randles J. W., Imperial J. S. 1985. Tinangaja and bristle top, coconut diseases of uncertain etiology in Guam, and their relationship to Cadang-cadang disease of coconut in the Philippines. *Phytopathology*. 71: 1104-1107

Brahama J., Lubis A.U., Desmier de Chenon R. 1992. Evolution of coconut bud rot disease and control strategies (observations in Bangun Purba, North Sumatra.). *Coconut Phytophthora. Workshop Proceedings 26-30 October, Manado, Indonesia. Colloques Cirad.85-92*

Brasier, C.M., and Griffin, M.J. 1979. Taxonomy of *Phytophthora palmivora* on cocoa. *Trans. Br. Mycol. Soc.* 72: 111–143.

Celino M.S. *et al.* 1970. Bud Rot Control. In: *Coconut production*. Ed. by R.G. Emata (Philippines) p. 72-75; cited by Ohler J.G. 1984. *Coconut, tree of life* FAO Plant production and protection paper 57: 446

Child R. 1974. *Coconuts*. Longman London, 2nd ed. *Trop. Agric. Ser. Group.* 335p.

Chinchilla C.M. 1992. El síndrome del anillo rojo-hoja pequeña en palma aceitera y cocotero. *Revista palmas.*13:33-56.

Concebido-Manohar E.C. and Blahe G. 1992. Identification of *Phytophthora* isolates pathogenic to coconut and closely related species by isoenzymes analysis through starch gel electrophoresis. *Coconut Phytophthora. Workshop Proceedings 26-30 October, Manado, Indonesia. Colloques Cirad.85-92*

Dollet M. 1984. Plant diseases caused by flagellate protozoa (*Phytomonas*). *Annual Review of Phytopathology*, 22 : 115-132.

Dollet M., Quaiçoe R., Pilet F. 2009. Review of coconut "Lethal Yellowing" type diseases. Diversity, variability and diagnosis. *Oléagineux Corps gras Lipides.* 16: 97-101

Dollet M., Fauquet C., Thouvenel J.C. 1976. *Sorghum arundinaceum*, a natural host of Peanut Clump Virus in Upper Volta. *Plant Disease Reporter.* 60: 1076-1080.

Dollet M., Mazzolini L., Bernard V. 1994. Research on viroid-like molecules in oil palm. In: "Coconut improvement in the South Pacific? M.A Foale and Lynch P.W. ed. *ACIAR Proceedings.* 53: 62-65.

Dollet M., Sturm N., Campbell D. 2001. The spliced leader RNA gene array in phloem-restricted plant trypanosomatids (*Phytomonas*) partitions into two groupings: epidemiological implications. *Parasitology.* 122: 89-297.

Doucet D., Delfosse P., Legavre A., Devis P.S. 1999. The Indian peanut clump virus is highly infectious on graminaceous plants. *Zeitschrift für Pflanzenkrankheiten und Pflanzenschutz .* 106: 418-424.

Elliot M.L, Broschat T.K., Uchida J.Y., Simone G.W. 2004. *Compendium of ornamental palm diseases and disorders.* The American Phytopathological society. 68 p.

- Esquivel Rios E. A. 2002. Reporte de posibles casos de porroca en pixbae (*Guilma gassipaes*) y palma real (*Roystonea regia*) en Panama. Info. Grupo Porroca N° 1/2002. OIRSA.
- Ferrand M. 1959. La porroca, enfermedad del cocotero observada de Monteria a Cartagena y Baranquilla. FAO report. 9pp. (in Urueta Sandino et al., 1982)
- Forde St. C.M., Leyritz M.J.P. 1968. A study of confluent orange spotting of the oil palm in Nigeria. J. of NIFOR. 4: 72
- Franqueville H. De, Renard J.L. 1989. Effectiveness of Phosethyl –Al in coconut *Phytophthora* control. Oléagineux.44: 351-358.
- Gascon J-P., Meunier J. 1979. Anomalies of genetic origin in the oil palm, *Elaeis*. Oléagineux. 34: 437-447.
- Genty P. 1981. Entomological research on the oil palm in Latin America. Oléagineux. 36: 585-594.
- Genty P., Reyes E. 1977. Un nouvel acarien du palmier à huile: l'eriophide *Retracus alaeis* Keifer. Oléagineux.32 : 255-260.
- Ghesquiere J., 1935. Rapport préliminaire sur l'état sanitaire de quelques palmeraies de la province de Coquilhatville. Publications de l'Institut National pour l'étude agronomique du Congo belge, série scientifique n° 3 : 37-40.
- Giblin-Davis R.M., Lehman P. S., Inserra R.N. 2003. A list of Exotic nematodes Plant pests of the agricultural and environmental significance to the United States. The society of nematologists/APHIS. <http://nematode.unl.edu/pest1.htm>
- Gilbert G.S., Parker I. P. 2008. Porroca: An emerging disease of coconut in Central America. Plant disease. 92: 826-830.
- Griffith R. 1987. Red ring disease of coconut palm. Plant Dis.71:193-196.
- Hanold D., Randles J.W. 1991. Coconut cadang-cadang disease and its viroid. Plant Disease. 75: 330-335.
- Hanold D., Randles J.W. 2003. CCCVd-related molecules in oil palms, coconut palms and other monocotyledons outside the Philippines. In: "Viroids. Hadidi A., Flores R., Randles J.W., Semancik S. Ed. CSIRO publishing; Science Publishers, Inc. p. 336-340.
- Harrison N., Helminck E.E., Elliot M.L. 2008. Lethal yellowing-type diseases of palms associated with phytoplasmas newly identified in Florida, USA. Ann. Appl. Biol.153: 85-94
- Harrison N., Helmick E.E., Elliot M.L. 2009. First report of a phytoplasma-associated lethal decline of Sabal palmetto in Florida. Plant pathology. 58: 792
- Haseloff J., Mohamed N. A., Symons R. H. 1982. Viroid RNAs of cadang-cadang disease of coconuts Nature.299: 316-321
- Hopkins D.L., Purcell A. H. 2002. *Xylella fastidiosa*: cause of Pierce's disease of grapevine and other emergent diseases. Plant Dis.86: 1056-1066.

- Imperial J. S., Bautista R. M., Randles J. W. 1985. Transmission of the coconut cadang-cadang viroid to six species of palm by inoculation with nucleic acid extracts Plant pathology.34:391-401.
- Imperial J. S., Rodriguez J. B., Randles J. W. 1981. Variation in the viroid-like RNA associated with cadang-cadang disease: evidence for an increase in molecular weight with disease progress. Journal of General Virology. 56: 77-85
- Jeyaprakash A., Sutton B.D., Halbert S.E., Schubert T.S. 2011. First report of a 16SrIV-D phytoplasma associated with Texas *Phoenix* palm decline on pigmy date palm (*Phoenix roebelinii*) in Florida. Plant Dis. 95: 1475.
- Keese P., Osorio-Keese M. E., Symons R. H. 1988. Coconut tinangaja viroid : sequence homology with coconut cadang-cadang viroid and other potato spindle tuber viroid related RNAs. Virology.162: 508-510.
- Lim T.K. and Chan L.G. 1986. Fruit Rot of Durian Caused by *Phytophthora palmivora*. Pertanika 9: 269 - 276
- Louise C., Dollet M., Mariau D. 1986. Recherches sur le Hartrot du cocotier, maladie à *Phytomonas* (Trypanosomatidae) et sur son vecteur *Lincus sp.* (Pentatomidae) en Guyane. Oléagineux, 41 (10) : 437-449.
- Martinez G., Varon F., Rodriguez J., Aya H., Velez D.C., Diana C., Salcedo S., Noreña C., Ariza J., Zuñiga F., Mestizo Y., Torres G.A., Sarria G.A. 2011. New developments in the study of the causal agent of oil palm bud rot disease in Colombia. Phytopathology.101: S254.
- Maramorosch K. 1964. A survey of coconut diseases of unknown etiology. FAO report. Rome 1964.
- Menon K. P. V. and Pandalai K. M. 1958. The coconut palm. A monograph. Indian Central Coconut Committee, Ernakulum, S. India. 384 p.
- Mohamed N. A., Bautista R., Buenaflor G., Imperial J. S. 1975. Purification and infectivity of the coconut cadang-cadang viroid. 75: 79-83
- Navaratnam S.J. (1966): Patch canker of the durian tree. Mal. Agri. J. 45(3): 291 - 294.
- Nowel W. 1923. Diseases of crop plants in the Lesser Antilles. The west India Committee, London
- Nowel W. 1925. Coconut bud-rot in Trinidad. Rep. Proc. Imp. Bot. Conf. London (from Trop.Agriculturist, 64: 257-277
- Ortiz Garcia C. and Blaha G. 1992. Rapid specific identification of coconut and cocoa *Phytophthora* strains through PGI and MDH isoenzymatic banding patterns. Coconut *Phytophthora*. Workshop Proceedings 26-30 October, Manado, Indonesia. Colloques Cirad.93-104;
- Owens R. A., Chen W., Hu Y., Hsu Y.H. 1995. Suppression of potato spindle tuber viroid replication and symptom expression by mutations wich stabilize the pathogenicity domain Virology.208: 554-564
- Parthasarathy, M. V., Van Slobbe, W. G. 1978. Hartrot or fatal wilt of palms I. Coconuts (*Cocos nucifera*). Principes.22:3-14.

- Quillec G., Renard J.L., Ghesquière H. 1984. *Phytophthora hevea* of coconut: role in bud rot and nutfall. *Oléagineux*. 39:477-485.
- Randles J. W., Boccardo G., Imperial J. S. 1980. Detection of the cadang-cadang associated RNA in african oil palm and buri palm. *Phytopathology*. 70:185-189.
- Randles J. W., Boccardo G., Retuerma M. L., Rillo E. P. 1977. Transmission of the RNA species associated with cadang-cadang of coconut palm, and the insensitivity of the disease of antibiotics. *Phytopathology*. 67: 1211-1216.
- Randles J.W., Rodriguez J.M.B., Hanold D., Vadamalai G. 2009. Coconut cadang-cadang viroid infection of oil palm. *The planter*. 85 :93-101.
- Richardson D.L. 1995. The history of oil palm breeding in the United Fruit Company. *ASD Oil Palm Papers*, 11: 1-22.
- Ridings W. H. 1972. *Phytophthora* bud rot of coconut palm. *Plant Pathology Circular No. 115* February. Fla. Dept. of Agr. and Cons. Serv. Division of Plant Industry.
- Rodriguez M. J. B., Randles J. W. 1993. Coconut cadang-cadang viroid (CCCVd) mutants associated with severe disease vary in both the pathogenicity domain and the central conserved region. *Nucleic acids research*. 21: 2771.
- Sanchez N.J., Ayala Samaca L., Alvarez E. Gomez Cuervo P.L. 1999. Patogenicidad, identificacion y caracterizacion molecular de *Phytophthora spp.* en palma de aceite. *Avances. Boletín Divulgativo Cenivances*. 60: 1-4.
- Stahel, G., 1954. Die Siebröhrenkrankheit (Phloëmnekrose, Flagellatose) des Kaffeebaumes. *Neth. J. Agric. Sci.* 4, 260±4.
- Steer J., Coates-Beckford P.L. 1990. Role of *Phytophthora katsurae*, *P. palmivora*, *Thielaviopsis paradoxa* and *Enterobacter sp.*, in budrot of coconut in Jamaica. *Oléagineux*; 45: 539-545.
- Tai L.H. 1970. Studies of *Phytophthora palmivora*, the causal organism of patch canker disease of durian. *Cawangan Perkembangan, Jabatan Pertanian, K. Lumpur*. 7 pp.
- Thévenin J.M. 1992. Coconut *Phytophthora* diseases in Indonesia. Etiological aspects. Coconut *Phytophthora*. In: *Workshop Proceedings 26-30 October, Manado, Indonesia*. Colloques Cirad.27-31
- Thévenin J.M., Motulo H.F.J., Kharie S., Mangindaan H.F., Warokka J.S. 1992. Epidemiological studies of coconut in North Sulawesi province, Indonesia. In: *Workshop Proceedings 26-30 October, Manado, Indonesia*. Colloques Cirad.21-25
- Torres G.A., Arria G.A., Varon F., Coffey M.D., Elliott M.L., Martinez G. 2010. First report of bud rot caused by *Phytophthora palmivora* on African oil palm in Colombia. *Plant Dis*. 94: 1163.
- Tsao P.H., and Alizadeh A. 1988. Recent advances in the taxonomy and nomenclature of the so-called *Phytophthora palmivora* MF4 occurring on cocoa and other tropical crops. X International Cocoa Research Conference, held at Santo Domingo, Dominican Republic, 17–23 May 1987. Cocoa Producers' Alliance, Lagos, Nigeria.

Turner P.D. 1981a. Oil palm diseases and disorders. Oxford University Press. 247-253.

Turner P.D. 1981b. Oil palm diseases and disorders. Oxford University Press. 228- 229

Uexküll von H.R., Fairhurst T.H. 1999. Some Nutritional Disorders in Oil Palm Better Crops International Vol. 13: 16-21

Urueta Sandino E.J., Atehortúa de la Cuesta M., Valencia Restrepo A. 1982. La Porroca u hoja pequena del cocotero. Secretaria de Agricultura y fomento de Antioquia. Publicación técnica N°1. Medellin.

Vadamalai G., Hanold D., Rezaian M.A., Randles J.W 2006. Variants of Coconut cadang-cadang viroid isolated from an African oil palm (*Elaeis guineensis* Jacq.) in Malaysia. Arch Virol. 151: 1447–1456

Vadamalai G., Perera A.A., Hanold D., Rezaian M.A., Randles J. W. 2009. Detection of Coconut cadang-cadang viroid sequences in oil and coconut palm by ribonuclease protection assay. Ann. Appl. Biol. 154: 117-125

Vadamalai G., Hanold D., Rezaian M. A., Randles J.W. 2006. Variants of Coconut cadang-cadang viroid isolated from an African oil palm (*Elaeis guineensis* Jacq.) in Malaysia. Arch. Vriol. 151: 1447-1456.

Wassenegger M., Spieker R. L., Thalmeir S., Gast F.U., Riedel L., Sanger H. L. 1996. A single nucleotide substitution converts potato spindle tuber viroid (PSTVd) from a noninfectious to an infectious RNA for *Nicotiana tabacum*. Virology. 226: 191-197

Yao N.R., N’Goran B., Allou K., Dogbo D.O., Konan K.J.L., Kouassi, P. 1990. Differential behaviour in pathogenicity and enzymatic activities of *Phytophthora katsurae* strains from coconut trees in Cˆte d’Ivoire. Journal of Applied Biosciences 21: 1246 - 1257

FIGURE LEGENDS

Plate1. Bud rot of coconut/*Phytophthora*.

From left to right, from top to bottom:

Coconut plantation affected by Bud rot; Spear rot, the early stage of bud rot; Browning/necrosis of a young inflorescence; Browning/necrosis inside the rachis of a young leaf in longitudinal section; Brown zone inside the trunk at one meter above the ground; Rot at the base of the rachis and on a young inflorescence; Brown zone in transversal section of the rachis of a middle leaf; External rot of the spear leaves above the meristematic zone.

Plate 2. Porroca/Little leaf.

From left to right, from top to bottom:

First stage of little leaf syndrome of coconut in French Guiana – asymmetric necrosis of the leaflets of young leaves; advanced stage showing drying of the intermediate leaves and stunting of young leaves; “Chronic stage” of little leaf; Little leaf symptom in oil palm in Venezuela.

Plate 3. Lixa grande/ Lixa pequena /Queima das folhas.

From left to right, from top to bottom:

Symptoms of Lixa pequena on a leaflet of coconut in Brazil; Wilt or blight – Queima das folhas caused by the fungus *Botryosphaeria cocogena*- in French Guiana; Coconut plantation where all coconuts are affected by Lixa/Queima das folhas in Brazil: all the lower leaves are desiccated; First lower leaf attacked by *Botryosphaeria cocogena*.

Plate 4. Stem bleeding.

From left to right, from top to bottom:

Reddish/dark–brown spots and sap flow down the stem; The same spots under the bark; Bud rot in an advanced stage of stem bleeding; Severe attack of stem bleeding leading to the death of the coconuts.

Plate 5. Hartrot/Marchitez sorpresiva.

From left to right, from top to bottom:

Medium stage of hartrot: all the lower leaves are brown/desiccated, yellowing of young leaves; Early stage of hartrot on a 3 year old coconut: yellowing of the lower leaves; Marchitez sorpresiva on a 18 year old oil palm: browning of the lower leaves; Yellow wilt of *Alpinia purpurata* caused by trypanosomatids identical to the *Phytomonas* associated with marchitez sorpresiva of oil palms; Electron microscopy picture of trypanosomatids associated with hartrot.

Plate 6. Red ring.

From left to right, from top to bottom:

Young coconut, affected by red ring disease; Red ring inside the trunk of an old coconut at one metre above the soil; Red ring in the trunk just under the meristematic zone.



Plate 1. Bud rot/*Phytophthora*.

Dollet *et al.*



Plate 6. Porroca/Little leaf.

Dollet *et al.*

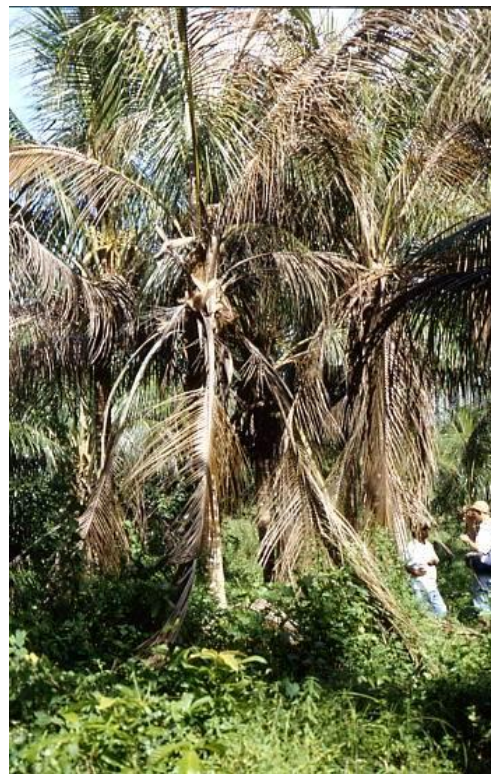


Plate 3. Lixa grande/Lixa pequena /Queima das folhas

Dollet *et al.*

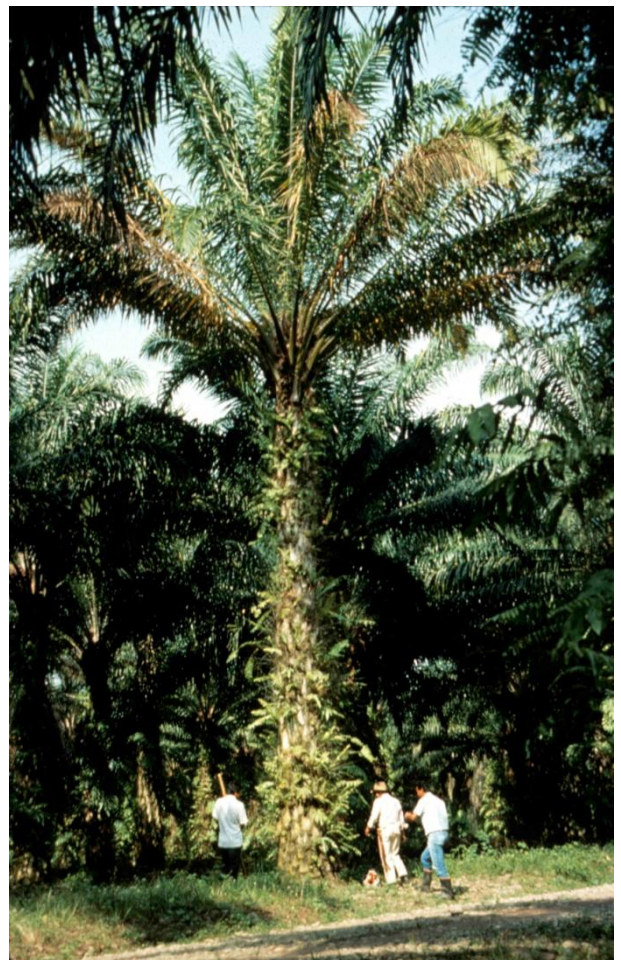


Plate 5. Hartrot/Marchitez sorpresiva

Dollet *et al.*



Plate 6. Red ring.

Dollet *et al.*

