Forest Science, Vol. 39, No. 1, pp. 41–54

Modeling Rubber Tree Root Disease Epidemics with a Markov Spatial Process

J. CHADOEUF
J.C. PIERRAT
D. NANDRIS
J.P. GEIGER
M. NICOLE

ABSTRACT. Root diseases of the rubber tree (*Hevea Brasiliensis*) due to *Rigidoporus lignosus* (Klotzsch) Imaz. and *Phellinus noxius* (Corner) G.H. Cunn spread along planting lines and cause serious damage. Various control methods exist. A mathematical model was developed which allows the influence of their various parameters on the dynamics of an epidemic to be evaluated. The health status of the trees on a planting line was considered as a Markov process. The model parameters were given a biological interpretation and estimated using the results of an epidemiological survey carried out in Côte d'Ivoire from 1977 to 1984. Hypotheses of influence of dead trees before the survey, stationarity of secondary infection, and development of initial infection were tested. By simulation of the main model, the parameters were linked to percentages of infected trees on each inspection and of healthy trees at the end of the epidemic, thereby allowing the effects of the control methods to be evaluated according to their nature, efficiency, and mode of application. FOR. SCI. 39(1):41–54.

ADDITIONAL KEY WORDS. *Hevea brasiliensis, Phellinus noxius, Rigidoporus lignosus,* epidemiology, mathematical model, Gibbs, pathogen spread, spatio-temporal.

IN THE CÔTE D'IVOIRE RUBBER TREE INDUSTRIAL PLANTATIONS, root rot diseases due to *Rigidoporus lignosus* and *Phellinus noxius* cause serious damage, attacking up to 50% of the trees planted. The root rotting agents are spread in the soil by mycelial filaments developing from infected residual stumps of forest trees and/or woody debris remaining in the soil after clearing of the stand prior to planting. *R. lignosus* is characterized by fast-growing mycelial filaments, i.e., rhizomorphs, and *P. noxius* by a slow-growing mycelial sleeve surrounding the root of the infected tree. During the spread phase, the mycelium runs along the lateral roots to reach the taproot. Then, the root penetration and rotting process begin. Once the infected tree has decayed, it in turn becomes an inoculum causing the infection center to enlarge by progressive infection of neighboring rubber trees. To reduce the consequent losses, it is now possible to decrease the amount of inoculum in the soil after clearing the forest prior to planting, or to protect the trees with fungicide.

Although the efficiency of a treatment may easily be measured for a given tree, at present it is experimentally impossible, particularly over a long time period, to

quantify its repercussions on the overall dynamics of the epidemic in the plantation. Up to the last 10 yr, most of modeling studies quantifying the dynamic process of epidemics in plants have dealt with pathogenic fungi of aboveground organs, especially in foliar diseases (Zadocks and Schein 1979), whereas few investigations have attempted to model root diseases due to soil-borne pathogens in general and to root rot fungi in particular. With new approaches to soil-borne pathogen epidemiology in temperate countries and particularly to forest pathology (Williams and Marsden 1982, Bloomberg 1983, Gilligan 1983, Shaw et al. 1985, Stage et al. 1990), these types of investigation are becoming more common. We present here a mathematical model of how the epidemic spreads in order to estimate the theoretical efficiency of various treatments by simulation. The model is based first of all on data concerning various biological aspects of these pathogens and their interactions with rubber trees (Nandris 1983, Nandris 1987, Geiger 1986, Nicole 1986), acquired during an epidemiological study carried out from 1977 to 1984 (Nandris 1988), and secondly on a recent study of probabilities of tree attack (Chadoeuf et al. 1988). In the latter, it was pointed out that the infection risk for a tree within a given line did not depend on the adjacent lines: the pathogens spread along the planting line, not laterally (no interline spread). This allowed us to reduce the study of the stand to that of a single line. Our 1988 study was based on a decomposition of the conditional probabilities similar to that in analysis of variance where, knowing the state of health of all trees in a given neighborhood, the conditional probability is the probability of finding a tree in a given state of health. It proved to be an effective method for testing various hypotheses on the spreading characteristics of the two pathogens. Being centered on the study of an individual tree, this method is easy to use but cannot, however, be used for studying the changes of a stand's health status without including some type of modeling logic that permits compilation of individual tree effects into stand effects.

In this paper, we propose a global model of the two pathogens' spread in the stands. According to the results of the previous paper, this model is defined using conditional probabilities, the neighborhood of a tree being restricted to the two nearest neighbors. This model can be seen as a Markovian spatial model at each date on the trees found healthy at the previous date. The parameters defined from the description proposed were given a biological interpretation. Parameter estimation was performed by maximum of pseudo-likelihood, allowing us to test various hypotheses on the pathogens' spread. The time course of these parameters was modeled. The model could thus be used to simulate changes within the stand, and to quantify the mean rates of pathogen spread and their variability. It was also shown how such a model could be used to quantify the effects of various treatments.

MATERIALS AND METHODS

Epidemiological Investigation

A population of 2,526 rubber trees 4 yr old at the beginning of the study was monitored for 4 yr. These trees, in 4 stands and taking part in a broader study, were inspected every 6 mo, at the end of the rainy and dry seasons, except for

the first two inspections, which were separated by 1 yr. Detecting root disease is particularly difficult because the pathogens develop underground, and because a decaying tree often remains without aboveground symptoms for a long time. The detection method adopted for this survey consisted in (1) looking for direct or indirect aboveground symptoms of infection, i.e., the presence of mycelium or sporophores on the lower part of the trunk, grooves on the trunk, distinct delay in growth, and wilting or discoloration of leaves, and (2) clearing the soil around the collar and taproot to look for and characterize the mycelium on that part of the root system which can be seen in the opening (30–40 cm radius and 10–20 cm deep); this is refilled after inspection. On each inspection, we recorded the newly infected trees by the following abbreviations: $x(i,t)$ (state of tree in position i along the line at inspection t) is denoted as follows:

0 for a healthy tree

m if the tree is found dead on the first inspection

L_j if the tree is found to have been infected by *R. lignosus* since inspection j

N_j if the tree is found to have been infected by *P. noxius* since j

When there is no ambiguity, we will set $x(i,t) = x_i$.

Statistical Model for a Planting Line: Markov Model

In a previous study on the determination of a tree's health status according to the state of its neighbors (Chadoeuf et al. 1988), it was pointed out that the health status of a given tree depends mostly on the state of its two nearest neighbors. If the latter is known, it makes sense to suppose independence from other trees: a tree located a certain distance beyond an inoculum is not influenced by it (the distance depends on how far the fungus rhizomorphs can travel without a woody substrate). This characteristic, which is typical of a Markovian process, makes it possible to base the model on conditional probabilities. First, the probability for a given tree to be in a certain state x_i, given the neighbors' states x_{i-1} and x_{i+1}, has been modeled (Kindermann and Snell 1980, Krickeberg 1980, Prum 1986, Ripley 1988) using two groups of parameters. Let $C(t)$ be the set of all possible new states at time t. A series of main parameters $a(t) = (a(y))_{y \in C(t)}$ determines the probability of a tree being in a given state if its neighbors are healthy:

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

with the convention $a(0) = 0$.

As an example, let us consider the case of a single disease, noted L, observed at $t = 0$. There is thus only one parameter which can be noted a and, if we ignore the notation t, the preceding definition becomes

$$P(x_i = L \mid x_j = 0, j \neq i) = \frac{\exp(a)}{1 + \exp(a)}$$

A second series of parameters, $b(t) = (b(u,v))_{u \in C(0), v \in C(0) \cup \ldots \cup C(t)}$, measures the increase of probability of being infected when the neighbors are infected, with

states u or v in comparison to the probability of being infected when the neighbors are healthy:

$$P\left(x(i,t) \middle| \begin{matrix} x(i-1,t) \\ x(i,t-1) = 0 \\ x(i+1,t) \end{matrix} \right) = \frac{\exp(a(x(i,t)) + b(x(i,t),x(i-1,t)) + b(x(i+1,t),x(i,t)))}{\sum_{y \in C(t)} \exp(a(y) + b(y,x(i-1,t)) + b(x(i+1,t),y))}$$

with $b(y,z) = 0$ if y or z is equal to 0.

Considering the example again, there is only one parameter, $b(x,y)$, different from 0; this will be noted b. Supposing that only one of the neighbors is healthy, the formula becomes

$$P(x_i = L \mid x_{i-1} = L, x_{i+1} = 0) = \frac{\exp(a+b)}{1 + \exp(a+b)}$$

If both neighbors are diseased, then it writes:

$$P(x_i = L \mid x_{i-1} = x_{i+1} = L) = \frac{\exp(a+2b)}{1 + \exp(a+2b)}$$

At time $t = 0$, the model includes three parameters: $a(m)$, $a(L_0)$, $a(N_0)$ and six parameters $b(x,y)$. For subsequent inspections, the model has two parameters $a(t)$ and $4t + 3$ parameters $b(t)$. Therefore, we have nine free parameters at time 0, and $4t + 5$ parameters at inspection t. All seven inspections pooled together, the most general model includes 135 free parameters.

The parameters $a(t)$ concern the initial infections of the young rubber trees' taproots due to hyphae spreading in the soil from a residual inoculum (stump or buried fragments). These diseased mature trees represent "autonomous" disease foci that can enlarge by mycelial spread from tree to tree. As pointed out by Nandris et al. (1988), pathogen spread through root contact between trees during the years of outbreak should be considered as Compound Interest Disease, since each newly diseased tree, as interest on "capital," would be included in the total amount of disease (Campbell and Powell 1980). Therefore, the initial phase could be considered as an incubation period (i.e., contact between decayed stumps and the roots of young rubber trees), as emphasized by Hirst and Schein (1965) and Jones (1978). Clearing the forest with great care prior to planting, i.e., eliminating woody waste, should influence this parameter. Considering that the increased probability of attack on a given tree when its neighbors are infected is mainly the result of pathogen spread from one tree to another, the parameters $b(t)$ describe the secondary infection or polycyclical development of the disease. A chemical treatment preventing the spread of hyphae or protecting its roots and collar will influence these parameters.

Second, the conditional probabilities defined before determine the law of probability for the various states of trees along a planting line (Krickeberg 1980). Let us set $\vec{X}(t) = (x(1,t),x(2,t), \ldots . x(n,t))$, the vector representing the stage of a segment with n trees, at inspection t. If, at inspection t-1, a segment is composed

of n healthy trees flanked by two trees in states $x(0,t-1)$ and $x(n+1,t-1)$, then

$$P\left(\vec{X}(t)=\vec{x}(t)\,\middle|\begin{matrix}y(0,t-1)\\ \vec{X}(t-1)=\vec{0}\\ x(n+1,t-1)\end{matrix}\right)=\frac{\exp\left(\sum_{i=1}^{n}a(t,x_i)+b(t,x_i,x_{i-1})\right)}{\sum_{z_i\in C_t}\exp\left(\sum_{i=1}^{n}a(t,z_i)+b(t,z_i,z_{i-1})\right)}$$

STATISTICAL ESTIMATION OF PARAMETERS

The Besag (1974) coding method was used. This method consists in keeping a maximum subset of the original lattice such that two trees of the subset are not neighbors. If the surroundings of the stands in question have been satisfactorily cleared, we assumed that a tree being at the edge of a stand would be equivalent to having a healthy tree as neighbor. We considered a set of 1263 "central" trees in which none of them was neighbor. Therefore, for a given date, t, each tree was conditionally independent of the others: the probability P_t of observing these trees simultaneously in a given health status when the state of their neighbors is known, is therefore equal to the product of the previous conditional probabilities

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

Finally, we considered the function of $F=\Pi_t P_t$; the estimates of the parameters are the parameters which maximize this function F as in the classical maximum of likelihood. Guyon (1985) has pointed out that the estimators obtained by this process are consistent and that tests similar to the likelihood ratio test can be performed using the pseudo-likelihood ratio. The function F and the probabilities used to define F look like those used in the General Log-linear model (Bishop et al. 1975), the health status of the central tree being the variable to be explained and the state of health of the neighbors being the explanatory variables. From a statistical standpoint however, our model greatly differs from the General Log-linear model in that the explanatory variables are also random. Moreover, the explanatory variables and the variable to be explained are not independent.

On one hand, we tried to test several biological hypotheses and, on the other, we adjusted the model by keeping only the relevant factors. We analyzed the influence of the trees missing before the survey, and the *R. lignosus–P. noxius* interaction. More precisely, we investigated whether both pathogens spread independently (if so, the parameters $b(t,L_i,N_j)$ are equal to zero), whether the presence of one influences that of the other in the vicinity (which means the $b(t,L_i,N_j)$ are positive), or whether the presence of one inhibits the other, leading to the development of pathogen-specific areas (which means negative parameters) and temporal stationarity of secondary infection. Therefore, after estimation of the more general model, including all the parameters, we then estimated the submodels resulting from removing the corresponding parameters and then, for each submodel, we tested it with the general model.

In addition, in order to describe the spread synthetically and, then, extrapolate, we modeled the parameters characterizing the initial infection and those related to the secondary infection as a function of time. We chose linear models because they are simple and fit well with the data. We thus obtain

$$a(L(t)) = a0(L) + ta1(L) + (-1)^t a2(L)$$

$$a(N(t)) = a0(N) + ta1(N) + (-1)^t a2(N).$$

Parameters $a0 = (a0(L), a0(N))$ represent the initial infections on the first inspection, $a1$ the linear decrease vs. time, and $a2$ the possible variations according to the season (rainy or dry).

The parameters concerning the secondary infection are as follows:

$$\begin{aligned} b(L(t),L(t')) &= b0(L,L) + (t - t')b1(L,L) \\ b(L(t),N(t')) &= b0(L,N) + (t - t')b1(L,N) \\ b(N(t),N(t')) &= b0(N,N) + (t - t')b1(N,N) \\ b(L(t),m) &= b0(L,m) + tb1(L,m) \\ b(N(t),m) &= b0(N,m) + tb2(N,m). \end{aligned}$$

As for the modeling of parameters $a(t)$, the possibility of a seasonal effect was eliminated, because the hypothesis of stationarity during the period of secondary infection was not rejected. Parameters $a0$, $a1$, $a2$, $b0$, and $b1$ were later on calculated and tested in comparison with zero.

Simulation of Health Status Changes within a Group (Population) of Trees

Aims of the Simulations

First, we simulated the health status changes of trees along one line with 2,500 and then 10,000 individuals from inspection 0 to inspection 20 in situations including trees being healthy, found dead on first inspection, or infected by both pathogens. Considering the state of the stands during the epidemiological survey, our model was validated by using the percentage of infected trees.

Second, we considered that the stands were subjected to a control method which decreased the initial infection (e.g., careful clearing before planting, or a protective treatment given to some trees and had no effect on the secondary infection). In the latter case, only the treated trees had a lower parameter *a*. Treatments with similar efficiency for both pathogens virtually decreasing this parameter by 30%, 50%, 70%, and 100% were considered and then applied to the trees surrounding the infected one of rank 1, 2, or 3. Third, we tested the impact of treatments on the secondary infection, e.g., trees flanking the infected trees were subjected to a special treatment, such as protective fungicides acting at the collar level, preventing the hyphae from settling down. We simulated the use of treatments which had similar modalities. Lastly, we considered a treatment acting on both parameters at the same time.

Statistical Aspects

A simulation was carried out for each inspection, distinguishing the first inspection from the following ones. Therefore, we generated pseudorandom uniform num-

bers (see Oakenfull, in McNeil 1979, Ripley 1987 for a detailed description). Let us denote

$$A(t) = \text{diagnoal matrix made of } \exp(a(u))_{u\in C(t)};\ \text{dimension } n \times n$$
$$B(t) = \text{matrix made of } bij = \exp(b(u,v))_{u,v\in C(t)};\ \text{dimension } n \times n$$
$$C(x0,t) = \text{diagonal matrix made of } \exp(b(xo,u))_{u\in C(t)};\ \text{dimension } n \times n$$
$$\mathbf{1} = (1, \ldots 1)'.$$

Let v and $R = (r_l)'$ be respectively the first eigenvalue and the first eigenvector of the matrix $A(0)B(0)$ and $M = (m_{lp})$ with $m_{lp} = b_{lp}a_{ll}r_p/vr_l$.

On the first inspection, the trees' health status on one line could be simulated using a Markov chain of transition matrix M (Spitzer 1973) when the number of trees in a line tends to infinity. The state of health of the trees is simulated sequentially from the first tree of the line to the last: the first tree is assumed to be healthy (since the lines are long enough, the statistics do not depend on the choice of the first tree's state); if tree number k is in state p, the probability of state l of tree $k + 1$ is m_{lp}.

During the following inspections, the line was composed of segments of healthy trees flanked by infected trees. We simulated the changes within these segments and the probabilities for each tree within a healthy segment to be in a given state on each inspection:

$$Pi(t) = \frac{C_{(x(i-1,t),t)}.(A(t)B(t))^{n-i}.C_{(x(n+1),t)}\mathbf{1}}{\mathbf{1}'C_{(x(i-1,t),t)}.(A(t)B(t))^{n-i}.C_{(x(n+1),t)}\mathbf{1}}$$

For the ith tree of a segment of length n, given the segment length, the states of the $i - 1$th tree and the $n + 1$th tree, the jth component of this vector gives the probability of being in stage j.

RESULTS AND DISCUSSION

Estimation of Parameters and Tests of Hypotheses

The most general model, including 135 free parameters, leads to a likelihood of 1134.45 for all seven inspections pooled together. As we shall see further on, this model will be simplified and hence we do not give any estimations of these parameters. First, we tested various biological hypotheses. These hypotheses do not naturally lead to nested models, so we preferred to test them by comparison with the most general model. Second, when modeling the parameters' temporal changes, we tested a series of nested models by comparing each model to the preceding model. Six hypotheses being tested, we performed each test with $\alpha = 0.008333$ (Miller 1981) so that the global risk is less than or equal to 5%.

Tests of the Various Hypotheses

Influence of the trees found dead on the first inspection. If such trees exerted no influence, there should be no increase in the attack probability for trees close to a dead one: hence, $b(L,m)$ and $b(N,m)$ are equal to zero for all the inspections. In this case, the model shows a likelihood of 1157.31, giving a 45.72 deviation with respect to the main model for 14 df. The χ^2 value for α of 0.00833

and 14 degrees of freedom is 29.72. Therefore, the hypothesis (no influence of the dead trees) was rejected.

Interaction between *R. lignosus* and *P. noxius*. If we consider that all the parameters $b(N,L)$ are equal to zero, meaning that the two pathogens represent two independently developing processes, the model's likelihood is 1204.7. We obtained a statistic of 70.3 for 49 df. The χ^2 value for $\alpha = 0.00833$ and 49 degrees of freedom is 75.1. Therefore, the noninteraction hypothesis was not rejected. Moreover, we noted that we were close to the 5% threshold on each inspection, whereas this threshold was not reached on the latest inspections, indicating a limited and decreasing dependence between the two pathogens. This may be due to the stand clearing method used (plant debris is collected and mixed), which may result in a substrate for both types of pathogen. If so, this effect will disappear with time, due to a progressive depletion of this inoculum.

Stationarity of the secondary infection. This hypothesis implies that the pathogenicity of the fungi does not decrease with time and that a healthy tree, whatever its age, always remains very susceptible to attacks. For example, a tree found attacked by *R. lignosus* on inspection 5 could constitute as much an inoculum (in terms of effectiveness against its neighbor) as a tree found infected on inspection 1: $b(L_6,L_5) = b(L_2,L_1)$. Therefore, the potential for infection of a diseased tree presents on the infection duration, not the date of attack. As in the case of host-pathogen interaction, this may result from the fact that the defense reactional capacity of a 5- or 9-yr-old tree does not affect the pathogen's ability to redifferentiate its rhizomorphs towards neighboring trees.

Mathematically, this hypothesis is equivalent to the equation $b(x,y) = b(u,v)$, where u and x denote the fact that a tree is attacked by the same pathogen after a time lag q, and v and y denote the neighboring tree is attacked by the same pathogen after the same time lag q. With a statistic of 80 and 109 df, the χ^2 value for α of 0.00833 and 109 degrees of freedom is 147.48, and this hypothesis cannot thus be rejected.

Temporal Modeling of Parameters

The linear model of the initial infection parameters was accepted since the value of the statistic is 5.66 and the χ^2 value for $\alpha = 0.00833$ and height degrees of freedom is 20.59. The linear model of secondary infection was also accepted with a statistic of 13.74 for 24 df; the χ^2 value for $\alpha = 0.00833$ and 24 degrees of freedom is 43.67. Furthermore, regarding the parameters of the previous model, we modeled the parameters of interaction with the trees missing before inspection, $b(L,m)$ and $b(N,m)$, by two constants. We then obtained a statistic of 35.6 for 12 df; the χ^2 value for $\alpha = 0.00833$ and 12 degrees of freedom in 26.77. However, since we cannot explain the deviation between the two models which appeared mainly during the third inspection, we will keep this model with 17 parameters. Analysis of the parameters (Table 1) revealed more significant initial infections for *R. lignosus* than for *P. noxius*, which means that $a0(L) > a0(N)$. The tendency as a function of time (parameter $a1$) was almost the same for *R. lignosus* as for *P. noxius*. On the other hand, the season strongly affected *R. lignosus* (it admits no 0 in its confidence interval), but did not affect *P. noxius*. The higher sensitivity of *R. lignosus* to climate may be responsible for differences between factors involved in pathogen spread. The parameters $a(t)$, which de-

TABLE 1.

Estimates and individual confidence intervals of the model parameters at the 95% level.

Parameter	Estimate	Upper bound	Lower bound
$a0(L)$	−4.38	−4.81	−4.00
$a1(L)$	−0.20	−0.30	−0.10
$a2(L)$	−0.33	−0.52	−0.14
$a0(N)$	−5.37	−6.06	−4.78
$a1(N)$	−0.19	−0.34	−0.03
$a2(N)$	−0.25	−0.54	0.03
$a0(M)$	−2.36	−2.59	−2.15
$b0(M,M)$	0.73	0.40	1.04
$b0(L,M)$	0.76	0.45	1.06
$b0(N,M)$	0.83	0.32	1.31
$b0(L,L)$	−0.21	−0.37	−0.06
$b1(L,L)$	2.86	2.46	3.29
$b0(N,N)$	−0.35	−0.68	−0.07
$b1(N,N)$	3.88	3.22	4.57
$b1(L,N)$	−0.19	−0.66	0.16
$b0(L,N)$	1.77	1.09	2.41
$b1(N,L)$	−0.12	−0.46	0.15

scribe the emergence of infection centers in noninfected areas, generally decreased with time due to the depletion of fungal strains.

The secondary infection, measured with parameters *b,* was higher for *P. noxius* than for *R. lignosus.* We therefore found a greater proportion of small centers with *R. lignosus* than with *P. noxius.* This is not immediately perceptible on the stand map because it is hidden by the greater number of trees infected by *R. lignosus* than by *P. noxius.* Similarly, slopes $b1(L,L)$ and $b1(N,N)$ are both negative; this indicates a decrease of the epidemic with time owing to the progressive depletion of the pathogens' infection potential in the root systems of the infected trees. Lastly, we also found the positive interaction between the two pathogens previously mentioned.

Treatment Simulation and Comparison

Simulation of Tree Lines in Survey Conditions

The estimates of attack percentages over 10 independent lines of 2,500 individuals are represented as a function of time in Figure 1. It can be seen that the random variations to which these percentages are subjected are relatively significant. The results of these simulations therefore mostly indicate tendencies. In order to stabilize the estimates, we then simulated lines of 10,000 individuals.

Figure 2 presents the values recorded in the field and the mean calculated values of the proportion of infected trees. In view of the great variability observed on the simulations presented in Figure 1, there was no significant discrepancy between simulated and observed values except for the percentage of trees attacked by *R. lignosus* on inspection 4. Moreover, a clear tendency for the infec-

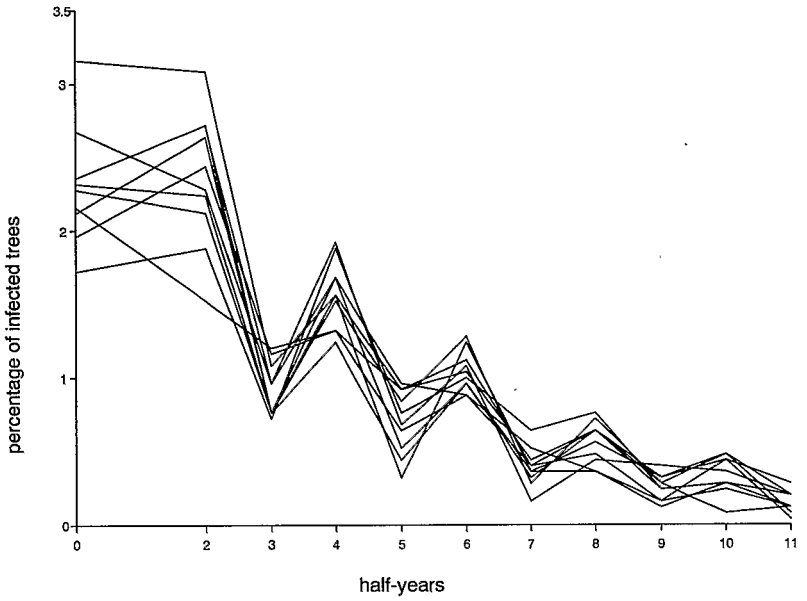

FIGURE 1. Variability of the simulated changes in number (in %) of rubber trees infected over time by *Rigidoporus lignosus*. Ten independent simulations of lines including 2,500 individuals were performed.

tion to stop was apparent. This slowing-down was due to the decrease of the initial infections (there were less and less new centers of infection) and the progressive decrease of the infection potential of infected trees (which reduced the extension of the disease centers).

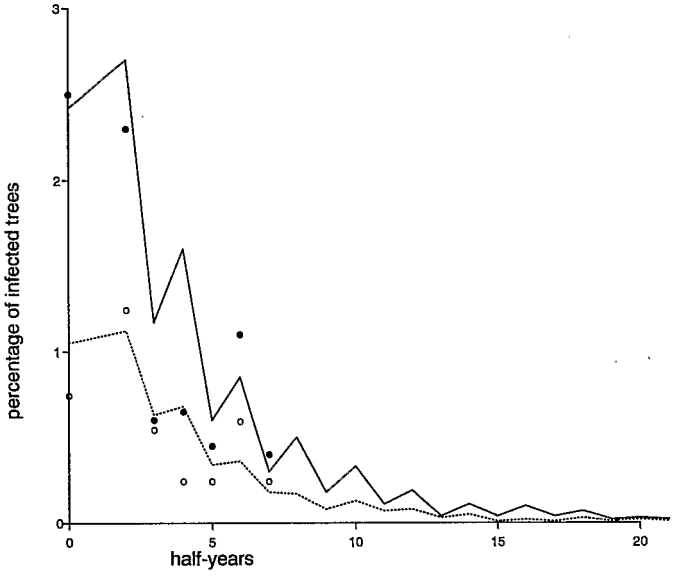

FIGURE 2. Simulation of the changes in number (in %) of trees infected over time by *Rigidoporus lignosus* and *Phellinus noxius*. ●: observed values for *Rigidoporus lignosus;* ○: observed values for *Phellinus noxius*.

Simulation of the Health Status Changes of Trees Submitted to Control Methods

Figures 3, 4, and 5 give the percentages of healthy trees on inspection 10, after 5 yr of treatment, according to efficiency of the following treatments:

- Treatment aiming at reducing initial infection (Figure 3): the curve increases as a function of the number of neighbors treated as well as of the treatment efficiency. It should be recalled that this treatment was applied only to trees neighboring infected ones. A more severe clearing would first result in lower parameter values for the trees in the stand, and second in increasing the number of healthy trees.
- Treatment aiming at reducing the secondary infection: a comparison of Figures 3 and 4 shows that this had a greater efficiency, i.e., increased number of healthy trees, than one acting on the initial infection. With highly efficient treatments the phenomenon was reversed. In fact, treatment efficiency against the secondary infection varied according to the health status of the neighbors: the effect increased with the number of infected neighbors.
- Treatment aiming at reducing both infections (Figure 5): the effects relative to each kind of parameter were not cumulative. Even a treatment 100% effective for the two types of parameters (a and b) did not stop the disease entirely. In a plantation, this would seem to result from the delayed emergence of new secondary centers outside the treated areas (3.3% for *R. lignosus,* 1.33% for *P. noxius*) in addition to the 10.8% of trees found dead on inspection 0.

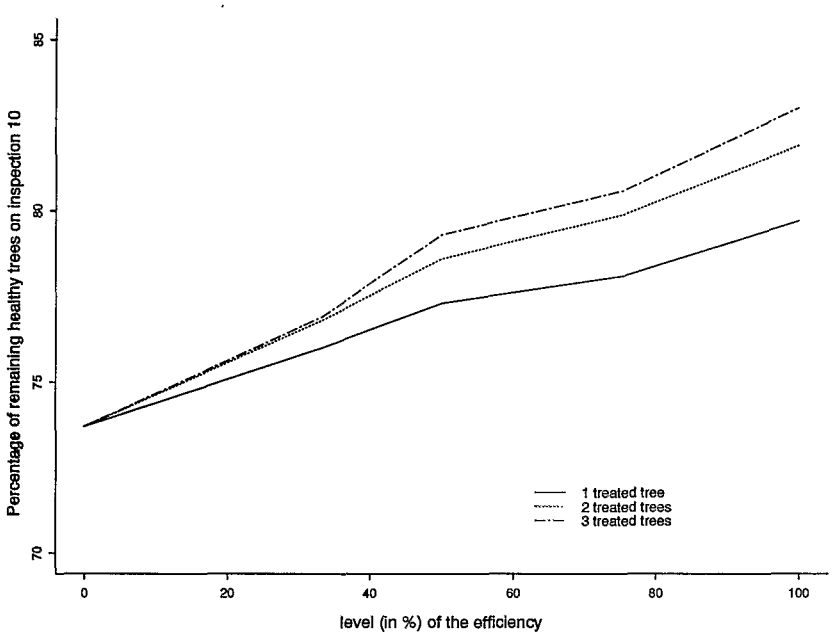

FIGURE 3. Simulated changes of root rot epidemics in plots where a control method acting on the initial infection had been used. The efficiency ranged through 0%, 33%, 50%, 66%, and 100%. The number of treated trees, except the infected rubber trees detected varied from 1 to 3. The number of remaining healthy trees was estimated on inspection 10.

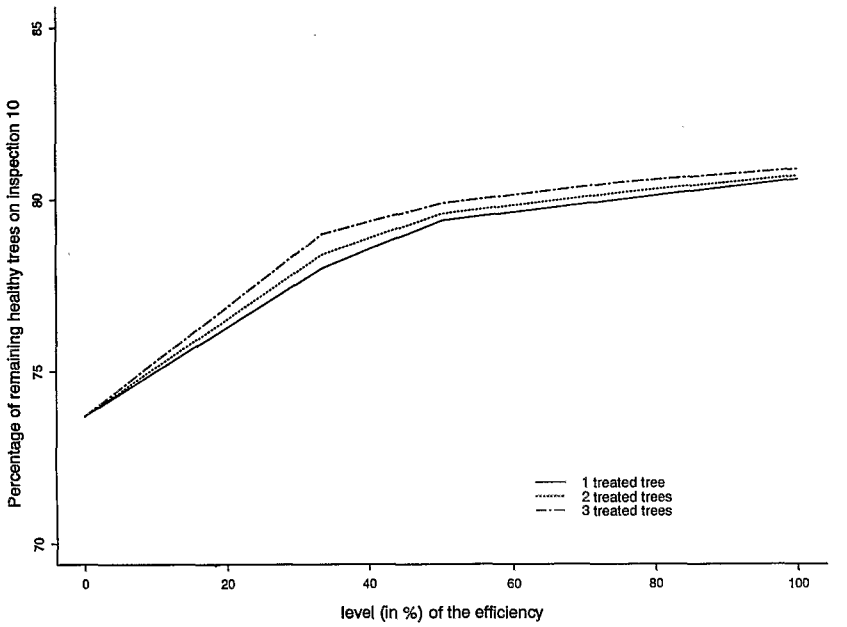

FIGURE 4. Simulated changes of root rot epidemics in plots where a control method acting on the secondary infection had been used. The efficiency ranged through 0%, 33%, 50%, 66%, and 100%. The number of treated trees except the infected rubber trees detected varied from 1 to 3. The number of remaining healthy trees was estimated on inspection 10.

CONCLUSIONS

The Markovian process allowed us to model epidemics and estimate the theoretical impact of various treatments according to their nature, efficiency, and mode of application. The parameters of local probabilities (or the probability of attack on a given tree, given the health status of its neighbors) were given a biological interpretation, which allowed for hypotheses to be tested on the influence of dead trees, time stationarity of the secondary infection, and development of the initial one. Moreover, by simulating the comprehensive model, these parameters were linked to the characteristics of the infection in the stands (percentage of infected trees on each inspection, percentage of healthy trees at the end of the infection), and the overall efficiency of the control methods was able be evaluated. Unlike the models of Williams and Marsden (1982), Bloomberg (1983), Gilligan (1983), Shaw et al. (1985), or Stage et al. (1990), which need a wide range of biological background, the present model is more a statistical model, the interest of which lies in hypothesis testing and prediction, than a deterministic one, the latter being used mainly to explain and incorporate biological phenomena. Our model, based on survey data, provides results which are, *a priori,* specific to this study. Further knowledge of the pathogens' spread characteristics according to soil and climate conditions would be necessary in order to extrapolate the model to other conditions. Such knowledge would in fact be worthwhile to be able to take climatic hazards into account. These methods are more particularly adapted to the study of industrial perennial crop plantations, where a lot of measured plants are available, but for which the biological background of the infecting pathogens is difficult to assess

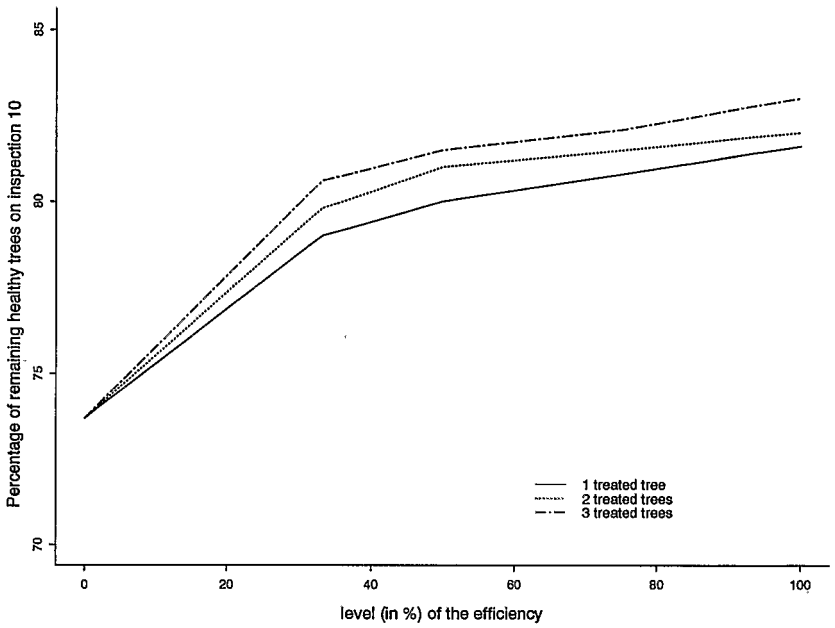

FIGURE 5. Simulated changes of root rot epidemics in plots where a control method acting simultaneously on the two types of infection had been used. The efficiency varied from 0%, 33%, 50%, 66%, and 100%. The number of treated trees, except the infected rubber trees detected varied from 1 to 3. The number of remaining healthy trees was estimated on inspection 10.

(especially, for root rotting agents). They allow to characterize the epidemic process through a few number of parameters, to test biological hypotheses concerning the primary and secondary infection, and at least to predict the evolution of the health status of a given stand together with its variability. Moreover, the parameters of such a model summarizing the infectious process can be used in order to compare various situations.

LITERATURE CITED

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

BISHOP, Y., S. FIENBERG, and P. HOLLAND. 1975. Discrete multivariate analysis. Theory and practice. MIT Press, Cambridge. 557 p.

BLOOMBERG, W.J. 1983. A ground survey method for estimating loss caused by *Phellinus weirii* root rot. III. Simulation of disease spread and impact (1–24). IV. Multiple-disease recording and stratification by infection intensity (1–16). Environ. Can., Can. For. Serv.

CAMPBELL, C.E., and N.T. POWELL. 1980. Progression of diseases induced by soilborne pathogens: Tobacco black shank. Prot. Ecol. 2:177–182.

CHADOEUF, J., H. JOANNES, D. NANDRIS, and J.C. PIERRAT. 1988. Mathematical analysis and modeling of epidemics of rubber tree root diseases: Probability of infection of an individual tree. For. Sci. 34(4):831–845.

GEIGER, J.P., M. NICOLE, D. NANDRIS, and B. RIO. 1986. Root rot diseases of *Hevea brasiliensis*. I. Physiological and biochemical aspects of host aggression. E.F.J.P. 16(1):22–37.

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

GUYON, X. 1985. Estimation d'un champ par pseudo-vraisemblance conditionnelle: étude asymptotique et application au cas markovien. Actes de la Sixième Rencontre Franco-Belge de Statisticiens. Nov 85. Editions des Facultés Universitaires de St-Loris, Bruxelles.

HIRST, J.M., and R.D. SCHEIN. 1965. Terminology of infection processes. Phytopathology 55:1157.

JONES, I.T. 1978. Components of adult plant resistance to powdery midew (*Erysiphe graminis* f.sp. *avenae*) in oats. Ann. Appl. Biol. 90:233–239.

KINDERMANN, R., and J.L. SNELL. 1980. Markov random fields and their applications. Am. Math. Soc., Providence, RI.

KRICKEBERG, K. 1980. Processus ponctuels en statistique. *In* Ecole d'été de probabilités de St Flour. X 1980. Springer Lecture Notes in Mathematics. 529 p.

MCNEIL, D.R. 1979. Interactive statistics. P. 17–37 *in* Proc. applied statistics conf. Sydney 8/9 February 1979. North Holland.

MILLER, R.G. 1981. Simultaneous statistical inference. Springer-Verlag. 299 p.

NANDRIS, D., M. NICOLE, and J.P. GEIGER. 1983. Infections artificielles de jeunes plants d'hévéa par *Rigidoporus lignosus* (Kl) Imaz et *Phellinus noxius* (Corner) G.H. Cunn. Eur. J. For. Pathol. 3(2):65–76.

NANDRIS, D., M. NICOLE, and J.P. GEIGER. 1987. Root rot diseases of rubber tree. Plant Dis. 71(4):298–306.

NANDRIS, D., M. NICOLE, and J.P. GEIGER. 1988. Root rot diseases of the rubber tree in the Ivory Coast. 1—Severity, dynamics and characterization of epidemics. Can. J. For. 18(10):1248–1254.

NICOLE, M., J.P. GEIGER, and D. NANDRIS. 1986. Root rot diseases of *Hevea Brasiliensis*. 2—Some hosts reactions. Eur. J. For. Pathol. 16(1):37–55.

PRUM, B. 1986. Processus sur un réseau et mesures de Gibbs. Masson. 190 p.

RIPLEY, B. 1987. Stochastic simulation. Wiley, New York. 237 p.

RIPLEY, B. 1988. Statistical inference for spatial processes. Cambridge University Press, Cambridge. 148 p.

SHAW, C.G. III, A.R. STAGE, and T.M. WEBB. 1985. Development of a root disease subroutine for use with stand growth models of western forests. P. 48-54 *in* Proc. 33rd Western Intern. For. Disease Work Conf.

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. 390 p.

STAGE, A.R., ET AL. 1990. User's manual for western root disease model. USDA Forest Service Gen. Tech. Rep. INT-267. 46 p.

WILLIAMS, R.E., and M.E. MARSDEN. 1982. Modeling probability of root disease centers occurrence in Northern Idaho forests. Can. J. For. Res. 12:876–882.

ZADOCKS, J.C., and R.D. SCHEIN. 1979. Epidemiology and plant disease management. Oxford University Press, New York. 427 p.

Manuscript received March 21, 1991

AUTHORS AND ACKNOWLEDGMENTS

J. Chadoeuf, Laboratoire de Biométrie, INRA, Domaine St Paul, 84000 Avignon, France; J.C. Pierrat, Laboratoire Science Forestière, INRA-ENGREF, 14 rue Girardet, 54000 Nancy, France; and D. Nandris, J.P. Geiger, and M. Nicole, Laboratoire de Phytopathologie Tropicale, ORSTOM, 911 avenue Agropolis, BP5045, 34032 Montpellier, France.