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Root-rot diseases of the rubber tree in the Ivory Coast. 1. Severity, dynamics, and characterization of epidemics

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An epidemiological survey was carried out in a rubber tree (*Hevea brasiliensis* Willd. ex A. Juss) plantation to study and describe the epidemics of the root-rotting fungi *Rigidoporus lignosus* (Klotzsch) Imazeki and *Phellinus noxius* (Corner) G. Cunn. Infection and mortality of the trees were systematically recorded half-yearly for 6 years. During the first year, the outbreak resulted in a considerable enlargement of the initial disease centers and the appearance of new ones. Pathogen activity was found to decrease 6 years after planting. This was attributed to the increase in defense reactions with the age of the tree and to disruption of trophic connections between forest inocula and decaying taproots of rubber trees. The type of root disease is discussed, mainly in terms of the kinetics of the creation of disease centers.

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Une enquête épidémiologique a été menée pendant 6 ans dans une jeune plantation d'hévéa (*Hevea brasiliensis* Willd. ex A. Juss) afin d'étudier et de prévoir le développement des champignons agents de pourridié, *Rigidoporus lignosus* (Klotzsch) Imazeki et *Phellinus noxius* (Corner) G. Cunn. Pendant cette période, la contamination et la mortalité de ces arbres ont été systématiquement prises en compte tous les 6 mois. Durant la première année, on a assisté à une explosion de la maladie se traduisant par l'accroissement des foyers initiaux et par l'apparition de nouveaux. À compter de la sixième année après la plantation, on a observé un net fléchissement des activités parasitaires possiblement lié à l'augmentation des capacités réactionnelles des arbres avec l'âge ainsi qu'à la rupture des liens trophiques entre l'inoculum et les racines des arbres en état de décomposition. Le type de maladie pouvant caractériser ces pourridiés est discuté, essentiellement, sur la base des cinétiques d'apparition des foyers parasitaires.

Introduction

As pointed out by Kranz (1980), some authors consider epidemiology to be the study of the effects of ecological factors on a disease; for some it concerns the dynamics of pathogens in the host population, while others think that the discipline deals only with disease spread and development. In fact, epidemiology addresses all these aspects and takes into account, at both the population and individual levels, the effects of interactions between pathogen, environment, and man.

The present epidemiological investigation was carried out following these principles. This work is part of an extensive multidisciplinary research project (biochemistry, biology, cytology, etc.) devoted to the study of root-rot diseases of the rubber tree, *Hevea brasiliensis* (Willd. ex A. Juss), in the Ivory Coast (e.g., Geiger et al. 1985; Nandris et al. 1984, 1987*a*, 1987*b*; Nicole et al. 1986). Rubber tree losses due to the two main root-rotting fungi, *Rigidoporus lignosus* (Klotzsch) Imazeki and *Phellinus noxius* (Corner) G. Cunn., have reached more than 50% in old plantations. Damage results in large patches of dead trees.

The aim of this investigation is to describe, from data collected during a 6-year survey, the dynamics of these root diseases in a young plantation established on a site freshly cleared of the indigenous primary forest. It is intended that such information be used to forecast the development of the disease elsewhere in this country and to assess and monitor the efficacy of control methods in rubber tree plantations.

The part of the study presented here deals first with the severity of root disease and its dynamics, secondly, it attempts to classify the type of disease recorded, and finally it deals with disease center enlargement. Detailed results concerning the infection processes, spatial spread of pathogens, tree mortality, and disease modeling will be published later.

Materials and methods

The host

The rubber tree can grow to a height of over 20 m. When fully developed, the root system is characterized by a verticil of lateral roots in the top 30 cm of soil and a taproot that acts as a food reservoir as well as an anchor (Otoul 1960). The mean lengths of the taproot and the lateral roots are 35 and 10 cm at 1 month, 3.8 and 6.5 m at 6 years, and 5 and 20 m at 24 years, respectively. The monitored rubber trees (clone GT1) were planted in 1973 on a contour-line pattern with 2 × 8 m spacing.

The pathogens

Rubber tree attack by root-rotting fungi leads to root system decay, often followed by death. After root colonization the fungi can live as perthophytes. Pathogen spread is essentially due to hyphae developing through soil and along roots, and only rarely via spores.

Rigidoporus lignosus, *white root rot*

In the Ivory Coast, this fungus (formerly called *Fomes lignosus* (Klotzsch) Bres. or *Leptoporus lignosus* (Klotzsch) Heim ex Pat.) is particularly polyphagous, but causes the highest incidence of disease in rubber tree plantations (Nandris et al. 1984). The pathogen forms numerous white, somewhat flattened rhizomorphs (1–2 mm thick) which grow on and adhere strongly to the surface of root bark. These rhizomorphs grow rapidly (21 cm per month) and may extend several metres through the soil in the absence of any woody substrate. Thus, healthy rubber trees can be infected by free rhizomorphs growing from old forest stumps or woody

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TABLE 1. Incidence (% infected trees) of root diseases in the 20 plots, recorded at the beginning of the survey (inspection 0)

	Plot no.																			
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
Rigidoporus lignosus	2.6	3.8	6.0	4.0	0.5	0.1	1.1	4.9	5.1	2.7	4.4	2.8	5.7	0.2	2.3	7.4	1.0	8.8	3.4	0.3
Phellinus noxius	0.6	0.9	1.3	2.6	0.7	0.0	0.0	1.7	1.0	0.0	2.2	0.2	0.0	0.7	0.7	2.1	0.5	0.5	0.9	0.3

debris in the ground, as well as by root contact. Internal progression of the fungus through root tissues lags well behind the front of epiphytic growth on infected roots.

Phellinus noxius, *brown root rot*

This fungus lacks rhizomorphs and forms a dark-brown mycelial sheath around roots. This slowly developing (6 cm per month) mucilaginous sleeve becomes encrusted with stand and gravel. Tree infection only occurs by direct contact between a root and the mycelium. Mycelial spread on the root surface precedes internal decay by only a few centimetres (Hodges and Tenerio 1984).

Detection methods

Detection of root disease is particularly difficult because of the insidious nature of the infection, because the pathogens develop underground, and because a decaying tree often remains for a long time without aboveground symptoms. Although imperfect and labor-intensive on a large scale, the detection method currently used in the Ivory Coast, which was adopted for this survey, consists of (*i*) looking for direct or indirect aboveground symptoms of infection, i.e., the presence of mycelium or sporophores on the lower part of the stem, grooves on the trunk, distinct delay in growth, wilting or discoloration of leaves, and abnormal seed production; and (*ii*) clearing the soil around the collar and taproot to look for and characterize the mycelium on the part of the root system that can be seen in the opening (30–40 cm radius and 10–20 cm depth), which is refilled after inspection. Secondary or subsequent infection (by *R. lignosus* or *P. noxius*) detected on the root system of an already infected tree (by one of the two pathogens) was not assessed.

Epidemiological survey procedure

The survey site was a rubber plantation (13 000 ha) located in the southwest of the country, in the dense rain-forest zone that borders the Gulf of Guinea. Before planting, the forest was cleared either mechanically with a tree-pusher or manually with saws. The soils are of a ferralitic desaturated clayey-sandy type. The climate of the region consists principally of two rainy seasons, with maxima in May–June and September–October, and two dry seasons. The driest period lasts from December to March and coincides with the annual defoliation of rubber trees in February. Annual precipitation ranges from 1300 to 2000 mm.

To take into account all possible variations of site conditions (soil and initial inoculum density), a preliminary survey was performed in 1977 on the area of the 1973 plantation. Then 20 plots (total area 15.5 ha, initial planting density 550 trees/ha in 1973) characterized by the presence of numerous infected trees were selected. For each plot, the locations of the planting lines and the positions of living and missing rubber trees were recorded on maps (scale 1:700). The first inspection (designated inspection 0), in October 1977, included 7636 living trees (healthy and diseased). The next assessment (inspection 1) was made in October 1978 and then inspections 2–11 were made systematically in April and October of each year until 1983, to include wet and dry periods. During each assessment, each tree was inspected and all cases of infection and mortality were systematically recorded and noted on the maps, with the number of the inspection (Fig. 1).

The results of the twice-yearly inspections of each tree were recorded on the map corresponding to each of the 20 plots. Contours of the disease centers at the end of the survey were also displayed on these maps. For each disease center, the earliest

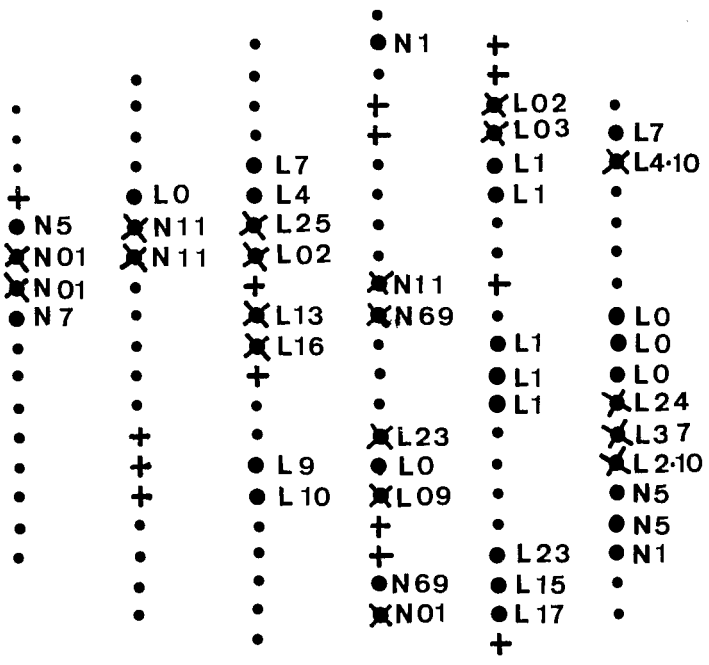

FIG. 1. Part of one of the maps used for data recording. A healthy tree is represented by a small solid circle, and a missing tree (bad growth or death caused by animal damage or early root-rot infection) by a cross. After detection, a diseased tree is indicated by a large solid circle, with a letter indicating the identity of the pathogen seen on the root system (L, *R. lignosus*; N, *P. noxius*). The first number indicates the inspection when the disease was detected (for example, ●L3 means that the tree was infected by *R. lignosus* and detected at inspection 3). Later, if the infected tree died, this is indicated by a cross and the inspection number (for example, ✖N06 means that the tree was infected by *P. noxius*, detected at inspection 0, and death was recorded at inspection 6).

detected diseased tree was considered the focus. Two criteria were considered in the infection center analysis: (*i*) the age of the center, i.e., the date when the first diseased tree was recorded; and (*ii*) the size of the center in terms of the number of diseased trees, a distinction being made between small ($n < 3$) and large ($n \geq 3$) centers. Percentages of infected and dead trees are given in terms of numbers of living trees at the beginning of the study ($n = 7636$).

Results

Initial infestation by root pathogens

At the beginning of the study, 4.1% of the tree population was infected; of 312 diseased trees, 245 (78.5%) were infected by *R. lignosus* and 67 (21.5%) by *P. noxius*. These diseased trees represented 102 and 48 disease centers whose average densities were evaluated to be 6.5 and 3.1 foci/ha for the respective pathogens. Two types of heterogeneity were observed within the plots (Table 1). (*i*) In some plots, nearly all infections were due to *R. lignosus*, whereas in others there was near parity between the numbers of infections caused by the two fungi. (*ii*) The numbers of diseased

TABLE 2. Number of trees newly infected or killed by *Phellinus noxius*, recorded at each inspection and related to wet and dry seasons

	Inspection no.										
	1	2	3	4	5	6	7	8	9	10	11
	Wet	Dry	Wet	Dry	Wet	Dry	Wet	Dry	Wet	Dry	Wet
Infected	105	24	44	23	40	20	33	3	21	3	4
Dead	97	26	13	15	14	23	18	21	12	12	3

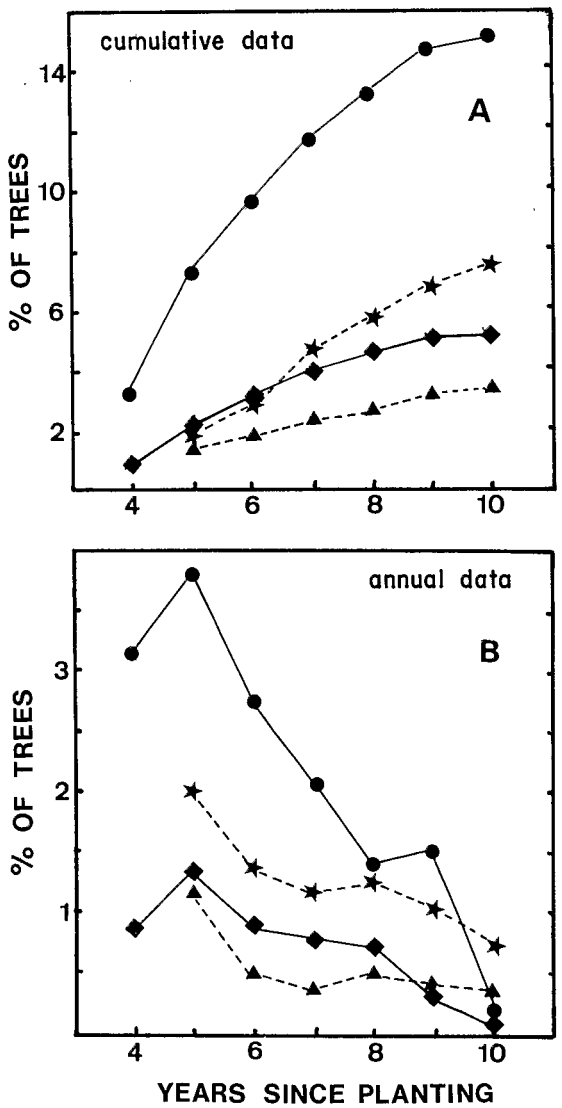

FIG. 2. Infection and mortality progress curves from years 4 and 10 after planting: ●, percentage of trees infected by *R. lignosus*; ★, percentage of trees killed by *R. lignosus*; ◆, percentage of trees infected by *P. noxius*; ▲, percentage of trees killed by *P. noxius*. Note that the value given for year 4 is cumulative from year of planting.

trees varied considerably among the plots, ranging from 0.8 to 8.8% (mean 4.1%). It is assumed that these variations among plots in a monoclonal area mainly reflect the distribution of the primary inoculum within the indigenous forest before planting (Nandris et al. 1984).

Incidence of root rot

Comparison of data from the beginning and end of the survey revealed the following points. (*i*) The number of diseased trees increased from 312 (4.1%) to 1538 (20.1%), corresponding to a multiplication rate of 4.9; in other words, 16% of the trees became infected during the 6 years, an average of 2.6% per year. The multiplication rates for newly infected trees within the plots varied from 0.5 to 33 for *R. lignosus* and from 0.9 to 13.6 for *P. noxius*. (*ii*) At the end of the survey, the proportions of trees infected by *R. lignosus* and *P. noxius* were 74.7 and 25.3%, respectively, indicating a slight increase in trees infected by *P. noxius* compared with the initial ratio. (*iii*) With regard to mortality caused by root pathogens during the survey, of the 1538 trees that became diseased, 829 (53.9%) died. This represents a loss of 10.8% of the initial number of living trees, or a mean annual loss of 1.8%. At the end of the survey, 65.3% of the trees infected by *P. noxius* were dead, compared with only 50% of those infected with *R. lignosus*; this reflects differences in aggressiveness between the two pathogens.

Dynamics of the pathogens

During the 1st year surveyed (year 5 after planting), 291 and 152 trees newly infected by *R. lignosus* and *P. noxius*, respectively, were detected (Fig. 2B). This rather abrupt increase corresponded to 36% of the total of newly infected trees detected during the entire survey. In the field, this was reflected by the enlargement of the existing disease centers and the appearance of new ones. From year 5 after planting, infection progress rates dropped noticeably, resulting in a virtual stagnation of disease development after year 9 (Fig. 2A). The annual infection rates recorded in year 10 are 15–20 times lower than those calculated at year 5. In April 1987 (4 years after the end of the survey), a new assessment at the same site confirmed this disease stagnation. The annual increase in the number of infected trees was then estimated to be 0.04–0.1%.

Although differences in pathogenicity were noted between the two pathogens, the mortality caused by both decreased significantly with time, particularly in the case of *R. lignosus* (Fig. 2B).

The twice-yearly inspections allowed the development of the disease in relation to climate to be studied throughout the survey. Contrary to that of *R. lignosus*, the disease pattern of *P. noxius* seems to vary with the season. The spread of pathogens in the soil (and consequently the infection of trees) occurred preferentially during rainy seasons, when soil moisture was high; root colonization and death of infected trees occurred mostly during dry seasons (Table 2), when water deficiencies were at a maximum.

Determination of disease type

Since Vanderplank's pioneer volume, *Plant Diseases: Epidemics and Control* (1963), most plant disease progress

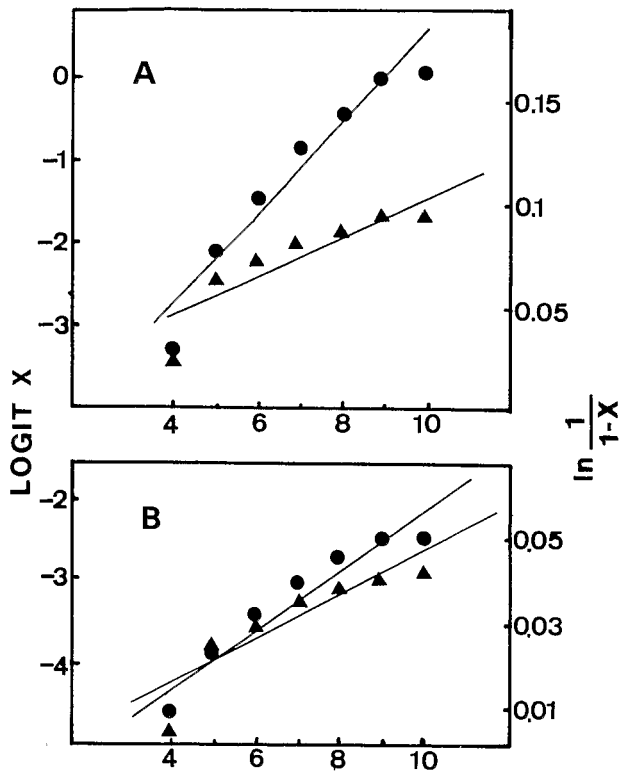

FIG. 3. Infection and mortality curves (between years 4 and 10 after planting) of (A) *R. lignosus* and (B) *P. noxius*, using the monomolecular (●) and logistic (▲) equations.

TABLE 3. Annual infection rates (r) for each type of disease, after mathematical transformation using simple interest ($r = 1/t_2 - t_1$ (ln x_2/x_1)) and compound interest ($r = 1/t_2 - t_1$ (logit x_2 − logit x_1)) formulae

	Year since planting					
	4–5	5–6	6–7	7–8	8–9	9–10
Rigidoporus lignosus	0.93	0.26	0.22	0.13	0.13	0.01
Phellinus noxius	1.05	0.33	0.25	0.17	0.07	0.02

curves have been classified as either simple interest (SID) or monocyclic, and compound interest (CID) or polycyclic. This method was also used for quantification of disease progression in forest pathology (Madden 1980; van der Pass 1981).

After transformation, the recorded data (Fig. 3) seem to fit the monomolecular equation (SID) slightly more closely, but on this mathematical basis, the differences are too small to enable root rot of rubber trees to be classified with any certainty as a monocyclic disease. Also, the inflexions of the disease progress curves (after transformation) at the beginning and end of the survey period indicate that the apparent infection rate, r, does not remain constant; calculation of the annual values of r reveals a significant decrease from years 4 to 10 (Table 3).

Appearance and enlargement of disease centers

Examination of the change with time of the number of disease centers between years 4 and 10 revealed that by year 8, new foci developed sparingly. The shape of the curves (Fig. 4) suggests that virtually all the foci that appeared during this first rubber tree cultivation cycle (30 years) were initiated before year 10 after planting. Differences in pathogen behavior can be seen in Fig. 4, as 65 and 51% of the disease centers caused by *P. noxius* and *R. lignosus*, respectively, appeared after year 4. Table 4 shows that on average over 5 years, almost 90% of the trees infected by *R. lignosus* each year after inspection 0 showed enlargement of existing disease centers, whereas more than 30% of infections due to *P. noxius* were recorded away from existing foci and thus could be considered new foci.

Table 5 shows that after their creation, many root disease centers did not enlarge through infection of the healthy neighboring trees, and that the capacity of the disease centers to expand is noticeably lower for those caused by *P. noxius* than for those caused by *R. lignosus*.

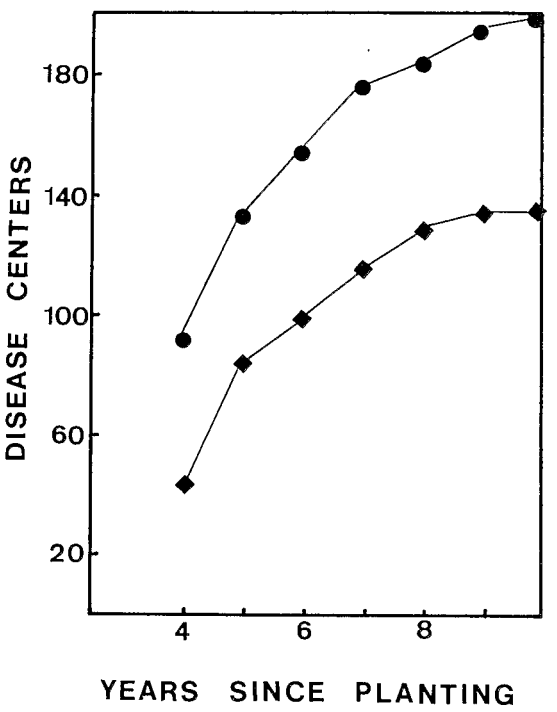

FIG. 4. Cumulative number of disease centers that were detected during the survey ●, *R. lignosus*; ◆, *P. noxius*.

Discussion and conclusions

In the site surveyed, the area to be planted with rubber trees (13 000 ha) was located in a total area of 35 000 ha in a dense rain forest. Initial sampling of the 20 plots on this site was designed to take into account the variability of local conditions. Thus, these plots could also be considered to represent 20 smallholder plantations covering the variety of soil conditions and inoculum densities of this part (southwest) of the Ivory Coast which now constitutes the main area of rubber cultivation in the country.

Interpretation of the data collected during the 6-year survey revealed first a sharp increase in the rates of infection and mortality, followed by a clear drop leading to current stagnation of the epidemic. The peak of pathogen activity was recorded during year 5 after planting. This appears to agree with the observations of Martin and Du Plessix (1965) concerning the progress of mortality in a rubber tree plan-

TABLE 4. Percentage of trees, newly infected by the two pathogens, that were detected away from existing disease centers at each inspection after year 4

	Inspection no.									
	2	3	4	5	6	7	8	9	10	11
Rigidoporus lignosus	9.9	15.3	10.5	20	10.2	7.3	12.7	17.7	25	0
Phellinus noxius	28.5	40	26	40	40	36.6	33	24	0	0

TABLE 5. Percentage of the different types of disease center that did not enlarge after creation in the field

	Type of disease center			
	Old	New	Large	Small
Rigidoporus lignosus	26	86	8	45
Phellinus noxius	42	85	23	48

NOTE: Old disease centers are those that were present at the first inspection; new centers were created after the first inspection; large centers comprise more than three trees; small centers comprise less than three trees.

tation in the southeastern part (sandy soils) of the Ivory Coast. In Sri Lanka, Riggenbach (1960) has observed that white root disease (*R. lignosus*) caused the greatest damage to rubber plants between the 2nd and 5th year of cultivation. Wijewantha (1964) reported a similar pattern in replanted areas, but found that the peak of pathogen activity occurred by the 2nd year of cultivation and was initially much more severe. In Malaya, too, John (1966) noticed a rubber tree root disease outbreak during the 3rd year.

Particular attention should be given to the "plateau phase" that is the next stage in the disease pattern, because it raises the question of mechanisms that are able to halt or slow the development of disease in the field. Indeed, more than 700 diseased trees (10.4%) remaining in the plots constitute a potential inoculum for infection of the healthy neighboring rubber trees.

Numerous observations made in the Ivory Coast, on mature rubber trees in various plantations, as well as on artificially infected rubber seedlings in the greenhouse (Nandris et al. 1983), have suggested that this stagnation of the epidemic could result from two attendant phenomena. (*i*) Defense reactions increase with age of the host and contain the pathogen in the decayed portion of the taproot. The cellular reactions of the tree's root system to fungal attack consist mainly of cellular hypertrophy and hyperplasia, stimulation of cambial activity, lignification and suberization of walls, callose deposition on pores of the sieve tubes, and formation of tyloses (Nicole et al. 1986). In addition, these reactions are characterized by changes in the enzymatic metabolism of the host, particularly peroxydases (Geiger et al. 1986). Anatomically, the formation of secondary taproots, cicatricial swellings, and hypertrophy of lateral roots have been observed *in situ* and in the greenhouse. The frequency and efficacy of these reactions, which compensate over time for the loss of the decayed taproot, have been shown to increase with age of inoculated rubber seedlings (Nicole et al. 1983). John (1966) also reported that the ability of *R. lignosus* to infect and kill a rubber tree was inversely related to the age of the host. The incidence of *Armillaria mellea* Vahl ex Fries in conifers (Greig 1962) and in indigenous and exotic trees (Swift 1972) was also recorded as becoming insignificant after 9 or 10 years of tree cultivation. (*ii*) Pathogen activity, i.e., the ability to kill a diseased tree and then spread towards a healthy neighboring host, decreases with time. Indeed, in enlarging disease centers, initial food bases (residual decayed stumps) became increasingly distant from the border of the center that represents the active infection zone; consequently, trophic connections through the mycelium with decaying taproots of rubber trees should become increasingly attenuated. Thus, the pathogen gradually passes from perthophytism to near-absolute parasitism. For "weak" pathogens like *R. lignosus* and *P. noxius*, such a phenomenon may induce a major decrease in activity and aggressiveness (Fassi 1964). However, this decrease does not mean degeneration or senescence of these fungi. Pathogen trapping (using sticks made of rubber tree wood buried in the soil around the collar for 3 weeks before examination) on diseased rubber trees surviving for 5 years (in the surveyed plots), and even for 20 years (in an old plantation elsewhere), have confirmed the persistence of *R. lignosus* in their root systems.

According to the monomolecular and logistic equations, where r is constant, and in view of allied problems emphasized by Pfender (1982), the large variation in annual infection rates makes it difficult to propose suitable mathematical characterizations of epidemics of rubber tree root-rot diseases in the Ivory Coast.

However, two major phases that qualitatively characterize these epidemics and the nature of disease cycle can be identified from field observations. (*i*) After forest felling, secondary disease centers consisting of young rubber trees infected from residual forest stumps (primary inoculum) appear during the first few years of cultivation. According to Vanderplank's (1963) definitions, this seems to be a typical SID pattern because infection and death are associated with the initial inoculum rather than with the spread of disease from tree to tree. Differences in vitality among fungal isolates (Nandris et al. 1987*b*) and in distance between inoculum and healthy roots could, nevertheless, cause a delay in the center's creation, thereby giving the illusion of enlargement or multiplication of disease foci over time. (*ii*) Until years 8 or 9 after planting, each diseased mature tree consists of an "autonomous" disease focus that can enlarge by mycelial spread from tree to tree. During these years of outbreak, pathogen spread through root contact between trees should be considered as compound (CID), because each newly diseased tree, as the interest on the "capital", would be included in the amount of disease (Campbell and Powell 1980). Therefore, during the first 10 years, fungal spread would consist mainly of CID-like development, the initial phase being considered an incubation period (i.e., contact between decayed stumps in the forest and the roots of young rubber trees), as emphasized by Hirst and Schein (1965) and Jones (1978).

It is interesting to study the comparative dynamics of *R. lignosus* and *P. noxius* with particular attention to the creation of disease centers in the field (Table 4). (*i*) After the beginning of the survey, there are many more new foci for *P. noxius* than for *R. lignosus*. (*ii*) In comparison with the initial number of disease trees (at inspections 0 and 1), the number of diseased trees in these new foci account for an increase of 47% for *P. noxius* and only 18% for *R. lignosus*. (*iii*) Of the cases of *P. noxius* detected every year, 34% appear external to any previously created focus (the remaining 66% result from infection from neighboring diseased trees in the center); thus, these trees reveal the activity of primary inoculum not previously initiated, and constitute secondary centers. In the case of *R. lignosus*, on the other hand, only 15% of the newly detected trees are localized away from already active disease centers.

These data concerning the localization of trees newly infected during the whole survey are consistent with the following observations. (*i*) The epidemics caused by *R. lignosus* seem to correspond more to a polycyclic type, because the multiplication of the initial inoculum by successive infections of trees takes precedence over the creation of new centers. As a result, the incubation period of the disease would be shorter or would occur earlier, but seems in any case to be finished practically at year 4 after planting. (*ii*) In comparison, the epidemics of *P. noxius* could be considered much more of the simple interest type, or as epidemics with a much longer incubation period; indeed, over the whole survey, pathogen activity essentially took the form of creation of secondary disease centers and rarely of their enlargement. These interpretations do not call into question the previously stated conclusions, but enable them to be modified according to the characteristics of each of the two types of epidemic.

More than 60% of *P. noxius* foci appeared in the field 4 years after planting. According to the biology of this pathogen, which lacks fast-growing rhizomorphs, this delay in initiation of secondary disease centers could be explained by the slow rate of growth of the mycelial sleeve on roots and the inability of the fungus to cover even short distances without a root substrate. However, as the wood-decaying ability of *P. noxius* is greater than that of *R. lignosus* (Geiger et al. 1985), the forest stumps rotted by *P. noxius* should decompose faster than stumps colonized by *R. lignosus*. Therefore, disease center initiation in plantations (after spread from stump to tree is complete) should have appeared earlier for *P. noxius*. How can these apparently contradictory phenomena be reconciled? In a root attacked by *P. noxius*, infection and wood decay occur almost simultaneously (Saccas 1975; Hodges and Tenerio 1984), whereas the epiphytic growth of *R. lignosus* occurs much in advance of the site of decay (Fox 1977). Thus, in contrast to *R. lignosus*, *P. noxius* was able to develop "autonomously" (but slowly) in the infected roots as soon as it contacted the host root system; consequently, disease centers of *P. noxius* can appear well after the disruption of trophic connections with the primary inocula.

Many disease centers did not enlarge after their creation, and differences in the characteristics of centers caused by each of the pathogens have been reported. These observations should be related not only to the host's defense mechanisms and the differences in mycelial spread between pathogens but also to the existence of important variations in pathogenicity among the various isolates constituting the population of each root pathogen in this plantation (Nandris et al. 1987*b*). This intraspecific variability is indeed a component to be considered in investigating the development of root-rot diseases in the rubber tree plantations of the Ivory Coast.

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