Impact of seasonality upon the dynamics of a novel pathogen in a seabird colony

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Abstract. A seasonally perturbed variant of the basic Susceptible-Infected-Recovered (SIR) model in epidemiology is considered in this paper. The effect of seasonality on an IR system of ordinary differential equations describing the dynamics of a novel pathogen, e.g., highly pathogenic avian influenza, in a seabird colony is investigated. The method of Lyapunov functions is used to determine the long-term behaviour of this system. Numerical simulations of the seasonally perturbed IR system indicate that the system exhibits complex dynamics as the amplitude of the seasonal perturbation term is increased. These findings suggest that seasonality may exert a considerable effect on the dynamics of epidemics in a seabird colony.

1. Introduction
Zoonotic emerging infectious diseases (EIDs), such as the highly pathogenic H5N1 avian influenza virus, are a major threat to global health [1]. Migratory birds have been implicated as a potential pathway by which the H5N1 virus could spread between countries [2, 3]. Consequently, the introduction of H5N1 avian influenza to a seabird colony with no existing herd-immunity would be of grave concern to marine conservationists and population ecologists [4]. Seabirds breed in very close proximity to each other in colonies numbering several thousands to millions of individuals [5]. This paper concentrates upon marine birds, which typically exhibit long life expectancy and low reproductive output, such as the northern gannet *Morus bassanus* and the common guillemot *Uria aalge*. These birds live in the North Atlantic; the seasonal variability of the climate may have a strong effect upon the life history of these birds [6].

Seasonality in the incidence of a disease may be caused by a variety of factors, including changes in host behaviour and changes in host immunity to infection [7, 8]. Environmental factors that change with the season, e.g., temperature, weather conditions, rainfall, etc., may also drive changes in disease incidence; in particular, these may affect the survival of the pathogen outside the host [8]. In addition, seasonality may cause changes in the recruitment of susceptibles into the population through annual pulses of host births and deaths [7]. For example, the timing of the breeding cycle of seabirds depends strongly on seasonal factors such as temperature and food availability [9]. It is also worth noting that multiple seasonal drivers may interact in complex ways [7]. Thus, seasonal variations may significantly drive the dynamics of an infection.

The most common approach for modelling seasonal dynamics is to subject the classical SIR model to periodical external forcing. The seasonally forced SIR model has been extensively studied [10, 11, 12]. Seasonally perturbed SIR models may induce a wide range of population dynamics, including annual oscillations, high-order cycles and even chaos [11, 13, 14, 15].
The purpose of this paper is to examine the effects of seasonality on the dynamics of a novel pathogen, e.g., highly pathogenic avian influenza, in a seabird colony. A seasonally perturbed variant of the Infected-Recovered IR model, proposed by O’Regan et al. [16], is developed to model the introduction of avian influenza into a seabird colony. In particular, the effect of seasonal variations upon a seabird colony is investigated by introducing a seasonal perturbation term into the IR model. The method of Lyapunov functions is used to analyse the long-term behaviour of this variation upon the basic IR model. Lyapunov functions are not only useful in determining global stability of a system; in the case of the seasonally perturbed IR model, a simple Lyapunov function is used to determine the region in which periodic solutions of the same frequency as the perturbation will remain for all future time. The results of numerical simulations of the seasonally perturbed IR model are also discussed.

2. Including Seasonality into the IR Model

O’Regan et al. [16] developed the following two-dimensional system of ordinary differential equations describing the dynamics of a novel pathogen, e.g., highly pathogenic avian influenza H5N1, in a seabird colony:

\[
\begin{align*}
\dot{I} &= (\beta(N - I - R) - (\gamma + \omega + \omega_I))I, \\
\dot{R} &= \gamma I - \omega R,
\end{align*}
\]

where a dot denotes the time derivative. System (1) will be referred to as the IR model. This two-dimensional system is equivalent to an unforced SIR model derived by O’Regan et al. [16].

The population \( N(t) \) is divided into three distinct classes: the susceptible population \( S(t) \), the infectious population \( I(t) \) and the recovered population \( R(t) \). The population \( N(t) \) is assumed to be constant, \( N(t) = N \). Since the population is constant, it suffices to consider system (1).

The parameter \( \beta > 0 \) is called the transmission parameter; the transmission term \( \beta(N - I - R)I \) determines the rate at which new infectives are produced. The parameter \( \beta \) includes the effect of basic reproductive number \( R_0 \), one of the most important concepts in epidemiological modelling. The basic reproductive number is defined to be the number of individuals infected by a single infected individual during its infectious period, in a population which is entirely susceptible. The formula described by Anderson and May [17] for calculating \( R_0 \) is used to calculate \( \beta \) here. The parameter \( \gamma > 0 \) is the rate of recovery. The positive parameters \( \omega \) and \( \omega_I \) are the death rates due to natural causes and the lethality of the pathogen respectively. The IR model is the system produced from a rigorous reduction of a SEIR model, originally developed by Clancy et al. [18], to investigate the impact of avian influenza in a seabird population.

The IR model (1) may be subject to seasonal variations. Seasonality may impact upon a variety of host-pathogen interactions, including altering components of the basic reproductive number \( R_0 \) by influencing parameters such as the transmission parameter or the length of recovery period [7]. The simplest method of including seasonality into epidemiological models is by perturbing the transmission parameter \( \beta \) [7, 12, 11, 14, 19]. However, other parameters in the system may also be seasonally forced, e.g., the recovery rate, as suggested by Altizer et al. [7].

Here, the parameter \( p = \beta N - (\gamma + \omega + \omega_I) \) in system (1) is subjected to external forcing. This combines the effect of the transmission parameter \( \beta \), the population \( N \), the recovery rate \( \gamma \) and the death rates \( \omega, \omega_I \). An increase in the number of susceptible birds congregating in the colony at certain times of the year, e.g., at the beginning of the breeding season, may cause a sudden jump in the transmission rate. This phenomenon has been observed in the case of measles, whereby significant increases in outbreaks of the disease have been recorded at the start of the school year [12, 20]. In wildlife species, social aggregation of house finches during the winter has coincided with epidemics of mycoplasmal conjunctivitis in North America [21]. A sharp increase
in transmission may, in turn, cause a sudden pulse of seabird deaths. Seasonal variation in host susceptibility may also be an additional factor. Immune systems of birds may be weakened at certain times during the year, e.g., during the winter or before the breeding season [7]. Seasonal changes in immunity may lower host recovery rates [7].

We consider the IR model

\[
\dot{I} = (p - \beta I - \beta R)I, \\
\dot{R} = \gamma I - \omega R,
\]

where \( p = \beta N - (\gamma + \omega + \omega_I) \). Subject the IR system to a seasonal perturbation \( \epsilon(t) \), assuming that

\[
p(t) = p(1 + \epsilon(t)).
\]

A sinusoidal perturbation represents a gradual, perhaps idealised, variation due to seasonality, e.g.,

\[
\epsilon(t) = p_1 \sin 2\pi t,
\]

where \( p_1 \) is the amplitude of the forcing. Alternatively, \( p \) may be forced by a step function, representing a sudden change in \( p \) from one season to another, i.e., \( \epsilon(t) = \pm p_1 \):

\[
p(t) = \begin{cases} 
p(1 - p_1), \\
p(1 + p_1). 
\end{cases}
\]

In the rigorous mathematical analysis that follows in Section 3, \( \epsilon(t) \) may be any type of function. Hence, the seasonally perturbed form of system (1) is given by the following system of ordinary differential equations:

\[
\dot{I} = (p(t) - \beta I - \beta R)I, \\
\dot{R} = \gamma I - \omega R.
\]

System (3) will be referred to as the **Seasonally Perturbed IR Model**.

### 3. Analysis of the Seasonally Perturbed IR Model

We will use Lyapunov’s direct method to investigate the behaviour of system (3). This method has been used extensively in mathematical epidemiology [22, 23, 24, 25].

The coordinates of the positive equilibrium of the unperturbed IR model, system (1), are given by

\[
I_* = \frac{\omega p}{\beta (\gamma + \omega)}, \quad R_* = \frac{\gamma p}{\beta (\gamma + \omega)}.
\]

The equilibrium cloud of the perturbed IR model is \(((1 + \epsilon(t))I_*, (1 + \epsilon(t))R_*)\), where \( I_*, R_* \) are the coordinates of the positive equilibrium state of system (1). O’Regan et al. [16] showed that the function

\[
V(I, R) = I - I_* \log I + \alpha(R - R_*)^2
\]

is a Lyapunov function for system (1). However, this expression also provides an estimate for the region of stability of system (3). Function (4) provides a rigorous estimate of a convex neighbourhood of \(((1 + \epsilon(t))I_*, (1 + \epsilon(t))R_*)\); periodic solutions of the same frequency as \( \epsilon(t) \) will remain in this region for all future time. However, function (4) does not tell us anything about the nature of these solutions; these solutions may have multiple period or may, perhaps, be more complicated in nature. Numerical simulations are required to determine the nature of these solutions.
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Now in system (5) we have,

\[ \dot{I} = I \{ \beta[(1 + \epsilon(t))I_\text{s} - I] + \beta[(1 + \epsilon(t))R_\text{s} - R] \}, \]
\[ \dot{R} = \gamma(I - I_\text{s}) - \omega(R - R_\text{s}). \]

Then, using expression (4), we find \( \dot{V}(I, R) \) with respect to system (5). Note that function (4) does not depend on \( t \) and hence, \( \frac{dV}{dt} = \frac{\partial V}{\partial I} \frac{dI}{dt} + \frac{\partial V}{\partial R} \frac{dR}{dt} \). The required partial derivatives are

\[ \frac{\partial V}{\partial I} = 1 - \frac{I_\text{s}}{I}, \quad \frac{\partial V}{\partial R} = 2\alpha(R - R_\text{s}). \]

Now in system (5) we have,

\[ \frac{dI}{dt} = I \{ \beta[(1 + \epsilon(t))I_\text{s} - I] + \beta[(1 + \epsilon(t))R_\text{s} - R] \}, \quad \frac{dR}{dt} = \gamma(I - I_\text{s}) - \omega(R - R_\text{s}). \]

For \( I, R > 0 \) we have,

\[ \frac{d}{dt} V(I, R) = \frac{\partial V}{\partial I} \frac{dI}{dt} + \frac{\partial V}{\partial R} \frac{dR}{dt} \]
\[ = \left( 1 - \frac{I_\text{s}}{I} \right) I \{ \beta[(1 + \epsilon(t))I_\text{s} - I] + \beta[(1 + \epsilon(t))R_\text{s} - R] \}
\[ \quad + (2\alpha(R - R_\text{s}))(\gamma(I - I_\text{s}) - \omega(R - R_\text{s})) \]
\[ = \beta(I - I_\text{s}) \left( \epsilon(t)(I_\text{s} + R_\text{s}) - (I - I_\text{s}) - (R - R_\text{s}) \right)
\[ \quad - 2\alpha\omega(R - R_\text{s})^2 + 2\alpha\gamma(I - I_\text{s})(R - R_\text{s}) \]
\[ = \beta(I - I_\text{s}) \epsilon(t)(I_\text{s} + R_\text{s}) - \beta(I - I_\text{s})^2
\[ \quad - 2\alpha\omega(R - R_\text{s})^2 + (2\alpha\gamma - \beta)(I - I_\text{s})(R - R_\text{s}). \]

Choosing \( \alpha = \beta/2\gamma \) and denoting \( b(t) = \epsilon(t)(I_\text{s} + R_\text{s})/2 \) in expression (6), we obtain

\[ \dot{V}(I, R) = -2\alpha\omega(R - R_\text{s})^2 - \beta \left[ (I - I_\text{s} - b(t))^2 - b(t)^2 \right]. \]

We are interested in the region in which expression (7) is negative. Expression (7) describes an ellipse if \( \dot{V}(I, R) = 0 \). The size of this ellipse is controlled by the parameter \( \epsilon(t) \), through \( b(t) \). Together with the Lyapunov function (4), this ellipse provides a bound for the region in which trajectories will enter and remain for all time. The results of numerical experiments, discussed in Section 4, suggest that the cloud determined by expressions (4) and (7) may be periodic or possibly more complicated in nature.

4. Numerical Simulations of the Seasonally Perturbed IR Model

Numerical simulations are required to gain insight into the dynamics of the seasonally perturbed IR model. We solved system (3) numerically using the Mathematica software package. In the computational experiments, we set the forcing as follows:

\[ p(t) = p(1 - p_1 \sin 2\pi t). \]
Thus, $p(t)$ is periodic with a period of 1 year. As mentioned in Section 2, a standard method of introducing seasonality to the SIR system is by perturbing the transmission parameter $\beta$. Numerous references [7, 10, 11] in the literature suggest that varying the amplitude of the perturbed transmission term may have a strong effect on the dynamics of the system. Here, however, the effect of varying the amplitude $p_1$ of the forcing function $p(t)$ is examined, whilst keeping all other parameters fixed. Table 1 gives the parameter values that were used for the experiments. These values are realistic for simulating the dynamics of avian influenza in a seabird colony [16].

| Table 1. Fixed Parameter Values |
|---------------------------------|
| $N$ | $R_0$ | $\beta$ | $\gamma$ | $\omega$ | $\omega_1$ | $p$ |
| 10000 | 10 | 0.0924 | 91.25 | 0.1 | 0.75 | 831.928 |

O’Regan et al. [16] proved that all solutions of system (1) converge to a globally asymptotically stable positive equilibrium in the long-term. In contrast to system (1), however, Figure 1 indicates that the seasonally perturbed IR system exhibits complicated dynamics as the amplitude $p_1$ is increased; a period-doubling route to chaos is observed. This observation agrees with the results of numerical simulations for other seasonally-forced SIR models [10, 11]. Figures 1–5 show the long-term dynamics of the seasonally perturbed IR model for various values of $p_1$; the value of $p_1$ is indicated on the top of each graph in each figure.

Figure 1 shows that for amplitude in the range $0 < p_1 \leq 0.015$, the infected population oscillates with a period of 1 year. Thus, the period is the same as $p(t)$. Small amplitude oscillations, such as those observed for $p_1 = 0.005$, may not be observed in reality; cycles of such small amplitude may be swamped by noise effects [14]. The amplitude is much larger when $p_1$ is increased to 0.01. In Figure 2, the amplitude of the oscillations changes abruptly when $p_1 = 0.00788$; sharp peaks in the infected population are observed. The duration of outbreaks become shorter but larger numbers of the population become infected. However, the infected population still oscillates with a period of 1 year. Note that the infected population also dies out for short periods of time each year.

The behaviour of the infected population remains 1-periodic for values of $p_1$ in the domain $0.00778 \leq p_1 < 0.019$. For $p_1 = 0.019$, the infected population oscillates with a period of 2 years, i.e., period-doubling is observed (see Fig. 1 for $p_1 = 0.02$). As $p_1$ is increased, the results of the numerical experiments show that a cascade of period-doublings occur, leading eventually to chaos. Figure 3 suggests that the period of the oscillations rapidly doubles in the range $0.0222 \leq p_1 \leq 0.0235$. The infected population oscillates with a period of 2 years when $p_1 = 0.0222$. Oscillations of period 4 are observed for $p_1 = 0.0225$. Oscillations of period 8 are observed at $p_1 = 0.0232$. For $p_1 = 0.0235$ however, it appears that the occurrences of outbreaks have become chaotic; the duration and amplitude of the outbreaks have become unpredictable.

As $p_1$ is further increased (Figs. 4 and 5), unpredictable behaviour of the infected populations is mostly observed with intermittent ranges of $p_1$ producing periodic outbreaks. Period 3 windows are observed in Figure 4 for $p_1 = 0.0275, 0.035$. Epidemics of greater magnitude are observed in Figure 5 compared to those observed for smaller $p_1$ (Figs. 1, 2, 3). The outbreaks of the disease also become less frequent; this is because the magnitude of $p_1$ is very large and thus, the infected population drops to very low values in between outbreaks.

Figure 6(a) clearly shows the unstable dynamics of avian influenza as the bifurcation parameter $p_1$ is increased from 0.0185 to 0.025. For each value of $p_1$, 900 years of transients were discarded before the maximum value of the infected population was recorded each year for
Figure 1. Progression from stable to more complicated behaviour as the amplitude $p_1$ of $p(t)$ increases. Note the appearance of period-doubling for $p_1 = 0.02$. The value of $p_1$ is indicated above each graph.

Figure 2. The amplitude of the oscillations of the infected population abruptly changes when $0.00787 < p_1 \leq 0.00788$. 


Figure 3. Period-doubling route to chaos: Oscillations of period 2 for $p_1 = 0.0222$, of period 4 for $p_1 = 0.0225$, of period 8 for $p_1 = 0.0232$; unpredictable outbreaks for $p_1 = 0.0235$.

Figure 4. Periodic windows are observed for $p_1 = 0.0275, 0.035$.

100 years. We observe a cascade of period-doubling bifurcations with increased seasonal forcing, eventually leading to chaos.

An exciting result is the discovery of a set that resembles the famous strange attractor of the Henon mapping (Figure 6(b)) for $p_1 = 0.025$. This set was produced by recording the values of the infected and recovered populations at the end of each year for 1500 years after discarding the
Figure 5. As $p_1$ increases, outbreaks of the disease become less frequent. The amplitude of the infected population is also of much greater magnitude compared to Figs. 1–4.

Figure 6. Numerical evidence of chaos in the seasonally perturbed IR model. The bifurcation diagram shows a cascade of period-doubling bifurcations and eventually, chaos.

first 500 transient values. The graph in Fig. 6(b) indicates the presence of potentially chaotic behaviour in the seasonally perturbed IR model for this value of $p_1$. This attractor will provide the foundation from which a rigorous proof of chaos in the model, for these particular parameter values, can be established, using the method of topological-degree theory and split-hyperbolicity [26]. It is noteworthy that this method has never previously been applied to an epidemiological model.

5. Discussion and Conclusions
In this paper, a model that may describe the dynamics of a novel pathogen in a seabird colony, subject to seasonality, was considered. Lyapunov’s method was used to find a convex region of stability for the seasonally perturbed IR model. Numerical simulations of this model show that seasonality has a significant effect on the dynamics of the disease. Small amounts of seasonal forcing can generate complex dynamics (Fig. 6(a)). For a large enough amplitude of perturbation, epidemics of unpredictable magnitude and duration may occur (Fig. 5). In particular, the period-doubling route to chaos observed in the numerical experiments is very interesting. However, it should be noted that for different parameter values, a cascade of period-
doublings may not be observed.

Seasonality is a complex force in nature. Much theoretical and computational work is required to obtain a basic understanding of how seasonality affects multiple processes in a seabird colony, including $R_0$, birth, transmission and recovery. It is important for biologists to understand how seasonality interacts with these processes. For example, seasonal variations may or may not be synchronous with each other. “Out of phase” seasonality can significantly affect the dynamics of epidemics in a bird population [21]. The application of seasonality to different model parameters may influence the resulting dynamics in various ways. It is important to emphasise this aspect has not been fully explored in this seasonally perturbed model. In particular, it may be interesting to perturb the transmission and recovery parameters and examine how these affect the system, especially when these parameters are out of phase.

Seasonal changes in host behaviour may also affect the dynamics of the disease significantly. This may be particularly true of a seabird colony, where birds congregate in crowded conditions during the breeding season. However, once the breeding season has finished, these birds disperse away from the colony. These assumptions could be more carefully incorporated into the model by using a step function to model seasonal changes in the transmission parameter. Step functions have been used in models for measles, where the transmission parameter drops during the school holidays and rises sharply at the beginning of the school term [8, 14].

Seasonality in host susceptibility could also be examined more closely. Environmental stresses such as physiologically challenging weather conditions may have multiple effects on hosts, e.g., increasing susceptibility to disease whilst decreasing the rate of reproduction [27] or affecting breeding success [9]. A related factor is resource availability, in particular, seasonal changes in the availability of food may also cause changes in disease dynamics. There is likely to be increased competition for food during the breeding season [9, 28, 29]. Another more recent development, for example, is the increase of snake pipefish *Entelurus aequoreus* in British and Irish waters since 2003. This species has poor nutritional value for seabirds and its distribution in the sea varies seasonally [30]. All of these processes may impact both on transmission and recovery.

Future work will include a rigorous proof of the existence of chaos in the seasonally perturbed IR model for certain values of $p_1$. This proof will be computer-aided and based upon methods from topological-degree theory. To find such complex behaviour in a simple, yet realistic, model is a very exciting development. Thus, problems in epidemiology may lead to new and interesting mathematical work. In turn, mathematics can help biologists understand the mechanisms leading to outbreaks of disease.

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