Qualitative and numerical investigations of the impact of a novel pathogen on a seabird colony

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Abstract. Understanding the dynamics of novel pathogens in dense populations is crucial to public and veterinary health as well as wildlife ecology. Seabirds live in crowded colonies numbering several thousands of individuals. The long-term dynamics of avian influenza H5N1 virus in a seabird colony with no existing herd immunity are investigated using sophisticated mathematical techniques. The key characteristics of seabird population biology and the H5N1 virus are incorporated into a Susceptible-Exposed-Infected-Recovered (SEIR) model. Using the theory of integral manifolds, the SEIR model is reduced to a simpler system of two differential equations depending on the infected and recovered populations only, termed the IR model. The results of numerical experiments indicate that the IR model and the SEIR model are in close agreement. Using Lyapunov’s direct method, the equilibria of the SEIR and the IR models are proven to be globally asymptotically stable in the positive quadrant.

1. Introduction
The modelling of the spread of infectious disease is a scientific field of major importance because epidemics continue to pose a substantial threat to humankind and to the world’s flora and fauna. Mathematical modelling may provide insights into the long-term dynamics of a particular micropathogen. Mathematical modelling is thus becoming an indispensable tool in understanding the epidemiology of infectious diseases. Consequently, mathematical modelling has been utilised by governments to assist decision making, e.g., during the outbreak of Foot and Mouth Disease in Britain in 2001 [1]. The strategies that were implemented to control the spread of the disease were predicted by models to be the most effective in the circumstances [2, 3]. Zoonotic emerging infectious diseases (EIDs), such as H5N1 avian influenza virus, are a significant growing threat to global health, with the majority of EID events caused by pathogens acquired by vertebrate animals [4]. Avian influenza is an infectious disease of birds caused by type A strains of the influenza virus. The most serious pandemic of avian influenza was the Spanish influenza virus (H1N1) in 1918–1919, which was responsible for the deaths of at least 40 million people [5]. Other less serious pandemics occurred in 1957 (Asian influenza, H2N2), 1968 (Hong Kong H3N2) and 1977 (Russian influenza H1N1) [5]. In 1997, the first known outbreak of the highly pathogenic H5N1 avian influenza virus in humans occurred in
Hong Kong, infecting 18 people and causing six fatalities [6]. The H5N1 virus has been endemic in poultry in southeast Asia since 2003 [7]. Recent outbreaks of H5N1 in humans in numerous countries throughout Asia, Africa and Europe [6] have raised concerns that a new influenza pandemic can occur in the near future. In 2005, over 1,500 migratory birds died as a result of H5N1 bird flu at the Qinghai Lake nature reserve in western China [7]. This was the first reported instance of a highly pathogenic strain of avian influenza causing mass die-offs in wild birds. These alarming events motivate the search for an intervention policy for the protection of humans and livestock [8].

In this paper, we concentrate on modelling the introduction and the spread of the H5N1 pathogen in a seabird colony. Marine birds, such as the northern gannet Morus bassanus and the common guillemot Uria aalge, have long life expectancy and low reproductive output. Seabirds congregate in large inaccessible colonies, numbering several thousands to millions of individuals [9] and tend to breed in very close proximity to each other on offshore islands. It is therefore, interesting to note that, despite apparently favorable conditions for the transmission of micropathogens, there are very few reported incidences of epidemics in seabirds.

Clancy et al. [10] incorporated the key characteristics of seabird population biology and the H5N1 virus into a Susceptible-Exposed-Infectious-Recovered (SEIR) model. The impact of the introduction of the H5N1 pathogen into a colony with no existing herd immunity was investigated using numerical simulations of the SEIR system. In this paper, we will build upon the work of Clancy et al. using Lyapunov’s direct method. In particular, we utilise the powerful method of Lyapunov functions to investigate the long-term dynamics of the H5N1 virus in a colony with no existing herd immunity. However, it should be noted that the model and methods described in this paper may be readily applied to a comparable novel pathogen introduced into a crowded population.

2. SEIR Model

2.1. SEIR Approach

One of the most widely used methods to predict the spread of infectious disease is the compartmental model devised by Kermack and McKendrick in 1927 [11]. This model involves dividing a population into the following disjoint classes: those who are susceptible to the infection, those who are infected with the disease and those who have recovered from the disease. The resulting system is termed a Susceptible-Infected-Recovered (SIR) model. Sets of ordinary differential equations based on this approach have been successful in modelling the spread of Foot and Mouth Disease [2], Severe Acute Respiratory Syndrome (SARS) [12] and the West Nile virus [13].

Here, in order to model the effect of the H5N1 influenza virus on a seabird colony, we divide the total population \(N\) into four disjoint subclasses. We let \(S(t)\) be the number of the susceptible population at time \(t\); denote by \(E(t)\) the number of birds which are latently infected, that is, those that have been infected with the virus but are not yet infectious (i.e., cannot yet transmit the virus); let \(I(t)\) be the number of birds which are infected and infectious and we denote by \(R(t)\) the number of birds that have recovered from the virus and are now immune. The size of a colony is constrained by the available nesting space rather then any other conditions, and this available space is constant. Therefore we assume that the total population \(N\) remains constant. That is, at any time \(t\) the equality

\[
S(t) + E(t) + I(t) + R(t) = N
\]

holds. Here the time \(t\) is measured in years.

2.2. Modelling Assumptions

The following assumptions were incorporated into the SEIR model:
Figure 1. The transfer diagram for the SEIR model describing the introduction of H5N1 into an adult seabird population.

- The population \( N \) is considered to be constant in time and homogeneous in space. This assumption makes sense because seabird colonies are very densely populated. Crespin et al. [14] record over 18,000 breeding pairs of the guillemot on the Isle of May, Scotland.

- The natural death rate \( \omega \) is assumed to be constant. Infected birds in the \( E \) class also die at a rate \( \omega_E \) due to the lethality (or virulence) of the disease. The corresponding lethality rate for infected members of the \( I \) class is \( \omega_I \).

- The members of the latently infected \( E \) class become infectious at a constant rate \( \lambda \). The time from the instant of infection to the beginning of the state of infectiousness is called the latent period. Thus the latent period of the virus is \( 1/\lambda \) years.

- The birds recover at a constant rate \( \gamma \).

- The transfer of individuals from the \( S \) class to the \( I \) class involves disease transmission. The transmission term informs us about the rate at which new infected individuals are produced. For a directly transmitted pathogen such as avian influenza H5N1, there has to be contact between susceptible and infected individuals. To derive the transmission term, we assume frequency-dependent (or mass-action) transmission. This describes the situation where the number of contacts between susceptibles and infectives is independent of population size. Due to the mass-action assumption, the transmission term in our model is \( \beta SI \), where \( \beta > 0 \). The transmission parameter \( \beta \) combines many epidemiological, environmental and social factors and cannot be measured directly. Keeling and Rohani [15] discuss the difference between frequency-dependent and density-dependent transmission in detail and also derive the transmission term from first principles.

- The recruitment rate of the susceptibles is assumed to be equal to the number of the vacant nesting spots; that is the recruitment of the new susceptibles balances the number of the deaths. For example, when a guillemot chick is born, it remains in the colony for about 22 days before leaving the colony. These chicks then return to the colony as adults after 4–5 years to breed [14]. We assume that all these birds return as susceptibles. However, since the colony is densely inhabited and space is scarce, these birds must wait for an appropriate nesting site. We make the assumption that when a bird in the colony dies, a bird in the “queue” will immediately take the space vacated by the dead bird. Hence \( S(t) \) is maintained.
by a return rate which matches the total number of adults dying, both naturally and as a result of the disease.

- We focus only on the adult seabird population. The colony is mainly comprised of breeding adults because chicks leave the colony and non-breeders (that is, the birds aged 2-4 years) tend to occupy exposed rocks at the edge of the colony [14]. Clancy et al. formulated two SEIR models describing the dynamics of the adult and chick populations respectively. Numerical experiments have indicated that the chicks have negligible impact on the overall dynamics of the disease in the colony. In addition, concentrating on the adult population only enables us to analyse the long-term dynamics of the disease using the method of Lyapunov functions.

The system can be thus described by the following set of ordinary differential equations, which are readily derived from an examination of Figure 1:

\[
\begin{align*}
\dot{S} &= \omega(S + E + I + R) + \omega_E E + \omega_I I - \beta SI - \omega S \\
\dot{E} &= \beta SI - (\lambda + \omega + \omega_E)E \\
\dot{I} &= \lambda E - (\gamma + \omega + \omega_I)I \\
\dot{R} &= \gamma I - \omega R,
\end{align*}
\]

where a dot denotes the time derivative.

2.3. Initial Conditions

It is important to investigate what will happen when a pathogen is introduced into a population in which there is no existing herd immunity, i.e., none of the population is immune. We require one initial condition for each equation in order to solve system (2) numerically. At time \( t = 0 \), \( N(0) = N \). We assume that at \( t = 0 \), there is a single infectious bird and thus \( I(0) = 1 \). Then \( S(0) = N - 1 \) since all other birds are susceptible to the virus, and \( E(0) = R(0) = 0 \). All numerical simulations were scaled by a factor of \( 1/N \).

2.4. The \( R_0 \) Parameter

One of the most important concepts in epidemiological modelling is the basic reproductive number, \( R_0 \), of a pathogen. The basic reproductive number of a pathogen is defined to be the average number of secondary cases arising from an average primary case in an entirely susceptible population. It describes the maximum reproduction potential of an infectious disease. Clearly, when \( R_0 < 1 \), each infected individual produces on average less than one infected individual and so the infection will eventually be no longer sustained within the population. If \( R_0 > 1 \) however, the virus will establish itself and remain in the population.

There is no definitive method for calculating the basic reproductive number. Heffernan, Smith and Wahl [16] give a comprehensive overview of the various methods used to calculate \( R_0 \). We assume that the host population is homogeneously mixed, i.e., epidemiological properties (e.g., genetic make-up) are intrinsically similar in the host population. The formula used to calculate \( R_0 \) here is that was suggested by Anderson and May [17],

\[ R_0 = \frac{N}{S_*}, \]

where \( S_* \) is the population of the susceptibles at the unique positive equilibrium state \( Q_* = (S_*, E_*, I_*, R_*) \), where the rates of change of the latently infected and infectious populations are zero.
2.5. Parameter Values

Values for each of the equation parameters were required to conduct numerical simulations of the model. The results of these experiments are discussed in Section 3. The parameter values were taken to accurately describe a large seabird colony and the H5N1 virus. Colonies of seabirds tend to be quite large; for example, there are more than 13,000 pairs of the Northern gannet on Stac Lee, a small 180m high sea rock at St Kilda, Western Isles off the coast of Scotland [18]. With this information, we conservatively chose the bird population \( N \) to be 10,000.

Seabirds are known to have long lifespan and high survival rates. Sandvik et al. [19] show that the annual survival rates for the common guillemot, razorbill \( Alca torda \) and Atlantic puffin \( Fratercula arctica \) in an island colony in Norway is over 90% for each species. Wanless et al. [20] give a similar rate for the gannet. Thus, we take the death rate \( \omega \) to be 0.1 in this analysis.

Gani et al. [21] take the latent period to be 2 days and the infectious period to be 4 days in their model of an influenza pandemic. Tiensin et al. [22] also cite a latent period of 1-2 days and an infectious period of 2-6 days. Here, we assume that the latent period to be 2 days, i.e., \( \lambda = \frac{365}{2} \) and the infectious period to be 4 days giving \( \gamma = \frac{365}{4} \).

It is difficult to quantify the rate of lethality induced by the H5N1 strain. Clancy et al. [10] varied the lethality parameters \( \omega_E \) and \( \omega_I \) in their model of the dynamics of the H5N1 virus in an isolated seabird colony to study their influence. They found that these lethality parameters governed the time between the outbreaks of the disease — the higher the lethality, the closer the occurrences. Clancy et al. [10] initially assumed that \( \omega_E = 0.5 \) and \( \omega_I = 0.75 \); these are reasonable estimates of the lethality rates. We will assume the same values here.

Finally, we require the transmission parameter \( \beta \). From equation (3) we have

\[
\beta = \frac{(\lambda + \omega + \omega_E)(\gamma + \omega + \omega_I)R_0}{\lambda N}.
\] (4)

Therefore, we can find a value for \( \beta \) by inputting a value for \( R_0 \). A precise value of \( R_0 \) for the H5N1 strain of avian influenza is not known because. Tiensin et al. [22] estimated \( R_0 \) to be between 2.2 and 2.7 during the 2004 H5N1 avian flu epidemic among chicken flocks in Thailand. Clancy et al. [10] varied \( R_0 \) from 2 up to 10 to examine the resulting dynamics.

3. Theoretical Analysis of the SEIR Model

3.1. Lyapunov Functions

The method of auxiliary functions (Lyapunov functions) was introduced by the Russian mathematician A. M. Lyapunov in 1899 to analyse the behaviour of nonlinear systems of differential equations. Using a Lyapunov function to analyse a nonlinear system of ODEs allows one to determine the long-term dynamics of such a system without solving the system explicitly. However, there are no generally applicable methods for finding Lyapunov functions. Trial and error and mathematical or physical insight are often used to construct an appropriate Lyapunov function. Hirsch, Smale and Devaney [23] comprehensively introduce the technique of Lyapunov functions. Lyapunov’s method has been widely used in population dynamics, particularly in conjunction with the Lotka-Volterra equations for modelling predator-prey interactions [24] and extensively in epidemiological models [25, 26, 27, 28]. In this paper, we use the following definition of a Lyapunov function.

A smooth function \( V = V(x, y) \) is a Lyapunov function in the positive quadrant for the system

\[
\dot{x} = f(x, y), \quad \dot{y} = g(x, y),
\] (5)

which has a unique positive equilibrium \((x_*, y_*)\), if the following conditions hold:

(i) \( \dot{V}(x, y) = \frac{\partial V}{\partial x} f + \frac{\partial V}{\partial y} g < 0 \) for \( x, y > 0 \) and \((x, y) \neq (x_*, y_*)\);
(ii) \( \lim V(x, y) = \infty \) as \( x \to 0 \), or \( y \to 0 \), or \( x \to \infty \), or \( y \to \infty \).

The existence of a Lyapunov function for system (5) guarantees that the equilibrium \((x_*, y_*)\) of system (5) is globally asymptotically stable in the positive quadrant. This means that in the long-term, all trajectories of the system will be attracted to the unique positive equilibrium \((x_*, y_*)\).

The method of Lyapunov functions is often used to describe the basin of attraction of an asymptotically stable equilibrium point. By definition, the basin of attraction of such a point is the set of all initial conditions whose solutions tend to the equilibrium point [23]. The basin of attraction is an open invariant set. If the equilibrium \((x_*, y_*)\) of system (5) is globally asymptotically stable, then the basin of attraction is the whole state space (in this case, the positive quadrant of the \(xy\) plane). Thus, after a perturbation, any trajectory will return to the equilibrium.

Global stability properties of general SEIR models have been analysed in detail by Korobeinikov [26, 27].

3.2. Global asymptotic properties of the SEIR model for the case \( \omega_E = 0 \)

For the particular case \( \omega_E = 0 \), the direct Lyapunov method enables us to analytically establish the global asymptotic properties of the model. The assumption \( \omega_E = 0 \) implies that there is no disease-induced death from the exposed compartments \( E \), and that all the disease-induced deaths are from the infectious compartment \( I \). Such an assumption appears to be fairly well justified for a SEIR model.

**Theorem 3.1.** Let \( \omega_E = 0 \). Then, (i) if \( R_0 \leq 1 \), then the model (2) has no positive equilibrium states, and the infection-free equilibrium state \( Q_0 = (N, 0, 0, 0) \) is globally asymptotically stable in \( \mathbb{R}^4_{>0} \).

(ii) If \( R_0 > 1 \), then the model has a positive equilibrium state \( Q_* \), which is globally asymptotically stable in \( \mathbb{R}^4_+ \).

**Proof.** Please note that if the population size \( N \) is assumed constant, then the equation for \( R(t) \) is decoupled from the first three equations, and hence these three equations can be considered separately. Then, assuming that \( \omega_E = 0 \), the system takes the form

\[
\begin{align*}
\dot{S} &= \omega N + \omega I I - \beta SI - \omega S, \\
\dot{E} &= \beta SI - (\lambda + \omega)E, \\
\dot{I} &= \lambda E - (\gamma + \omega + \omega I)I.
\end{align*}
\]

For this system,

\[
R_0 = \frac{\lambda \beta N}{(\lambda + \omega)(\gamma + \omega + \omega I)}.
\]

To establish the properties of the model (2), it suffices to consider this system.

1. **Stability of the infection-free equilibrium state \( Q_0 \).** Let consider the Lyapunov function

\[
U(S, E, I) = S - S_0 \ln S + BE + CI,
\]

where \( B = 1 - \omega I / \beta N, C = B(\lambda + \omega) / \lambda \). It is easy to see that at the point \( Q_0 \) this function reaches its global minimum in \( \mathbb{R}^3_{\geq 0} \). For the SEIR model this function satisfies

\[
\frac{dU}{dt} = \omega N + \omega I I - \beta SI - \omega S - \omega N S_0 \frac{S_0}{S} - \omega I S_0 \frac{S_0}{S} + \beta S_0 I + \omega S_0 + B(\beta SI - (\lambda + \omega)E) + C(\lambda E - (\gamma + \omega + \omega I)I)
\]
Therefore, \( \frac{dV}{dt} \leq 0 \) for all \( S, E, I \geq 0 \), where \( \frac{dU}{dt} = 0 \) holds only when \( R_0 = 1 \) for \( S = S_0 \). It is easy to verify that the infection-free equilibrium state \( Q_0 \) is the only fixed point of the systems in the sub-space \( S = S_0 \), and hence, by the Lyapunov-LaSalle asymptotic stability theorem (cf. [29, p. 28] or [30, p. 58]), the equilibrium state \( Q_0 \) is globally asymptotically stable.

2. **Uniqueness of the infection-free equilibrium state.** The derivative of a Lyapunov function must be equal to zero at an equilibrium state. Since \( \frac{dU}{dt} = 0 \) holds only in the sub-space \( S = S_0 \) (for a particular case \( R_0 = 1 \)), and \( Q_0 \) is the only fixed point in this sub-space, this system has no equilibria in \( \mathbb{R}^3_{>0} \) apart from \( Q_0 \).

3. **Stability and uniqueness of the positive (endemic) equilibrium state.** We consider a Lyapunov function

\[
V(S, E, I) = S - S_* \ln S + B (E - E_* \ln E) + C (I - I_* \ln I),
\]

where \( B = 1 - \omega_I / \beta S_* \), \( C = B(\lambda + \omega)/\lambda \). The point \( Q_* \) is the only stationary point of this function and its global minimum. This function satisfies

\[
\dot{V} = (\omega N + \omega_I I) \left( 2 - \frac{S_0}{S} - \frac{S}{\frac{S_0}{S}} \right) - (B(\lambda + \omega) - C\lambda) E
\]

\[
+ (\beta S_0 - \omega_I - C(\gamma + \omega + \omega_I)) I
\]

\[
= (\omega N + \omega_I I) \left( 2 - \frac{S_0}{S} - \frac{S}{\frac{S_0}{S}} \right) + C(\gamma + \omega + \omega_I)(R_0 - 1) I.
\]

(Here we used \( S_0 = N \) and \( B(\lambda + \omega) = C\lambda \).) That is, for this model \( R_0 \leq 1 \) ensures that \( \frac{dV}{dt} \leq 0 \) for all \( S, E, I \geq 0 \), where \( \frac{dU}{dt} = 0 \) holds only when \( R_0 = 1 \) for \( S = S_0 \). It is easy to see that the infection-free equilibrium state \( Q_0 \) is the only fixed point of the systems in the sub-space \( S = S_0 \), and hence, by the Lyapunov-LaSalle asymptotic stability theorem (cf. [29, p. 28] or [30, p. 58]), the equilibrium state \( Q_0 \) is globally asymptotically stable.

We obtain

\[
\dot{V} = (\omega N + \omega_I I) \left( 2 - \frac{S_0}{S} - \frac{S}{\frac{S_0}{S}} \right)
\]

\[
+ B \beta S_* I S_0 \left( 3 - \frac{S_* E_i}{S_* E_i} - \frac{S_* E_i}{S_* E_i} \right).
\]

Here,

\[
\frac{S E_i I}{S E_i I} + \frac{E_i S_* E_i}{S_* E_i} + \frac{S_* S_*}{S_* S_*} \geq 3, \quad \frac{S}{S} + \frac{S}{S} \geq 2
\]

for all \( S, E, I \geq 0 \), because the arithmetic mean is greater then, or equal to the geometric mean. Therefore, \( \frac{dV}{dt} \leq 0 \) holds for all \( S, E, I \geq 0 \), provided that \( S_*, E_*, I_0 \) are nonnegative, where the equality \( \frac{dV}{dt} = 0 \) holds only on the straight line \( S = S_*, I_0/I_0 = E/E_* \). It is easy to see that for this system \( Q_* \) is the only equilibrium state on this line. Therefore, by Lyapunov-La Salle asymptotic stability theorem [29, 30], the positive equilibrium state \( Q_* \) is globally asymptotically stable in the positive region \( \mathbb{R}^3_+ \).

This completes the proof. \( \Box \)
4. Reducing the SEIR Model to the IR Model
The latent period of an infection is the time interval from the instant of infection to the beginning of the state of infectiousness. The latent period of avian flu is believed to be short, approximately two days [21, 22]. The latently infected population \( E(t) \) become infectious at a constant rate \( \lambda = \frac{365}{2} \). The parameter \( \lambda \) is large relative to the other parameters in the SEIR system and thus \( 1/\lambda \) is a small parameter. Hence, the latently infected population decreases at a much faster rate relative to the proportions of the susceptible, infected and recovered populations. Therefore, due to the short duration of the latent period, it is reasonable to combine the latently infected and infectious classes. The theory of integral manifolds is an elegant mathematical tool to analyse the resulting system. Sobolev [31] and Goldfarb et al. [32] provide comprehensive introductions to the subject.

Let \( U = E + I \). Then \( I = U - E \), and thus we have,

\[
\begin{align*}
\dot{S} &= \omega(S + U + R) - \omega S + (\beta S + \omega E - \omega I) E + (\omega I - \beta S) U \\
\dot{E} &= \beta SU - (\beta S + \lambda + \omega + \omega E) E \\
\dot{U} &= (\beta S - \gamma - \omega I - \omega) U - (\beta S - \gamma - \omega I + \omega E) E \\
\dot{R} &= \gamma U - \gamma E - \omega R.
\end{align*}
\]

We note that the parameter \( \lambda \) is very large relative to \( \beta, \omega \) and \( \omega E \) and so \( E(t) \) is decreasing rapidly. The rate at which \( E(t) \) decreases, \( \dot{E} \), is much faster relative to \( \dot{S}, \dot{U} \) and \( \dot{R} \). Let \( 1/\lambda = \epsilon << 1 \). Then the system becomes:

\[
\begin{align*}
\epsilon \dot{E} &= \epsilon \beta SU - [1 + \epsilon(\beta S + \omega + \omega E)] E \\
\dot{S} &= \omega(S + U + R) - \omega S + (\beta S + \omega E - \omega I) E + (\omega I - \beta S) U \\
\dot{U} &= (\beta S - \gamma - \omega I - \omega) U - (\beta S - \gamma - \omega I + \omega E) E \\
\dot{R} &= \gamma U - \gamma E - \omega R.
\end{align*}
\]

System (8) is a singularly perturbed system of ordinary differential equations. The system of equations

\[
\begin{align*}
\dot{S} &= \omega(S + U + R) - \omega S + (\beta S + \omega E - \omega I) E + (\omega I - \beta S) U \\
\dot{U} &= (\beta S - \gamma - \omega I - \omega) U - (\beta S - \gamma - \omega I + \omega E) E \\
\dot{R} &= \gamma U - \gamma E - \omega R
\end{align*}
\]

represents the slow subsystem, and the equation

\[ \epsilon \dot{E} = \epsilon \beta SU - [1 + \epsilon(\beta S + \omega + \omega E)] E \]

represents the fast subsystem. We will analyse system (8) using the theory of integral manifolds. This will allow us to replace system (8) by another system on an integral manifold with dimension equal to that of the slow subsystem. In this case we will replace system (8) by a system with three ordinary differential equations. Thus we require the following terminology:

A smooth surface \( y = h(x, \epsilon) \) in \( \mathbb{R}^m \times \mathbb{R}^n \) is a slow invariant manifold of the system

\[
\begin{align*}
\dot{x} &= f(x, y, \epsilon) \\
\epsilon \dot{y} &= g(x, y, \epsilon)
\end{align*}
\]

if any trajectory \( x = x(t, \epsilon), y = y(t, \epsilon) \) of this system that has at least one point \( x = x_0, y = y_0 \) in common with this surface lies entirely in this surface, i.e. if \( y_0 = h(x_0, \epsilon) \), then

\[ y(t, \epsilon) = h(x(t, \epsilon), \epsilon). \]
The motion along an invariant manifold is governed by the equation
\[
\dot{x} = f(x, h(x, \epsilon), \epsilon).
\]

If \(x(t, \epsilon)\) is a solution of this equation, then the pair \((x(t, \epsilon), y(t, \epsilon))\), where \(y(t, \epsilon) = h(x(t, \epsilon), \epsilon)\) is a solution of the original system (10), since it defines a trajectory on the invariant manifold.

The theory of integral (invariant) manifolds states that system (10) has an unique integral manifold of the form
\[
M := \{(x, y, t) : y = h(x, t, \epsilon)\}
\]
that can be represented as the asymptotic expansion of \(h(x, t, \epsilon)\) in integer powers of the small parameter \(\epsilon\):
\[
h(x, t, \epsilon) = h_0(x, t) + \epsilon h_1(x, t) + \epsilon^2 h_2(x, t) + \ldots + \epsilon^k h_k(x, t) + \ldots
\]

We will find the slow integral manifold \(E = E(S, U, R, \epsilon)\) of (8) in the form of an asymptotic expansion:
\[
E = E_0(S, U, R) + \epsilon E_1(S, U, R) + O(\epsilon^2).
\]

We note that
\[
\epsilon \frac{dE}{dt} = \epsilon \left[ \frac{\partial E}{\partial S} \dot{S} + \frac{\partial E}{\partial U} \dot{U} + \frac{\partial E}{\partial R} \dot{R} \right],
\]
and we substitute the asymptotic expansion of \(E\) into the fast subsystem of (8):
\[
\epsilon \beta SU - [1 + \epsilon (\beta S + \omega + \omega_E)](E_0 + \epsilon E_1 + \ldots) = \\
\epsilon \frac{\partial E_0}{\partial S} \{ \omega(S + U + R) - \omega S + (\beta S + \omega_E - \omega_I)E_0 + (\omega_I - \beta S)U \} \\
+ \frac{\partial E_0}{\partial U} \{ (\beta S - \gamma - \omega_I - \omega)U - (\beta S - \gamma - \omega_I + \omega_E)E_0 \} \\
+ \frac{\partial E_0}{\partial R} \{ \gamma U - \gamma E_0 - \omega R \} + O(\epsilon^2).
\]

From (11), we obtain
\[
\epsilon^0 : \quad E_0 = 0.
\]
\[
\epsilon^1 : \quad \beta SU - (\beta S + \omega + \omega_E)E_0 - E_1 = 0 \Rightarrow E_1 = \beta SU.
\]

Thus, the slow integral manifold \(E = E(S, U, R, \epsilon)\) of (8) is given by
\[
E = E(S, U, R, \epsilon) = \epsilon \beta SU + O(\epsilon^2),
\]
and consequently \(I = U - \epsilon \beta SU + O(\epsilon^2)\). Therefore, on substituting equation (12) into system (9), we have
\[
\dot{S} = \omega(S + U + R) - \omega S + (\omega_I - \beta S)U + \epsilon \beta SU(\beta S + \omega_E - \omega_I) + O(\epsilon^2)
\]
\[
\dot{U} = (\beta S - \gamma - \omega_I - \omega)U - (\beta S - \gamma - \omega_I + \omega_E)\epsilon \beta SU + O(\epsilon^2)
\]
\[
\dot{R} = \gamma U - \omega R - \gamma \epsilon \beta SU + O(\epsilon^2).
\]

In the limiting case, i.e., \(\epsilon \to 0\), system (13) becomes the following:
\[
\dot{S} = \omega(S + U + R) - \omega S + (\omega_I - \beta S)U
\]
\[
\dot{U} = (\beta S - \gamma - \omega_I - \omega)U
\]
\[
\dot{R} = \gamma U - \omega R.
\]
Figure 2. Comparing solutions of the SEIR model to the IR solution for various $\lambda$ values,
$t = 0.5$. The convergence of the SEIR solution to the IR solution becomes more pronounced as $\lambda$
grows larger.

Since $I = U - \epsilon \beta SU + O(\epsilon^2)$, then in the limiting case $I \approx U$. Thus, we may replace $U$
with the more intuitively clear variable $I$, standing for the infected (and infectious) population, to
obtain a Susceptible-Infected-Recovered (SIR) system:

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

Finally, since we have assumed the total population $N$ to be constant (equality (1)), it suffices
to consider a two-dimensional system. It is conventional to omit the $R$ equation to obtain a
Susceptible-Infected SI system. However, against intuition, we omit the $S$ equation and obtain
the following set of equations, which we term the IR model:

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

The results of numerical experiments indicate that the IR model and the SEIR model of the
seabird colony are in close agreement. The convergence of the SEIR system to the IR system
was examined for various values of $\lambda$, the rate that individuals move from the $E$
class to the $I$
class, using tools provided by the mathematical software package Mathematica. As the value of $\lambda$
increases, the latent period of the disease becomes shorter; the IR model assumes that there is no
latent period of the disease. Figure 2 confirms that the IR model is a good approximation to the
SEIR model. The convergence of the SEIR system to the IR system becomes more pronounced
for larger values of $\lambda$ (or as the latent period tends to zero). Figure 2 also illustrates that there
is a significant outbreak of the disease in the first year after the pathogen is introduced into the
colony. This agrees with the results of numerical simulations of the SEIR model conducted by
Clancy et al. [10].
5. Analysis of the IR Model

5.1. Lyapunov Analysis

A simple and elegant Lyapunov function may be constructed for the IR system of ODEs and therefore, enabling us to prove global stability of the IR model. Consider the rate of change of the infected population in system (15):

$$\dot{I} = \beta(N - I - R)I - (\gamma + \omega + \omega_I)I.$$

Set

$$p = \beta N - (\gamma + \omega + \omega_I).$$

Then \(p\) is a positive constant and system (15) becomes

$$\begin{align*}
\dot{I} &= (p - \beta I - \beta R)I \\
\dot{R} &= \gamma I - \omega R.
\end{align*}$$

The coordinates of the positive equilibrium state of system (16) are given by

$$\begin{align*}
I^* &= \frac{\omega p}{\beta(\gamma + \omega)}, \\
R^* &= \frac{\gamma p}{\beta(\gamma + \omega)}.
\end{align*}$$

Hence,

$$\begin{align*}(p - \beta I^* - \beta R^*) &= 0 \Rightarrow p = \beta(I^* + R^*), \\
\gamma I^* - \omega R^* &= 0.
\end{align*}$$

Therefore, system (16) becomes

$$\begin{align*}
\dot{I} &= I(\beta(I^* - I) + \beta(R^* - R)) \\
\dot{R} &= \gamma(I - I^*) - \omega(R - R^*).
\end{align*}$$

Construct a trial Lyapunov function for system (17):

$$V(I, R) = I - I^* \ln I + \alpha(R - R^*)^2.$$  \hspace{1cm} (18)

To verify that the function (18) is a Lyapunov function for system (17), and hence, for system (15), we must show that

$$\dot{V}(I, R) = \frac{\partial V}{\partial I} f(I, R) + \frac{\partial V}{\partial R} g(I, R) < 0$$

for \(I, R > 0\) and \((I, R) \neq (I^*, R^*)\). The required partial derivatives are given by:

$$\begin{align*}
\frac{\partial V}{\partial I} &= 1 - \frac{I^*}{I}, \\
\frac{\partial V}{\partial R} &= 2\alpha(R - R^*).
\end{align*}$$

For our system we have,

$$f(I, R) = I(\beta(I^* - I) + \beta(R^* - R)), \quad g(I, R) = \gamma(I - I^*) - \omega(R - R^*).$$
For $I, R > 0$ we have

$$\frac{d}{dt} V(I, R) = \frac{\partial V}{\partial I} f(I, R) + \frac{\partial V}{\partial R} g(I, R)$$

$$= (2\alpha(R - R_*)((\gamma(I - I_*)) - \omega(R - R_*))) + I \left(\frac{-I_*}{I} + 1\right) (\beta(I_* - I) + \beta(R_* - R))$$

$$= -2\alpha\omega(R - R_*)^2 + 2\alpha\gamma(R - R_*)(I - I_*) - \beta(R - R_*)(I - I_*)^2$$

$$= -2\alpha\omega(R - R_*)^2 + (2\alpha\gamma - \beta)(R - R_*)(I - I_*) - \beta(I_* - I)^2.$$

(19)

Now choose $\alpha = \beta/2\gamma$. Then equation (19) becomes

$$\dot{V}(I, R) = -2\alpha\omega(R - R_*)^2 - \beta(I_* - I)^2 < 0, I \neq I_*, R \neq R_*.$$

(20)

Clearly, equation (20) is always negative except at equilibrium. Thus, all trajectories will eventually tend to the equilibrium $(I_*, R_*)$.

We also have

$$V(I, R) \to \infty \text{ as } I \to 0, I \to \infty \text{ and } V(I, R) \to \infty \text{ as } R \to 0, R \to \infty.$$

We can therefore conclude that equation (18) is a Lyapunov function for system (15) and that the equilibrium $(I_*, R_*)$ is globally asymptotically stable in the positive quadrant.

5.2. Numerical Analysis of IR Model

Theoretical analysis of the IR model using the method of Lyapunov functions verified all solutions of system (15) approach an equilibrium in the long-term. Numerical experiments confirm this prediction. Using the parameters described in Section 2.5 and setting $R_0 = 2, 10$, the IR system of equations was solved numerically in Mathematica. In Figures 3 and 4, we see that the disease does not die out; it persists in the seabird population. The infectious population tends to an asymptotically stable positive equilibrium in the long-term. As time increases, the infectious population $I(t)$ oscillates with decreasing amplitude. If $R_0 = 2$, it takes approximately 80 years for no further oscillations of $I(t)$ to be observed; however, if $R_0 = 10$ the system reaches a stable equilibrium in approximately 20 years. We also note that for larger $R_0$, the oscillations of $I(t)$ are of greater amplitude and occur more frequently. This is because the transmission parameter $\beta$ is large.

6. Discussion and Conclusions

In this paper, we formulated a SEIR model which incorporates the features of a seabird colony and the H5N1 virus. Using the theory of integral manifolds, we rigorously reduced the SEIR system to the IR model, a system dependent on the infected and recovered populations only. By constructing Lyapunov functions, we analysed the long-term behaviour of the SEIR model for a particular case when the disease-induced mortality of the exposed hosts is absent, and the IR model. All trajectories tend to an asymptotically stable positive equilibrium in the long-term, i.e., the disease persists in the population.

From the results of numerical experiments, we saw that the IR model retains the essential features of the SEIR model. A major epidemic occurs in the first year. When $R_0 = 2$, the number of infectives rises sharply to a maximum of 1,500 and then decreases to almost zero in a period of 0.2 years. Subsequent outbreaks are smaller; damped oscillations of the infective and
recovered populations are observed. The size and frequency of these outbreaks depend upon $R_0$. When $R_0 = 10$, outbreaks are more severe and occur with greater frequency compared to those observed when $R_0 = 2$. Therefore, the infected and recovered populations damp to equilibrium in a much shorter time frame, about 20 years. Once an individual enters the $R$ class, it acquires permanent immunity from the disease. Since more individuals become infected when $R_0$ is large, consequently, more individuals recover and cannot return to the susceptible class. Thus, the pool of susceptibles becomes exhausted in a much shorter time frame. Therefore, the system approaches equilibrium faster.

System (2) is a multi-scaled system. Influenza dynamics in seabirds exhibit fast dynamics at the individual and colony levels when compared to seabird demography. The average lifespan of a seabird (about 10-20 years) is thousands of times greater than the duration of the latent
period of H5N1 (about 1-2 days). Using singular perturbation theory greatly simplifies the four-dimensional SEIR system. The global stability analysis of the two-dimensional IR model is much simpler compared to the corresponding analysis for the SEIR system.

Reducing the SEIR model to the IR system also enables us to readily develop and analyse interesting and useful variants of the basic model. For example, the IR model may be subject to perturbations from seasonality. Environmental factors that change seasonally, e.g., weather conditions, may have a significant impact on the dynamics of a disease. Altizer et al. [33] provide a thorough introduction to the incorporation of seasonality into epidemiological models. Another interesting point worth investigation is the effect of nonlinearity in the transmission term, e.g., $\beta SI^{\alpha}$, on the model. Korobeinikov [28] gives reasons why a modification to the standard transmission term $\beta SI$ may be necessary. Lyapunov functions may be used to analyse the resulting perturbed nonlinear systems.

Future work may include incorporating the effect of different strains of the H5N1 virus. Casagrandi et al. [34] developed a simple ODE model to study the consequences of the drift mechanism for influenza A viruses. Studying this in the context of seabird colonies may be potentially very interesting and significant.

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