The effect of seasonal host birth rates on population dynamics: the importance of resonance

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Abstract

Many of the simple mathematical models currently in use often fail to capture important biological factors. Here we extend current models of insect–pathogen interactions to include seasonality in the birth rate. In particular, we consider the SIR model with self-regulation when applied to specific cases — rabbit haemorrhagic disease and fox rabies. In this paper, we briefly summarize the results of the model with a constant time-independent birth rate, $a$, which we then replace with the time dependent birth rate $a(t)$, to investigate how this affects the dynamics of the host population. We can split parameter space into an area in which the model without seasonality has no oscillations, in which case a simple averaging rule predicts the behaviour. Alternatively, in the area where oscillations to the equilibrium do occur in the non-seasonal model, disease persistence is more complicated and we get more complex dynamical behaviour in this case. We apply resonance techniques to discover the structure of the subharmonic modes of the SIR model with self-regulation. We then look at whether many biological systems are likely to display these “resonant” dynamics and find that we would expect them to be widespread.

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1. Introduction

It is important to understand how diseases interact with their host, how they persist and conversely, how they can be controlled. This is vital for both public health and economic reasons. Mathematical models have already been used, very successfully, to give insight into important disease systems such as HIV/AIDS (e.g. Anderson and May, 1992).

However, many of the simple mathematical models currently in use often fail to capture important biological factors. In ecological systems, the nonlinear dynamics within the population and the underlying exogenous environmental factors interact making it extremely difficult to understand observed population fluctuations. We wish to examine the dynamical consequences of allowing the host birth rate, $a$, to be a function which varies over time. This turns out to be a complex and interesting problem.

In this paper, the SIR (susceptible, infected, recovered) model with self-regulation will be considered and parameterized to describe two specific examples — rabbit haemorrhagic disease and fox rabies. The models are based on the work of Anderson and May (1979, 1981); Norman (1994, 1995); Mena-Lorca and Hethcote (1992); Gao and Hethcote (1992) and Zhou and Hethcote (1994) and are rewritten to include a seasonal function in the birth rate. Although the work of Anderson and May (1981) has been expanded in several ways to include seasonality, the impact of seasonal host birth rate on host–microparasite interactions has not been fully investigated.

Williams and Dye (1997) investigated the case when transmission varies seasonally and they illustrate (to a
very good approximation) that for a general class of infected functions the arithmetic mean transmission rate gives an accurate value of the threshold parameter, $R_0$ — often referred to as the basic reproductive ratio (when $R_0 > 1$, a disease can invade and establish itself within a population). That is, the average transmission rate can be used to calculate the appropriate threshold parameters.

Roberts and Kao (1998) do look at seasonality in the birth rate, but they consider only a pulsed birth rate, where animals give birth in the wild only during a single short period of the year, and apply this to an example of tuberculosis in the possum. One drawback is that the analysis requires complex numerical evaluation. The issues raised suggest that further research incorporating other forms of seasonality would be beneficial, as a pulsed birth rate is unrealistic for most biological systems.

White et al. (1996) considered the effect that seasonal host reproduction has on host–macroparasite interactions and concluded that if seasonal effects are disregarded, regulation of the hosts by the parasite population are overestimated.

Our main objective in this paper is to study the impact of a disease on a seasonally cycling population. We show that although under some circumstances the effect of seasonality can be understood easily, complications arise when there are underlying oscillations towards a stable equilibrium in the non-seasonal model — this is the case for rabbit haemorrhagic disease and fox rabies. When we add seasonal forcing, it combines with the oscillations and resonates, thus exciting several different states for the system. Our second objective is to understand the structure of the different subharmonics that can be excited and to determine the type of complex dynamics we might expect to see in the RHD and fox rabies systems. Also, to discuss how important this resonance behaviour might be for infectious disease systems in general.

2. The SIR model with self-regulation

Anderson and May (1979) and Goa and Hethcote (1992) describe details of the simplest SIR model with self-regulation when the birth rate takes a constant value over the entire year, but they are summarized here. The host population is split into three distinct classes: susceptibles ($X$), infecteds ($Y$) and immunes ($Z$); therefore, our total population density $H = X + Y + Z$. The following set of coupled, non-linear differential equations describes the system:

$$\frac{dX}{dt} = aH - (b + sH)X - \beta XY,$$

$$\frac{dY}{dt} = \beta XY - (r - sH)Y - \gamma Y,$$

$$\frac{dZ}{dt} = \gamma Y - (b + sH)Y.$$

The parameters are listed for clarity in Table 1. We have incorporated density dependence into the death rate here — partly for biological realism but also to avoid direct interaction between the seasonality and non-linearities in the density dependence term of their research.

We can solve for the equilibrium by setting the derivatives in eqs. (1)–(4) equal to zero. This results in three possible equilibria. There is, the origin ($H = 0$, $Y = 0$, $Z = 0$), in which case nothing is present, and ($H = K$, $Y = 0$, $Z = 0$), in which case, the host is at the carrying capacity, $K = r/s$, and there is no disease. Finally, where the disease and host co-exist, there is the equilibrium,

$$Y = Y_0 = rH_0 - sH_0^2/2,$$

$$Z = Z_0 = \gamma(rH_0 - sH_0^2)/a(b + sH_0)$$

$$H = H_0 = (rH_0 - sH_0^2/2) + (\Gamma + sH_0/\beta)$$

$$+ \gamma(rH_0 - sH_0^2)/a(b + sH_0))$$

A cubic can now be formed, $e(H_0)$, which can be solved to find $H_0$:

$$e(H_0) = \beta s^2 H_0^3 + H_0^2 [\alpha \beta s + \beta bs - \beta sr - s^2 \alpha + \lambda \beta s] + H_0 [2 \beta b - \beta br - \Gamma \alpha s - h s \alpha - \lambda \beta r] - \Gamma x b = 0.$$  

2.1. Stability analysis

Having solved for equilibrium, we are concerned with when these equilibria are stable. This can be determined by considering the Jacobian of the system at equilibrium and its eigenvalues (the roots of the characteristic equation). If the eigenvalues have negative real parts then the equilibrium is stable. We make use of the Routh Hurwitz criteria to determine stability. The above is a standard method of analysis described in Anderson and May (1981). If the eigenvalues have negative real parts

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Meaning</th>
<th>Units</th>
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<tbody>
<tr>
<td>$a$</td>
<td>Per capita seasonal birth rate</td>
<td>time$^{-1}$</td>
</tr>
<tr>
<td>$b$</td>
<td>Per capita natural death rate</td>
<td>time$^{-1}$</td>
</tr>
<tr>
<td>$z$</td>
<td>Per capita death rate due to the disease</td>
<td>time$^{-1}$</td>
</tr>
<tr>
<td>$\beta$</td>
<td>Transmission parameter</td>
<td>density$^{-1}$</td>
</tr>
<tr>
<td>$s$</td>
<td>Density dependent parameter</td>
<td>time$^{-1}$</td>
</tr>
<tr>
<td>$\gamma$</td>
<td>Per capita rate of recovery from the disease</td>
<td>time$^{-1}$</td>
</tr>
<tr>
<td>$r$</td>
<td>Per capita population growth rate, $a/b$</td>
<td>time$^{-1}$</td>
</tr>
<tr>
<td>$\Gamma$</td>
<td>$\alpha + b + \gamma$</td>
<td>time$^{-1}$</td>
</tr>
</tbody>
</table>
then the equilibrium is stable. It can therefore be shown that the origin, (0,0,0), is only stable if \( b > a \). The \((K,0,0)\) equilibrium is only stable if \( a > b \) and \( H_T > K \), where the threshold density, \( H_T = \Gamma / (\beta - s) \), and due to the algebraic complexities involved in these analyses, we assume that the disease can be maintained, i.e. we have a co-existence equilibrium or possibly cycles, only if \( a > b \) and \( H_T < K \). This assumption is backed up by numerical simulations.

For this model, we have not been able to prove that the population does not exhibit stable limit cycles but extensive numerical simulations have not shown them. However, there has been seen to be oscillations towards the equilibrium for certain parameter values. This is because under these circumstances, the eigenvalues have negative real parts but are complex, indicating that we have a spiral point in state space.

3. Model development

The simple theory shown above makes the assumption that the host birth rate takes a constant value over the whole year. However, in reality, many animal species have seasonal birth rates, and this may affect the number of susceptible individuals available at a given time of year and will certainly effect the dynamics of the system.

We now wish to represent the birth rate, \( a \), by a function which varies over time, \( a(t) \). Many models of childhood diseases have assumed that seasonal forcing could be modelled by a periodic function (Keeling et al., 2001; Finkenstadt and Grenfell, 2000).

Therefore, in accordance with these papers, the following sine function has been chosen for simplicity, \( a(t) = a_0(1 + a_1 \sin(2\pi t)) \).

It turns out that if there are no underlying oscillations, i.e. the eigenvalues of the Jacobian at the coexistence equilibrium have no imaginary part then we can use a maximum/minimum approach to analyse the condition of disease persistence, since \( a(t) \) is bounded. Since \( K \) in the model becomes dependant on time, then the sine wave is incorporated into the carrying capacity \( K(t) \). In this simple case, if \( 1 < (K(t)/H_T) \) on averaging over the year using an arithmetic mean, then the disease persists and the host population settles to a stable, oscillating equilibrium else, the disease dies out and the host population regulates at the carrying capacity, \( K(t) \) (Ireland and Norman, 2003). However, if the non-seasonal model does have underlying oscillations then the dynamics of the system become much more complicated. We will initially look at how important this distinction is likely to be and then look in detail at the possible dynamics.

In the case where there are no underlying oscillations, we can apply the simple averaging rules outlined above to describe the effect of seasonal birth rates on the system. To describe what happens when there are underlying oscillations, things are much more complicated. The seasonality and the oscillations resonate to cause more complicated dynamics.

4. The resonance approach

In order to understand these complexities more fully, we carry out the following analysis (Greenman et al., 2004). We scale the model equations (1)–(4) by a constant factor \( p^{-1} \) to change the model into one with resonant behaviour and to enable us to obtain a more thorough understanding of the deterministic behaviour of the seasonal SIR model with self-regulation. This scaling factor, \( p^{-1} \), is incorporated into the model parameters:

\[ b' = b_0/p; \ s' = s/p; \ \beta' = \beta/p; \ \gamma' = \gamma/p; \ a'_0 = a_0/p. \]

We also have to rescale time: \( t' = tp \).

It is more suitable to drop the primes, and therefore, the rescaled equations are as follows:

\[ dX/dt = aH - (b + sH)X - \beta XY, \]

\[ dY/dt = \beta XY - (\Gamma + sH)Y, \]

\[ dZ/dt = \gamma Y - (b + sH)Y, \]

\[ dH/dt = ((a - b) - sH)H - aY, \]

where \( a = a_0(1 + a_1 \sin(2\pi t/p)) \).

We then substitute in the parameter values for RHD. These parameter values are held constant as we change the factor \( p \), which allows us to draw a path through the initial parameter space and reveals the different resonances excited by the external force. We now have a model in which there is an external force with period \( p \) now a parameter (Greenman et al., 2004). As \( p \) varies, we are effectively looking at a different member of the family of models defined by Eqs. (5)–(8). The resonance response curves are then constructed for the above model. We will illustrate the methods of analysis and possible dynamics caused by the resonance by considering rabbit haemorrhagic disease and fox rabies.

5. Some biological systems

As discussed above, in the model where the birth rate is constant, it turns out we can use the results of stability analysis to split parameter space into an area where there are no oscillations, i.e. the eigenvalues of the Jacobian at the equilibrium are real, and an area where oscillations do occur, i.e. where the eigenvalues have negative real parts, but are complex. This is illustrated in Fig. 1 for the model with parameters for rabbit haemorrhagic disease (RHD) in \( \alpha-\beta \) space. RHD
has extreme parameter values, high $a$, high $b$, and lies in the area of underlying oscillations. Table 2 lists some important disease-host systems, which could be described by the model and for which parameters could be easily found in the literature, and lists whether or not the non-seasonal model with these parameters contains decaying underlying oscillations, indicating a stable spiral point in state space.

### 6. Rabbit haemorrhagic disease

Rabbit haemorrhagic disease (RHD) is a highly virulent disease of rabbits, which first originated in China in 1984, and has become established throughout much of Western Europe and Australasia. RHD has significantly reduced the number of rabbits, especially in drier regions of southern Spain, inland Australia and South Island New Zealand (Cooke, 2002) It can kill up to 95% of infected rabbits within 48 h (White, 2002).

RHD damages the liver, intestines and lymphatic tissue and causes blood clots (WWW1). There is no treatment for the infection and it is often not detected until the rabbits show terminal clinical signs (WWW2). RHD is transmitted via contact with a rabbit which is infected by the disease or their excretes. Also it is transmitted via insects, rodents, rabbit products such as meat and fur skin, and also contaminated objects such as cages and feeders (WWW3). The disease is spread to other rabbits from surviving rabbits which can become carriers. RHD is of interest for two reasons, firstly it has been used to try and control rabbits in Australasia and secondly it is having a devastating impact on European rabbits, which is having a knock on effect to the environment and predators.

Seasonality is important in this system because rabbit birth rates are highly seasonal and this may effect the number of susceptible rabbits available at a given time of year. The European rabbit *Oryctolagus cuniculus*, is said to be the only species which is susceptible.

Two different rabbit populations will be considered here — one from the United Kingdom and one from New Zealand because parameter values were easily obtained in the literature. Table 3 lists the parameter values. The UK parameters come from White et al., (2001) and the NZ parameter values are based on Barlow and Kean (1998). The NZ parameters come from a semi-arid environment where there is a high density of rabbits.

#### 6.1. Rabbit haemorrhagic disease and resonance

In Europe, RHD spread rapidly. For reasons that are yet unknown, the impact on the rabbit populations is unpredictable. This contrasts with the steady spread of myxomatosis through France and England in the 1950s (White, 2002). The first UK outbreaks of RHD in wild rabbit populations were recorded in 1994 (White et al., 2001).

In the UK, the European rabbit plays a vital role in maintaining biodiversity—it is a prey item for rare species, such as, wild cats, polecats and red kites.

Using the model outlined in Section 2, parameter values were chosen which corresponded to RHD levels in UK rabbit populations, see Table 3. Once again, the constant birth rate was compared to that of the seasonal birth rate.

<table>
<thead>
<tr>
<th>Biological Disease</th>
<th>Underlying Oscillations</th>
<th>$p_0$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rabbit haemorrhagic disease (UK)</td>
<td>Yes</td>
<td>0.23</td>
</tr>
<tr>
<td>Rabbit haemorrhagic disease (NZ)</td>
<td>Yes</td>
<td>0.81</td>
</tr>
<tr>
<td>Fox rabies</td>
<td>Yes</td>
<td>1.41</td>
</tr>
<tr>
<td>Rinderpest in wild ungulates</td>
<td>Yes</td>
<td>2.23</td>
</tr>
<tr>
<td>Bovine TB in badgers</td>
<td>Yes</td>
<td>6.95</td>
</tr>
<tr>
<td>Cowpox in bank voles</td>
<td>No</td>
<td>N/A</td>
</tr>
</tbody>
</table>

### Table 3

<table>
<thead>
<tr>
<th>Disease parameters (per day)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symbol</td>
</tr>
<tr>
<td>$a_0$</td>
</tr>
<tr>
<td>$a_1$</td>
</tr>
<tr>
<td>$b$</td>
</tr>
<tr>
<td>$g$</td>
</tr>
<tr>
<td>$\beta$</td>
</tr>
<tr>
<td>$s$</td>
</tr>
<tr>
<td>$\gamma$</td>
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</tbody>
</table>
6.1.1. Oscillation area

In the case without seasonality, our system is stable, but it achieves stability through decaying oscillations, we will let \( p_0 \) denote the natural period of oscillation of the system. Our objective is to understand the structure of the different subharmonics that can be excited when these oscillations interact with the seasonal forcing. We will therefore make use of the resonance techniques described in Section 4.

6.1.2. Graph illustrating subharmonics initiated as external forcing increases

The types of complex behaviour seen in this model depends on a combination of the strength of seasonality (\( a_1 \)) and the initial conditions. Let us commence by constructing a bifurcation map to illustrate the subharmonics which are excited as the external forcing strength, \( a_1 \), is increased, Fig. 2. This involves plotting the maximum infection level against the strength of the external force, thus illustrating the sensitivity of the model to the seasonal forcing and furnishing us with a simple graph illustrating the behaviour of the system. When the magnitude of the external forcing parameter, \( a_1 \), is less than 0.43, the system reacts with oscillations of period 1, e.g. one cycle per year, the same (annual) period as the external force — see for example Fig. 3(a).

As the external forcing increases slightly, subharmonics of period 2 and period 3 emerge, illustrated in Figs. 3(b) and (c). In other words, as \( a_1 \) increases, the system responds with stable subharmonics which are integer multiples of the external force, although these are not necessarily smooth like the sine wave. For example Fig. 3(b), the dynamics are of period two but there are complex dynamics even within a year.

Chaos eventually results from increasing the external forcing parameter, as illustrated in Figs. 3(d) and (e) when \( a_1 = 0.5 \). In accordance with the findings of Greenman et al, when they studied subharmonic behaviour in a range of human diseases, the amplitudes of all the subharmonic oscillations exceed that of the base mode (one cycle per year) for RHD. Note that as we increase the level of the seasonal forcing in Fig. 2, the subharmonics can disappear just as quickly. For a choice of \( a_1 \) between 0.43 and 0.48, the system has a choice of states. Take for example, \( a_1 = 0.45 \). The system can access stable modes of oscillation with periods 2 or 3, or an unstable chaotic mode. The state achieved by the model depends on the initial conditions used in the simulation.

6.1.3. Resonance response curves

If a frequency or driving force is equal to the natural frequency of the system its driving, then the phenomenon of resonance occurs. This causes the forced oscillations to grow rapidly in size and acquire large amplitudes. Therefore, for our example, the systems natural mode of oscillation reacts with the external force to create the resonance peaks—in other words, the response curve has a resonance when the systems response is a maximum.

Fig. 4(a) is a graphical representation of the resonance response curves for \( a_1 = 0.3 \). The maximum susceptible population is plotted against the external period, \( p \). For a more thorough investigation of the background theory, see for example (Jordan and Smith, 1999).

There are three main resonance peaks, which emerge due to the interaction between the natural mode of oscillation from the system and the external force. As mentioned previously, we can denote the natural period of oscillation by \( p_0 = 2\pi/\omega \) where \( \omega \) is the imaginary part of the eigenvalues of the Jacobian. Resonance then occurs when \( p = p_0 \).

The nonlinearity also results in resonance when \( p \cong p_0/2 \) and \( p \cong 2p_0 \). These time periods of interest should be kept in mind whilst analysing the response curves. The dominant peak is rooted at \( p = 0.23 \) (\( p_0 \)) (denoted with a dashed line on Fig. 4(a)). It is distorted to the right, due to the nonlinearities in the system. Therefore, the maximum of the peak occurs at \( p \cong 0.52 \). Lesser peaks originate from \( p = 0.115p_0/2 \), and \( p = 0.46(2p_0) \). We can see that these vertical peaks are also distorted. In the case of the dominant peak, where the forcing and natural period are equivalent (\( p = p_0 \)), the system reacts to the external forcing with oscillations of the same period, \( p \). Notice that a subharmonic has been produced, with period 2\( p \), for the peak at \( p \cong p_0/2 \). If we look at the response curves in depth, we discover that period doubling occurs at the onset of the first resonance peak, which generates the period two peak. At this peak, the stable and unstable pair of period 2 limit cycles are connected by a saddle-node bifurcation. A pitchfork bifurcation, which unites the unstable period \( p \) and period 2\( p \) cycles to enable a stable period \( p \) oscillation to emerge, occurs at point \( E \). At the dominant peak, \( F \), a period \( p \) saddle-node bifurcation occurs and then again at points \( G, H \) and \( I \). From Fig. 4(a), one can also note an isolated component of period 3\( p \) and this will be discussed in more detail shortly.
Increasing the level of external forcing — this time $a_1 = 0.5$, we can see from Figs. 4(b) that whilst the response curves retain their shape, they grow higher and stretch out more. At this higher level of seasonality, more "spray" components are available, which increase in length in both directions. As was found in Greenman et al., (2003) on each component, a succession of period doubling bifurcations take place, which extend from the middle, thus producing a nested hierarchy of subharmonic sections which finally result in an innermost section for which the system is chaotic.

6.1.4. Relating resonance response curves and Fig. 3

It is not complicated to see the relationship between Figs. 2 and 4. Looking as the response diagrams of Fig. 4, we look at the line $p = 1$, to discover the active modes for the SIR model with self-regulation incorporating the RHD parameter values. Take for example Fig. 4(a) — if we look at the line $p = 1$, we discover there is only one intersection it intersects the base mode (period 1). This is consistent with Fig. 2, when we look at the vertical line $a_1 = 0.3$. If we consider Fig. 4(b) and look at the line $p = 1$, we discover there are two intersections, both of which are in the chaotic mode. This is consistent with Fig. 2 when this time, we look at the vertical line $a_1 = 0.5$.

The value $p_0$ is critical in determining which subharmonics are accessible. In this instance, for RHD, $p_0 = 0.23$, whereas this value may alter significantly if the model parameters change. A vast amount of information is embedded in the resonance response diagrams. If we were to take another vertical line, $p = a$, 

![Graphs](image-url)
then the intersection properties of that line relate to the SIR model with self-regulation with the parameters scaled by a factor \(a\).

If we parameterize the equations for RHD in New Zealand (Table 3) we obtain a different value for \(p_0 (= 0.81447)\). Again we can construct the resonance response curves and an extract is illustrated in Fig. 5. Initially the system reacts with oscillations of period 1, this cascades towards chaos and the extract illustrated here shows period halving. You need only look at the line \(p = 1\) to see the behaviour of the system. It can be seen that the line intersects an active mode of \(2p\). In other words, the system responds with oscillations of period 2, which is an integer multiple of the external force. The pattern is the expected two-cycle seen in field observations (Barlow and Kean, 1998).

7. Fox rabies in Europe

Many major epidemics of rabies have been recorded during the past seven centuries, with the dog acting as the main host and transmitter to man (Anderson et al., 1981). While the risk of contacting the virus is low, it is still a possibility. Several Western European countries have been free from rabies at various times in their history, this is because dog rabies were eradicated by vaccinating domestic animals and eliminating stray dogs.

Adaptation of rabies to the fox allowed the virus to invade many countries after the Second World War (WWW4). At the moment, you can avoid coming into contact with a rabid animal in the British Isles. However, the fox has become the main carrier in Europe (WWW5). The disease is transmitted mostly as a result of a non-infected animal being bitten by an infected animal. All mammals are thought to be susceptible, however, humans, cats and dogs are regarded as mildly susceptible, whereas skunks, foxes, bats and raccoons are highly susceptible. Using the SIR model with self-regulation, adding seasonality to the birth rate and using resonance techniques, we predict the dynamics of rabies in foxes in Europe and discuss this in the context of field observations. The parameters used are taken from Anderson et al. (1981) (Table 3).
In the case of Fox Rabies, there is no recovery or immunity ($\gamma = 0$) and therefore, the underlying model is simpler. As with RHD, the non-seasonal model is stable but with decaying oscillations whose natural period is $p_0 = 1.41$.

7.1. Graph illustrating subharmonics initiated as external forcing increases

We commence by constructing the bifurcation map which illustrates the subharmonics which are excited as the external forcing strength, $a_1$, is increased, Fig. 6. This involves plotting the maximum infection level against the strength of the external force. This is clearly more complicated than for RHD (Fig. 2). When the magnitude of the external forcing parameter, $a_1$, is less than 0.2, the system reacts with oscillations of period 1, e.g. one cycle per year, the same (annual) period as the external force. As the external forcing increases slightly, subharmonics of period 3 emerge and increasing the parameter $a_1$ again, we discover periods $4p$, $6p$ and a chaotic mode can also be accessed at different external forcing values. In other words, as $a_1$ increases, the system responds with stable subharmonics which are integer multiples of the external force. The state achieved by the model depends on the initial conditions used in the simulation.

7.2. Resonance response curves

Fig. 7(a) is a graphical representation of the resonance response curves for seasonal forcing, $a_1 = 0.31$. The maximum susceptible population is plotted against the external period, $p$. There are two main resonance peaks visible in the scale of this graph, which have emerged here due to the interaction between the natural mode of oscillation from the system and the external force. As mentioned previously, we can denote the natural period of oscillation by $p_0$. The dominant peak is rooted at $p = 1.41(p_0)$, again the nonlinearities, mean it is extremely distorted to the right, with the peak occurring above $p \cong 8$. A lesser peak occurs at $p = 0.75(p_0/2)$. We can see that these vertical peaks are distorted — again due to the nonlinearities within the system and also more than in the RHD example. Once again, we only need look at the line $p = 1$ to discover the behaviour of the system. We see it intersects a mode of $1p$, $2p$, $3p$ or $6p$. Fig. 7(b) is an extract from the resonance response curves for $a_1 = 0.36$. Looking again at $p = 1$, we discover it intersects a mode of $1p$, $3p$, $4p$ or $6p$. The results are sensitive to small changes in parameter values—however they are still consistent with the expected 3–5 year cycle seen in observed field dynamics (Anderson et al., 1981).

8. Discussion

We have shown here, that although under some circumstances, the addition of seasonality in the birth
rate of a simple model can be analysed relatively trivially and intuitively, if the underlying model has non-seasonal oscillations then more complex dynamics arise which depend on both the level of seasonal forcing and the initial conditions of the system. The analysis in this case is much more complicated. When the nonlinear system is driven by small degrees of seasonality, we observe that the system reacts by producing oscillations which have the same annual period as that of the external force. As the external forcing increases, stable subharmonics can be generated whereby the system oscillates with a period that is an integer multiple of the external force. The value of $p_0$ is crucial in determining which subharmonics may be accessed.

We would expect even more complicated dynamics (Greenman et al., 2004), in systems with density dependence in the birth rate or seasonality in the transmission term, i.e. models in which seasonality goes directly into a nonlinear term. Greenman et al. have shown that if stochasticity is added to these systems then it is possible for dynamics to flip between states due effectively to the dependence of behaviour on initial conditions.

Our understanding of the effect of seasonality on specific systems clearly depends on how good our parameter estimates for these systems are, since this will tell us about underlying non-seasonal dynamics.

It would also be helpful to know what level of seasonal forcing we would expect to see in real systems. We can get a ballpark estimate of this from the UK RHD data since we know that real rabbit populations can fluctuate between 50 and 170 over the course of a year (White et al., 2001). In order to achieve this, we need, $a_1$ approximately equal to 0.5. This means that we would expect to see something like the complex dynamics illustrated in Fig. 4(a), unfortunately, time series data to verify these predictions are unavailable. However, we know from Kean and Barlow, 1998, that the strength of seasonality in the birth rate of the New Zealand population was 0.8. In the New Zealand rabbit population with this level of forcing, the model predicts a two cycle, this is in accordance with the field data.

In addition, despite the fact the model dynamics are sensitive to small changes in parameter values, using the model to describe Rabies also gives results which are qualitatively consistent with observations. This means that the addition of a disease to a seasonally cycling population could explain the dynamics seen empirically in these systems and we already know that the addition of seasonality to the transmission rate of a model such as this can explain the dynamics seen in childhood disease systems (Greenman et al., 2004).

In order to determine whether this behaviour is dependent on the shape of the seasonality term, we have looked at a number of alternatives. We have repeated the simulations using a step function for the birth rate in all three cases. We found that the dynamics are much more sensitive to seasonal forcing, with lower levels of seasonal forcing giving us more complex behaviour. However, if the step function is adjusted so that the variances of the step function and sine wave are similar in value then this disparity disappears, i.e. the two periodic functions generate essentially the same dynamical behaviour (Greenman et al., 2004). For the systems we have investigated, we do not believe a pulse birth rate is a realistic approximation. However, we have considered something closer to a pulse birth rate in the case of fox rabies, and preliminary analysis suggests that there might be differences in structure which merit further study.

Resonance behaviour is characterized by high peaks but also low troughs where the populations reach sufficiently low levels as to raise questions about the validity of the model at these points. There are two ways to deal with this problem. One is to have an open population modelled by adding to the system, a term for the immigration of infecteds (Bartlett, 1960; Rand and Wilson, 1991). This extension to the model is extremely useful because it avoids having to re-start the calculations after every fade out (Bolker and Grenfell, 1995). The second option is to use a stochastic model with integer population dynamics (Bailey, 1957) if the focus is on the possibility of extinction.

One question which arises from this work is, how important these resonance effects might be in other infectious disease systems. To determine this, we have looked at some other systems to see whether their appropriately parameterized, non-seasonal models have underlying oscillations and if so, what the natural period of those oscillations is. The results are presented in Table 2. It can be seen that we would expect this phenomenon to be important in most of the disease systems that we have been able to parametrize.

Since most biological systems have some level of seasonality in them, we expect the results presented here to have wide ranging consequences for our ability to predict population dynamics. These higher order subharmonics can be excited in a variety of wildlife and human diseases and hence are important in our path to understanding the population dynamics of real systems and the impact of disease on a population. What we have shown here in effect, is that the addition of a disease to a seasonally cycling population can cause complex dynamics and even chaos and that this is likely to occur in many real systems.

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References