A Review of a Century of Studies on South American Leaf Blight of the Rubber Tree

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Natural rubber is an essential raw material for many uses as it offers some physical characteristics that industrialists are unable to reproduce in synthetic rubber. World natural rubber production was around 12 million t in 2013 from around 10 million ha, of which 91.5% came from Asia (FAOSTAT, <http://www.fao.org/faostat/en/#data>). The rubber tree is mostly exposed to fungal diseases, including South American leaf blight (SALB), which devastated the first plantations planted in Guyana in 1909, then in other countries (Hilton 1955). This disease, for which no economical and effective control has yet been developed, remains a threat to rubber growing worldwide. Although it was first observed at the beginning of the 20th century, the disease and its pathogen are fairly poorly documented and little known. The studies on the disease took place in successive phases, which were summed up by Langford (1945), Holliday (1970), Chee and Holliday (1986), and Lieberei (2007).

This article is a fresh overview of the disease, more particularly focusing on the most recent epidemiological studies, and on the biochemical and molecular aspects of resistance.

The Disease

SALB is a fungal disease of the aboveground organs of the rubber tree. It primarily occurs on leaves (Fig. 1A), but sometimes on petioles and stems in the event of very severe attacks (Fig. 1B). The first symptoms are a discoloration of the lamina, then grayish-green spots with a powdery appearance on the underside of leaves, corresponding to conidial sporulation. Young necrosis differ from necrosis due to *Colletotrichum* sp. or *Helminthosporium* sp. by the irregularity of

their margin and the easily visible asexual sporulation. When the leaves reach maturity, the affected zones become necrotic and conidial sporulation disappears. Then the sexual forms appear, comprising black stromata around the edges of the necroses, mainly visible on the upper side. If infections occur on very young leaves, the lamina stops growing in the diseased zones, leading to deformation. Growth may stop completely and the leaflet abscises. Severely necrotized leaflets often fall after reaching maturity. When massive attacks occur in wet periods in the presence of abundant young foliage (refoliation period), the refoliation process, which is interrupted by the disease, keeps on for as long as the tree is able to emit young leaves and conditions remain suitable for the parasite (Rao 1973). The young stems become less and less vigorous, resulting in low foliage density with numerous dry points (stems drying out from their tip). This phenomenon usually occurs each year and the trees gradually weaken (Fig. 2). Growth is slowed down, production is low, and the tree may die.

Hennings (Stahel 1917) reported SALB for the first time in 1904, on samples of wild rubber trees taken in the Upper Amazon, near the border between Peru and Brazil. However, the economic incidence of the disease only appeared with the extension of rubber plantations at the beginning of the 20th century. The pathogen responsible for the disease, the Ascomycete fungus *Pseudocercospora ulei* was, like its host, native to the Amazon Basin, but it spread throughout South America with the expansion of rubber growing. At the beginning of the 1950s, *P. ulei* was found from southern Mexico (El Palmar) in the north, as far as Ilheus (state of Bahia in Brazil) in the south, as well as in Trinidad. Later, it was reported in all the major rubber growing regions, including some regions very far from its place of origin, such as the state of São Paulo in Brazil (da Cunha 1966) and all the islands of the Caribbean where the rubber tree was introduced (Hilton 1955). *P. ulei* is only found in Central and South America. SALB is responsible for the very small share of world natural rubber production from America (3.2%) (FAOSTAT,

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Accepted for publication 20 December 2017.

Fig. 1. South American leaf blight symptoms on a rubber tree **A**, on a leaf, **B**, on a stem.

<http://www.fao.org/faostat/en/#data>), because most of the large plantations have been destroyed by the disease.

Rubber cultivation extended firstly to Asia with trees obtained from seeds collected by Wickham in Pará State (Brazil) by the end of the 19th century (Dean 1987). Rubber trees from this area are genetically close to those from Mato Grosso (de Souza et al. 2015), which are poorly resistant to SALB (Le Guen et al. 2002). It is probably for this reason that all the clones planted in Asia and later in West Africa (called “Wickham clones”) are highly susceptible to SALB. All the plantations in South America using Wickham clones in the early 20th century were unsuccessful. Chee and Holliday (1986) and Hilton (1955) gave detailed consequences of the damages caused by this disease during the first half of the 20th century. Some other examples of destructions of plantations were reported by Rands (1924), Bodkin (1922), and Francis (1923) in Dutch Guiana; and by Bancroft (1913) in British Guiana. Altson (1923) estimated the yield decrease due to SALB at 93% and reported the giving up of a plantation by Goodyear in Panama five years after it has been set. Other examples were cited in Costa Rica (Blandin 1941) and in Colombia (Sorensen 1945). Dean (1987) reported the difficulties encountered by the Pirelli company on its plantation in Una (Bahia, Brazil) in the 1970s. However, the most representative example was the destruction of almost all the 3,200 ha of the Fordlandia plantation and of the 6,478 ha of the Belterra plantation created and then abandoned by the American industrialist Henry Ford from 1934 to 1943 (Braudeau 2003; Chee and Holliday 1986). Barré, former manager of the Michelin plantation in Bahia, estimated the loss of yield caused by SALB at between 1,300 and 1,400 kg/ha/an compared with the yield obtained in Nigeria with 600 to 700 kg/ha/an and 2,000 kg/ha/an, respectively (Lepetit 2006). However, there are no precise data concerning the loss of yield of trees less or more defoliated by SALB and, to have an idea of the consequences of SALB, we have to report to evaluations obtained on other leaf diseases of rubber trees. So, in conditions favorable to the disease, a tree can lose all its leaves and, at a plantation scale, the loss of foliage can exceed 20% (Rocha and Vasconcelos Filho 1978). During defoliation due to *Phytophthora* sp., Chee (1969) observed a defoliation reaching 75% and causing a 30 to 50% loss of latex yield. In Gabon, in large scale trials

Fig. 2. Defoliation of rubber trees caused by South American leaf blight.

for the control of *Colletotrichum* secondary leaf fall of rubber tree (SLF), Guyot et al. (2001) estimated the gain of yield obtained when fighting SLF with artificial defoliation at 450 to 500 kg/ha/an (including the stimulating effect on yield of the ethephon used for defoliation). In Malaysia, Azaldin and Rao (1974) evaluated the increase in yield obtained with the control of SLF by artificial defoliation at 33% when the RRIM (Radziah and Hashim 1990) estimated the increase at 30%. In Cameroon, Senechal (1986) evaluated the gain of yield between 48 and 115 kg/ha/an according the age of the trees on the clone GT 1 when treated by artificial defoliation against SLF. However, it is necessary to state that the most productive clones have been selected in Asia and Africa, and it is impossible to plant them in areas where SALB is present because of their extreme susceptibility. Consequently, there is a limit of yield due to the clones planted in areas where SALB is present.

The failure of rubber cultivation in tropical America is clearly linked to SALB (Langford 1945; Stevenson 1935) and Holliday stated that the ineffectiveness of disease control methods is one of the main factors stopping the growth of large-scale natural rubber production on the subcontinent (Krantz et al. 1981). Control with fungicides, difficult in plantations, has been developed only in nurseries. Control with escape using artificial defoliation has been tested, apparently with some success (Chee 1980a; Rogers and Peterson 1975), but it has never been developed on a large scale, probably because climatic conditions are too often favorable to the fungus. For this reason, SALB control relies mainly on genetic improvement in order to produce clones that combine reduced SALB susceptibility and high productivity in latex. Recent results in that direction are encouraging, but these results have to be confirmed in large scale experiments.

Using genetic tools on some populations sampled in four regions of South America (Brazil, Ecuador, Guatemala, and French Guiana), where the first reports of the fungus occurred in the early 20th century, the 1970s, the 1950s, and 1982, respectively, Barrès et al. (2012) proposed a scenario for the colonization of Latin America by *P. ulei*, which brings into play two independent founding events. The population of French Guiana would appear to be derived from the original population existing before the introduction of *Hevea brasiliensis* (around 30 years ago). The population present in French Guiana on planted rubber trees probably originates from the primary forest where *P. ulei* was found on wild rubber trees (Guyot, personal communication). Indeed, French Guiana is the only locality among the four localities studied by Barrès et al. (2012) where the rubber tree is native, so the chances that primary inoculum leading to formation of the French Guiana population originates from the Amazon forest are high (Barrès et al. 2012). The populations from the other three regions would appear to come from a series of independent and long-distance introductions from the same unidentified distant zone of origin. This dissemination may have been natural by long-distance dispersal events of ascospores or manmade by transporting infected planting material.

The Fungus

Morphology. The mycelium is segmented and its development is strictly intercellular (Blasquez and Owen 1963; Stahel 1917), mostly subcuticular at first to produce conidiophores. The mycelium then invades the palisade tissues and parenchyma. The hyphae only develop in young, strongly growing organs. They die at the same time as the diseased tissues (Stahel 1917). From the mycelium, which aggregates on the surface of the epidermis (Roger 1953), conidiophores appear on the underside of the lesions. Conidial sporulation mostly takes place on immature leaves (Fig. 3A and B), and unlike the asexual spores of *Colletotrichum* sp., the conidia of *P. ulei* do not need water for their dispersal and are easily dispersed by wind. The conidia (Fig. 3C) are usually bicellular (Stahel 1917). When the leaf lamina starts to harden, pycnidia appear in the form of small black dots. Pycnidiospores (Fig. 4) are present as soon as the leaf reaches full development (stage D, Hallé and Martin 1968). They then become gradually less abundant, but some can still be found more than 6 months after stage D. They are dumbbell shaped. The perfect form

of the fungus develops shortly afterward on leaves that are already mature (Fig. 3D). The perithecia form in groups, called stromata (Fig. 3E), mainly on the upper side of the leaves, but sometimes also on the underside (Chee et al. 1985). They cover the surface of the lesion if it is very small but remain around the edge when the lesion is large (Blasquez and Owen 1963). The perithecia contain asci whose swollen, apical end bursts when ascospores are released. The perithecia contain ascospores within 20 days after stage D (Guyot and Eveno 2015). The hyaline ascospores comprise two cells (Fig. 3F). The pycnidia and perithecia may live together on the same organ (Griffon and Maublanc 1913; Guyot and Eveno 2015). Stahel (1917) considered them to be two different structures. However, according to Stahel (1917), the two types of structures develop very close to each other and are often entangled with shared walls, but this observation has not been developed any more by other authors and the relationship between pycnidia and asci is not clear.

Taxonomy. In 1904, Hennings described two fungi, one bearing perithecia (*Dothidella ulei*), the other pycnidia (*Aposphaeria ulei*). Based on samples collected in Paramaribo (Suriname), Kuijper (1912) described a conidial form of the fungus called *Fusicladium macrosporum*. He also noted the existence of pycnidia produced by the same fungus. In 1912, some rubber tree leaves collected in nurseries near Belem (Pará, Brazil), on the estuary of the Amazon River, carried a fungus that Griffon and Maublanc (1913) considered to be identical to the fungus described as *D. ulei* by Hennings, but they did not find the pycnidia mentioned by Hennings for *A. ulei*. However, they did report the existence of a conidial form identical to that described by Kuijper. They classed it under the genus *Scolecotrichum*. Later, Vincens (1915) described that fungus under the name *Scolecotrichum heveae*. In 1913, in Suriname, Bancroft described a disease on cultivated rubber trees whose pathogen was called *Passalora heveae* by Masse (Stahel 1917). Cayla (1913) and Petch (1914) established a link between *D. ulei* P. Henn., *A. ulei* P. Henn., *F. macrosporum* Kuijper, and *P. heveae* Masse, and considered them to be different forms of the same fungus. Vincens (1915) observed the disease from leaf necroses caused by *F. macrosporum* up to some sporulating forms of *D. ulei* that occurred gradually. The same year, in Suriname, Stahel mentioned the simultaneous presence of perithecia, conidia (*F. macrosporum*), and pycnidia (*A. ulei*), and suggested calling these three forms by the single name of *Melanopsammopsis heveae* (Stahel 1917). As Stahel found that all the previously described forms were linked to the same species, he called it *Melanopsammopsis ulei* (Stahel 1917). In 1962, Müller and von Arx classed that fungus in the genus *Microcyclus* Saccardo due to the morphology of its stromata (Holliday 1970). Lastly, *Microcyclus ulei* (P. Henn.) v. Arx bore two names: *D. ulei* P. Henn. and *Melanopsammopsis ulei* (P. Henn.) Stahel. The pycnidial form was *A. ulei* P. Henn. and the conidial form was *F. macrosporum* Kuijper. The genus *Microcyclus* was placed in the family of the Mycosphaerellaceae (order of the Capnodiales) by Eriksson and Hawksworth (1993) in reference to the morphology of its stromata, then moved to the family of the Planistromellaceae (order of the Dothideales) (Barr 1996; Lumbsch and Huhndorf 2010). Despite some more recent work based on molecular approaches, the position of the genus *Microcyclus* and of *M. ulei* remains uncertain (Minnis et al. 2012). In fact, the three forms of spores of this fungus can be classed in different groups: Planistromellaceae for the sexual form, Venturiaceae for the conidial form, and Lophiostomataceae for the pycnidial form. The lack of DNA for each of these forms added to the confusion (da Hora Junior et al. 2014). Given that confusion, da Hora Junior et al. (2014) very recently used molecular tools on the three forms of the fungus to revise the taxonomic position of *M. ulei*. They classed these three forms in the family of the Mycosphaerellaceae s. str. (Capnodiales [Dothideomycetes]), and in the genus *Pseudocercospora*.

Epidemiology

Knowledge of the SALB epidemic process suffers from a lack of field observations, particularly those comparing all the variables involved in the course of an epidemic and the severity of attacks: the

environment, the host, and the parasite. Some claims, based more on comparison with other pathogens than on precise observations, have subsequently been refuted. To date, only eight publications have covered the epidemiology of SALB: Gasparotto linked climatic conditions and disease levels in Viana (Esperito Santo) (Gasparotto 1988) and in Ponte Nova (Minas Gerais) (Gasparotto et al. 1989), De Camargo et al. (1967) did likewise in the region of São Paulo, Rocha and Vasconcelos Filho (1978) worked in Itabuna (Bahia), Chee (1976c) in Trinidad, and Guyot et al. (2008, 2010, 2014) in French Guiana in an equatorial climate. However, the inoculum was not taken into account by most of these authors. Only Rocha and Vasconcelos Filho (1978), Chee (1976c), and Guyot et al. (2014) included the inoculum in their studies, but only Chee in Trinidad and Guyot in French Guiana took ascospores into consideration.

Incubation latency. Only young leaves are susceptible to infections. Susceptibility decreases with the age of the leaf. Mature leaves (stage D) can no longer be infected. The more leaves are infected at advanced stages, the smaller the diameter of the lesions. Junqueira

et al. (1990) also found that the infection stage had an effect on sporulation. Some precise data obtained under controlled conditions or in the field indicated incubation and latency periods of 5 and 7 days respectively, though they could vary depending on the strains (Junqueira et al. 1988, 1990) and the rubber tree clones (Guyot et al. 2008; Rivano 1992).

Asexual sporulation. Asexual or conidial sporulation takes place on young leaves, from the elongation stage (B-C) and into the first days of maturity (D). It lasts a maximum of 3 weeks (Guyot et al. 2008). Conidia can be released and dispersed by splashing but this may happen without rainfall or wetting, as they can be released and transported dry by the wind, which enables the fungus to spread in dry periods. The dissemination ability appears to be limited to a few dozen meters (Guyot et al. 2014; Rivano 1992). However, in strong vegetation periods (i.e., the rainy season), conidia are especially abundant (Guyot et al. 2014; Holliday 1969; Rocha and Vasconcelos Filho 1978). They are scarce in periods when the tree is producing few new leaves, especially in periods of low rainfall.

Fig. 3. Sporulation of *Pseudocercospora ulei*. **A, B**, Conidial sporulation; **C**, conidia (30 to 55×8 to $10 \mu\text{m}$); **D, E**, sexual sporulation; **F**, perithecium with asci discharging ascospores (10 to $20 \times 2.5 \mu\text{m}$). **B**: photo by C. Mattos, Michelin Company; **C**: photo by F. Pinard, Cirad.

They play a particularly large role in disease propagation in the rainy season, and over a short distance, but cannot guarantee the survival of the fungus if there is a complete break in the production of new leaves, notably if the unsuitable season (dry or cool) is long.

Sexual sporulation. Even recently, the sexual phase of the fungus was less well known than the asexual phase because it has never been

Fig. 4. Pycnidiospores of *Pseudocercospora ulei*. Size: $5 \times 9 \times 1 \times 2 \mu\text{m}$. (Kuijper 1912; Stahel 1917).

observed in in vitro cultures (Fig. 5). In all likelihood, it begins with the formation of pycnidia in which pycnidiospores are produced. The pycnidiospores germinate in 3 to 4 h but the germ tubes, which do not adhere to the cuticle, stop growing very quickly (Stahel 1917). da Hora Junior et al. (2014) did not achieve in vitro germination and all the authors agreed in saying that the pycnidiospores were incapable of infecting the plant. Pycnidiospores are considered as male gametes and they most probably act as an ascogonium fertilizer, as found for the pycnospores of *Mycosphaerella capsellae* (Inman et al. 1999), *Monilinia fruticola* (Agrios 2005), and *Pseudocercospora fijiensis* (Churchill 2011). Sexual sporulation does not take place on an artificial medium.

The maturation speed and viability of the perithecia have been the subject of numerous studies that gave results so variable that it was impossible to precisely describe (Chee 1976b; Holliday 1970; Langford 1945; Medeiros 1976; Stahel 1917). More recently, Guyot and Eveno (2015) established unambiguously that the perithecia contain ascospores from the second week following leaf hardening (start of stage D). Ascospore production is strong in the period ranging from 50 to 120 days following the start of stage D. However, the perithecia continue to produce ascospores beyond 200 days after the start of stage D. Some pycnidiospores are found up to 200 days after the start of stage D. Pycnidiospore and ascospore production occurs more or less early depending on the clones (Guyot and Eveno 2015; Guyot et al. 2008). Some perithecia containing viable ascospores can be found on 6-month-old leaves (Guyot and Eveno 2015). The fungus can therefore survive on organs in the unsuitable months and start a new epidemic when more suitable climatic conditions return. However, in a very wet climate, the disease may persist due to strong overnight wetting of the leaves. It is more the absence of susceptible organs in the dry season and old leaf fall that slow down the epidemic (Guyot et al. 2010). Ascospore release depends more on a high relative humidity and the existence of dew than on rainfall (Chee 1976c; Guyot et al. 2014). Chee (1976c) found a delay of around 30 min between perithecia wetting and ascospore release, while Guyot and Eveno (2015) observed a faster release and found that 80% of ascospores were released within 30 min after wetting. Ascospores are not released without leaf wetting even in a saturated atmosphere. On the other hand, just very slight wetting enables release. Guyot and Eveno (2015) found that for this to occur, wetting equivalent to a small amount of rainfall ($<0.5 \text{ mm}$) at low intensity was enough. It

Fig. 5. In vitro culture of *Pseudocercospora ulei* (photo by F. Doaré, Cirad).

has sometimes been indicated that cool temperatures ($<20^{\circ}\text{C}$) were necessary for ascospore release (Chee 1976b; Holliday 1970; Lantheaume 1989), but this information was refuted by Guyot et al. (2014). The two ascospore trapping studies undertaken to date showed them to be present throughout the year, in both Trinidad (Chee 1976c) and French Guiana in an Amazonian climate (Guyot et al. 2014). However, some ascospore trapping peaks were detected over the year. They occurred in the rainy season in Trinidad, and at the start of the rainy season in French Guiana. The role of ascospores is therefore to remain on the old foliage during the seasons unsuitable for rubber tree vegetation. The resumption of rainfall creates conditions that are suitable for the emergence of young, susceptible leaves and the massive release of ascospores, enabling a rapid resumption of the epidemic (Guyot et al. 2014). The dissemination ability of ascospores has been estimated to be at least several hundred meters (Guyot et al. 2014; Rivano 1992), but the conditions of those experiments did not enable a very precise estimation of that distance, which may be much greater. A second role of ascospores is probably to disperse the fungus over quite large distances, not only enabling the disease to spread, but also increasing the chances of the fungus finding a receptive substrate.

Dissemination. The dissemination of SALB is a fundamental issue in risk prevention. Yet, data on the subject are scarce and very insufficient for enabling an assessment of the risk if *P. ulei* is introduced into Asia or Africa. During observations in Costa Rica, Langford (1945) mentioned rapid dissemination of SALB up to 180 m in a diseased plot, with the disease decreasing with the distance from the inoculum source. Rivano (1992) studied spore dissemination using microscope slides smeared with Vaseline (70%) and paraffin (30%) positioned 20, 50, and 100 m from an infected plot. He did not find any conidia beyond 100 m and estimated that ascospores were disseminated further than conidia: 50 m away from the source, he still found 80% of the ascospores emitted in the focus and at 100 m, the quantity of ascospores trapped still amounted to 40% of the quantity emitted in the focus.

The survival time for spores is a question that has been greatly studied to assess the risks of disease spread in Asia. However, some highly variable results have been proposed, ranging from a viability of under a day (Stahel 1917) to more than 6 weeks (Chee 1976b) for conidia, with great variability depending on the humidity, temperature, and lighting (Chee 1976b; Holliday 1970). What is sure is that conidia are highly sensitive to ultraviolet wavelengths, but here again the data

Fig. 6. Theoretical biological cycle of *Pseudocercospora ulei* (da Hora Junior et al. 2014, under the Open Access license CC-BY, no changes were made). **A**, Asexual morph with conidiophores and conidia (bar = 35 μm) and conidiogenous cells with conidia at different stages of conidium formation. Pictures: Lesions with which the asexual form is associated (left), and close-up of leaf bearing typical lesions (right). **B**, Spermogonial morph with stroma, spermogonia (bar = 30 μm) and spermatia (bar = 7 μm). Pictures: Lesions with which the spermogonial morph is associated (left), and close-up of the same lesions (right). **C**, Sexual morph with stroma, pseudothecia, asci, and ascospores (bar = 60 μm). Pictures: Lesions with which the sexual morph is associated (left), and close-up of stomata (right). Dotted arrows indicate that both ascospores and conidia can infect young leaves.

are fairly variable, ranging from a few minutes to an hour (Brookson 1963; Chee 1976b; De Jonge 1962; Kai-Ming et al. 1986; Lebaijuri et al. 1997). In general, conidia are better conserved when they are in groups or kept on the necroses than when dispersed or isolated (Chee 1976b). Langford (1945) found that conidia deposited on leaves then dried for several days were able to infect the leaf after remoistening. Ascospore viability was only studied by Chee (1976b) who made the same findings as for conidia, but with a shorter survival time (21 days at the most). Extreme temperatures and excess moisture are, as for conidia, detrimental to ascospore survival.

The biological cycle of *P. ulei* was diagrammatically represented by da Hora Junior et al. (2014) (Fig. 6).

Influence of climatic factors. Epidemiological observations in the field are scarce. Although several authors have suggested the negative role of heavy rain on the severity of epidemics (Chee 1976b, 1980; Holliday 1969; Stahl 1917), it has been shown that the amount of rainfall does not play a major role in the disease (De Camargo et al. 1967; Gasparotto 1988; Gasparotto et al. 1989; Guyot et al. 2010). The duration and repetition of periods of very high relative humidity (>90%) and leaf wetting are the climatic factors that most affect the severity of epidemics (Gasparotto 1988; Gasparotto et al. 1989; Guyot et al. 2010). Amazonian conditions meet the requirements of *P. ulei* throughout the year, even though damage in the dry season is less severe than in the rainy season (Gasparotto et al. 1991; Guyot et al. 2010).

Fungus diversity. The diversity of the fungus has long been characterized by observing the pathogenicity of isolates in relation to ranges of rubber tree clones displaying variable resistance levels. In a review in 1970, Holliday considered the existence of four physiological races of *P. ulei* based on the work by Langford (1945, 1946), Langdon (1963, 1965), and Miller (1966). Then, in a second review, Chee and Holliday (1986) found that the diversity of *P. ulei* was much greater and proposed nine physiological races of different geographical origins, primarily based on their virulence in relation to *Hevea benthamiana* and some populations of *Hevea brasiliensis* from the Upper Amazon. Zhang and Chee (1986) found differential susceptibility between races to benomyl fungicide. Many studies undertaken in the 1980s and 90s showed the very great diversity of the fungus: Hashim and De Almeida (1987), Junqueira et al. (1986, 1987, 1988, 1989), and Rivano (1997). Comparisons between these studies are difficult, sometimes giving contradictory results, possibly due in large part to methodological differences: different ranges of differential clones, inoculations on leaf discs in some cases, on leaves in situ in other cases, and different assessment criteria. In the 1990s, CIRAD and the Michelin tire company, who have been partners since 1993 in a research project on SALB, established a range of differential clones and some methods for inoculating and assessing the virulence of *P. ulei*. The differential range comprised 12 clones (Mattos et al. 2003): FX 2784, FX 3899, IAN 3087, IAN 717, FX 2804, and IAN 6158, which are hybrids between *H. benthamiana* and *H. brasiliensis*, PA 31 (primary clone of *H. pauciflora*), MDF 180 (primary clone of *H. brasiliensis*), from the Madre de Dios region in the Peruvian Amazon, and FX 3864, FX 985, FX 2261, and FX 4098, which are derived from crosses between various clones of *H. brasiliensis*. Lieberei (2007) concluded from some work on the physiological diversity of *P. ulei* that there existed 68 races of the fungus displaying different reaction spectra.

Le Guen et al. (2004) used molecular tools based on microsatellite markers to characterize the diversity of the fungus. Eleven polymorphic markers were identified using 11 isolates from Brazil (Mato Grosso, Bahia, and Amazonas), French Guiana, and Guatemala. Another study on the genetic diversity of the fungus using 17 microsatellite markers was conducted by da Hora Junior (2012) on 15 Brazilian populations originating from rubber tree cultivation areas spreading from East Amazonia to coastal regions. This study indicated that the genetic diversity of the fungus in Brazil has probably been influenced by human activity and also results from a genetic drift caused by a disconnection between Amazonian populations and the populations of the coastal regions.

Influence of the tree phenology in the epidemiology of the disease.

Only the genus *Hevea* is known as a host for *P. ulei*. Four of the 10 species in that genus are susceptible to *P. ulei*. Leaves are only susceptible in their immature phase (15 to 21 days), but the susceptibility decreases after stage B2 (Hallé and Martin 1968). Thus, the period of maximum leaf susceptibility lasts 10 to 12 days (Guyot et al. 2008). Once leaves are mature (stage D), they are no longer susceptible to SALB. When observing three clones with different susceptibility, Guyot et al. (2008) did not find any difference in leaf development rate that might have been responsible for the different levels of susceptibility.

In the first 5 to 6 years, rubber trees grow by a succession of leaf flushes throughout the year. The time taken for a leaf flush to develop from bud break to full maturity is around 1 month, and there is one flush every 1.5 to 2 months depending on the environmental conditions. Older trees reach phenological maturity with an annual defoliation-refoliation cycle: old leaves fall and are replaced by new leaves at a precise time of year. The defoliation-refoliation scheme (earliness, uniformity, speed) varies considerably depending on the clone (Guyot et al. 2008). The phenology of the rubber tree therefore has major consequences for the impact of SALB. Clonal differences in phenology inside a plantation may make it possible to escape the disease or may lead to severe damage. The existence of neighboring plots with different clones, each with their own defoliation-refoliation scheme, may be a factor involved in inoculum conservation and dissemination between plots (Furtado 1990; Rivano 1992).

Search for Resistant Varieties

SALB control naturally turned to clonal selection with a view to producing planting material that is less susceptible to SALB with high yields of good quality rubber. Starting in 1935 at the Henry Ford plantations, where sorting of some tolerant material of Brazilian origin and of the first crosses had been undertaken, selection work was then taken up by the Instituto Agronomico do Norte (IAN) in collaboration with USDA (United States Department of Agriculture). The first attempts to create new *Hevea* varieties that were both SALB-resistant and high-yielding were made by the agronomists at the Ford plantations and date back to 1943 (Townsend 1960). There was a string of several other attempts in the following decades (IAN, Firestone), still based on an empirical approach. However, the first true hybridization and SALB-resistant clone selection program (Bos and McIndoe 1965) was conducted in 1949 by the Firestone Plantations Company at the Harbel plantation in Liberia, in Guatemala, and in the state of Bahia in Brazil. The initial tolerant material was made up of the least susceptible clones selected at the Ford plantations then at IAN, clones introduced from the Madre de Dios region in Peru, and the species *Hevea pauciflora* displaying a high level of resistance.

It was not until the 1970s that the first studies were published developing standardized methodologies for the resistance screening of *Hevea* genotypes, with systematic trials of the new clones being developed. Some teams then endeavored to describe the biochemical and histological aspects of SALB resistance in the rubber tree, while others sought to localize resistance factors on the genetic map of resistant genotypes. The most recent work has focused on how the genes involved in SALB resistance are expressed, whether it be genes directly involved in the metabolic pathways, or expression factors.

The first work by Chee (1976a) focused on describing reliable and repeatable ways of estimating the susceptibility of *Hevea* clones. That work was resumed and fine-tuned by Junqueira et al. (1988) then by Rivano (1992), leading to the first characterizations of clonal resistance. Such resistance can take on various forms: slightly later, less abundant, or more staggered sexual and asexual sporulation (Guyot et al. 2008; Guyot and Eveno 2015).

The clones derived from 30 years of clonal selection in the Firestone plantations underwent only limited development (Le Guen, personal communication). Those clones, completed by some planting material derived from surveys carried out in 1981 in the Brazilian states of Acre, Rondônia, and Mato Grosso, were then used again in

the selection program undertaken by the Michelin Company in its plantations in Brazil in collaboration with the Centre de Coopération Internationale en Recherche Agronomique pour le Développement (CIRAD) (Garcia et al. 2004; Le Guen et al. 2002). Previously, starting with some clones derived from the Ford selections and some wild planting material, Brazilian public research embarked, for its part, on a genetic improvement program seeking resistance to *P. ulei* (Darmono and Chee 1985; Junqueira et al. 1988). Thus, for many years, knowledge of SALB resistance mechanisms in *Hevea* (Garcia et al. 1999; Sanier et al. 1992) and clonal selection with a view to combining latex production and resistance have been the target in SALB research programs (Chee 1976a; de Araujo et al. 2001; Rivano 1997). It is necessary to highlight an essential difference between the Ford breeding program initiated in the 1930s (continued by IAN, Table 1) and the program developed by Firestone in the 1950s and later amplified by Michelin and Cirad (CMB project). While the first one was based on the use of resistances found in wild trees in Pará and Upper Amazonia, of which several belonged to the species *H. benthamiana*, Firestone and later CMB have used the resistances of trees from the Madre de Dios region in Peru. The rubber trees originating from this region are genetically close to those of Acre in Brazil (Le Guen et al. 2009), where a high percentage of rubber trees were found resistant to SALB (Le Guen et al. 2002). The very different origin of the resistance sources used by Firestone and then by CMB probably explains that the resistance of clones selected by the CMB breeding project exhibit a more durable SALB resistance than the clones and derivative clones selected by Ford. Some current clones (FDR 5788, CDC 312, MDX 608, PMB 1) combine a good level of SALB resistance, good growth, and high rubber productivity, and can be recommended in the SALB area of southeastern Bahia (Cardoso et al. 2014). FDR 5788, CDC 312, and PMB 1 are free of sexual sporulation as MDF 180, while only a few stromata are found on MDX 608. Some other clones (CDC 429, SIAL 893, TP 875, FDR 4461) exhibit a good level of resistance either characterized by moderate asexual or sexual sporulation, but do not have a sufficient yield for large scale recommendation (Cardoso et al. 2014). These clones were developed by the program for support of small-holder rubber plantations near the Michelin rubber plantation in Bahia. In 2011, 866 ha of FDR 5788 (64%), 262 ha of CDC 312 (19%), and 98 ha of PMB (7%) were planted (Garcia, personal communication). To evaluate them on a large scale and under diverse environmental conditions, the most promising clones for resistance to SALB and latex production obtained by the Cirad-Michelin selection project have been introduced into Colombia, Guatemala, Ghana, and Cambodia, and into the Cirad rubber tree collection in French Guiana. However, combining good SALB resistance traits and high yield traits could not be fully achieved, particularly as some strains of *P. ulei* gradually appeared that were capable of overcoming a fair number of resistances, as it happened for the clone FX 3864 in the Michelin plantation of Bahia (not documented). Currently, along

with assessments of clonal resistance in field trials and under controlled conditions, research is being carried out to identify QTLs of resistance to *P. ulei* with a view to developing early marker-assisted selection techniques (Le Guen et al. 2003; Lespinasse 1999; Lespinasse et al. 2000a).

Biochemical Resistance

The first mention of a substance produced by the rubber tree in reaction to infection by a fungal pathogen, *Colletotrichum gloeosporioides*, was in work by Tan and Low (1975). The substance revealed a blue color under UV light (365 nm). Taking their lead from that study, Giesemann et al. (1986) showed that some *Hevea* leaves that were resistant to *P. ulei* produced a blue fluorescent substance (bfs) identical to that found by Tan and Low (1975), while leaves of susceptible cultivars did not. The production of that bfs could also be induced by spores of fungi that were not pathogenic on *Hevea*. They identified the substance as scopoletin, a coumarin whose accumulation in tissues near the hyphae of the pathogen likened it to a phytoalexin. The rapid and durable accumulation of scopoletin was associated with total resistance or to a high degree of resistance to *P. ulei* (Garcia et al. 1995b). The level of partial resistance was positively correlated to the scopoletin concentration up to 48 h after infection. The fungitoxic effect of that molecule was also shown in vitro, affecting both conidium germination and elongation of the germ tubes. Infections with other parasites (*C. gloeosporioides*, *Corynespora cassiicola*) showed that it was necessary to double the concentration of scopoletin to achieve the same effect on germination and elongation. Garcia et al. (1995a) confirmed that the degree of *Hevea* cultivar resistance was directly linked to the scopoletin accumulation rate. Its extracellular location suggested a phytoalexin-like action that blocked conidium germination and penetration by hyphae. The same study also revealed later lignin accumulation, which seemed to play a major role in keeping the fungus around the edge of the necrotic zone in the case of cultivars with partial resistance. Systematic tests carried out under controlled conditions to inoculate 36 *Hevea* cultivars with *P. ulei* (Garcia et al. 1999) revealed a strong correlation between scopoletin and the level of resistance, along with a frequent association between lignin accumulation and the duration of stroma generation. However, although the absence of scopoletin and lignin was often associated with severe symptoms, their presence alone could not account for all the resistances observed.

At the same time, the inhibiting action of cyanogenesis was explored on the defense reactions of the rubber tree. *Hevea* releases hydrocyanic acid (HCN) when subjected to stress or a wound, causing cellular decompartmentalization, which brings linamarin (a precursor of HCN) and linamarase (Lieberei 1986) into contact. It was found that the release of HCN can notably occur after infection by a pathogen and especially *P. ulei*. The quantity of HCN released after infection by *P. ulei* was high in the leaves of susceptible plants and low in the leaves of resistant plants (Lieberei 1988). This difference

Table 1. Origin of the clones used in the different breeding programs

Research organism	Abbreviation	Meaning	State (Country)	Date	Type of material
Ford plantations	F	Ford	Pará (Brazil)	1930s	Primary clones
	FX	Ford cross	Pará (Brazil)	1940s	F × Wickham
Peruvian Ministry of Agriculture	MDF	Madre de Dios Firestone	Madre de Dios (Peru)	1948	Primary clones
Firestone plantations	CD	Clavellina Disease	(Guatemala)	1950s	MDF × Wickham
	CDC	Clavellina Disease Clone	(Guatemala)	1950s	MDF × Wickham
	MDX	Madre de Dios cross	(Guatemala)	1950s	MDF × Wickham
	FDR	Firestone Dothidella Resistant	(Liberia)	1950s	MDF × Wickham
	TP	Três Pancadas	Bahia (Brazil)	1980s	Primary clones
Brazilian public research centers	IAN	Instituto Agrônômico do Norte	Pará (Brazil)	1950s	Ford × Wickham
	SIAL	Seleção do Instituto Agrônômico do Leste	Bahia (Brazil)	1980s	Primary clones
Michelin plantations and CIRAD	PMB	Plantações Michelin da Bahia	Bahia (Brazil)	1990s	Primary clones
	CMB	Cirad Michelin Brazil	Mato Grosso and Bahia (Brazil)	1990s to 2000s	Firestone × Wickham

in HCN release after infection by *P. ulei* was not due to pre-existing differences in the content of cyanogenic precursors. Lieberei suggested that the difference might be explained by differences in the activity of detoxification enzymes that metabolize HCN (such as rhodanese or β -cyanoalanine synthase) or by differences in the histological distribution of cyanogenic precursors. The presence of HCN was found to reduce resistance reactions in *Hevea*. In addition, Lieberei et al. (1983) showed that when applied in moderate concentrations at early stages of pathogen development (germinated spores, young mycelium), HCN had a positive effect on *P. ulei* growth in vitro.

Later work by Lieberei et al. (1989) sought to reveal the effect of cyanogenesis on the defense reaction after inoculation with *P. ulei*. To that end, some isolates of *P. ulei* classed in two distinct virulence groups were inoculated on some cultivars reacting in different ways to the two groups, and on some cultivars susceptible to both groups. Following infection, the resistant cultivars released small quantities of HCN, and high concentrations of scopoletin were observed near the fungus penetration points. Conversely, the susceptible cultivars released large quantities of HCN and displayed a low accumulation of scopoletin. It would appear that the accumulation of HCN inhibited scopoletin production, hence the resistance of the cultivars. This high HCN production after an infection in these cultivars was a consequence of a high cyanogenic potential not compensated for by sufficient activity of HCN metabolism (or detoxification) enzymes. Thus, HCN production by the plant beyond a certain threshold would seem to inhibit its own defense mechanisms against pathogens. One of the resistance factors of a cultivar would therefore seem to be the outcome of an appropriate combination of the rate of HCN emission and HCN detoxification by the plant.

Lastly, some more recent work (Lieberei 2006) focused on characterizing the behavior of *P. ulei* in the presence of HCN. It turned out that the fungus has a β -glucosidase with a stronger affinity for *Hevea* cyanogenic precursors than that of the corresponding enzyme of the plant itself. The parasite would therefore seem able to develop contents sufficient to hinder the plant's defense mechanisms, notably scopoletin production, in the presence of HCN.

However, this interpretation hardly accounts for the differences in resistance observed between cultivars depending on the pathogen virulence groups. While cyanogenic potential plays a role in defense mechanisms in general, it cannot alone explain the difference observed between cultivars for their resistance to different pathogenic fungi, and different pathotypes of the same species. It is at least necessary to envisage the upstream action of processes modulating cyanogenic potential, with those processes themselves being triggered by specific plant-pathogen interactions. Neither does this hypothesis exclude other defense processes also triggered by plant-pathogen reactions. In addition, some later processes come into play, such as lignin accumulation in totally or partially resistant clones, around 96 h after penetration, which limits fungus propagation in the leaves (Garcia et al. 1995b).

Recent advances in gene expression analysis have enabled new insights into the mechanisms involved in SALB resistance. A transcriptomic analysis conducted by Fang et al. (2016) highlighted differences in gene expression involved in cyanogenesis as well as in lignin and anthocyanin synthesis in the leaves at different stages of their development. These differences are correlated, according to the authors, with the modifications in the resistance to several leaf diseases including SALB.

Another study of Koop et al. (2016) aimed at characterizing the genes differentially expressed by three rubber tree clones, a totally resistant clone, a partially resistant clone, and a susceptible clone when inoculated by *P. ulei*. The study dealt more specifically with the genes encoding for enzymes involved in the redox system and it revealed that the differences in the levels of resistance are linked to the capacity of these clones to maintain an equilibrium between the production and the scavenging of the reactive oxygen species (ROS). After an infection, if this equilibrium is broken, there is an accumulation of H_2O_2 in the cells, causing the programmed death of the cell (apoptosis).

Genetics of the Resistance

In the mid-1990s, studies started being developed that took advantage of progress in the molecular marking of plant genomes. Generalized use of DNA markers led to decisive progress for many cultivated species in fields as diverse as variety identification, checking the legitimacy of crosses, population genetics, and identifying loci linked to quantitative traits (QTL) or to major genes. They particularly make it possible to create tools that improved the efficiency of the varietal breeding strategies, whether it be by selecting genotypes with alleles favorable to the markers of QTLs of interest, steering generations of back-crosses or RILs (recombinant inbred lines), setting in place marker-assisted recurrent selection schemes, or by genomic selection that took into account the genetic information provided by the whole genome for the study population. These different approaches can be referred to with the generic term marker assisted selection (MAS).

The geneticists and breeders working on *Hevea* quite quickly understood that resistance to *P. ulei* was a trait for which it ought to be quite easy, thanks to molecular markers, to identify some resistance factors and thereby set up MAS schemes based on biparental populations obtained by carrying out crosses between resistant and susceptible individuals (Lespinnasse et al. 1997). The first DNA markers developed in *Hevea* were nuclear RFLPs associated with microsatellite probes (Besse et al. 1993). However, the heterozygous nature of the *Hevea* genome and the impossibility of direct access to the parental haplotypes were obstacles to establishing genetic maps until the calculations of recombination frequencies between genetic markers in this particular case were defined (Ritter et al. 1990) and a pseudo-testcross type efficient strategy for mapping based on populations was established (Grattapaglia and Sederoff 1994). The first saturated genetic linkage map for *Hevea* (Lespinnasse et al. 2000b) comprised DNA markers of the RFLP, AFLP, and microsatellite type, along with a few protein markers (isozymes). That map, obtained from the cross between an individual of the species *H. brasiliensis* susceptible to *P. ulei* (clone PB260 created in Malaysia) and a resistant individual derived from the interspecific hybridization of *H. brasiliensis* $\times$ *H. benthamiana* (clone Fx3899 created in Brazil), comprised 717 marker loci and was dense enough to localize any segregating genetic factors among the progenies of the cross.

The individuals of that progeny that established the genetic map were therefore also inoculated under controlled conditions with five strains of *P. ulei* exhibiting different virulence levels. They were individually scored according to two parameters able to account for the level of resistance (Le Guen et al. 2008): the lesion diameter and the type of reaction on a scale of 1 to 5. Depending on the aggressiveness of the inoculated strains, the number of resistance QTLs identified varied from 1 to 5, be it for the lesion diameter or for the type of reaction. Two of those QTLs could have a major effect in resistance expression, explaining from 18 to 34% of the phenotypic variance of the trait. In particular, the QTL located on the g13 linkage group was identified with each of the five strains inoculated and for the two traits observed. All the QTLs detected came from the Fx3899 parent derived from the interspecific hybridization, and more particularly from the *H. benthamiana* grandparent. The other major information provided by that study was that for the four strains against which the Fx3899 parent displayed total resistance, three to five resistance QTLs were detected, while the partial resistance of Fx3899 to strain G77 only relied on the sole detected presence of the major QTL of the g13 linkage group. Thus, unlike the interaction models usually described, it was a matter here of polygenic total resistance and monogenic partial resistance.

The same progeny was then assessed for its resistance to *P. ulei* under natural infestation conditions in a field trial planted in French Guiana (Le Guen et al. 2003). In such an experimental design, the inoculum coming into contact with the test plants was not controlled, which can complicate interpretation. On the other hand, it made it possible to observe some perithecia containing the asci and ascospores, and hence detect any resistance QTLs effective against the sexual phase of the fungus cycle. Symptoms

were observed 20 times over a period of 18 months. The major QTL previously identified on the g13 linkage group was found once again in this study, effective both for the type of reaction and for the sexual phase (existence of stromata) and able to explain up to 91% of the phenotypic variance of the latter trait. With such values, it is reasonable to wonder whether that QTL might not behave like a major resistance gene whose highly contrasting expression when observing the sexual phase of the fungus might be assimilated to Mendelian segregation. In fact, 1:1 segregation was clearly seen for the “presence/absence of stromata” trait

within this progeny in the field. Four other minor QTLs of resistance to *P. ulei* were also discovered in this study.

Lastly, another experiment under controlled conditions on the same progeny was conducted in French Guiana, this time with three strains of *P. ulei* against which the Fx3899 interspecific parent only displayed low resistance or none at all (Le Guen et al. 2007). In all, eight efficient QTLs were detected for partial resistance to one of the strains, including the major QTL of the g13 linkage group. Only one of those QTLs (on group g12) was found for partial resistance to the second strain, and no efficient QTL was detected for the strain for

Fig. 7. Summary of the QTLs and resistance genes identified in four *Hevea* progenies (Le Guen, unpublished).

which the Fx3899 parent did not display any resistance. Thus, although the resistance of this Fx3899 genotype was polygenic, with up to eight QTLs detected for resistance to some strains, all these resistance loci could be overcome by the same isolate. In such a case, the genotype therefore becomes highly susceptible to an isolate of this type.

The same resistance QTL analysis approach was applied to some other genotypes that seemed to be of potential interest for creating cultivars that are both productive and resistant. These included the primary clone MDF180 originating from a natural population located in the Madre de Dios region of Peru. This clone displayed a unique particularity: despite being a well-known clone for which numerous resistance observations had been carried out both in plantations located in regions infected by *P. ulei* and under conditions of controlled inoculation with the fungus, the sexual form of the fungus (teleomorph) was never found on its leaves or any other tissues (Le Guen et al. 2008). It was also seen that, in general, the development of the asexual phase (anamorph) was reduced on this clone, whatever the strain inoculated. This clone did not display a latex production level that would make it attractive to growers, but it was a very interesting candidate as a parent for selection populations within which one would hope to identify some resistant and productive genotypes. It was therefore crossed with the susceptible Asian clone PB260 to create a biparental population of 351 genotypes, of which 298 were assessed in the field in French Guiana under natural *P. ulei* infestation conditions. Of those 298 genotypes, 195 were also assessed under controlled inoculation conditions with two different strains of the pathogen (Le Guen et al. 2011). The genetic linkage map established by genotyping the 351 individuals of the population with some microsatellite and AFLP markers revealed six loci with a significant effect on resistance to *P. ulei*. Of those six loci, four corresponded to QTLs with a limited effect. One QTL with a strong effect was identified on the g13 linkage group and was effective against the Brazilian strains but not against the Guianan strains, whether under controlled inoculation conditions or with natural infestation. Lastly, one of these loci located on the g15 linkage group corresponded to a major resistance gene, effective against the three strains tested under controlled conditions, and against the natural Guianan strain. It can be said that this locus corresponded to a major gene because the LOD levels were very high (>80 for some traits) as was the percentage of observed phenotypic variance explained (>90% for some traits). In addition, when individuals were assigned to a “susceptible” or “resistant” phenotypic class based on the existence or lack of stromata, this qualitative trait then behaved like a Mendelian marker localized exactly at the peak of the QTL in g15.

Another genotype attracted interest for the detection of resistance QTLs: Fx2784. This clone had the particularity of being totally resistant in many situations, be it in the field or when inoculated under controlled conditions with some known strains of *P. ulei*. However, that resistance could be overcome by a small number of strains and Fx2784 then displayed very strong susceptibility, as can be found in the state of Bahia, Brazil (Mattos et al. 2003). This situation suggests monogenic resistance. Consequently, this clone was also crossed with the susceptible Asian clone PB260 in order to create a segregating biparental population for this trait. The population was planted out in French Guiana, and in the state of Mato Grosso in Brazil, two places where the resistance of Fx2784 has yet to be overcome by the pathogen (Le Guen et al. 2013). Segregation (1:1) of the resistance trait was observed in both places, strengthening our initial hypothesis. The targeted genotyping of the individuals of this progeny following a bulk segregant analysis (BSA) strategy revealed a major resistance gene located on the g2 linkage group.

Thus, the various studies of QTLs of resistance to *P. ulei* undertaken to date have revealed numerous QTLs with varied effects, along with some major resistance genes (Fig. 7). This is very important information for steering ongoing selection schemes as it enables early genotype screening within progenies involving resistant parents for which attempts are being made to improve rubber yield characteristics.

Conclusion

Slightly more than a century after the large-scale development of rubber growing, SALB is managed in Latin America by growing rubber in areas where SALB occurrence is less severe, though with new constraints as the rubber trees are no longer under optimum growing conditions. Consequently, selection work has been geared toward clones adapted to these so-called escape zones. The selection of both disease-resistant and high-yielding clones has yet to be fully achieved, mostly because of the considerable adaptability of the fungus. At present, several researchers are working on the definition of the escape areas for planting rubber trees, but few articles have been published on this topic (Cecilio et al. 2006; Jaimes et al. 2016).

Among the clones shortlisted as part of the CMB project (joint work by CIRAD and the Michelin tire company), many display resistance and yield characteristics that suggest it will be possible in the medium term to disseminate some good cultivars adapted to the regions infested by SALB.

The ways in which *Hevea* resists the fungus and the epidemiology of SALB are now fairly well known, apart from the dissemination conditions, which have remained virtually unstudied. Despite that, they remain a major element in the risk assessment for regions that are still free of the disease, namely Africa and especially Asia, which supplies around 90% of the world's natural rubber production. On those two continents, rubber growing relies on clones that are highly susceptible to SALB as they were selected without the disease pressure.

Despite the risk, the fact that SALB has yet to appear on those continents has led to a reduction in research on the subject, which is only considered occasionally, in very fundamental aspects. However, if the most suitable clones and complementary control methods are to be chosen, it would be wise to make a more in-depth examination of escape conditions (phenological, climatic). It would thus also be possible to more effectively assess the risks of SALB being introduced into Africa and Asia, and the most exposed regions.

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Jean Guyot

Jean Guyot began his career as an entomologist and an agronomist, first on Reunion Island (stem borer on sugarcane, *Pseudaulacaspis pentagona* on peach tree), later in the French West Indies (*Thrips palmi* on eggplant), and then in Cameroon. He entered Cirad in 1991 to develop a program of experiments to improve the control of rubber tree diseases in large plantations in Gabon, especially root diseases (*Rigidoporus lignosus* and *Armillaria* spp.) and secondary leaf fall (SLF) due to *Colletotrichum* spp. He developed artificial defoliation to control SLF at a large scale in Gabon. Then he worked in the Ivory Coast in smallholders plantations and was sent to French Guiana in 2000 for research work on South American leaf blight of rubber. Now, he is responsible for developing cultural practices in French Guiana to reduce the use of chemicals and fertilizers in several crops (pineapple, citrus, eggplant, tomato), including agroforestry and covering plants.

Vincent Le Guen

Vincent Le Guen is a senior researcher at Cirad who specializes in genetics and crop breeding. Since the beginning of his career, he focused on the genetics of tropical tree-crops. He has spent several years in Brazil and French Guiana, where he worked on the genetics of rubber tree resistance to the fungus *Pseudocercospora ulei*, responsible for South American leaf blight (SALB). These studies continued at a laboratory scale when he moved to Montpellier, France. He used molecular markers to complete field observations or inoculations under controlled conditions made in South America. This work enabled the identification of genetic factors linked to SALB resistance in different resistant genotypes. These studies have also helped to understand the distribution of SALB resistance among wild and domesticated populations of rubber tree. They provided a rational explanation to the question: why are all widespread performing rubber tree cultivars highly susceptible to SALB? Along with his colleagues in Cirad, he is involved in similar research on resistance to the fungus *Corynespora cassiicola*, a severe pathogen of rubber tree in Asia and Africa.

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