
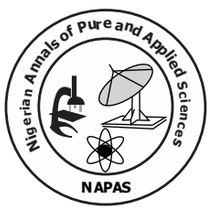

Deterministic and Stochastic Models of the Transmission Dynamics of Chicken Pox.

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Abstract

In this work a deterministic and stochastic model is developed and used to investigate the transmission dynamics of chicken pox. The models involve the Susceptible, Vaccinated, Exposed, Infectious and Recovered individuals. In the deterministic model the Disease free Equilibrium is computed and proved to be globally asymptotically stable when $R_0 < 1$.The deterministic model is transformed into a stochastic model which was solved using the Euler Maruyama method. Numerical simulations of the stochastic Model show that as the vaccine rate wanes, the number of individuals susceptible to the chicken pox epidemic increases.

Introduction

Before routine immunization was introduced the number of cases occurring each year was similar to the number of people born. But immunization has helped to reduce number of infections recorded.

In 2015, chicken pox resulted in 6400 deaths globally down from 8900 in 1990.Death occurs in about 1 per 60,000 cases. There is need therefore to develop appropriate mathematical model that can help to address the trend of the disease and suggest possible ways of combating the epidemic.

The chicken pox vaccine is very safe and effective and generally has few side effects. Its side effects includes: mild reactions such as fever, redness or swelling at the injection site. Two doses of the vaccine are about 90% effective at preventing chicken pox.

Individuals are encouraged to get vaccinated in order to protect yourself and others in the community who cannot get vaccinated such as those with weakened immune systems or pregnant women. People that have been vaccinated can still be infected with the disease but it is usually milder with fewer blisters, little or no fever. Chicken pox vaccine should be given to children, first dose at 12 months through 15months old and second dose at 4 through 6 years old. As in any viral infection, chicken pox-infected patients should be encouraged to drink plenty of water, fruit juices, tea or lemonade. These will be used to replace fluids lost through sweat and heat during febrile episodes. Feed chicken pox patients with mashed vegetable, soup, pilaf, mashed meat, perris and food prepared using steam cooking. You may also give the patient yoghurt, bananas, apples and carrots lynx. Children with chicken pox need more rest to recover. Usually the child can safely return to school after 7-10 days after the fever and the rash have disappeared. However, a medical consultation may be necessary to diagnose the exact state of the episode.

A lot of mathematical model have been developed to understand the dynamics of infectious diseases. London and Yorke (1973) stated that recurrent outbreaks of measles, chickenpox and mumps in cities were studied using a mathematical model of ordinary delay differential equations. They estimated the mean contact rate from the monthly reported cases over a 30- to 35-year period. The mean monthly contact rate for each disease was 1.7 to 2 times higher in the winter months than in the summer months. They showed that the seasonal variation was attributed primarily to the gathering of children in school. The two-year period of chicken pox outbreaks was the signature of an endemic infectious disease that would exhaust itself and become non-endemic if there were a minor increase in infectivity or a decrease in the length of the incubation period. Allen *et. al.*, (1991) considered a discrete-time, age-independent Susceptible-Infected-Recovered (*SIR)* type epidemic model of chicken pox. They verified three mathematically important properties for the model. Their solutions were non-negative, the population size was time-invariant, and the epidemic concluded with all individuals either remaining susceptible or becoming immuned. Lloyd (2001) used mathematical model to study the epidemiology of childhood viral diseases, such as chicken pox. He described the period of infectiousness by an exponential distribution. He used Susceptible Infectious Recovered (*SIR*) model in his study. He observed that less dispersed distributions were seen to have two important epidemiological consequences. First, less stable behaviour was seen within the model: incidence patterns became more complex. Second, disease persistence was diminished: in models with a finite population, the minimum population size needed to allow disease persistence increased. The assumption made concerning the infectious period distribution was of a kind routinely made in the formulation of mathematical models in population biology. He detected that a major effect on the central issues of population persistence and dynamics were observed. The results of his study have broad implications for mathematical modelers of a wide range of biological systems. Grenfell (1992) examined the impact of seasonality and chaotic dynamics in simple models for the population dynamics of chicken pox on the probability of fade-out of infection. His results indicated a significant degree of fade-out of infection, which was not consistent with previously derived criteria for the persistence of chicken pox. A simple non-linear analysis of the simulated series was presented, and the epidemiological implications of those results were discussed. According to Zaman *et.*

al. (2007), almost all mathematical models of diseases start from the same basic premise. The population could be subdivided into a set of distinct classes dependent upon experience with respect to the relevant disease. They used Susceptible Infected Recovered (*SIR*). In their paper, they described an *SIR* epidemic model with three components; *S*, *I* and *R*. They described their study of stability analysis theory to find the equilibrium for the model. In order to achieve control of the disease, they considered a control program relative to the *SIR* model. A percentage of the susceptible population was vaccinated in that model. They showed that an optimal control exists for the control problem and they used *Runge-Kutta* fourth order procedure to describe the numerical simulations. Their results were consistent with a system that was driven by an oscillation in the transmission parameter(Duncan *et. al.,* 1999).Infectious diseases provide a particularly clear illustration of the spatiotemporal underpinnings of consumer-resource dynamics. The paradigm was provided by extremely contagious, acute, immunizing childhood infections. Partially synchronized, unstable oscillations were punctuated by local extinctions. That, in turn, could result in spatial differentiation in the timing of epidemics and, depending on the nature of spatial contagion, might result in travelling waves. They used the basis of a gravity coupling model and a Time series Susceptible Infected- Recovered (TSIR) model for local dynamics. They proposed a meta-population model for regional chicken pox dynamics. Their model could capture all the major spatiotemporal properties in pre-vaccination epidemics of chicken pox in England and Wales (Yingcun *et. al*., 2004). Earn *et al*. (2000) showed that dramatic changes in patterns of epidemics had been observed throughout the twentieth century. They observed that for childhood infectious diseases such as chicken pox, the major transitions were between regular cycles and irregular, possibly chaotic epidemics and from regionally synchronized oscillations to complex, spatially incoherent epidemics. Lloyd (2000) illustrated how detailed dynamical properties of a model might depend in an important way on the assumptions made in the formulation of the model. According to his study most mathematical models used to understand the dynamical patterns seen in the incidence of childhood viral diseases, such as chicken poxs, employ a simple, but epidemiologically unrealistic, description of the infection and recovery process. The inclusion of more realistic descriptions of the recovery process was shown to cause a significant destabilization of the model. When there was seasonal variation in disease transmission that destabilization leads to the appearance of complex dynamical patterns with much lower levels of seasonality than previously predicted. Trottier and Philippe (2001) also presented a deterministic model as applied to the population dynamics of infectious diseases. They used SEIR deterministic model to provide useful insights into the mechanic of many common childhood diseases such as chicken pox. They showed that deterministic models exhibit damped oscillations, showed random variations and predicted the spread of infectious diseases. Tarwater and Martin (2001) evaluated the effect of population density on the epidemic outbreak of chicken pox. They used average-number contacts with susceptible individuals per infectious individual as a measure of population density, an analytical model for the distribution of the non-stationary stochastic process of susceptible contact was presented. They used a 5-dimensional lattice simulation model of disease spread to evaluate the effects of four different population densities. Analysis of the simulation results identified a decrease in a susceptible contact rate from four to three, resulted in a dramatic effect on the distribution of contacts over time, the magnitude of the outbreak, and, ultimately, the spread of the disease. Keeling and Grenfell (2002) revealed that the use of constant infectious and incubation periods, rather than the more convenient exponential forms, had been presented as a simple means of obtaining realistic persistence levels. They considered the persistence of chicken pox: reconciling theory, simulation and observation. They used a deterministic approach to parameterize a variety of models to fit the observed biennial attractor that determined the level of seasonality by the choice of model. Wallinga*et al*. (2005) estimated the chicken pox reproduction ratio for eight Western European vaccination programmes.

Since many plausible age-structured transmission patterns results in a similar

description of the observations, it is not possible to estimate a unique value of the reproduction ratio. They developed a method to estimate bounds and confidence intervals for plausible values of the reproduction ratios. Trottier and Philippe (2006) presented univariate time series analysis of pertussis, mumps, measles and rubella based on Box-Jenkins or Auto-Regressive Integrated Moving Average (ARIMA) modeling. The objective of their paper was to analyze the stochastic dynamics of childhood infectious disease using time series analysis. Their method, which enables the dependency structure embedded in time series data to be modelled, had potential research applications in studies of infectious disease dynamics.

Siabouh and Adetunde (2013) developed a deterministic SEIR model for the study of chicken pox in cape Coast Metropolis, Ghana. Data from the Central Region Hospital, Cape Coast were used to analyze the rate of chicken pox infection in the metropolis. They observed that the population of infected individuals at the beginning rise sharply as the contact rate increases and then fall uniformly as time increases. Garnett *et al* (1992) were the first to explore the relationship between varicella and zoster using mathematical models; they tested the impact of vaccination on long term equilibrium incidence of the diseases. They ignored the short term medium. Poletti *et al.*, (2013) developed a mathematical model on perspective of the impact of varicella immunization on herpes zoster .Their model was a multi country of VZV transfer and reactivation. They used the model to examine the impact of varicella vaccination on herpes zoster epidemiology in Italy, Finland and Uk.

Model Formulation

The Deterministic Model Formation.

We formulate a model of chicken pox with vaccination. In this model all the newborns are assumed to be susceptible or vaccinated. The population is divided into five compartments $S(t), V(t), E(t), I(t)$ and $R(t)$ known as Susceptible, Vaccinated, Exposed, Infectious and Recovered individuals where t represents time.The model for the dynamics of chicken pox is described by the following system of non –linear ordinary differential equation (The parameters are described in Table 1.

$$\left.\begin{aligned}
\frac{dS}{dt} &= (1-\rho)\pi + \eta V - \lambda S - \mu S \\
\frac{dV}{dt} &= \rho\pi - \lambda(1-\xi)V - \eta V - \mu V \\
\frac{dE}{dt} &= \lambda S + \lambda(1-\xi)V - (\alpha+\mu)E \\
\frac{dI}{dt} &= \alpha E - (\mu+\delta+\sigma)I \\
\frac{dR}{dt} &= \sigma I - \mu R
\end{aligned}\right\} \quad (1)$$

Table 1: Model parameters and their description

Parameters	Description
π	Recruitment rate
ρ	Fraction of vaccinated individuals
β	Contact rate
α	Rate of progression to an infectious stage
σ	Recovery rate
μ	Natural death rate
δ	Disease induced death rate
η	Waning vaccine rate.

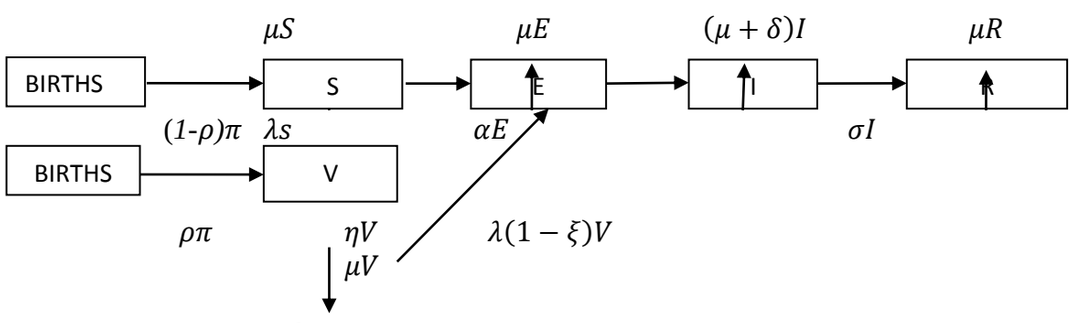

Figure 1: Flow diagram for the SVEIR Model

Assumptions of the SVEIR Model

i. susceptible individuals are equally likely to be infected by the infectious individuals in a case of contact including those who are vaccinated.
ii. Recovered individuals are permanently immune
iii. Some newborns are vaccinated at birth while some are not.
iv. Those in each class can die as a result of natural death.
v. vaccinated individuals get infected at a reduced rate $(1-\xi)\lambda$, compared to the susceptible individuals where ξ is the chicken pox vaccine efficacy.
vi. there is a tendency that those in the infected class can die as a result of chicken pox.(ie chicken pox induced death can occur).

Analysis of the Model

In this chapter we analyze the chicken pox deterministic and stochastic models, we first prove that the set of solution is confined in a feasible region and then show that all the solutions are positive. We investigate the existence of the equilibrium point. Further we computed the basic reproduction number. We also proved global stability of the disease free equilibrium (DFE) using the lyapunov function. Finally we considered the numerical solution of the model using simulation.

Basic Properties of the Deterministic Model

Invariant Property

Using the same procedure in Omame *et. al.*,(2018), the following result can be established:

Theorem 1: The closed set $D = \{(S,E,V,I,R) \in R^5{}_+ : S+E+V+I+R \geq 0\}$ is positively invariant and attracting with respect to the model (1).

Positivity of Solution.

Theorem 2 (Lakshmikantham, 1989): The solutions of the chicken pox model with positive initial values in the feasible region D remains positive at all time$t > 0$.

Using the first modeling procedure developed by Allen *et. al.*, (2008), we derive the stochastic model for the deterministic model (1) above

The drift vector is defined as

$$\vec{f} = \sum_{j=1}^{12} P_{i\vec{\lambda}_j}, \qquad (2)$$

Where $\overrightarrow{\lambda_j}$ and $p_j$are the random changes and the transition probabilities defined in the table 2.

Table2: Table of Transition Probabilities

Change	Probability	Event
$[1\ 0\ 0\ 0\ 0]^T$	$p_1 = (1-\rho)\pi\ \Delta t$	Birth of an unvaccinated susceptible
$[0\ 1\ 0\ 0\ 0]^T$	$p_2 = (\rho)\pi\ \Delta t$	Vaccination of a susceptible individual
$[1\text{-}1\ 0\ 0\ 0]^T$	$P_3 = \eta V \Delta t$	Vaccinated susceptible loses immunity
$[0\ \text{-}1\ 0\ 0\ 0]^T$	$p_4 = \mu V\ \Delta t$	Vaccinated individual dies natural death
$[\text{-}1\ 0\ 0\ 0\ 0]^T$	$p_5 = \mu S\ \Delta t$	Susceptible dies natural death
$[\text{-}1\ 0\ 1\ 0\ 0]^T$	$p_6 = \lambda S\ \Delta t$	Susceptible becomes exposed
$[0\ 0\ \text{-}1\ 0\ 0]^T$	$p_7 = \mu E\ \Delta t$	Exposed dies natural death
$[0\ 0\ \text{-}1\ 1\ 0]^T$	$p_8 = \alpha E\ \Delta t$	Exposed becomes infected.
$[0\ 0\ 0\ \text{-}1\ 0]^T$	$p_9 = (\mu + \delta) I\ \Delta t$	Infected dies natural death due to the disease
$[0\ 0\ 0\ \text{-}1\ 0]^T$	$p_{10} = \sigma I\ \Delta t$	Infected becomes recovered.
$[0\ 0\ 0\ 0\ \text{-}1]^T$	$p_{11} = \mu R\ \Delta t$	Recovered dies natural death
$[0\ \text{-}1\ 1\ 0\ 0]^T$	$p_{12} = \lambda(1-\xi)V\ \Delta t$	Vaccinated becomes exposed.

Hence, the drift vector $\vec{f}$ of order 5×1, is given by

$$\begin{bmatrix} (1-\rho)\pi + \eta V - S(\mu+\lambda) \\ \rho\pi - V(\eta+\mu) - \lambda(1-\xi)V \\ \lambda S + \lambda(1-\xi)V - (\alpha+\mu)E \\ \alpha E - (\mu+\delta+\sigma)I \\ \sigma I - \mu R \end{bmatrix} \qquad (3)$$

The covariance matrix is defined as V=$\sum_{j=1}^{12} p_j\, \vec{\lambda}_j (\vec{\lambda}_j)^T$

and is given by

$$\begin{Bmatrix} (1-\rho)\pi+\eta V+\mu S+\lambda S & -\eta V & -\lambda S & 0 & 0 \\ -\eta V & \rho\pi+\eta V+\mu V+\lambda(1-\xi)V & -\lambda(1-\xi)V & 0 & 0 \\ -\lambda S & -\lambda(1-\xi)V & -\lambda S+\alpha E+\lambda(1-\xi)V & \mu E+\alpha E & 0 \\ 0 & 0 & -\alpha E & \alpha E+(\mu+\delta)I+\sigma I & -\sigma I \\ 0 & 0 & 0 & -\sigma \mathrm{I} & \sigma I-\sigma I \end{Bmatrix} \quad (4)$$

The stochastic model is therefore given by

$$\begin{Bmatrix} d\vec{X}(t)=\vec{f}(t,\vec{X}(t)dt+V^{\frac{1}{2}}(t,\vec{X}(t))d\vec{W}(t) \\ \vec{X}(0)=[X_1(0,X_2(0),X_3(0),X_4(0),X_5(0)]^T \end{Bmatrix}. \quad (5)$$

Existence and Uniqueness of solution for SDEs

The existence and uniqueness of solutions of SDEs is well established by Allen *et al* (1998). Hence we uphold that the Stochastic model has unique solution.

Discussion of Results

Numerical Simulations

Many phenomena of interest in biology that can be modelled by the use of diffusion processes satisfying a nonlinear stochastic differential equation are not easy to solve analytically, it is advantageous to proceed via computer simulations. There will be time when the physical system is too complicated for analytical modelling; in such a case, simulation would be an appropriate tool (Feldan and Valdez-Flores, 2010). We therefore solve the model numerically using the MATLAB software (see appendix). We set year as a unit of time. The natural mortality or natural death rate μ is postulated to be equal to the inverse of the life expectancy at birth, which is about 54.5 years in Nigeria (UNAIDS-WHO, 2015); that is$\mu = 1/54.5 = 0.018yr^{-1}$. The recruitment rate π controls the total population size, because$N = \pi/\mu$. We set$\pi = \mu \times 300yr^{-1}$, following Song, *et al (2002*). η is the vaccination rate and is taken to be 0.05 yr^{-1} (Moghadas *et al*(2003)). β is the rate at which susceptibles children becomes exposed and is taken to be 0.00002 yr^{-1} (Moghadas and Gumel, 2003). α is the rate at which an exposed individual becomes infectious per unit time and is taken to be 45.6 yr^{-1}, the infectious period for Africa (Moghadas and Gumel, 2003). σ is the rate at which an infectious individual recovers per unit time and is taken to be 7.50 yr^{-1}(Moghadas and Gumel, 2003). δ is the differential mortality due to chicken pox and is taken to be 0.5 yr^{-1} (Mohammed, *et al* (2010)). $S(0)$= 80, $V(0) = 20, E(0)$= 80, $I(0)$= 80, $R(0)$= 40. The initial populations were based on the real data for the outbreak of chicken pox in okujagu-ama a rural community in Rivers state,Nigeria between september and october 2017.A total of three hundred households were studied in the house to house search for cases in okujagu-ama village.

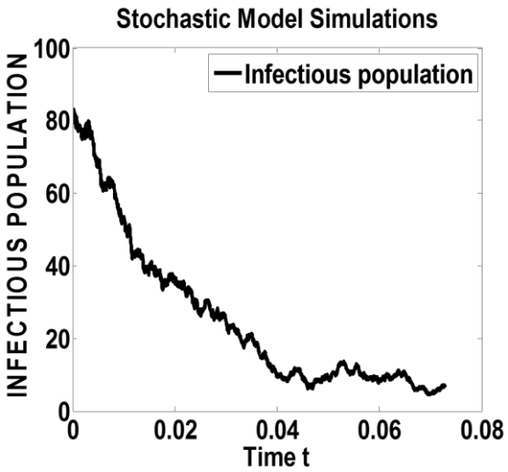

Figure 4.1: The stochastic plot of infectious population over time when the treatment rate $\sigma = 10.0$.

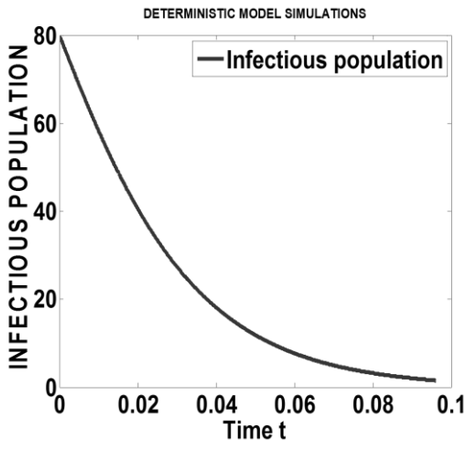

Figure 4.2: Deterministic plot of the infectious population over time when the treatment rate σ= 10.0.

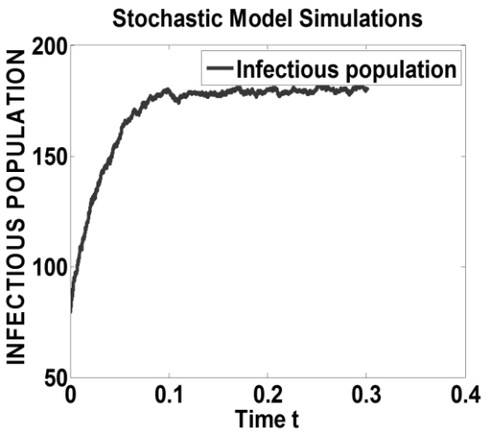

Figure 4.3: Stochastic plot of the infectious population over time when the treatment is rate is low σ= 1.0. (low treatment rate).

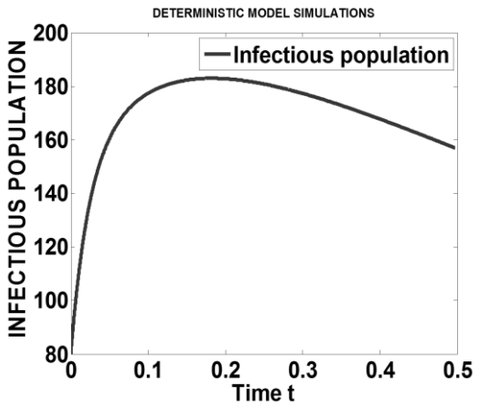

Figure 4.4: The deterministic plot of the infectious population over time when the treatment rate is low σ= 1.0.(low treatment rate).

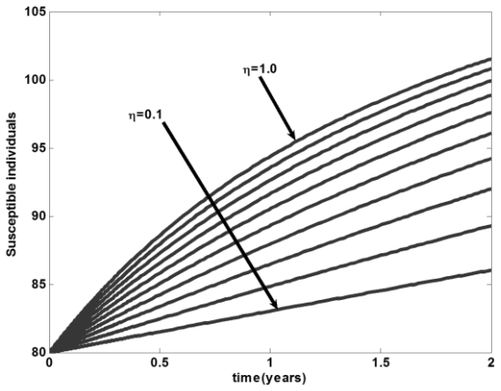

Figure 4.5: *The deterministic plot of the susceptible individuals over time at different values of vaccine waning rates.*

Discussion of Results

The simulations of the model (both deterministic and stochastic) using parameter values relevant to the outbreak of Chicken pox in Okujagu-Ama community in Okirika Local Government Area of Rivers State, Southern Nigeria is presented in the figures 1-5.

From Figure 4.1, we observe that when the reproduction number is less than one, the disease dies out with time, as expected (based on the epidemiological interpretation of the reproduction number)., though there is a random fluctuation as it the infectious population decreases with time.

In Figure 4.2, we observe that the infectious population, like in Figure 1, decreases over time since the treatment rate is very high. However, the random fluctuation is not observed in the deterministic plot.

Figure 4.3 shows the stochastic profile of the infectious population over time when the treatment rate is very low. It is observed from this graph, that the infectious population increases with time and there is a random fluctuation along the path as time increases. That is to say, there are moments when the infectious individuals decrease and then increase again over the years. There are other environmental factors that might have cause this to occur, a phenomenon that is captured only in a stochastic model plot.

The deterministic profile of the infectious population over time when the treatment rate is very low depicted in Figure 4.4, shows that the disease persists in the population over time.

Finally, the deterministic plot of the susceptible population over time, varying the waning vaccine rates, depicted in Figure 4.5, shows that as the waning vaccine rate increases,

the number of unvaccinated susceptible increases with time. This, if not checked leads to increase of the disease burden in the population. More people join the susceptible population since the susceptible individuals are more likely to be infected with the disease this will cause epidemic over time. Hence this model strongly recommends a vaccination for chicken pox which completely offers full protection against the disease in order to eliminate chicken pox from the population.

The study clearly points to the importance of adequate and proper treatment as well as the use of a perfect vaccine as the major tools in the global fight against chicken pox disease.

Conclusion

In this work a deterministic and a stochastic differential equation models is developed and investigated for the transmission dynamics of chicken pox epidemic. The model, which is a multidimensional diffusion process, includes susceptible individuals, vaccinated individuals, latent (exposed), infected and recovered individual. A deterministic model was formed and the resulting model was transformed into a stochastic differential equation model by applying the procedure proposed by Allen *et al* (2008). As most nonlinear Stochastic Differential Equations (SDEs) are not easy to solve analytically, Euler Maruyama Method for SDEs is used to solve and analyze the model with the aid of MATLAB software (see appendix). The result shows that increased vaccination rate will lead to chicken pox disease reduction and possible extinction. The result has also shown that proper and supervised treatment of the infectious individuals can cause a major reduction in chicken pox disease transmission and possible extinction.

Based on the result of our formulated model, the following steps should be taken into consideration to ensure eradication of measles in our country.

1. The National Health infectious diseases Control Programme should emphasize on the improvement in early detection of chicken pox cases so that the disease transmission can be minimized through treatment.
2. Not only should mass vaccination exercise be encouraged to cover the majority of the population whenever there is an outbreak of the disease but also, chicken pox prevention must be a public health priority.

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