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RESEARCH ARTICLE

INVESTIGATION OF DETERMINISTIC AND STOCHASTIC SIR EPIDEMIOLOGICAL MODEL WITH NONLINEAR INCIDENCE RATE

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Abstract

In this study, an expanded SIR-type model is provided that takes behavioral and environmental factors into account when analyzing the dynamics of transmission. Equilibrium points and their local stability are explored in a deterministic framework, and the fundamental reproduction number is also calculated. The model is then reconstructed using a discrete-time Markov chain (DTMC) technique to represent the random character of illness propagation in real-world settings. The evolution of the epidemic can be analyzed probabilistically using transition probabilities thanks to this stochastic framework. Numerical simulations are used to verify the outcomes of the deterministic and stochastic versions, and a comparison of their predictive tendencies is made. The results have demonstrated the need to include stochasticity in epidemiological models, particularly when taking variability and uncertainty in transmission dynamics into consideration. This dual viewpoint gives useful insights for public health policies as well as a fuller knowledge of how diseases spread.

Keywords

SIR Model,
Stochastic Modeling,
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1. INTRODUCTION

Comprehending the dynamics of infectious illness transmission is essential for developing public health initiatives and controlling epidemics [1]. In order to facilitate these endeavors, mathematical modeling has emerged as a crucial instrument for the quantitative examination of epidemiological processes [2]. The classical SIR (Susceptible–Infected–Recovered) model is notable among these models due to its simplicity and deterministic structure, which captures the fundamental principles of disease recovery and dissemination [2, 3].

The fundamental work of Kermack and McKendrick in 1927 [2] is where the SIR model got its start, and it has subsequently sparked a large amount of study in both deterministic and stochastic contexts [4, 5]. The SIR system may be used to reasonably mimic a number of infectious illnesses, particularly those with a relatively simple pattern of transmission and those in which immunity develops after infection. Some examples of diseases can be modeled as an SIR system include: Mumps, rubella, chickenpox, influenza etc.

Nonetheless, empirical observations and real-world data reveal that the progression of epidemics is subject to various uncertainties and random fluctuations [6, 7]. These stochastic influences often arise from heterogeneous contact patterns, environmental variability, and behavioral responses.

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According to a number of studies, including stochastic elements is crucial to enhancing model realism and capturing important dynamics like outbreak variability, extinction likelihood, or epidemic initiation latency [8, 9]. Therefore, depending exclusively on deterministic models might lead to simplistic evaluations, which emphasizes the need for stochastic components in epidemiological models [4].

In this work, we create an expanded SIR-type model by adding more parameters that take into consideration outside variables that affect the spread of illness. These variables are intended to represent behavioral feedback mechanisms, social interaction levels, and environmental influences [10, 11].

In the deterministic context, we first determine the fundamental reproduction number R_0 and examine the equilibrium points of the model together with their local stability criteria [12]. A discrete-time Markov chain (DTMC) technique is then used to reframe the model within a stochastic framework, allowing for a probabilistic examination of the system dynamics through transition probabilities [7, 9, 13]. Lastly, numerical simulations are used to confirm the theoretical results, and the predictive behavior of the stochastic and deterministic formulations is contrasted.

This technique is innovative because it provides a more thorough and accurate depiction of epidemic evolution by directly integrating behavioral and environmental factors into the transmission function. Furthermore, a more sophisticated comprehension of system stability and variability in real-world scenarios is made possible by the dual analysis, which combines deterministic and stochastic elements [1, 4, 13].

The structure of the paper is as follows: We look at the equilibrium states and their stability characteristics in Section 2. Section 3 involves a stochastic reformulation of the model, theoretical calculations within the Markov chain framework, and a detailed examination of the epidemic dynamics using the resulting transition probabilities. Section 4 provides numerical simulations to validate the theoretical insights and compares the deterministic and stochastic models. In conclusion, Section 5 provides a recap of the main findings and discusses possible directions for future studies.

2. DETERMINISTIC MODEL

Mathematical epidemiology employs a variety of compartmental models to capture the transmission dynamics of infectious diseases within populations. These models classify individuals according to their epidemiological status, such as susceptible (S), infected (I), and recovered (R), resulting in widely studied frameworks including SIS, SIR, SEIR, SEIRS, and SI models. The nomenclature directly reflects the compartments considered, each tailored to the biological and immunological characteristics of specific diseases [2, 4, 14]. Over time, these models have evolved to incorporate additional biological realism, control strategies, and environmental factors, enhancing their applicability across diverse epidemic contexts [5, 6]. In this study, we focus on the classical SIR model, which explicitly accounts for immunity acquired post-infection. We undertake a deterministic analysis of the model dynamics, investigating the influence of key epidemiological parameters and stability properties of equilibria.

In the presented model, the variables $S(t)$, $I(t)$, and $R(t)$ represent the counts of susceptible, infected, and recovered individuals at time t, respectively. The total population size is given by $N = S(t) + I(t) + R(t)$. The initial conditions satisfy $S(0) + I(0) + R(0) = N$. We assume that the birth rate equals the death rate, so that the total population size is constant, $\frac{dN}{dt} = 0$. The natural mortality rate and recovery rate of infected people are denoted by the values μ and r, respectively. While $\beta > 0$ indicates the transmission rate, the recently added parameters α_1, α_2, and α_3 represent the impact of environmental factors, social interaction, and population density on disease transmission [7, 12]. The fact that disease transmission is influenced by larger social and environmental factors in addition to interpersonal interaction is reflected in this formulation.

The following figure represents the population's dynamic transitions:

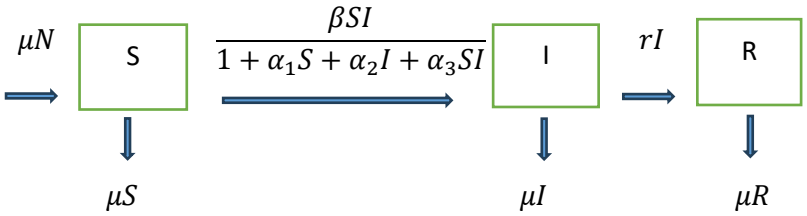

Figure 1. SIR Compartmental Diagram

Based on the previously mentioned hypotheses, differential equations that describe the dynamics of a SIR epidemic model have the following form:

$$\begin{aligned}\frac{dS}{dt} &= \mu N - \mu S - \frac{\beta SI}{1+\alpha_1 S+\alpha_2 I+\alpha_3 SI},\\ \frac{dI}{dt} &= \frac{\beta SI}{1+\alpha_1 S+\alpha_2 I+\alpha_3 SI} - (\mu + r)I,\\ \frac{dR}{dt} &= rI - \mu R.\end{aligned} \tag{2.1}$$

This system describes the evolution of the population compartments by incorporating both demographic processes (births and deaths) and disease-specific dynamics, infection and recovery. The introduction of the α_i parameters allows the model to account for behavioral adaptations, policy interventions, and environmental variability—thus enhancing its applicability to complex epidemic scenarios [6].

It can be observed that that in the first two equations of (2.1), the compartment $R = R(t)$ is absent. The last equation of the system (2.1) , $R = N - S - I$, can be used to determine R. Consequently, we can think about the sub-system provided by

$$\begin{aligned}\frac{dS}{dt} &= \mu N - \mu S - \frac{\beta SI}{1+\alpha_1 S+\alpha_2 I+\alpha_3 SI},\\ \frac{dI}{dt} &= \frac{\beta SI}{1+\alpha_1 S+\alpha_2 I+\alpha_3 SI} - (\mu + r)I.\end{aligned} \tag{2.2}$$

The reduced system (2.2) allows for a more concise analysis of the interaction between susceptible and infected individuals [8].

The basic reproduction number, R_0, is the number of secondary infections caused by one infected individual in an entirely susceptible population [3, 4, 6, 15].To calculate the basic reproduction number, R_0, it is necessary to determine the appearance of new infections and exits from the system. R_0 can be found using the *next generation matrix* method introduced by Van Den Driessche & Watmough. In this method, the emergence of new infections caused by infected individuals, and their exits from the system (i.e., through recovery or death), are analyzed.

The part containing new infections appears only in the differential equation of the I variable. If we denote the new infections by $\mathcal{F}$, then for our system, the new infections are given by

$$\mathcal{F} = \frac{\beta SI}{1 + \alpha_1 S + \alpha_2 I + \alpha_3 SI}.$$

The exits from the infections are represented as

$$\mathcal{V} = (\mu + r)I.$$

The $\mathcal{V}$ equation describes the departure of individuals from the system due to recovery (rI) and natural death (μI). The basic reproduction number R_0 is

$$R_0 = \rho(\mathcal{F}\mathcal{V}^{-1}),$$

where ρ denotes the largest eigenvalue of the matrix.

Remark 2.1 To calculate R_0, we need to consider the disease spread in the "initial outbreak" situation. That is, when the disease is just starting to spread, we assume $S \approx S_0$ (the population is mostly susceptible, with only a few infected individuals). Therefore, we will replace S with S_0 in the **F** matrix.

$$\mathbf{F} = \left[\frac{\beta S_0}{1 + \alpha_1 S_0}\right]$$

$$R_0 = \rho(FV^{-1})$$

Here, FV^{-1} represents the matrix that shows the spread of the infection, and $\rho(FV^{-1})$ denotes the largest eigenvalue of the FV^{-1} matrix.

$$V = [\mu + r]$$

$$V^{-1} = \left[\frac{1}{\mu + r}\right]$$

$$FV^{-1} = \left[\frac{\beta S_0}{1 + \alpha_1 S_0}\right]\left[\frac{1}{\mu + r}\right]$$

$$= \left[\frac{\beta S_0}{(1 + \alpha_1 S_0)(\mu + r)}\right]$$

Since the resulting matrix is of size 1×1, its only eigenvalue is the matrix itself. Therefore for model (2.2), the basic reproduction number is defined as follows:

$$R_0 = \frac{\beta S_0}{(1 + \alpha_1 S_0)(\mu + r)}.$$

Here, S_0 was taken for values where the disease is absent or minimal. Thus, for $I = 0$ or $I \approx 0$, we have $S_0 \approx S^* = N$. That is, the basic reproduction number is given by

$$R_0 = \frac{\beta N}{(1 + \alpha_1 N)(\mu + r)}.$$

Theorem 2.2

- If $R_0 \leq 1$, then system (2.2) has a unique disease-free equilibrium of the form $E_0(S_0, 0) = (N, 0)$.
- If $R_0 > 1$, the disease-free equilibrium is still present and system (2.2) has a unique endemic equilibrium of the form $E^*(S^*, I^*) = \left(S^*, \frac{\mu N - \mu S^*}{X}\right)$, where

$$S^* = N + \frac{X\left(\alpha_1 X - \beta - \alpha_3 \mu N - \alpha_2 \mu + \sqrt{\Delta}\right)}{2\mu\alpha_3 X},$$
$$I^* = -\frac{\left(\alpha_1 X - \beta - \alpha_3 \mu N - \alpha_2 \mu + \sqrt{\Delta}\right)}{2\alpha_3 X},$$

with $X = \mu + r$ and $\Delta = \alpha_1^2\mu^2 + 2\alpha_1^2\mu r + \alpha_1^2 r^2 - 2\alpha_1\alpha_2\mu^2 - 2\alpha_1\alpha_2\mu r + 2\alpha_1\alpha_3\mu^2 N + 2\alpha_1\alpha_3\mu N r - 2\beta\alpha_1\mu - 2\beta\alpha_1 r + \alpha_2^2\mu^2 + 2\alpha_2\alpha_3\mu^2 N + 2\beta\alpha_2\mu + \alpha_3^2\mu N^2 - 2\beta\alpha_3\mu N + 4\alpha_3\mu^2 + 4\alpha_3\mu r + \beta^2$

Δ can be simplified as $\Delta = (\beta - \alpha_1 X + \alpha_2\mu - \alpha_3\mu N)^2 + 4\alpha_3\mu(X + \alpha_2\mu N)$.

Next, we study the local stability of the disease-free equilibrium $E_0(S_0, 0)$ and the endemic equilibrium $E^*(S^*, I^*)$. We define the Jacobian matrix of system (2.2) at any equilibrium $E(S, I)$ by

$$J_{E(S,I)} = \begin{pmatrix} -\mu - \frac{\beta I(1+\alpha_2 I)}{(1+\alpha_1 S+\alpha_2 I+\alpha_3 SI)^2} & \frac{-\beta S(1+\alpha_1 S)}{(1+\alpha_1 S+\alpha_2 I+\alpha_3 SI)^2} \\ \frac{\beta I(1+\alpha_2 I)}{(1+\alpha_1 S+\alpha_2 I+\alpha_3 SI)^2} & \frac{\beta S(1+\alpha_1 S)}{(1+\alpha_1 S+\alpha_2 I+\alpha_3 SI)^2} - X \end{pmatrix}. \tag{2.3}$$

Theorem 2.3. The disease-free equilibrium $E_0(S_0, 0)$ is locally asymptotically stable if $R_0 \leq 1$ and unstable whenever $R_0 > 1$.

Proof. The equilibrium point of the system is $E_0(S_0, 0) = (N, 0)$, and at $E_0(S_0, 0)$, (2.3) becomes

$$J(E_0) = \begin{pmatrix} -\mu & \frac{-\beta\mu N}{\mu(\alpha_1 N+1)} \\ 0 & \frac{\beta\mu N}{\mu(\alpha_1 N+1)} - \mu - r \end{pmatrix} \tag{2.4}$$

$$\det(J(E_0) - kI) = \begin{vmatrix} -\mu - k & \frac{-\beta\mu N}{\mu(\alpha_1 N+1)} \\ 0 & \frac{\beta\mu N}{\mu(\alpha_1 N+1)} - \mu - r - k \end{vmatrix}$$

$$\det(J(E_0) - kI) = (-\mu - k) \cdot \left(\frac{\beta\mu N}{\mu(\alpha_1 N+1)} - \mu - r - k\right) - 0 = 0$$

$$(-\mu - k) \cdot \left(\frac{\beta\mu N}{\mu(\alpha_1 N+1)} - \mu - r - k\right) = 0$$

$$-\mu - k = 0 \quad \Rightarrow \quad k_1 = -\mu$$

$$\frac{\beta\mu N}{\mu(\alpha_1 N+1)} - \mu - r - k = 0$$

$$\frac{\beta\mu N}{\mu(\alpha_1 N+1)} - \mu - r = k_2$$

Thus, the eigenvalues k_1 and k_2 are obtained. Since $k_1 < 0$, this condition is already satisfied for this eigenvalue. Now, let's analyze the eigenvalue k_2.

$$k_2 \leq 0 \Rightarrow \frac{\beta\mu N}{\mu(\alpha_1 N + 1)} - \mu - r \leq 0$$

$$\Rightarrow \frac{\beta\mu N}{\alpha_1\mu N + \mu} - (\mu + r) \leq 0$$

$$\Rightarrow (\mu + r)\left[\frac{\beta N}{(\alpha_1 N + 1)(\mu + r)} - 1\right] \leq 0$$

$$\Rightarrow (\mu + r)[R_0 - 1] \leq 0$$

$$\Rightarrow [R_0 - 1] \leq 0$$

$$R_0 \leq 1$$

Hence, the eigenvalues k_1 and k_2 of system (2.4) are negative when $R_0 \leq 1$. Therefore, the equilibrium point E_0 is locally asymptotically stable for $R_0 \leq 1$, and becomes unstable whenever $R_0 > 1$. ▫

Theorem 2.4 The endemic equilibrium $E^*(S^*, \frac{\mu N - \mu S^*}{X})$ is locally asymptotically stable if $R_0 > 1$ and unstable whenever $R_0 \leq 1$.

Proof. At $E^*(S^*, I^*)$, (2.3) becomes

$$J(E^*(S^*, I^*)) = \begin{pmatrix} -\mu - \frac{\beta I^*(1+\alpha_2 I^*)}{(1+\alpha_1 S^* + \alpha_2 I^* + \alpha_3 S^* I^*)^2} & \frac{-\beta S^*(1+\alpha_1 S^*)}{(1+\alpha_1 S^* + \alpha_2 I^* + \alpha_3 S^* I^*)^2} \\ \frac{\beta I^*(1+\alpha_2 I^*)}{(1+\alpha_1 S^* + \alpha_2 I^* + \alpha_3 S^* I^*)^2} & \frac{\beta S^*(1+\alpha_1 S^*)}{(1+\alpha_1 S^* + \alpha_2 I^* + \alpha_3 S^* I^*)^2} - X \end{pmatrix} \tag{2.5}$$

For simplicity, let us assume the following equalities:

- $\frac{\beta I^*(1+\alpha_2 I^*)}{(1+\alpha_1 S^* + \alpha_2 I^* + \alpha_3 S^* I^*)^2} = G$
- $\frac{\beta S^*(1+\alpha_1 S^*)}{(1+\alpha_1 S^* + \alpha_2 I^* + \alpha_3 S^* I^*)^2} = H$

Thus, we can rewrite the (2.5) as:

$$J\big(E^*(S^*, I^*)\big) = \begin{pmatrix} -\mu - G & -H \\ G & H - X \end{pmatrix}$$

$$\det\big(J(E^*) - kI\big) = \begin{vmatrix} -\mu - G - k & -H \\ G & H - X - k \end{vmatrix}$$

$$= (-\mu - G - k)(H - X - k) + GH = 0$$

$$= k^2 + (G - H + X + \mu)k + GX - \mu H + \mu X = 0$$

$$\det(J(E^*) - kI) = \begin{vmatrix} -\mu - G - k & -H \\ G & H - X - k \end{vmatrix}$$

$$= (-\mu - G - k)(H - X - k) + GH = 0$$

$$= k^2 + (G - H + X + \mu)k + GX - \mu H + \mu X = 0$$

Hence, the eigenvalues of $J(E^*)$ are

$$k_{1,2} = \frac{-(G-H+X+\mu) \pm \sqrt{(G-H+X+\mu)^2 - 4(GX - \mu H + \mu X)}}{2} \tag{2.6}$$

The characteristic equation of the (2.5) is

$$k^2 + a_1 k + a_0 = 0$$

where

- $a_0 = GX - \mu H + \mu X$,
- $a_1 = (G - H + X + \mu)$.

We assume that $R_0 > 1$. Hence, $a_0 > 0$ and $a_1 > 0$ whenever $R_0 > 1$. Thus , by Routh-Hurwitz criterion [19] , all the eigenvalues of the (2.5) system of equations defining the model have negative real parts at point E^* for $R_0 > 1$, which ensures the locally asymptotic stability of equilibrium point for $R_0 > 1$ and unstable whenever $R_0 \leq 1$.

3. FORMULATION OF DTMC SIR EPIDEMIC MODEL

Stochastic models, such as discrete-time Markov chains (DTMC), provide a valuable framework for capturing the randomness inherent in epidemic spread within finite populations [4, 13].

In this section, we formulate the extended SIR model as a DTMC by defining the state space and transition probabilities, incorporating environmental and behavioral factors into the transmission dynamics. Beyond deterministic predictions, this stochastic approach allows for a thorough probabilistic examination of illness course [6, 16].

A Markov chain with a limited state space is used to formulate the discrete-time stochastic SIR model. In a population of fixed size N, the state space is defined as the set of ordered pairs $\{(N,0),\ (N-1,0),\ \dots,\ (0,0),\ (N-1,1),\ (N-2,1),\ \dots,\ (0,1),\ \dots,\ (0,N)\}$, where each (S,I) combination corresponds to the susceptible and infected population sizes at a given time point.

The stochastic SIR model is a bivariate process characterized by the random variables S and I, representing the number of susceptible and infected individuals at time t, respectively. The number of recovered individuals at time t is calculated as $R(t) = N - S(t) - I(t)$.

Within the DTMC epidemic framework, time evolves in discrete intervals $t \in \{0, \Delta t, 2\Delta t, \dots\}$, where Δt denotes the constant time step. The system assumes that at most one event (infection or recovery) occurs during each time interval [7, 13, 17].

The stochastic SIR model has a joint probability function,

$$p_{(s,i)}(t) = Prob(S(t) = s, I(t) = i) \tag{3.1}$$

where $s, i = 0,1,2, \dots, N$ and $0 \leq s + i \leq N$.

Let, $\frac{\beta SI}{1+\alpha_1 S+\alpha_2 I+\alpha_3 SI}\Delta t,\ rI\Delta t$ and $1 - \left[\frac{\beta SI}{1+\alpha_1 S+\alpha_2 I+\alpha_3 SI} + rI\right]\Delta t$ denote the probability of infection of a susceptible individual, the probability of recovery of an infective or the probability of no change, respectively:

$$\begin{aligned} &Prob[S_{t+\Delta t} = s-1, I_{t+\Delta t} = i+1 \ ] \mid S_t = s, I_t = i] = \frac{\beta SI}{1+\alpha_1 S+\alpha_2 I+\alpha_3 SI}\Delta t \\ &Prob[S_{t+\Delta t} = s, I_{t+\Delta t} = i-1 \ ] \mid S_t = s, I_t = i] = \ rI\Delta t \\ &Prop[S_{t+\Delta t} = s, I_{t+\Delta t} = i \ ] \mid S_t = s, I_t = i] = 1 - \left[\frac{\beta SI}{1+\alpha_1 S+\alpha_2 I+\alpha_3 SI} + rI\right]\Delta t \end{aligned} \tag{3.2}$$

Each death is balanced by a corresponding birth, maintaining a constant population size.

The difference equations satisfied by the joint probability $p_{(s,i)}(t)$ are

$$\begin{aligned} p_{(s,i)}(t+\Delta t) = &\frac{\beta(s+1)(i-1)}{1+\alpha_1(s+1)+\alpha_2(i-1)+\alpha_3(s+1)(i-1)}\Delta t p_{(s+1,i-1)}(t) \\ &+r(i+1)\Delta t p_{(s,i+1)}(t) \\ &+\left[1 - \left(\frac{\beta si}{1+\alpha_1 s+\alpha_2 i+\alpha_3 si} + ri\right)\Delta t\right] p_{(s,i)}(t) \end{aligned} \tag{3.3}$$

where $s, i = 0,1,2, \dots, N$ and $p_{(s,i)}(t) = 0$ if $s, i \notin \{0,1, \dots, N\}$. To ensure that the transition probabilities are positive and bounded by one, it is required that

$$\sum_{\substack{s+i=0,1,\dots,N \\ s+i \leq N}} p_{(s,i)}(t) \leq 1.$$

The inequality above is satisfied if Δt is chosen sufficiently small. We express equation (3.3) in matrix and vector notation as below, where $t = n\Delta t$ and $p(t)$ is a row vector of probabilities for the states (s, i) at time t:

$$p(t+\Delta t) = p(t)P = p(0)P^{n+1}$$

The bivariate process exhibits Markov property and is time-homogeneous by nature. By selecting a sufficiently small-time step Δt, it can be assumed that at most one transition occurs within each interval. Let the initial state of the population at time $t = 0$ be $(S_0, I_0) = (s_0, i_0)$, implying $P[(S_0, I_0) = (s_0, i_0)] = 1$. To predict future states, we use the *Markov property* [17, 18], which states that the future state depends only on the current state, not on the past. This is particularly expressed by the following equation (3.4).

$$p_{(s+m,i+n),(s,i)}(\Delta t) = P[(S_{t+\Delta t}, I_{t+\Delta t}) = (m,n) \mid (S_t, I_t) = (s,i), \dots, (S_0, I_0) = (s_0, i_0).]$$

$$p_{(s+m,i+n),(s,i)}(\Delta t) = P[(S_{t+\Delta t}, I_{t+\Delta t}) = (m,n) \mid (S_t, I_t) = (s,i).] \tag{3.4}$$

Thus, the transition probabilities of the SIR model can be represented as follows:

$$p_{(s,i),(m,n)}= \begin{cases} \dfrac{\beta SI}{1+\alpha_1 S+\alpha_2 I+\alpha_3 SI}\Delta t, & (m,n)=(s-1,i+1) \\ rI\Delta t\ , & (m,n)=(s,i-1) \\ 1-\left[\dfrac{\beta SI}{1+\alpha_1 S+\alpha_2 I+\alpha_3 SI}+rI\right]\Delta t, & (m,n)=(s,i\,) \\ 0\,, & otherwise \end{cases} \tag{3.5}$$

The only absorbing states in the model are of the form $(S,0)$, where $S=0,1,\dots,N$, corresponding to the extinction of the disease. Once the system reaches any such state, it remains there permanently, meaning that the transition probability satisfies $p_{(s,0),(s,0)}=1$. Hence, there are $N+1$ absorbing states in total and the transition matrix P as follows:

$$P=\begin{pmatrix} \text{States} & (N,0) & (N-1,0) & \cdots & (1,0) & (0,0) & (N-1,1) & (\text{N}-2,1) & \dots & (0,1) & \dots & (0,N) \\ (N,0) & 1 & 0 & \cdots & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ (N-1,0) & 0 & 1 & \ddots & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \vdots & \vdots & \vdots & \ddots & \vdots & \vdots & \vdots & \vdots & \vdots & \vdots & \vdots & \vdots \\ (1,0) & 0 & 0 & \dots & 1 & 0 & 0 & 0 & 0 & 0 & \dots & 0 \\ (0,0) & 0 & 0 & \dots & 0 & 1 & 0 & 0 & 0 & 0 & \dots & 0 \\ (N-1,1) & 0 & r\Delta t & \dots & 0 & 0 & 1-\left[\dfrac{\beta(N-1)}{1+\alpha_1(N-1)+\alpha_2+\alpha_3(N-1)}+r\right]\Delta t & 0 & 0 & 0 & 0 & 0 \\ (\text{N}-2,1) & 0 & 0 & 0 & \dots & 0 & 0 & 1-\left[\dfrac{\beta(N-2)}{1+\alpha_1(N-2)+\alpha_2+\alpha_3(N-2)}+r\right]\Delta t & \dots & 0 & \dots & 0 \\ \vdots & \vdots & \vdots & \vdots & \ddots & \vdots & \vdots & \vdots & \ddots & 0 & 0 & \vdots \\ (0,1) & 0 & 0 & \dots & 0 & r\Delta t & 0 & 0 & \dots & 1-r\Delta t & \dots & 0 \\ \vdots & \vdots & \vdots & \vdots & \vdots & \vdots & \vdots & \vdots & \vdots & \vdots & \ddots & \vdots \\ (0,N) & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1-Nr\Delta t \end{pmatrix}$$

4. NUMERICAL SIMULATIONS

In this section, we present numerical simulations to illustrate the dynamical behavior of both the deterministic and stochastic SIR epidemic models under consideration. The simulations are designed to give a thorough comparison between the discrete-time Markov chain (DTMC) formulation of the model and the standard deterministic method.

As a starting point for comprehending the general dynamics of the epidemic, we start by looking at the temporal evolution of the susceptible, infected, and recovered populations in the deterministic SIR model.

We next use many sample routes produced by the DTMC SIR epidemic model to investigate the stochastic behavior of the system. To illustrate the intrinsic unpredictability brought about by stochastic influences, these trajectories are placed next to the deterministic solution (shown by a dotted curve).

Finally, we analyze the probability distribution of the states in the DTMC SIR model. This distribution is computed using the iterative formula

$$p(t+\Delta t)=p(t)P=p(0)P^{n+1},$$

where P denotes the transition probability matrix, $p(t)$ is the state probability vector at time t, and Δt is the discrete time step. This analysis provides insight into the likelihood of the system residing in various states over time.

- *Case I:* $R_0 > 1$

When the basic reproduction number exceeds unity ($R_0 > 1$), the epidemic tends to spread through the population. Figure 2 displays the temporal evolution of the susceptible, infected, and recovered compartments according to the deterministic SIR model. As observed, the infected population grows rapidly, reaching a peak before declining to an endemic equilibrium.

Figure 3 shows three sample paths of the discrete-time Markov chain (DTMC) SIR model, plotted alongside the deterministic solution (dashed line). The stochastic trajectories exhibit variability but generally align with the deterministic trend, emphasizing the role of randomness in disease spread.

Finally, Figure 4 illustrates the evolution of the probability distribution of states in the stochastic model. The distribution concentrates around the endemic equilibrium over time, reflecting the persistence of the infection in the population.

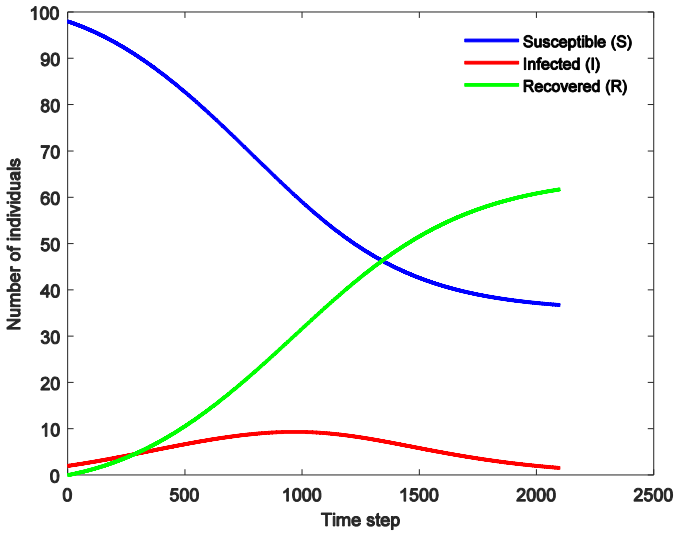

Figure 2. Time-course dynamics of epidemic spread in the SIR epidemic model for Case I.

The simulation parameters are set as follows: $\Delta t = 0.01$, $N = 100$, $\beta = 0.1$, $\alpha_1 = 0.1$, $\alpha_2 = 0.02$, $\alpha_3 = 0.003$, $\mu = 0.1$, $r = 0.5$ with initial condition $(S(0), I(0)) = (98,2)$. In the stochastic formulation, the initial condition holds with certainty, i.e. ,

$$P((S(0), I(0)) = (98,2)) = 1.$$

Both the basic reproduction number and the initial replacement number exceed unity, with $R_0 = 1.5152 > 1$. The epidemic outbreak is clearly observable in the trajectory of the deterministic solution, while each of the three sample paths similarly displays a characteristic epidemic curve.

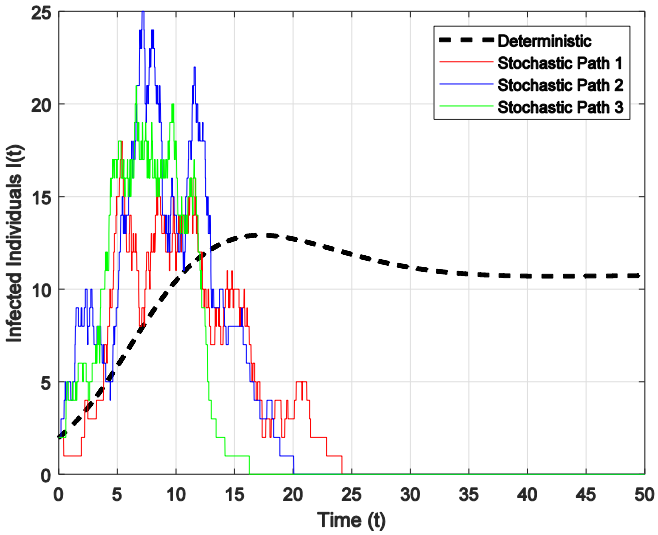

Figure 3. Three sample paths of the DTMC SIR epidemic model are graphed with the deterministic solution (dashed curve). Parameter values are the same as in Figure 2

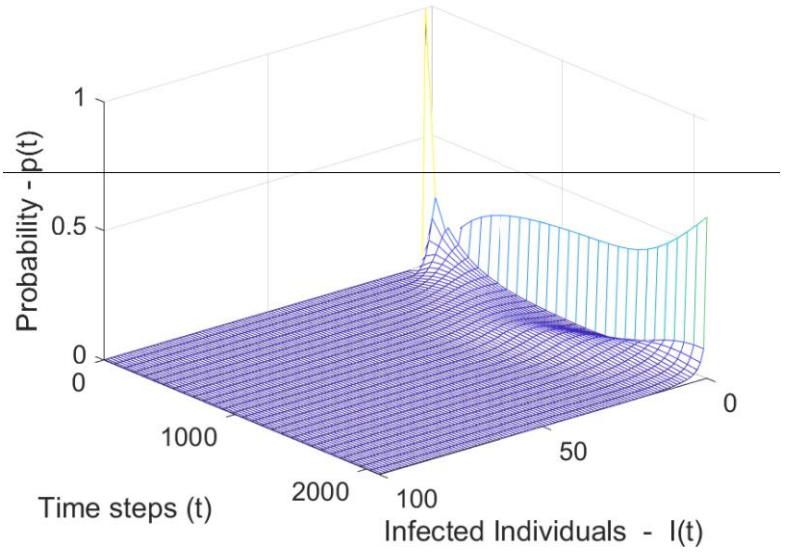

Figure 4. Probability distribution of the DTMC SIR epidemic model, $(I(t), t, p(t))$. Parameter values are the same as in Figure 2

- *Case II:* $R_0 \leq 1$

For $R_0 \leq 1$, the infection is expected to die out, and the disease-free equilibrium is stable. This is demonstrated in Figure 5, where the deterministic infected population declines monotonically to zero.

The stochastic sample paths presented in Figure 6 also show that the infection eventually disappears in all realizations, confirming the extinction of the disease.

Moreover, the probability distribution depicted in Figure 7 increasingly concentrates on the disease-free state as time progresses, highlighting the absorbing nature of this equilibrium in the stochastic

framework. Now, we choose $\beta = 0.05$, and we keep the other parameter values. In this case $R_0 = 0.75758 \leq 1$.

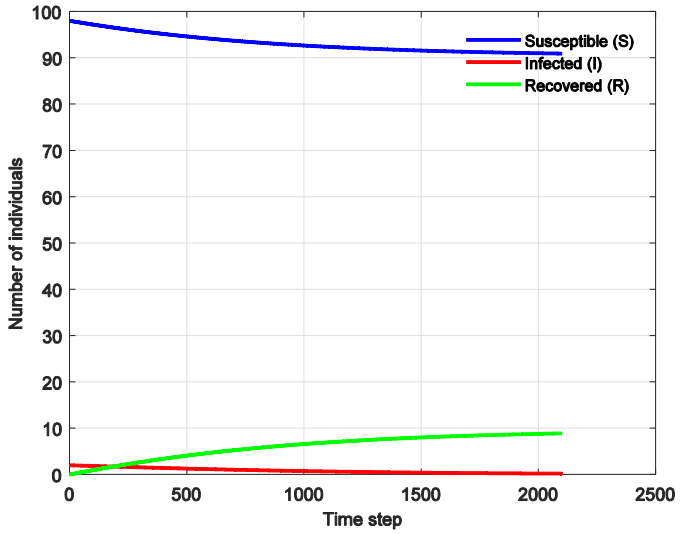

Figure 5. Time-course dynamics of epidemic spread in the SIR epidemic model for Case II.

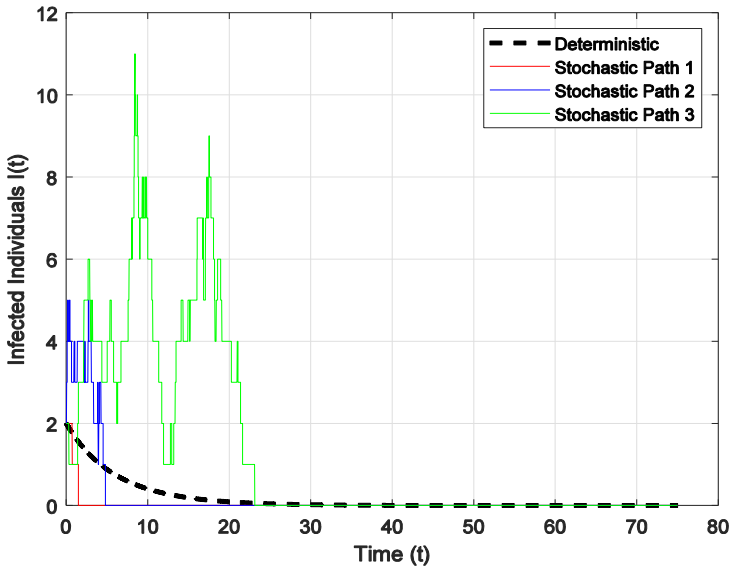

Figure 6. Three sample paths of the DTMC SIR epidemic model are graphed with the deterministic solution (dashed curve). Parameter values are the same as in Fig.5

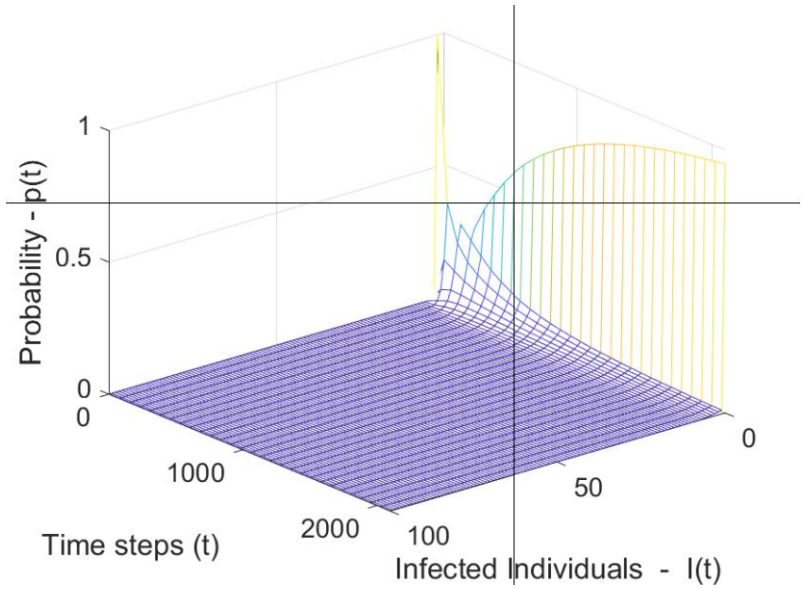

Figure 7. Probability distribution of the DTMC SIR epidemic model, $\big(I(t), t, p(t)\big)$. Parameter values are the same as in Figure 5

5. CONCLUSION

In this study, we investigated the dynamics of an epidemic using both deterministic and stochastic formulations of the SIR model. The deterministic model was analyzed in terms of equilibrium points and the basic reproduction number R_0, providing a foundational understanding of the system's long-term behavior.

To capture random fluctuations inherent in real-world epidemic processes, the model was extended to a discrete-time Markov chain (DTMC) framework. The transition probabilities between states were derived, and the corresponding transition matrix was expressed in canonical form. The transient and absorbing states were identified and interpreted in the context of epidemic spread and extinction.

Numerical simulations were carried out to visualize and compare the evolution of the epidemic under both deterministic and stochastic settings. For values of $R_0 \leq 1$, the infection was shown to die out, aligning with the theoretical expectation of global stability at the disease-free equilibrium. When $R_0 > 1$, both the deterministic trajectory and the stochastic sample paths indicated sustained transmission, with the probability distribution eventually spreading over a range of infected states rather than collapsing to the disease-free condition.

Overall, the results demonstrate the importance of considering stochastic effects in epidemic modeling, particularly for small population sizes or near the critical threshold $R_0 = 1$, where random fluctuations may significantly alter the system's outcome. The integration of deterministic analysis and stochastic simulation provides a comprehensive view of epidemic dynamics and enhances our understanding of the probabilistic nature of disease transmission.

CONFLICT OF INTEREST

The authors stated that there are no conflicts of interest regarding the publication of this article.

CRediT AUTHOR STATEMENT

Ali Serdar Nazlıpınar: Supervision, Project administration, Conceptualization, Methodology,
Kübra Erol: Formal analysis, Investigation, Writing – Original Draft, Visualization.

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