Expl Agric. (2003), volume 39, pp. 225–240 © *2003 Cambridge University Press*
DOI: 10.1017/S0014479703001315 *Printed in the United Kingdom*

REVIEW PAPER
OIL PALM BUD ROT IN LATIN AMERICA†

By H. DE FRANQUEVILLE‡

Tree Crops Department, CIRAD-CP, TA 80/02 - Avenue Agropolis, F34398 Montpellier Cedex 5 – France

(*Accepted 6 December 2002*)

SUMMARY

In South and Central America, tens of thousands of hectares of oil palms (*Elaeis guineensis* Jacq.) are affected by bud-rot types of disease. Having destroyed entire estates in Panama, Colombia, Suriname, Brazil and Ecuador, they are holding back the development of oil palm cultivation in Latin America. The cause is unknown. Indeed, 30 years after these diseases first wreaked havoc on a large scale, it is still not known whether we are dealing with one or more diseases of infectious origin, or with a physiological disorder. Despite lengthy research launched in the early 1980s, no pathogens or insect vectors have been clearly identified. At present, genetics look likely to offer a solution in the medium- to long term, using traits of resistance transmitted by the native species on the American continent, *Elaeis oleifera*, to the interspecific hybrid *E. oleifera* × *E. guineensis*.

INTRODUCTION

Oil palm (*Elaeis guineensis* Jacq.) cultivation is undergoing substantial development in Latin America. Approximately 370 000 ha are currently under mature crops. Only 60 000 ha were harvested in 1970, 100 000 ha in 1980 and 245 000 ha in 1990. In 2000, the 135 000 and 96 000 ha harvested respectively in Colombia and Ecuador accounted for more than 60 % of Latin American oil palm plantings (FAOSTAT, 2001). For several years, Colombia has been the fourth largest producer in the world, behind Malaysia, Indonesia and Nigeria.

Unfortunately, oil palm cultivation in this geographical zone is confronted with a wide range of pests and diseases, most of which jeopardize its sustainability. In particular, it is bud-rot type diseases that pose the greatest threat. These begin with rotting of the spear. If the infection spreads to the meristem, palm death occurs. Examples include *pudrición del cogollo* (PC) and *amarelecimento fatal* (AF). They are found primarily in Ecuador, Colombia, Brazil, Panama and Suriname and are of considerable economic importance, as they have already caused the complete destruction of the oil palm crops on several estates.

Reference is made to 'bud-rot type' diseases as they can take on two forms: a lethal form, which is predominant in Ecuadorian and Brazilian Amazonia and in certain zones of Colombia and Suriname; and a non-lethal form, with a high recovery

† This paper is based on a review published on the internet: www.burotrop.org
‡ Email address of author: hubert.de_franqueville@cirad.fr

rate, which is found mainly in the Colombian *Llanos*. It is not known whether these are different manifestations of the same disorder, or whether distinct pathological or physiological phenomena are involved.

No etiological agent has been found yet and the biotic or abiotic origin, of what is referred to as 'PC' in the rest of this document, remains a subject of controversy. Some teams of researchers feel that agro-physiological factors, such as drainage or the toxicity of certain mineral nutrients lie behind this problem. Others consider that one or more diseases are responsible. Following infection, symptoms spread from plot to plot, developing exponentially from a given threshold or a given age, depending on the situation.

In the 1960s, much work was devoted to PC research some of which came to a standstill faced with the size of the problem. Many hypotheses were put forward but none has been verified. Of numerous factors considered to be propitious to PC development, most came up against counter-examples that refuted the proposed argument.

The purpose of this paper is to review the different areas that have been explored, and to identify those hypotheses that can be rejected, and those that are worth taking further in an attempt to shed light on the origin of PC with a view to developing either integrated or non-integrated control strategies.

THE BUD ROT SITUATION

Oil palm bud rot: a brief history

The first large-scale PC damage occurred during the 1960s. It was from 1965 onwards that the private company-owned La Arenosa estate in the Turbo region of northern Colombia suffered substantial losses. In a few years PC swept across virtually all the 2800 ha that had been planted to *Elaeis guineensis* in 1960–1961. Turner (1981) reported that in 1968 and 1969, PC destroyed 49 000 oil palms over some 1800 ha of that plantation, i.e. almost 20 %. By 1973, only 850 ha were still in bearing. By 1975 no palms were left. Attempts, in 1971, to replant had failed.

Interspecific hybrids of *E. oleifera* × *E. guineensis* planted in 1968, however, were apparently resistant to PC. From 1973 to 1976, therefore, the company embarked on the conversion of 1945 ha to hybrid plantings. Certain social and political events forced the company into liquidation in 1981 and the plots were subsequently redistributed and reconverted to food crop or banana fields (Ospina Bozzi, 1998).

It was probably not a new disease, nor a disorder that had so far gone undetected. According to Richardson (1995), quoting observations by Reiking at the Almirante estate, a similar syndrome had been reported as early as 1928 in Panama. That was the first time that PC was recorded on the, at that time, recently introduced crop. Around 1970, the Icacal estate in Panama (Colon), between the Almirante and Turbo zones, was destroyed (F. Corrado, personal communication). Between the 1930s and 1960s, events in this area remain confused.

In that period, however, Ghesquière (1935) reported the occurrence of a bud-rot syndrome in the Coquilhatville region of the Belgian Congo (now the Mbandaka

region of the Democratic Republic of Congo), and Bachy (1954) described similar symptoms in the Sibiti region, in Congo Brazzaville. Various organizations then launched research work on this syndrome, but political disturbances in central Africa at the beginning of the 1960s soon put an end to promising investigations.

The end of the 1960s saw the emergence of PC in the Colombian *Llanos* (Corrado, 1970), though with less of an explosion in terms of kinetics and mortality than had occurred at Turbo. In 1985, the disease was detected in the Tumaco region (Jimenez, 1991). From 1988 onwards, PC occurrence increased in the *Llanos* (Nieto and Gomez, 1991; Gomez, 1995). It also appeared at that time in the northern and western zones of Colombia, and started spreading considerably in 1994 (Gomez, 1995). To date, however, the devastation of the Turbo scenario has not been repeated in this area, notably due to the high proportion of diseased palms that recover.

Such is not the case, however, at the Victoria estate in Suriname where 1700 ha of oil palm were established in 1971. Around 1976, the disease was detected here on four-year-old palms (Van de Lande, 1986; 1991; 1993). PC incidence trebled from 1984 to 1987 and caused 85 % losses in 1992. Interestingly, FAO statistics show that oil palm plantings in Suriname covered a harvested area of 5425 ha in 1990 and only 40 ha in 2000 (FAOSTAT, 2001). Though the serious political events between 1986 and 1988 (that led to a halt in operations on oil palm estates) partly explain this situation, the figures highlight the severity of the damage that PC can cause. It can lead to oil palm cultivation being abandoned in certain zones.

The 1980s were also marked by the development, in Brazil, of a disorder similar to PC (Van Slobbe, 1987, Kastelein *et al*., 1990). Known locally as *Amarelecimento fatal* (AF), the first cases were recorded in 1974 in palms planted from 1967 onwards on estates belonging to Denpasa, at Paricatuba, near Belém in the state of Para (Mariau *et al*., 1992). Initially the cases were few and scattered. Then, from 1983–1984 onwards, foci began to form. Between 1988 and 1990, 2000 of the 5300 ha initially planted were devastated by AF. The later plantings subsequently disappeared in the 1990s.

It was in 1988 that the first cases were observed on crops planted in 1984–1985 at the Emade estate, near Tefe, in Brazilian Amazonia (Mariau, 1992). The estate was obliterated in just a few short years. On the other hand, no cases of PC have been recorded in the Manaus region, or in the state of Bahia where semi-wild oil palm groves have existed since the 16th century. In the province of Amapa, AF caused some damage initially in 1987, but cases have remained scattered and the disease does not seem to have taken on a serious dimension.

In Ecuador, the first precise observations on bud rot date from 1976, but it is likely that the disease had already been rife there for several years (Dzido *et al*., 1978). The first cases were recorded on the Pacific slopes of the Ecuadorian cordillera, at Palmeras de los Andes, on three- to four-year-old palms. Subsequently, damage remained relatively limited and cases of recovery were recorded regularly. In the same article, Dzido *et al*. reported that, to their knowledge, no case had been reported on palms younger than three years old in any other Ecuadorian plantations. At that time, their study was limited to the Pacific slopes. Soon after, in 1979, PC appeared on two-year-old palms on the Amazonian slopes, foreshadowing one of the most devastating

explosions of the disease. It began in 1992–1993 near Shushufindi (affecting more than 5000 ha) and especially at Huashito (also more than 5000 ha) where virtually no palms had survived by 2000.

Colombia, Ecuador and Brazil form the epicentre of problems linked to bud-rot type diseases. That does not mean that neighbouring countries are PC-free. However, there is little information available about its incidence in those zones.

In Peru, the largest estates have been spared so far (for example, Palmas del Espino), though Mariau (1995) recorded symptoms similar to those of PC in the Iquitos region. The first cases seem to have occurred in 1992–1993 on palms planted in 1984–1985.

In Venezuela, Díaz and Castellano (1988) reported the existence of PC in a pilot plot in the state of Monagas, emphasizing its endemic nature. They did not provide any further information as to the date of first occurrence, or on the degree of damage.

Oil palm estates in Costa Rica are affected by common spear rot (CSR), which seems to be related to Crown Disease (CD). The latter has been known for a long time on certain types of planting material and in seed gardens in West Africa (de Berchoux and Gascon, 1963). Chinchilla and Durán (1999) considered that a continuum existed, between CSR/CD and lethal forms of PC, within which a set of factors led to more- or less-severe expression of the symptoms and their development towards either recovery or death. In the same spirit, Alvarado *et al*. (1996) felt that the knowledge obtained through a study of factors propitious to CSR might be useful in understanding the mechanisms that culminated in bud-rot type diseases. Chinchilla and Durán (1999) also mentioned the appearance in 1992 of dry spear rot in the Quepos region, on a plantation of 3000 ha, some plots of which were up to 70 % affected. This dry rot also fitted into the continuum mentioned above.

Description and symptomatology

Various authors have described the symptomatology of PC or PCs, usually with respect to the zones in which they worked. Mariau *et al*. (1992) and Swinburne (1993) presented overviews. In the early stages of disease development, there is a fairly constant general symptomatology, but it may evolve differently depending on the situations in which affected palms are found. The distinctions that can be made frequently concern the speed with which the rot spreads to the meristem, and the powers of remission of the observed symptoms.

Generally speaking, the first symptoms involve chlorosis of young fronds. Wet rot can be found on the leaflets of spear fronds from where it spreads by contact from one leaflet to the next. Deliquescence of the tissues is then seen at the base of these fronds. This spreads towards the growth point.

In Ecuador, three types of apparent symptoms exist. On the Pacific slopes, rot spreads slowly and is rarely lethal. On the Amazon slopes, rot spreads very rapidly to the meristem, sometimes goes around it, and invades all the youngest tissues. In the last few years in Ecuadorian Amazonia, cases of a rot that spreads even more slowly have occurred alongside the previous two types. It is accompanied by a shortening of fronds (Perthuis and de Franqueville, 1999).

At Paricatuba (Para, Brazil), rot spreads more slowly than in Ecuador, but the yellowing of young fronds, which then turn brown, is more marked. There can be partial or temporary remissions resulting in the emission of small, irregular fronds. At Tefe, the symptoms resemble those observed in Ecuador. In Amapa, they are similar to those in Paricatuba, and Mariau *et al*. (1992) reported the frequent formation of a scar barrier over the meristem, which prevents the rot from spreading.

In Suriname, the symptomatology begins with leaflet chlorosis on unopened fronds 1, 2 and 3 (Van de Lande, 1993), accompanied by necrotic patches. The chlorosis and necrotic patches spread to the young fronds and fronds in the middle crown. At an advanced stage, the spears snap and the central leaf cabbage disappears. Remission may occur, resulting in the emission of apparently healthy, but shorter-than-normal fronds. The rot affects the meristem in young palms more frequently than in adult palms. The same palm can pass through the symptom expression/remission phases several times.

In the *Llanos* of Colombia, the first symptoms are a gradual desiccation of the spear, but the young fronds remain green longer than in the situations described above. The rot develops more rapidly to the terminal bud in young palms than in older palms. In many cases, the rot stops spreading before it reaches the meristem. This cessation in symptom spread results firstly in the emission of new fronds that are small and deformed, then in the emission of normal fronds, leading to total remission.

Epidemiological processes

After PC appears, it spreads linearly for a few years, then exponentially from a given moment. The duration of these successive phases varies. Some estates have yet to experience an exponential phase (for example, the Pacific slopes of Ecuador).

There is little experience for use in judging phenomena that occur in replantings. Obviously, more information is available on oil palms in their first cropping cycle. In Ecuadorian Amazonia, the linear phase (phase 1) lasts for approximately twelve years, with an average rate of 1 % of palms affected per year. The exponential phase (phase 2) starts when approximately 12–15 % accumulated losses are reached after 12 to 13 years of cultivation. The phenomena are more pronounced at Huashito than at Shushufindi. At Paricatuba, in Brazil (Denpasa), phase 2 begins when only 2–3 % of accumulated losses have been recorded. In Suriname, the phenomena observed are similar to those in Brazil.

The so-called linear phase corresponds to random dispersion of PC cases. It does not entail any economically heavy losses below 1 % palm loss per year. Moreover, partial compensation occurs in production gains recorded on palms next to dead palms. The so-called exponential phase is triggered once foci begin to form and join up with increasing rapidity.

Gradients have been found in the spatial development of PC, at least once it has appeared. In Ecuadorian Amazonia, a clear border effect has been detected, with the first cases occurring in plots located on the edge of the forest. In Brazil and Suriname,

the disease spreads in the direction of the prevailing winds, from east to west (Van Slobbe and Rocha de Souza, 1991; Mariau *et al*., 1992; Van de Lande, 1993).

A spatio-temporal analysis of the disease in Suriname was carried out by Van de Lande (1993) and completed by Van de Lande and Zadoks (1999). The main conclusions indicated or confirmed that disease dispersion followed a preferential direction. The data were compatible with the following hypotheses: (i) PC – or AF – is a disease resulting from an infectious process; (ii) the causal agent is conveyed by a wind-borne vector; (iii) PC occurs in two distinct phases, phase 1 being characterized by a few palms distributed at random, and phase 2 by the development of foci from these isolated cases. For these authors, the causes of the switch from phase 1 to phase 2 remained unknown, hence they did not rule out the hypothesis that two different populations of vectors might be involved.

Using data from Denpasa, Bergamin Filho *et al*. (1998) and Laranjeira *et al*. (1998) drew very different conclusions from their analyses. According to these authors, the conventional epidemiological models do not explain the data gathered. There is no preferential direction for disease spread, which rules out the involvement of a vector borne by the wind, but rather a tendency to find diseased palms near watercourses. As disease development is not explained by the propagation of any pathogen, the general conclusion of this study was that PC – or AF – is due to an abiotic phenomenon.

In all places where it was possible to replant devastated plots, it has been found that PC occurs much earlier than first-time plantings in extended areas. As in Brazil and Suriname, the first cases in Ecuador appear between nine months and a year after replanting. That was also the case at Turbo, in Colombia, after the disaster at the end of the 1960s. This suggests that there is a major soil effect. So far, little assessment has been made of its cause.

Genetic resistance and susceptibility to bud rot

The massive losses recorded at the La Arenosa estate in Colombia during the 1960s, spared the *Elaeis oleifera* × *Elaeis guineensis* interspecific hybrid populations (Turner, 1981; Renard and Quillec, 1984). In the Ecuadorian Oriente, where the lethal form of PC predominates on *E. guineensis*, PC symptoms can be found sporadically on interspecific hybrids but, usually, development of the symptoms soon stops. This generally corresponds to the formation of a scar-tissue barrier over the meristem, which prevents the rot from spreading.

There is a source of resistance, therefore, in *E. oleifera* and in its hybrid, and use of such resistance is probably going to be the only long-term solution for the PC problem (Meunier, 1991). However, any strategy along such lines comes up against the problem that, due to partial sterility, the interspecific hybrid produces less oil than does *E. guineensis* (around 30 %). Hence its planting in a monoculture encounters pollination problems. The hybrid material is highly feminine, and the rare male inflorescences that appear produce only a small amount of viable pollen. Pollinating insects are not particularly drawn to the male inflorescences, whilst the attraction of

female inflorescences on these hybrids is good. The degree of hybrid sterility varies depending on the origin of the parents used.

Currently, the only possible option is to study and choose the best interspecific hybrids with good pollen viability. Some hybrids are known for their good fresh fruit bunch (FFB) production, which is at least equivalent to that of the best *Elaeis guineensis*.

In the longer term, the search for high-yielding material with resistance to PC requires introgression of *E. oleifera* resistance in high-yielding *E. guineensis*, and it is this avenue that is being explored by CIRAD geneticists. *In vitro* culture of backcross embryos is being used in the search for molecular markers of resistance to PC. Planting of such markers in severely affected zones of Ecuador, Colombia and Brazil will make it possible to monitor the introgression of resistance in the best *E. guineensis*.

Sources of resistance to PC among the pure populations of *E. guineensis* do not seem to exist despite differences in performance depending on origin. Several statistical-design trials have been established at Shushufindi to compare the performance of various categories of planting material. In a trial with four different categories, planted in 1983, one was shown to be more susceptible than the others, insofar as the exponential phase began at around 12 years, whereas the other categories only reached that phase two years later. It is undoubtedly not insignificant for growers, but the disappearance of those materials seems to be unavoidable.

It can be said, therefore, that there are differences in susceptibility, enabling longer exploitation of the first cultivation cycle depending on the origin of the planting material. There is as yet no degree of resistance, however, that can be used to embark on a second cultivation cycle in a replanting. Trials were set up at the beginning of 2000 in Ecuador to confirm this observation.

Priority needs to be given to genetic improvement. Given the length of the selection cycles, however, this will require time (about 10 years) and continuity. A tool to enable reproduction of the disease under controlled conditions at an early stage could considerably reduce the time lapse imposed by the biology of the oil palm. In this regard, the development of a screening test for planting material would be a spectacular step forward. In order to achieve this, however, it would be necessary to be able to induce PC symptoms, with all their lethal consequences, through a vector or an identified pathogen. It would be necessary also to know the factors that make the oil palm liable to infection.

Current knowledge is insufficient to develop any tool. The following section reviews the hypotheses that have been explored.

REVIEW OF WORK AND OF THE HYPOTHESES EXPLORED

Hypotheses of biotic origin

The role of insects. A lot of effort has been invested to discover the biotic origin of PC and elucidate its etiology. Much of this work has concentrated on the search for an airborne insect vector. This line of research seemed logical and a priority, given the aerial expression of the disease symptoms and the existence of gradients from the plantation boundaries at Shushufindi, and in line with the prevailing winds at Denpasa. It also

seemed logical due to the analogies found with other diseases transmitted by insects, for which research had been successful.

In Brazil and in Ecuadorian Amazonia, inventories were made of the entomofauna on oil palms and in the plantation surroundings (Louise, 1990; Perthuis, 1990; Perthuis, 1990). The purpose was to identify suspect species, whose population variations bore a direct relation with disease incidence. At the same time, trials were carried out to try to transmit PC using these and numerous other species, in the hope of detecting the role of one or more of them in transmitting the disease.

Several hundred different species were found to visit oil palms. In Belém, there were at least 400 with three predominant families: Derbidae, Cicadellidae and Membracidae. In Ecuador, the same families were found, and the total number of species encountered was even larger.

In both cases, and over several years, hundreds of thousands of insects belonging to the most common species, and also many less common species, were released into cages; dozens of cages in Brazil and hundreds in Ecuador. No positive results were obtained.

Trials with insecticide treatments were set up in the two countries in an attempt to show indirectly the role played by insects in disease transmission. No reduction in disease spread was seen in the treated plots compared with the control plots, in either country.

There was, therefore, no indication that Homoptera played a role in PC transmission in either Brazil or Ecuador.

It was also not possible to establish a correlation between the insects observed and PC incidence from inventories of the entomofauna found on oil palm in Colombia. Insecticide trials did not give any significant results either: the treated plots, in which insecticide populations were zero, suffered from as many cases of PC as did the untreated control plots that were rich in entomofauna (Gomez, 1995).

All this work shows that the hypothesis of air-borne transmission by Coleoptera, Lepidoptera and most Homoptera can be ruled out. However, other groups of Arthropods, such as mites, can be involved in the transmission of disease, but no entomological studies have been carried out on these groups. Also, with the exception of the pollinating Curculionidae of the genera *Mystrops* and *Eleaidobius*, the fauna so specific to inflorescences have not been inventoried let alone tested. The same applies for fauna associated with wounds.

Little is known of any relevant soil-borne entomofauna. In view of PC dispersion, which now suggests that the origin of the disease is more soil-borne than air-borne, this is an avenue that needs to be explored.

The introduction of pollinating insects was a biological event that marked the 1980s (Mariau and Genty, 1988). PC is not related to this introduction: La Arenosa estate was destroyed before the role of pollinating insects was discovered in Africa (Syed, 1979) and introductions took place after the appearance of large foci at Denpasa.

Furthermore, the possible role of pollen in disease transmission has been tested in Brazil. At least 1000 apparently healthy female oil palm inflorescences were pollinated

with pollen taken from 500 different diseased palms, but no significant result was obtained (C. Louise, personal communication).

The role of fungi and of bacteria. As early as 1928, Reinking – quoted by Richardson (1995) – set out to identify microorganisms associated with bud rot in the Almirante region of Panama. He discovered the presence of bacteria, *Fusarium moniliforme* and a possible *Phytophthora*.

A little later, in central Africa, Ghesquière (1935) reported the association between *Phytophthora palmivora*, *Bacillus coli* and bud rot in the oil palm plantings of Coquilhatville province in the Belgian Congo. *Thielaviopsis paradoxa* has been mentioned as being capable of invading oil palm affected by *Phytophthora*. At Sibiti, in Congo-Brazzaville, Bachy (1954) noted a close association between bud rot, bacteria and *Fusarium*, primarily *F. oxysporum*, *F. solani* and *F. roseum*. Artificial inoculation with these different microorganisms did not give any results. Some years later, Duff (1963) invariably isolated a bacterium of the genus *Erwinia*, similar to *Erwinia lathyri* (= *E. herbicola*) from diseased tissues, with which he obtained rot symptoms on oil palm seedlings after inducing a physiological shock causing retarded growth. This work was not continued.

At Turbo in Latin America also, microbiological isolations showed the predominance of *Fusarium* (*F. oxysporum* and *F. solani*) and bacteria in diseased tissues. Attempts to artificially inoculate with these microorganisms, separately or combined, did not lead to the production of disease symptoms (Renard, 1976).

In Ecuador, studies by Quillec (1983) led to similar conclusions as to the nature of the microflora associated with bud rot. The *Phytophthora* avenue was explored again (Quillec had isolated a Pythiaceae) but the very low rate of positive isolations led him to conclude that Pythiaceae were not involved in the bud rot syndrome in Ecuador. At the same time, trials over several successive years with Fosetyl-Al (Aliette) or Metalaxil treatments – fungicides specific to Pythiaceae – drew a blank. Similarly, fungicide and antibiotic treatments against other potential pathogens were inconclusive.

In 1998, de Franqueville inoculated the soil at the nursery stage with *Fusarium*. A few months later, half the inoculated plants received a bacterial suspension applied to the spear. This trial was based on the hypothesis of a soil-borne origin for PC, caused by hypovirulent strains of *Fusarium* migrating towards the bud systemically, and then promoting bacterial development. Disease symptoms were not reproduced (unpublished results).

With the exception of work at Turbo early in the 1960s, perhaps, few studies have been made of interactions between *Fusarium* sp. and enterobacteria of the genus *Erwinia*, which make up the microbiological constants of PC; *Erwinia* has generally been considered as a secondary agent, whose inoculation alone has never led to any symptoms.

Apparently, early stages of spear rot have been induced on several occasions by distinct inoculations of *Fusarium* or *Erwinia*, be it in the nursery or in the field. There has always been remission, however, and the lethal consequences of PC have never been observed.

In Colombia, the bacterial microflora did not particularly attract the attention of researchers. They concentrated more on the hypothesis of a fungal origin to the disease. Thus, they discovered various fungal organisms associated with spear rot and bud rot symptoms. The Colombian researchers focused first on various *Fusarium* species, notably *Fusarium solani* (Nieto and Gomez, 1991), then turned to a fungal complex comprising *Fusarium* spp., *Pythium* spp. and *Thielaviopsis paradoxa* (Nieto, 1996). Further work suggested strongly that *T. paradoxa* played a vital role in pathogenesis (Gomez *et al*., 2000).

In Ecuador and in Brazil, only rarely did hundreds or even thousands of isolations give rise to *Thielaviopsis* sp. cultures. Consequently, it is difficult to link this fungus to PC in those countries with any certainty. Should it be assumed, therefore, that the etiology of PC is not the same everywhere? Are different diseases or abnormalities responsible, depending on the country?

The role of viruses, viroids or phytoplasmas. The search for a possible vector insect was logically to go hand in hand with virological research, and substantial resources were devoted to that task from 1983 to 1990 (Dollet, 1991). This work was carried out in two main phases: the first, from 1983 to 1987, involved techniques employing *in vitro* culture by somatic embryogenesis, and electron microscopy; the second, from 1987 to 1990, involved nucleic acid studies. The methodologies used fitted in with a general strategy for studying diseases of unknown etiology, recapitulated in Dollet (1992).

At Shushufindi, field samples taken from palms at different stages of the disease, and from several organs (leaflets, base of spears, inflorescences, meristematic zone and roots) were examined under the electron microscope. These examinations did not reveal any phytoplasma or virus-type pathogens.

Faced with the impossibility of visualizing a possible virus, nucleic acids extracted from healthy and diseased plants were compared in the hope of detecting a replicating form (double-stranded RNA) of the viral genome (Dollet, 1991; 1992; Dollet *et al*., 1994) or the presence of a viroid. Detection of numerous double-stranded RNAs in the oil palm showed that they were in much higher concentrations in diseased palms than in palms assumed to be healthy, but no clear additional band appeared among the double-stranded RNA from diseased palms that might suggest the existence of a viral genome. Moreover, molecular hybridization trials with viroid probes did not enable a specific link to be established with PC at Shushufindi. Similar observations have been carried out on AF in Brazil (Beuther *et al*., 1992).

Unfortunately, this work was halted prematurely at the beginning of the 1990s. It can be imagined that, had this work been continued, it would have benefited from the new techniques and methodologies developed over the last decade.

The role of nematodes. Depending on the species, nematodes can cause or transmit diseases, be they viral or otherwise. Transmission trials were conducted by regularly applying soil from around diseased palms over several months. Other trials involving nematode inoculations in the bud, alone or in combination with bacteria, were launched. None of these trials gave any results (IRHO, 1986; 1987).

More recently, in Colombia, Guevara and Nieto (1999) examined this subject without being able to establish a direct relation between PC and the existence of nematodes known for their pathogenicity or vector role, thus confirming the observations reported by Gomez (1995).

Hypotheses of an abiotic origin

The line dividing the study of biotic factors propitious to the disease from that of abiotic factors that would seem to be strictly responsible, is very vague.

In reference to spear rot observed in the Quepos region of Costa Rica, Chinchilla and Durán (1999) proposed a scheme by which spear infections belonged to a continuum whose extremes consisted of Crown Disease on the one hand, and bud rot in its lethal form on the other hand.

For these authors, spear infections were always associated with a poorly developed root system, with multiple malformations linked to the conditions to which the palms were subjected. However, their observations did not tally with those of other authors, such as Renard (1976), Quillec (1983), Mariau *et al*. (1992), Jimenez (1991), or Braida (1999). For this latter group, the root system remained healthy for a long time after symptoms appeared and its structures were not altered, enabling middle and upper fronds to remain green for several months.

Also according to Chinchilla and Durán, the severity of such infections would seem to depend on a multitude of factors, including, in addition to climatic factors, soil aeration, various nutritional disorders, such as low phosphorus and potassium levels, very high magnesium and calcium levels, excess nitrogen, low levels of zinc and copper, and also the levels of iron and manganese in the soil.

The role of soil type. Bud rot occurs on all types of soils. This is notably the case at Denpasa, in Brazil, where the diversity of sandy or clay soils has not differentially limited the spread and generalization of PC. Nevertheless, numerous authors consider that PC is directly linked to poor drainage, heavy compact soils, and root system asphyxia. As soon as PC appeared at Turbo, in Colombia, the absence of drainage and inadequate fertilization were suspected of being the cause (Hartley, 1965). In also referring to Turbo, De Rojas Pea and Ruiz (1975), quoted by Gomez *et al*. (2000), suggested that PC might be caused by an accumulation of nitrites linked to the lack of oxygenation in the heavy, poorly drained soils. Poor drainage, compaction and low hydraulic conductivity were also held to be responsible in Brazil (Rodrigues *et al*., 2000) and in Colombia (Gomez *et al*., 2000).

Soil acidity has been mentioned as a possible factor inducing or favouring PC, as has the role of aluminium, which increases in toxicity in line with pH acidity. It is true that most of the zones affected by PC are very rich in aluminium, but that is also true for PC-free zones, such as the Manaus region (Ochs, 1989). Be that as it may, an in-depth examination of PC/aluminium content relations undoubtedly needs to be undertaken.

Turner (1981) does not seem to share the opinion that edaphic or nutritional factors directly cause PC. He observed that vigorous, well-fertilized palms were just as severely attacked as were palms clearly affected by various disorders.

The role of mineral nutrition. Mineral trials conducted in Ecuador in the 1980s did not show any significant effect that could link fertilizer management to PC development. However, those trials were conducted when the disease only appeared in a random and dispersed manner, i.e. before the exponential phase of PC development. Two fertilization trials were launched at Denpasa in Brazil during the first planting campaigns in 1968 (Pacheco *et al*., 1985) and continued up to the start of the exponential phase in 1987 (Van Slobbe, 1996). Observations carried out up to 1988 showed that the different treatments did not have any effect on disease development and that N, P, K, Mg or Ca were therefore not involved in predisposing the palms to PC.

Several teams of researchers endeavoured to use leaf analyses to compare concentrations of macro−elements and trace elements between healthy palms and diseased palms, or between palms in PC-free zones and palms in affected zones. Viegas *et al*. (2000) measured Cu, Fe, Mn, and Zn concentrations in leaves 1, 9 and 17 of healthy palms, diseased palms and interspecific hybrids. Copper contents were slightly lower in diseased *E. guineensis* palms than in healthy palms, and much lower in *E. guineensis* palms than in the hybrids, irrespective of phytosanitary condition and the leaf rank analysed. Iron contents were roughly the same in leaves 1 and 9 of healthy and diseased E. *guineensis* palms and hybrids, but higher in leaf 17 in the diseased palms. This suggested to the authors that toxicity of that nutrient might be linked to PC. Manganese concentrations were the same in leaf 1, irrespective of the planting material. In leaves 9 and 17 Mn concentrations were the same between diseased palms and hybrids in leaves 9 and 17, though lower than the concentrations found in healthy palms. Lastly, the Zn contents were much lower in the diseased palms, irrespective of the leaf rank analysed. Viegas and his colleagues suspected therefore that these four elements played a decisive role in the disease. Their opinion tallies with that expressed shortly before by Chinchilla and Durán (1999).

Gomez (1995), with reference to the *Llanos* in Colombia, considered no major or minor element to be individually associated with PC. More recent work, launched by Cenipalma in 1999 (Gomez, 2000) compared the concentrations of different elements in rank 9 leaves of diseased palms and palms located in healthy zones. The differences involved P, K, Ca, Mg and Cu, and the Ca:B, N:K, Ca:K and N:P ratios. Gomez concluded with the need to initiate agricultural trials designed to evaluate these parameters effectively.

Effect of other cultural practices. Numerous cultural techniques have been examined in the light of the effect they might have on PC appearance. At Shushufindi, castration or severe pruning was not found to have any effect, thereby minimizing the hypothesis of possible transmission by harvesting tools (IRHO, 1986). No effect of grasses or cover crops could be detected (IRHO, 1986; Van Slobbe and Rocha de Souza, 1991).

The elimination of diseased palms is generally recommended. This is justified if a biological agent is responsible for PC, but less so if PC is of abiotic origin, apart from limiting the risks of proliferation of *Rhynchophorus* or other harmful insects, whose larval sites are located in rotting stems. However, there is no formal proof of the beneficial effect of this measure, which is difficult to demonstrate experimentally. Van Slobbe and Rocha de Souza (1991) reported that nine months after starting a campaign of systematic eradication of diseased palms, the speed with which PC spread at Denpasa had fallen by 50 %. At the same time, however, they noted a reduction of around the same degree in plots without eradication. In Ecuador, mortality spread more rapidly at Huashito, where systematic felling was abandoned, than at Shushufindi, where elimination of diseased palms is continuing. It is difficult, however, to establish a cause-and-effect link.

At Denpasa, an observation was carried out which, whilst it may not lead to a technically recommendable cultural practice, is worth reporting (A. S. Veiga, personal communication). A plot was subjected to burning prior to replanting, in 1992. All the stems of palms from the first generation were removed and the periphery of the plot slashed over a width of 100 m. The first cases of the disease did not occur until two years after planting, i.e. more than a year after the first cases were recorded in plots that had not be subjected to this treatment. This suggests that there may have been decontamination of the soil, albeit transient, or elimination of a host plant. If it was a matter of soil decontamination, it can be imagined that the treatment had an effect on a pathogen present in the soil or in stem tissues, or on a population of soil-borne vectors.

DISCUSSION

The preceding notes may sound like a long catalogue of negative or not very conclusive results. Indeed, thirty years after Turbo, the role of an etiological agent in the bud-rot syndrome has never been clearly demonstrated in all the affected areas. Also, the disease, with all its lethal consequences when it exists, has never been reproduced by artificial inoculation of a fungus, bacterium or nematode, or by releasing a potential vector into a cage. The search for viruses, viroids or phytoplasmas has drawn a blank. Koch's postulates remain to be verified in the different areas.

PC is a specifically Latin American phenomenon[1]. Yet it can be said that, from Africa to Southeast Asia, oil palm is a crop that is planted on highly varied soils, is too often faced with a multitude of adverse factors, is subjected to inappropriate cultural practices, and an ongoing lack of upkeep, poor drainage and/or toxicity. *Fusarium*, *Thielaviopsis* and *Erwinia* are ubiquitous, sometimes pathogenic it is true, but they also exist in zones where they do not cause any damage. The genetic background of the planting material, whatever the commercial origin, is also widely spread throughout the oil palm growing area, and does not vary fundamentally between Ecuador, Brazil,

[1] Little information is available on the development of rot diseases observed at one time in the Congo (Brazzaville and Kinshasa).

the Ivory Coast or Indonesia. Indeed, sporadic cases of spear- or bud-rot are recorded just about everywhere, but nowhere does the damage caused reach the severity seen in Latin America.

The origin of 'bud rot' remains an enigma, despite the multidisciplinary approach taken in trying to solve it. This approach needs to be continued through close collaboration between the different countries in which it is a real or potential threat.

Such collaboration would make it possible to conclude or advance on the following points:

- Is the disease the same throughout the continent? This is the essential question. A more in-depth study of the symptomatology and epidemiology, which are so variable from one site to the next, should make it possible to make some headway. For the time being, the diversity of symptoms and ways in which the disease spreads suggest that there are several diseases or different syndromes.
- What are the relative roles of abiotic and biotic factors in the syndrome(s)?
- Does PC result from soil-borne or air-borne factors?

REFERENCES

Alvarado, A., Chinchilla, C. M., Bulgarelli, J. and Sterling, F. (1996). Agronomic factors associated to Common Spear Rot/Crown disease in oil palm. *ASD Oil Palm Papers* 15:8–28.

Bachy, A. (1954). Contribution à l'étude de la pourriture du cœur du palmier à huile. *Oléagineux* 9 (8–9):619–627.

Berchoux, C. de and Gascon, J. P. (1963). L'arcure défoliée du palmier à huile: éléments pour l'obtention de lignées résistantes. *Oléagineux* 18 (11):713–715.

Bergamin Filho, A., Amorim, L., Larenjeira, F. F., Berger, R. D. and Hau, B. (1998). Análise temporal do amarelecimento fatal do dendezeiro como ferramenta para elucider sua etiologia. *Fitopatologia Brasileira* 23(3):391–396.

Beuther, E., Wiese, U., Lukács, N., van Slobbe, W. G. and Riesner, D. (1992). Fatal yellowing of oil palms: search for viroids and doubled-stranded RNA. *Journal of Phytopathology* 136:297–311.

Braida, L. (1999). Contribution à l'étude des caractères cytohistochimiques de l'appareil racinaire du palmier à huile en relation avec la maladie de la pourriture du cœur. *Rapport commandé par Palmeras del Ecuador S.A. Institut de Recherches pour le Développement (IRD) pp. 26* .

Chinchilla, C. M. and Durán, N. (1999). Nature and management of spear rot-like problem in oil palm: a case study in Costa Rica. *Proceedings of the 1999 Porim International Palm Oil Congress (Agriculture) A:29 97–126.*

Corrado, F. (1970). La maladie du palmier à huile dans les Llanos de Colombie. Conseils de l'IRHO No. 97. *Oléagineux* 25 (7):383–384.

Diaz, A. and Castellano, G. (1988). Problemas fitopatologicos de la palma aceitera (*Elaeis guineensis* Jacq.) en Venezuela. *Seminario Problemas fitopatologicos de la palma Africana, Bucaramanga, Colombie, 21–25 mars 1988:134–141.*

Dollet, M. (1991). Etiologia de la pudricion de cogollo. Investigaciones virologicas conducidas por el IRHO. *Palmas* 12 (2):33–37.

Dollet, M. (1992). Strategies used in etiological research on coconut and oil palm diseases of unknown origin. In *Plant Diseases of Viral, Viroid, Mycoplasma and Uncertain Etiology,* 184 pp. (Ed. Maramorosh, K.). Oxford and IBH Publishing Co. Pvt. Ltd.

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

Duff, A. D. S. (1963). The bud rot/little-leaf disease of the oil palm. *Journal of WAIFOR (West African Institute for Oil Palm Research)* 4 (14):176–190.

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

FAOSTAT (2001). Statistical Databases, Food and Agricultural Organization of the UN, http://www.fao.org

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* .

Gomez, P. L. (1995). Estado actual de la investigación sobre pudrición de cogollo. *Palmas* 16 (1):9–23.

Gomez, P. L., Ayala, L. and Munevar, F. (2000). Characteristics and management of bud rot, a disease of oil palm. *Proceedings of the International Planters Conference* , 17–20 *May 2000: 545–553*.

Guevara Avila, L. A. and Nieto, L. E. (1999). Nematodos asociados con palmas de aceite (*Elaeis guineensis* Jacq.) afectadas con pudrición de cogollo. *Palmas* 20 (1):93–99.

Guevara, Y., Rondón, A., Arnal, E., Suárez, Z., Solórzano, R. and Navas, R. (1992). La pudrición bacterial del tallo de la yuca en Venezuela. *Fitopatologia Venezuela* 5 (2):33–36.

Hartley, C. W. S. (1965). Some notes on the oil palm in Latin America. *Oléagineux* 20 (6):359–363.

IRHO (1986). Troisième bilan des recherches sur la pourriture du cœur dans l'Oriente Equatorien. *Document Institut de Recherches pour les Huiles et Oléagineux 2013, octobre 1986 pp. 70*.

IRHO (1987). Quatrième bilan des recherches sur la pourriture du cœur dans l'Oriente Equatorien. *Document Institut de Recherches pour les Huiles et Oléagineux 2085, octobre 1987, pp. 77+annexes*.

Jimenez, O. (1991). Pudricion de cogollo en la palma aceitera en la region de Tumaco, Colombia. *Palmas* 12 (2):45–48.

Kastelein, P., van Slobbe, W. G. and de Leeuw, G. T. N. (1990). Symptomatological and histopathological observations on oil palms from Brazil and Ecuador affected by fatal yellowing. *Netherlands Journal of Plant Pathology* 96 (2):113–117.

Laranjeira, F. F., Amorim, L., Bergamin Filho, A., Berger, R. D. and Hau, B. (1998). Análise espacial do amarelecimento fatal do dendezeiro como ferramenta para elucider sua etiologia. *Fitopatologia Brasileira* 23 (3):397–403.

Louise, C. (1990). Inventory of Homoptera and Heteroptera in relation to the Amarelecimento Fatal disease. In *Spear Rot of Oil Palm in Tropical America. Proceedings of the first international seminar on the identification and control of the organism(s) and/or other factor(s) causing the spear rot syndrome in oil palm. Paramaribo, Surinam, 8–12 March 1998*: *36–46*.

Mariau, D. (1992). Embrapa – CIRAD/IRHO. Mission entomologique, 24 septembre-3octobre (1991).. *Document Institut de Recherches pour les Huiles et Oléagineux (IRHO) 2413, mars 1992, pp. 31+annexes*.

Mariau, D. (1995). Visite de la plantation de palmier à huile de Maniti (Iquitos) pour le compte de Emrepalma, 22–23 *mai 1995. Document CIRAD-Cp 410, juin 1995, pp. 11+annexes*.

Mariau, D. and Genty, P. (1988). Contribution de l'Irho à l'étude des insectes pollinisateurs du palmier à huile en Afrique, Amérique du Sud et Indonésie. *Oléagineux* 43 (6):233–240.

Mariau, D., van de Lande, 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 - Epidémiologie - Incidence. *Oléagineux* 47 (11):605–618.

Meunier, J. (1991). Una posible solucion genetica para el control de la pudricion de cogollo en la palma aceitera. Hibrido interpacifico *Elaeis oleifera* × *Elaeis guineensis*. *Palmas* 12 (2):39–42.

Nieto, P. L. E. and Gomez Cuervo, P. L. (1991). Estado actual de la investigacion sobre el complejo Pudricion de Cogollo de la palma de aceite en Colombia. *Palmas* 12 (2):57–67.

Ochs, R. (1989). Mission d'agronomie 1989. Embrapa – CIRAD/IRHO. 8 – 20 mai 1989. *Document Institut de Recherches pour les Huiles et Oléagineux 2228. pp. 23+annexes*.

Ospina Bozzi, M. L. (1998). *La palma africana en Colombia. Apuntes y memorias. Volumen 2*. Publication of the National Federation of Oil Palm Growers (FEDEPALMA), Santafé de Bogatá, Colombia.

Pacheco, A. R., Tailliez, B. J., Rocha de Souza, R. L. and de Mima, E. J. (1985). Les déficiences minérales du palmier à huile (*Elaeis guineensis* Jacq.) dans la région de Bélem (Pará, Brésil). *Oléagineux* 40 (6):295–309.

Perthuis, B. (1990). Investigation about the pudrición del cogollo disease (PC) of oil palm in the Amazonian part of Ecuador: search of the insect vector. In *Spear rot of oil palm in Tropical America. Proceedings of the first international seminar on the identification and control of the organism(s) and/or other factor(s) causing the spear rot syndrome in oil palm. Paramaribo, Surinam, 8–12 March 1998* : *51–56*.

Perthuis, B. (1991). Busqueda de la etiologia de la pudricion de cogollo en el Ecuador Oriental. *Palmas* 12 (2):25–30.

Perthuis, B., Franqueville, H. de. (1999). Les maladies de type pourriture du cœur du palmier à huile en Equateur. *Rapport de mission à Shushufindi. Document CIRAD-Cp 1090. pp. 19*.

Quillec, G. (1983). Etude de la pourriture du cœur de Shushufindi (Equateur). *Document Institut de Recherches pour les Huiles et Oléagineux (IRHO) 1757, février 1983. pp. 47* .

Renard, J. L. (1976). Diseases in Africa and South America. In *Oil Palm Research* , 447–466. (Eds Corley, R. H. V. Hardon, J. J. and Wood, B. J.). Amsterdam: Elsevier Scientific Publishing Company.

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

Richardson, D. L. (1995). The history of oil palm breeding in the United Fruit Company. *ASD Oil Palm Papers* , 11:1–22.

Rodriguez, T. E., Viegas, I. de J. M., Trindade, D. R., Martins e Silva, H., Frazao D. A. C. and Cordeiro, R. A. M. (2000). Influência das propriedades fisicas do solo na ocorrência do amarelecimento fatal do dendezeiro. Poster présenté au *Seminário Internacional, Agronegócio do dendê: uma alternativa social, econômica e ambiental para o desenvolvimento sustentável da Amazônia. Belém, Pará, Brésil, 16–20 octobre 2000.*

Swinburne, T. R. (1993). Fatal yellows bud rot and spear rot of African oil palm. A comparison of the symptoms of these diseases in Brazil, Ecuador and Columbia. *Planter* 69 (802):15–23.

Syed, R. A. (1979). Studies on oil palm pollination by insects. *Bulletin of Entomology Research* 69:213–224.

Turner, P. D. (1981). *Oil Palm Diseases and Disorders,* pp. 280. Kuala Lumpur: Oxford University Press.

Van de Lande, H. L. (1986). Diseases of fatal character to oil palm in Suriname and North-Brazil. *Surinaamse Landbouw* 34 (1–3):15–32.

Van de Lande, H. L. (1991). Pudricion de cogollo en palma aceitera en Suriname. Investigaciones desde 1986 hasta 1990. *Palmas* 12 (2):11–15.

Van de Lande, H. L. (1993). *Studies on the Epidemiology of Spear Rot in Oil Palm* (Elaeis guineensis *Jacq.) in Suriname*, pp. 153. Wageningen, NLD: Landbouwuniversiteit.

Van de Lande, H. L. and Zadocks, J. C. (1999). Spatial patterns of spear rot in oil palm plantations in Suriname. *Plant Pathology* 48 (2):189–201.

Van Slobbe, W. G. (1987). Spear rot (Pudrición del Cogollo) en Suriname y Brasil. *Proceedings of the IV Mesa Redonda: Valledupar. Colombia. June 1986. FAO. Santiago, 110–112.*

Van Slobbe, W. G. (1996). Oil palm estate Denpasa and other oil palm plantings in Pará. II. *Second Interim Report of Short-Term Consultancy on the Progress of Fatal Yellowing Disease (Amarelecimento fatal – AF). 19 August-17 November 1996.*

Van Slobbe, W. G and Rocha De Souza, R. L. (1991). Amarillamiento fatal o pudricion de cogollo en Denpasa-Brasil. *Palmas* 12 (2):17–23.

Vieigas, I. de J. M., Frazao, D. A. C., Furlan Júnior, J., Trindade, D. R., and Thomaz, M. A. A. (2000). Teores de micronutrientes em folhas de dendezeiros sadios e com sintomas de amarelecimento fatal. Poster présenté au *Seminário Internacional, Agronegócio do dendê: uma alternativa social, econômica e ambiental para o desenvolvimento sustentável da Amazônia. Belém, Pará, Brésil, 16–20 octobre 2000.*