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STOCHASTIC ANALYSIS OF A DETERMINISTIC AND SEASONALLY FORCED SEI MODEL FOR IMPROVED DISEASE SPREAD SIMULATION

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ABSTRACT

The geographic distribution of different viruses has developed widely, giving rise to an escalating number of cases during the past two decades. The deterministic **S**usceptible, **E**xposed, **I**nfectious (**SEI**) models can demonstrate the spatio-temporal dynamics of the diseases and have been used extensively in modern mathematical and mechano-biological simulations. This article presents a functional technique to model the stochastic effects and seasonal forcing in a reliable manner by satisfying the Lipschitz criteria. We have emphasized that the graphical portrayal can prove to be a powerful tool to demonstrate the stability analysis of the deterministic as well as the stochastic modeling. Emphasis is made on the dynamical effects of the force of infection. Such analysis based on the parametric sweep can prove to be helpful in predicting the disease spread in urban as well as rural areas and should be of interest to mathematical biosciences researchers.

Keywords: *Chaos; Deterministic; Stochastic; Endemic equilibrium; Epidemic disease; Lipschitz criteria; mathematical virus simulations.*

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1. INTRODUCTION

Mathematical modeling plays an important role in improving understanding of the transmission of infections and in the estimation of the potential impact of control programs. Applications

include determining optimal control strategies against new or growing infections, such as swine flu or Ebola, or against HIV, dengue and malaria. The modeling helps in predicting the impact of vaccination strategies against common infections such as measles and rubella. The role of stochasticity and its relationship with nonlinearity are recent issues in the study of the infectious diseases. Childhood diseases like chicken pox, measles or deadly diseases like AIDS have provided important case studies to build up and check mathematical models with practical application to epidemiology [1]. A wide range of temporal behaviors, including annual, biennial, multi-annual and irregular fluctuations have been described by Alonso *et. al.* [2] for time-series data on childhood diseases, including whooping cough and measles. The *method of stochastic modelling* has attracted a lot of attention since it can address the unrevealed factors, responsible for the perturbation in epidemic modelling. Wilkinson [3] provided a detailed analysis of stochastic modelling and emphasized on its applications in computational and system biology. Recently Liu [4] analysed a stochastic SEIR model and established useful results using the continuation theorem. Studies on epidemic models of SEIR or SEIRS type with stochastic effects (i.e. when the deterministic model is seasonally forced) are limited in the literature. Seasonal forcing plays an important role in the dynamics of many infectious diseases. In seasonally forced systems, qualitatively different dynamical patterns can be stable for any specific combination of parametric values. Seasonality plays a vital dynamical role in shaping the population fluctuations of the vector-borne diseases both in humans (such as malaria or dengue) and in animals (such as Cryptococcus neoformans, foot and mouth diseases and Toxocara canis etc). Many animals give birth during a short breeding season. This means that the population dynamics of the host undergoes significant seasonal fluctuations. Seasonality flows periodically in infectious diseases which vary according to the seasonal variations in temperature and rainfall that are occurring every calendar year [5]. Seasonality can alter the spread and persistence of infectious diseases. Although seasonal variation is a well-known phenomenon in the epidemiology of vector-borne diseases [6, 7] in both *temperate* and *tropical* climates but the mechanisms responsible for seasonal disease incidence, and the epidemiological consequences of seasonality, are rarely discussed in the literature. Keeling and Rohani [8] examined the impact of seasonally varying parameters as a forcing mechanism and reported the relevant dynamical consequences. They demonstrated that the temporally forced models better capture the observed pattern of recurrent epidemics in contrast to unforced models, which predict oscillations that are damped toward equilibrium. Although seasonal oscillation of infectious diseases can be easily simulated using simple transmission models, but it is not always possible for certain cases where the seasonal effects are intricate, therefore

seasonality and its effects exhibit a rich area for future research. The recent advances in the study of spatial disease dynamics can demonstrate the network models and their construction; the thresholds; scaling of parameters; heterogeneity and the interaction (long distance) [11]. Variations in population spread within different regions allow global persistence even if the disease dies out locally. Thus stochastic modelling together with the network modelling can help to address the disease spreaders. Maksim [15] has identified several effcient disease spreaders. Their study proved that networks portray a multitude of interactions through which infectious diseases propagate within a population. The circumstances where the best spreaders do not correspond to the most highly connected people were discussed. It was observed that the most efficient spreaders were located within the core of the network as identified by the *k*-shell decomposition analysis (details of the analysis are provided by Carmi *et al.* [16]). To explain the population spread processes, Mollison [18] discussed the use of linear deterministic models. Their main advantages are that their assumptions are relatively transparent and that they are easy to analyze. Generally they give the same velocity as more multifaceted linear stochastic and nonlinear deterministic models. The network modelling is thus another emerging area of research. An important sub-branch of the epidemic modelling is the incubation. Literature on epidemic models mostly assumes that the disease incubation is insignificant such that, once infected, each susceptible individual (in the class *S*) instantaneously becomes infectious (in the class *I*) and later recovers (in the class *R*) with a permanent or temporary attained immunity [14].

Data-driven modelling is another way to run epidemic analysis. The epidemic models can be well synchronized with the real data using various robust numerical and statistical techniques [10]. Recently, Perra and Goncalves [33] presented robust epidemiological models and demonstrated the infectious disease spreading. Their realistic analysis was based on the data driven models, implemented at various geographical locations. Despite the fact that the SEIR model as well as the stochastic SEIR model have been discussed in detail in the literature [8, 3], we aim to propose a stochastic model which will be helpful to synchronize the data with the model, using a parametric sweep. In short, thorough epidemic modelling (by keeping in view the stochasticity, incubation, seasonality and parametric values linked with data) can prove to be a useful tool to control the spread of the deadly diseases. We have thus made an effort to initiate with a *multi-patch* population model for spatial heterogeneity in epidemics. Latter, we have extended the model by taking into account the perturbations through the force of infection. The stochastic effects have been studied by considering the Brownian motion. We have discussed the advantages as well as the limitations of the stochastic modelling.

2. MATHEMATICAL MODEL

A simple model can demonstrate the complex dynamical transitions in epidemics. To start with the basic epidemic analysis, we have considered the following SEIR model:

$$\begin{cases} \dfrac{dS}{dt} = m_D N - (m_D + \lambda_D)S \\ \dfrac{dE}{dt} = \lambda_D S - (m_D + \sigma_D)E \\ \dfrac{dI}{dt} = \sigma_D E - (m_D + g_D)I \end{cases} \qquad (2)$$

and the initial conditions are $S(0) = S_0$, $E(0) = E_0$, $I(0) = I_0$. The model governs the number of susceptible (S), the number of exposed but not yet infectious (E), the number of infectious (I) and recovered (R) individuals. A similar model was reported by Lloyd & May [17]. For computational simplicity, it is assumed that the total population size ($N = S + E + I + R$) in a specific region during a certain period (T) remains fixed (the number of births balances the number of deaths). A schematic diagram is shown in **Figure 1**. The mean of the life expectancy ($1/m_D$), latent period (the time taken to move from class E to I) ($1/\sigma_D$) and the infectious period $1/\gamma$ contribute in the model equations. The net infection rate per susceptible, proportional to the number of infectious I and represented as $\lambda_D = \beta I$, is often called the force of infection [17], [21]. The parameter β is the constant involved in the term $\lambda_D S$ (or more precisely βSI), and measures the rate at which each infective makes contact with the susceptible.

3. NUMERICAL SOLUTION

3.1 The Euler-Maruyama Method

Almost all epidemic diseases exhibits recurrent epidemics however often with biennial cycles such oscillations are sustained in the model if a stochastic formulation of the SEIR equations is used as the random effects prevent the system from settling into the stable endemic equilibrium. In the deterministic framework oscillations can be sustained if the contact rate is allowed to vary seasonally. When the deterministic model is seasonally forced (strongly), a wide range of complex dynamic behaviour is seen including chaos and coexisting cycles of different periods (for details, see [17] and references therein). Let us now consider the stochastic effects which are introduced in the system through the perturbations (in the force of

infection and average latent period of diseases). We can write a general form of the system of first order differential Eqs. (1), as follows:

$$\frac{dX^k}{dt} = f_k(t, X^i) \tag{2a}$$

Or more precisely:

$$dX^k = f_k(t, X^i)dt \tag{2b}$$

where k here stands for the susceptible $k = 1$, exposed $k = 2$ and infectious $k = 3$.

$$\begin{aligned} f_1(t, X^i) &= m_D N - m_D X^1 - \beta X^1 X^3; \\ f_2(t, X^i) &= \beta X^1 X^3 - (m_D + \sigma_D) X^2 \\ f_3(t, X^i) &= \sigma_D X^2 - (m_D + g_D) X^3 \end{aligned} \tag{3}$$

The general form when extended to stochastic form provides the system:

$$dX^k = f_k(t, X^i)dt + g_k(t, X^i)dW \tag{4}$$

It follows that a solution to Eq.(4) can be approximated by using the robust Euler-Maruyama Method. The first step is to discretize the temporal domain into M equal patches of size $h = T/M$, i.e., $t_j = j_h$ and the variables evaluated at that j^{th} instant are $X_j^k = X^k(t_j)$ for $k = 1, 2, 3$. Eq. (4) now takes the integral form:

$$X^k(t_j) = X^k(t_{j-1}) + \int_{t_{j-1}}^{t_j} f_k(s, X^i)ds + \int_{t_{j-1}}^{t_j} g_k(s, X^i)dW(s) \tag{5}$$

This can be further modified to:

$$X_j^k = X_{j-1}^k + f_k(t_{j-1}, X^i{}_{j-1})(t_j - t_{j-1}) + g_k(t_{j-1}, X^i{}_{j-1})((W(t_j) - (W(t_{j-1}))$$

i.e.

$$X_j^k = X_{j-1}^k + f_k(t_{j-1}, X^i{}_{j-1})h + g_k(t_{j-1}, X^i{}_{j-1})((W(t_j) - (W(t_{j-1})) \tag{6}$$

Inspection of eqn. (6) reveals that the Euler-Maruyama scheme converges to the basic Euler's scheme in the absence of stochastic effects i.e., for $g_k \equiv 0$. The stochastic effects involved in the model are numerically addressed by computing the *Brownian paths* which are in turn

implemented to generate $W(t_j) - W(t_{j-1})$. A detailed analysis of the Brownian path generation and convergence is available in [22]. We have solved the numerical scheme (6) using the Matlab interface. The values used under the given conditions are listed in **Table 1**.

3.2 Lipschitz Condition

One important point to be considered while defining $g_k(t,X^i)$ is that the solution to Eqs. (4) (for k = 1, 2, 3) must exist and the stochastic diff erential equation always adopts the same process under equivalent conditions. We now mention the Existence-Uniqueness Theorem [23, 24] which shows that under reasonable modeling conditions stochastic differential Eqs.(4) do indeed satisfy this prerequisite.

Theorem

For the stochastic differential equation:

$$dX^k = f_k(t, X^i)dt + g_k(t, X^i)dW \tag{7}$$

Assume:

1. *Both* $f_k(t,X^i)$ *and* $g_k(t,X^i)$ *(i;k = 1,2,3) are continuous on* $(t,\mathbf{X};\ \mathbf{X} = [X^1,X^2,X^3]) \in [t_0,T] \times R^3$

2. *The coefficient functions* f_k *and* g_k *satisfy a Lipschitz condition:*

$$|f_k(t,X^i) - f_k(t,Y^i)| + |g_k(t,X^i) - g_k(t,Y^i)| \leq K|\mathbf{X} - \mathbf{Y}| \tag{8}$$

3. *The coefficient functions* f_k *and* g_k *satisfy a growth condition in* $\mathbf{X}$ *such that:*

$$|f_k(t,X^i)|^2 + |g_k(t,X_i)|^2 \leq H(1 + (X_1)^2 + (X^2)^2 + (X^3)^2) \tag{9}$$

Then the stochastic differential equation has a *strong solution* on $[t_0, T]$ that is continuous with probability 1. The first definition of a solution of a stochastic differential equation reflects the interpretation that the solution process X at time t is determined by the equation and the exogenous input of the initial condition and the path of the Brownian motion up to time t. Mathematically, this is translated into a measurability condition on Xt or equivalently into the smallest reasonable choice of the filtration to which X should be adapted. The strong solution is a solution that is continuous and has probability 1. It follows that:

$$\max\ (E[\mathbf{X}^2(t)]) < \infty \qquad \forall\ t \in [t_0,T] \tag{10}$$

Consequently for every Wiener process $W(t)$, the strong solutions are pathwise unique.

In the light of these conditions, careful selection of $g_k(t,X^i)$ is made for the stochastic model and the *extended* model for the stochastic analysis is presented below.

3.2.1 Perturbed Model

We now consider the system of stochastic differential equations

$$dX^k = f_k(t,X^i)dt + g_k(t,X^i)dW;\ k = 1, 2, 3 \quad (11)$$

such that:

$$f_1(t,X^i) = m_D N - m_D X^1 - \beta X^1 X^3, \quad f_2(t,X^i) = \beta X^1 X^3 - (m_D + \sigma_D)X^2,$$
$$f_3(t,X^i) = \sigma_D X^2 - (m_D + g_D)X^3, \quad g_1(t,X^i) = m_D X^1 + \beta^* X^1 X^3,$$
$$g_2(t,X^i) = \beta^* X^1 X^3 + (m_D + \sigma_D)X^2; \ g_3(t,X^i) = 0. \quad (12)$$

The main idea is to carefully select of the perturbation terms which will (a) demonstrate the random effects caused by the force of infection and (b) provide a convergent stochastic approximate solution. We have followed a strategy similar to [25] to select the perturbation terms. These equations are solved numerically after satisfying the stability criteria of the discretization scheme. In the next section, we have presented the graphical results to demonstrate the dynamics.

4. RESULTS AND DISCUSSION

The spatial heterogeneity may address many of the deficiencies of the SEIR model. These heterogeneities are included by taking into account the *immigration rate*, where infective individuals enter the system at some constant rate (Olsen *et al*. [26]). This clearly allows the persistence of the disease since if it dies out in one region then the arrival of an infective from elsewhere can trigger another epidemic. Climate is treated as an *independent factor* in the observed expansion of epidemic transmission [29]. Recent approaches seek to combine climate data with projected societal changes, including increased population and economic development in tropical/subtropical domains [30]. A more sophisticated way of introducing spatial effects into the model is to divide the population into p sub-populations of size(s) N_i; $_i = 1, 2,....,p$ and allow infective individuals in one patch to infect susceptible individuals in another. A detailed description of the contact rate variation relative to number of patches is given elsewhere [17]. During this discussion, we have presented the results which demonstrate the dependence of the pandemic and epidemic diseases spread [27, 28] on the population sizes. When the analysis is made on two different populations such that the size is almost doubled, surprising chaotic results are obtained. The relative importance of stochasticity depends on the population size and thus this effect is most visible in simulations with small populations. In **Figure 2** a dynamical analysis relative to the non-stochastic model is presented. The number of infectives when plotted with respect to their size after a period of T and $2T$ revealed multifarious dynamics. For three different values of the force of infections, the rate of infectives

is revolutionized and when the population size is doubled, the dynamics are totally sundry. In all simulations, the numerical solutions converged to a fixed point. More precisely, at $\beta = 0.001$ faster damping can be seen when population size is doubled. Also the damping dynamical behaviour is inversely proportional to β.

In **figure 3**, where the stochastic effects *are effective*, the change in the number of infectives under the effects of (a) force of infection, (b) population size and (c) the stochastic effects are plotted. This figure demonstrates clearly that time series has two main streams of oscillation; smaller and higher for all the plots. It is diffcult to understand the dynamics for immense population size with stronger force of infection i.e. as β increases from 0.001 to 0.003. **Figure 4** presents the deterministic and stochastic analysis of S, E and I relative to time (transient analysis). There are more fluctuations in stochastic model as compared to the deterministic model. At the initial stage of infectious spread there are high oscillations and in the interval 10-20 days there is damping. After passing 20 days, it seems that *the infection dies out* in deterministic model but it still remains in the population which can be seen from the stochastic modelling. **Figure 5** depicts the orbital relation between the population of susceptible, exposed and infectious. There is a twist in the portraits relative to change in size of population. We can see that as the force of infection increases, the orbital span decreases, since the three sub-populations are strongly correlated in that scenario. Precisely, for N_a the infectives population oscillates around $10^{2.45}$ and around $10^{2.75}$ for N_b. **Figure 6** exhibits an interesting behavior for the stochastic version of the *S-E-I* model. When we change the size of the population, there are random distributions and the results are irregular. In the deterministic model, the orbital span is *not overlapping* with the layers while in the stochastic model, evidently the orbital span is jumbled. The orbital span increases in stochastic perturbations as compared to the deterministic model relative to an increase in seasonality. We observe that random effects are *more dominant* when the *population is small* and there is no twist in the phase space portraits when population size is doubled. The solution converges to an equilibrium point as we increase the value of β from 0.001 to 0.003 and the critical points are more precisely defined when β changes from 0.003 to 0.006.

5. CONCLUSIONS

Favourable climate factors are prerequisite to allow the expansion of disease spread observed over the last four decades. Besides that, human factors, including growing global population, urbanization, and socio-economic limitations on control measures contribute to the spread of

many of the epidemic diseases [31]. The basic *SEIR* model is first converted to the *SEI* model and then the most suitable stochastic model is considered, based on two important aspects. These are firstly the *Lipschitz criteria* and secondly the *stability analysis* of the model with graphical analysis and phase space portraits. It is hoped that the proposed analysis and the results will aid mathematical biologists in conducting research in different domains where stochastic modelling is applicable [31-42]. Trends in current human settlement, together with rapidly expanded urban areas, exploding population density, and limited socio-economic resources, suggest that the human factors, in addition to climate factors are important components in understanding current and future risks of disease transmission [1, 32]. Settlement and socio-economic factors together with climatic suitably, globalized travel and trade, suggest that human populations and their collective actions strongly contribute to the pandemic and epidemic diseases spread. The present study has shown that a deterministic model oscillates in phase as compared to a stochastic model. Complex dynamical behaviour has been reported in the seasonally forced spatial model along with the coexistence of perturbed patterns. Chaotic solutions are observed for higher values of seasonal forcing. An important conclusion from the present analysis is that it is necessary to consider not only the natural structure and spread of the population *but also the random effects*. In this discussion we have limited our analysis to *forced perturbations*. The size of population matters for both deterministic and stochastic models. However from this *extended model*, we have demonstrated that it may be essential to consider some important biological factors including heterogeneity, stochasticity and geographic aspects. Future work, it is envisaged, will focus on the stochastic analysis of delayed differential equations, where the delay will be based on the *incubation period*. In this paper we discussed the stochastic analysis using the Euler-Maruyama method. In subsequent investigations, it is feasible to deploy the *s*-stage diagonally implicit stochastic Runge-Kutta methods (where $s \geq 2$) with strong order for strong solutions. Such methods have a large stability region. We will also consider hybrid stochastic Runge-Kutta methods which are the combination of semi-implicit Runge-Kutta methods and implicit Runge-Kutta methods.

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TABLES

Parameter	N	m_D *(per year)*	β *(per year per infective)*	σ_D *(per year)*	γ_D *(per year)*
range	10^6-10^7	0.01-0.04	0.0005-0.01	40-50	60-80

Table 1: Epidemic model parametric values

FIGURES

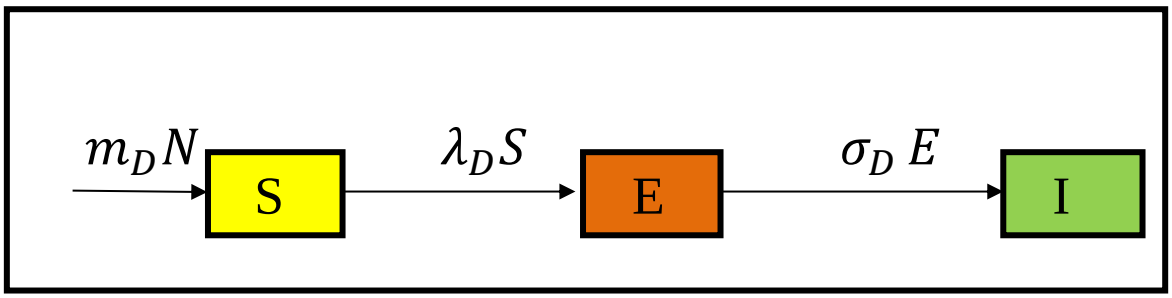

Figure 1: Schematic diagram of disease spread.

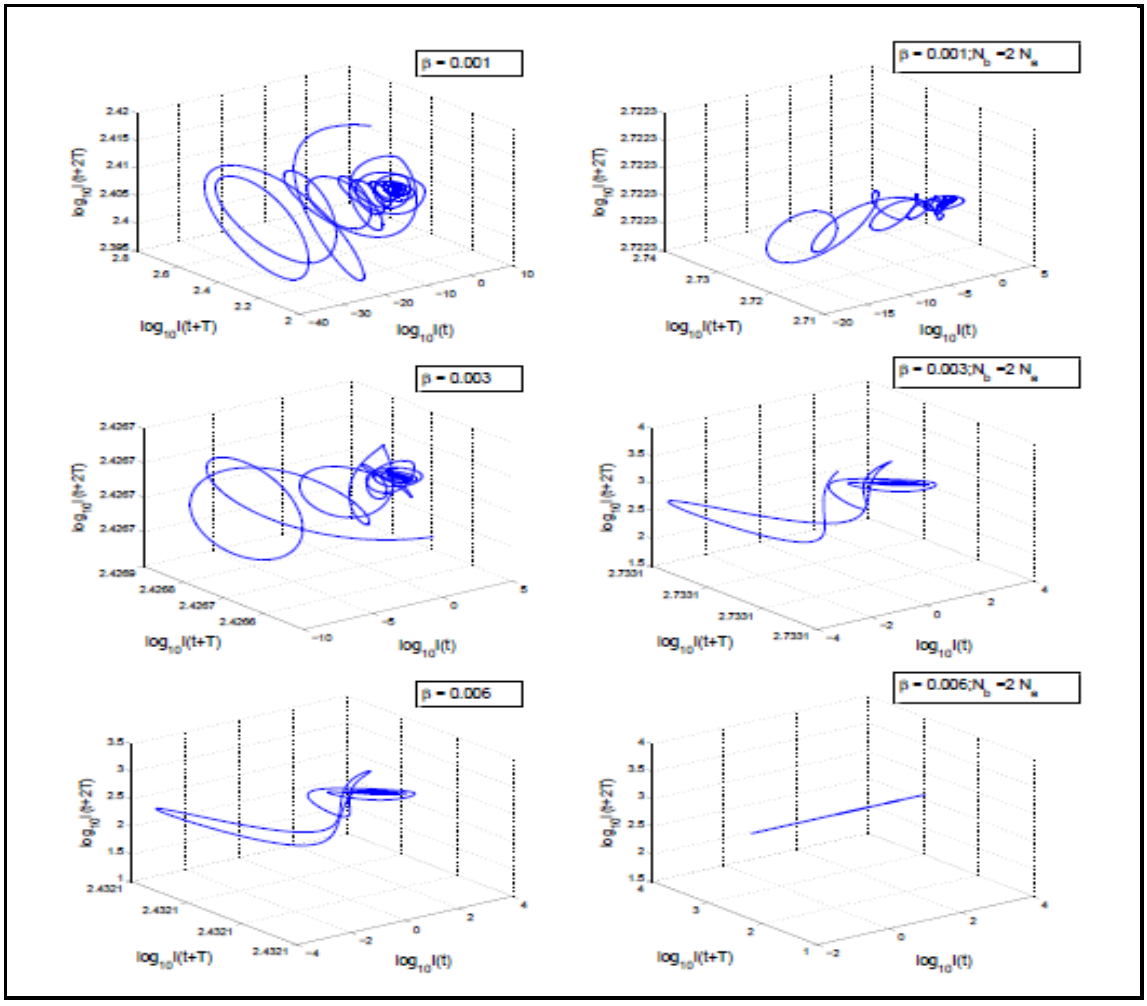

Figure 2: Periodic spread for $N = Na$ (left panel) and (b) $N = N_b$ (right panel).

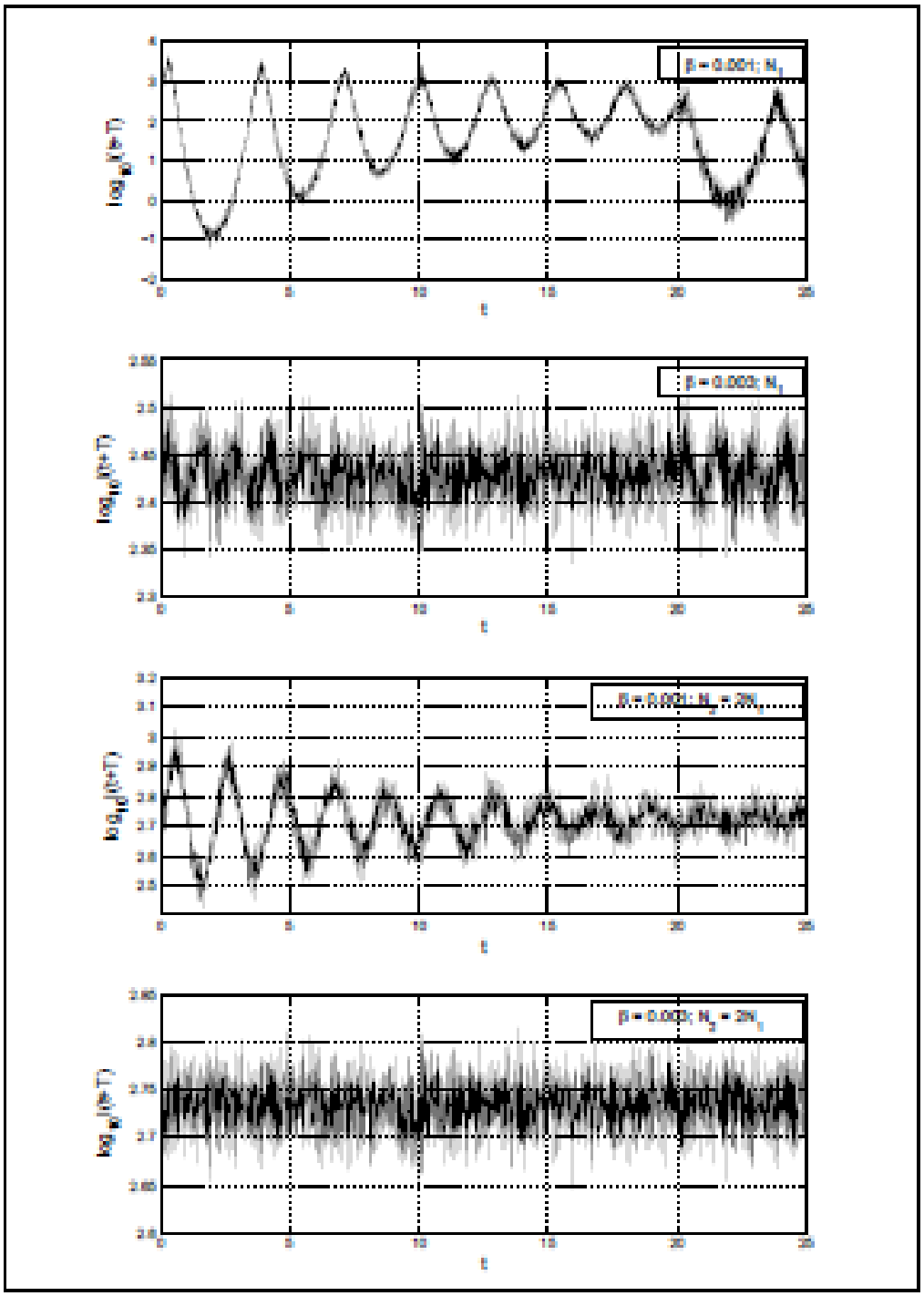

Figure 3: Dynamical analysis of the number of infectives relative to the stochastic effects, force of infection and the size of population.

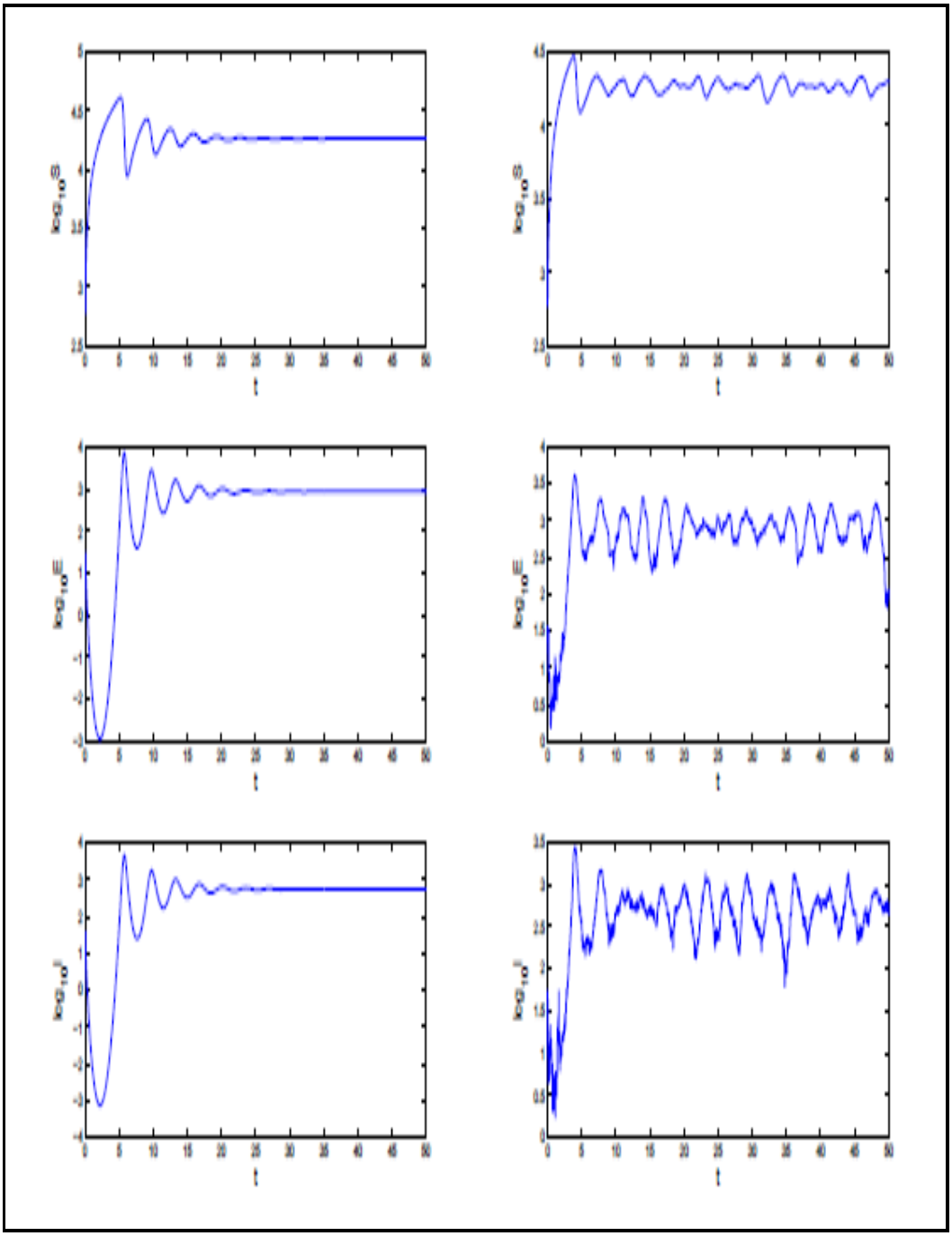

Figure 4: Transient analysis of the deterministic model (left panel) and stochastic model (right panel) *S*, *E* and *I*.

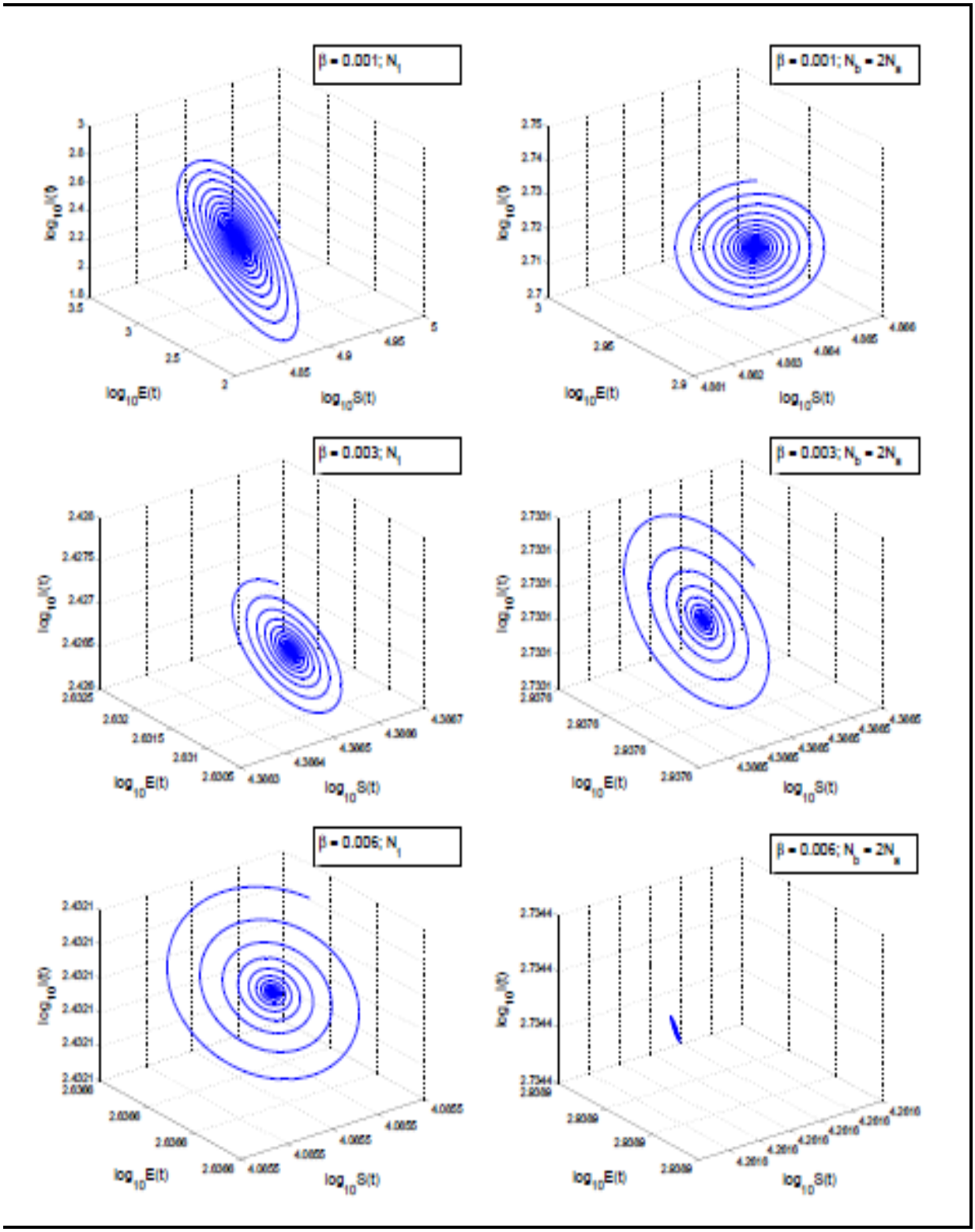

Figure 5: Phase space portraits of *S*, *E* and *I* for deterministic model.

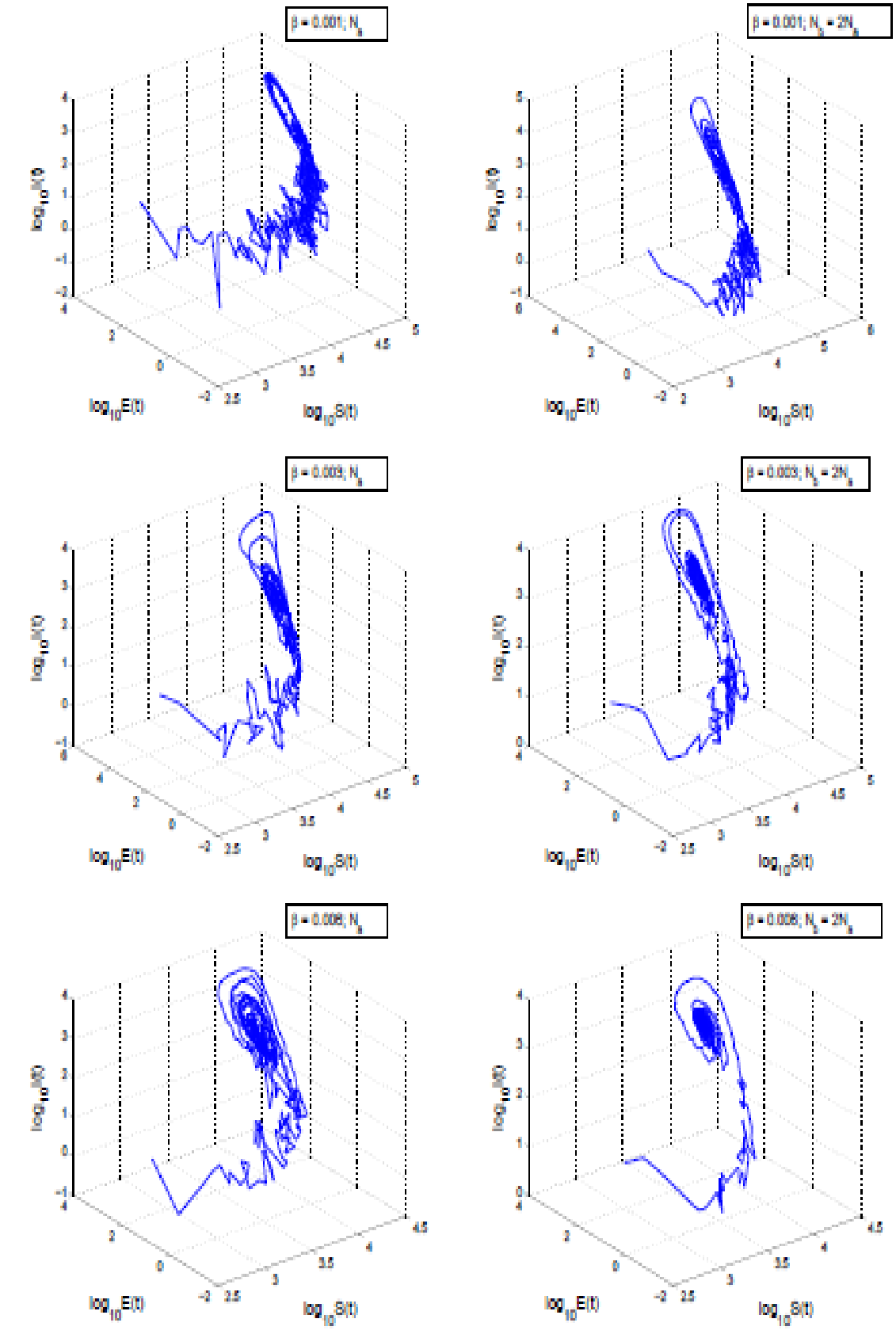
β = 0.001; N0
β = 0.001; N0 = 2N0
β = 0.003; N0
β = 0.003; N0 = 2N0
β = 0.006; N0
β = 0.006; N0 = 2N0
log10I(t)
log10E(t)
log10S(t)

Figure 6: Phase space portraits of *S, E* and *I* for stochastic model.