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DOI

[10.1007/s002850050047](https://doi.org/10.1007/s002850050047)

Publication date

1966

Published in

Journal of Mathematical Biology

[Link to publication](#)

Citation for published version (APA):

van den Bosch, F., & de Roos, A. M. (1966). The dynamics of infectious diseases in orchards with roguing and replanting as control strategy. *Journal of Mathematical Biology*, 35, 129-157. <https://doi.org/10.1007/s002850050047>

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The dynamics of infectious diseases in orchards with roguing and replanting as control strategy

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Received 2 September 1994

Abstract. Roguing and replanting is a widely adopted control strategy of infectious diseases in orchards. Little is known about the effect of this type of management on the dynamics of the infectious disease. In this paper we analyze a structured population model for the dynamics of an S-I-R type epidemic under roguing and replanting management. The model is structured with respect to the total number of infections and the number of post-infectious infections on a tree. Trees are assumed to be rogued, and replaced by uninfected trees, when the total number of infections on the tree reaches a threshold value. Stability analysis and numerical exploration of the model show that for specific parameter combinations the internal equilibrium can become unstable and large amplitude periodic fluctuations arise. Several hypothesis on the mechanism causing the destabilisation of the steady-state are considered. The mechanism leading to the large amplitude fluctuations is identified and biologically interpreted.

Key words: Structural population model – Stability analysis – Numerical solutions

1 Introduction

Fungal, viral and bacterial diseases cause severe losses in many orchards. One seeks to influence the course of disease development by applying curative chemicals in some situations, but in others such means are not available to date. Moreover, for economic or environmental safety reasons other disease control options are often considered. The control strategy adopted in such situations has often been to rogue infected trees (Zadoks and Schein, 1979) and replace them with disease free trees. This control strategy often resulted in large numbers of trees being rogued. Attempts to control cocoa swollen shoot disease in Ghana, for example, resulted in 190 million trees rogued since 1946

(Ollennu et al. 1989). These measures have not prevented cocoa swollen shoot disease to become one of the most serious threats to cocoa plantations. In other cases roguing and replanting proved to be a useful means of disease control. Roguing against citrus tristeza disease in Israel, Spain and the USA, for example, is successful (Fishman et al., 1983; Thresh, 1988). Other examples where roguing is adopted as control strategy are the bacterial disease 'fire leaf' (*Erwinia amylovora*) in pear and apple, the viral disease causing banana bunchy top in Australia (Allen 1977, 1978a, 1978b; Thresh, 1988), the fungal disease 'silver leaf' (*Strereum purpeum*) in plum, the viral sugarcane Fiji disease (Hayes, 1977) and the plume pox virus in England (Adams, 1978).

It has been recognised that roguing and replanting is a useful strategy for the control of plant disease with limited reproductive capacity only. Diseases with high reproductive capacity infect the orchard quickly, and trees have to be rogued before they become productive in an economic sense. Roguing and replanting management is developed for each disease on an ad-hoc basis, such that trial and error are the only factors stirring the development of the management strategy. Little is known about the dynamics of the disease under the roguing management. Roguing programs would benefit from models which give insight in the consequences of roguing and replanting on the population dynamics of the pathogen.

Chan and Jeger (1994) developed an S(usceptible)-E(xposed)-I(nfective)-R(emoved) model for a viral disease in an orchard. Effects of roguing in the various disease categories was investigated. They concluded that at low contact rates, roguing trees in the infectious stage only is sufficient to eradicate the disease. At higher contact rates, additional roguing in the latent category is needed for disease control. Such latently infected trees can be detected using bio-assays on virus related enzymes. Fishman and Marcus (1984) modeled the citrus tristeza virus using an S-I model with periodic inspection and roguing in an orchard consisting of a number of interacting plant rows. They calculated an inequality between detection rates and the parameters of the disease that must be satisfied for disease control. Using this model Fishman et al. (1983) made an economic analysis of the roguing management. They concluded that roguing management is economically justified in the case of citrus tristeza virus. For this model Thieme and Heesterbeek (1989) considered criteria from which one can conclude whether the control efforts are adequate. Their criterion is based on a combination of data on the number of trees rogued in each row during inspection rounds. Allen (1983) developed a model for the spatial and temporal spread of a virus disease. He applied the model to several virus diseases with the aim to investigate the usefulness of roguing and replanting management for disease control.

All these models distinguish only one or two different types of infected trees. For a systemic virus disease, which infects the entire tree within a short time span, the 'infected tree' is a suitable conceptual unit for modelling. Many diseases, however, have localized infections on leaves or branches. The tree can then not be classified as exposed or infected, but has to be characterized by its number of infections. The number of infections gradually increases,

depending on the force of infection from all trees in the orchard. The roguing criterion in this case usually relates to the number of infections passing a threshold value. Modelling the dynamics of these diseases requires a physiologically structured population model in the sense of Metz and Diekmann (1986).

In this paper we develop such a structured population model of disease dynamics in an orchard with roguing and replanting. The dynamics of the system is investigated using steady-state and stability analysis and numerical simulations of the model.

2 The model

In this section we develop a model for the dynamics of a disease with localized infections in an orchard where roguing is used as control strategy. We use the Murphy (1983) model for the development of a size structured population with constant size at birth (see also Metz and Diekmann, 1986). The disease is assumed to be of S(usceptible)-I(nfective)-R(removed) type with constant transition rate, μ , from infectious, I , to remove, R , state. We will call both I and R state *infected*. The I state will be called *infectious*. A tree has a fixed number, α , of so-called sites. A site is a limited area of the tree that can support one infection. We leave tree growth out of consideration. A tree is rogued when the number of infected sites reaches a preset value, to be called the *roguing threshold*. Each rogued tree is instantaneously replaced by a completely disease free tree. Trees die due to other causes but the disease with constant rate, ω . Dead trees are also replaced by disease free trees. Replacing rogued and dead trees implies that the total number of trees in the orchard is constant.

The model has to keep track of the total number of infected sites on a tree, $L(t, a)$, since this determines when the tree is rogued. The model also has to keep track of the number of post-infectious sites, $R(t, a)$, since these do not contribute to the initiation of new infections. The structuring individual characteristics of the model are thus $L(t, a)$ and $R(t, a)$. The evolution of $L(t, a)$ and $R(t, a)$ can be described by

$$\frac{\partial \sigma}{\partial t} + \frac{\partial \sigma}{\partial a} = g(t, \sigma); \quad \sigma(t, 0) = \sigma_0 \quad (1)$$

where σ is replaced by $L(t, a)$ and $R(t, a)$, respectively, and $g(t, \sigma)$ is the growth rate of the relevant structuring individual characteristic.

The growth rate of the number of infected sites on a tree, $g_1(t, L)$, depends on the total number of infectious sites in the entire orchard, denoted by $I(t)$, and on the number of susceptible sites on the tree, $\alpha - L$. Consider an infectious unit produced by and infectious site in the orchard. We assume that this infectious unit is homogeneously mixed in the air-plankton above the canopy. The infectious unit finally settles down either on a tree or on the

ground. The probability to settle on a tree is proportional to the density of trees in the orchard, D_{eff} , we can thus write,

$$g(t, \sigma) := g_1(t, L) = \Psi D_{\text{eff}} I(t)(\alpha - L) . \quad (2)$$

The parameter Ψ is a compound parameter incorporating the rate of production of infectious units by an infectious site, catchment area of a tree, the area of the orchard and the probability that an infectious unit, landed on a susceptible site, will cause an infection. We chose to incorporate tree density explicitly because this parameter is an obvious candidate in the development of orchard management strategies.

Since trees are planted disease free, the boundary condition is,

$$L(t, 0) = 0 . \quad (3)$$

The growth rate of the number of post-infectious sites on a tree, $g_2(t, R)$, depends on the current number of infectious sites on that tree, $L - R$. Since transition rate from infectious to post-infectious is constant we have,

$$g(t, \sigma) := g_2(t, R) = \mu(L - R) , \quad (4)$$

where μ is the transition rate.

Trees are planted disease free and the boundary condition reads,

$$R(t, 0) = 0 . \quad (5)$$

Trees are rogued when $L(t, a)$ equals the roguing threshold L_{max} . Therefore, the age at roguing, $A_{\text{max}}(t)$, is the solution of

$$L(t, A_{\text{max}}(t)) = L_{\text{max}} . \quad (6)$$

The number of infectious sites on a tree of age a at time t is given by $L(t, a) - R(t, a)$. Multiplying this number by the age density of the trees, $M(t, a)$, and integrating over all possible tree ages, we obtain for the total number of infectious sites in the orchard

$$I(t) = \int_0^{A_{\text{max}}(t)} [L(t, a) - R(t, a)] M(t, a) da . \quad (7)$$

Finally the evolution of the age distribution of the trees in the orchard, $M(t, a)$, is described by

$$\frac{\partial M}{\partial t} + \frac{\partial M}{\partial a} = -\omega M \quad (8)$$

where ω is the tree death-rate. Since tree rogued or died is replaced instantaneously, the total number of tree in the orchard, N , remains constant. The boundary condition can thus be written

$$M(t, 0) = \omega M + \left(1 - \frac{dA_{\text{max}}(t)}{dt}\right)^+ M(t, A_{\text{max}}(t)) \quad (9)$$

The first term to the right of this boundary condition is the number of natural deaths per unit of time and the second term is the positive part of the flux through the cross-section $a = A_{\max}(t)$. This flux of course equals the number of trees rogued per time unit. One has to take the positive part to correctly model the fact that if the roguing age growth more rapidly than the age of the tree, no tree will reach the roguing age.

Note: In this model there is a relation between $A_{\max}(t)$, $L_{\max}$ and $I(t)$ which ensures that the flux is always positive and taking the positive part is superfluous. This can be seen as follows: integrating the differential equation for $L(t, a)$ along characteristics we find

$$L(t, a) = \alpha - \exp\left(-\psi D_{\text{eff}} \int_{t-a}^t I(\xi) d\xi\right) \quad (10)$$

Using Eq. (6)

$$\int_{t-A_{\max}(t)}^t I(\xi) d\xi = -\frac{1}{\psi D_{\text{eff}}} \ln(\alpha - L_{\max}) \quad (11)$$

and differentiating with respect to t yields

$$\frac{dA_{\max}(t)}{dt} = 1 - \frac{I(t)}{I(t - A_{\max}(t))} \quad (12)$$

which implies that $dA_{\max}/dt \leq 1$.

The variables are scaled as follows

$$\begin{aligned} l(t, a) &= \frac{L(t, a)}{\alpha}; & r(t, a) &= \frac{R(t, a)}{\alpha}; & i(t) &= \frac{I(t)}{N\alpha}; \\ m(t, a) &= \frac{M(t, a)}{N}; & \psi &= N\Psi\alpha. \end{aligned} \quad (13)$$

The scaled variables can be interpreted as fractions of the total. Putting all pieces together the model reads,

$$\frac{\partial l}{\partial t} + \frac{\partial l}{\partial a} = \psi D_{\text{eff}} i(t)(1 - l) \quad (14)$$

$$l(t, 0) = 0 \quad (15)$$

$$A_{\max}(t) \text{ is the solution of } l(t, A_{\max}(t)) = l_{\max} \quad (16)$$

$$\frac{\partial r}{\partial t} + \frac{\partial r}{\partial a} = \mu(l - r) \quad (17)$$

$$r(t, 0) = 0 \quad (18)$$

$$i(t) = \int_0^{A_{\max}(t)} [l(t, a) - r(t, a)] m(t, a) da \quad (19)$$

$$\frac{\partial m}{\partial t} + \frac{\partial m}{\partial a} = -\omega m(t, a); \quad a \leq A_{\max}(t) \quad (20)$$

$$m(t, 0) = \omega + \left(1 - \frac{dA_{\max}(t)}{dt}\right) m(t, A_{\max}(t)) \quad (21)$$

where $l_{\max}$ is the fraction sites of a tree infected when it is rogued, the (scaled) *roguing threshold*. The parameter combination ψD_{eff} will, in the following, loosely be called the ‘effective tree density’ in the orchard. The scaled model has only four parameters, which is few for a physiologically structured model. This enables us to study parameter space in detail.

In our analysis we concentrate on biologically reasonable ranges of parameter values. The lifetime of a disease free tree in an orchard is roughly between 1 and 50 years. A reasonable range of tree death-rates thus is $[0; 1.0]$, per year. Plant disease infections have a lifetime between a few weeks, for fungal infections, and a few years, for bacterial cancers. We therefore take $\mu \in [0; 20]$, per year. The net-reproductive number is defined as the total number of new infections caused by one infection during the whole course of its life when it is placed in a completely disease free orchard. About the net-reproductive number of diseases controlled by roguing management little information is found in the literature. Only for virus diseases parameter values can be estimated. In their model Chan and Jeger use parameter values which results in R_0 approximately 1.5. For fungal diseases the net reproduction would, probably, be larger. We take a range of R_0 between 1 and 5. With $\omega = 0.1$ and $\mu = 10$ this yields $\psi D_{\text{eff}} \in [0; 50]$, infections per infection per year. Finally the roguing level $l_{\max}$ can theoretically have values between 0 and 1. In the discussions of results we will always use year as our time unit.

3 Reformulation of the model

For the steady-state and stability analysis we reformulate the model as a set of integral and differential equations with delay terms. Since we are only interested in the behaviour of solutions for large t , where the influence of the initial condition has vanished, we will only consider $a < t$.

In scaled form Eq. (11) reads

$$\int_{t-A_{\max}(t)}^t i(\xi) d\xi = -\frac{1}{\psi D_{\text{eff}}} \ln(1 - l_m) \quad (22)$$

Integrating (20) along characteristics, substituting this expression in (21) and defining $m(t, 0) =: b(t)$ we arrive at,

$$b(t) = b(t - A_{\max}(t)) \left(1 - \frac{dA_{\max}(t)}{dt} \right) e^{-\omega A_{\max}(t)} + \omega, \quad (23)$$

where $b(t)$ is the rate of planting disease free trees at time t .

Integration of (14), (17) and (20) along characteristics and substitution of these expressions in (19) yield,

$$\begin{aligned} i(t) = & \int_0^{A_{\max}(t)} \left\{ e^{-\mu a} \left(1 + \mu \int_0^a e^{\mu \sigma} e^{-\psi D_{\text{eff}} \int_{t-a}^{t-a+\sigma} i(\xi) d\xi} d\sigma \right) - e^{-\psi D_{\text{eff}} \int_{t-a}^t i(\xi) d\xi} \right\} \\ & \times b(t-a) e^{-\omega a} da. \end{aligned} \quad (24)$$

Model system (14) through (21) is thus equivalent with the system (22), (23) and (24). This system will be subject to steady-state and stability analysis.

The function $b(t)$ is the rate of planting disease free trees. Replanting is the result of (i) roguing diseased trees and (ii) the death of trees due to all causes but the disease. In the discussion on the model output we often have to distinguish between these two sources of removing trees from the orchard. To simplify the discussion we introduce an additional variable; the 'roguing rate', $r(t)$. The roguing rate is the fraction of trees rogued per year. Obviously, roguing rate, the death-rate, ω , and the replanting rate, $b(t)$, are related as $r(t) = b(t) - \omega$.

4 Steady-states

The system admits a trivial solution of the form

$$(\hat{i}_1, \hat{b}_1, \hat{A}_1) = (0, \omega, \infty). \quad (25)$$

The non-trivial steady-states are given by

$$(\hat{i}_2, \hat{b}_2, \hat{A}_2) = \left(\hat{i}, \frac{\omega}{1 - e^{-\omega \hat{A}_2}}, -\frac{1}{\psi D_{\text{eff}} \hat{i}} \ln(1 - l_{\text{max}}) \right), \quad (26)$$

where $\hat{i}$ is found from (see Eq. (24))

$$\begin{aligned} & \frac{\omega \psi D_{\text{eff}}}{\mu - \psi D_{\text{eff}} \hat{i}} (1 - (1 - l_{\text{max}})^{\omega / (\psi D_{\text{eff}} \hat{i})})^{-1} \\ & \times \left\{ \frac{1 - (1 - l_{\text{max}})^{1 + (\omega / \psi D_{\text{eff}} \hat{i})}}{\omega + \psi D_{\text{eff}} \hat{i}} - \frac{1 - (1 - l_{\text{max}})^{(\mu + \omega) / (\psi D_{\text{eff}} \hat{i})}}{\mu + \omega} \right\} = 1. \end{aligned} \quad (27)$$

Denote the left hand side of (27) by $F(\hat{i})$. Then

$$\lim_{\hat{i} \rightarrow 0} F(\hat{i}) = \frac{\psi D_{\text{eff}}}{\mu + \omega} \quad (28)$$

and $F(1) < 1$. It follows by continuity that if

$$\frac{\psi D_{\text{eff}}}{\mu + \omega} \geq 1 \quad (29)$$

Eq. (27) has at least one root in $[0, 1)$. This combination of parameters has a clear biological interpretation. Consider a disease free orchard. Introduce an infinitesimal small number of infections. In this orchard $\mu + \omega$ is the rate of removal of infectious sites. The average lifetime of an infectious site is $1/(\mu + \omega)$. In a disease free orchard the rate at which an infectious site produces new infections equals ψD_{eff} . The total number of new infections produced by one infected site during the whole course of its life thus equals $\psi D_{\text{eff}}/(\mu + \omega)$. If this number is larger than one, an infection produces on

Fig. 1. Non-trivial steady-state values of the fraction of infectious sites, $\hat{i}$, the age at roguing, $\hat{A}_{\max}$, and the roguing rate, r , as a function of the roguing threshold, $l_{\max}$, the tree death-rate, ω , the transition rate of infections from infectious to post-infectious, μ , and the effective tree density in the orchard, ψD_{eff}

average more than one new infection, and the disease invades the disease free orchard. In the ecological literature this number is known as the net-reproductive number denoted by R_0 . For a thorough discussion on net-reproductive numbers we refer to Heesterbeek (1992).

We have not been able to show analytically that Eq. (27) has only one root. Extensive numerical calculations however always yield only one non-trivial steady-state value for $\hat{i}$. This also implies only one value for $\hat{A}$ and b . Before discussing the steady-states we note again that the roguing rate, $r(t)$, is the replanting rate, $b(t)$, minus the tree death-rate ω . Figure 1 shows the dependence of the steady-state on parameters values. Scales on the abscissa are equal to allow easy comparison.

Clearly, biologically reasonable values of the tree death-rate ω have virtually no influence on steady-states. All other parameters do have a distinct effect on steady-state values. Steady-state values have a simple and biologically plausible dependence on the parameters involved in the infection process (μ and ψD_{eff}). Changing these parameters such that the net-reproductive number increases, results in an increasing fraction of infectious sites, an increasing roguing rate and a decreasing tree age at roguing. Decreasing tree density, D_{eff} , can cause the net-reproductive number to become smaller than unity and therewith the disease to go extinct. A remarkable feature is that decreasing, D_{eff} , the age at roguing increases sharply, when D_{eff} is still considerably larger than the density needed to drive the disease to extinction. The same holds for increasing the death-rate of infectious sites, μ . The implications for orchard management will be discussed.

The effect of the roguing threshold, $l_{\max}$, on steady-state values of the variables is slightly more complicated. Increasing $l_{\max}$ from zero to unity the fraction of infectious sites shows an increase followed by a decrease. Contrary to the fraction of infectious sites, the age at roguing increases continually when increasing the roguing threshold $l_{\max}$. At first sight this seems contradictory. An increase in the fraction of infectious sites is expected to decrease the age at roguing. Consider, however, Eq. (14) through (16) assuming $i(t) = \hat{i}$. Calculating the age at roguing results in the same expression as for $\hat{A}_2$ in (26). The term $\ln(1 - l_{\max})$ thus expresses the ‘direct’ effect of the roguing threshold on the age at roguing. The fraction of infectious sites, $\hat{i}$, also depends on $l_{\max}$. This represents the ‘indirect’ effect of the roguing threshold on the age at roguing. Combining the observation that $\hat{i}$ can both increase or decrease with increasing roguing threshold, with the observation that the age at roguing increases with the roguing threshold implies that the ‘direct’ effect of the roguing threshold on the age at roguing is always stronger than the ‘indirect’ effect through the fraction of infectious sites.

5 The stability analysis

Linearizing about the trivial steady-state yields the characteristic equation,

$$\frac{1}{\lambda + \omega} \left(\frac{\psi D_{\text{eff}}}{\mu + \omega + \lambda} - 1 \right) = 0. \quad (30)$$

The trivial steady state is stable if

$$\frac{\psi D_{\text{eff}}}{\mu + \omega} < 1, \quad (31)$$

and unstable if the inequality holds in the other direction. The interpretation of this quantity discussed above, implies that the trivial steady-state loses stability when the disease can invade a disease free orchard.

In Appendix C we show that the characteristic equation associated with linearizing about the non-trivial steady-state reads,

$$\begin{vmatrix} \frac{\hat{b}\psi D_{\text{eff}}}{\lambda} [-\mu F(\lambda) + G(\lambda)] - 1 & H(\lambda) & C \\ 0 & \frac{1}{\lambda + \omega} (1 - e^{-(\lambda + \omega)\hat{A}}) & \hat{b}e^{-\omega\hat{A}} \\ -\frac{1}{\hat{i}\lambda} (1 - e^{-\lambda\hat{A}}) & 0 & -1 \end{vmatrix} = 0 \quad (32)$$

where

$$\begin{aligned} F(\lambda) = & \frac{1}{\mu - \hat{i}\psi D_{\text{eff}} + \lambda} \left[\frac{-1}{\omega + \hat{i}\psi D_{\text{eff}}} (e^{-(\omega + \hat{i}\psi D_{\text{eff}})\hat{A}} - 1) \right. \\ & \left. + \frac{1}{\mu + \omega + \lambda} (e^{-(\mu + \omega + \lambda)\hat{A}} - 1) \right] - H(\lambda) \frac{1}{\hat{i}\psi D_{\text{eff}}}, \end{aligned} \quad (33)$$

$$G(\lambda) = \frac{-1}{\omega + \hat{b}\psi D_{\text{eff}}} (e^{-(\omega + \hat{b}\psi D_{\text{eff}})\hat{A}} - 1) + \frac{1}{\omega + \hat{b}\psi D_{\text{eff}}} (e^{-(\omega + iD_{\text{eff}})\hat{A}} - 1), \quad (34)$$

$$H(\lambda) = \hat{b}\psi D_{\text{eff}} \frac{1}{\mu - \hat{b}\psi D_{\text{eff}}} \left[\frac{-1}{\omega + \hat{b}\psi D_{\text{eff}} + \lambda} (e^{-(\omega + \hat{b}\psi D_{\text{eff}} + \lambda)\hat{A}} - 1) + \frac{1}{\mu + \omega + \lambda} (e^{-(\mu + \omega + \lambda)\hat{A}} - 1) \right], \quad (35)$$

$$C = -\hat{b}\hat{b}\psi D_{\text{eff}} \frac{1}{\mu - \hat{b}\psi D_{\text{eff}}} (e^{-(\mu + \omega)\hat{A}} - e^{-(\omega + \hat{b}\psi D_{\text{eff}})\hat{A}}). \quad (36)$$

This characteristic equation is too unwieldy to be dealt with analytically and we have to take recourse to numerical techniques. The non-trivial steady-state might lose stability through a hopf-bifurcation. Consider a path in a two dimensional parameter space which intersects the stability boundary. Along this path a pair of complex conjugated roots crosses the imaginary axis while all other roots lie in the left half plane. We study this hopf-bifurcation numerically by substituting $\lambda = i\theta$ in (32) through (36) and solving the resulting complex equation for θ and a free parameter.

Figure 2 shows the computed stability boundaries for combinations of the roguing threshold, l_{max} , and one of the three remaining parameters. We only show one set of stability diagrams. Other parameter combination hardly change the *form* of the stability boundaries, although they do change the precise location. Each loop in the figure represents the set of parameter combinations where one pair of eigenvalues lies on the imaginary axis. We have found three such loops. Studying the characteristic equation numerically the first (largest, lower) loop was easily found. The second (intermediate sized, middle) loop took a lot more time and the third (smallest, upper) loop was only found after several long computer sessions. We cannot exclude the possibility that more loops can be found. However, the parameter regions for values of l_{max} smaller than that of the largest loop are searched through extensively, but no other stability boundaries were found. Moreover, the numerical simulations, to be discussed in the next section, also show convergence to the non-trivial steady-state in these latter regions. Additional, probably still smaller, loops could thus be positioned only on top of the third loop. Such additional loops will not change the appearance of the stability diagrams. Moreover, they will not change any of the interpretations and conclusions to be discussed in the following sections. Numerical solutions of the model show that the internal steady-state is unstable inside the loops and stable in the remainder of parameter space.

Figure 2 gives insight in the relative importance of parameter values on stability. The roguing threshold, l_{max} , has a profound effect on stability. Roguing thresholds close to zero and close to unity always result in stable steady-states. Except for parameter combinations close to the extinction boundary and for large tree death-rates, steady-states are instable for intermediate roguing thresholds. Comparing Figs. 1 and 2 shows that the

Fig. 2. Stability diagram of the full model. On the abscissa: the fraction of sites infected at which a tree is rogued and replaced by an uninfected tree, $l_{\max}$. On the ordinate: Figure A: The density of trees in the orchard, ψD_{eff} . Figure B: the transition rate of infections from infectious to post-infectious, μ . Figure C: tree death-rate, ω . In each figure the values of the parameters not displayed on the axis have the default value (see Sect. 3). For parameter values in the regions marked with EXTINCTION the disease goes extinct. Inside the loops the internal steady-state is unstable. In the rest of the parameter space the internal steady-state is locally stable. Dotted line: final value of the Kermack and McKendrick epidemic (see text)

non-trivial steady-state becomes unstable for values of $l_{\max}$ where the fraction of infectious sites sharply decreases and the age at roguing sharply increases. Though tree death-rate, ω , has little effect on steady-state values (Fig. 1d) it does influence stability. When tree death-rate is small it has little effect on the value of the roguing threshold where the steady-state becomes unstable. However, for larger tree death-rates the unstable region becomes unstable and the internal steady-state becomes stable. We have found this effect of tree death-rate on stability for all other parameter values investigated. The explanation of the stabilising effect of increasing tree death-rate is given in Sect. 8. Like for the roguing threshold, the non-trivial steady-state is unstable for intermediate values of the tree density, ψD_{eff} , as well as intermediate values of the death-rate of infectious sites, μ . Note that also for these parameters the unstable region coincides with the region of steep increase in the age at roguing and sharp decrease in the fraction of infectious sites (Fig. 1).

6 Dynamic behaviour

De Roos (1988) developed a method especially appropriate for the numerical integration of partial differential equations occurring in structured population models. This method was used to check stability boundaries computed in the previous section. Furthermore we studied the dynamic behaviour of the model using numerical simulations. Figure 3 shows two simulations for parameter combinations where the non-trivial steady-state is unstable. The two examples show qualitatively similar dynamics. All simulations carried out show the same, qualitative pattern of fluctuations in the fraction of infected sites, the fraction of infectious sites and the number of trees rogued per year. The sequence of events during one period of oscillation, can be summarized as follows: a large number of trees is rogued and replaced by uninfected trees, which constitutes an explosive increase in the number of susceptible sites and leads to an epidemic outbreak. The disease ‘consumes’ a large part of its resource during this epidemic outbreak. Due to the decrease in the number of susceptible sites the disease is not able to maintain a population of infectious sites. The epidemic peters out and a phase of very low epidemic activity follows. During this phase the total number of infected sites in the orchard gradually decreases. This slow decrease is not the result of replanting of rogued trees since no trees are being rogued. Plotted on a log-scale it is seen that the number of infected sites decrease exponentially with rate parameter 0.1. This decrease is thus due to tree death not associated with the disease ($\omega = 0.1$). Because of the replanting of these dying trees, the number of susceptible sites gradually increases. When the number of susceptible sites

Fig. 3. Periodic solutions of the model for parameter values $\psi D_{\text{eff}} = 20$, $\mu = 10$, $\omega = 0.1$. Figure A: Simulation for $l_{\text{max}} = 0.82$. Figure B: Simulation for $l_{\text{max}} = 0.88$. Top panel the total fraction of sites infected, middle panel of the fraction of trees in the orchard rogued per year, bottom panel the fraction of infectious sites

passes the threshold value for epidemic increase, the disease starts to build up slowly and the number of infected sites increases. As a consequence more and more trees reach the roguing threshold, are rogued and replaced by disease free trees, which in turn increases the number of susceptible sites and speeds up the epidemic. Due to the speed up eventually all remaining trees pass the roguing threshold and are rogued. This causes again the sharp increase in the number of susceptible sites, the epidemic explodes anew and the cycle starts all over.

Contrary to the qualitative similarity, the examples shown are quite different quantitatively. Figure 3a shows a simulation for parameter values in the lower loop of the stability diagram ($l_{\max} = 0.82$ $\psi D_{\text{eff}} = 20$, see Fig. 2a). Peak values of the fraction of infectious sites are approximately 0.1. Peak values of the roguing rate are approximately 0.2. The period of the oscillations is approximately 10 year. Adding the fraction of trees died during the phase without epidemic activity to the fraction of trees rogued during the roguing peak shows that within one cycle the whole orchard is replanted. Figure 3b shows a simulation for parameter values in the second loop ($l_{\max} = 0.88$ $\psi D_{\text{eff}} = 20$, see Fig. 2a). Peak values of the fraction of infectious sites and the fraction of trees rogued per year are 0.04 and 0.03, respectively, which is considerably lower than for the simulation with $l_{\max} = 0.82$. The period is approximately 7 year. This implies that less trees died during the phase without epidemic activity than for $l_{\max} = 0.82$. Since also the replanting peak is considerably smaller we conclude that only part of the orchard is replanted during one cycle. This quantitative difference between the periodic solutions in the lower loop and in the middle loop is found throughout parameter space.

For the simulations shown in Fig. 3 the evolution of the population distribution as a function of the fraction of infected sites during one cycle period is shown in Fig. 4. First consider Fig. 4a, corresponding to the

Fig. 4. The evolution of the population distribution as a function of the fraction of infected sites. Figure A: parameter values as in Fig. 3a. Figure 3B: parameter values as in Fig. 3b. Top panel development in the period without epidemic activity. Lower panel development during the epidemic outbreak

simulation in Fig. 3a. The top panel shows the development of the population distribution during the period without epidemic activity. All trees have l values close to the roguing threshold. During this, approximately eight year lasting phase, trees only die due to all causes but roguing. The dying trees are replaced by uninfected trees, that remain uninfected because there is no epidemic activity. During the phase of epidemic outbreak (lower panel) all remaining trees are rogued and replaced. The newly planted cohort of trees increases in infection level during an approximately two year period. When the epidemic peters out all trees have again l values close to the roguing threshold. Figure 8b shows the development of the same population distribution during one period for a parameter combination in the second loop (compare Fig. 2a). Here two peaks are found. During each epidemic outbreak the peak with largest values of l passes the roguing threshold. The peak with the smaller l values moves during the outbreak to l values close to the roguing threshold. In line with these findings one would expect to find three groups of trees with distinct fractions of infected sites for parameter combinations in the upper loop. The numerical procedure however is not accurate enough to get a clear view of the dynamics of the system for parameter values close to the upper stability boundary.

A number of runs was done for a range of values of $l_{\max}$, two different values of ψD_{eff} and $\omega = 0.1$, $\mu = 10$. Figure 5 shows the minimum and maximum of the fraction of infected sites and of the age at roguing (compare with Fig. 2a) observed during the run. For values of $l_{\max}$ only a few percent larger than its value on the stability boundary, the periodic solution showed large amplitude oscillations. For values few percent smaller than the value on the stability boundary the trajectories converged to the stable steady-state. From the model simulations it is not possible to determine whether the

Fig. 5. Steady state, minimum and maximum value of the age-at-roguing, $\hat{A}$ (top panel), and the fraction of infected sites, l (bottom panel) as a function of the roguing threshold, $l_{\max}$. Dotted line indicates the unstable steady-state values. Figure A: effective tree density, ψD_{eff} is 13. Figure B: effective tree density, ψD_{eff} is 20. Other parameters have default values (see Sect. 3)

hopf-bifurcation is sub- or supercritical. For other parameter sets we also found that increasing $l_{\max}$ to values only slightly larger than its value on the stability boundary leads to large amplitude fluctuations. In large parts of the parameter region where the internal steady-state is unstable the range of values of the age at tree roguing does not contain its steady-state value. The explanation of this phenomena will be given in the next sections.

7 The destabilising mechanism

Which mechanism is responsible for the loss of stability of the internal steady-state? In this section we will answer this question by considering various variants of the full model. First we completely strip the model from its roguing and replanting component to consider the population dynamics of the infectious disease separated from the orchard management. Next we investigate the effect of a roguing management, in which trees are rogued and replaced after a delay time $A_{\max}$. The difference between a fixed delay time and variable delay time shows to be crucial.

Unstable steady-states and periodic solutions can be the result of the population dynamics of the infectious disease itself and not due to the roguing and replanting management. To test this idea consider an orchard where trees are not rogued and replaced by new trees. To this end let $l_{\max} \uparrow 1$ in the full model. This implies that $A_{\max}(t) \uparrow \infty$. Now define:

$$S(t) = \int_0^\infty (1 - l(t, a))m(t, a)da; \quad \text{the fraction of susceptible sites}, \quad (37)$$

$$I(t) = \int_0^\infty (l(t, a) - r(t, a))m(t, a)da; \quad \text{the fraction of infectious sites}, \quad (38)$$

$$R(t) = \int_0^\infty r(t, a)m(t, a)da; \quad \text{the fraction of post-infectious sites}. \quad (39)$$

Note that $I(t)$ equals $i(t)$ in (19) if $A_{\max}(t)$ is replaced by ∞ . In Appendix A it is shown that in an orchard without roguing the model boils down to the well known S-I-R model with constant death-rate in all classes in a population of constant size. In the terminology of our model this S-I-R model reads,

$$\begin{aligned} \frac{dS}{dt} &= \omega - \psi D_{\text{eff}} I(t) S(t) - \omega S(t) \\ \frac{dI}{dt} &= \psi D_{\text{eff}} I(t) S(t) - (\mu + \omega) I(t) \\ \frac{dR}{dt} &= \mu I(t) - \omega R(t). \end{aligned} \quad (40)$$

This model has been studied extensively. Its internal steady-state is, if it exists stable. From this we conclude that the instability of the internal steady-state of the full model is a result of the roguing and replanting management and is not solely due to the population dynamics of the infectious disease.

How can the time delay $A_{\max}$ destabilize the internal steady-state? We first consider the epidemic process and the roguing and replanting process separately and subsequently combine the two. Consider the epidemic described by (4) to be in steady-state. Given a small perturbation from its steady-state the S-I-R system returns to it through damped oscillations. The period of these oscillations is approximately

$$P = \frac{2\pi}{|\text{Im}\lambda|}, \quad (41)$$

where $\text{Im}\lambda$ is the imaginary part of the eigenvalue of the system linearised around the internal steady-state. The eigenvalues are:

$$\lambda = -\frac{1}{2}\omega \frac{\psi D_{\text{eff}}}{\mu + \omega} \pm \frac{1}{2}\sqrt{\left(\frac{\psi D_{\text{eff}}}{\mu + \omega}\right)^2 \omega^2 - 4\omega\psi D_{\text{eff}}\left(1 - \frac{\mu + \omega}{\psi D_{\text{eff}}}\right)}. \quad (42)$$

The time delay $A_{\max}$ may interact with this characteristic period in the following way: Assume that the age distribution of the trees in the orchard is in steady-state, implying that the age density decreases exponentially, up to the age at roguing $A_{\max}$. If this steady-state were to be perturbed by removing a number of infected trees and replacing them with uninfected trees of age zero, those trees involved in the perturbation that do not die through natural mortality, will be rogued and replanted after a period $A_{\max}$, leading to a second influx of uninfected trees.

The initial perturbation with uninfected trees also induces an increase in the spreading rate of the epidemic, a phenomenon that damps out in an oscillatory manner. If the secondary influx of uninfected trees coincides with a maximum in the (damping) rate of epidemic spread, the latter might be reinforced. In this situation the epidemic will increase faster with the roguing management than it would have done without. One can imagine that the resonance between the epidemic and the roguing process can destabilise the internal steady-state. Destabilisation due to resonance is expected when $\hat{A}_{\max}$ is equal to, or a multiple of, the period of the S-I-R model. Conversely, when the age at roguing is half, one and a half, two and a half, etc. of the period of the S-I-R epidemic one might expect stabilisation due to interference. We thus have to compare $\hat{A}_{\max}$ calculated from the full model with the period P , (41). To this end the steady-state Eqs. (26) and (27) are used to calculate the age at roguing $\hat{A}_{\max}$ for each parameter combination μ , ω , ψD_{eff} and $l_{\max}$. Period P is calculated from (41) and (42) for the same parameters values. Figure 6 plots those parameter combinations where $\hat{A}_{\max} = \alpha P$, with $\alpha = 1, 2, 3, \dots$. Here resonance is expected. The figure also indicates parameter combinations where $\hat{A}_{\max} = \alpha P$, with $\alpha = 0.5, 1.5, 2.5, \dots$. Here interference is expected.

Fig. 6. Parameter values where resonance or interference is expected between the dynamics of the infectious disease and the dynamics of the roguing and replanting management. Solid lines represent the stability boundaries as presented in Fig. 2a. Heavy dotted lines are parameter values where the period of the S-I-R epidemic is a multiple of the age-at-roguing. Thin dotted lines are parameter values where the period of the S-I-R epidemic is 0.5, 1.5, 2.5 and 3.5 times the age-at-roguing. For explanation see the text. Other parameter values as in Fig. 2a

These parameter combinations are presented together with the stability diagram of the full model. The curves of parameter combinations where resonance is expected go midway through each loop of the stability diagram. Curves of parameter combinations where interference is expected are very close to the upper and lower branch of each loop. We conclude that parameter combinations where resonance phenomena are expected correspond to an unstable internal steady-state of the full model, suggesting that the delay term is responsible for the destabilisation of the internal steady-state.

In the full model the age at roguing is a dynamic variable. Does the instability of the internal steady-state hinge upon the fact that $A_{\max}$ is a dynamically varying time delay or would a fixed delay also result in periodic solutions? To answer this question we compare the full model with a model with fixed age at roguing. To derive the model with fixed age at roguing, substitute τ for $A_{\max}(t)$ in Eqs. (19), (20) and (21) and delete Eq. (16) from the model. Using definitions (37), (38) and (39) with the upper bound of the integrants equal to τ and defining,

$$U(t) = \exp\left(-\psi D_{\text{eff}} \int_{t-\tau}^t i(\xi) d\xi\right);$$

the fraction of susceptible sites on a tree of age τ , (43)

$$V(t) = \int_0^\tau \exp\left(-\mu\sigma - \psi D_{\text{eff}} \int_{t-\tau}^{t-\sigma} i(\xi) d\xi\right) d\sigma;$$

the fraction of infectious sites on a tree of age τ , (44)

the model can be written as the system of delay differential equations (Appendix B),

$$\begin{aligned}
 \frac{dS}{dt} &= -\psi D_{\text{eff}} IS + \omega(1 - S) + \frac{\omega}{e^{\omega\tau} - 1}(1 - U) \\
 \frac{dI}{dt} &= \psi D_{\text{eff}} IS - (\mu + \omega)I - \frac{\omega}{e^{\omega\tau} - 1}(e^{-\mu\tau} - U + \mu V) \\
 \frac{dU}{dt} &= \psi D_{\text{eff}}(I(t - \tau) - I(t))U \\
 \frac{dV}{dt} &= \psi D_{\text{eff}} I(t - \tau)V - e^{-\mu\tau} + U - \mu V \\
 \frac{dR}{dt} &= \mu I - \omega R .
 \end{aligned} \tag{45}$$

Here we are only interested in the stability characteristics of the non-trivial steady-state of the model equations (45) and we will not go into other details of the analysis of this model. The non-trivial steady-state is given by,

$$(\hat{S}_2, \hat{I}_2, \hat{R}_2, \hat{U}_2, \hat{V}_2) = \left(S^*, I^*, \frac{\mu}{\omega} I^*, e^{-\psi D_{\text{eff}} I^* \tau}, \frac{1 - e^{-(\mu - \psi D_{\text{eff}} I^*)\tau}}{\mu - \psi D_{\text{eff}} I^*} e^{-\psi D_{\text{eff}} I^* \tau} \right) \tag{46}$$

where

$$S^* = \frac{1}{\psi D_{\text{eff}} I^* + \omega} \frac{\omega}{1 - e^{-\omega\tau}} (1 - e^{-(\psi D_{\text{eff}} I^* + \omega)\tau}) \tag{47}$$

and I^* is calculated from

$$\begin{aligned}
 &\left\{ \frac{\psi D_{\text{eff}}}{\psi D_{\text{eff}} I^* + \omega} \frac{\omega}{1 - e^{-\omega\tau}} (1 - e^{-(\psi D_{\text{eff}} I^* + \omega)\tau}) - \mu - \omega \right\} I^* \\
 &- \frac{\omega}{1 - e^{-\omega\tau}} \frac{\psi D_{\text{eff}} I^*}{\mu - \psi D_{\text{eff}} I^*} (e^{-(\psi D_{\text{eff}} I^* + \omega)\tau} - e^{-(\mu + \omega)\tau}) = 0 .
 \end{aligned} \tag{48}$$

Linearizing about the non-trivial steady-state yields the characteristic equation,

$$\begin{vmatrix} \psi D_{\text{eff}} I^* + \omega + \lambda & Q(\lambda) \\ \psi D_{\text{eff}} I^* & R(\lambda) \end{vmatrix} = 0 \tag{49}$$

where

$$Q(\lambda) = \psi D_{\text{eff}} S^* - \frac{\omega \psi D_{\text{eff}}}{1 - e^{-\omega\tau}} e^{-(\psi D_{\text{eff}} I^* + \omega)\tau} \frac{1 - e^{-\lambda\tau}}{\lambda} , \tag{50}$$

$$\begin{aligned}
 R(\lambda) &= \psi D_{\text{eff}} S^* - \mu - \omega - \lambda - \frac{\omega \psi D_{\text{eff}}}{1 - e^{-\omega\tau}} e^{-(\psi D_{\text{eff}} I^* + \omega)\tau} \\
 &\times \left\{ \frac{1 - e^{-\lambda\tau}}{\lambda} - \mu \frac{1 - e^{-(\lambda + \mu - \psi D_{\text{eff}} I^*)\tau}}{\lambda(\lambda + \mu - \psi D_{\text{eff}} I^*)} + \mu \frac{e^{-\lambda\tau} - e^{-(\lambda + \mu - \psi D_{\text{eff}} I^*)\tau}}{\lambda(\mu - \psi D_{\text{eff}} I^*)} \right\} .
 \end{aligned} \tag{51}$$

Fig. 7. Dynamics of the model with fixed age-at-roguing for parameter values as used in Fig. 3a

For an introduction in the stability analysis of delay differential equations we refer to Gurney and Nisbet (1985). Following the procedure outlined in Sect. 6 the characteristic equation was solved for pairs of complex eigenvalues on the imaginary axis. τ was considered to be a function of the other parameters in such a way that for any given combination ($l_{\max}$, ψD_{eff} , μ , ω) the value of τ equalled the value of $\hat{A}_{\max}$ in the full model for this parameter combination. If a fixed delay would result in unstable steady-states, one expects to find a stability diagram closely resembling that of the full model. Despite extensive searches throughout the parameter space, paying special attention to the parameter regions where stability boundaries are expected, we have not found any parameter combination for which eigenvalues lie on the imaginary axis. From this we conclude that the non-trivial steady-state (46) is stable irrespective of parameter values. To verify this conclusion the model was numerically solved using the program SOLVER (Mass et al., 1984). This package is especially developed for the numerical solution of delay differential equations. These simulations also show convergence to the non-trivial steady-state irrespective of parameter values. An example, using the parameter values as in Fig. 3a, is given in Fig. 7. We conclude that a fixed age at roguing does not result in periodic solutions.

From the above discussion we conclude that periodic solutions are not a feature of the population dynamics of the infectious disease. Periodic solutions are due to the interaction of the infectious disease with the orchard management. This management introduces a time delay in the form of a maximum age of a tree, $A_{\max}$. The fact that $A_{\max}$ is a dynamically varying time delay results in periodic solutions of the model. A fixed delay would not result in periodic solutions.

8 A biological interpretation

In this section we discuss the loss of stability of the internal steady state once more. We will argue that the loss of stability results from the interplay between the final value of an S-I-R epidemic without death (Kermack and

McKendrick, 1927) and the roguing and replanting management. Consider again the evolution of the population distribution as a function of the fraction of infected sites, during one cycle period, for parameter combinations in the lower loop of the stability diagram (Fig. 2). During the phase of low epidemic activity, almost all trees have l values just below the roguing threshold. The number of trees in this cohort decreases due to death-rate ω . During the phase of epidemic outbreak all remaining trees in this cohort are rogued and replaced. The new cohort of trees increases in infection level during an approximately two year period. When the epidemic peters out all trees have again l values close to the roguing threshold.

Now consider the situation where, from an unstable situation where the above description applies, we suddenly decrease the roguing threshold. This causes the trees, increasing in l during the epidemic outbreak, to be pushed over the roguing threshold before the epidemic has petered out. The period without epidemic activity, where trees have a fraction of infected sites just below the roguing threshold, disappears. Consequently the number of infectious sites shows less fluctuations. The roguing and replanting peak will flatten out due to the tree death-rate ω . One can imagine that this finally leads to a stable steady-state.

Obviously the petering out of the epidemic results in a cohort of trees 'waiting' for a long period at values of the fraction of infected sites slightly smaller than the roguing threshold. Combining this with the observation that during one cycle the whole orchard is replanted we can conclude that after the roguing and replanting peak the orchard consists mainly of completely susceptible trees. The dynamics of the system can then, caricaturally, be separated in two distinct phases. First the time span where there is no epidemic activity and all trees have l values close to the roguing threshold. Secondly a phase that starts with a completely uninfected orchard. In the later phase an S-I-R type epidemic takes place. The S-I-R epidemic finally peters out, leaving the trees at their 'final' infection level. Separating the two phases in this way, during the second phase a Kermack and McKendrick (1927) type epidemic takes place. In our notation this model reads:

$$\begin{aligned}\frac{dS}{dt} &= -\psi D_{\text{eff}} SI \\ \frac{dI}{dt} &= \psi D_{\text{eff}} SI - (\mu + \omega)I \\ \frac{dR}{dt} &= (\mu + \omega)I\end{aligned}\tag{52}$$

where S , I and R are the total fraction of susceptible sites, infectious sites and removed sites in the orchard, respectively. The fraction of infected sites the trees will have after the epidemic is given by the final value theorem developed by Kermack and McKendrick (1927) (see also Metz, 1978). The final fraction

of infected sites, l_∞ , is calculated from

$$l_\infty = 1 - \exp \left\{ \frac{\psi D_{\text{eff}}}{\omega + \mu} l_\infty \right\}. \quad (53)$$

The mechanism discussed above implies that the internal steady-state is stable when $l_{\text{max}} < l_\infty$. The internal steady-state loses stability when the roguing threshold is increased above the final value of the S-I-R epidemic. Figure 2 shows that the final value l_∞ accurately predicts the lower stability boundary accurately.

Effects of parameter changes on the dynamics of the system can be explained if we assume that the ‘piling’ up just below the roguing threshold is the correct explanation of the loss of stability of the internal steady-state. Increasing tree death-rate increases the rate of inflow of susceptible material during the phase of low epidemic activity. The threshold for epidemic increase is then passed after a shorter time interval than for smaller tree death-rates. Increasing ω thus should result in shorter periods of the periodic solutions. For $\mu = 10$, $\psi D_{\text{eff}} = 20$, $l_{\text{max}} = 0.82$ we found for $\omega = 0.10, 0.15, 0.20, 0.25$ a period of approximately 7.9, 6.3, 5.1, 4.0 year, respectively (Fig. 2c). When the average lifetime of a tree is close to but below the duration of the epidemic phase the threshold for epidemic increase is passed before the epidemic has petered out. Increasing tree death-rate will thus finally result in stable-states. This explains the stabilising effect of increasing ω as seen in Fig. 2c. Passing the stability boundary by increasing l_{max} , suddenly adds a long phase of no epidemic activity to the dynamics of the system. This results in a sudden increase in the age at roguing, which explains the large ‘jump’ in the age at roguing shown in Fig. 5. In the stable situation there is no phase of low epidemic activity. This thus also explains why the range of ages at roguing observed for periodic solutions does not contain the steady-state value. Increasing the death-rate of infectious sites the final value of the S-I-R epidemic becomes smaller. This implies that for large death-rates l_∞ is smaller than l_{max} resulting in the loss of stability of the internal steady-state. This effect of infectious site death-rate is observed in Fig. 2b. The same arguments hold for decreasing tree density.

The mechanism by which the epidemic outbreak increases the fraction infected sites to close to the roguing threshold, where the trees ‘pile up’ and remain till the next epidemic outbreak, explains all dependencies of the stability characteristics as discussed. We thus conclude that this mechanism is responsible for the loss of stability of the internal steady-state.

The above discussion is based on results of simulations in the lower loop (Fig. 2). For parameter combinations in other parts of the unstable domain additional phenomena are observed. In the second loop two peaks are found. During each epidemic outbreak the peak with largest values of l passes the roguing threshold. The peak with the smaller l values moves during the outbreak to l values close to the roguing threshold. We have not found a quantitative explanation comparable with the final value mechanism for this phenomenon. Intuitively the development of two peaks can however be

understood. For values of $l_{\max}$ considerable larger than l_{∞} the S-I-R epidemic leaves the trees at a value of the fraction of infected sites far below the roguing threshold. During the next epidemic outbreak trees replanted during the phase without epidemic activity start to increase in fraction of infected sites sharply. It will however still take a considerable time before the other trees, already infected, reach the roguing threshold. When the epidemic has petered out before these trees reach the roguing threshold for groups of trees, with distinct infection levels, are left.

9 Concluding remarks

In this paper we investigated the effect of the much applied roguing and replanting management strategy for disease control, on the dynamics of an S-I-R epidemic in an orchard with constant tree density. It is shown that the interaction between the population dynamics of the infectious disease and the roguing and replanting management leads to the observed periodic solutions. Roguing when the number of infections on a tree reaches a threshold, results in a dynamically varying age at roguing. It is this dynamically varying time delay that results in periodic solutions. The effect of dynamically varying time delays on the stability of steady-states of population dynamic models, is little studied. De Roos et al. (1990) found a dynamically varying juvenile period to be destabilising in a model of *Daphnia* populations. Nisbet and Gurney (1983) found a dynamically varying juvenile period to be stabilising in a model for a damselfly population.

The objective of orchard holders obviously is to reduce disease severity and therewith to increase the trees productive lifespan. The steady-state analysis shows that increasing the roguing threshold beyond a certain 'key-value' sharply increases tree lifespan. This 'key-value' depends on the model parameters. Whether an increase of the roguing threshold increases the productive life-span of the tree or actually decreases production due to the effects of the disease on tree physiology depends on the disease under consideration. Increasing the death-rate of infectious sites, by applying chemical curatives, also increases tree lifespan. The same hold for decreasing the density of trees in the orchard. Figure 1 shows that tree lifespan becomes virtually infinite for death-rates of infectious sites and tree densities far below the values needed to drive the disease to extinction. This implies that one does not have to drive the disease to extinction to get an economically productive orchard. The stability analysis shows that changing the parameters such that the tree lifespan is sufficiently large also implies that large amplitude periodic fluctuations are likely to occur. When the life history parameters of the disease are such that the chosen roguing threshold is slightly above the final value of the S-I-R epidemic, periodic outbreaks of the disease are to be expected. Periodic outbreaks in turn have consequences for orchard management. It is beyond the scope of this paper to treat this interplay between orchard management and disease dynamics in more detail.

Acknowledgements. We wish to thank Michael Shaw (Reading University, England) and Mike Jeger (Natural Resource Institute, England) for putting us on this track. We are grateful to Odo Diekmann (Centre of Mathematics and Computer Science, The Netherlands) and Joost van Opheusden (Agricultural University, The Netherlands) for helpful discussions during the research, and J. Grassman for discussions and comments on an early draft of the manuscript. The comments of an anonymous referee helped us improving the paper.

Appendix A: The model without roguing

In this appendix we derive the S-I-R model discussed in Sect. 8, from the full model developed in Sect. 3. When there is no roguing $l_{\max} = 1.0$. If we let $l_{\max} \uparrow 1$, $A_{\max}(t) \uparrow \infty$. From (21) it is seen that in this limit $m(t, 0) = \omega$, which is consistent with no roguing. Integrating (20) along characteristics now yields, $m(t, a) = \omega \exp(-\omega a)$ for $a \geq 0$. Now consider the definitions (37), (38) and (39). Differentiating (37) with respect to time we find

$$\frac{dS}{dt} = \int_0^\infty \left\{ -\frac{\partial l}{\partial t} m + \frac{\partial m}{\partial t} (1-l) \right\} da. \quad (\text{A.1})$$

Substituting (14) and (20) leads to

$$\frac{dS}{dt} = - \int_0^\infty \frac{\partial(1-l)m(t, a)}{\partial a} da - \{\psi D_{\text{eff}} i(t) + \omega\} \int_0^\infty (1-l)m da. \quad (\text{A.2})$$

Integrating (20) along characteristics, using the definition of $S(t)$ and boundary condition (15) we arrive at the o.d.e.

$$\frac{dS}{dt} = -\psi D_{\text{eff}} I(t) S(t) + \omega(1-S(t)). \quad (\text{A.3})$$

Upon differentiating (38) one obtains

$$\frac{dI}{dt} = \int_0^\infty \left\{ \frac{\partial(l-r)}{\partial t} m + \frac{\partial m}{\partial t} (l-r) \right\} da. \quad (\text{A.4})$$

Substituting (14) and (17) leads to

$$\begin{aligned} \frac{dI}{dt} = & \int_0^\infty -\omega \frac{\partial(l-r)e^{-\omega a}}{\partial a} da + \psi D_{\text{eff}} i(t) \int_0^\infty (1-l)m da \\ & - (\mu + \omega) \int_0^\infty (l-r)m da. \end{aligned} \quad (\text{A.5})$$

With the boundary conditions (15) and (18) and our definitions of the variables this leads to

$$\frac{dI}{dt} = \psi D_{\text{eff}} I(t) S(t) - (\mu + \omega) I(t). \quad (\text{A.6})$$

Differentiating (39) yields

$$\frac{dR}{dt} = \int_0^\infty \left\{ \frac{\partial r}{\partial t} m + \frac{\partial m}{\partial t} r \right\} da . \quad (\text{A.7})$$

Substituting (17) and (20) leads to

$$\frac{dR}{dt} = \int_0^\infty -\omega \frac{\partial r e^{-\omega a}}{\partial a} da + \mu \int_0^\infty (l - r) m da - \omega \int_0^\infty m r da . \quad (\text{A.8})$$

With boundary condition (18) and the definitions of the variables

$$\frac{dR}{dt} = \mu I(t) - \omega R(t) . \quad (\text{A.9})$$

Appendix B: roguing at a fixed age

In this appendix we derive the delay differential equation formulation of the model with roguing at a fixed age. In terms of the full model, Eqs. (14) through (21), this amounts to substituting τ for $A_{\max}(t)$ in (19), (20) and (21), and deleting (16) from the model. To ease the derivation we introduce an additional equation for the dynamics of the fraction of infectious sites on a tree of age a at time t , $s(t, a)$. Since $s(t, a) = l(t, a) - r(t, a)$ we can derive from (14), (15), (17) and (18) that the dynamics of $s(t, a)$ is described by

$$\frac{\partial s}{\partial t} + \frac{\partial s}{\partial a} = \psi D_{\text{eff}} i(t)(1 - l) - \mu s \quad (\text{B.1})$$

with boundary condition

$$s(t, 0) = 0 . \quad (\text{B.2})$$

Substitution of (22) in (B.1) and subsequently integrating along characteristics we find

$$s(t, a) = \left\{ e^{-\mu a} - e^{-\psi D_{\text{eff}} \int_{t-a}^t I(\xi) d\xi} + \mu \int_0^a e^{-\psi D_{\text{eff}} \int_{t-a}^{t-\sigma} I(\xi) d\xi} e^{-\mu \sigma} d\sigma \right\} . \quad (\text{B.3})$$

The equations for S and I: Definition (38) for the fraction of infectious sites now takes the form

$$I(t) = \int_0^\tau s(t, a) m(t, a) da . \quad (\text{B.4})$$

Differentiating with respect to time yields

$$\frac{dI}{dt} = \int_0^\tau \left\{ \frac{\partial s}{\partial t} m + \frac{\partial m}{\partial t} s \right\} da . \quad (\text{B.5})$$

Substituting (B.1) and (20) we find

$$\frac{dI}{dt} = \int_0^\tau -\frac{\partial sm}{\partial a} da + \psi D_{\text{eff}} I \int_0^\tau (1-l)m da - (\omega + \mu) \int_0^\tau sm da . \quad (\text{B.6})$$

Using the definitions of the variables and boundary condition (B.2) yields

$$\frac{dI}{dt} = -s(t, \tau)m(t, \tau) + \psi D_{\text{eff}} IS - (\omega + \mu)I . \quad (\text{B.7})$$

Differentiating (37), where the upper integrant is replaced by τ , with respect to time and substituting (14) and (20) yields (A.2), here again the upper integrant is replaced by τ . Finally we use the definitions of the variables and boundary condition (15) to arrive at

$$\frac{dS}{dt} = -\psi D_{\text{eff}} IS - \omega S - (1 - l(t, \tau))m(t, \tau) + m(t, 0) . \quad (\text{B.8})$$

Both equations (B.7) and (B.8) contain a factor $m(t, \tau)$. Differentiating (21) with respect to time yields

$$m(t, 0) = m(t, \tau) + \omega . \quad (\text{B.9})$$

Integrating (20) along characteristics and recursive substitution of (B.9) we find

$$\begin{aligned} m(t, \tau) &= m(t - \tau, 0)e^{-\omega\tau} = \{m(t - \tau, \tau) + \omega\}e^{-\omega\tau} \\ &= \{m(t - 2\tau, 0)e^{-\omega\tau} + \omega\}e^{-\omega\tau} \\ &= \{\{m(t - 2\tau, \tau) + \omega\}e^{-\omega\tau} + \omega\}e^{-\omega\tau} + \dots \\ &= \left\{ \sum_{k=0}^{\infty} \omega(e^{-\omega\tau})^k \right\} e^{-\omega\tau} = \frac{\omega}{e^{\omega\tau} - 1} . \end{aligned} \quad (\text{B.10})$$

Next use definitions (43) and (44) to write

$$l(t, \tau) = (1 - U(t)) \quad (\text{B.11})$$

$$s(t, \tau) = \{e^{-\mu\tau} - U(t) + \mu V(t)\} .$$

Finally (B.7) and (B.8) take the form

$$\begin{aligned} \frac{dS}{dt} &= -\psi D_{\text{eff}} IS + \omega(1 - S) + \frac{\omega}{e^{\omega\tau} - 1}(1 - U) \\ \frac{dI}{dt} &= \psi D_{\text{eff}} IS - (\mu + \omega)I - \frac{\omega}{e^{\omega\tau} - 1}(e^{-\mu\tau} - U + \mu V) . \end{aligned} \quad (\text{B.12})$$

The equations for U and V . Differentiating (43) with respect to time yields

$$\frac{dU}{dt} = U \frac{d}{dt} \left\{ -\psi D_{\text{eff}} \int_{t-\tau}^t I(\xi) d\xi \right\}. \quad (\text{B.13})$$

Which leads to

$$\frac{dU}{dt} = \psi D_{\text{eff}} \{I(t - \tau) - I(t)\} U. \quad (\text{B.14})$$

Upon differentiating (44) with respect to time we find

$$\frac{dV}{dt} = \psi D_{\text{eff}} I(t - \tau) V(t) - \int_0^\tau e^{-u\sigma} e^{-\psi D_{\text{eff}} \int_{t-\tau}^{t-\sigma} I(\xi) d\xi} \psi D_{\text{eff}} I(t - \sigma) d\sigma. \quad (\text{B.15})$$

Integrating by part the second term on the right hand side and substituting the definitions of the variables finally yields

$$\frac{dV}{dt} = \psi D_{\text{eff}} I(t - \tau) V(t) - e^{-\mu\tau} + U - \mu V. \quad (\text{B.16})$$

Appendix C: the characteristic equation of the full model

In this appendix we will present the derivation of the characteristic Eq. (32). First define a small perturbation from the steady state as:

$$x(t) = \hat{i} - i(t); \quad y(t) = \hat{b} - b(t); \quad z(t) = \hat{A}_{\text{max}} - A_{\text{max}}(t). \quad (\text{C.1})$$

Using these definitions it is readily seen that

$$e^{-\psi D_{\text{eff}} \int i(t) dt} = e^{-\psi D_{\text{eff}} \int (\hat{i} + x(t)) dt} \approx e^{-\psi D_{\text{eff}} \hat{i}} \left(1 - \psi D_{\text{eff}} \int x(t) dt \right). \quad (\text{C.2})$$

$$\begin{aligned} \int_0^{A_{\text{max}}(t)} f(\xi) d\xi &= \int_0^{\hat{A}_{\text{max}} + z(t)} f(\xi) d\xi = \int_0^{\hat{A}} f(\xi) d\xi + \int_{\hat{A}}^{\hat{A} + z(t)} f(\xi) d\xi \\ &\approx \int_0^{\hat{A}} f(\xi) d\xi + f(\hat{A}) z(t). \end{aligned} \quad (\text{C.3})$$

The second part shows how the perturbation in the age at roguing is accounted for in the linearization.

We first linearize Eq. (24). Using the definitions (C.1) and Eq. (C.2) we find

$$\begin{aligned} \hat{i} + x(t) &\approx \int_0^{\hat{A} + z(t)} \hat{b} e^{-(\mu + \omega)a} \left(1 + \mu \int_0^a e^{(\mu - \psi D_{\text{eff}} \hat{i})\sigma} d\sigma \right. \\ &\quad \left. - \mu \psi D_{\text{eff}} \int_0^a e^{(\mu - \psi D_{\text{eff}} \hat{i})\sigma} \int_{t-a}^{t-a+\sigma} x(\xi) d\xi d\sigma \right) da \\ &\quad + \int_0^{\hat{A} + z(t)} e^{-(\mu + \omega)a} y(t-a) \left(1 + \mu \int_0^a e^{(\mu - \psi D_{\text{eff}} \hat{i})\sigma} d\sigma \right) da \end{aligned}$$

$$\begin{aligned}
& - \int_0^{\hat{A} + z(t)} \hat{b} e^{-(\omega + \psi D_{\text{eff}} \hat{t})a} \left(1 - \psi D_{\text{eff}} \int_{t-a}^t x(\xi) d\xi \right) da \\
& - \int_0^{\hat{A} + z(t)} e^{-(\omega + \psi D_{\text{eff}} \hat{t})a} y(t-a) da
\end{aligned} \tag{C.4}$$

and applying (C.3) we arrive at the linear equation

$$\begin{aligned}
x(t) = & - \hat{b} \mu \psi D_{\text{eff}} \int_0^{\hat{A}} e^{-(\mu + \omega)a} \int_0^a e^{(\mu - \psi D_{\text{eff}} \hat{t})\sigma} \int_{t-a}^{t-a+\sigma} x(\xi) d\xi d\sigma da \\
& + \hat{b} \psi D_{\text{eff}} \int_0^{\hat{A}} e^{-(\omega + \psi D_{\text{eff}} \hat{t})a} \int_{t-a}^t x(\xi) d\xi da \\
& + \int_0^{\hat{A}} e^{-(\mu + \omega)a} \left(1 + \mu \int_0^a e^{(\mu - \psi D_{\text{eff}} \hat{t})\sigma} d\sigma \right) y(t-a) da \\
& - \int_0^{\hat{A}} e^{-(\omega + \psi D_{\text{eff}} \hat{t})a} y(t-a) da + C^* z(t)
\end{aligned} \tag{C.5}$$

where

$$C^* = - \frac{\hat{b} \psi D_{\text{eff}} \hat{t}}{\mu - \psi D_{\text{eff}} \hat{t}} (e^{-(\mu + \omega)\hat{A}} - e^{-(\omega + \psi D_{\text{eff}} \hat{t})\hat{A}}) . \tag{C.6}$$

Next, substituting (C.1) in Eq. (22) we find

$$\int_{t-\hat{A}-z(t)}^t (\hat{i} + x(\xi)) d\xi \approx \hat{i} \hat{A} + \hat{i} z(t) + \int_{t-\hat{A}}^t x(\xi) d\xi = - \frac{1}{\psi D_{\text{eff}}} \ln(1 - l_{\max}) \tag{C.7}$$

which gives us the linear equation

$$z(t) = - \frac{1}{\hat{t}} \int_{t-\hat{A}}^t x(\xi) d\xi . \tag{C.8}$$

Finally, substituting (C.1) in (23) gives

$$\begin{aligned}
\hat{b} + y(t) = & (\hat{b} + y(t - \hat{A} - z(t))) \left(1 - \frac{d\hat{A} + z(t)}{dt} \right) e^{-\omega(\hat{A} + z(t))} + \omega \\
& \approx \hat{b} e^{-\omega \hat{A}} - \hat{b} e^{-\omega \hat{A}} \left(\omega z(t) + \frac{dz(t)}{dt} \right) + e^{-\omega \hat{A}} y(t - \hat{A}) + \omega
\end{aligned} \tag{C.9}$$

which yields the linear equation

$$0 = \hat{b} e^{-\omega \hat{A}} \left(\frac{dz}{dt} + \omega z(t) \right) - y(t - \hat{A}) e^{-\omega \hat{A}} + y(t) . \tag{C.10}$$

Finally substituting the trial solution

$$x(t) = x_0 e^{\lambda t}; \quad y(t) = y_0 e^{\lambda t}; \quad z(t) = z_0 e^{\lambda t} \quad (\text{C.11})$$

we arrive at the characteristic Eq. (32).

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