Eur J Plant Pathol (2009) 125:435–444
DOI 10.1007/s10658-009-9493-4

Modelling and control of non-persistent plant virus transmission for annual production cycles

Diego Viteri · Luis F. Gordillo

Received: 14 September 2008 / Accepted: 28 May 2009 / Published online: 1 July 2009

Abstract When an insect carrying a non-persistent virus punctures a plant it loses part, or may be all, of its viral load. Using a differential equation model, we show that this is a critical factor affecting disease incidence levels when crops are under annual production cycles. Computer simulations suggest that relatively low vector pressure at the beginning of planting cycles decrease the disease progression. The model provides also approximations to disease incidences in subsequent plantings. Conditions for incidences to decrease or increase in time are supplied, which may be useful to assess the impact of some control strategies.

Keywords Non-persistent virus transmission · Annual production cycle · Plant virus dynamics

D. Viteri
Department of Crop Protection,
University of Puerto Rico-RUM,
Mayagüez, Puerto Rico

L. F. Gordillo (✉)
Department of Mathematical Sciences,
University of Puerto Rico-RUM,
Mayagüez, Puerto Rico
e-mail: luis.f.gordillo@gmail.com

Introduction

The majority of viruses on plants depend on having an efficient method of plant-to-plant dispersal for their survival. Plant viruses generally rely on specific types of vectors for their spread. The most common plant virus vectors are arthropods, and of these, the great majority are of the insect order Hemiptera (Burnett and Kawchuk 2002; Ng and Falk 2006). Plant viruses take advantage of the highly invasive character of some vector species to spread, (American Association for the Advancement of Science 2007; Morales and Anderson 2001), causing outbreaks of viral diseases in vegetable crops every year, damaging world's food supplies and bio-fuel sources as well as causing severe economic losses worldwide. For instance, in the USA alone, crop diseases cause billions of dollars in losses each year, mainly attributed to invasive microorganisms, (Pimentel et al. 2000).

The processes of virus transmission between plants and vectors have a high degree of complexity, each case with its own biological characteristics. The majority of plant viruses use the non-circulative method of transmission. In this method, viruses do not cross vector cell membranes and are transported either on the vector's surface or within its mouth parts or foregut, (Gray and Banerjee 1999; Nault 1997). Non-circulative viruses are further classified into non-persistent and semi-persistent. Viruses in the latter class

remain in the vector foregut and are retained for several days or weeks. Non-persistent viruses however, attach to the vector stylet and are retained just for several minutes or hours (usually < 12 h), sometimes without latency time, as occurs with the transmission of *Copwea mild mottle virus* (CPMMV), (Muniyappa and Reddy 1983), a type of Carlavirus transmitted by *Bemisia tabaci*. Other examples of this type of viruses include Alfamoviruses (transmitted by *Myzus persicae*) and Machlomoviruses (transmitted by *Diabrotica* sp.), among others, (Gray and Banerjee 1999; Morales and Anderson 2001).

Although important technical advances in molecular biology have been made in recent years, the intricate process of transmission of non-persistent viruses in plants has not been completely understood. Intense efforts have been made studying the processes that lead to successful virus transmission, inside plant cells and within insect mouth parts, (Ng and Falk 2006). For example, it was recently discovered, through the use of electron microscopy, how the tip of the maxillary stylets of aphids may carry *Cauliflower mosaic virus*, (Uzest et al. 2007). Also, it has been observed in experiments that the process of inoculation of non-persistent viruses is done in several stages and takes just several seconds, after which the virus load carried by the vector is completely lost or, at least, significantly reduced, (Martin et al. 1997; Iwaki et al. 1982).

Even though the biology of the transmission processes at the molecular level is complex, it is possible to create mathematical models to simulate the dynamics of the disease at the population level including relevant information from molecular phenomena. Our aim is to present a differential equation model which describes the dynamics of the transmission of a non-persistent virus in a plant population where vectors reduce or lose their virus load after inoculation, as mentioned in the previous paragraph. We found that this becomes important when the plant population is under an annual production cycle. This scenario can be observed, for instance, in soybean production, where in general there is a three month period between planting and harvesting. In tropical countries that produce soybean, appropriate weather conditions allow these periodic production cycles within a year. Not long ago, a Carlavirus related to CPMMV damaged large areas of soybean plantations in west central Brazil, (Almeida et al. 2005), and recently another has been detected in Puerto Rico, (Rodriguez et al. 2008).

The use of mathematical models to describe the dynamics of diseases in populations, showing how the infection spreads among susceptible individuals, has been extensive in theoretical epidemiology. The mathematical modelling approach is successful in describing the spread of diseases in human and animals. For vector-transmitted diseases in plants, the models were inspired from the work of malariologists at the beginning of the 20^{th} century. The first efforts in plant virus disease modelling did not consider specific forms of transmission characteristics, (Chan and Jeger 1994); however, subsequent models incorporate vector population and epidemiological dynamics, giving more adequate descriptions of disease development. Although very general models can be constructed, (Jeger et al. 1998; Madden et al. 2000), they have to be adjusted to specific disease features due to specific biological characteristics. An excellent review of differential equation models for plant virus spread can be found in Jeger et al. (2004).

Materials and methods

Compartmentalisation

For modelling purposes we only consider the interactions between plants and vectors, isolated from the rest of the environment. Plants can be classified as susceptible or infected. Similarly, vectors can be classified as effective carriers (vectors ready to transmit the virus) or virus-free. Sometimes a vector carrying the virus may be unable to transmit the disease, because the virions were ingested or suffered genetic mutations, (Gray and Banerjee 1999). We will include these cases in the virus-free state. Thus, we have divided both populations, plants and vectors, in two compartments each, where individuals may move from one state to another. However, the flow between compartments will be different in the two populations. The change from susceptible to infected

plants will be considered irreversible, for instance, Carlavirus may cause irreparable stem necrosis on soybean. The symptoms include vein and petiole necrosis, stunting, molting and seed malformation. In severe infection, plants die before reproductive stages, reducing seed production, (Almeida et al. 2005; Hartman et al. 1999; Rodriguez et al. 2008). Vectors, playing the role of transporters, switch their status between effective carriers and virus-free as long as they live.

Modelling hypothesis

The life time of non-persistent viruses is short if they are not able to find an appropriate host. If inoculation is not made soon after acquisition, the vector becomes virus-free, on average, after a period of time $1/g$. For non-persistent viruses this time can vary from being very short, 30 min, to several hours, (Jeger et al. 1998).

We suppose that susceptible and infected vectors are homogeneously mixed among the hosts, which are uniformly distributed in space; thus the probability of each host receiving a bite from a vector is the same for all of them. The rate at which susceptible plants are infected, b, can be described as $b = h_i \sigma \hat{T}$, where $\hat{T}$ is the average time spent per visit on a plant, h_i^{-1} is the mean time an insect must feed on a plant for inoculation to occur and σ is the number of plants visited per day per vector. Similarly, the rate at which susceptible vectors are infected, a, can be written as $a = h_a \sigma \hat{T}$, where h_a^{-1} is the mean time an insect must feed on a plant for aquisition to occur. Estimated values for these parameters can be found in Jeger et al. (1998), where $h_{i,a} \approx 10^3$ and $0 \leq \sigma \hat{T} \leq 0.02$ for non-persistent viruses. We have assumed that rates of acquisition and inoculation are equal (Jeger et al. 1998; Madden et al. 2000; Zhang et al. 2000).

We represent the number of susceptible and infected hosts at time t with $x(t)$ and $y(t)$ respectively. The number of virus-free vectors at time t is $z(t)$ and effective carriers, $w(t)$. For notational convenience we write x, y, z, w instead of $x(t)$, $y(t)$, $z(t)$, $w(t)$. We also incorporate the following set of additional assumptions:

- The number of hosts is constant and equal to K, i.e. $x + y = K$ for all times.
- The number of vectors has reached its saturation level in the environment and is constant, equal to M, i.e. $z + w = M$ for all times. In real contexts, fluctuations around this number are, of course, expected. Local losses are assumed always to be balanced by immigrants from the same compartmental category.
- The age structure in the host and vector populations is ignored.
- The existence of only one class of susceptible (infected) vector and host.
- Susceptible hosts acquire the virus only through the bite of an infected vector.
- Absence of latency periods in hosts.

The period for the crop production process (the time between sowing and harvest) is denoted with T. We assume the time between a harvest and the next sowing as negligible. During this time, a reduction in the vector population is expected, due to the lack of an appropriate environment for survival. However, we want to assess the scenario where the vector population has a continuous ideal environment to develop, because of a large area of cropping, or because of the existence of adjacent plantations, not necessarily the same crop, that are able to sustain the vector population during short lapses of time. Thus, the reduction in the vector population may be thought as negligible. If the time between harvest and the next sowing is sufficiently long, the vector population may decrease substantially due to death or migration. Our case study is, consequently, the worst case scenario.

Flow between compartments

We now describe how the flow of individuals is modelled between compartments. The amount of susceptible hosts is reduced at a rate which is proportional to the number of contacts between the susceptible hosts and effective carriers present at time t. Denoting the derivative of a function with respect to time with $'$, we can express the claim in mathematical terms using the differential equation $x' = -b\,x\frac{w}{M}$, where b and M are the constants defined above. In epidemiology this is called standard incidence (Diekmann and Heesterbeek

2000). The rate at which the group of susceptible plants decreases is the same as the rate at which the group of infected plants increases, giving us the second equation, $y' = bx\frac{w}{M}$. The rate at which the number of effective carriers changes is composed of the rate at which virus-free vectors puncture diseased plants and acquire the virus, $bz\frac{y}{K}$, the rate at which effective carriers become virus-free because the virus has not been inoculated on time, $-gw$, and the rate at which effective carriers become virus-free because they lose the virus load after puncturing a susceptible host, $-\phi bx\frac{w}{M}$, where ϕ represents the average fraction of vectors that becomes virus-free. Thus, $z' = -bz\frac{y}{K} + \phi bx\frac{w}{M} + gw$ and, similarly, $w' = bz\frac{y}{K} - \phi bx\frac{w}{M} - gw$.

Because $x = K - y$ and $z = M - w$, we can reduce the study of the four equations for x', y', z' and w' to the study of only two:

$$y' = b(K - y)\frac{w}{M}, \tag{1}$$

$$w' = b(M - w)\frac{y}{K} - \phi b(K - y)\frac{w}{M} - gw. \tag{2}$$

These are the governing equations in each cycle, or in other words, when $t \neq kT$, $k = 0, 1, 2, \ldots$. At the time points of harvest, that is $t = kT$, the number of infected plants evidently reduces to zero, that is, the infected population of plants has a negative increment $\Delta y = -\lambda(t)$, where $\lambda(t)$ is equal to the total amount of individuals at the end of a cycle (see Appendix). We have defined what is known as an impulse differential equation of a simple type. The general theory for equations with impulses can be found, for example, in Lakshmikantham et al. (1989). We can make our equations to look simpler after replacing $\hat{y} = y/K$, $\hat{w} = w/M$, re-scaling the time to $\hat{t} = bt$ and dropping the hats:

$$y' = (1 - y)w, \tag{3}$$

$$w' = (1 - w)y - \phi P(1 - y)w - Qw, \tag{4}$$

with $P = K/M$ and $Q = g/b$.

Results

We can extract valuable information about the system by solving our system numerically (see Appendix). In Fig. 1, we use parameter values corresponding to the transmission of Carlavirus by whiteflies among soybean plants to perform computations with the model. Our interest is to observe what will be the overall behaviour of the disease incidence at the end of each cycle of production. In (a), the incidences increase in time, opposed to what happens in (b), where they decrease. Figure 1 only shows computations along three cycles, but the same phenomenon is seen if we extend to a larger number of them. The difference between the two figures is because in (b) the solution was computed with a lower value of vector population, M, than in (a). This suggests the existence of a threshold value for the vector population, say M_0, that will cause increasing incidences if $M > M_0$ and decreasing incidences if $M < M_0$ (it is possible to rule out any other kind of behaviour, see Appendix). Incidence of the disease in plants becomes smaller if the vector population is reduced. The best scenario is when $M = 0$, which, in practice, could be a difficult goal to achieve. However, by just reducing the value of M under the threshold, without killing all the vector population, it is possible to eventually drive the disease to extinction. Therefore, we would like to compute the threshold value M_0 or, at least, an estimate, M_{ap}. An approximation to the solution of our model allows us to compute this estimate, providing a condition for increasing disease incidences in subsequent planting cycles:

$$M_{ap} = \frac{\rho \cosh\left(\frac{\rho T}{2}\right) - D \sinh\left(\frac{\rho T}{2}\right)}{\rho e^{\frac{DT}{2}} - 2w((k-1)T)\sinh\left(\frac{\rho T}{2}\right)} > 1, \tag{5}$$

where $D = 1 + \phi K/M + g/b$ and $\rho^2 = D^2 + 4$. Figure 2a shows the graph of M_{ap} depending on the vector population M, computed for $\phi = 1$ and with the other parameters adjusted to values corresponding to the transmission of Carlavirus by whiteflies in soybean. The agreement with the results in Fig. 1 is evident. However, it has to be pointed out that these results have to be handled with caution: we have neglected random perturbations, and made approximations to the average behaviour of the system.

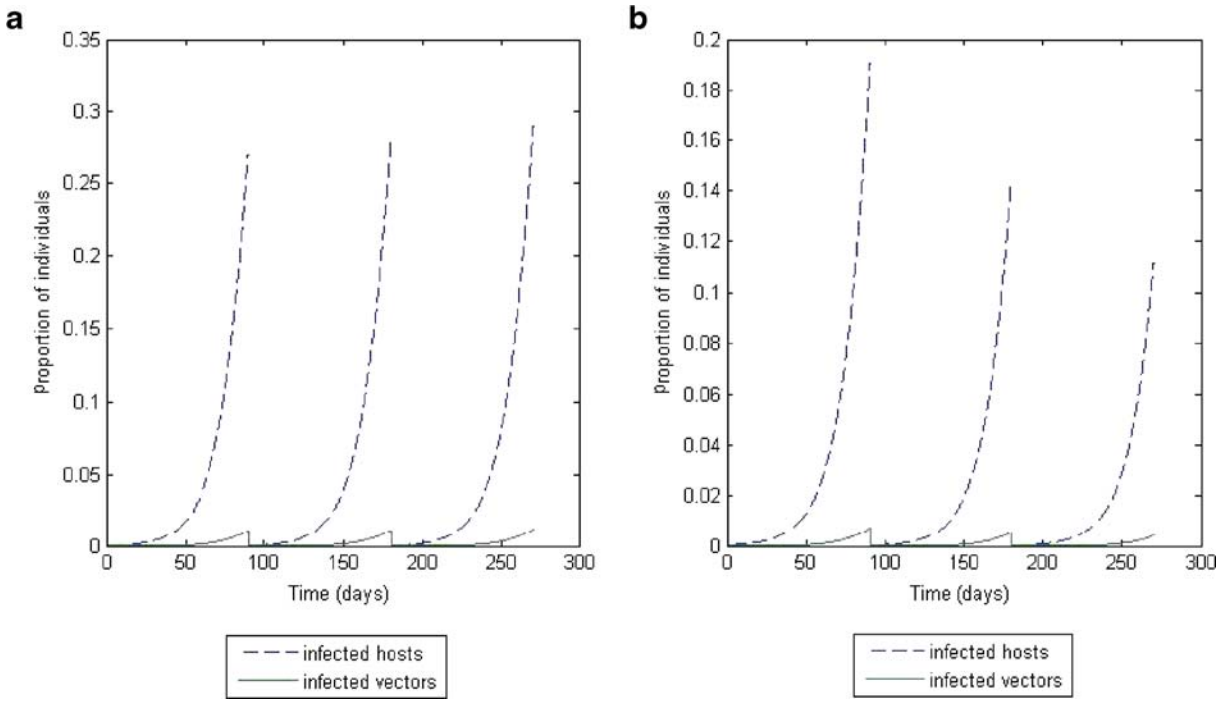

Fig. 1 Proportion of infected hosts (*dashes*) and infected vectors (*continuous line*). **a** With values of $K = 1,000$ and $M = 2,500$ the sequence y_k increases. **b** If $M = 500$ then the monotonicity changes. For this figure, we used parameter values $b = 1.93$, $1/g = 30$ min (Jeger et al. 1998), $\phi = 1$, $w_0 = 0.01$, and assume three periods of sowing-harvesting of 90 days each. These parameter values correspond to Carlavirus transmission by whiteflies in soybean

We can also infer from the model that the value of the threshold strongly depends on the parameter ϕ: if, for example, we do not take into account the possibility that a vector loses its capacity to infect after a puncture, that is $\phi = 0$, numerical simulations are expected to show only increasing incidences. In fact, in Fig. 2b, we have plotted the values of M_{ap} as a function of the vector

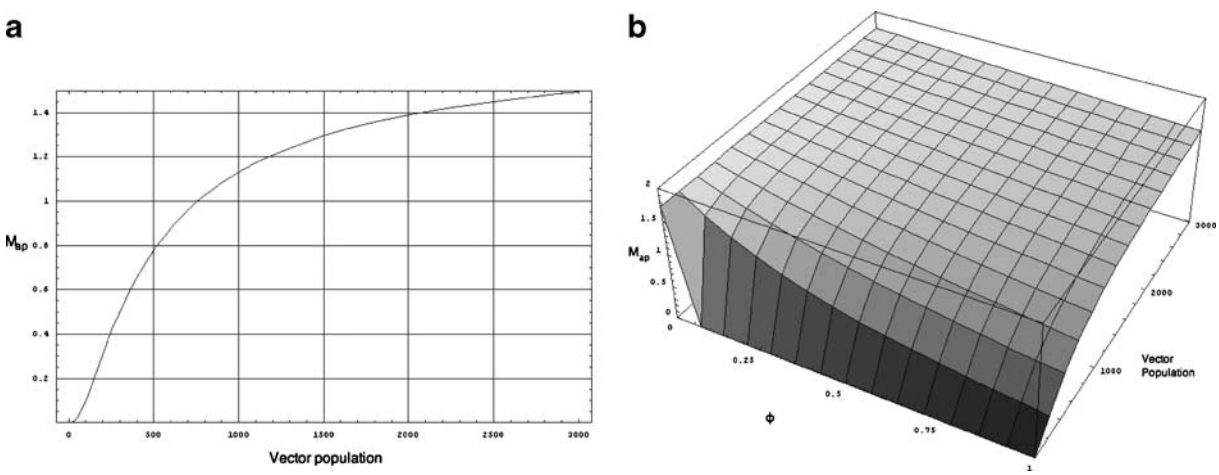

Fig. 2 **a** Plot of M_{ap} as function of the vector population M, $\phi = 1$. **b** Dependence of M_{ap} on the values of ϕ. For $\phi = 0$ the model predicts only increasing incidences. The other parameter values are the same as in Fig. 1

population M and ϕ, and for $\phi = 0$, the value of M_{ap} is bigger than one for all the positive values of the vector population, implying increasing levels of disease incidence in upcoming cycles.

In conclusion, the model suggests that a reduction of vector population, by insecticides, natural predators or any other means, jointly with a short production cycle with harvest and renewal of plant population, may halt the amount of infected plants growing at the end of each cycle, because the virus is not able to spread fast enough to the new generations of plants. If the vector population is shortened to a level below the threshold, the incidence may be reduced over consecutive cycles, eventually driving the disease to extinction.

Discussion

In the proposed model we have made numerous simplifying hypotheses. From these equations, we determined that the disease-free state is always sensitive to small perturbations (unstable), meaning that the introduction of the pathogen, even in small quantities, will start a disease outbreak. Equation 1 tells us that the number of infected plants, which begins at zero in each cycle, will always increase. Our threshold quantity does not determine if an outbreak will happen or not, as does the well known basic reproduction number in epidemiology. Instead, it tells us if the final size in a cycle will decrease or increase relative to the final size of the previous cycle. However, from the simulations and the schematic Fig. 4, it is apparent that once the process of reducing incidence begins, it will continue. The threshold for each cycle depends on $w((k-1)T)$, the final amount of vector population at the end of the previous cycle. For practical purposes, these numbers can be approximated using field measurements and parameter estimation techniques.

The study of the spread of vector-borne viruses in annual or perennial crops with seasonal growth is not new. For instance, in Madden and van den Bosch (2002), conditions for invasion and persistence of plant diseases due to the the introduction of pathogens are studied carefully. Our equations integrate, within this context, the vector's diminished ability to transmit the virus after a puncture, suggesting that this fact may have a critical impact on disease spread.

The model presented would help to assess the impact that certain control measures applied directly to the vector population, like insecticide application, may have on the incidence of the disease on crops. In fact, an optimal insecticide should reduce the vector population below the threshold to guarantee a decreased incidence for the next production cycle. The use of the model can be extended to analyse other type of measures. For instance, whiteflies can show high levels of resistance to several insecticide groups, including pyrethroids (Morales and Anderson 2001; Dennehy et al. 2005), allowing other pest management strategies, like oil application or virus resistant cultivars, to be used instead. It is known that spraying certain non-lethal oils may inhibit the transmission of non-persistent viruses (Perring et al. 1999). It is not completely clear if stylet contact with the oil occurring during probing of treated hosts may affect the virus attachment or the release from stylets, or if the infection process is impeded even though virus is transmitted. It also has not been understood to what extent this method could reduce the incidence. Our equations can be easily modified to integrate these types of conditions and it would be possible to obtain similar mathematical conditions that guarantee increasing or decreasing incidences in consecutive production cycles.

Appendix

Let T be the duration of the production cycle. At the beginning of each period, the number of infected hosts is to 0. Therefore, by integrating Eq. 3 we obtain the number of infected plants at time t in the k^{th} period, $k = 1, 2, ...,$

$$y(t) = \begin{cases} 1 - e^{-\int_{(k-1)T}^{t} w(s)ds} & \text{if } t \in [(k-1)T, kT), \\ 0 & \text{if } t = kT. \end{cases} \tag{6}$$

Let y_k be defined by

$$y_k := \lim_{t \to kT^-} y(t) = 1 - e^{-\int_{(k-1)T}^{kT} w(s)ds}.$$

Therefore, $\lambda(kT) = -y_k$. In Fig. 2 we observe how the monotonicity of the sequence (y_k) changes in response to changes of the value $P = K/M$. Essentially, if the number of hosts is constant, the incidence at the end of each harvest can be reduced if the number of vectors, M, is kept at low levels, corresponding with what is expected to happen in a real scenario. This fact is not captured by the model if the term $\phi P(1-y)w$ is not included.

For practical purposes, it makes sense only to study the set $\Omega = \{(w, y) : 0 \le w \le 1, 0 \le y \le 1\}$, which is invariant. The system without impulses has only two equilibria, $E_u = (0, 0)$ (unstable) and $E_s = (1/(1+Q), 1)$ (stable). Solving for w in Eq. 3 and replacing it in Eq. 4 produces the following second order nonlinear equation for the k^{th} time interval of length T,

$$y'' + \left(\frac{1}{1-y} - (\phi P - 1)y + \phi P + Q\right) y' - y = 0, \tag{7}$$

with $y((k-1)T) = 0$ and $y'((k-1)T) = w((k-1)T), k = 1, 2, \ldots$. A closed solution of Eq. 7 seems difficult to achieve. However, our interest is on the monotone nature of the sequence (y_k), the infected plant proportion at the end of each production cycle. We consider instead a differential equation which approximates Eq. 7, with the sequence of points at the end of each cycle having the same monotonicity as the sequence (y_k). For small values of y, and for periods of time T not too large, our approximation is obtained from Eq. 7 by eliminating the terms of order higher than two,

$$y'' + Dy' - y = 0, \tag{8}$$

where $D = 1 + \phi P + Q$. Contrary to Eq. 7, the solutions of this equation are easy to obtain and have an apparent closeness at the beginning of stages to the numeric solutions of Eq. 7, see Fig. 3. Let y and Y be solutions of Eqs. 7 and 8 respectively, then, from Eq. 3, $Y'((k-1)T) \approx y'((k-$

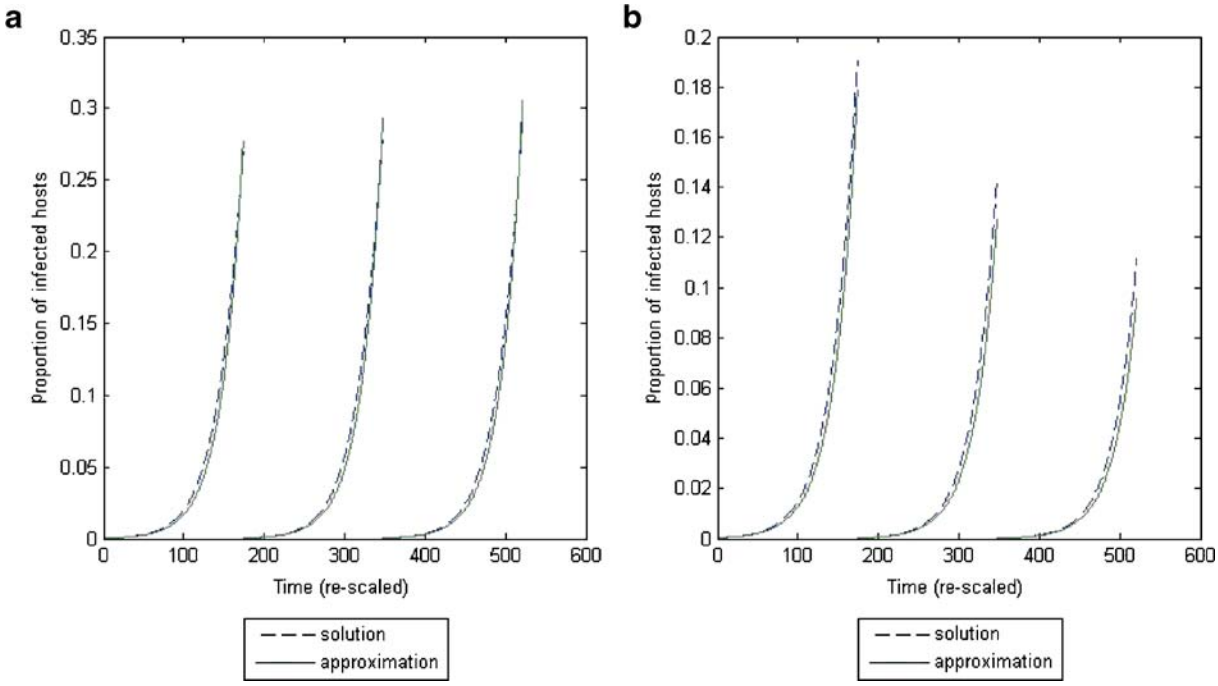

Fig. 3 Solutions to the model equations (*dashes*) and to the approximate differential equation (*continuous line*). The parameter values are the same as in **a** and **b** in Fig. 2. For both the final incidences have the same monotone character; the agreement between the curves is reasonable

$1)T) = w((k-1)T)$, and the solutions of Eq. 8 in the k^{th} time interval are given by:

$$Y(t) = \frac{2}{\rho} w((k-1)T) e^{-\frac{D}{2}(t-(k-1)T)} \times \sinh\left(\frac{\rho}{2}(t-(k-1)T)\right), \quad t \in [(k-1)T, kT), \tag{9}$$

with $\rho^2 = D^2 + 4$. Therefore, defining

$$Y_k := \lim_{t \to kT^-} Y(t) = \frac{2}{\rho} w((k-1)T) e^{-\frac{DT}{2}} \sinh\frac{\rho T}{2}, \tag{10}$$

we see that the three differences, $y_{k+1} - y_k$, $Y_{k+1} - Y_k$ and $w(kT) - w((k-1)T)$ should have the same sign for any k, so if one of the three sequences (y_k), (Y_k) and $(w(kT))$ is monotone so are the other two, being the three of them increasing or decreasing. Figure 4 shows a schematic picture of some trajectories on the phase plane which illustrate how it is possible to obtain increasing or decreasing sequences when the initial proportion of infected vectors is $< 1/(1+Q)$. If the time τ required for a trajectory to cross the line $w = w((k-1)T)$ in the wy-plane is larger (shorter) than T then the starting point of the

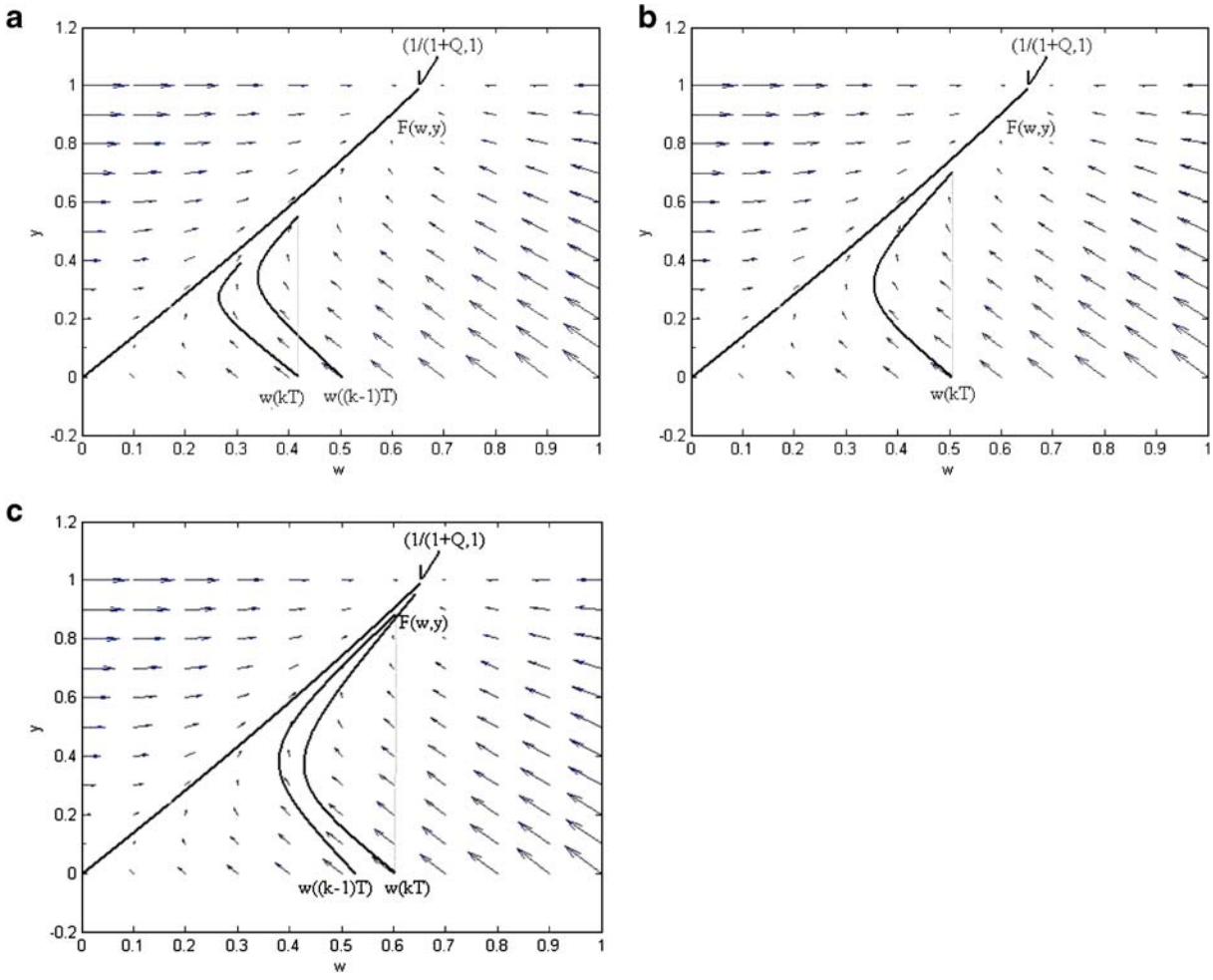

Fig. 4 Schematic representation of solution paths on the wy-plane. **a** The time T is not enough to reach the line $w = w((k-1)T$, causing the next path to start on the left of $w((k-1)T)$ and implying $y_{k+1} < y_k$. **b** The sequence is constant when the starting and endpoint of the path are in the same horizontal position. **c** When the path crosses $w = w((k-1)T$ the starting point of the next path is on the left of starting point of the previous path. As a consequence, $y_{k+1} < y_k$

next trajectory will be to the left (right) of $w((k-1)T)$ and consequently the monotonicity of (y_k) can be determined. This is exactly the situation observed in Figs. 2a and b. If the initial proportion of infected vectors is $>1/(1+Q)$ then, initially, there will be a reduction in the infected vector population eventually reducing this case to the one previously mentioned. Because $Y(t) \approx y(t)$, we can write

$$\frac{Y'(t)}{1-Y(t)} \approx w(t). \tag{11}$$

Therefore, the inequality

$$\lim_{t \to kT^-} \frac{Y'(t)}{1-Y(t)} > w((k-1)T) \tag{12}$$

may be used as a condition to anticipate if the position of the final endpoint corresponding to the k^{th} path will be to the right of the starting point. This condition can be rewritten as

$$\frac{\rho \cosh\left(\frac{\rho T}{2}\right) - D \sinh\left(\frac{\rho T}{2}\right)}{\rho e^{\frac{DT}{2}} - 2w((k-1)T)\sinh\left(\frac{\rho T}{2}\right)} > 1, \tag{13}$$

and can be thought of as a condition determining that the spread of the disease will increase during the next cycle. Similarly,

$$\lim_{t \to kT^-} \frac{Y'(t)}{1-Y(t)} < w((k-1)T), \tag{14}$$

or the corresponding expression analogue to Eq. 13 with the inequality reversed, indicates that the spread of the disease will decrease in the next cycle.

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