Eur. J. For. Path. 26 (1996) 25–44

ISSN 0300–1237

Institut Français de Recherche Scientifique pour le Développement en Coopération, Laboratoires de Phytopathologie, Centres ORSTOM, Nouméa[1] et Montpellier[2]; Institut National de la Recherche Agronomique, Laboratoire de Biométrie, CRA Montfavet[3]; Station de Biométrie, CNRF Champenoux[4] et CRF Nancy[5] Laboratoire de Recherche Forestière, INRA/ENGREF, Nancy, France

Modelling rubber-tree root diseases, simulations of various inoculum rates and methods of control

By D. NANDRIS[1], J. CHADOEUF[3], J. C. PIERRAT[5], H. JOANNES[4]
J. P. GEIGER[2] and M. NICOLE[2]

Summary

Two root-rot fungi, *Rigidoporus lignosus* and *Phellinus noxius* are the causal agents of severe damage in Ivory Coast rubber tree (*Hevea brasiliensis*) plantations. Mathematical investigations were made using data from a previous 6 year long epidemiological survey. Modelling of spatial spread of the pathogens and mortality of the infected trees, lead to simulations of the quantitative evolution vs. time of root diseases. The theoretical kinetics obtained revealed intra- and interspecific variability among the studied stands with respect to the damage caused. Using this model, different phytosanitary scenarios were simulated in relation to the density of the initial inoculum, the control measures, the specificity of the fungicide, the efficiency of the treatment, and the optimal time for application. Their influence on root-disease dynamics was quantified. The characteristics of the model, its applicability in plantations and its use in predicting disease or stand development are discussed.

1 Introduction

Since the development of rubber-tree cultivation (*Hevea brasiliensis* [Wild. ex Adr. de Juss] Mull. Arg.) in Western Africa, and more particularly in the Ivory Coast, the root-rot fungi *Rigidoporus lignosus* (K1.) Imazeki and *Phellinus noxius* Corner G.H. Cunn have represented a constraint both for smallholders and for managers of industrial plantations (MARTIN and DU PLESSIX 1969; NANDRIS 1985).

Consequently, a multi-methodological research programme was carried out in the Ivory Coast for several years (GEIGER et al. 1986; NICOLE et al. 1986; NANDRIS et al. 1987). At the end of the epidemiological investigations, a model of root-disease dynamics was developed using data collected *in situ* (NANDRIS et al. 1988a). The aim of this mathematical approach was to elaborate a decision-making tool able to give rational information on the dynamics of the epidemic (i.e. forecasts of potential losses caused by these root pathogens) and on the expected performance of available control measures. Such data are needed to help decision makers choose the most appropriate pest-control method (MUMFORD 1987).

The first step of the modelling was to quantify the two sequential phases of the infection chain: spread of pathogens and time of mortality of diseased trees (CHADOEUF et al. 1988). Then, assuming that these spatial and temporal phenomena are independent, root-disease evolution was simulated using the model. The effectiveness of current control methods was investigated with particular attention to the respective impact of various means and timing of application. Results obtained on these topics and applications of the model are presented in this paper.

0300–1237/96/2601–0025 $11.50/0

2 Materials and Methods

2.1 Biological data

The root-rotting fungi, *R. lignosus* and *P. noxius*, have been shown to spread in the soil by developing hyphae from infected residual stumps of forest trees, and/or from woody debris buried in the soil after stand clearing. On the root system of the rubber tree, *R. lignosus* and *P. noxius* are identifiable by fast-growing rhizomorphs and slow-growing ectotrophic mycelial sleeves, respectively. In the spread phase, the mycelium runs along the lateral roots and reaches the tap-root. The fungus penetrates the wood and begins the internal rotting process. Once infected and decaying, the diseased rubber tree in turn constitutes a new inoculum source allowing the infection centre to expand by successive infestations of neighbouring trees.

The epidemiological survey was performed on 4-year-old rubber trees located in several stands of an industrial plantation set up after primary forest clearing. The rubber trees were planted in a standard layout, every 2 m along the row, with 8 m between rows. This survey initially consisted of detecting the disease on the root system of each of the rubber trees, then mapping all healthy and infested trees. The plots were monitored for 6 years with 6-monthly inspection readings. Further information on the procedure was detailed previously by NANDRIS et al. (1988a).

2.2 Characteristics of the model

For this modelling, only the stands characterized by mechanical ground operations prior to planting (clearing, windrowing and burning of the woody debris) were selected, because these correspond to current standard practice in industrial rubber tree plantations. The high density of trees (> 500) within each of the three selected stands was suitable for estimating the model parameters.

Considering previous results on the underground rate and type of mycelial spread, the propagation of pathogen from one tree to the trees located on the adjacent row (8 m) is a particularly rare event. Between two successive inspections, only the two nearest-neighbour trees (on the same row, 2 m apart) have an influence on the state of health of a given healthy tree (CHADOEUF et al. 1988). Moreover, once diseased (even on a small portion of the root system), an infected tree remains diseased. Therefore, the spread of the root pathogens at each date was modelled as a spatial Markovian model (KRICKEBERG 1975; PRUM 1986) on the remaining healthy trees at the previous inspection (NANDRIS 1985):

For each row, let $x(i, t)$ be the infection status of the ith tree. Let $C(t)$ be the set of all possible states at time t of a tree healthy at $t-1$. These states are: healthy, 0; infected by *R. lignosus* since time t, $l(t)$; and infected by *P. noxius*, $n(t)$. A particular state related to trees found dead or missing at $t=0$ was added. As the mortality of such trees can be due to initial planting problems or previous pathological decay, they cannot be considered directly and they will be coded m.

Pathogen spread is modelled using two main groups of parameters:

- if the trees $i-1$ and $i+1$ are healthy at time t, then the probability of finding the tree i in a given status of healthy — knowing the status of health of all other trees — and knowing that the tree i is healthy at $t-1$ will be modelled by:

$$P\left(x(i,t)\,\middle|\,\begin{matrix}x(j,t),\ j\neq i\\ x(i,t-1)=0\end{matrix}\right)=\mathrm{P}\left(\mathrm{x}(i,t)\,\middle|\,\begin{matrix}x(i-1,t)=0\\ \mathrm{x}(\mathrm{i},t-1)=0\\ x(i+1,t)=0\end{matrix}\right)=\frac{\exp\left(a(x(i,t))\right.}{\sum\limits_{y\in C(t)}\exp\ (a(y))}$$

with the convention $a(0)=0$. In this formula, the parameter a represents the probabilities of a tree becoming infected while its two neighbours remain healthy. This describes the concept of initial infection due to mycelium spreading in the soil from a forest residual inoculum, which may have direct contact with the root system of sapling rubber trees. The level of efficacy in clearing the forest prior to planting could influence this parameter whereby an effective stump removal would lead to a decrease in the density of remaining infected wood debris.

- if the trees $i-1$ and $i+1$ are infected, the probability of finding the ith tree in a given status is:

$$P\left(x(i,t)\,\middle|\,\begin{matrix}x(j,t),\ j\neq i\\ x(i,t-1)=0\end{matrix}\right)=P\left(x(i,t)\,\middle|\,\begin{matrix}x(i-1,t)\\ x(i,t-1)=0\\ x(i+1,t)\end{matrix}\right)$$

$$=\frac{\exp\left(a(x(i,t)+b(x(i,t),x(i-1,t))+b(x(i+1,t),x(i,t))\right)}{\sum_{y\in C(t)}\exp\left(a(y)+b(y,x(i-1,t))+b(x(i+1,t),y)\right)}$$

where the function b verifies the condition $b(x(l-1,t),x(l,t))=0$, if $x(l-1,t)=0$ or $x(l,t)=0$ for every position l and any date t, i.e. no interaction between two trees as long as one of them is healthy. The parameter of interaction b describes variations in the probability of tree infection due to the existence of infected neighbours. Related to pathogen spread from tree-to-tree, the parameter b will characterize tree infection by contagion, i.e. the polycyclic development of the disease, as described in VANDERPLANK (1963).

Let us recall that a diseased tree at time t remains diseased at $t+1$. A detailed description of the model of spread, its estimation per codage (BESAG 1974) and simulation procedure can be found in CHADOEUF et al. (1992). Various submodels of the above general model were tested. The final model is described as follows:

$$a(l(t))=a_0(l)+ta_1(l)+(-1)^t a_2(l) \qquad b(l(t),l(t'))=b_0(l,l)+(t-t')b_1(l,l)$$

$$a(n(t))=a_0(n)+ta_1(n)+(-1)^t a_2(n) \qquad b(n(t),(t'))=b_0(n,n)+(t-t')b_1(n,n)$$

$$b(l(t),m)=b_0(l,m)+tb_1(l,m) \qquad b(n(t),m)=b_0(n,m)+tb_2(n,m)$$

$$b(l(t),n(t'))=b_0(l,n)+(t-t')b_1(l,n)$$

The results of the estimation of the parameters are presented in Table 1 (CHADOEUF et al. 1993).

2.3 Methodology for simulation

As previous results on root disease activity have definitely revealed the existence of a plateau phase from the 10th year of planting (NANDRIS 1988a), simulations of epidemics were conducted over a 13-year period. For each analysis, 50 simulations of the development of root-disease infection and tree-death mortality were carried out. The corresponding variation range between the repetitions is illustrated in Figure 1a. Results are presented as cumulated values (Fig. 1b) allowing determination of the level of the plateau phase, the inflexion point, and the variation range recorded at the end of the simulation time. In order to compare various scenarios on the same graph, each set of 50 simulations was reduced to a single curve going through the median of the box-plots (Fig. 2). The values of the median at the plateau stage, of the inter-quartile interval and of the confidence interval (risk of

Table 1. Estimates of the parameters of the model (spread and mortality), with individual confidence intervals at 95% (parts of this table have been previously published in Biometrics 48, 1165–1175). The parameter $a_0(u)$ represents the forest residual inoculum at the beginning of the survey; $a_1(l)$ corresponds to a linear decrease vs. time of the potential of this inoculum; $a_2(u)$ concerns a seasonal effect on this process; $b_0(u, v)$ describes the capacity of a given infected tree to be contagious (at the time of disease detection) and $b_1(u, v)$ its decrease vs. time; M and μ describe the probability of death for a given infested tree and the time between initial infection of roots and tree death, respectively

Parameter	Estimate	Lower limit	Upper limit
$a_0(l)$	−4.38	−4.81	−4.00
$a_1(l)$	−0.20	−0.30	−0.10
$a_2(l)$	−0.33	−0.52	−0.14
$a_0(n)$	−5.37	−6.06	−4.78
$a_1(n)$	−0.19	−0.34	−0.03
$a_2(n)$	−0.25	−0.54	0.03
$a_0(m)$	−2.36	−2.59	−2.15
$b_0(m, m)$	0.73	0.40	1.04
$b_0(l, m)$	0.76	0.45	1.06
$b_0(l, m)$	0.83	0.32	1.31
$b_0(l, l)$	2.86	2.46	3.29
$b_1(l, l)$	−0.21	−0.37	−0.06
$b_0(n, n)$	3.88	3.22	4.57
$b_1(n, n)$	−0.35	−0.68	−0.07
$b_1(l, n)$	−0.19	−0.66	0.16
$b_0(l, n)$	1.77	1.09	2.41
$b_1(n, l)$	−0.12	−0.46	0.15
$\mu(l)$	0.96	0.86	1.06
$M(l)$	0.55	0.43	0.67
$\mu_0(n)$	0.72	0.64	0.90
$M_0(n)$	0.70	0.56	0.84
$\mu_1(n)$	1.93	1.49	2.37
$\alpha(n)$	−0.14	−0.57	−0.05
$\beta(n)$	1.15	1.06	2.05

10%) corresponding to each simulation, are gathered together in Table 2. In a first set of simulations, for each stand, the pathological condition that was recorded at the first inspection only was taken into account; in a second set of simulations, an entire process, including also the simulation of the initial infestation, was performed.

2.4 Simulating root-disease spread, with (or without) control methods

Because the rows are independent, the evolution of a stand, considered as a group of rows, was simulated by simulating independently the evolution of each row. A row was simulated as follows. At the first date, it was simulated by using the equivalent four-state Markov chain (SPITZER 1973). At other dates, because the segments of consecutive healthy trees were independent, their evolution was simulated independently by calculating, for each tree, its probability either of becoming infested by one pathogen or of remaining healthy, knowing the state of health of the neighbouring trees at this date and the distance to the end of the segment (CHADOEUF et al. 1993).

Several values of parameters a (linked to the probability of young trees being in contact with an inoculum source) and of parameters b (contagion of the disease by successive infection of trees at the perimeter of infection centres) were used in order to estimate their influence on the epidemic dynamics. Results concerning changes in the parameters $a_0(l)$, i.e.

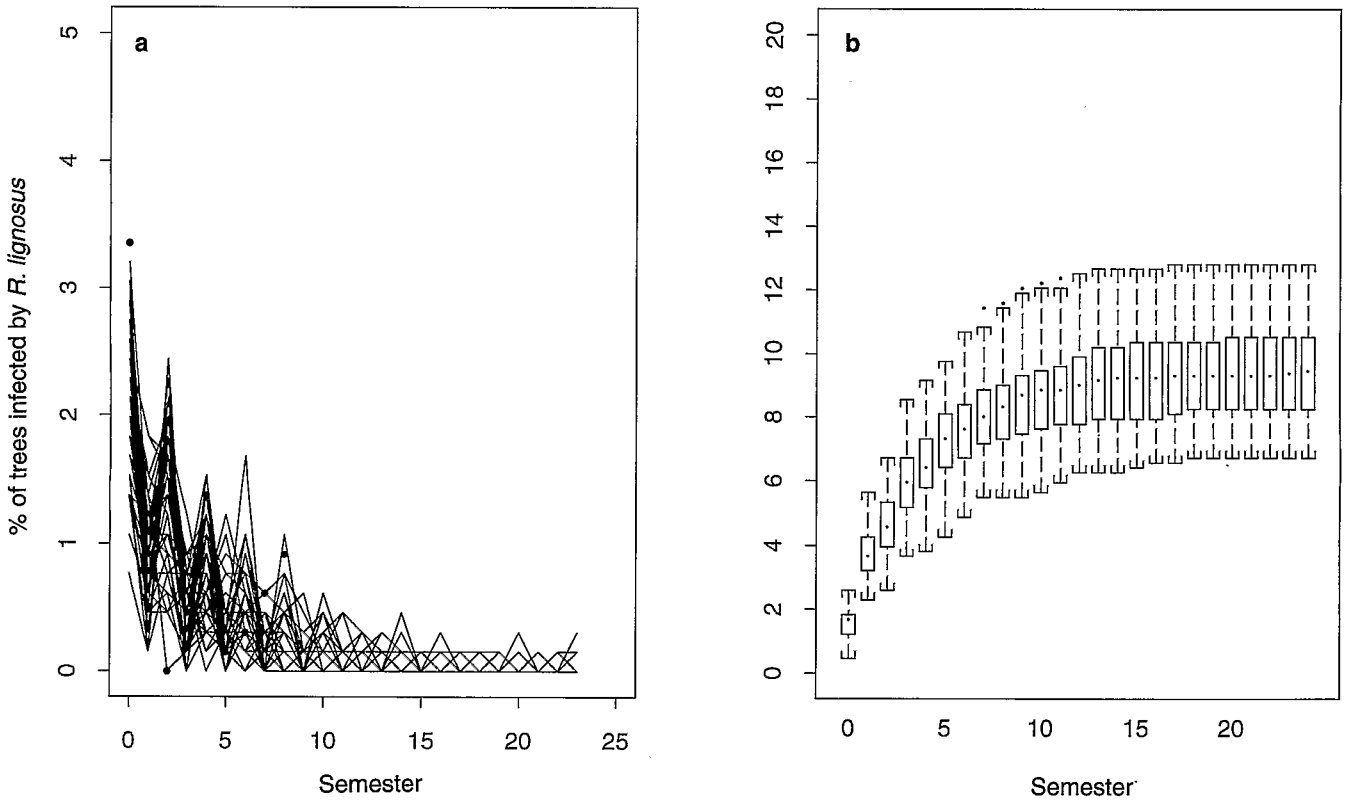

Fig. 1. Simulated kinetics of root rot disease: a. Frequency (%) of trees infested by *R. lignosus* (within the whole number of trees surveyed), 50 successive simulations; b. Synthetic display of the cumulated data using boxplots (CHAMBERS et al. 1983)

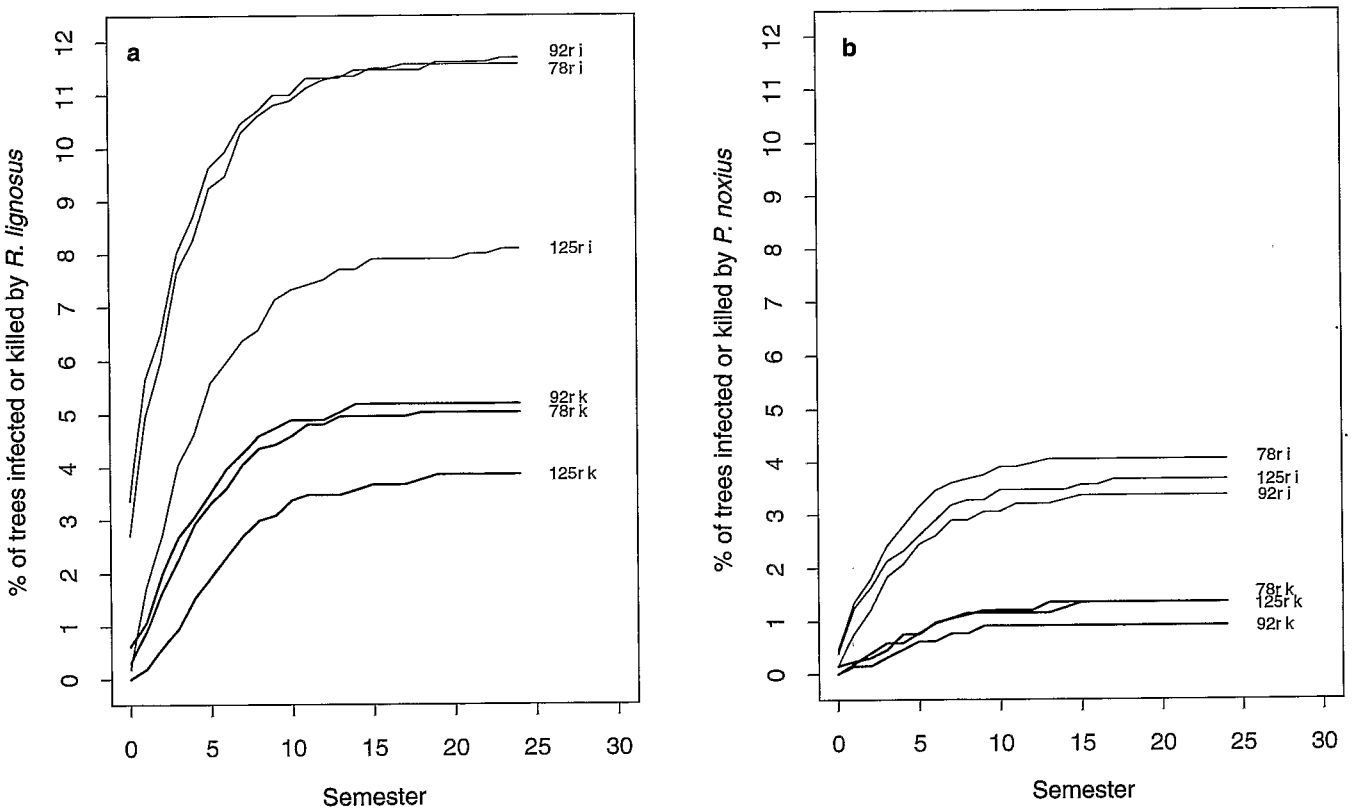

Fig. 2. Simulated dynamics of root-rot diseases based on the initial infestation of the stands (using diseased and healthy trees): a. Frequency (%) of infected (thin lines, code i) or killed (thick lines, code k) trees by *R. lignosus* (within the whole number of trees surveyed), respectively; b. Frequency (%) of infected (thin lines, code i) or killed (thick lines, code k) trees by *P. noxius*. 92r, 78r and 125r: stands studied

Table 2. Simulated infestation of rubber trees by *Rigidoporus lignosus* or by *Phellinus noxius* with incidence of root disease after 12 years (% of infected or dead trees within the total number of trees surveyed). 92r: initial actual configuration in stand#92 (simulation based on diseased sapling trees only); 92s: simulation of initial configuration according to diseased and missing trees; M: median, IQR: interquartile range (25–75%); CI: half-length of the Confidence Interval (10%). Idem stands#78 and 125

Stand	% of trees infected by *R. lignosus*			% of trees killed by *R. lignosus*			% of trees infected by *P. noxius*			% of trees killed by *P. noxius*		
	M	IQR	CI	M	IQR	CI	M	IQR	CI	M	IQR	CI
92s	10.08	2.35	0.52	4.47	1.09	0.24	4.22	1.62	0.36	1.46	0.77	0.17
78s	10.51	2.21	0.49	4.57	1.50	0.33	4.27	1.35	0.30	1.65	0.90	0.20
125s	10.01	2.40	0.53	4.43	1.54	0.34	4.43	1.34	0.29	1.54	1.10	0.24
92r	11.67	1.79	0.39	5.19	1.18	0.26	3.35	1.22	0.27	0.91	0.45	0.10
78r	11.56	1.65	0.36	5.03	1.12	0.25	4.05	0.86	0.19	1.35	0.56	0.12
125r	8.09	1.49	0.30	3.85	0.77	0.17	3.66	0.96	0.21	1.34	0.57	0.12

initial infection parameter for *R. lignosus*, and $b_0(l, l)$, i.e. contagion parameter for *R. lignosus*, are presented below. They are referred to as *a* or *b*.

- Firstly, each of the two parameters a and b were made to vary separately, the other being equal to the estimated value, so that the infection rate by *R. lignosus* varied from 0.5 to 40% in the first case, and from 5 to 40% in the second case. Subsequently, the two parameters were made to vary simultaneously to calculate the incidence of various combinations on stand evolution.
- The use of various current control methods was also simulated in order to estimate their respective efficacy in reducing root-disease development:
  - (i) treatment decreasing the 'initial infection risk', i.e. for example, careful clearing of the residual forest vegetation by stump removal, and/or protection of the tap-roots of young rubber trees by fungicide fumigation;
  - (ii) treatment acting mainly on the 'contagion risk', by preventing mycelial spread from the decaying woody tissues; i.e. specific fungicide inhibiting fungal morphogenesis (GOHET et al. 1991);
  - (iii) treatment acting simultaneously on both infection risks.

In each of these scenarios, the treatment (whose effect is supposed to be similar for each of the two pathogens) was characterized by two hypothetical efficacy rates (50 and 100%). These two levels should be considered as indicating either, the efficacy of fungicide application in the field, or its intensity of diffusion underground and/or efficacy of ground operations like stump removal. To evaluate the effectiveness of treatments with increasing application cost within large areas, these prophylactic operations were also simulated according to an increasing number (1, 2 or 3) of treated neighbours along the row, apart from each newly or previously infected tree.

2.5 Simulating infected tree mortality

In this industrial plantation, rubber trees are grown for nearly 30 years only. After this time, these 'old' trees are removed to be replaced by seedlings. During the epidemiological survey, the probability of pathological mortality of the surveyed trees was estimated. Thus, it was shown that many infested trees were able to stop the internal root decay for several years (NANDRIS et al. 1988b). This was also confirmed by stick-trapping experiments around the collars of 30-year-old rubber trees. In fact, many apparently healthy trees have been shown to support root-pathogen decay (NANDRIS et al. 1987).

Tree mortality was modelled using the ERLANG-2 model (HAHN and SHAPIRO 1967). The two parameters were M = probability of infected tree death and μ = time after initial infection for maximum mortality. In this model, tree mortality (for natural or pathological causes) that intervened after a period of 30 years was not taken into account. The maximum-likelihood method was used to infer this model (DOBSON 1983).

For a tree infected at time $t = k$, the probability of dying at time l after the infection of its root system, is:

$$p(l) = \frac{M(l-k)}{\mu} \exp\left(-\frac{(l-k)}{\mu}\right)$$

The parameters of the retained model are:

$$M(l(t)) = M(l), \quad M(n(t)) = M_0(n)1_{t=0} + (\alpha(n)t + \beta(n))1_{t>0}$$

$$\mu(l(t)) = \mu(l), \quad \mu(n(t)) = \mu_0(n)1_{t=0} + \mu_1(n)1_{t>0}$$

their estimations are presented in Table 1. During the time of survey, the overall probability

of pathological mortality of the infested trees was estimated to be only 0.5 and the mean duration of the infectious cycle (for the two pathogens) was 2 years (NANDRIS et al. 1988b).

3 Results

3.1 Simulating root-rot-disease dynamics

The first simulation considered the initial root-pathogen density (at the beginning of the survey) in the three stands in order to estimate the epidemic development vs. time. Figures 2a and 2b show the severity of the damage caused by each of the two pathogens. After a 6-year simulated growth of the trees, the simulated evolution of root disease is characterized by a great increase in the number of trees affected, then by a definite plateau phase (Fig. 2). During the period, inter-stand variations are more noticeable for *R. lignosus* than for *P. noxius*. Variations exist between the 50 repetitions of a stand simulation (Table 2) and show differences that can be attributed to the density of the infection centres and/or to their distribution within the stand. However, these inter-stand differences remain low. Therefore, only mean values are presented in Tables 3–5. At the end of the simulation period, the average level of the infections due to *R. lignosus* ranged from 8 to 11% and from 3.5 to 5% for those caused by *P. noxius*. The percentages of trees dead after root decay were evaluated at 3.5 and 1.5% respectively.

A second simulation considered the potential variations of the initial infestation by taking into account the parameters estimated at date 0 of the survey (Fig. 3). Using this method, approximately 10% of trees were infected by *R. lignosus* and 4.3% by *P. noxius*. This simulation might have a forecast value for plantations located in similar areas, since the inter-stand variability was lower than within-stand variation (Fig. 3; Table 2). These results were considered as reference values for calibrating and comparing further simulations.

3.2 Effect of modifying the epidemiological parameters

The interest of the model lies in the possibility of simulating various scenarios, such as initial pathogen infestation and/or enlargement of infection centres etc. To facilitate the presentation of the results, only the infections caused by *R. lignosus* were considered; those for *P. noxius* were qualitatively similar.

3.2.1 Variation of the initial infection risk

Figure 4 reveals the range of the variations corresponding to increasing values of the a parameter. This corresponds to a simulated continuous emergence of new centres (i.e. newly infected sapling trees) until the natural attrition of the epidemic dynamics. In contrast to the control ($a = -4.41$), the plateau stage of the epidemic is of about 25 and 40% of diseased trees, when $a = -3.82$ or $a = -3.47$, i.e. when the initial number of diseased rubber trees is multiplied by 2.5 or by 5; the level of mortality of these trees varies between 10 and 18%.

3.2.2 Variation of the contagion risk

Some increasing values of the parameter b were also considered. The kinetics of mortality obtained are quite similar to those obtained previously (Fig. 4). On the other hand, when comparisons are made with the reference values of Table 2, the corresponding variability (CI) is definitely higher (Table 3). This phenomenon arises from the fact that by increasing the lateral infection rate, it is possible to simulate the enlargement of the infection centres

Table 3. Simulated evolution of *R. lignosus* infestation vs time (% of infected or dead trees, after 12 years) according to various levels of the parameters *a* and *b* describing the initial infection and the contagion risks, respectively; SDI: Standard Disease Incidence, i.e. without any control-method application

		% of trees infected by *R. lignosus*			% of trees killed by *R. lignosus*			% of trees infected by *P. noxius*			% of trees killed by *P. noxius*		
a	*b*	M	IQR	CI	M	IQR	CI	M	IQR	CI	M	IQR	CI
SDI		10.08	2.35	0.52	4.47	1.09	0.24	4.22	1.62	0.36	1.46	0.77	0.17
−3.47	2.86	40.65	5.20	1.15	18.37	2.76	0.61	4.39	1.30	0.28	1.46	0.77	0.17
−3.82	2.86	24.22	3.69	0.82	10.65	1.66	0.37	4.55	1.74	0.38	1.62	1.09	0.24
−4.41	2.86	9.67	2.11	0.46	4.39	1.30	0.28	4.30	1.62	0.36	1.46	0.81	0.18
−7.08	2.86	0.48	0.44	0.09	0.16	0.28	0.06	4.06	1.13	0.25	1.30	0.77	0.17
−4.38	4.11	37.64	6.58	1.46	16.99	2.84	0.63	3.73	1.42	0.31	1.62	0.77	0.17
−4.38	3.84	23.33	4.51	1.00	10.73	2.27	0.50	4.14	1.38	0.30	1.62	0.97	0.21
−4.38	2.80	9.43	2.27	0.50	4.14	0.97	0.21	3.90	1.26	0.27	1.46	0.97	0.21
−4.38	0.00	6.01	1.38	0.30	2.76	0.77	0.17	4.06	1.58	0.35	1.46	0.77	0.17
−3.47	0.00	13.98	1.58	0.35	6.26	1.09	0.24	5.04	1.78	0.39	1.95	0.93	0.20
−3.47	3.84	74.31	4.27	0.95	10.73	2.27	0.50	2.11	0.89	0.19	0.97	0.65	0.14
−3.47	4.11	94.63	0.97	0.21	42.92	2.76	0.61	0.32	0.32	0.07	0.16	0.32	0.07
−7.08	0.00	0.48	0.28	0.06	0.16	0.32	0.07	3.98	1.13	0.25	1.46	0.93	0.20
−7.08	4.11	0.48	0.32	0.07	0.16	0.16	0.03	3.90	1.50	0.33	1.38	0.65	0.14

Table 4. Simulated impact of various control methods on root disease evolution vs. time (% of infected and dead trees after 12 years): treatment acting either on *a* (initial infection risk), on *b* (contagion risk) or on both. The number of the treated healthy neighbours and the efficacy of the treatment varies from 1 to 3 and from 50 to 100%, respectively. M: median; CI: Confidence Interval at 10%; SDI: Standard Disease Incidence, i.e. without any control-method application

Parameter	Number of treated trees	Hypothetic level of efficacy (%)	% of trees infected by *R. lignosus*			% of trees killed by *R. lignosus*			% of trees infected by *P. noxius*			% of trees killed by *P. noxius*		
			M	IQR	CI	M	IQR	CI	M	IQR	CI	M	IQR	CI
	SDI		10.08	2.35	0.52	4.47	1.09	0.24	4.22	1.62	0.36	1.46	0.77	0.17
a	1	50	7.72	1.58	0.35	3.57	0.97	0.21	3.25	1.09	0.24	1.21	0.77	0.17
a	1	100	5.69	1.58	0.35	2.52	0.81	0.18	2.27	0.65	0.14	0.97	0.48	0.10
a	2	50	6.91	1.95	0.43	3.08	0.93	0.20	2.84	1.26	0.27	0.97	0.48	0.10
a	2	100	3.73	1.54	0.34	1.78	0.77	0.17	1.62	0.48	0.10	0.81	0.65	0.14
a	3	50	6.26	1.30	0.28	2.68	0.97	0.21	2.43	1.05	0.23	0.89	0.65	0.14
a	3	100	3.25	1.30	0.28	1.46	0.48	0.10	1.30	0.93	0.20	0.65	0.65	0.14
b	1	50	6.66	1.62	0.36	2.92	0.93	0.20	2.11	0.97	0.21	0.89	0.60	0.13
b	1	100	5.12	1.46	0.32	2.43	0.93	0.20	2.11	0.77	0.17	0.97	0.60	0.13
b	2	50	6.58	1.62	0.36	2.92	0.81	0.18	2.11	0.81	0.18	0.81	0.48	0.10
b	2	100	5.44	1.21	0.27	2.43	0.77	0.17	2.11	0.65	0.14	0.81	0.48	0.10
b	3	50	6.01	1.58	0.35	2.76	0.81	0.18	2.03	1.09	0.24	0.81	0.48	0.10
b	3	100	5.36	1.09	0.24	2.35	0.81	0.18	2.11	0.65	0.14	0.81	0.48	0.10
ab	1	50	5.85	1.54	0.34	2.60	0.81	0.18	2.11	0.65	0.14	0.81	0.60	0.13
ab	1	100	4.39	1.30	0.28	1.86	0.81	0.18	1.62	0.81	0.18	0.65	0.44	0.09
ab	2	50	4.95	1.46	0.32	2.35	1.09	0.24	1.78	0.81	0.18	0.65	0.48	0.10
ab	2	100	3.73	1.54	0.34	1.78	0.77	0.17	1.62	0.48	0.10	0.81	0.65	0.14
ab	3	50	4.79	1.42	0.31	2.11	0.65	0.14	1.78	0.81	0.18	0.81	0.44	0.09
ab	3	100	3.25	1.30	0.28	1.46	0.48	0.10	1.30	0.93	0.20	0.65	0.65	0.14

Table 5. Simulated impact of various control methods on root-disease evolution with increasing periods of time (1.5–6 years) between disease detection on a given tree and treatment of its neighbours. The number of these treated healthy neighbours varies from 1 to 3, respectively; 1T: reference data, concerning simulation with only one lateral tree treated as soon as disease detection; 2T: two neighbouring treated trees; 3T: idem for three treated trees; SDI: Standard Disease Incidence, i.e. without any control-method application

Number of treated trees	Delay (years)	% of trees infected by *R. lignosus* M	IQR	CI	% of trees killed by *R. lignosus* M	IQR	CI	% of trees infected by *P. noxius* M	IQR	CI	% of trees killed by *P. noxius* M	IQR	CI
SDI		10.08	2.35	0.52	4.47	1.09	0.24	4.22	1.62	0.36	1.46	0.77	0.17
1T		1.39	1.30	0.28	1.87	0.81	0.18	1.63	0.81	0.18	0.65	0.44	0.10
2T		3.73	1.54	0.34	1.78	0.77	0.17	1.62	0.48	0.10	0.81	0.65	0.14
3T		3.25	1.30	0.28	1.46	0.48	0.10	1.30	0.93	0.20	0.65	0.65	0.13
1	1.5	7.31	1.86	0.41	3.33	1.09	0.24	2.76	1.26	0.27	1.30	0.60	0.17
1	3	8.94	1.46	0.32	3.98	0.97	0.21	3.41	1.09	0.24	1.46	0.77	0.14
1	6	10.24	1.78	0.39	4.71	1.13	0.25	3.73	1.30	0.28	1.46	0.65	0.14
2	1.5	6.99	1.58	0.35	3.25	1.26	0.27	2.92	1.26	0.27	1.46	0.65	0.16
2	3	8.69	1.86	0.41	4.06	1.30	0.28	3.41	1.46	0.32	1.38	0.73	0.13
2	6	10.16	2.56	0.56	4.55	0.81	0.18	4.06	1.13	0.25	1.46	0.60	0.13
3	1.5	6.99	1.91	0.42	3.17	1.09	0.24	2.76	1.13	0.25	1.46	0.97	0.21
3	3	8.78	2.56	0.56	3.82	1.26	0.27	3.25	1.09	0.24	1.30	0.77	0.17
3	6	9.26	1.99	0.44	4.22	1.46	0.32	3.73	1.54	0.34	1.46	0.73	0.16

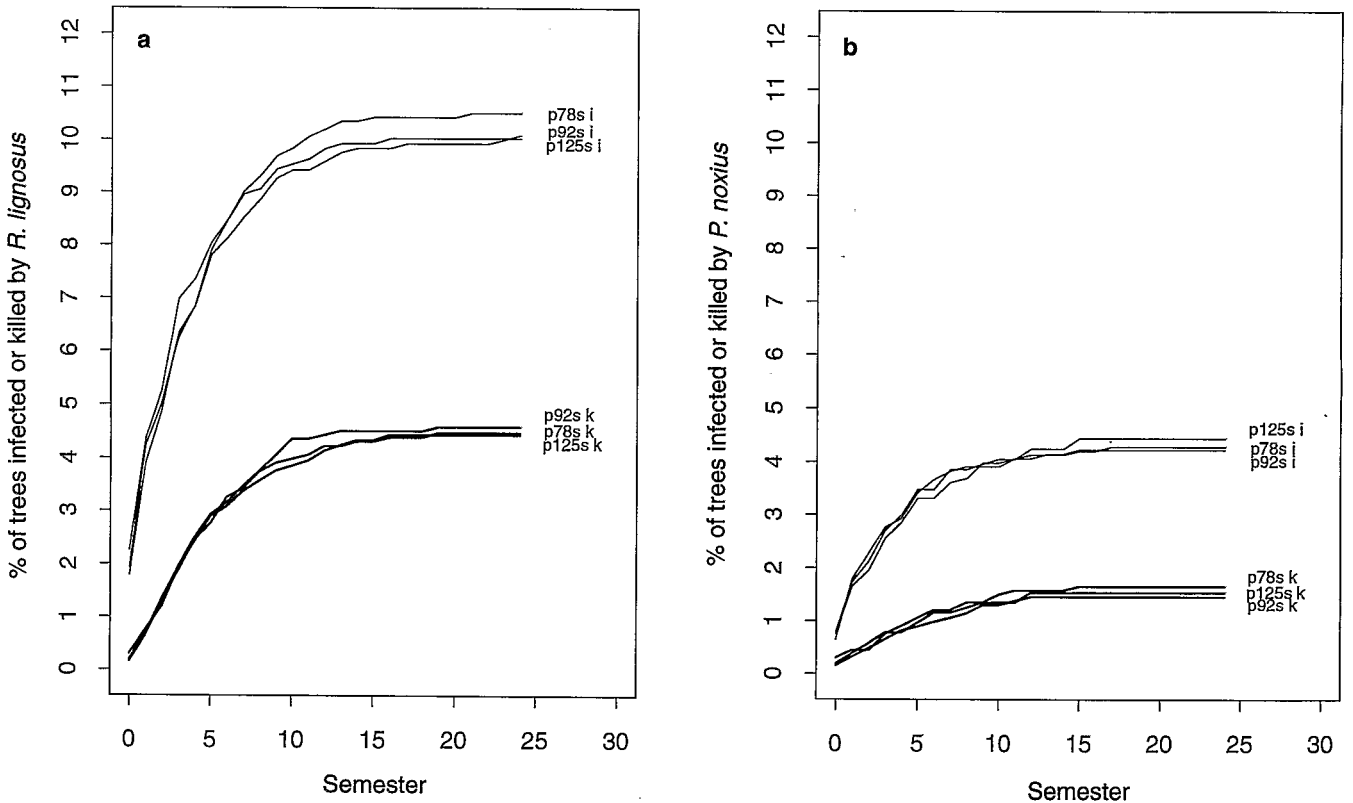

Fig. 3. Simulated dynamics of root-rot diseases based on the initial infestation including diseased, healthy or missing trees (i.e. infested trees dead before the beginning of the survey): a. Frequency (%) of infected (thin lines, code i) or killed (thick lines, code k) trees by *R. lignosus* (within the whole number of trees surveyed), respectively; b. Frequency (%) of infected (thin lines, code i) or killed (thick lines, code k) trees by *P. noxius*. 92r, 78r and 125r: stands studied

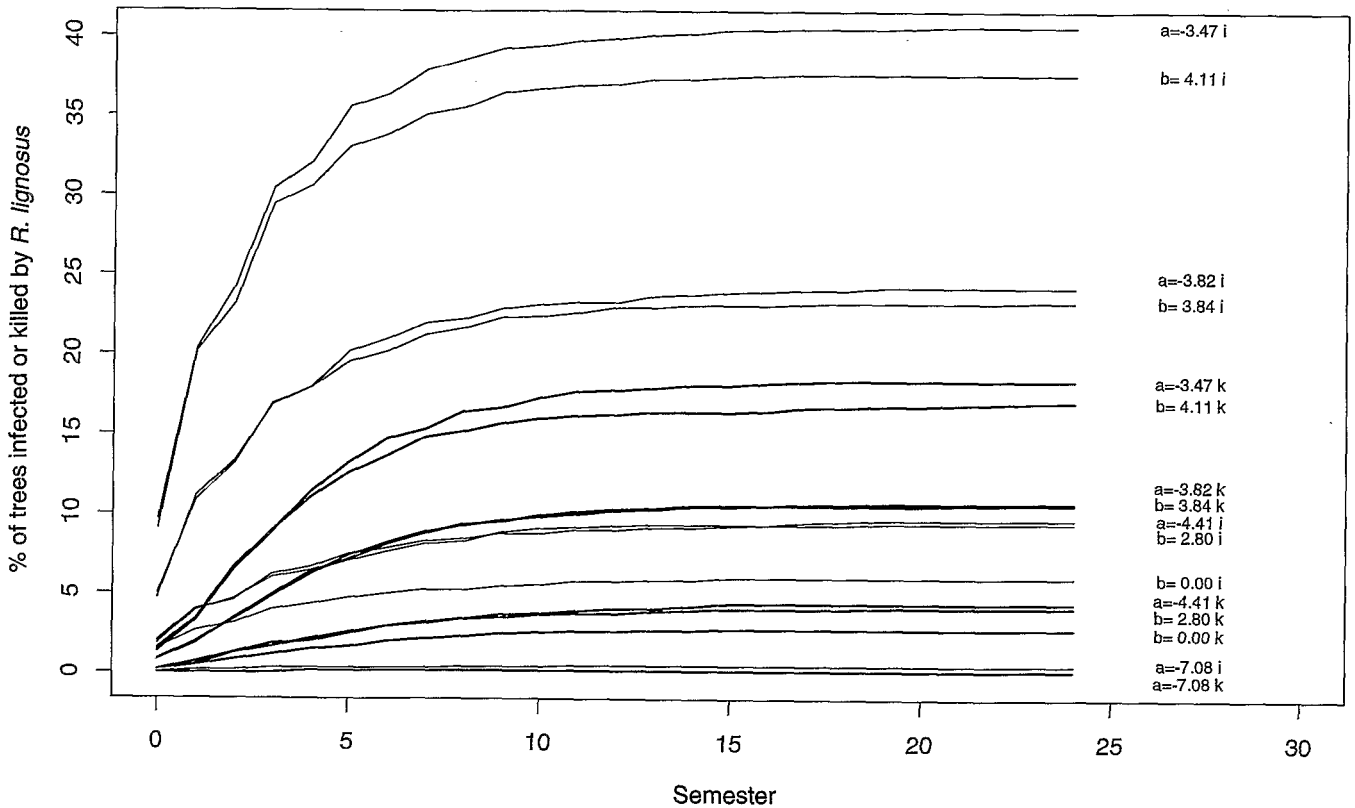

Fig. 4. Simulations of root-disease evolution vs. time, according to various levels of the parameters *a* or *b* describing the initial infection and the contagion risks, respectively: Frequency (%) of infected (thin lines, code i) or killed (thick lines, code k) trees by *R. lignosus* (within the whole number of trees surveyed)

that progressively merge. The number of centres and the accuracy of the averages obtained are therefore reduced as a result of fewer reference samples.

3.2.3 Related variation of parameters a and b

Figure 5 describes the curves obtained after simulating simultaneous variations of parameters a and b. They show the prominence of the initial infection over the secondary one. Varying the values of the b rate, a sizeable decrease in a brings the number of centres below a threshold, which stops the epidemic development. On the other hand, a simultaneous increase in both parameters ($a = -3.47$ and $b = 3.84$) would lead to an initial explosion of root diseases and their subsequent proliferation (Table 3). In this case, the infection and mortality rates were estimated at 74.3 and 32.6%. In the most serious cases ($a = -3.47 + b = 4.11$), these rates would reach 94.6 and 42.9%, respectively.

3.3 Simulating control-method applications

The results of simulating various types of control methods, degrees of efficiency expected for the treatment and numbers of treated neighbouring trees are given in Table 4.

3.3.1 Treatments with specific efficiency on a and b parameters

Consider first the case of a control method acting more specifically on the a parameter. On average, with a 100% efficiency rate, the treatment of one neighbour on both sides of a given infested tree would lead to a decrease of nearly half of the percentage of infected trees (Table 4). If this protection is extended to two lateral trees, the epidemic would be reduced in a significant way. The treatment of three lateral trees appears to be the most efficient as it reduces the progression of root disease by a factor of 2.8. These results apply to simulations with both *R. lignosus* and *P. noxius*.

By contrast, a control method acting more specifically on the b parameter would have quite different consequences. The treatment of a single tree on both sides of an infested one already forms a barrier that protects the neighbouring healthy trees. The systematic treatment of two or three neighbouring trees would not actually lead to better results.

The effectiveness of control methods acting on a and b would not be higher than the efficiency of a treatment on three neighbouring healthy trees that limit mainly the initial infection risk (Table 4). When taking into account financial costs, if only one tree can be treated, it appears to be better to act on the contagion risk. Indeed, treatments limiting the initial infection risk should actually be applied to all neighbouring trees to obtain maximum efficiency. However, in any case, the epidemic could not be totally stopped as new infections can arise in the segments of rows not already treated in the stand.

The effects of these control methods on infection and mortality rates appear to be quite similar. This suggests that the effect of fungicides on root-rotting fungi may be considered more preventative (by limiting the initial infection) than curative. In this case, it always remains very difficult to counter a pathogen located in the root system.

Due to the low initial inoculum rate, similar simulations with *P. noxius* lead to differences too small for interpretation.

3.3.2 Deferred treatments

The importance of the time factor in maximizing the efficiency of treatments applied to reduce the rate of pathogen infestation is enhanced by the simulation of deferred applications of the previous control methods (Table 5). Results correspond to treatments set up 1.5, 3

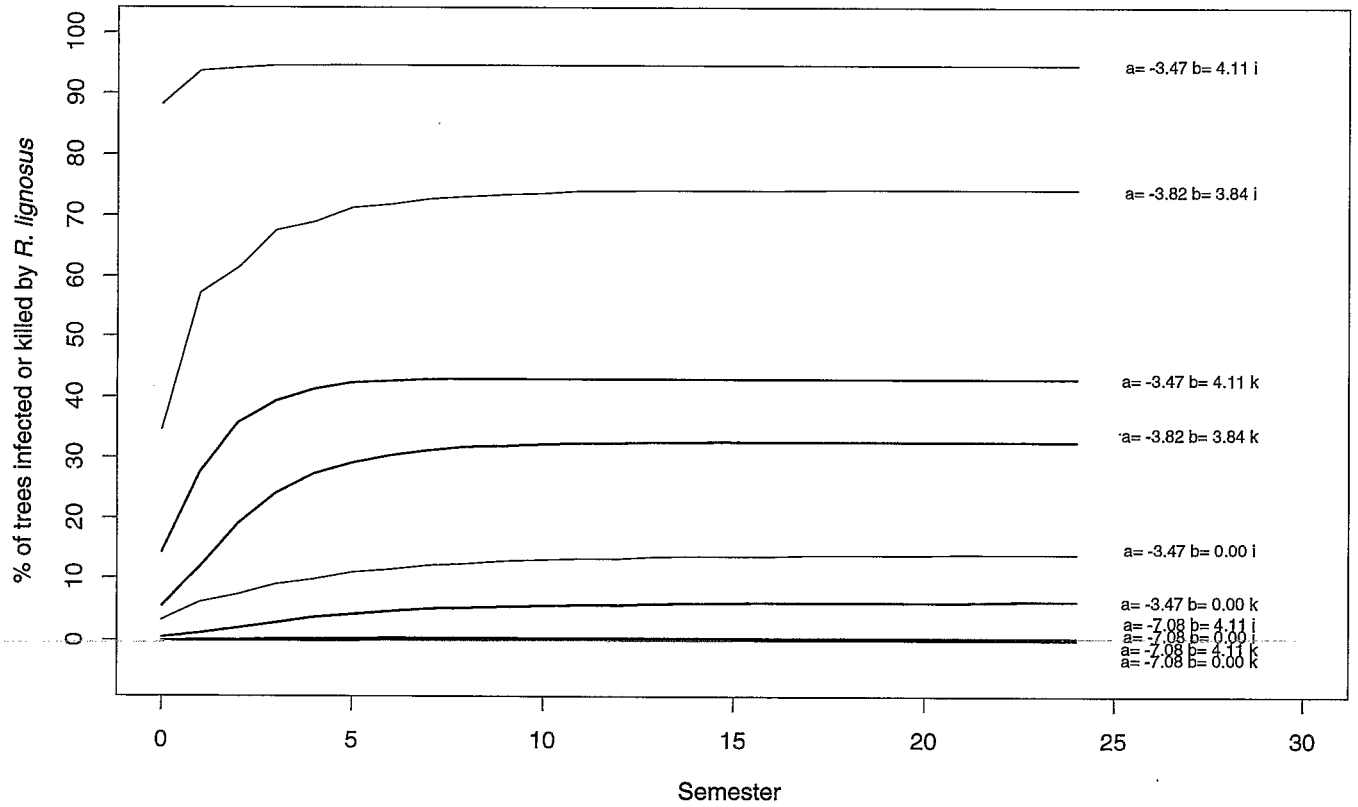

Fig. 5. Simulations of root-disease evolution vs. time, with simultaneous variations of both parameters *a* or *b* describing the initial infection and the contagion risks, respectively: Frequency (%) of infected (thin lines, code i) or killed (thick lines, code k) trees by *R. lignosus* (within the whole number of trees surveyed)

or 6 years after the beginning of the survey. They must be interpreted in association with the control (i.e. without any treatment) and with simulated treatments (100% efficiency rate), applied to one, two or three neighbouring trees (Table 5).

Some treatments used after 1.5 years indicate a definite effectiveness in halting the propagation of root diseases as they were applied before the stabilization of the epidemic. After this period, any delay in the application of these treatments would greatly reduce their efficiency, whatever the number of treated rubber trees. The impact on root-disease spread remains low compared with the untreated control, and always less effective than control methods used immediately after detecting new centres. These results reinforce the importance of the earliest possible intervention to stop the progression of root diseases at the emergence of the first sources.

4 Discussion

The general pattern of the theoretical curves obtained after simulation is quite similar to the observed kinetics of root diseases in this plantation (NANDRIS et al. 1988a) and also similar to other rubber-growing areas (RIGGENBACH 1960; MARTIN and DU PLESSIX 1969). These curves show a progressive reduction in fungal spread. According to FASSI (1964), this balance or latency stage is assumed to be a return to the endemic stage that prevailed in the forest prior to clearing.

For control-methods simulation, two efficiency levels (50 and 100%) were used to take into account the problems related to the quality of the applications *in situ* on a large scale. Until recently, many fungitoxic products have shown mediocre results in the countries of rubber-tree cultivation where they have been tested (SHARPLES 1936; RRIM 1974). The probability of deep infection of the taproot—here used as parameter a—can actually be reduced by using an injection device and some fumigating products such as Vapam (PICHEL 1956). Calixine and a few other products are currently recommended in Western Africa for the management of rubber-tree plantations (GOHET et al. 1991). They are used as a prophylactic fungicide for protecting healthy trees close to infected ones (i.e. parameter b).

The simulation of severe infestations gives the expected impact of root diseases, but similar conditions are still readily observed in many small-holder plantations that have been cleared manually without any forest-stump removal. Conversely, planting in low-infected sites or in sites carefully cleared may reduce the explosion phase of epidemics and thus limit cumulative rates of infection and mortality to less than 1%.

Simulating increasing rates of diseases made it possible to further investigate the potential economic implications of root-rotting agents. Consequently, the rate of survival of decayed trees must be considered in order to forecast the amount of residual inoculum at the end of the first cultural cycle (after 30 years). If no specific operations such as poisoning or removal of the old rubber stumps (HUTCHINSON 1961) are carried out at the beginning of the second cultivation cycle, the residual stumps would directly represent an infectious potential for the new rubber seedlings planted halfway from the initial rows. According to the various theoretical infestation levels used in this study, estimates of rubber tree losses and of residual inoculum rates may assist in rational determination of the type of control methods to be used before the replanting operations.

Many biological phenomena are involved in plant epidemics. Models of such complex processes mainly summarize their most important features. Consequently, they strongly depend on the objective of the model and the available knowledge of the epidemic (KRANZ 1974; GILLIGAN 1994). In forest pathology, several models attempt to describe these biological phenomena (BLOOMBERG et al. 1980; WILLIAMS and MARSDEN 1982; GILLIGAN 1983; SHAW et al. 1985; STAGE et al. 1990), generally by using deterministic functions that

concern the average trend. Often, they are based on a voluminous biological-data background (i.e. pathogen behaviour, surface of infecting sources, growing dynamics of the host etc.). These models, essentially deterministic, point out the key-steps of the epidemic processes. They can also be used to predict the mean epidemic spread, depending on edaphic or climatic conditions, as soon as their influence has been introduced into the model.

Such predictions mainly take into account the mean characteristics of the epidemic components, but broad individual variability, and more particularly spatial variability, is not really considered. Therefore, these predictions are more relevant when dealing with large areas where these random fluctuations will vanish. By contrast, for smaller-scale investigations such as with regard to industrial rubber-tree plantations, this variability cannot be ignored if the economic impact of the pests and the efficiency of treatments are concerned.

Moreover the present modelling presented here is characterized by a basic knowledge of the biology of the root pathogens, and by a large number of individual measurements. Accordingly, we chose to model the epidemic by a stochastic process where the main features of the epidemic are summarized in some statistical parameters, but where the spatial variability is explicitly taken into account. Such an approach was developed on purely spatial processes (NAVAS and GOULARD 1991) or spatio-temporal processes (BESAG 1977; DURRETT and NEUHAUSER 1990). It permits building of predictions of the pathogen spread, together with confidence intervals, and also allows evaluation of the influence of local treatments based on the spatial pattern of diseased trees. Consequently, the extrapolation of the model to other locations is more difficult. Indeed, the edaphic and climatic parameters were not explicitly taken into account, and their global effect on the pathogen spread were only observed and evaluated afterwards.

The model in this study basically rests on a reduction of the spatial structure of the stand to a juxtaposition of rows of trees considered as nearly 'independent' from the others; nevertheless, application of this model could be tested for the new planting patterns currently recommended in Malaysia (IYER 1985). Furthermore, the model has the advantage of being easy to implement. On the basis of field readings, only broad parameters were used to implicitly integrate the various biological steps of host–pathogen interactions. For example, parameter a (probability of a healthy tree of becoming infected while its neighbours are healthy) could be used to integrate the density of the infected woody masses buried in the soil and their proximity to the taproots of young rubber trees. This information constitutes a background for the micro-environment features that are not available to an observer at the beginning of a survey. Consequently, the low number of model parameters facilitated modelling and, as far as methodology is concerned, should constitute an asset for applications in other plantations. The simplifications of the data needed for modelling were assisted by several specificities of this disease survey. Rubber-tree plantations are a single-species crop, set up according to a strict layout, and easily accessible throughout the year. Within such plots, it is easy to map tree by tree and to conduct frequent epidemiological observations. This forest-pathology study is therefore much more similar to investigations in research stations than to the working context of pathologists in large natural forests.

Acknowledgements

The authors are grateful to the three referees for their constructive comments.

Résumé

Modélisation des maladies racinaires de l'hévéa, simulations de différents taux d'inoculum et de plusieurs types de méthodes de lutte

Deux champignons phytopathogènes *Rigidoporus lignosus* et *Phellinus noxius* occasionnent des dégâts conséquents dans les plantations ivoiriennes d'hévéa (*Hevea brasiliensis*). Sur la base des résultats d'une étude épidémiologique menée en plantation industrielle pendant 6 années, a été réalisée une étude mathématique des processus spatio-temporels caractérisant le développement *in situ* de ces maladies du système racinaire. La modélisation des phases d'extension de la maladie et de colonisation du système racinaire des arbres infectés a débouché sur des simulations de la dynamique des épidémies. Les cinétiques théoriques obtenues indiquent à la fois, une variabilité intraspécifique (au sein des parcelles étudiées) ainsin qu'une amplitude de variation interspécifique en terme de dommages causés à la population d'hévéas. Sur la base du modèle mis au point et au travers de paramètres synthétiques, il a été possible de simuler différentes situations phytosanitaires mettant en cause les facteurs suivants: nature et quantité d'inoculum (primaire et secondaire), modes d'action de fongicides, degré d'efficacité des méthodes de lutte, calendrier d'application des traitements après la date de détection. Les caractéristiques du modèle utilisé, son usage potential pour gérer les problèmes santaires en plantation et certaines préconisations sont discutés.

Zusammenfassung

Modellierung von Wurzelfäulekrankheiten beim Gummibaum: Simulation unterschiedlicher Inokulummenge und Bekämpfungsmethoden

Die Wurzelfäuleerreger *Rigidoporus lignosus* und *Phellinus noxius* verursachen an der Elfenbeinküste große Schäden in Plantagen von *Hevea brasiliensis*. Basierend auf Beobachtungen, die über einen Zeitraum von sechs Jahren angestellt wurden, konnte mit mathematischen Methoden ein Modell der räumlichen Ausbreitung der beiden Pathogene und der durch diese bedingten Baummortalität entwickelt werden. Dieses Modell erlaubte die Simulation der quantitativen Entwicklung der Wurzelschäden in Abhängigkeit von der Zeit. Der theoretische Verlauf dieser Entwicklung zeigte bezüglich der beiden Erreger inter- und intraspezifische Variabilität und standortabhängige Unterschiede. Mit Hilfe dieses Modells wurden unter Einbezug der Menge an Inokulum, der Bekämpfungsmaßnahme, der Spezifität des Fungizids, der Wirksamkeit und des Zeitpunkts der Behandlung unterschiedliche phytosanitäre Szenarien simuliert. Der Einfluß dieser Faktoren auf die Dynamik der beiden Wurzelfäulekrankheiten wurde quantifiziert. Die Eigenschaften des Modells, seine Übertragbarkeit auf die Praxis und seine Aussagekraft über den zukünftigen Krankheitsverlauf und die Entwicklung der Plantagen werden diskutiert.

References

BESAG, J., 1974: Spatial interaction and the statistical analysis of lattice systems. J. Royal Stat. Soc. **B36,** 192–236.

–, 1977: Some methods of statistical analysis for spatial data. Bulletin of the International Statistical Institute **47,** 77–92.

BLOOMBERG, W. J.; CUMBERBIRCH, P. M.; WALLIS, G. W., 1980: A Ground Survey Method for Estimating Loss Caused by *Phellinus weirii* Root Rot. I. Development of Survey Design. Environment Canada, Forestry Service, Can. J. For. Serv. Rep. BC-R 3.

CHADOEUF, J.; JOANNES, H.; NANDRIS, D.; PIERRAT, J. C. 1988: Mathematical analysis and modelling of rubber tree root diseases: probability of infection of an individual tree. For. Science **34,** 831–845.

–; NANDRIS, D.; NICOLE, M.; GEIGER, J. P.; PIERRAT, J. C., 1992: Modélisation spatio-temporelle d'une épidémie par un processus de Gibbs: estimation et tests. Biometrics **48,** 1165–1175.

–; PIERRAT, J. C.; NANDRIS, D.; GEIGER, J. P.; NICOLE, M., 1993: Modelling Rubber Tree Root Disease Epidemics with a Markov Spatial Process. Forest Science **39,** 41–54.

CHAMBERS, J. M.; CLEVELAND, W. S.; KLEINER, B.; TUKEY, P. A., 1983: Graphical Methods for Data Analysis. Pacific Grove: Wadsworth and Brooks/Cole Publish. Comp. Advanced Books and Software.

DOBSON, A. J., 1983: An Introduction to Statistical Modelling. London: Chapman and Hall.

DURRETT, R.; NEUHAUSER, C., 1990: Epidemics with recovery in D = 2. Ann. Appl. Probab. **20,** 1–15.

FASSI, B. 1964: Evolution du pourridié blanc, dû au *Fomes lignosus* (Kl.) Bres., dans une Plantation de *Hevea brasiliensis* Aménagée Immédiatement après l'Abattage de la Forêt. Publ. Inst. Nat. Et. Agr. du Congo, INEAC 105.

GEIGER, J. P.; NICOLE, M.; NANDRIS, D.; RIO, B., 1986: Root rot diseases of *Hevea brasiliensis*. I. Physiological and biochemical aspects of root aggression. Eur. J. For. Path. **16**, 22–37.

GILLIGAN, C. A. 1983: Modelling of soil-borne pathogens. Ann. Rev. Phytopath. **21**, 45–64.

–, 1994: Problems, solutions and analyses of non-linear models to disease progress and other curves. 7th Int. Workshop Epidemiology of Plant Diseases. Papendal Arnhem, 10–15 April, 1994. p. 78.

GOHET, E.; TRAN VAN CANH; LOUANCHI, M.; DESPREAUX, D., 1991: New developments in chemical control of white root disease of *Hevea brasiliensis* in Africa. Crop Protection **10**, 234–238.

HAHN, G. J.; SHAPIRO, S. S., 1967: Statistical Models in Engineering. New York: Wiley. pp. 83–91.

HUTCHINSON, F. W., 1961: Factors affecting root disease incidence and control in replantings. Proc. Nat. Rubb. Res. Conf., Kuala Lumpur **1960**, 483–495.

IYER, D. M., 1985: Spacings trials in plantation of *Hevea brasiliensis*. J. of the Rub. Res. Inst. of Malaysia **1985**, 28–35.

KRANZ, J., 1974: Comparison of epidemics. Ann. Rev. Phytopathol. **12**, 355–374.

KRICKEBERG K., 1975: Processus Ponctuels en Statistique. Ecole d'été de probabilité de St. Flour, X. Springer Lecture Notes in Mathematics. Berlin: Springer.

MUMFORD, J. D., 1987: Analysis of decision making in pest management. In: TENG, P. S. (ed.), Crop Loss Assessment and Pest Management. APS Press. pp. 201–208.

MARTIN, R.; DU PLESSIX, C. J., 1969: White root rot (*Leptoporus lignosus*) of rubber on lower Ivory Coast. J. Rubb. Res. Inst. Malaya **21**, 96–106.

NANDRIS, D., 1985: Pathogénèse et Epidémiologie des pourridiés de l'*Hevea brasiliensis*. Thèse Doctorat es Sciences, Univ. P. et M. Curie, Paris VI, 16 avril 1985.

–; CHADOEUF, J.; JOANNES, H.; PIERRAT, J. C.; GEIGER, J. P.; NICOLE, M., 1988b: Rubber root rot diseases: investigations on pathogen spread and tree mortality. In: Proc. 7th Int. Conf. IUFRO, WP S2-06, Victoria, Canada, 9–17 August, 1988. pp. 286–295.

–; NICOLE, M.; GEIGER, J. P., 1987: Root rot diseases of rubber trees. Plant Disease. **71**, 298–306.

–; –; –, 1988a: Root rot diseases of the rubber tree in the Ivory Coast. 1. Severity, dynamics, and characterization of epidemics. Can. J. For. Res. **18**, 1248–1254.

NAVAS, M. L.; GOULARD, M., 1991: Spatial Pattern of a Clonal Perennial Weed, *Rubia peregrina* (Rubiaceae) in Vineyards of Southern France. Journal of Applied Ecology **28**, 1118–1129.

NICOLE, M.; GEIGER, J. P.; NANDRIS, D., 1986: Root rot diseases of *Hevea brasiliensis*. II-Some host reactions. Eur. J. For. Path. **16**, 37–55.

PICHEL, R. J., 1956: Les Pourridiés de l'Hévéa dans la Cuvette Congolaise. INEAC Ser. Techn. 49.

PRUM, B., 1986: Processus sur un réseau et Mesures de Gibbs. Paris: Masson Ed.

RIGGENBACH, A., 1960: On *Fomes lignosus* (Klotzsch) Bres., the causative agent of the white root disease of the para rubber tree *Hevea brasiliensis* Muell. Arg. Phytopathol. Zeits **40**, 187–212.

RUBBER RESEARCH INSTITUTE OF MALAYSIA (RRIM), 1974: Root Diseases of Hevea. Planters Bulletin **133**, 109–120.

SHARPLES, A., 1936: Diseases and Pests of the Rubber Tree, London: MacMillan and Co.

SHAW, C. G., III; STAGE, A. R.; WEBB, T. M., 1985: Development of a root disease subroutine for use with stand growth models of western forests. In: Proc. 33rd Western Int. For. Disease Work Conf., 48–54.

SPITZER, F. L., 1973: Introduction aux processus de Markov à paramètres dans Zv. In: Ecole d'été de Probabilités de St. Flour III. Springer Lecture Notes in Mathematics. Berlin: Springer.

STAGE, A. R.; SHAW, C. G., III; MARSDEN, M. A.; BYLER, J. W.; RENNER, D. L.; EAV, B. B.; MCNAMEE, P. J.; SUTHERLAND, G. D.; WEBB, T. M., 1990: User's Manual for Western Root Disease Model. USDA Forest Service, Intermountain Research Station, Gen. Techn. Rep. INT-267.

VANDERPLANK, 1963: Plant Diseases: Epidemics and Control. New York: Academic Press.

WILLIAMS, R. E.; MARSDEN, M. E., 1982: Modelling probability of root disease centres occurrence in Northern Idaho forests. Can. J. For. Res. **12**, 876–882.

Authors' addresses: D. NANDRIS, Laboratoire de Phytopathologie, Centre ORSTOM, BP A5, 98848 Nouméa, New Caledonia; J. CHADŒUF, Laboratoire de Biométrie, INRA Domaine St. Paul, F-84140 Montfavet, France; J. C. PIERRAT, Laboratoire de Sciences Forestières, INRA/ENGREF, 14 Rue Girardet, F-54000 Nancy, France; H. JOANNES, Station de Biométrie, CNRF Champenoux, F-54280 Seichamps, France; M. NICOLE et J. P. GEIGER, Laboratoire de Phytopathologie, Centre ORSTOM, BP 5045, F-34032 Montpellier, France

Received: 26.1.1994; accepted: 3.11.1995