FORUM

Simulation Model for Phytomona Epidemics in Coconut Trees

Ricardo B. Sgrillo[1], José I.L. Moura[1] and Katia R.P.A. Sgrillo[2]

[1]Centro de Pesquisas do Cacau - CEPLAC - Rod. Ilhéus-Itabuna, km 22, 45650-000, Ilhéus, BA
[2]Depto. Ciências Biológicas - UESC - Rod. Ilhéus-Itabuna, km 16, 45650-000, Ilhéus, BA

Modelo de Simulação Para Epidemias de Fitomonas em Coqueiro

RESUMO - Este trabalho apresenta um modelo matemático determinístico compartimental do sistema composto pela planta hospedeira – coqueiro, o microorganismo patógeno – *Phytomonas staheli* McGhee & McGhee, e o inseto vetor – *Lincus lobuliger* Bred. (Hemiptera: Pentatomidae). Inicialmente um inseto sadio torna-se infectado após alimentar-se de uma planta infectada; conseqüentemente ele pode iniciar uma epidemia, disseminando *P. staheli* para plantas sadias. Esse processo inicia um crescimento exponencial da doença, que decresce conforme aumenta o numero de plantas infectadas. O programa Vensim DSS® foi utilizado para desenvolver um modelo de simulação da dinâmica da doença. Compartimentos para as plantas sadias, infectadas e erradicadas representam os componentes do sistema correspondentes ao hospedeiro. Existem dois sub modelos para a população do inseto vetor, onde os compartimentos representam os estágios de desenvolvimento (ovos, ninfas e adultos) das populações de vetores sadios e infectados, respectivamente. Os resultados da simulação foram comparados com dados de uma epidemia de fitomonas que teve inicio na Estação Experimental Lemos Maia (CEPLAC/CEPEC) localizada no município de Una, Bahia, Brasil. São apresentados resultados extensivos da análise da sensitividade do modelo às variáveis do sistema assim como resultados referentes à simulação da aplicação de diferentes métodos de controle. O modelo indica que a aplicação de técnicas de controle do vetor retarda a disseminação da doença, e que seria mais conveniente não aplicar técnicas de controle do *L. lobuliger* em áreas onde a doença não está ocorrendo.

PALAVRAS-CHAVE: Modelo matemático, inseto vetor, *Lincus*

ABSTRACT - A mathematical deterministic compartmental model of a system composed by the host plant – coconut palms, the pathogen microorganism – *Phytomonas staheli* McGhee & McGhee, and the insect vector – *Lincus lobuliger* Bred. (Hemiptera: Pentatomidae) was developed. A healthy insect becomes infected after feeding on a diseased tree starting an epidemic by disseminating *P. staheli* to healthy palms. This process initiates an exponential disease growth that decreases when the number of infected plants increases. The software Vensim DSS® was used as a tool to build a simulation model of disease dynamics. Compartments (stocks) for healthy, infected and eradicated plants represented the plant components of the system. There are two sub models for the insect vector population; the compartments represent each life stage of the healthy and infected vector population. The simulation outputs were compared with data from a recorded epidemic of *Phytomonas* that occurred at the Experimental Station Lemos Maia (Cocoa Research Center, CEPLAC/CEPEC), located at Una, Bahia. Here we present extensive data on the sensitivity analysis of the parameters and results from simulations of application of epidemics control methods. The model indicates that control techniques for the vector only delays the spread of the disease, and that it would be more convenient not to apply control techniques for *Lincus* sp. in areas where the disease is absent.

KEY WORDS: Mathematical model, insect vector, *Lincus*

In 2004, the State of Bahia produced more than 400,000 tons of coconut in 78,000 ha, at an approximate value of US$ 50 million (SEI 2002). Compared with other agricultural activities coconut profitability is high, especially in the southern coast of the state.

Phytomonas staheli (McGhee & McGhee) (Protozoa: Trypazomatidae) was detected in Brazil in 1982, in coconut trees in southern Bahia (Bezerra & Figueiredo 1982). The

parasite (Fig. 1) transmits hartroot (Fig. 2), a disease that causes plant death. Hartroot is so far a serious problem because its control is still unknown. Recently, hartroot was also detected in the States of Alagoas, Sergipe, Pernambuco, and Paraíba (Warwik *et al*. 1999).

Several hemipterans (Lygaeidae and Coreidae) have been reported as vectors of *Phytomonas* (Dolle 1984). Resende *et al.* (1986) recorded, for the first time in Brazil, the transmission of *P. staheli* to African palm oil trees, by *Lincus lobuliger* Bred. (Fig. 3), captured from diseased coconut trees. Although hartroot has been considered a serious constraint to coconut production, few studies on the vector, plant, and the disease interactions have been conducted. System Dynamics, firstly developed by Forrester (1961), offers a rather useful method to understand and describe such interactions.

System Dynamics was originally developed for the areas of engineering and administration, being increasingly applied to social, economic, chemical, biological, and ecological system analysis. A system is defined as a set of elements that interact continuously as a unit. System components and their relations and interactions form the structure of the system, which is dynamic and undergoes changes. System structure defines system behavior. The definition of the system structure (model) allows simulating its behavior in time, as function of different parameters. This work aims at developing and
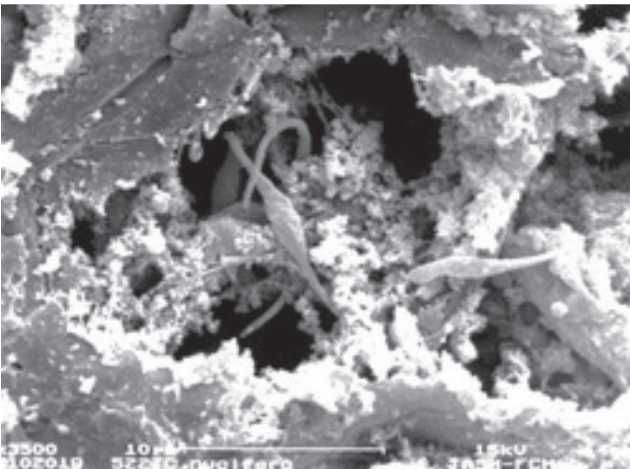

Figure 1. *P. staheli* in vegetal tissue. (x 3,500 - Photo by Flavio C. Miguens – UENF)

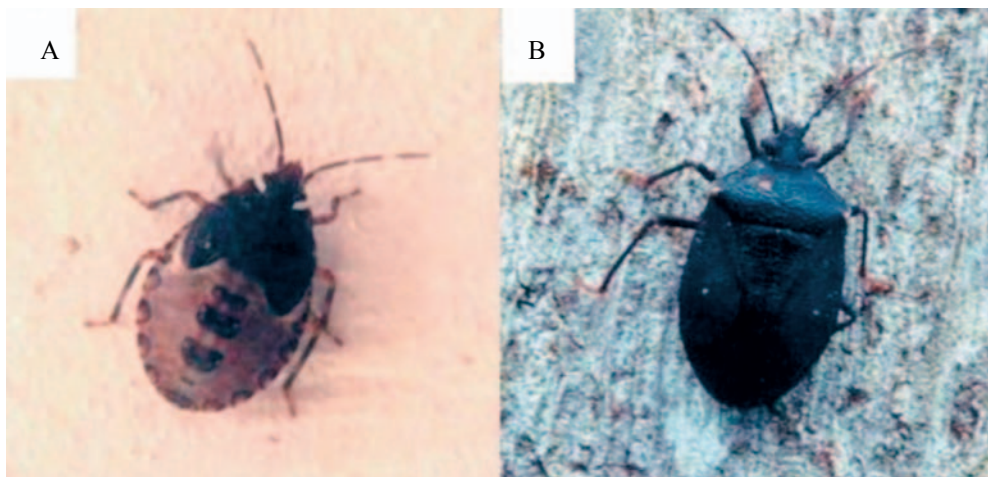Figure 2. Hartroot symptom in coconut tree. Una, BA, 2003

Figure 3. *L. lobuliger*. A. Nymph; B. Adult.

evaluating a model that represents a systemic structure composed by the coconut tree, *Phytomonas* and its vector, *L. lobuliger.*

The System. Although insects belonging to the genus *Lincus* have been considered vectors, the genus is not yet well known, and most of its species were described only after 1983 (Howard 2001). These insects can colonize hosts such as the coconut tree, African oil palm, banana tree, and several species of plants with unknown economic value.

On coconut trees, adults agglomerate on leaf axils, usually on the lower face of the petiole, and on the stipules, where they feed by sucking sap. Females normally lay eggs in line (Howard 2001). De Chenon (1984) recorded 16 to 18 eggs/ mass for *Lincus* sp. in Ecuador, and Louise *et al.* (1986) observed 7-9 eggs/mass in Guiana. The nymphal period varies from two months in *L. latifer* to five in *L. fumidifrons*. Adults of *L. latifer* live for approximately one month (Howard 2001). *L. lobuliger* is active at night, adults and nymphs being found on the ground after sunset (J.I.L. Moura, unpubl.).

The disease spreads in coconut trees after infected insects feed on healthy plants. Initially, the last two or three leaves turn brown (Fig. 2) and mid-sized fruits fall. As the disease progresses, partial or total fruit loss occurs; young leaves show yellowish and later brownish color; and necrosis of opened or closed inflorescences, and of immature leaves, take place. In the final stage, necrosis and rotting of the stem

apices and sometimes of roots occur (Moura *et al.* 2002). First symptoms appear four months after infestation (Resende *et al.* 1968), and time interval between first symptoms and plant death varies (Waters 1978).

Because the emerged nymphs feed on infected sap from the colonized plant, progeny will also be infected. The rate of newly infected plants is directly proportional to the number of infected vectors and healthy plants. Similarly, the rate of vectors that become infected is directly proportional to the number of healthy vectors and infected plants.

The diagram of causalities (Fig. 4) represents the basic structure of a system, where arrows show the cause '! effect relations. A positive sign indicates direct proportionality of cause and effect, and the negative sign indicates a relation of inverse proportionality.

The system is characterized by four main relationships: a) as the number of infected plants increases, the probability of encounters between healthy vectors and infected plants and the number of infected vectors increase, resulting in a higher rate of disease growth; b) as the epidemic advances, the number of healthy plants decreases, increasing the number of infected plants. As the number of healthy plants decreases, the probability of infected vectors finding healthy plants also decreases, thus reducing the rate of disease development; c) as the number of infected plants increases, so do the number of infected vectors, thus decreasing the
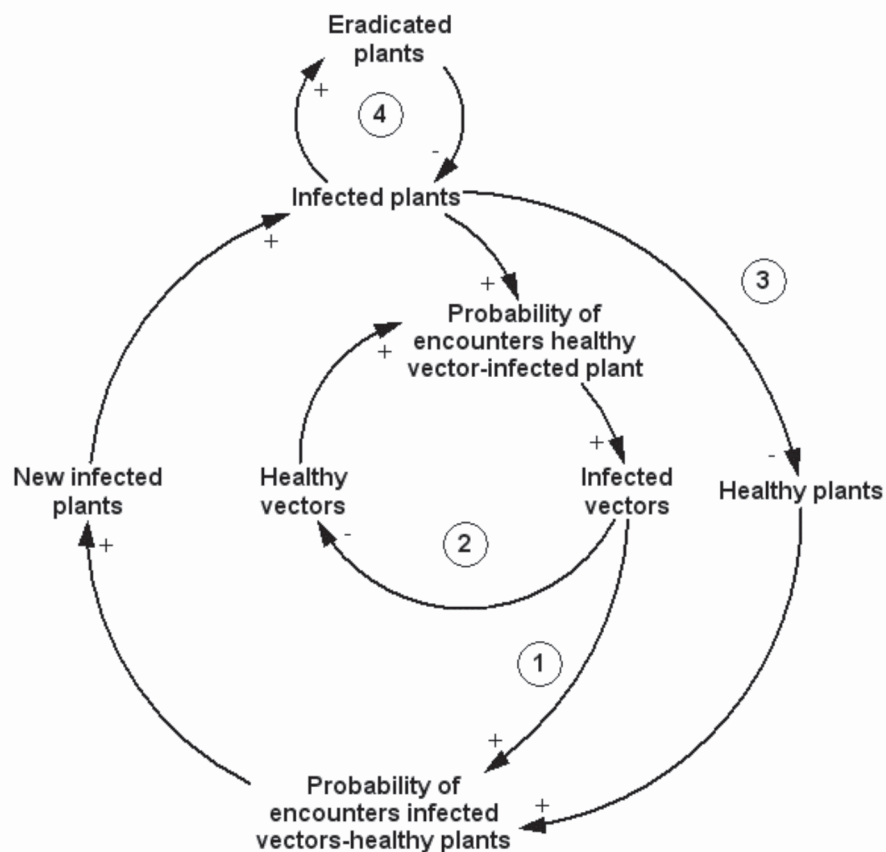

Figure 4. Diagram of causes of the system plant-vector-disease (see text for explanation).

number of healthy vectors. This reduces the probability of encounters of healthy vectors and infected plants, mitigating the progression rate of the epidemic; d) if infected plants are eliminated when disease symptoms are noticed, both the number of infected plants and the disease progress rate are reduced (Fig. 4).

This system presents an exponential increase (positive feedback) when the number of infected plants is small, followed by a phase of asymptotic increase (negative feedback) as the number of infected plants increases (feedbacks 2, 3, and 4).

Model Development

The methodology presented by Sgrillo & Araújo (1996) was adopted to develop this model, and the software Vensim DSS® version 5.3a, from Ventana Systems, Inc. (Ventana 2003) was used.

Two interrelated sub-models were developed: one for the plant, and one for healthy and infected vectors. Compartments that represent quantities regulated by in- and outflows compose each sub-model. Mathematically, the flows are represented by a system of differential equations that are numerically integrated. The coefficients of differential equations are calculated separately, at different

stages, by a set of algebraic equations. A Forrester diagram (Forrester 1961) of the model, showing only the most important variables, is shown in Fig. 5.

Plant Sub-Model. The plant sub-model has three compartments that represent, respectively, the numbers of healthy plants, of infected plants, and of eradicated plants. Healthy plants become infected through the rate of infection presented on equation 1:

$$\frac{dHealthyPlants}{dt} = -PInfecRate$$

where *HealthyPlants* (unit: plants) represents the total number of healthy plants in the area; *PInfecRate* (unit: plants/day) is the rate of plant infection or the number of healthy plants infected per unit of time. Rate calculations were based on the hypothesis that the vector (adult and nymph) moves randomly in the area and, in so doing, the probability of a vector to meet a host plant can be estimated by the ratio trunk area of a plant and total area; this ratio is named *Search Efficacy* (unit: m^2/m^2). The rate of infection is calculated by equation 2:

$$PInfecRate = \\ VetInfec \times (SearchEfficacy \times HealthyPlants) \times EficTransmVect$$
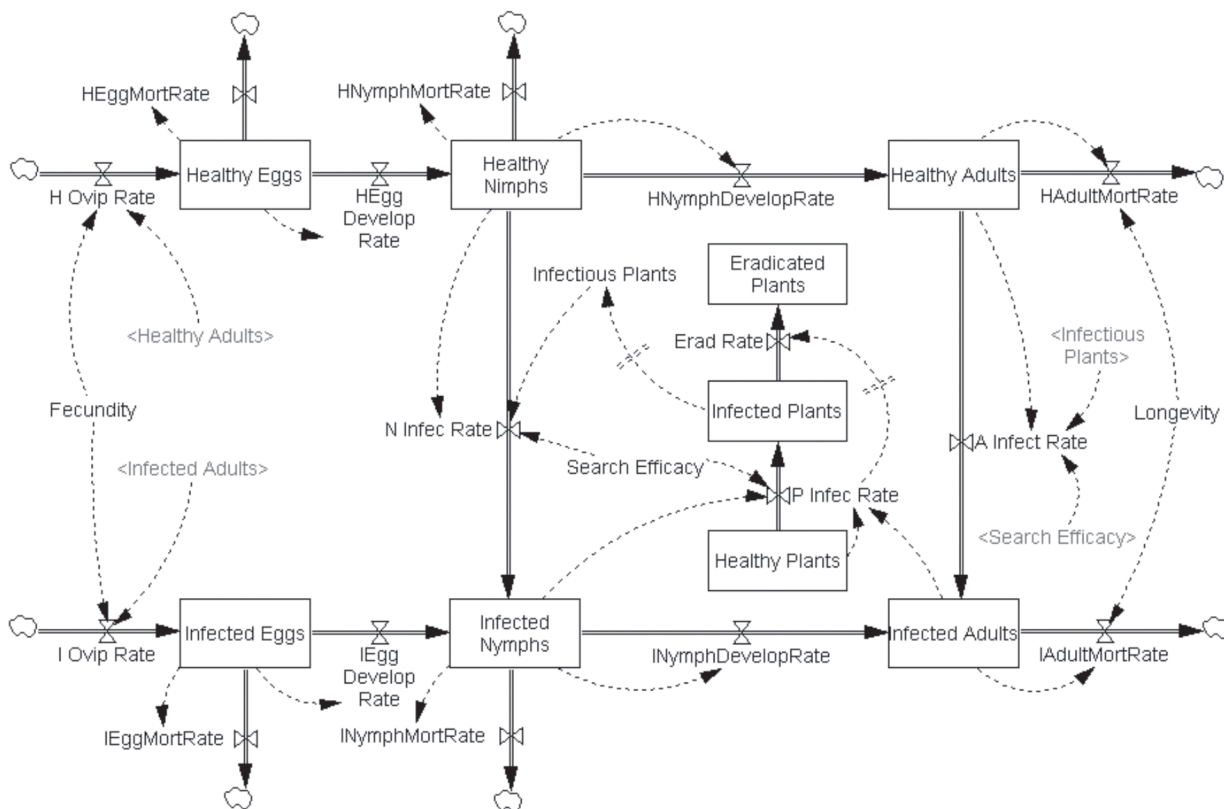

Figure 5. Simplified Forrester diagram of the model. Squares represent quantities (plants or vectors); double arrow and valve symbols represent flows among compartments; dotted lines show dependency relations among variables; and names outside symbols are some of the constants.

where *VetInfec* (unit: vectors) is the total number (adult and nymph) of infected vectors. The expression in parentheses represents the number of plants found per day by each vector. This expression, multiplied by the number of infected vectors represents the number of healthy plants found per day by all infected vectors. *EficTransmVect* (unit: 1/vector/day) is the proportion of healthy plants that become infected per unit of time, where 0.7 for the variable means that 70% of the healthy plants found each day by the infected vectors will be infected.

Variation in number of infected plants is calculated as difference between infection and eradication rates, according to equation 3:

$$\frac{dInfectedPlants}{dt} = PInfecRate - EradRate$$

where *InfectedPlants* (unit: plants) is the number of infected plants in the area and *EradRate* (unit: plants/day) is the rate of eradication, i.e., the number of eradicated (cut and burned) plants per unit of time. Plant eradication does not occur immediately after infection because the first symptoms are noticed 4-8 months later. The model presumes that plants will be eradicated as soon as disease symptoms are noticed. However, in practice, the incubation period in the model represents the time between plant infection and eradication. Thus, eradication rate will be equal to infection rate, with a delay equivalent to the incubation period, as in expression 4:

$$EradRate = \text{DELAYFIXED}(PInfecRate, IncubPeriod, 0)$$

where DELAYFIXED is a function of Vensim, which delays the input value (*PInfecRate*) for a period of time (*IncubPeriod*). The third variable of the function (0) is the value that will be given to *EradRate*, at the beginning of each simulation. Therefore, *IncubPeriod* (unit: days) represents the time needed for the outbreak of the first symptoms.

The diagram in Fig. 3 is a simplified representation of the real system. The number of infected vectors depends on the number of infected plants. However, a recently infected plant cannot immediately infect new vectors, and probably needs time for the pathogen to be distributed throughout the plant. Thus, a new variable is established representing the number of infectious plants, according to expression 5:

$$InfectiousPlants = \text{DELAYFIXED}(InfectedPlants, LatencyPeriod, 0)$$

where *InfectiousPlants* (unit: plants) is the number of plants with infecting capacity and *LatencyPeriod* (unit: days) is the time needed for an infected plant to infect healthy vectors.

The compartment of eradicated plants accumulates plants that were cut due to disease symptoms. This compartment has only one inflow, *EradRate*, calculated by equation 4 above.

Healthy Vector Sub-Model. The sub-model of healthy vectors has three compartments, representing numbers of eggs, nymphs and adults, respectively. Changes in egg numbers are represented by equation 6:

$$\frac{dHealthyEggs}{dt} = HOvipRate - HEggDevelopRate - HEggMortRate$$

where *HealthyEggs* (unit: vectors) represents the number of healthy eggs, *HOvipRate* (unit: vectors/day) the number of eggs laid per day (daily posture), *HEggDevelopRate* (unit: vectors/day) the rate of development of healthy eggs (number of eggs hatching per day), and *HEggMortRate* (unit: vectors/day), the daily egg mortality rate.

The oviposition rate is calculated according to equation 7:

$$HOvipRate = Fecundity \times \frac{HealthyAdults}{2} \times DensDepSurvRate$$

where *Fecundity* (unit: vector/vector/day) is the number of eggs laid per female per day (for both healthy and infected vectors), and *HealthyAdults* (unit: vectors) is the number of healthy adults, which is divided by two, assuming that 50% of the insects are females. *DensDepSurvRate* (no dimension unit) is the density-dependent survival rate, which regulates the maximum-total vector population size. Assuming a linear relation between population density and survival (theoretical logistic population growth), *DensDepSurvRate* will have a value of one (all eggs laid) when the population is minimal (zero), and value zero (no egg laid) when the population reaches the maximum value (maximum density). In the Vensim system, these values are shown in a table (*DensDepSurvRateTab),* and the values of *DensDepSurvRate* values are generated at each integration interval, as represented by expression 8:

$$DensDepSurvRate = DensDepSurvRateTab(VectorsbyPlant)$$

where *VectorsbyPlant* (unit: vector/plant) is obtained by dividing the total number of vectors (both healthy and infected nymphs and adults) by the total number of plants (healthy and infected); thus, population density is calculated continuously, and a survival value is generated by linear interpolation through the survival table. The use of vector/plant as measure of population density implies that the host plant is the only or main vector host. In equation 6, *HEggDevelopRate* (unit: vectors/day), which represents the number of nymphs emerging per day, is calculated considering a first degree delay, determined by dividing the number of eggs by the egg development period, following equation 9:

$$HEggDevelopRate = \frac{HealthyEggs \times \text{LN}(2)}{EggPeriod}$$

where *EggPeriod* (unit: days) is the egg development period of both healthy and infected vectors. Considering that equation 9 represents a delay of first order (exponential decrease), *EggPeriod* must be divided by the natural logarithm (LN) of 2, so that 50% of the population (*HealthyEggs*) can complete their development in the period, which is equivalent to multiplying *HealthyEggs* by LN(2), as shown by the equation above.

HEggMortRate, or daily mortality rate of healthy eggs, is calculated by multiplying the number of eggs by the proportion of daily mortality, according to equation 10:

$$HEggMortRate = HealthyEggs \times DailyEggMort$$

where *DailyEggMort* (unit: 1/day) is the proportion of healthy and infected eggs that die each day. This proportion is calculated based on the total egg mortality during the development period. Daily mortality is calculated according to equation 11:

$$DailyEggMort = 1 - \left((1 - EggMort)^{\frac{1}{EggPeriod}} \right)$$

where *EggMort* (dimensionless) is the proportion of constant mortality (inherent to the specie) at the egg stage. A value of 0.8, for example, means that 80% of the eggs cannot survive.

The compartment of healthy nymphs possesses one inflow and three outflows. Changes in this compartment are expressed in equation 12:

$$\frac{dHealthyNymphs}{dt} =$$
$$HEggDevelopRate - HNymphDevelopRate - HNymphMortRate - NymphInfecRate$$

where *HealthyNymphs* (unit: vectors) is the number of healthy nymphs. The inflow is given by the egg development rate, *HEggDevelopRate* (equation 9). From this compartment, the healthy nymphs will become healthy adults with a daily development rate *HNymphDevelopRate* (unit: vectors/day); the nymphs that die per day, are expressed by *HNymphMortRate* (unit: *vectors/day*). Healthy nymphs that are daily infected move to the infected nymph compartment by the rate *NymphInfecRate* (unit: vectors/day).

The daily development rate of nymphs to become adults, *HNymphDevelopRate* is calculated as egg development rate (equation 9), shown by equation 13:

$$HNymphDevelopRate = \frac{HealthyNymphs \times LN(2)}{NymphPeriod}$$

where *NymphPeriod* (unit: days) is the development period of healthy and infected nymphs. The use of LN(2) is explained in the comments of equation 9.

The healthy nymphs mortality parameter, *HnymphMortRate*, has three components: a) constant mortality, determined by the population genetic characteristics; b) variable mortality, which depends on population density, and c) mortality caused by occasional control techniques, such as insecticides, as expressed by the equation 14:

$$HNymphMortRate =$$
$$HealthyNymphs \times (1 - DensDepSurvRate) \times DailyNymphMort$$

where 1-*DensDepSurvRate* (equation 8) represents the mortality caused by factors that depend on population density, and *DailyNymphMort* (unit 1/day) is the daily mortality of healthy and infected nymphs, due to constant mortality and/ or mortality caused by control techniques, according to expression 15:

$$DailyNymphMort =$$
$$MAX\left(1 - \left((1 - NymphMort)^{\frac{1}{NymphPeriod}} \right), DailyCtrMort \right)$$

The function MAX has two arguments, separated by commas, and returns the argument with the highest numerical value. The first argument represents the constant daily mortality of both healthy and infected nymphs, and is calculated in a way similar to the constant egg mortality calculations (equation 11). *NymphMort* (dimensionless) is the constant nymph mortality during the development period; and *NymphPeriod* (unit: day) is the development period of healthy and infected nymphs. *DailyCtrMort* (unit: 1/day) is the daily mortality caused by control techniques, and is calculated by expression 16:

$$DailyCtrMort =$$
$$PULSETRAIN\ (StartD, Length, Interval, FinalD) \times \left(1 - \left((1 - EficCtr)^{\frac{1}{Lenght}} \right) \right)$$

The function PULSETRAIN normally returns to the value zero, but generates a repeated pulse with value one, beginning when simulation time is equal to *StartD,* during the time-units specified in *Length* and repeated at each *Interval,* until the time specified in *FinalD*. This function simulates an insecticide application. *StartD* represents the day of first application; *Length,* the period needed for the product to work; *Interval* represents the time between applications; and *FinalD,* the day when applications ceased.

In equation 16, the expression in parentheses, which multiplies the function PULSETRAIN, represents the daily mortality caused by the control technique, where *EficCtr* represents insecticide efficiency (mortality) or the proportion of population that will die during *Length*. *DailyCtrMort* will have zero value for no product application. In this case, *DailyNymphMort* (equation 15) will have its value determined by nymph constant mortality. Otherwise, *DailyNymphMort* will have the value specified by the insecticide efficiency.

In equation 12, the last outflow of the healthy nymph compartment represents the daily number of healthy nymphs infected, or *NymphInfecRate*. Calculating this variable is similar to calculating the rate of plant infection (equation 2), and is performed according to equation 17:

$$NymphInfecRate =$$
$$HealthyNymphs \times (SearchEfficacy \times InfectiousPlants) \times EficTransmPlants$$

As *SearchEfficacy* represents the probability of a vector to find a plant, the second term in parentheses (*Infectious Plants*) represents the number of infective plants found by each vector per day. This expression, when multiplied by *HealthyNymphs,* produces the total number of infective plants found by all healthy nymphs per day. *EficTransmPlants* (unit: 1/plant/day) is the proportion of healthy nymphs that meet infected plants and become infected by unit of time.

Variations in the healthy adult compartment are calculated by equation 18:

$$\frac{dHealthyAdults}{dt} =$$
$$HNymphDevelopRate - HAdultMortRate - AdultInfecRate$$

where *HealthyAdults* (unit: vectors) is the number of healthy adults; *HNymphDevelopRate* is the nymph to adult development rate, calculated by equation 13; *HAdultMortRate* (unit: vectors/day) is the adult daily mortality rate, calculated according to the equation 19:

$$HAdultMortRate =$$
$$HealthyAdults \times DailyCtrMort \times (1 - DensDepSurvRate) + \frac{HealthyAdults \times LN(2)}{AdLongevity}$$

where *DailyCtrMort* (daily mortality due to control) is calculated by equation 6 and *DensDepSurvRate* (survival dependent on population density) is calculated by equation 8. The last term of the equation incorporates mortality due to longevity of both healthy and infected adults *AdLongevity* (unit: days), calculated by a delay of first order. The use of LN(2) is explained in equation 9 (comments column).

    Healthy adult infection rate, *AdultInfecRate*, equation 18, is calculated as in equation 17, replacing *HealthyNymphs* for *HealthyAdults* (20)*:*

$$AdultInfecRate =$$
$$HealthyAdults \times (SearchEfficacy \times InfectiousPlants) \times EficTransmPlants$$

Infected Vector. The sub-model for infected vectors is practically equal to that for healthy vectors, except for the nymph and adult compartments. The healthy nymph compartments (see equation 12) consider an outflow corresponding to the infection rate of healthy nymphs (*NymphInfecRate)*. This is an inflow at the infected nymph compartment (21):

$$\frac{dInfectedNymphs}{dt} =$$
$$IEggDevelopRate - INymphDevelopRate - INymphMortRate + NymphInfecRate$$

    Similarly, *AdultInfecRate,* an outflow of the healthy adult compartment (see equation 18), becomes an inflow of the infected adult compartment (22):

$$\frac{dInfectedAdults}{dt} =$$
$$INymphDevelopRate - IAdultMortRate + AdultInfecRate$$

Model Evaluation

    To evaluate the model, data of a *Phytomona* epidemic on coconut trees were used. The epidemic started at the Experimental Station Lemos Maia (CEPLAC/CEPEC) in 1998, in Una county, Bahia, Brazil (15^0 17' S, 39^0 04' W). The regional climate is hot and humid without a defined dry season (Moura *et al*. unpubl.).

    The epidemic developed in 5 ha (1100 coconut plants) cultivated with cv. Anão-Verde-do-Brasil-de-Una, banana, cocoa, and cupuassu trees (*Theobroma grandiflorum* (Wild. ex Spring) Schumann). The Anão-Verde-do-Brasil-de-Una cultivar was introduced in 1987 and showed no symptoms of hartroot until 1997, when three plants were found infected. Thereafter, monthly inspections were conducted and plants with symptoms were eliminated. In 2002, insecticide (Chlorpyrifos) was applied to the soil every tree months.

    The model was evaluated by comparing real data (annual and accumulated number of eradicated plants) with data generated by applying the model. Because we did not aim at validating the model, statistical tests comparing the real and simulated results were not performed.

Constants. Simulations for model evaluation were conducted with constant values, as described bellow (Table1).

Model Exploration. Sensitivity analysis can help researchers to identify the most relevant variables and processes for the system, to establish the needed precision for estimation, and to understand the behavior of the real system. The analysis consists of changing the values of selected constants and evaluating changes caused on the model output, by this variation. A standard simulation was performed (Table 1), except for the control efficiency, which was fixed in zero, thus simulating natural disease development without human intervention. For each constant, we provided values of + and - 30%, 20%, and 10% in relation to values of the standard simulation.

    To measure the effect of these variations on model behavior, we used a disease progression rate (expressed as the reciprocal of the time needed for 50% of the plants to be eradicated). When the relationships between percentages of constant variation and of disease progression rate were linear, regression parameters between these variables were estimated, the regression coefficient being a measure of system sensitivity (sensitivity index). This parameter expresses the percent variation on the disease progression rate for every 1% variation in the value of the constant. Results of sensitivity analyses depend on the set of values used (Table 1). This analysis was performed by changing one parameter at a time and keeping the others constant, with their standard values. Because model behavior is the result of a set of interactions between constants, using different values generates differing sensitivity results.

Control Techniques. Simulations explored chemical control for vectors. Biological control and reduction of host access were evaluated through sensitivity analyses.

Results

Model Evaluation. The real and simulated results referring to annual and accumulated numbers of eradicated plants (Table 2) are similar, as shown in Figs. 6 and 7.

    The simulated results showed a reducton in number of eradicated plants during part of the years 2002 and 2003, due to insecticide application. Although the simulated results repeated the real values, input values (Table 1) did not necessarily correspond to the real system parameters. In fact, any other set of self-compensating parameters, such as higher fecundity and mortality could lead to the same results. Even though the simulated results were similar to the real values, statistical validation was not performed due to lack of data for the model constants. However, the structure (interrelations) of the model properly describes the real system because it considers the basic principles of plant disease epidemiology (Van Der Plank 1963).

Model Exploration. Results of the sensitivity analysis are presented in Table 3 and in Fig. 8. There is a linear relation for

Table 1. Value of constants used for model evaluation

Constant	Value	Source/comment
First day of simulation	07/01/1997	Hypothetic value. Because three infected plants were found by the end of 1997, perhaps one infected vector had entered the area on July 1st of that year.
Initial number of healthy plants	1,100 plants	Real value
Initial number of infected plants	0 plant	Real value
Initial number of healthy vectors	1,000 adults	Hypothetic value
Initial number of infected vectors	1 adult	Hypothetic value (see first day of simulation)
Incubation period (period for symptoms to appear), *IncubPeriod*	120 days	Resende *et al*. (1986)
Latency period (period for the infected plant to became infective), *LatencyPeriod*	60 days	Hypothetic value. Bibliographic information not found.
Search efficiency (probability of a vector finding a plant in the area), *SearchEfficacy*	6E-6	Area of a coconut tree trunk with 31 cm diameter divided by the total area (50,000 m^2).
Infection efficiency from vector to plant *EficTransmVect*	0.7/vector/day	70% of the plants exposed to infected *L. lobuliger* presented symptoms (Resende *et al*. 1986).
Infection efficiency from plant to vector, *EficTransmPlants*	1/plant/day	We hypothesized that 100% of the vectors that feed on infective plants become infected.
Egg period, *EggPeriod*	5 days	Hypothetical. Bibliographic information on *L. lobuliger* not found.
Nymphal period, *NymphPeriod*	90 days	Hypothetical. Howard (2001) reports 60 days for *L. lathifer* and 150 days for *L. tumidifrons*.
Adult longevity, *AdLongevity*	30 days	Supposed. De Chenon (1984) reports 30 days as longevity for *L. lathifer* adults.
Fecundity, *Fecundity*	1 egg/female/day	Hypothetical. Bibliographic information not found.
Constant egg mortality, *EggMort*	0.8	Hypothetical. Bibliographic information not found.
Constant nymph mortality, *NymphMort*	0.6	Hypothetical. Bibliographic information not found.
Maximum population density (population density where fecundity and survival of eggs and nymphs have zero values)	5 vectors/plant	Hypothetical. Louise (1986) reports a 4.2 average (adults + nymphs of *Lincus* sp.) on the first three leaves of coconut in a French Guiana area. Resende *et al*. (1986) presents average of nearly three vectors by healthy plant.
First day of chemical treatment, *StartD*	02/01/2002	Real value
Interval between treatments, *Interva*	90 days	Real value
Period of insecticide effect, *Length*	30 days	Hypothetical
Effectiveness of insecticide *EficCtr*	0.99	Hypothetical. Means that in 30 days the insecticide kills 99% of nymph and adult vector population.
Number of insecticide applications	4	Real value

search efficiency among variables. Model sensitivity to search efficacy has a sensitivity index higher than one (1.62), meaning that errors in estimating this variable are amplified. Because this is one of the most important parameters for determining system behavior, estimation should be precise. However, because estimation requires a complex experimental process, its value could be achieved by calibration.

The transmission efficiency effect of an infected vector to plants, on the model behavior, is similar to the search efficacy effect, but at lower intensity (sensitivity index of 0.87). Despite this, it should be precisely estimated because ca. 90% of

estimated error will be passed to the system behavior.

System response to the efficiency of plant transmission is similar to the efficiency of vector transmission to plants, but for plants the system presents a lower sensitivity index (0.74), i.e., 74% of the estimated error will be passed to the system.

Sensitivity rate was estimated at 0.66, where 1% decrease or increase in the incubation period would cause approximately 6.6% decrease or increase, respectively, of the disease progression rate. In practical terms, disease progression is delayed if infected plants are eradicated as soon as the first disease symptoms are identfied. A still

Table 2. Annual and cumulative number of coconut trees infected by *Phytomonas*. Real and simulated values. Una, BA, 1997-2002.

Year	N. of plants eradicated per year		Cumulative n. of plants eradicated	
	Real	Simulated	Real	Simulated
1997	3	0.5	3	0.8
1998	2	9.1	5	9.6
1999	18	20.3	23	29.8
2000	48	43.9	71	73.7
2001	78	78.1	149	151.9
2002	68	68.7	217	220.5
Mean	36.2	36.8	78.0	81.3

inexistent method for early diagnosis could delay the spreading of *Phytomonas* considerably.

The relationship between disease progression rate and latency period showed an inverse linear relation. As expected, a longer latency period requires more time for healthy vectors to be infected. Sensitivity to latency period is low (sensitivity index estimated ca. 0.08); therefore, estimating this variable does not need much accuracy because for every 10% error, only 0.8% variation will be introduced in the disease progression rate.

Variables Referring to Vector Biology. Results of the sensitivity analysis of variables for vector biology are presented in Table 4 and in Fig. 9. Sensitivity indexes were not calculated because variation in the disease progression rate shows a non-linear relationship, with value variation for most variables.

Nymphal Period. System sensitivity was high when the nymphal period was reduced, as compared to the standard simulation: a 30% period reduction caused a 72% reduction of the disease progress rate, whereas a 30% increase of the nymph development period caused an increase of ca. 32% in the disease progress rate. We expect that reducing the nymphal period will lower the disease progress rate, because nymphs will have less time to find diseased hosts and to be infected. Disease progression can be paralyzed when the nymphal period is very short, but develop faster when the period is longer. The latter, however, is an asymptotic relation.

Nymph and Egg Mortality. Variation in the disease progression rate is inversely proportional to variation in nymph mortality rate. As expected, high nymph mortality rates reduce the vector population size, thereby decreasing plant infection rate and slowing disease progress. Egg mortality affects disease progression rate slightly when its value is lower than the standard simulation value; as the value increases, the progression rate rapidly decreases.

Fecundity. Vector fecundity had minimal effect on the disease spreading rate, probably due to mortality factors dependent on population density. Apparently, fecundity is important to determine the speed the vector population reaches or returns to equilibrium (when the population decreases due to phytosanitary techniques, or when vectors
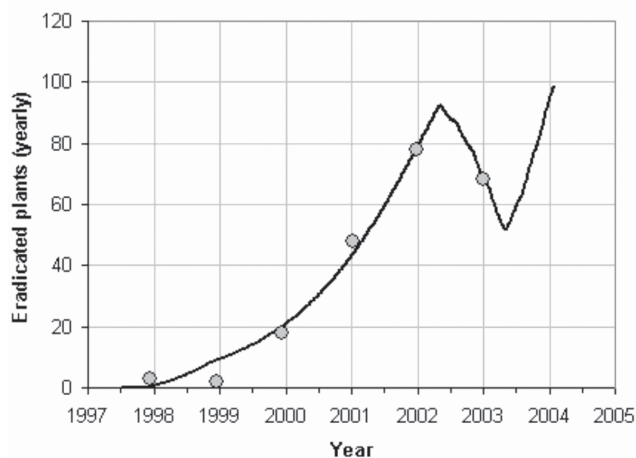
Figure 6. Real (dots) and simulated (lines) values of numbers of plants eradicated per year. Una, BA, Brazil, 1997-2002.
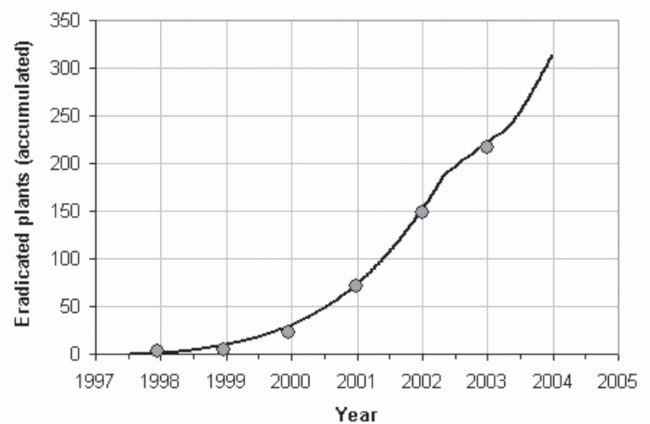
Figure 7. Real (dots) and simulated (lines) values of accumulated numbers of eradicated plants per year. Una, BA, Brazil, 1997-2002.

Table 3. Variation on the disease progression rate as a function of the variations of the constants: a) efficiency of search (*SearchEfficacy*, equation 2); b) efficiency of transmission from vector to plant (*EficTransmVect*, equation 2); c) efficiency of transmission from plant to vector (*EficTransmPlant*, equation 17); d) period of incubation (*IncubPeriod*, equation 4); and e) period of latency (*LatencyPeriod*, equation 5), and respective sensitivity index.

Variation on the constants (%)	Variation on the disease progression rate (%)				
	SearchEfficacy	*EficTransmVect*	*EficTransmPlant*	*IncubPeriod*	*LatencyPeriod*
-30	-45.37	-26.81	-23.87	-22.51	2.37
-20	-31.21	-18.23	-15.62	-14.51	1.57
-10	-16.10	-9.11	-7.75	-7.10	0.65
0	0.00	0.00	0.00	0.00	0.00
10	16.29	8.51	7.02	6.14	-1.02
20	33.15	16.99	14.08	11.94	-1.89
30	52.55	25.28	20.81	17.35	-2.51
Sensitivity index	1.62	0.87	0.74	0.66	-0.08

are introduced in a non-infected area). Nevertheless, according to the sensitivity analysis of maximum population density (see below), the dissemination rate seems to be proportional to the number of infected vectors in the population in equilibrium.

Longevity of Adults. We expected that, if adult longevity were high, population increase rate would also be high because females would lay eggs for a longer period of time, therefore speeding the dissemination of the disease. However, sensitivity data show that this did not occur and that as longevity increases, dissemination rate decreases.

Available information on the behavior of *Lincus* spp. show that both healthy and infected adults and progenies compete for the same resources, and because they have the same biological parameters, the preferred progeny will have the highest initial population. In standard simulation, the initial vector population presented one infected vector and 1000 healthy vectors (Table 1); higher longevity values for healthy

Figure 8. Variation (%) in disease progression rate, due to changes (%) in variables for the interactions plant-pathogen-vector.

populations reduce the disease dissemination rate. Thus, it does not seem convenient to keep the vector population under control whether the disease has or has not been found in the area. In this case, the disease progression rate is directly proportional to the vector longevity, as normally expected (Fig. 10).

Initial Population of Healthy Vectors. Disease progression rate increases exponentially as the number of healthy vectors in the area decreases when an infected vector is introduced (Fig.11). At a first glance, these data may not make sense, because the greater the number of healthy vectors in the area, the fastest the development of the disease, and therefore, the higher the number of infected vectors. However, the model structure comprises a survival factor that depends on the vector population density (see equation 8), and acts on both healthy and infected vectors (a larger population results in lower survival). Therefore, there might be a competition between healthy and infected populations, for environmental resources. Thus, if healthy vectors have a low initial population density, the infected vector population will increase faster because of the low competition for environmental resources, and the disease will spread faster. Therefore, control techniques for *Lincus* sp. should not be applied in areas where the disease is not occurring.

Maximum Population Density. The dissemination rate of *Phytomonas* is a linear function and directly proportional to the maximum population density; that is, the greater the number of vectors supported by the environment, the faster the disease will spread (Fig. 9).

Simulation of Control Techniques

Biological Control. Biological control agents should keep the population low and affect disease spread as the reduction of the maximum population density (Fig. 9), where a reduction of the disease dissemination rate is directly proportional to the reduction of the maximum population density.
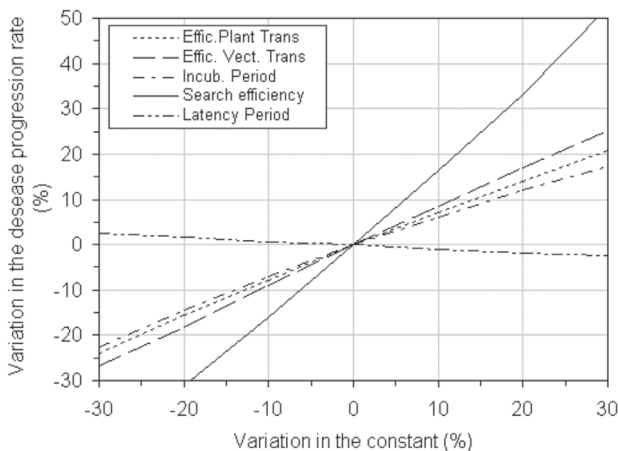
Table 4. Percent variation on the rate of disease progression related to variations on the nymphal period (*NymphPeriod*, equation 13), nymphs constant mortality (*NymphMort*, equation 15), eggs constant mortality (*EggMort*, equation 11), females' fecundity (*Fecundity*, equation 7), adults' longevity (*AdLongevity*, equation 19) and maximum population density (*MaxPopDen*, see equations 7 and 8).

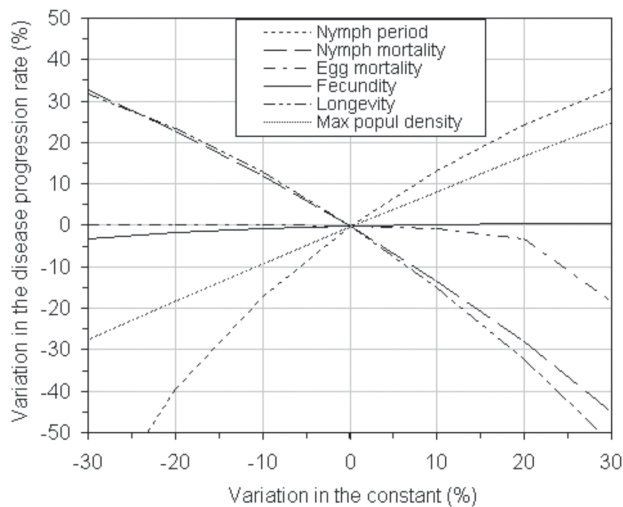| Variation on the Constants (%)	Variation on the rate o disease progression (%)					
	NymphPeriod	*NymphMort*	*EggMort*	*Fecundity*	*AdLongevity*	*MaxPopDen*
-30	-71.93	32.54	0.26	-3.11	31.86	-27.36
-20	-39.50	22.71	0.30	-1.64	23.30	-18.11
-10	-17.06	11.78	0.14	-0.64	12.75	-9.01
0	0.00	0.00	0.00	0.00	0.00	0.00
10	13.08	-13.36	-0.64	0.26	-15.16	8.36
20	24.28	-28.10	-3.23	0.39	-32.41	16.73
30	32.99	-44.94	-18.36	0.52	-52.62	24.84

Figure 9. Variation (%) in disease progression rate, as function of changes (%) in vector biology variables.

Figure 11. Variation (%) in disease progression rate, as function of changes (%) among initial populations of healthy adults.
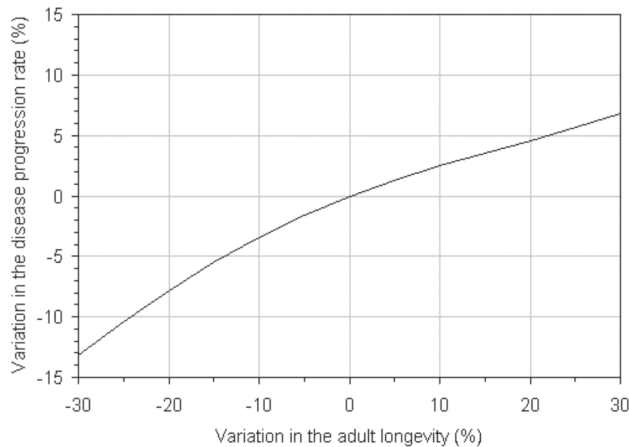

Figure 10. Variation (%) in disease progression rate, as function of changes (%) in adult longevity (initial populations = one infected and one healthy vector).
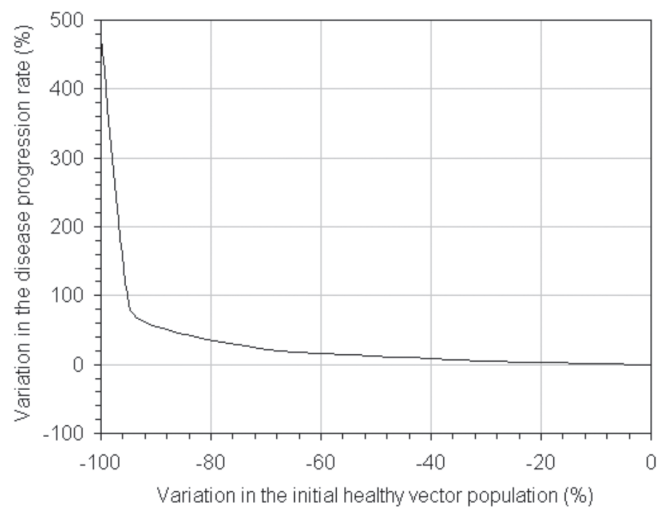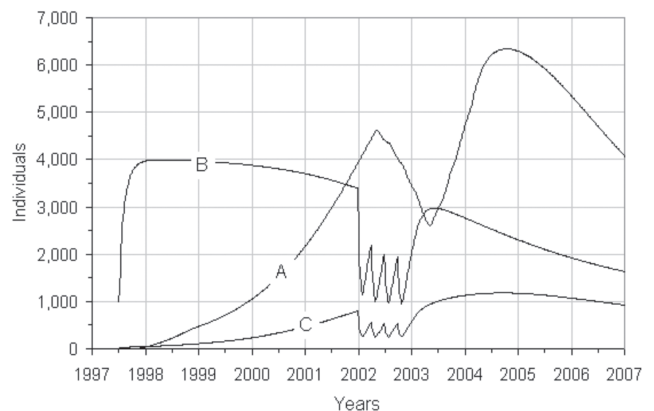

Figure 12. Effects of phytosanitary products on vectors and on numbers of eradicated plants. A = number of eradicated plants per year (x 50); B = healthy nymphs and adults; C = infected nymphs and adults (x 10).

Chemical Control. Studies that evaluated the efficiency of chemical control of *L. lobuliger* (Moura & Rezende 1995, Warwik *et al*. 1999) indicate the possibility of vector control. However, Fig. 12 shows the effect of phytosanitary products on the number of nymphs and adults (both healthy and infected), and on the number of annually eradicated plants; these products reduce vector populations only while the sanitary effect continues. Both healthy and infected populations quickly return to equilibrium when the phytosanitary effect ceases, allowing the disease dissemination rate to increase again. Therefore, phytosanitary products delay the disease progress, but total control can be reached only by eradicating vectors.

Reduction of Host Access. *L. lobuliger* seems to search the host by chance, by exploring the soil (Moura & Rezende 1995), and the probability of access to the host could be reduced by placing adhesive belts or cone-shaped metallic rings on the coconut tree. This practice would be equivalent to reducing the efficacy of vector search and would diminish *Phytomona* spreading rate (Fig. 8). For example, if only 10% of the hosts are reached by the vector (equivalent to a 10-fold reduction in search efficiency), only ca. 1% of the plants would be infected 20 years later.

Conclusions and Suggestions

The model helps to understand the interactions among system components. Variables regarding the disease (like latency and incubation periods, and efficiency of vector-plant and plant-vector transmission), as well as variables concerning vector biology and habits are important for determining the system behavior. Variables should be estimated by taking into account their relative importance (Figs. 8 to 11). The model indicates that chemical or other control techniques only delay the spread of the disease and that as soon as the effect ceases, the disease spreads at its original rate again. The solution to this problem should come from plant breeding programs. The original VENSIM model (23 kb) is available, from the first author.

Acknowledgments

Literature Cited

Bezerra, J. & L. Figueiredo. 1982. Ocorrência de *Phytomonas staheli* McGhee & McGhee em coqueiro (*Coco nucifera* L.) no estado da Bahia, Brasil. Fitopatol. Bras. 7: 139-143.

Chenon, R.D. De. 1984. Reserches sur le genre *Lincus* Stål. Hemiptera, Pentatomidae, Discocephalinae st son role eventual dans la tramission de la Marcitez de palmier à huile et du Hart-Rot du cocotier. Oleagineux 39: 1-4.

Dollet, M. 1984. Plant diseases caused by flagellate protozoa (Phytomonas). Annu. Rev. Phytopathol. 22: 115-132.

Forrester, J.W. 1961. Industrial dynamics. Cambridge, Massachusetts, The MIT Press, 1v.

Howard, F. 2001. Sap-feeders on Palms. In F.W. Howard, R. Giblin-Davis, D. Moore and R. Abad, insects on palms. CABI Bioscience, Ascot, UK, 400p.

Louise, C., M. Dollet & D. Mariau. 1986. Recherches sur le Hartrot du cocotier, maladie à Phytomionas (Trypanosomatidae) et sur son vecteur *Lincus* sp. (Pentatomidae) en Guyane. Oleagineux 41: 437-446.

Moura, J.I.L. & M.L. Rezende. 1995. Eficiência de monocrotofós aplicado via raiz no controle de *Lincus lobuliger* Brad. em coqueiro. Ann. Soc. Entomol. Brasil 24: 1-6.

Moura, J.I.L., R.B. Sgrillo & F.C.M. Miguens. 2002. Manejo integrado das principais pragas do coqueiro, p.67-90. In Manejo Integrado das Principais Pragas e Doenças de Cultivos Amazônicos, 1ª ed., Belem, EMBRAPA v.1, 284p.

Resende, M.L.V., R.E.L. Borges, J.L. Bezerra & D.P. Oliveira. 1986. Transmissão da murcha-de-Phytomonas a coqueiros e dendezeiros *por Lincus lobuliger* (Hemiptera, Pentatomidae): Resultados preliminares. Rev. Theobroma 16: 149-154.

SEI – Superintendência de Estudos Econômicos e Sociais da Bahia. 2002. Anuário Estatístico 2002. (http://www.sei.ba.gov.br/)

Sgrillo, R.B. & K.R.P. Araújo. 1996. Modelaje y simulación de sistemas epizoóticos, p.293-311. In R.E. Lecuon, (ed), Microorganismos patógenos empleados en el control microbiano de insectos plaga. E.R.Lecuona ed. Buenos Aires, 338p.

Vanderplank, J.E. 1963. Plant diseases: Epidemics and control. Academic Press, New York, 349p.

Ventana. 2003. Vensim software. Linking systems thinking to powerful dynamic models. http://www.vensim.com/software.html

Warwick, D., J.L. Moura & M.L. Leal. 1999. Eficiência do manejo integrado na redução da murcha de Phytomonas em coqueiro anão amarelo. Agrotropica 11: 177-182.