



Simulating Deterministic and Stochastic Differential Equation Models of Measles Outbreak Considering Population Size and Initial Vaccination Regime

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Abstract In this paper a stochastic differential equation model as a version of the classical deterministic SIR model by Kermack and McKendrick (1927) is developed and both the stochastic and the deterministic models are applied to measles outbreaks. The objectives are to compare the solutions of these models for varying susceptible sizes; and assess the feasibility of measles control for varying initial vaccination regimes. The models were solved numerically using Euler method and the patterns of measles outbreaks were compared. Numerical results reveal that the solutions of the stochastic model display strong stochastic components for small susceptible population sizes. However, the solutions of the stochastic model demonstrate weak chance effects for larger susceptible population sizes. Thus, the solution of the deterministic model is a limit of the solutions of the stochastic counterpart for larger susceptible population sizes. The results further show that adequate vaccination coverage before invasion of infectious individuals into the population can stop measles outbreak.

Keywords stochastic model, deterministic model, simulation, measles

1. Introduction

Measles is a highly infectious illness caused by a paramyxovirus, of genus morbilivirus. The virus lives in the mucus of the nose and the throat of people with this infection. It is one of the first and worst childhood diseases. Sign and symptoms of measles include cough, runny nose, inflamed eyes, sore throat, fever and a red blotchy skin rash. It can lead to serious and fatal complications including pneumonia, diarrhea, encephalitis, blindness, deafness or impaired vision. Physical contact, coughing and sneezing can spread the infection. Once quiet common, measles can now almost always be prevented with a vaccines [1].

Measles is still a leading cause of death among young children, despite the availability of an effective vaccine for the past 40years. Although it is rare in many developed countries, it remains a common illness in many developing countries and more than half a million people, mostly children, died from measles in 2003 [2]. As reported in [3], measles caused an estimated 2.6 million deaths in 1980, 75% decrease in deaths from 2000 through 2013, 145,700 deaths in 2013, and estimated 20 million cases every year.

As reported in [5], Allen and his collaborators, in 1991, studied a discrete-time model with vaccination for measles epidemic. They used a discrete-time, age-independent *SIR*-type epidemic model. They applied their model to measles epidemic on a university campus. [6] developed a simple stochastic Mathematical model to investigate the dynamics of measles epidemic. Their model is a multi-dimensional diffusion process with SEIR compartments. An analysis on extensive simulations of a stochastic metapopulation model (SEIR type) focusing on Seasonality and extinction in Chaotic metapopulations can be seen in [7].

[8] presented a detailed analysis of the pattern of measles outbreaks in the small isolated community of the Faroe Islands. Measles outbreaks in that population showed frequent fade-out of infection resulting in long intervals when the disease was absent from the islands. They used a Lattice-based epidemic model to provide a

theoretical estimate of the scaling exponents. A mathematical model for the simulation of a localized measles epidemic was presented in [9]. Susceptible-Exposed-Infected-Recovered (SEIR) model was used in [10] to study the transmission dynamics of measles. A univariate time series analysis on pertussis, mumps, measles and rubella based on Box-Jenkins or Auto-Regressive Integrated Moving Average (ARIMA) model was carried in [11].

As reported in [11], most mathematical models are used to study the epidemiology of childhood viral diseases, such as measles. He described the period of infectiousness by an exponential distribution. He used Susceptible Infectious Recovered (SIR) model in his study. [13] used SEIR deterministic model to provide useful insights into the mechanic of many common childhood diseases such as measles. A survey of stochastic epidemic models can be seen in [14].

The plan of this paper is as follows. In section 2, we present model development and analysis. Numerical simulation and result are presented in sections 3. Finally, discussion and conclusive remarks are passed in sections 4 and 5 respectively.

2. Model Development and Analysis

As in [3], the SIR model, first published by Kermack and McKendrick in 1927, is undoubtedly the most famous mathematical model for the spread of an infectious disease. Here, people are characterized into three classes: susceptible *S*, infectious *I* and removed *R*. Removed individuals are no longer susceptible neither infectious for whatever reason; for example, they have recovered from the disease and are now immune, or they have been vaccinated, or they have been isolated from the rest of the population, or perhaps they have died from the disease. We assume that infectious individuals leave the *I* class at a constant rate γ , and move directly into the *R* class. The deterministic flow diagram and the model are as follows.

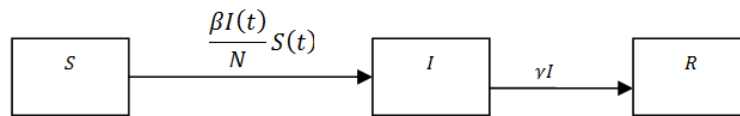


Figure 1: Transmission flow diagram

2.1. The Deterministic Model

$$\left. \begin{aligned}
 \frac{dS}{dt} &= \frac{-\beta I(t)}{N} S(t) \\
 \frac{dI}{dt} &= \frac{\beta I(t)}{N} S(t) - \gamma I(t) \\
 \frac{dR}{dt} &= \gamma I(t) \\
 N &= S(t) + I(t) + R(t)
 \end{aligned} \right\} (2.1)$$

We apply this SIR model to measles outbreaks based on the following assumptions. It is observed that measles outbreaks take place on a relatively short timescale in order of weeks or months. Also demographic processes in humans take place at much slower timescales. Therefore, birth and death do not have a major impact on the disease dynamics and are ignored. The process of vaccination only changes the fraction of susceptible individuals slowly and so vaccination can enter into the model in form of the fraction of susceptible individuals initially immunized [2].

Since the recovered individuals do not contribute in the transmission process, the system of equations (2.1) can be reduced as follows.

$$\left. \begin{aligned}
 \frac{dS}{dt} &= \frac{-\beta I(t)}{N} S(t) \\
 \frac{dI}{dt} &= \frac{\beta I(t)}{N} S(t) - \gamma I(t) \\
 R(t) &= N - S(t) - I(t)
 \end{aligned} \right\} (2.2)$$

From (2.2), the basic reproduction number, $R_0 = \frac{\beta}{\gamma}$.

2.2. Stochastic Model for Measles Outbreaks

We follow the method in [4]. Let $X_i, i = 1, 2, 3$ denote the random variables for *SIR* respectively. There are three possibilities in changes in the vector $X = [X_1, X_2, X_3]^T$ for a small interval Δt assuming at most one change can occur. The transition probabilities are represented by the following equation:

$$p(s + k, I + j) = \begin{cases} \frac{\beta I(t)}{N} S(t) \Delta t & (k, j) = (-1, 1) \\ \gamma I(t) \Delta t & (k, j) = (0, -1) \\ 1 - \left[\frac{\beta I(t)}{N} S(t) - \gamma I(t) \right] \Delta t & (k, j) = (0, 0) \end{cases}$$

To form the stochastic differential equations (SDEs) using the procedure adopted in [4], the expectation $E(\Delta X)$ and covariance $E((\Delta X)(\Delta X))^T$ need to be computed.

This can of course, be derived directly from the differential equations in (2.2). The compartmental changes in small time period Δt is shown in Table 1.

Table 1: The compartmental changes in small time period Δt

Transition	Probability
$(\Delta X)_1 = [-1, 1]^T$	$p_1 = \frac{\beta I(t) S(t)}{N} \Delta t$
$(\Delta X)_2 = [0, -1]^T$	$p_2 = \gamma I(t) \Delta t$

The system (2.2) will be changed into the stochastic differential equations in the form;

$$\Delta X(t) = F(t, X(t))dt - B(t, X(t))dw(t).$$

$$\begin{aligned} E(\Delta X) &= \sum_{i=1}^2 p_i (\Delta X)_i \\ &= p_1 \begin{pmatrix} -1 \\ 1 \end{pmatrix} + p_2 \begin{pmatrix} 0 \\ -1 \end{pmatrix} \\ &= \left(\frac{-\beta I(t) S(t)}{N} - \gamma I(t) \right) \Delta t. \end{aligned}$$

Covariance matrix $C = E((\Delta X)(\Delta X))^T$

$$\begin{aligned} &= \sum_{i=1}^2 p_i (\Delta X)_i (\Delta X)_i (\Delta X)^T \\ &= p_1 \begin{pmatrix} -1 \\ 1 \end{pmatrix} \begin{pmatrix} -1 & 1 \end{pmatrix} + p_2 \begin{pmatrix} 0 \\ -1 \end{pmatrix} \begin{pmatrix} 0 & -1 \end{pmatrix}. \\ C &= \begin{pmatrix} \frac{\beta I(t)}{N} S(t) & -\frac{\beta I(t)}{N} S(t) \\ -\frac{\beta I(t)}{N} S(t) & \frac{\beta I(t)}{N} S(t) + \gamma I(t) \end{pmatrix} \Delta t \end{aligned}$$

Since the matrix C is a positive definite matrix, $B = \sqrt{C}$ can be calculated as follows.

$$\begin{aligned} B &= \frac{1}{D} \begin{pmatrix} \frac{\beta I(t)}{N} S(t) + G & -\frac{\beta I(t)}{N} S(t) \\ -\frac{\beta I(t)}{N} S(t) & \frac{\beta I(t)}{N} S(t) + \gamma I(t) + G \end{pmatrix} \Delta t \\ G &= \sqrt{\det C} \\ D &= \sqrt{c_{11} + c_{22} + 2G} \\ G &= \sqrt{\frac{\beta I(t) S(t)}{N} \gamma I(t)}. \\ D &= \sqrt{2 \frac{\beta I(t) S(t)}{N} + \gamma I(t) + 2G} \end{aligned}$$

Table 2: Parameters for numerical simulations

Parameters	Definition	Parameter value	Source
β	Transmission parameter	2	estimated



γ	Fixed probability for recovery	0.1429	[2, 15]
c	Proportion vaccinated before start of outbreak	0-0.95	variable

3. Numerical Simulations and Results

In this section, we carry out numerical simulations of the models. The aim of these simulations is to demonstrate numerically the size of epidemics for different population sizes and initial vaccination regimes. To achieve this, the Euler algorithm was coded in MATLAB to integrate the models. The values of our model parameters are based on published epidemiological data shown in Table 2.

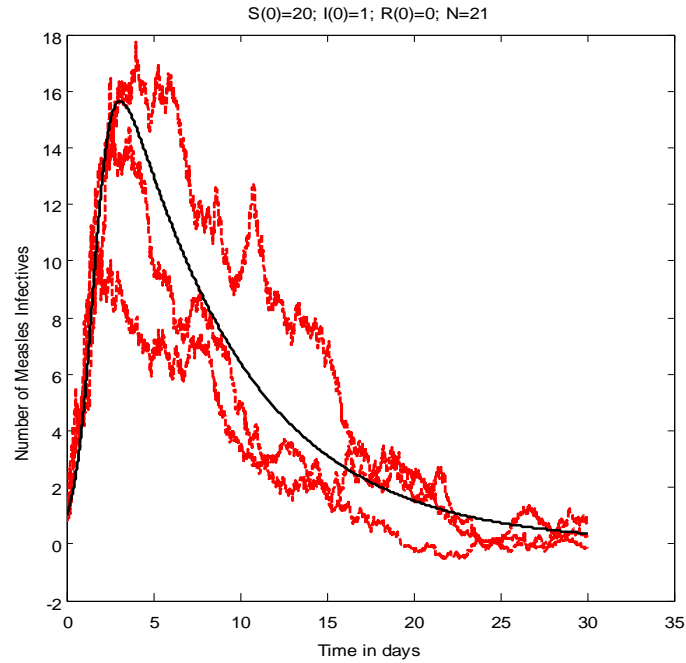


Figure 2: No initial vaccination regime, $R_0 = 14$

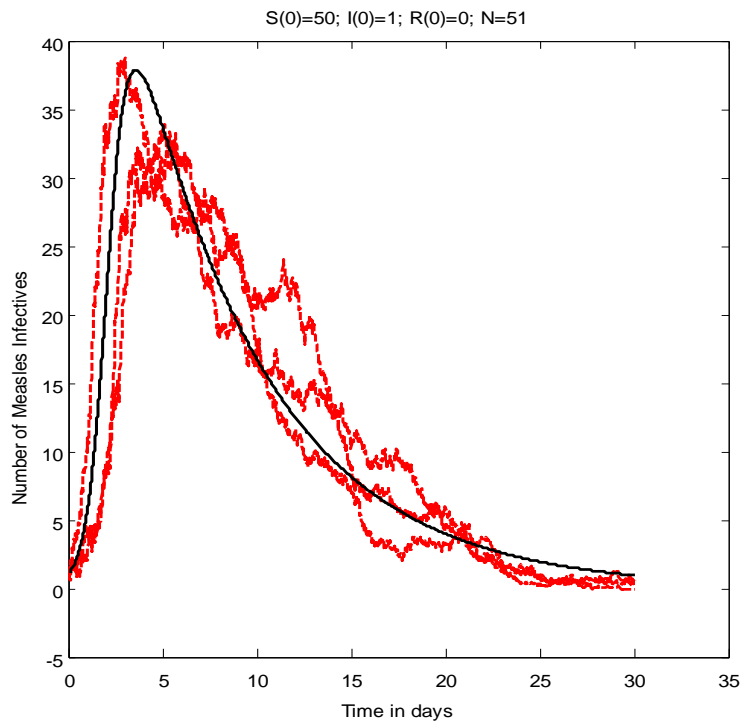


Figure 3: No initial vaccination regime, $R_0 = 14$

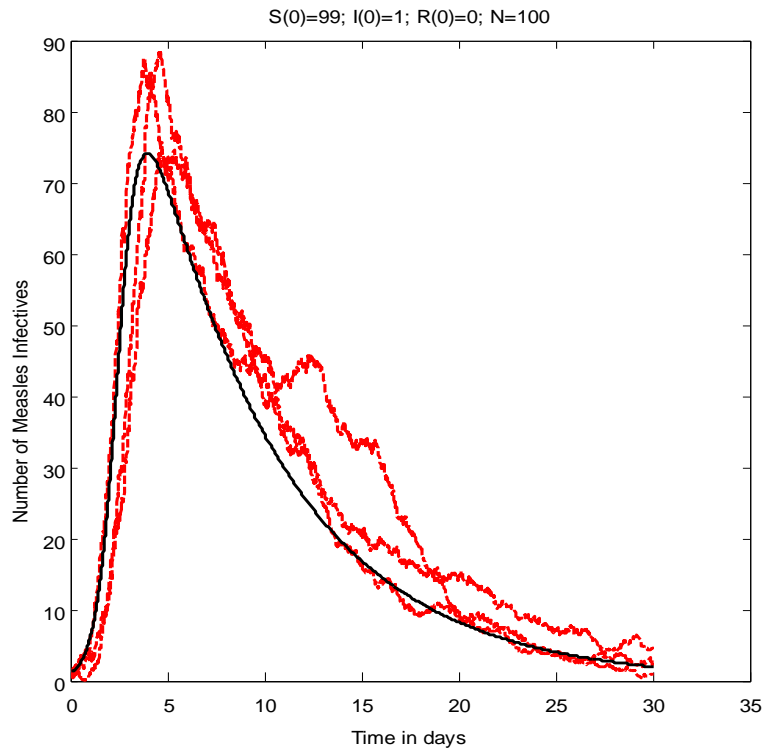


Figure 4: No initial vaccination regime, $R_0 = 14$

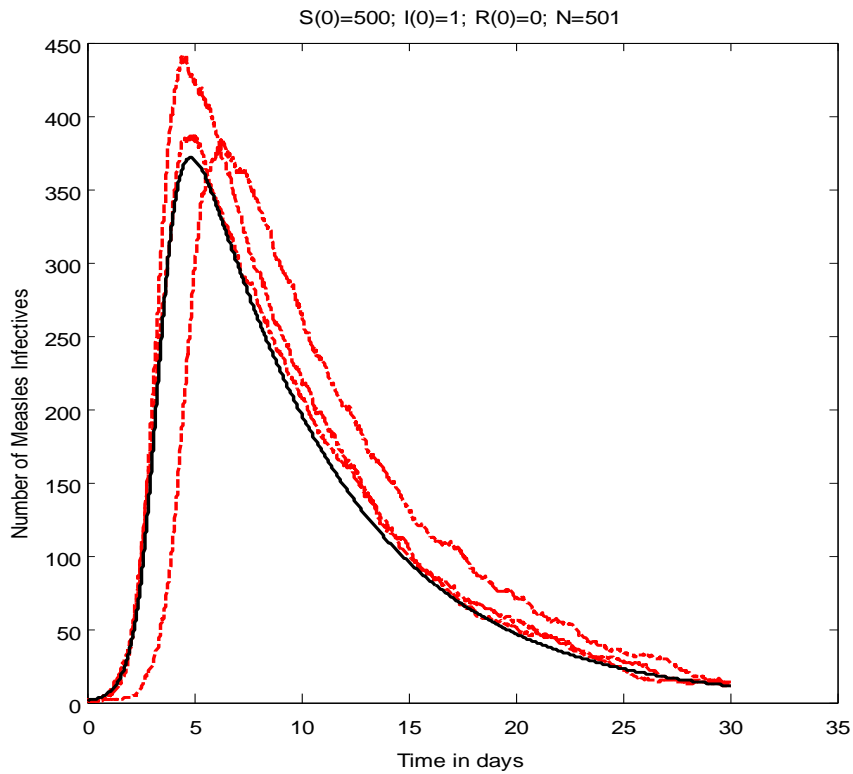


Figure 5: No initial vaccination regime, $R_0 = 14$

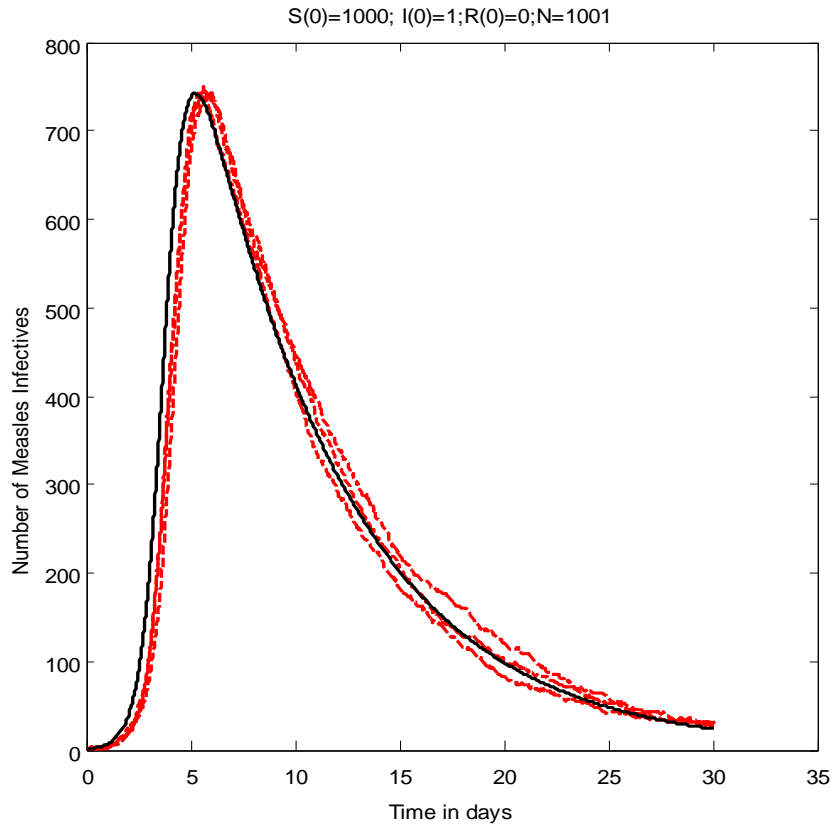


Figure 6: No initial vaccination regime, $R_0 = 14$

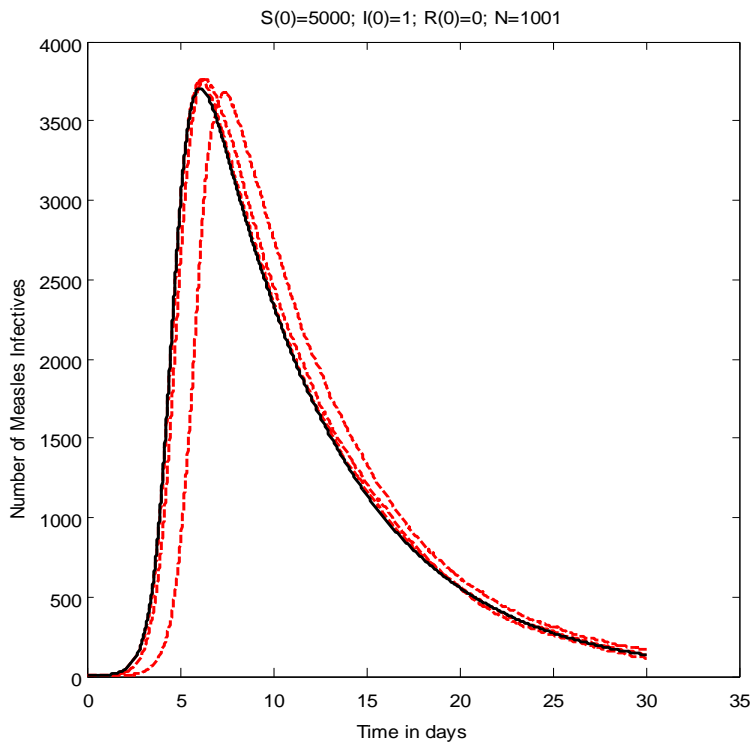


Figure 7: No initial vaccination regime, $R_0 = 14$.

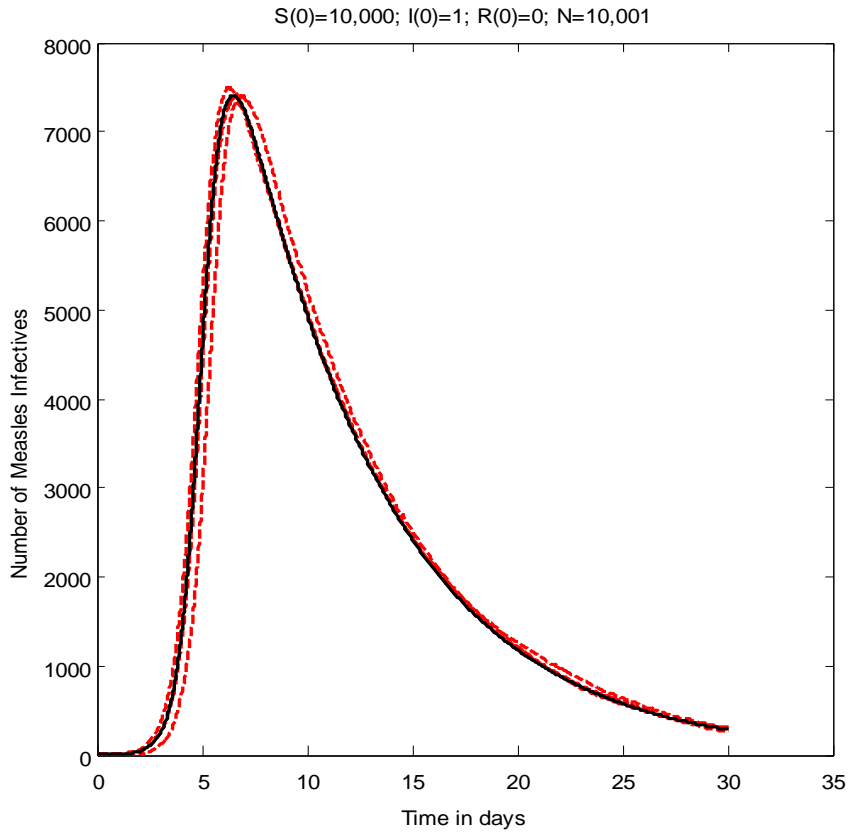


Figure 8: No initial vaccination regime, $R_0 = 14$.

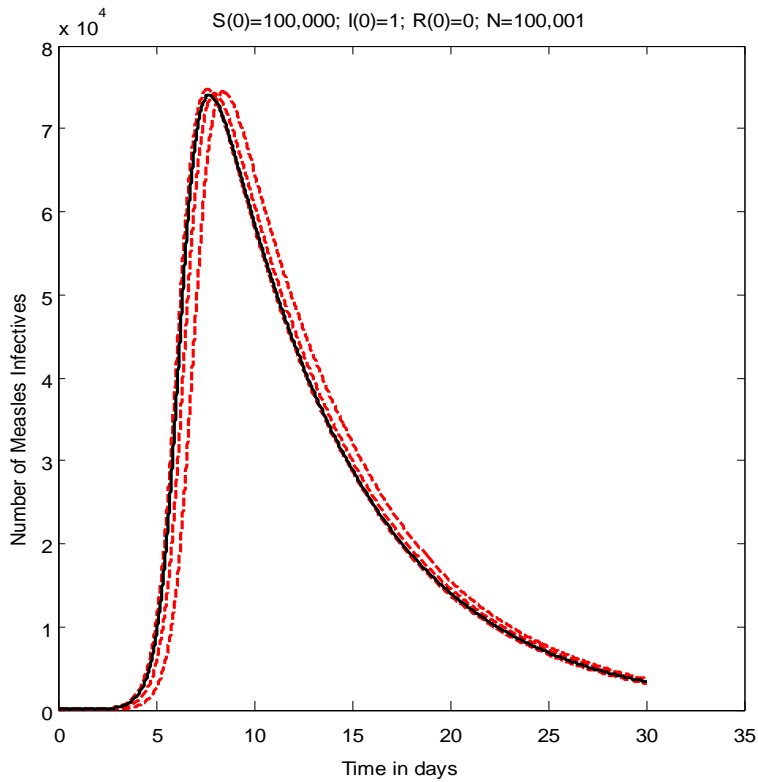


Figure 9: No initial vaccination regime, $R_0 = 14$

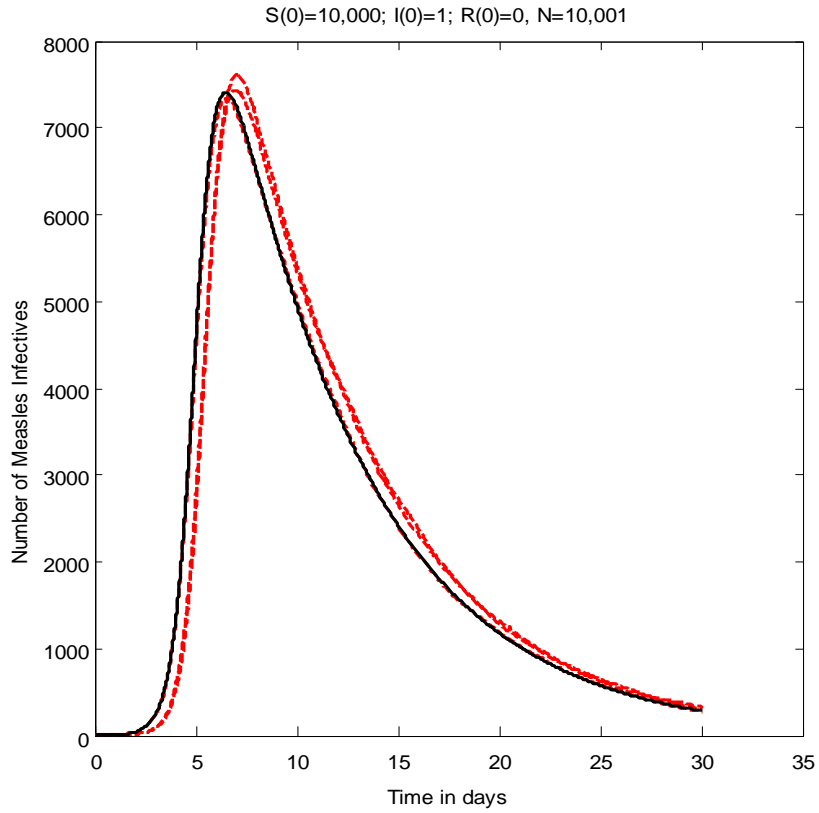


Figure 10: No initial vaccination regime, $R_0 = 14$

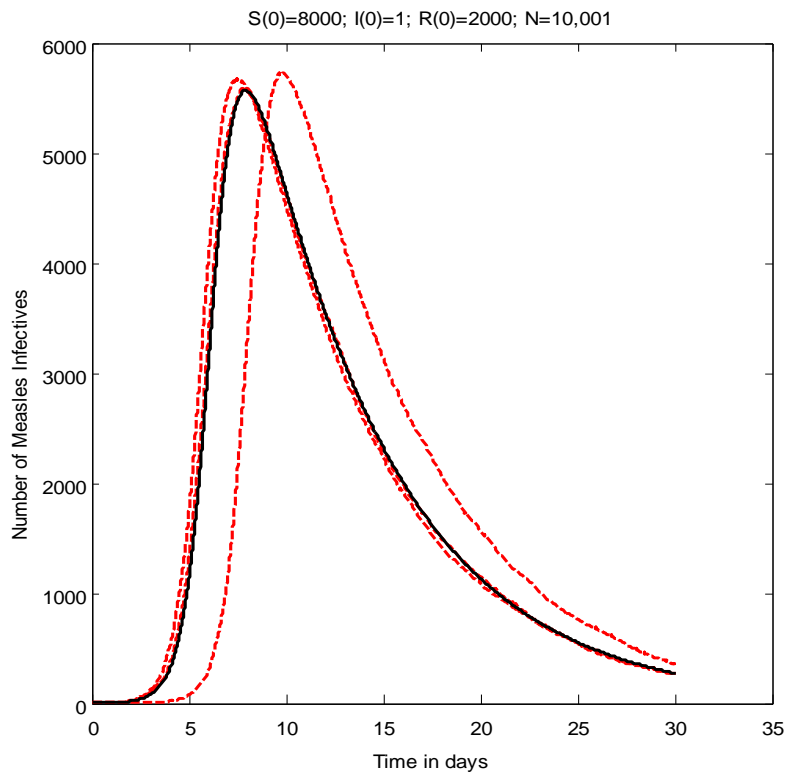


Figure 1: 20% initially vaccinated, $R_0(1 - c) = 11$

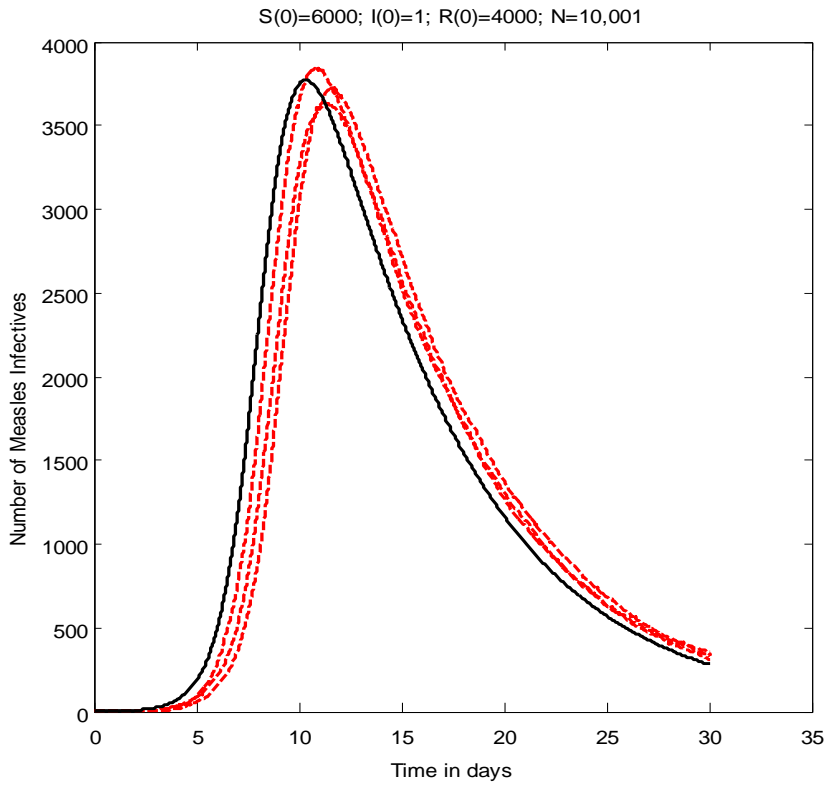


Figure 2: 40% initially vaccinated, $R_0(1 - c) = 8$.

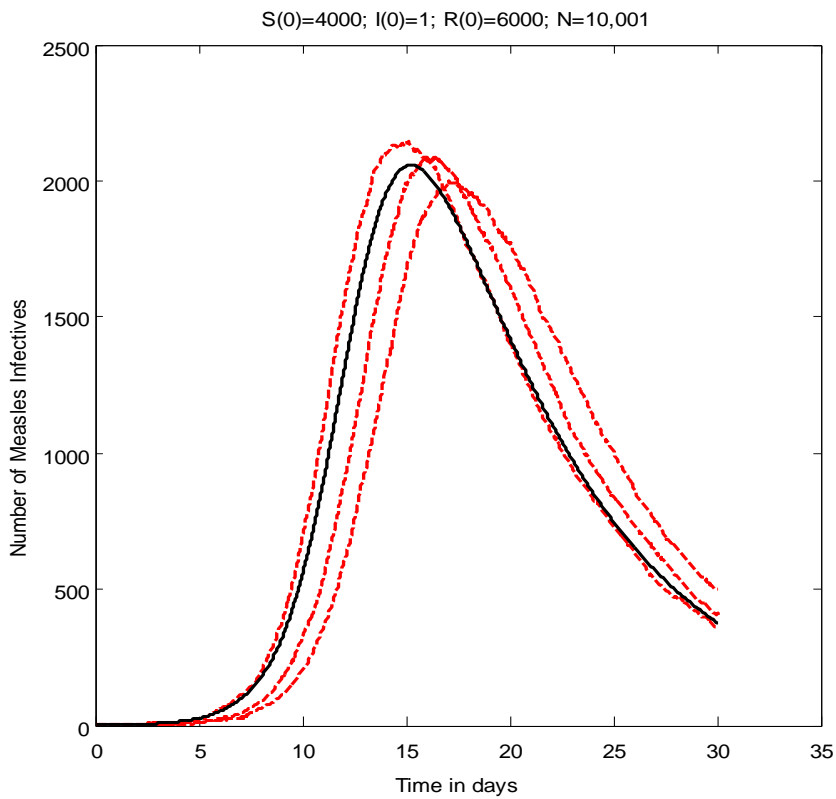


Figure 3: 60% initially vaccinated, $R_0(1 - c) = 5.6$

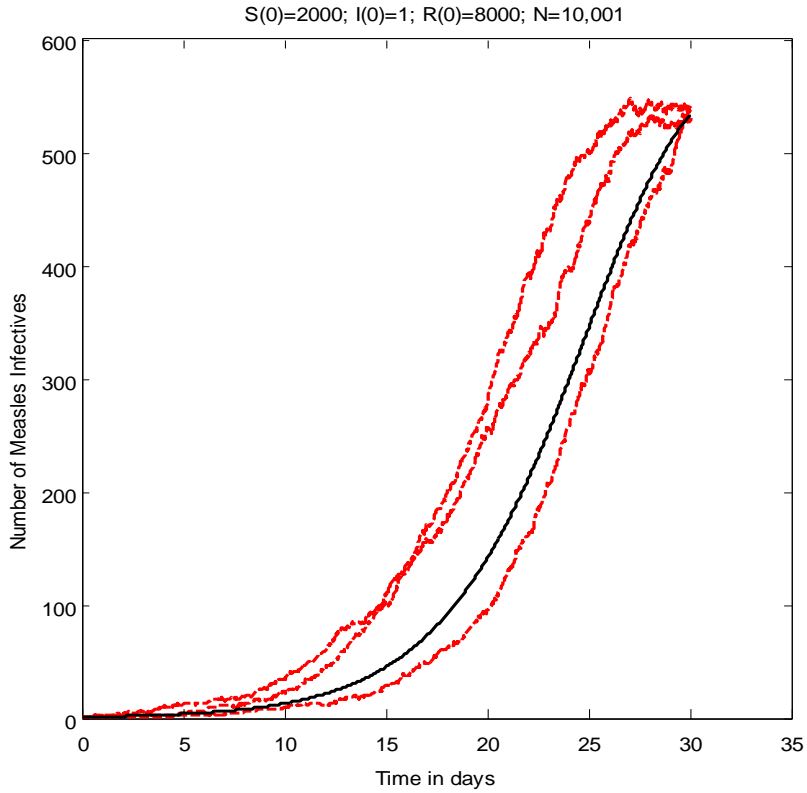


Figure 4: 80% initially vaccinated, $R_0(1 - c) = 2.8$.

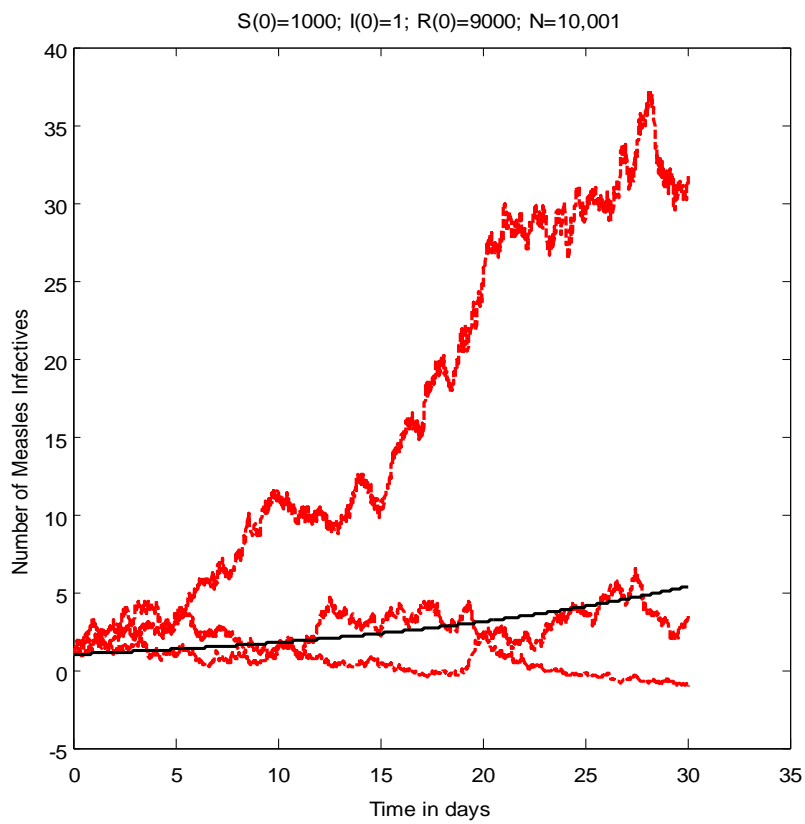


Figure 5: 90% initially vaccinated, $R_0(1 - c) = 1.4$.

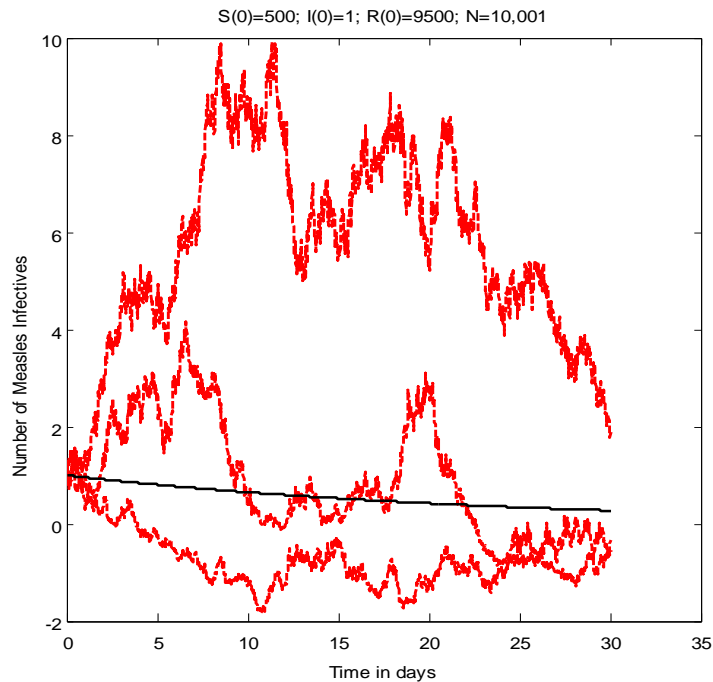


Figure 6: 95% initially vaccinated, $R_0(1 - c) = 0.7$.

4. Discussion

The results obtained from joint simulations of both deterministic and stochastic models of measles outbreaks are in two phases. In the first phase, results were obtained, considering different sizes of susceptible population. The results are shown in Figures 2 through 10. The results show that the size of measles epidemic increases with the size of the susceptible population. Another pointer from these results is that the two models exhibit strong components of stochasticity for smaller population sizes. This can be seen in Figures 2 through 5. However, the models demonstrate weak stochasticity for larger populations. See Figures 6 through 11. Thus, for larger populations, deterministic model is a good approximation of the stochastic counterpart.

In the second phase, results were obtained, considering the proportion of susceptible individuals that were already immunized before the outbreak. This can be seen in Figures 11 through 16. The results show that the size of outbreak decreases with the proportion of susceptible individuals initially immunized.

5. Conclusion

In this paper, deterministic and stochastic models of measles outbreak are presented. The models were solved numerically, considering, first, the size of the susceptible population and secondly, the effects of initially vaccinated individuals. The results show that stochastic model is more robust for smaller population sizes, and the deterministic model is a good approximation for larger population sizes. The results further stress that effective vaccination before the take off of an outbreak is crucial to the control of epidemic.

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