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JUMP-DIFFUSION MODEL FOR THE GLOBAL SPREAD OF AN AMPHIBIAN DISEASE

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Abstract. A system of jump-diffusion stochastic differential equations is considered for modelling the dynamics of the spread of an amphibian disease. In this investigation, it is assumed that the amphibians are located in M regions which are widely and uniformly spaced on the surface of the earth and that the disease is present initially in only one region. Within each region, the amphibians live in N separate patches. A jump-diffusion stochastic system is derived for the number of infected patches in each of the M regions. Computational simulations are performed and compared with results predicted by a deterministic SIS model, a continuous-trajectory stochastic differential equation model, and Monte Carlo calculations. It is seen that the rate of spread predicted by the jump-diffusion model agrees well with that predicted by Monte Carlo calculations. Indeed, if there is a step increase in the transmission rates or a step decrease in the recovery rates, then the disease can spread globally from region to region at an exponential rate.

Key Words. amphibian disease, stochastic differential equation, jump diffusion, chytridiomycosis

1. Introduction

Since the 1970's, populations of amphibians have declined or vanished worldwide (see, e.g., Berger et al., 1998, Carey et al., 1999, Daszak et al., 1999, Morell, 1999). Mass mortalities have been reported in North America, Central America, South America, Europe, and Australia. The severity, the rapid rate, and the abruptness of the amphibian population declines have led to much scientific interest and several hypotheses for the causes of the population declines have been proposed. It was hypothesized that changes in the global environment, such as increased ultraviolet light, global warming, and pollutants, were responsible (see, e.g. Alexander and Eisched, 2001, Corn and Muths, 2002, Stallard 2001). After the amphibian chytrid fungus (*Batrachochytrium dendrobatidis*) was found at sites of mass mortality in Australia and Central America (Daszak et al., 2001), it was hypothesized that emerging infectious diseases (EIDs) were responsible for the amphibian die-offs. In addition, it has been hypothesized that a combination of factors is responsible (Daszak, et al., 2001, Rollins-Smith, et al., 2002). For example, global warming may

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have changed the behavior of montane amphibians resulting in increased transmission rates of a disease or, possibly, increased ultraviolet light has decreased the resistance of the amphibians to infections (Daszak et al., 2001).

Of particular interest has been the global spread of chytrid fungus with the fungus recorded in the United States in the 1970's, Australia, Central America, and South America in the 1980's, and Europe and Africa in the 1990's (Berger et al., 1999, Speare and Berger, 2000). Chytrid fungus is interesting as it has emerged in pristine sites, infected a wide variety of hosts, and has caused severe population declines in disparate regions (Daszak et al., 1999, 2000). Chytrid fungus spreads through waterborne zoospores and apparently prefers cool temperatures for optimal growth (Daszak et al., 2001, Sparrow, 1968). As a result, montane amphibians have been the most susceptible to the disease (Berger et al., 1999, Bosch, et al., 2001) even though the populations are generally located at widely separated regions on the earth. The international trade in amphibians, which has undoubtedly increased since the 1970's, has been a possible mechanism for the introduction of chytrid fungus (Daszak et al., 2001).

In the present investigation, the dynamics of the global spread of an amphibian disease are studied. Of particular interest is determining how increased transmission rates or increased susceptibility affect the rate of global spread of a disease. To study these effects, the assumption is made that the disease initially is present at only one location on the earth's surface. (For example, a virulent strain may evolve at a certain location.) Unfortunately, with regard to a particular disease such as chytrid fungus, appropriate data may never be obtained to support or disprove such an assumption. For example, one may argue that chytrid fungus has been distributed worldwide for thousands of years and we have just recently begun to identify the effects of the fungus.

In addition to the assumption that the disease originates at one location, it is also assumed that the amphibian populations susceptible to the disease are located at disparate regions on the surface of the earth. With these assumptions, a stochastic model is formulated for the global spread of an amphibian disease, in particular, a jump-diffusion model is introduced and studied. Often, in mathematical models for dispersal and growth, small fractions of the population can immediately diffuse with the result that migrated populations can quickly increase. However, a jump-diffusion model realizes that the populations are discrete and that migrated populations cannot undergo growth unless at least one individual has moved. Jump-diffusion models have recently become popular in mathematical finance in order to account for random discrete jumps in prices (see, e.g., Hanson and Westman, 2002, Runggaldier, 2003). In the present investigation, a jump-diffusion model is considered for modelling the spread of amphibian infections. (In addition, it is worthwhile to note that the mathematical model developed here is not a small-world network (Collins and Chow, 1998, Watts and Strogatz, 1998) as all regions are interconnected. However, the problem studied may be considered as a metapopulation since a collection of amphibian subpopulations exist on a system of habitat patches (Hanski, 1999, Marsh and Trenham, 2001).) The purpose of the investigation is to assess the impact of increased transmission coefficients and decreased recovery coefficients on the rate that an amphibian disease can spread globally. The model may help improve our understanding of how a disease spreads rapidly through widely separated populations and whether changing global conditions are partly responsible for the amphibian population declines.

2. Mathematical Model

2.1. Introduction. It is first useful to explain the geometrical assumptions made concerning the amphibian populations. A rather simple set of geometric assumptions are made in the present investigation. One advantage of such simple assumptions is that they readily allow the calculational results to be duplicated and compared using different mathematical models for the spread of the disease. First, it is assumed that the amphibian populations are located in M regions which are approximately uniformly spaced on the surface of the earth. Within each region the amphibians live in N patches. Hence, the amphibians are located in a total of NM patches. In each patch, more than one species of amphibian may be present that is susceptible to the disease. The transmission and recovery rates are assumed to be the same for all the susceptible species. For the computational simulations, described in a later section, M is taken as 30 and N is taken as 25 and each region is assumed to be 250 km by 250 km. Four regions are located at 60° North, four at 60° South, seven at 30° North, seven at 30° South, and eight at 0°. At each latitude, the regions are evenly spaced with respect to longitude. For example, the eight regions at the equator have longitudes: 0°, 45°, 90°, 135°, 180° East, and 45°, 90°, and 135° West. The regions are thus uniformly distributed on the surface of the earth and the average distance between the 30 regions is 9999 km. (For comparison, the average distance between two randomly chosen points on the earth's surface is $\frac{\pi}{2} \times 6378$ km $= 10019$ km.) In addition, it is assumed that the 25 patches are randomly spaced within each region.

2.2. A Deterministic SIS Model. Before deriving a stochastic jump-diffusion model, it is useful to consider a standard deterministic metapopulation mathematical model (see, e.g. Hanski, 1999) for the spread of the infection. Let $S_j(t)$ and $I_j(t)$ be the number of susceptible patches and infected patches, respectively, in the jth region at time t. In the present investigation, the disease is assumed to spread rapidly and vigorously within a patch and the population of each patch is therefore assumed at any time to be either totally susceptible or totally infected. That is, the population in each patch is never partially infected or partially susceptible. In addition, it is assumed that a patch may recover from the infection albeit with perhaps a significant loss of the amphibian population from the patch. However, it is assumed that a patch cannot disappear or completely die out; a patch is either susceptible or infected. It follows then that $I_j(t) + S_j(t) = N$ for $j = 1, 2, \ldots, M$ as there are N patches for each region.

Now, let $\gamma_j I_j(t)$ be the rate that an infected patch recovers from the infection, i.e., a patch changes from infected to susceptible at this rate. Let $\alpha_j I_j(t) S_j(t)$ be the rate that infected patches infect susceptible patches in the jth region. Let $\beta_{kj} I_k(t) S_j(t)$ be the rate that infected patches in region k infect patches in region j. An SIS (see, e.g., Allen, 2003, Brauer and Castillo-Chávez, 2001, Hethcote, 2000, Hethcote and Yorke, 1984) deterministic model for the rate of change of the number of infected and susceptible patches has the form:

$$(1) \qquad \begin{cases} \dfrac{dS_j(t)}{dt} = \gamma_j I_j(t) - \alpha_j I_j(t) S_j(t) - \left(\sum_{k=1, k\neq j}^{M} \beta_{kj} I_k(t)\right) S_j(t) \\ \dfrac{dI_j(t)}{dt} = -\gamma_j I_j(t) + \alpha_j I_j(t) S_j(t) + \left(\sum_{k=1, k\neq j}^{M} \beta_{kj} I_k(t)\right) S_j(t) \end{cases}$$

for $j = 1, 2, \ldots, M$. Notice that

$$\frac{d(S_j(t) + I_j(t))}{dt} = 0$$

so that $S_j(t) + I_j(t) = S_j(0) + I_j(0) = N$ for each j. Also, notice that the infection spreads between patches within the jth region at a rate determined by the parameter α_j. That is, each infected patch in a region is equally effective at spreading the disease to any susceptible patch in the region. However, the rate that the infection spreads between two regions, say regions j and k, is determined by the parameter β_{jk}. Thus, regions may not be equally effective in spreading the disease. Indeed, it is assumed, in the present investigation, that $\beta_{jk} = \beta_R/d_{jk}$ where d_{jk} is the distance on the earth's surface between regions j and k and β_R is a constant. Also, it is assumed in the computational examples that α_j is the same for each region, specifically, $\alpha_j = \beta_P/d_{avg}$ where β_P is a constant and $d_{avg} = 130$ km is the average distance between patches in the region. (The average distance between two randomly chosen points in a square of side length 250 km is approximately $.52 \times 250$ km $= 130$ km.)

Before developing a stochastic model for the dynamics of the infection, it is useful to understand the equilibrium solutions of (1). If $N - \gamma_j/\alpha_j > 0$ then the disease persists in region j independently of the presence or absence of the infection in the other regions and the equilibrium number of infected patches in region j is greater than or equal to $N - \gamma_j/\alpha_j$. Indeed, for the SIS model, the basic reproduction number is a useful parameter. The basic reproduction number is defined to be the average number of secondary infections produced when an infected individual is introduced into a population where everyone is susceptible (Anderson and May, 1991, Dietz, 1975, and Hethcote, 2000). For only one region $M = 1$, the basic reproduction number is $\mathcal{R}_0 = \frac{\alpha_1 N}{\gamma_1}$. If $\mathcal{R}_0 > 1$, then the disease can persist in the region in the absence of inter-regional spread. However, if $\mathcal{R}_0 < 1$, then the disease-free equilibrium is locally asymptotically stable. For the full SIS epidemic model (1) with $M > 1$, the basic reproduction number is the spectral radius of the following matrix (Diekmann, 1990, and van den Driessche and Watmough, 2002):

$$N \begin{bmatrix} \frac{\alpha_1}{\gamma_1} & \frac{\beta_{21}}{\gamma_2} & \frac{\beta_{31}}{\gamma_3} & \cdots & \frac{\beta_{M1}}{\gamma_M} \\ \frac{\beta_{12}}{\gamma_1} & \frac{\alpha_2}{\gamma_2} & \frac{\beta_{32}}{\gamma_3} & \cdots & \frac{\beta_{M2}}{\gamma_M} \\ \vdots & \vdots & \vdots & \cdots & \vdots \\ \frac{\beta_{1M}}{\gamma_1} & \frac{\beta_{2M}}{\gamma_2} & \frac{\beta_{3M}}{\gamma_3} & \cdots & \frac{\alpha_M}{\gamma_M} \end{bmatrix},$$

which indicates the importance of the ratios $\frac{\alpha_j}{\gamma_j}$ and $\frac{\beta_{jk}}{\gamma_j}$. Furthermore, assuming that $N > \gamma_j/\alpha_j$, good approximations to the equilibrium numbers of susceptible and infected patches in the jth region are given by:

$$(2) \qquad \begin{cases} S_j^{eq} \approx (N - \frac{\gamma_j}{\alpha_j}) / \left(\frac{\alpha_j}{\gamma_j}(N - \frac{\gamma_j}{\alpha_j}) + \sum_{k=1, k\neq j}^{M} \frac{\beta_{kj}}{\gamma_j}(N - \frac{\gamma_k}{\alpha_k}) \right) \\ I_j^{eq} \approx N - S_j^{eq} \end{cases}$$

for $j = 1, 2, \ldots, M$.

Now suppose that at some time there is a step increase in the transmission coefficient β_R or a step decrease in the recovery coefficient γ_j for all regions. That is, at some time, the transmission rate of the disease has increased or the recovery rate has decreased. Then (1) predicts that the disease will spread rapidly at an exponential rate at least initially. To see this, suppose, for simplicity, that $\alpha_j =$

$\alpha, \gamma_j = \gamma$ and $\beta_{kj} = \beta$ for all j and k and the initial conditions for each region are the same so that $I_j(t) = I_k(t)$ for $t \geq 0$ and $1 \leq j, k \leq M$. Then the equation for the number of infected patches in the jth region has the form:

$$\frac{dI_j}{dt} = -\gamma I_j + \alpha I_j(N - I_j) + \tilde{\beta} I_j(N - I_j) \tag{3}$$

where $\tilde{\beta} = (M-1)\beta$. This equation can be solved exactly to yield:

$$I_j(t) = \left(\frac{aI_j(0)}{a - bI_j(0)} \exp(at)\right) / \left(\left(\frac{bI_j(0)}{a - bI_j(0)} \exp(at)\right) + 1\right) \tag{4}$$

where $a = \alpha N + \tilde{\beta} N - \gamma$ and $b = \alpha + \tilde{\beta}$. For $I_j(0)$ small compared with a/b, equation (4) exhibits an exponential rate of increase before approaching the equilibrium value of a/b.

Although the deterministic model (1) is useful for estimating equilibrium values and for developing an understanding of the approximate dynamics of the infection, equation (1) considerably overestimates the rate that the disease spreads among the widely separated regions for a given set of transmission and recovery coefficients. To see why, suppose that the disease is initially only present in one region, say region 1. (Such an assumption is made later in the computational simulations.) The deterministic model (1) predicts that immediately for any time $t > 0$, the disease has spread to every region from region 1. This is due to the inherent assumption in (1) that $I_j(t)$ and $S_j(t)$, $j = 1, 2 \ldots, M$, are continuous variables. Therefore, although $I_j(t) + S_j(t) = N$ for each j, $I_j(t)$ and $S_j(t)$ are fractional quantities for $t > 0$. Hence, for $t > 0$, $I_j(t) > 0$ for each region j and the infection builds up exponentially as indicated by (4) for each region. The infection thus increases at an unrealistically rapid rate because there is only a small probability in any given time interval that the disease actually spreads from region 1 to the other regions. To remedy this problem, a stochastic model is required. One way to formulate a stochastic model is to add the appropriate stochastic terms to (1) as in Allen (1999), Allen and Victory, (2003), or Allen (2003) to account properly for the randomness in the disease transmission and recovery. The result is a system of Itô stochastic differential equations (Øksendahl, 1985) involving an M-dimensional Wiener process and which can be approximately computed using various numerical procedures (Kloeden et al., 1997, Schurz, 2002, Talay, 1995). However, this approach only partially remedies the problem as fractional increases in the infection from one region to another still occur. However, by considering the problem using Poisson processes as in the next section, a jump-diffusion stochastic differential equation system is obtained that accurately takes into account the random nature of the disease propagation and, indeed, agrees consistently with Monte Carlo simulations.

2.3. A Jump-Diffusion Model. A jump-diffusion stochastic model for the spread of the infection is developed in this section. The model consists of a system of stochastic differential equations and models the random nature of the discrete jumps in the dynamics of the infection. Let Δt be a small interval in time. During this time interval, there are three possibilities: no change occurs, an infected patch in some region recovers, or a susceptible patch becomes infected. Before deriving the jump-diffusion model, it is useful to understand a Monte Carlo approach. In a Monte Carlo procedure, at each time step, each patch in every region is individually considered. The probability for the patch to change its status (e.g., infected to susceptible) is computed and this probability is compared with an appropriately selected random number generated uniformly on $[0, 1]$. If the random number is

smaller in magnitude than the probability, then the status of the patch is changed, otherwise, no change is made for that patch. This continues for all patches for each time step until the final time is reached. Although the Monte Carlo approach is accurate, simple, and straightforward, the approach provides little insight into the dynamics of the spread of the infection and Monte Carlo computations are generally slow.

In the present investigation, the random process is modelled by a system of jump-diffusion stochastic equations which give some insight into the phenomenon and can be rapidly approximately computed. Consider the two possibilities for a change in time Δt. Let $\begin{bmatrix} \Delta S_j \\ \Delta I_j \end{bmatrix}^{(1)} = \begin{bmatrix} 1 \\ -1 \end{bmatrix}$ represent the recovery of an infected patch in region j during time interval Δt. Furthermore, let $\begin{bmatrix} \Delta S_j \\ \Delta I_j \end{bmatrix}^{(2)} = \begin{bmatrix} -1 \\ 1 \end{bmatrix}$ represent a susceptible patch becoming infected in region j during time interval Δt. These are Poisson processes and the probabilities for the changes in a small time interval Δt are (to order $(\Delta t)^2$), respectively,

$$p_j^{(1)} = \lambda_j^{(1)}(t)\Delta t = \gamma_j I_j(t)\Delta t$$

and

$$p_j^{(2)} = \lambda_j^{(2)}(t)\Delta t = \alpha_j I_j(t) S_j(t)\Delta t + \left(\sum_{k=1, k\neq j}^{M} \beta_{kj} I_k(t) \right) S_j(t)\Delta t.$$

where the above equations also define $\lambda_j^{(1)}(t)$ and $\lambda_j^{(2)}(t)$. Finding the mean and covariance matrix of the change, one obtains:

$$\mathrm{E}\begin{bmatrix} \Delta S_j \\ \Delta I_j \end{bmatrix} = p_j^{(1)} \begin{bmatrix} \Delta S_j \\ \Delta I_j \end{bmatrix}^{(1)} + p_j^{(2)} \begin{bmatrix} \Delta S_j \\ \Delta I_j \end{bmatrix}^{(2)} = \begin{bmatrix} \lambda_j^{(1)}(t) - \lambda_j^{(2)}(t) \\ \lambda_j^{(2)}(t) - \lambda_j^{(1)}(t) \end{bmatrix} \Delta t$$

and

$$\mathrm{E}\left[\begin{bmatrix} \Delta S_j \\ \Delta I_j \end{bmatrix} [\Delta S_j \;\; \Delta I_j] \right] = \begin{bmatrix} \lambda_j^{(1)}(t) + \lambda_j^{(2)}(t) & -\lambda_j^{(1)}(t) - \lambda_j^{(2)}(t) \\ -\lambda_j^{(1)}(t) - \lambda_j^{(2)}(t) & \lambda_j^{(1)}(t) + \lambda_j^{(2)}(t) \end{bmatrix} \Delta t.$$

The above considerations imply that the number of susceptible patches and infected patches in region j satisfy the following equations for small Δt:

$$\begin{cases} S_j(t+\Delta t) = S_j(t) + \Delta q_j^{(1)}(t) - \Delta q_j^{(2)}(t) \\ \\ I_j(t+\Delta t) = I_j(t) - \Delta q_j^{(1)}(t) + \Delta q_j^{(2)}(t) \end{cases} \tag{5}$$

for $j = 1, 2, \ldots, M$ where $\Delta q_j^{(1)}(t)$ and $\Delta q_j^{(2)}(t)$ are Poisson processes with intensities $\lambda_j^{(1)}(t)$ and $\lambda_j^{(2)}(t)$. That is, for a small time interval Δt,

$$\Delta q_j^{(1)}(t) = \begin{cases} 1 \;\text{ with probability }\; \lambda_j^{(1)}(t)\Delta t \\ \\ 0 \;\text{ with probability }\; 1 - \lambda_j^{(1)}(t)\Delta t \end{cases}$$

and

$$\Delta q_j^{(2)}(t) = \begin{cases} 1 \;\text{ with probability }\; \lambda_j^{(2)}(t)\Delta t \\ \\ 0 \;\text{ with probability }\; 1 - \lambda_j^{(2)}(t)\Delta t. \end{cases}$$

As $\Delta t \to 0$, equation (5) has the form of a jump-diffusion stochastic system:

$$\begin{cases} \dfrac{dS_j(t)}{dt} = \dfrac{dq_j^{(1)}(t)}{dt} - \dfrac{dq_j^{(2)}(t)}{dt} \\ \\ \dfrac{dI_j(t)}{dt} = -\dfrac{dq_j^{(1)}(t)}{dt} + \dfrac{dq_j^{(2)}(t)}{dt} \end{cases} \tag{6}$$

for $j = 1, 2, \ldots, M$ where $q_j^{(1)}(t)$ and $q_j^{(2)}(t)$ are doubly stochastic Poisson processes (Runggaldier, 2003) with time-dependent random intensities

$$\lambda_j^{(1)}(t) = \gamma_j I_j(t) \tag{7}$$

and

$$\lambda_j^{(2)}(t) = \alpha_j I_j(t) S_j(t) + \left(\sum_{k=1, k\neq j}^{M} \beta_{kj} I_k(t) \right) S_j(t). \tag{8}$$

Equation (6) is a jump-diffusion system of stochastic differential equations. It is called jump-diffusion as the values of $S_j(t)$ and $I_j(t)$ experience jump changes at random times determined by the intensities. In addition, as the intensities $\lambda_j^{(1)}(t)$ and $\lambda_j^{(2)}(t)$ depend on the stochastic quantities $S_j(t)$ and $I_j(t)$, $q_j^{(1)}(t)$ and $q_j^{(2)}(t)$ are referred to as doubly stochastic Poisson processes. System (6) can be approximated in a stepwise manner for small Δt using equation (5) which is a form of Euler's method (Hausenblaus, 2002, Kubilius and Platen, 2002, Liu and Li, 2000, Protter and Talay, 1997) for solving (6).

Equation (6) will be simplified by noticing that $d(I_j(t) + S_j(t))/dt = 0$ for $j = 1, 2, \ldots, M$. However, it is first worthwhile to compare (6) with a continuous trajectory stochastic differential system that agrees with (6) in having the identical values of $\mathrm{E}[I_j(t+\Delta t) - I_j(t)]$, $\mathrm{E}[I_j(t+\Delta t) - I_j(t)]^2$, $\mathrm{E}[S_j(t+\Delta t) - S_j(t)]$, $\mathrm{E}[S_j(t+\Delta t) - S_j(t)]^2$ to order $(\Delta t)^2$. Indeed, using the procedure described in (Allen, 1999, Allen and Victory, 2003, Allen, 2003), one obtains the continuous-trajectory Itô stochastic differential equation system:

$$\begin{cases} \dfrac{dS_j}{dt} = \lambda_j^{(1)} - \lambda_j^{(2)} + \sqrt{\frac{1}{2}(\lambda_j^{(1)} + \lambda_j^{(2)})} \left(\dfrac{dW_j^{(1)}}{dt} - \dfrac{dW_j^{(2)}}{dt} \right) \\ \\ \dfrac{dI_j}{dt} = -\lambda_j^{(1)} + \lambda_j^{(2)} + \sqrt{\frac{1}{2}(\lambda_j^{(1)} + \lambda_j^{(2)})} \left(-\dfrac{dW_j^{(1)}}{dt} + \dfrac{dW_j^{(2)}}{dt} \right) \end{cases} \tag{9}$$

for $j = 1, 2, \ldots, M$ where $\lambda_j^{(1)}(t)$ and $\lambda_j^{(2)}(t)$ are given by (7) and (8) and $W_j^{(1)}(t)$ and $W_j^{(2)}(t)$ are independent Wiener processes for each j. Notice that (9) also satisfies $d(I_j(t)+S_j(t))/dt = 0$ for $j = 1, 2, \ldots, M$. Generally, under most initial conditions, the continuous-trajectory stochastic system (9) would agree well with Monte Carlo simulations as discussed, for example, in (Allen and Allen, 2003). However, if the infected population sizes in many regions are initially zero, then stochastic system (9) overestimates the rate of spread of the infection. This is because $S_j(t)$ and $I_j(t)$ are continuous variables in (9) rather than discrete variables as in (6) and changes occur continuously rather than discretely allowing the infection to spread too rapidly. For small initial population sizes, the jump-diffusion model (6) agrees better with Monte Carlo calculations than model (9).

One can simplify (6) by using the fact that $I_j(t) + S_j(t) = N$ for $t \geq 0$ and $j = 1, 2, \ldots, M$. Indeed, setting $S_j(t) = N - I_j(t)$ in (6), one obtains that:

$$\frac{dI_j(t)}{dt} = -\frac{dq_j^{(1)}(t)}{dt} + \frac{dq_j^{(2)}(t)}{dt} \tag{10}$$

for $j = 1, 2, \ldots, M$ where $q_j^{(1)}(t)$ and $q_j^{(2)}(t)$ are doubly stochastic Poisson processes with intensities:

$$\begin{cases} \lambda_j^{(1)}(t) = \gamma_j I_j(t) \\ \\ \lambda_j^{(2)}(t) = \alpha_j I_j(t)(N - I_j(t)) + (\sum_{k=1, k\neq j}^{M} \beta_{kj} I_k(t))(N - I_j(t)). \end{cases} \tag{11}$$

Using Euler's method, stochastic system (10) can be approximated in a stepwise manner using

$$I_j(t + \Delta t) = I_j(t) - \Delta q_j^{(1)}(t) + \Delta q_j^{(2)}(t) \tag{12}$$

for $j = 1, 2, \ldots, M$ where for small Δt,

$$\Delta q_j^{(m)}(t) = \begin{cases} 1 \text{ with probability } \lambda_j^{(m)}(t)\Delta t \\ \\ 0 \text{ with probability } 1 - \lambda_j^{(m)}(t)\Delta t \end{cases} \tag{13}$$

for $m = 1, 2$.

In the next section, model (10) is compared with Monte Carlo simulations and with the deterministic SIS system (1).

3. Computational Simulations

Several computations were made to see how well model (10) compares with Monte Carlo calculations and with the deterministic model. In addition, computations were carried out to determine if the jump-diffusion model could predict a rapid global expansion in an infection if the transmission rates undergo a step increase or if the recovery rates undergo a step decrease at some time. The parameters in the model were set equal to the values given in Table 1 for two different sets of calculations. Unless explicitly specified in the table, the parameter values are constant for $t \geq 0$. In addition, $\Delta t = \frac{1}{15}$ in the calculations as smaller values of Δt produced only small changes in the results. To better understand the values given, recall that $\beta_{k,j} = \beta_R/d_{jk}$ is the transmission rate for the spread of the disease between patches in regions k and j where d_{jk} is the (great-circle) distance in kilometers between the two regions. Recall that the latitudes and longitudes of the 30 regions are given in the second section and the average distance between the regions is 9999 km. Recall also that $\alpha_j = \beta_P/d_{avg}$ is the transmission rate for the infection between patches in region j and γ_j is the recovery rate. Also, the rate of spread of the infection between regions is related to the value of the ratio β_R/γ_j and the rate of spread between patches is related to the value of the ratio β_P/γ_j. Consider again the values in Table 1 for the two sets of calculations. In the first set of calculations, the region-to-region transmission rate undergoes a step increase at time $t = 10$. (Although the time units are not specified here, time could be regarded, for example, having units of years.) Initially, $S_j(0) = N = 25$ for $j \neq 1$ and $S_1(0) = 0$. That is, the infection begins in region 1, where the recovery rate is lowest, and $I_1(0) = 25$ for this region. (Region 1 has latitude 60° North and longitude 0°. Although the infection is assumed to originate in region 1 in the present investigation, similar calculational results would be expected if the infection

is assumed to originate in another region.) In the second set of calculations, the transmission rates remain fixed. However, the recovery rates, for all regions other than region 1, undergo a step decrease at time $t = 10$. This change also produces a rapid global expansion in the disease.

In Figures 1-3, the results of the calculations are displayed for calculational set 1 in which the transmission rates have a step increase at time $t = 10$. In each figure, the total number of infected patches is given versus time. Notice that the total number of infected patches is bounded above by $NM = 750$. Two of the curves in each figure are for individual sample paths of the spread of the infection. The smooth curve in each figure is the average of 400 independent sample paths. The average number of infected patches estimated by the jump-diffusion model given in Figure 2 agree quite well with those estimated by the Monte Carlo calculations. In comparison, the continuous-trajectory stochastic model (9), with the calculated results given in Figure 3, overestimates the rate that the disease progresses. For example, at time $t = 18$, the Monte Carlo, jump-diffusion, and continuous-trajectory stochastic models estimate the average number of infected patches to be 393.5, 378.6, and 520.7, respectively.

Parameter	Value (Set 1)	Value (Set 2)
β_P	3.25	3.25
$\beta_R,\ t < 10$	0.1625	3.25
$\beta_R,\ t \geq 10$	3.25	3.25
d_{avg}	130.0	130.0
$\gamma_j,\ j \neq 1,\ t < 10$	0.25	2.0
$\gamma_j,\ j \neq 1,\ t \geq 10$	0.25	0.25
γ_1	0.125	0.125
$I_j(0),\ j \neq 1$	0	0
$I_1(0)$	25	25
N	25	25
M	30	30

TABLE 1. The values of the parameters used in the calculations for set 1 and set 2.

In Figure 4, the deterministic model (1) is compared with the average number of infected patches predicted by the Monte Carlo calculations, the continuous-trajectory stochastic model, and the jump-diffusion model. The deterministic model and the continuous-trajectory stochastic model clearly overestimate the rate of spread of the infection. Based on Figures 1-4, the jump-diffusion equations (10) accurately estimate, in comparison with Monte Carlo simulations, the spread of the infection. A least squares fit to the total number of infected patches for time $t \geq 10$ is given by

$$\hat{I}(t) = \sum_{j=1}^{M} I_j(t) \approx 535 \exp(0.512t)/(\exp(0.512t) + 3350),$$

where $\hat{I}(t)$ is the total number of patches infected. This relation indicates that, after a step increase in the transmission rates, the infection spreads initially at an exponential rate of increase and approaches an equilibrium value of approximately 535. For comparison, the deterministic model predicts an equilibrium value of

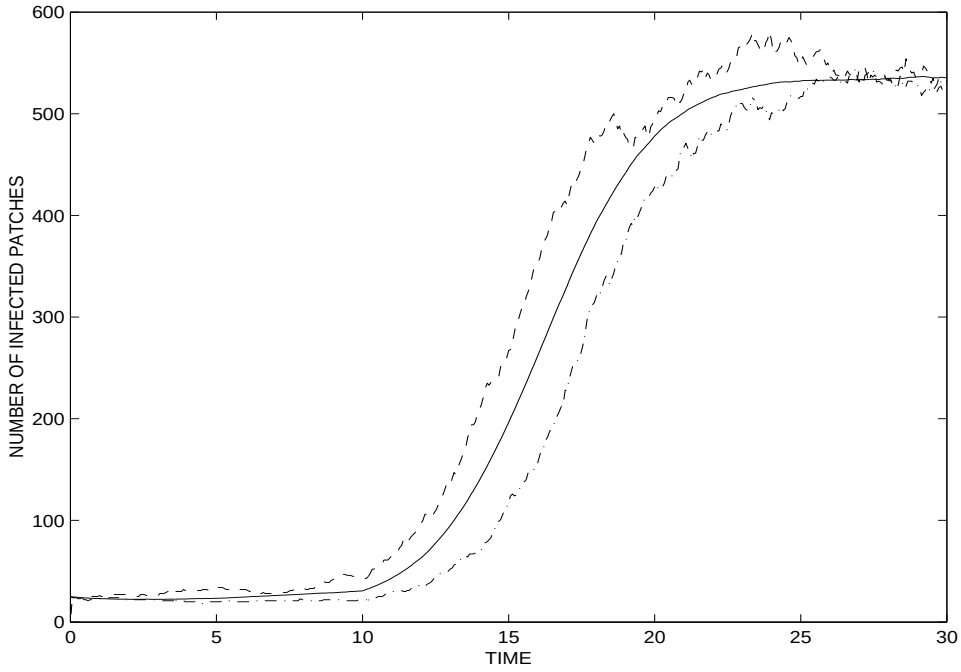

FIGURE 1. Number of infected patches as predicted by Monte Carlo simulations when a step increase in transmission rates is introduced at $t = 10$. The smooth curve is the average of 400 trajectories.

about 546 infected patches. (Recall that the total number of infected patches cannot exceed $NM = 750$.)

Notice that the susceptibility/resistance of the amphibians to the disease is related to the values of β_P and β_R as well as to the values of γ_j. In other words, if the susceptibilty of the amphibians to the disease decreases, then the values of β_P and β_R may increase as the likelihood of contracting the disease after exposure increases with reduced resistance. However, the values of β_P and β_R are also related to the transmission rate of the disease. Hence, global environmental changes, such as increased ultraviolet light, increased pollution levels, or global warming, may affect the values of β_P and β_R. In addition, the increased ability for a viable infection to spread from region to region due to increasingly rapid means of transportation affects the values of β_P and β_R. However, the recovery rates γ_j for $1 \leq j \leq M$ are independent of the transmission rates. The recovery rate can also be related to the susceptibility/resistance of the infection with the values of γ_j increasing with resistivity to the infection. A large value of γ_j would signify rapid recovery and hence, γ_j is kind of measure of resistance to the infection. Therefore, in the second set of calculations, the transmission rates β_P and β_R were held constant for time $t \geq 0$. However, the recovery rate γ_j, for regions $2 \leq j \leq M$, underwent a step decrease at $t = 10$ to model a change in environmental conditions that affected the ability of the amphibians to recover from the infection. The total number of infected patches as estimated by the jump-diffusion model are given in Figure 5. The smooth curve is again the average of 400 different sample paths whereas the two rough curves are for two individual sample paths for comparison. The interesting

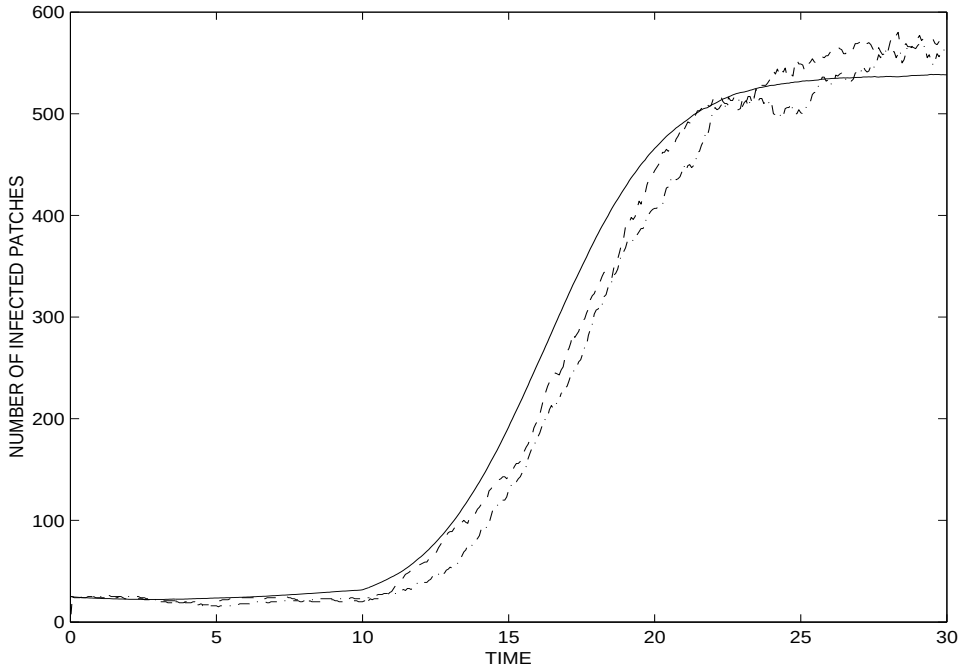

FIGURE 2. Number of infected patches as predicted by the jump-diffusion model (10) when a step increase in transmission rates is introduced at $t = 10$. The smooth curve is the average of 400 trajectories.

feature of Figure (5) is the close similarity of its curves with the curves given in Figures 1 and 2. Indeed, the values of the parameters for this set of calculations were adjusted so that the number of infected patches after a step decrease in the recovery rates would be similar to the number of infected patches following a step increase in the transmission rates. The conclusion reached, based on the results of the two sets of calculations, is that given only information regarding population size decreases, the model would not be able to accurately differentiate between amphibian population declines due to changes in transmission rates, changes in recovery rates, or to changes in a combination of factors.

4. Discussion

A jump-diffusion stochastic model was developed in the present investigation for modelling the global spread of an amphibian infection. The jump-diffusion model is a stochastic version of a deterministic SIS model for the spread of the disease. In the present investigation, the disease spread was modelled in terms of 30 regions widely dispersed on the earth's surface with 25 patches in each region. Each patch was assumed to consist of one or more populations of amphibians equally susceptible to the disease. It was assumed that the infection was initially present only in one region while the other regions were free of the disease. At time $t = 10$, it was assumed that there was a step change in the transmission rates or a step change in the recovery rates of the infection. The deterministic SIS model overestimated the rate of spread of the disease in comparison with Monte Carlo simulations while the jump-diffusion model and the Monte Carlo simulations were in good agreement.

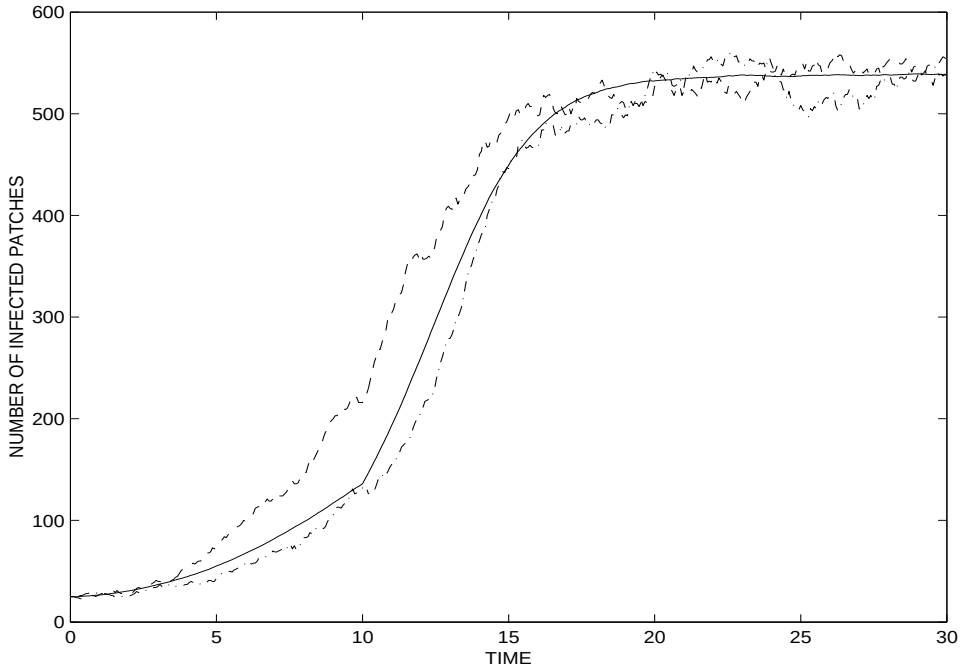

FIGURE 3. Number of infected patches as predicted by the continuous-trajectory stochastic model (9) when a step increase in transmission rates is introduced at $t = 10$. The smooth curve is the average of 400 trajectories.

The calculational results indicate that the disease can spread globally at an exponential rate following a step change in the transmission or recovery rates. Unfortunately, given the spread of a particular disease, the mathematical model, as presently formulated, cannot differentiate between the two effects. That is, although the mathematical model predicts that an infection can rapidly spread through a system of disparate regions, the model cannot be used to determine whether the spread of the infection is due to changes in the transmission rates of the infection, to changes in the recovery rates of the infection, or to some combination of changes in the transmission and recovery rates.

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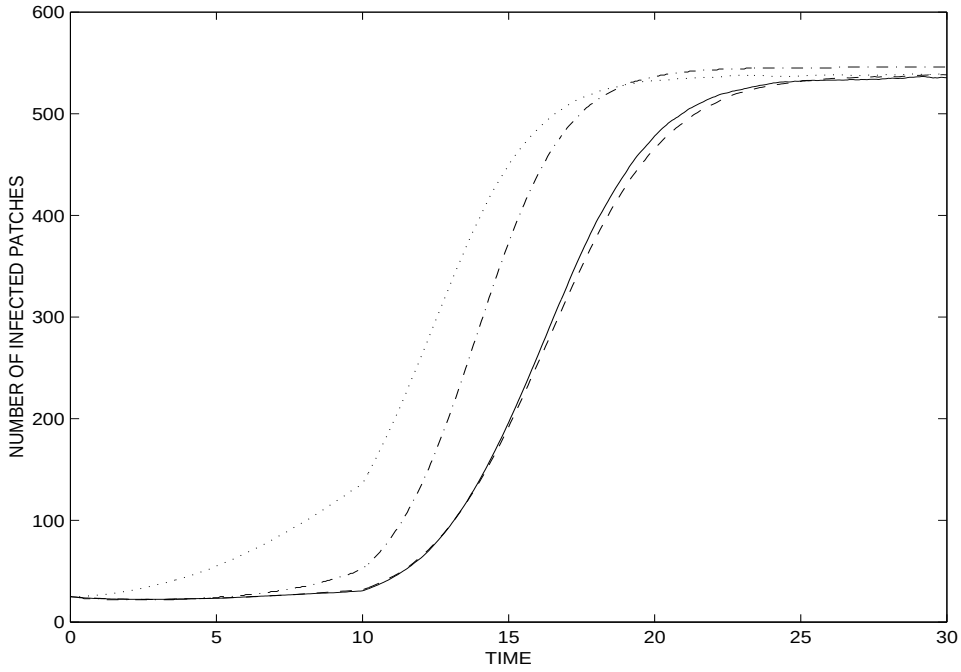

FIGURE 4. Average number of infected patches as predicted by the the continuous-trajectory stochastic model (dots), the deterministic model (dash-dot), Monte Carlo simulations (solid), and the jump-diffusion model (dash) when a step increase in transmission rates is introduced at $t = 10$.

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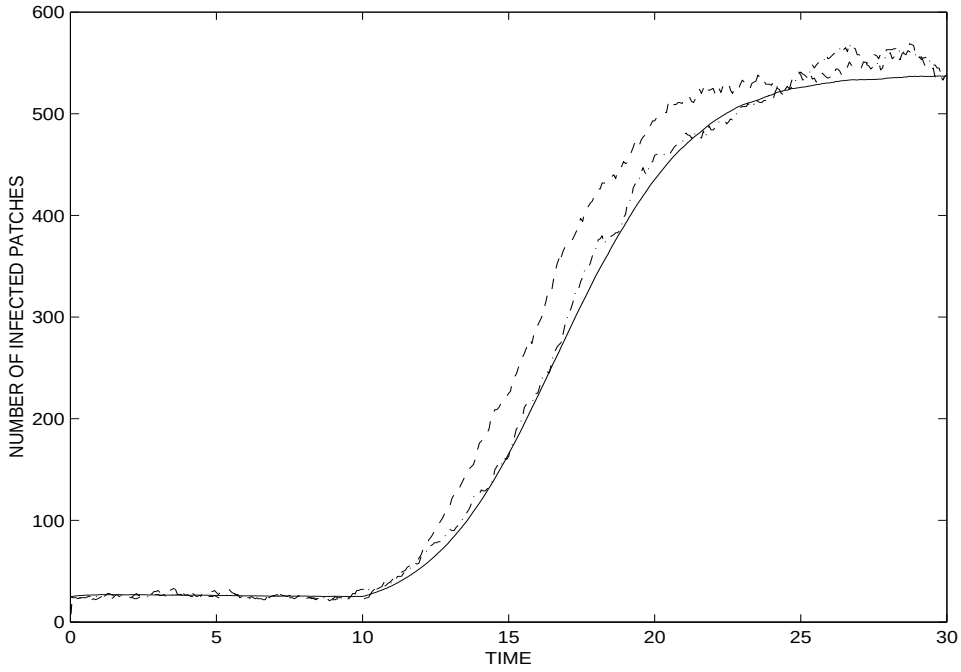

FIGURE 5. Number of infected patches as predicted by the jump-diffusion model (10) when a step decrease in recovery rates is introduced at $t = 10$. The smooth curve is the average of 400 trajectories.

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