A Discrete SEIRS Model for Pandemic Periodic Infectious Diseases

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Abstract

A deterministic SEIRS epidemiological model that captures the essential properties of pandemic recurrent diseases is developed here, in terms of a system of delay-difference equations. A non-linear system of difference equations is proposed, based on the assumptions of exponential incidence and of constant periods of latency, infectiousness and immunity. The model is able to reproduce observed data behaviors such as sustained oscillations and seasonality, typical of many childhood epidemics. We explore the stability of the system in parameter space and show its robustness and versatility to be applied to many infectious diseases. This new model is able to exhibit a wider range of dynamics than previous epidemiological models which make it ideal for a wide use in several infectious diseases. In order to illustrate this, we use this model in combination with a deliberately simplified spatially distributed population, and verify that it captures the essential features of communicable disease spread, such as fade-outs, and a great variability in epidemic sizes. We conclude that realistic models of geographical spread of infections could be improved by describing separately the properties of the diseases, from the social and human mobility aspects of the process. This model helps to fill the theoretical gap that exists between the classical compartmental models and the true stochastic nature of the spread of pandemics.

Keywords: epidemic model, pandemics, delay-difference equations, non-linear incidence, stability analysis, oscillatory and quasi-periodic dynamics

1. Introduction

Epidemiological mathematical models are useful in proposing and testing theories, and in comparing, planning, implementing and evaluating various detection, prevention, therapy, and control programs.

An astounding amount of epidemiological models have been elaborated and applied to infectious diseases [3, 44, 27]. There are models that consider passive immunity, gradual loss of vaccine and disease-acquired immunity, age structure, stages of infection, vertical transmission, disease vectors, vaccination, quarantine, chemotherapy (for a review see [3, 44, 27]), and more recently spatio-temporal spread over a country or all over the world [39, 5, 48, 19, 14, 15, 29, 20, 13, 43]. However, few of the models are able to reproduce some essential aspects of childhood epidemics, for instance, sustained periodic oscillations, and seasonality, common to many endemic diseases, as measles, rotavirus, rubella, whooping cough, influenza and many others.
The typical model used for epidemics is the continuous SIR model, in which a population with $N$ individuals is divided into three different compartments: susceptibles, infectious, and recovered or immune [3]. This model has been used for describing public health interventions during the 1918 influenza pandemic [10, 24]. However, transmission and recovery are intrinsically stochastic processes, and the deterministic SIR model does not account for fluctuations and sustained oscillations. Such models have also been extended to model the geographical spread of infectious diseases by considering reaction diffusion kinetics [44] and/or by including stochasticity in a closely interconnected and interdependent world [39, 6, 14, 15, 29]. During the last century, most epidemiological models were essentially deterministic (for a review see [3, 44]). Notable exceptions are the seminal works of Bartlett [7] and Bailey [8]. Stochastic effects were largely ignored because chance events were considered to be of relevance only for large populations [41, 33] and because it was assumed that they merely add a blurring effect on the evolution of a deterministic epidemic system. Furthermore, in isolation any stochastic population model will eventually become extinct. However, in nonlinear systems with dynamical noise, the latter might act as a driving term in the equations of motion and could drastically modify the deterministic dynamics. Currently, stochastic simulation models of the dynamics of infectious diseases are publicly available [11].

Most epidemiological models make use of the mass action principle, i.e., the assumption that new cases arise in a simple proportion to the product of the number of individuals who are susceptible and the number who are infectious [3, 44]. However, this principle has limited validity and in discrete models this principle leads to non-biological results unless severe restrictions are imposed in the parameters [35]. Exponential incidence in the rate of transmission is more sensible biologically for discrete epidemiological models [35]. Furthermore, the mass action principle intrinsically implies that on contact, both the susceptible and infectious agents can be affected, which is obviously not the case in infectious diseases.

Infectious diseases may be absent over certain periods of time, i.e. there are fade-outs. In this regard, it was suggested [38, 50] that seasonal forcing in transmission rates might be the most likely explanation of fade-outs even in large communities. The simplest model for homogeneous mixing populations is considered to be the seasonally forced SEIR model, in which there is a new compartment of exposed but not yet infectious people. However, it has been reported that in continuous stochastic SEIR models, with or without seasonal contact rates and age structure, it is not possible to reproduce sustained periodic oscillations, fade-outs, and power-law distributions observed for measles incidence [45, 46]. This is also applied to SEIRS models, in which the recovered people might become susceptible again. Forest-fire models have been used to generate fade-outs and sustained oscillations without the need of seasonal-forcing terms [30, 34]. Such periodicities may be driven by extrinsic factors, as reflected in periodic transmission rates, e.g. seasonality [4], time delays [25], age structure [49], or non-linearity in incidence rates [37].

In the present work, we have developed a simple epidemiological model that combines the precise ingredients of a discrete model with exponential incidence, vital dynamics, and
constant periods of latency, infectiousness and transient immunity. This model captures the
dominant features of dynamics of communicable diseases.

This paper is organized as follows: In Section 2, we justify the use of exponential
incidence, and in Section 3 we formulate the model in terms of a non-linear system of
difference equations. In Section 4, a detailed sensitivity analysis of the various parameters
of the model is presented. The model exhibits a rich variety of behaviors: regular
oscillations of different period, damped oscillations, quasi-periodicity, and stable regimes.
In Section 5, we illustrate the model in combination with a deliberately simple spatially
distributed population in order to unravel underlying mechanisms of infectious disease
transmission. This analysis reveals that this model can be confidently used to investigate
the development of many infectious diseases and could serve a basic tool to be applied in
more complicated models aiming to explore the fundamental mechanisms of the
geographical spread of epidemics. Finally, in Section 6 we discuss the results and draw
some important conclusions.

2. Exponential incidence in homogeneous and uniformly mixing
populations

Most epidemiological models make use of the mass action principle, which leads to a
transmission rate $P/\Delta t = \beta Y/\Delta t$ (Anderson and May, 1991). However, we have recently
shown that in continuous epidemic models this principle has limited validity [35]. Let us
calculate the incidence function by considering the probability of contracting the viral
disease by contact with infected people. This surely must include the product of the number
of susceptibles and the probability of being infected as a result of contacts with infectious
people.

We assume that the population is large and homogeneous, in the sense that individuals are
not distinguishable. One could be infected by any number of contacts from 1 to $N$, and the
probability of being infected by exactly $k$ contacts is very small. Under these conditions the
probability of being infected in exactly $k$ contacts follows a Poisson distribution

$$P_k(\lambda) = \frac{\lambda^k}{k!} e^{-\lambda},$$

(2.1)

where $\lambda$ should be proportional to the probability of encountering an infectious individual.
This number can be associated to the strength of the contact with an infectious agent, and
should take into account the virulence of the disease. Therefore this factor is a characteristic
property of the viral disease.

Obviously a susceptible can become infected not only with exactly $k$ contacts, since one is
likely to get the infection with any number of contacts. Therefore the total probability of
being infected is:
The most probable (and also the mean) value of $\lambda$, is $Np$, where $p$ is the probability of having a single contact with an infectious agent. At a given time $t$ this should be proportional to the number of infectious people $Y$, that is, one could write $\lambda = \beta Y$, where $\beta$ is related to the degree of transmission of the particular viral disease. Therefore, the incidence function can be written as

$$G_t = X_t(1-e^{-\beta Y}), (2.3)$$

It is evident that when the exponent is small, Eq. (2.3) can be expanded as a Taylor series, whose leading term gives exactly the law of mass action $G = \beta XY$. It is worth pointing out that the factor $\beta$ has a meaning that differs from the usual connotation in classic SIR models, since in Eq. (2.3) it can have any positive value.

3. SEIR(S) epidemiological model

Models for viral and bacterial infections commonly divide the population into categories or compartments – those who have not yet experienced infection (susceptibles, $X$); infected but not yet infectious (exposed, $E$); infectious ($Y$); recovered and thereby immune ($Z$) – with various rate processes determining the flows into and out of each category. We consider direct transmission of an infectious disease that after recovery it confers immunity, from transient to long-life.

A novel feature of the present model is that the probability that a susceptible do become infective is assumed to be of the form $P = (1-e^{-\beta Y})$ (see Section 2), where the dimensionless constant $\beta$ represents the transmission parameter.

It is also assumed that the periods of latency, infectiousness and immunity are constants denoted by $\varepsilon, \sigma$ and $\omega$, respectively, which can be made dimensionless by using a constant timescale $\tau$. This means that the probability of sojourn in one of these compartments $\tau$ units of time after entrance is one. Demography is incorporated assuming an exponential functional form with a constant mortality rate $\mu = 1/L$, where $L$ is the life expectancy and all newborns are considered to be susceptible. For simplicity the total population $N = X + Y + E + Z$, is considered constant, although the model can deal with the general case in which $N$ is not conserved. We suppose that the individual infection is a consequence of daily contacts, so that, the time step of our discrete model is a basic cycle.
of the population life history, a quantum of time, which we take as one day. Considering all
the foregoing assumptions, we can now express the flow rates per day of all variables by
the following system of dimensionless delay-difference equations:

\[
\begin{align*}
X_{t+1} &= q[(X_t - G_t + S q' G_{t-1-b})] + \mu N \\
E_{t+1} &= q[(E_t + G_t - q' G_{t-\varepsilon})] \\
Y_{t+1} &= q[Y_t + q' G_{t-1-\varepsilon} - q' G_{t-1-\omega}] \\
Z_{t+1} &= q[Z_t + q' G_{t-1-\omega} - q' G_{t-1-\alpha}]
\end{align*}
\] (3.1)

where \( q = (1 - \mu), a = (\varepsilon + \sigma), b = (\varepsilon + \sigma + \omega) \) and the incidence function
is \( G_a = X_a (1 - e^{-\beta a}) \). We have introduced a resilience parameter \( S \) that takes into account
the fact that only a fraction of recovered people may become susceptible again, because
timely acquired immunity and/or changes in social behavior and/or mutations of the infectious
agents. Note that if this parameter is different from one, the total population is no longer
constant.

The solution of the system (3.1) can be found, provided that the following nonnegative
initial conditions are given: \( t \in [-\omega, 0] \) for \( X, \) and \( Z, \) \( t \in [-\varepsilon, 0] \) for \( E, \) and \( t \in [-\sigma, 0] \)
for \( Y. \) The initial values \( E_0, Y_0 \) and \( Z_0 \) have to satisfy the following equations:

\[
\begin{align*}
E_0 &= \sum_{t=1}^{\varepsilon} (1 - \mu)^t G_{t-\varepsilon} \\
Y_0 &= \sum_{t=1}^{\sigma} (1 - \mu)^{t+\varepsilon} G_{t-\varepsilon-\omega} \\
Z_0 &= \sum_{t=1}^{\varepsilon} (1 - \mu)^{t+\sigma+\varepsilon} G_{t-\varepsilon-\omega-\alpha}
\end{align*}
\] (3.2)

It can easily be shown (see [35], Lemma 1) that the following sum rules are satisfied at all
times for \( S = 1:\)

\[
\begin{align*}
E_t &= \sum_{t=1}^{\varepsilon} (1 - \mu)^t G_{t-\varepsilon} \\
Y_t &= \sum_{t=1}^{\sigma} (1 - \mu)^{t+\varepsilon} G_{t-\varepsilon-\omega} \\
Z_t &= \sum_{t=1}^{\varepsilon} (1 - \mu)^{t+\sigma+\varepsilon} G_{t-\varepsilon-\omega-\alpha}
\end{align*}
\] (3.3)
In this case one can use directly these latter equations with the conservation condition for $N$, or numerically integrate Eqs. (3.1), with initial conditions given by Eqs.(3.2). Therefore, the dynamics of other infectious diseases can be modeled with the same system of Eqs. (3.1) or (3.3). We shall give specific values of these parameters when applied to the specific case of rotavirus, taken from published studies [31, 12, 40].

4. SEIR(S) dynamics: Analysis and stability

The set of non-linear difference equations with memory presented in the above section displays a rich variety of behaviors as a consequence of the symmetry-breaking non-linearities brought in by the memory effects. The model presents various fixed points, of which the point $[X,Y,E,Z]=[1,0,0,0]$ is stable for small values of $\beta$. Then, there is a change of behaviour from a regime in which this fixed point is stable\(^1\) to a regime in which it becomes unstable. The bifurcation parameter that unleashes a series of instabilities is known in the literature as the basic reproductive number $R_0$ [3, 44]. For a discrete SIS model it has been proved that $R_0 = \beta\sigma N$ [35]. In the case of our SEIRS model when $S=1$, each infection produces $R_0$ new infections in its lifetime of duration, $a = \epsilon + \sigma$. By definition, $R_0(X_t / N) = 1$. Hence, in our model this quantity is:

$$R_0 = \beta(1 - \mu)^{\epsilon+\sigma}\sigma X_{t=0}. \quad (4.1)$$

This quantity represents the expected number of infections generated by a single primary infection in a fully susceptible population. The critical value is $R_0 = 1$. For $R_0 < 1$ the disease-free equilibrium point is asymptotically stable and the disease dies out. For $R_0 > 1$ the system shows oscillatory behavior due to the stabilization of a limit cycle. There is also the possibility of no closed paths leading to quasi-periodic behavior. There is a transition region in which one can observe period doubling instabilities. The exact location of the critical point in parameter space depends on the survival parameter $S$. In Figure 1 we show the critical line in the space for four key values of $S$, and we also show the location of the parameters $\beta$ and $\sigma$ used in the application of the model to rotavirus infection. Note that, for example, the SEIRS model ($S=1$) predicts an endemic behavior for rotavirus infection. However, the estimated value of rotavirus is $R_0 = 2.79$, and therefore we can predict that if the value of $S < 0.4$, the disease would fade out. We are assuming that susceptible

\(^1\)Meaning that after the initial perturbation ($Y_0 = \eta$), the system returns to this fixed point. Therefore, in the time history one gets a single peak in the number of infectious people.
individuals are infected, recover and are transiently immune, then become susceptible again, with reduced susceptibility to infection and disease following one or more previous infections. Since $\mu$ is very small (of the order of $10^{-5}$ for a reasonable value of life expectancy of various decades) the parameter $\varepsilon$ has little influence on the bifurcation parameter (see Eq. (4.1)).

**Figure 1.** Critical behaviour of the SEIR(S) model. The solid-black curve is the critical line using $S=1$ (that corresponds to the SEIR model) separating the regions of asymptotical stability (underneath the curve) from the region of existence of a limit cycle (above the curve). This critical line depends on the value of the survival parameter $S$. The dotted -blue curve corresponds to $S = 0.4$; the dashed-red curve is for $S = 0.2$, and the green
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-dashed-dotted curve has value of $S = 0.1$. (The SEIRS dynamics corresponds to $S < 1$). The location of the point that corresponds to the parameters used in the application to the influenza pandemics is shown as a red rhombus lying in close to the dotted-blue curve $S = 0.4$. Observe that this point is located above but very near the critical line for $S = 1$, and that by lowering the value of $S$ one can cross to the stable region.

In Figure 2 we show an example calculation of the time history of susceptible, infectious and recovered individuals when the system is in the region of regular oscillations. Note the sustained regular oscillations of the infectious individuals alternating with the oscillatory behavior of both susceptible and recovered individuals. For definitiveness, the values of the parameters used in Figure 2 are appropriate for rotavirus epidemics, particularly, the time periods are $\varepsilon = 3$ days, $\sigma = 4$ days [31, 12, 40] and $\omega = 365$ days (adjusted to give an average of one outbreak per year).

Now, let us analyze the behavior of the model in regions of parameter space near the chosen values. We start by looking at the effect of varying the transmission parameter $\beta$ while maintaining all the other parameters fixed. In Figure 3(a) we show the time history of the number of infectious people as a function of $\beta$. Notice that in the region where $R_0 > 1$ there are regular oscillations whose period varies smoothly with the transmission parameter. The lower panel (Fig. 3(b)) shows the calculation using a small value for the survival parameter $(0.1)$ and the oscillations are very much damped. This is to be expected, since the population of susceptibles entering the dynamics becomes very small as time grows.

In order to show the wealth of behavior of this model and its versatility in its applications to different viral diseases we examine positions in parameter space far from the chosen values. In Figure 4 we show the phase portraits of the model under four different conditions. Observe that in addition to the regular (Fig. 4(a) or damped oscillations (Fig. 4(c)) one could have a series of double period bifurcations (Fig. 4(b)) and a seemingly disordered map (Fig. 4(d)).

In Figure 5 we show the temporal behavior of the model as the latency period $\varepsilon$ varies from 0 to very high values. It is interesting to note that if $\varepsilon$ is smaller than the infectious period, regular oscillations appear. For intermediate values oscillations may be present or not, but for large values there is the possibility of initial small oscillations (with non-zero minima) followed by a sudden burst of epidemic activity. This is an interesting prediction of the model.

The effect of varying the other time parameters is an almost linear variation of the period of the oscillations. For large values of $\sigma$ and $\omega$ the activity is lost, and the number of infected people either dies off or is zero.
Figure 2. Example of regular oscillations obtained with the SEIR model. Population of susceptible ($X$) (red curve), infectious ($Y$) (blue curve), and recovered ($Z$) (black curve) individuals as a function of time. The parameter values used in this calculation are: transmission parameter $\beta = 0.7$, latent period $\varepsilon = 3$, infectious period $\sigma = 4$, immunity period $\omega = 365$, mortality rate $\mu = 1/(365 \times 70)$, and the survival parameter $S = 1$. The population has been conveniently normalised to $N = 1$. The initial conditions were $X_0 = 1$, $Y_0 = \eta = 0.0001$, $E_0 = Z_0 = 0$. 
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(a)
Figure 3. Time history of the population of infectious people as the transmission parameter $\beta$ varies. (a) Regular oscillations with varying period for large values of $\beta$. This calculation corresponds to $S = 1$. The lower panel shows the same calculation using $S = 0.1$, (see Figure 3b). Observe that in this case only a single peak appears before the bifurcation and a very small second oscillation, at around $t = 125$, is visible after the bifurcation has taken place.
Figure 4. Phase portraits of the model for different points in parameter space (a) Interior limit cycle. (b) Exterior limit cycle. (c) Poincaré cycle. (d) Seemingly chaotic trajectory. The parameters used are indicated in each figure and the number of days in the calculations was 45,000. The values assigned to the parameters are as follows: Figure 4(a): $\beta = 1.92; \epsilon = 5; \sigma = 8; \omega = 90$; Figure 4(b): $\beta = 1.92; \epsilon = 250; \sigma = 8; \omega = 90$; Figure 4(c): $\beta = 0.18; \epsilon = 5; \sigma = 8; \omega = 90$; Figure 4(d) $\beta = 1.92; \epsilon = 1; \sigma = 1; \omega = 20$. 
Figure 5. Effect of varying the latency parameter $\varepsilon$. Observe the increase on the period of the oscillations as the parameter grows and also the various changes of behaviour of $Y_i$.

5. How to model a pandemic with the SEIRS model

Herein, we hypothesized that the specific epidemiological details that distinguish different strains become irrelevant and the dynamics should be dominated by nearest-neighbor spread in some social network. The time series of infectious diseases are nonlinear process operating at multiple scales and far from equilibrium; bursts of epidemic activity of varying sizes in the number of infected individuals are followed by longer or shorter periods of quiescence (fade-outs) (e.g. [32]).

In order to model a pandemic, we consider essential to add information about spatial spread of the disease. We define a two-dimensional grid of cells $(i, j)$, in which we put an independent SEIR(S) dynamical system in each one of them. The lattice-based model used
in this work consists of a two-dimensional grid of size 5x5. Since people tend to move randomly between cells, noise has to be considered, and in order to simulate this we introduce a standard Monte-Carlo procedure for communication between cells. In this spirit, an infection event between cells is admitted if a random number \( p \in [0,1] \) is smaller than a mobility parameter \( v \in [0,1] \). That will have the effect of varying initial conditions of the model in each cell dynamically.

In Figure 6(a), a sample of 1500 consecutive points of the time-series obtained with our SEIR(S) model assuming only one serotype is displayed. Note that the model is able to reproduce certain variability in the heights of the epidemic outbursts, which are interrupted by variable periods of quiescence. This great variation in the heights and the presence of fade-outs cannot be reproduced by a simple seasonally forced model [46, 47]. Notice that in the simulation there are no strict fade-outs, as in some observed data. This might be due to the fact that not all cases are reported and detected in the data. In general, only primary and secondary infections result in severe cases of infection, which is usually reported. Subsequent infections are usually asymptomatic or present mild symptoms. Thus they are not notified.

Introducing randomness in the SEIRS model alone leads to similar random outbreaks, but the amplitude decays rapidly to extinction.

A phase portrait derived from the simulated time series of the model (Figure 6(b)). Note that the model is able to mimic the cyclic dynamics of several infectious diseases and that the phase portrait displays the frequent visits to fade-outs.
Figure 6. Modeling a pandemic. Figure 6(a) Monthly incidence of infection with the SEIRS model; Figure 6(b) Phase portrait from the simulated time series. The values assigned to the parameters are as follows: $\beta=0.7$, $\epsilon=3$, $\sigma=4$, $\omega=365$, $Y_0=\eta=0.0001$, $v=1.5\times10^{-5}$. 
6. Discussion

In this work we present a new SEIR(S) model that, when combined with spatial spread, is able to mimic the dynamics of actual infectious diseases. We illustrate its potential applicability by modeling a simple pandemic. The deterministic SEIR(S) model captures the biological properties of an infectious illness which do not change during the course of an epidemic and the spread occurs in a stochastic manner due to nearest neighbor social interactions. In order to reproduce the variability of the time-dependent behavior of the actual incidence of various infectious diseases, we may be forced to complement the model with a mechanism of spatial propagation based on a mobility parameter. We conclude that in actual data the contributions from the spatial spread between cities is as important as the properties of the disease.

An early work on the spatially heterogeneous population arbitrarily subdivided into \( n \) groups, with one transmission rate among individuals within any one group, and another, lower transmission rate between groups was developed in 1984 by May [42]. This model has been used as a basis for modeling the geographical spread of infectious diseases like influenza [5, 14, 15, 43]. There is a gap in the literature about how one can couple a discrete compartmental model with spatial spread. This work aims to fill in part, this gap.

A SEIRS model that is able to reproduce the observed epidemiological patterns of age-prevalence of diarrheal episodes associated to *Escherichia coli* and rotavirus was developed almost two decades ago [31]. However, this compartmental model was unable to reproduce both the observed self-similar scaling and the time-dependent behavior of rotavirus epidemics. In contrast, forest-fire models have been used to model the spreading of rotavirus [34] and measles [46, 47] using only one or two parameters.

The SEIRS model proposed here differs from other models with delays because it is formulated with a system of delay-difference equations. SI, SIS and SIR epidemic models in discrete time (difference equations) have been analyzed [3, 1, 2]. Complex models like discrete time SEIRS are complicated to solve analytically. However, nowadays personal computers are powerful enough to make extensive numerical exploration of this kind of models with very simple programs.

Periodic solutions have been found in different classes of epidemic models like the ones that incorporate periodic parameters [4, 16, 23]. Besides this extrinsic forcing other mechanisms have demonstrated to lead to an oscillatory behavior in an autonomous form [26, 22, 51].

It has been shown that epidemic models for infectious diseases with temporary immunity that incorporate a time delay in the recovered class can have periodic solutions. For example, Hoppensteadt and Waltman[28] found numerically periodic solutions of a SEIRS epidemic model with three delays. Using bifurcation techniques, Green [21] observed that periodic solutions can exist for some parameter values of a SIRS model with delays in the transfer of infectives and recovered. Hethcote et al. [25], found periodic solutions of a SIRS model where the recovered class was divided into a chain of \( n \) subclasses, causing a delay in this class. A SIRS model with time delays in the latent and immune class and variable
population size and that generates periodic solutions was analyzed by Cooke and van den Driessche [17]. Most epidemics models with delays, like those mentioned above, have been formulated by systems of integro-differential equations. The periodic solutions of these kind of models have been found by either numerical or Hopf bifurcation techniques.

In a population of constant size, births are continually taking place. In several infectious diseases, the immigration among cities involves the emergence or re-emergence of a particular strain for which the community may be at least partially susceptible. In contrast to infections like measles in which a single infection produces life-long immunity (a SEIR model), in the case of rotavirus repeated attacks of acute gastroenteritis is the rule (SEIRS model), particularly in developing countries. It is clear that a single attack confers no persistent and general immunity, and yet it is equally certain that resistance increases with age, i.e. reinfections are more sporadic each time. Immunity is gradually acquired and takes few years to become pronounced. Resistance to rotavirus infection seems to develop very early in life, since most diarrheal cases are confined to the first 2–3 years of life [9].

Predicting the spread of an epidemic is difficult owing to the complexity of modern human societies. Human mobility patterns affect the spatiotemporal dynamics of an epidemic. The spatial structure of the population has an impact on the spread. The heterogeneity of the population itself can play an important role in the spread of an epidemic [18]. However, an accurate model of the important features of the disease is essential to be used in a model of geographical spread of epidemics, if one wants to make realistic and accurate predictions of pandemics.

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References


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