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Stochastic model of measles transmission dynamics with double dose vaccination

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ABSTRACT

In this paper we developed a stochastic model of measles transmission dynamics with double dose vaccination. The total population in this model was sub-divided in to five compartments, namely Susceptible $S(t)$, Infected $I(t)$, Vaccinated first dose $V_1(t)$, Vaccinated second dose $V_2(t)$ and Recovered $R(t)$. First the model was developed by deterministic approach and then transformed into stochastic one, which is known to play a significant role by providing additional degree of realism compared to the deterministic approach. The analysis of the model was done in both approaches. The qualitative behavior of the model, like conditions for positivity of solutions, invariant region of the solution, the existence of equilibrium points of the model and their stability, and also sensitivity analysis of the model were analyzed. We showed that in both deterministic and stochastic cases if the basic reproduction number is less than 1 or greater than 1 the disease free equilibrium point is stable or unstable respectively, so that the disease dies out or persists within the population. Numerical simulations were carried out using MATLAB to support our analytical solutions. These simulations show that how double dose vaccination affect the dynamics of human population.

1. Introduction

Measles is one of the most acute viral infectious human diseases and can cause serious illness, life-long complications and death (Moss & Griffin, 2012). Measles is an acute, highly contagious viral disease caused by *paramyxovirus*. This virus is transmitted primarily by airborne spray to mucous in the upper respiratory tract and it can live in the nose and throat mucus of an infected person. It can be transmitted by direct contact with infected nasal transmission when an infected person cough or sneezes. Humans are the only natural hosts of measles virus. It can be divided into four stages of illness phases such as incubation**,** prodrome, rash and recovery phase (C (Center of Disease Co, 2008–2015). Complications of measles are more common among children younger than 5 years of age and adults 20 years of age and older. These include pneumonia, ear and sinus infections, mouth ulcers, persistent diarrhea, Otis, blindness, malnutrition, and brain damage (Ethiopian health and nutr, 2012).

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Measles is highly communicable, with greater than 90% secondary attack rates among susceptible persons. It is still a public health problem in many developing countries, particularly in parts of Asia and Africa. According to the WHO, more than 20 million people are affected by measles each year with more than 95% of measles deaths occur in developing countries especially with more than half of measles deaths occur in sub-Saharan Africa and this burden accounted for 15% of all under-five mortality (O (World Health Organiz, 2012). Measles vaccine is the best way to reduce the risk of contracting measles. It is safe, effective and inexpensive. Unvaccinated young children and pregnant women are at highest risk of measles and its complications, including death. Immunity conferred by vaccination against measles has been shown to persist for at least 20 years and is generally thought to be life-long for most individuals. Measles vaccine efficacy is expected to be 85% at 9–11 months of age and increases to 97% after a second dose given at greater than 12 months (O (World Health Organiz, 2012).

Treatment aims to ease symptoms until the body's immune system clears the infection. There is no specific medicine that kills the measles virus. For most cases, rest and simple measures to reduce a fever are all that are needed for a full recovery. People with measles need bed rest, fluids, and control of fever and pains, and antibiotics (C (Center of Disease Co, 2000–2010).

Mathematical modeling is the process of expressing real world phenomena using mathematical principles and formula. Mathematical Modeling can be classified into deterministic models and stochastic models based on certainty. Mathematical models have been applied to infectious diseases since the middle of the 20th century. Infectious diseases like measles have been analyzed by both deterministic and stochastic epidemiology models.

The deterministic approach has some limitations in the mathematical modeling transmission of an infectious disease. They are simple to analyses but give less information, hard to perform estimation because it is not probabilistic and also repeated simulation from identical model result to one realization. The stochastic model of a process describes the uncertainty about the process development. Uncertainty is generated by randomness, which is a characteristic feature of the evolution of universe, and by ignorance, which is a characteristic feature of mankind.

Thus, in order to describe uncertainty in a realistic way, the stochastic model must explicitly include both sources of uncertainty (Wolfgang et al., 2006). Stochastic models play a significant role in various branches of applied sciences including measles transmission dynamics, as they provide some additional degree of realism compared to their deterministic model (KermackMcKendrick, 1927).

Few essential researches have been done on the transmission dynamics of measles in the last decade. The study done by (Ochoche & Gweryina, 2014) performed a SIR mathematical model of measles with vaccination and two phases of infectiousness. Their study realized that the disease will certainly be eliminated if all susceptible are vaccinated. They therefore suggested that the measles vaccine should be made compulsory such that no child is allowed to enter school without evidence of at least two dose measles vaccination (Raymond, 2016). studied stochastic modeling of the transmission dynamics of measles with vaccination control. The study has shown the effectiveness of stochastic analysis in studying the dynamics of measles compared to deterministic analysis. Moreover, the study done by (Edward et al., 2015) performed a mathematical model for control and elimination of the transmission dynamics of measles. Elimination of measles requires maintaining the effective reproduction number less than 1, as well as achieving low levels of susceptibility. Simulations of variables of the model have been performed and sensitivity analysis of different parameters has been done using MATLAB. Additionally (Christopher, Ibrahim, & Shamaki, 2017), developed a mathematical model for the dynamics of measles under the combined effect of vaccination at the susceptible class, and administering measles drug therapy to screened infected individuals in the exposed class. The results of the numerical experiments revealed that eradicating measles is more efficient if susceptible individuals are vaccinated and followed by drug therapy to screened infected individuals in the exposed class. The study by (NigusiePurnachandra, 2017) also considered and investigated SEIR mathematical modeling and simulation study for the control and transmission dynamics of measles. Infected has been split into two: Infected catarrh, and infected eruption. Numerical simulation was conducted using ode 45 of MATLAB. Similarly (Abu & Okutachi, 2017), studied simulating deterministic and stochastic differential equation models of measles outbreak considering population size and initial vaccination regime. Numerical results reveal that the solutions of the stochastic model display strong stochastic components for small 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 (SowoleSangare et al., 2019). also studied on the existence, uniqueness, stability of solution and numerical simulations of a mathematical model for measles disease. They carried out the stability of the model, established the existence and uniqueness of the solution to the model. Runge-Kutta fourth order method was used to solve the model numerically. This was used to do a simulation of the model by using MATLAB to determine the best strategies to adopt in controlling the measles disease.

All the above studies have developed deterministic as well as stochastic mathematical models of measles transmission dynamics. But there is no research done so far focusing on stochastic model of measles transmission dynamics with double dose vaccination by partitioning first and second dose vaccinated classes to the best of the knowledge the authors. But we need to be sure about the mentioned gap.

The remaining part of the paper is organized as follows. Section 2 introduces model formulation and description about proposed measles model. In Section 3, analysis of the model is discussed. Section 4 discuss about numerical simulation of the model. Finally, section 5 contains discussion and conclusions.

2. Model description and formulation

The proposed model of measles transmission dynamics total population size is divided into five distinct sub-classes which are: Susceptible *S(t),*Vaccinated first dose $V_1(t)$, Vaccinated second dose V_2(t), Infected I(t) and Recovered *R(t).* Susceptible(S): individuals who have not yet been infected with the disease, but are susceptible to the disease, and so might become infected. Vaccinated first dose (V_1): individuals who have received first dose of vaccine. Vaccinated second dose (V_2): individuals who have received second dose of vaccine. Infected (I): individuals who have been infected with the disease and are capable of spreading the disease to susceptible. Recovered (R): individuals who have been infected and then recovered from the disease, those are not able to be infected again.

Model assumptions

1. The population is uniform and mixes homogeneously,
2. The latent period is not crucial for the susceptible-infective interaction, so the compartment of exposed is omitted,
3. The recruited newborns who received first dose of vaccine join the Vaccinated class, but recruited newborns who have not received first dose of vaccine join the susceptible class.
4. An infected individual makes contact and is able to transmit the disease,
5. There is no treatment failure if you receive first and second doses of vaccine, a patient will either recover or die.

Susceptible class is increased by recruited of newborn at a rate $\boldsymbol{\pi}$**,** and waning for first dose of vaccine at rate $\boldsymbol{\theta V_1}$**,** and decreased due to contact with infected at rate $\boldsymbol{\beta SI}$**,** individuals who receive first dose of vaccine to susceptible at rate **aS.** Infected class is increased by contact with susceptible class by rate $\boldsymbol{\beta SI}$ and recovered from the infected class at a rate $\boldsymbol{\alpha I}$**.** Recovered class is increased due to the recovered from the infected class at a rate $\boldsymbol{\alpha I}$ and receive second dose of vaccine to recover at rate $\boldsymbol{bV_2}$**.** Vaccinated first dose class is increased by the Vaccinated recruited of newborn at a rate **K,** receive first dose of vaccine to susceptible at rate **aS,** and decreased due to waning for first dose of vaccine to susceptible at rate $\boldsymbol{\theta V_1}$**,** receive first dose of vaccine to second dose of vaccine at a rate $\boldsymbol{cV_1}$**.** Vaccinated second dose class is increased by receive first dose of vaccine to second dose at a rate $\boldsymbol{cV_1}$ and decreased due to receive second dose of vaccine to recovery at rate $\boldsymbol{bV_2}$. In all sub-classes decreased due to natural death rate $\boldsymbol{\mu}$, and the disease death rate $\boldsymbol{\eta}$ for infected class only.

The total population size at time t is denoted by N (t) where $N(t) = S(t) + V_1(t) + V_2(t) + I(t) + R(t)$.

The dynamics of the measles disease can be depicted in Fig. 1 below.

2.2. Model formulations

From the above schematic diagram (Fig. 1) the following system of differential equations is obtained for the deterministic model of measles transmission with double dose vaccination.

$$\begin{cases} \dfrac{dS}{dt} = \pi + \theta V_1 - \beta SI - aS - \mu S \\ \dfrac{dV_1}{dt} = K + aS - \theta V_1 - cV_1 - \mu V_1 \\ \dfrac{dV_2}{dt} = cV_1 - bV_2 - \mu V_2 \\ \dfrac{dI}{dt} = \beta SI - \alpha I - \eta I - \mu I \\ \dfrac{dR}{dt} = \alpha I + bV_2 - \mu R \end{cases} \tag{1}$$

With initial condition

$$S(0) = S_0 \geq 0, V_1(0) = V_{10} \geq 0, V_2(0) = V_{20} \geq 0, I(0) = I_0 \geq 0, R(0) = R_0 \geq 0$$

Stochastic measles model formulation.

Let us now consider the model (1) with the perturbation on transmission parameter given by white noise. The use of white noise is a good hypothesis in this model since it is assumed that the transmission parameter oscillates randomly around some average value, due to sometime varying disturbances. In this way, if a stochastic perturbation is made on the transmission parameter then we obtain Itôs type stochastic deferential system.

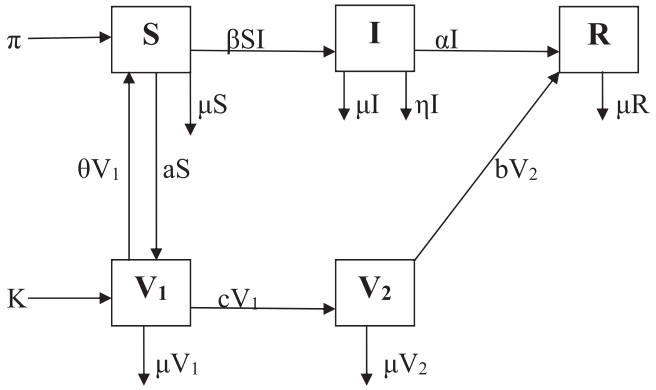

Fig. 1. The schematic model of measles transmission.

$$\begin{cases} dS = [\pi + \theta V_1 - \beta SI - aS - \mu S]dt + \rho_1 S dW_1 \\ dV_1 = [K + aS - \theta V_1 - cV_1 - \mu V_1]dt + \rho_2 V_1 dW_2 \\ dV_2 = [cV_1 - bV_2 - \mu V_2]dt + \rho_3 V_2 dW_3 \\ dI = [\beta SI - \alpha I - \eta \mathrm{I} - \mu \mathrm{I}]dt + \rho_4 I dW_4 \\ dR = [\alpha I + bV_2 - \mu R]dt + \rho_5 R dW_5 \end{cases} \tag{2}$$

with initial condition. $(S(0), V_1(0), V_2(0), I(0), R(0))^T = (S_0, V_{10}, V_{20}, I_0, R_0)^T \in R_+^5$ where $\rho_1, \rho_2, \rho_3, \rho_4, \rho_5$ *are*constants intensity fluctuations of each compartment and W_1, W_2, W_3, W_4, W_5 are the Brownian motions of each compartment.

3. Model analysis

In this section, the invariant region, positivity of solution, disease free equilibrium point, endemic equilibrium point, basic reproduction number, stability analysis and sensitivity analysis are discussed.

3.1. Invariant region

In this sub-section we obtained a region in which solutions of the models (1) and (2) are uniformly bounded in the proper subsetΩ of R^5.

Theorem 1. The feasible solution set $\Omega = (\mathrm{S}, V_1, V_2, \mathrm{I}, \mathrm{R})$ of the model (1) and (2) with initial conditions $S(0) = S_0 \geq 0, V_1(0) = V_{10} \geq 0, V_2(0) = V_{20} \geq 0, I(0) = I_0 \geq 0, R(0) = R_0 \geq 0$ *then* $0 \leq \mathrm{N} \leq \frac{\lambda}{\mu}\ \Omega = \left\{(\mathrm{S}, V_1, V_2, \mathrm{I}, \mathrm{R}) \varepsilon \mathrm{R}^5 : 0 \leq \mathrm{N} \leq \frac{\lambda}{\mu}\right\}$ *where* $\lambda = \pi + K$ is bounded region.

Proof. For the model in our consideration the total population is given by

$$N(t) = S(t) + V_1(t) + V_2(t) + I(t) + R(t) \tag{3}$$

Then differentiating N with respect to t and using equation (1) gives us;

$$\begin{aligned} &\frac{\mathrm{dN}}{\mathrm{dt}} = \frac{\mathrm{dS}}{\mathrm{dt}} + \frac{\mathrm{d}V_1}{\mathrm{dt}} + \frac{\mathrm{d}V_2}{\mathrm{dt}} + \frac{\mathrm{dI}}{\mathrm{dt}} + \frac{\mathrm{dR}}{\mathrm{dt}} \\ &\frac{dN}{dt} = \pi + K - \mu N - \eta \mathrm{I} \end{aligned} \tag{4}$$

In the absence of death due to measles or if there is no infected individual (i.e I=0), equation (4) becomes

$$\frac{dN}{dt} \leq \pi + K - \mu N, \text{ letting } (\pi + K) = \lambda$$

$$\int \frac{dN}{\lambda - \mu N} \leq \int dt \text{ Integrating both sides}$$

$$-\frac{1}{\mu}\ln(\lambda-\mu N)\leq t+C,\ \text{where C is a constant}$$

$$\ln(\lambda-\mu N)\geq -\mu(t+C)$$

$$e^{\ln(\lambda-\mu N)}\geq e^{-\mu(t+C)}$$

$$(\lambda-\mu N)\geq e^{-\mu t}.e^{-\mu C},\ \text{since } N_0=e^{-\mu C}\ \&\ \lim_{t\to\infty}(e^{-\mu t})=0$$

$$(\lambda-\mu N)\geq N_0.\ 0$$

$$N\leq\frac{\lambda}{\mu}$$

Hence, $0\ \leq\ N\ \leq\frac{\lambda}{\mu}$

Therefore, $\Omega=\left\{(S,V_1,V_2,I,R)\ \varepsilon R^5:0\leq N\leq\frac{\Lambda}{\mu}\right\}$ is positive invariant set for the system (1) and also for model (2) because stochastic model are deterministic model adding stochastic perturbations.

3.2. Positivity of solutions

In this sub-section, to obtain the solution of the model is non-negative we stated and proved the following theorem. To verify the model (1) to be epidemiologically meaningful and well posed we have to pave that all state variables are positive for all $t\geq 0$.

Theorem 2. *If* $S(0)\geq 0, V_1(0)\geq 0, V_2(0)\geq 0, I(0)\geq 0$ and $R(0)\geq 0$ *then the solution set* $\{S(t),V_1(t),V_2(t),I(t),R(t)\}$ *of the model* (1) *is positive for all* $t\geq 0$.

Proof. From the system of differential equation (1), let us take the first equation;

$$\Rightarrow\frac{dS}{dt}=\pi+\theta V_1-\beta SI-aS-\mu S$$

$$\frac{dS}{dt}\geq -(\beta I+a+\mu)S$$

$\frac{dS}{S}\geq -(\beta I+a+\mu)dt$ By using separating of variables and integrating both sides, we get$S(t)\geq S(0)e^{-(\beta I+a+\mu)t}>0$ *since* $e^C=S(0)$, *where C is a constant*

Hence, the proof holds if $S(t)\neq 0$ *for all* $t\geq 0$.

$V_1(t)\geq V_1(0)e^{-(\theta+c+\mu)t}>0$

$$V_2(t)\geq V_2(0)e^{-(b+\mu)t}>0$$

$$I(t)\geq I(0)e^{-(\mu+\eta+\alpha)t}>0$$

$$R(t)\geq R(0)e^{-(\mu)t}>0$$

Hence, the solution of $\{S,V_1,V_2,I,R\}$ for $t\geq 0$ in the region. Then we can deduce that the state variables of the system are all positive for all t>0.

3.3. Disease-free equilibrium point (DFEP)

In this sub-section we obtain the equilibrium point at which the epidemic is eradicated from the population. Letting all the right hand sides of (1) to zero and $I=R=0$, leads to

$$\begin{cases}\pi+\theta V_1^*-(a+\mu)S_0=0\\ K+aS_0-(\theta+c+\mu)V_1^*=0\\ cV_1^*-(b+\mu)V_2^*=0\end{cases}\tag{5}$$

by rearranging equation (5) and after substituting each other, we got,

$$S_0=\frac{\pi(\theta+c+\mu)+\theta K}{(\theta+c+\mu)(a+\mu)-\theta a}, V_1^*=\frac{K(a+\mu)+a\pi}{(\theta+c+\mu)(a+\mu)-\theta a}V_2^*=\frac{Kc(a+\mu)+\pi ac}{(b+\mu)[(\theta+c+\mu)(a+\mu)-\theta a]}$$

Therefore, DFE is given by,

$$(S_0,V_1^*,V_2^*,0,0)=\left(\frac{\pi(\theta+c+\mu)+\theta K}{(\theta+c+\mu)(a+\mu)-\theta a},\frac{K(a+\mu)+a\pi}{(\theta+c+\mu)(a+\mu)-\theta a},\frac{Kc(a+\mu)+\pi ac}{(b+\mu)[(\theta+c+\mu)(a+\mu)-\theta a]},0,0\right)$$

that represents the state in which there is no infection in the community.

3.4. Basic reproduction number($\boldsymbol{R}_0$)

In this sub-section we obtained basic reproduction numbers for deterministic as well as stochastic models.

3.4.1. Deterministic basic reproduction number ($\boldsymbol{R_o^D}$)

The deterministic model basic reproduction number $\boldsymbol{R_o^D}$ can be determined using the method of next-generation matrix (Diekmann & Heesterbeek, 2000). The basic reproduction number is the eigen-value of largest magnitude of the next generation matrix, that is, the number of all new infectious types in the next generation. $\boldsymbol{R_o^D}=\rho(FV^{-1})$

F be the rate of appearance of new infections in compartments I and V be the rate of transfer of individuals out of compartment I.

Based on system (1) one infectious compartment (m = 1) i.e mxm = 1 × 1 matrices, we may compute F and V as follow:

$$\frac{dI}{dt}=\beta SI-\mu\mathrm{I}-\eta\mathrm{I}-\alpha I \tag{6}$$

Note that equation (5) is made of one compartments I which are disease transmission. These are used for the determination of $\boldsymbol{R_o^D}$.

$$\mathrm{f}=\beta SI\ \ \mathrm{v}=(\mu+\eta+\alpha\ )\mathrm{I}$$

$$F=\frac{\partial f}{\partial I}=\beta S\ \ V=\frac{\partial v}{\partial I}=(\mu+\eta+\alpha\ )$$

$$\mathrm{F}_{\mathrm{DFE}(S_0,V_1^*,V_2^*,0,0)}=\beta S_0\ \text{Where}\ S_0=\frac{\pi(\theta+c+\mu)+\theta K}{(\theta+c+\mu)(a+\mu)-\theta a}$$

The next generation matrix of equation (6) is. $\boldsymbol{R_o^D}=FV^{-1}$

Thus, we need to find the spectral radius of FV^{-1}:

$$F=\beta S_0\ \ V^{-1}=\frac{1}{(\mu+\eta+\alpha\ )}$$

$$FV^{-1}=\frac{\beta S_0}{(\mu+\eta+\alpha\ )}=\frac{\beta S_0}{(\mu+\eta+\alpha\ )}$$

The Eigen-value of FV^{-1} can be obtain by. $\left|\frac{\beta S_0}{(\mu+\eta+\alpha\ )}-\lambda\right|=0$

Hence, with next generation matrix rule the largest Eigen-value of the next generation matrix is the basic reproduction number.

Therefore, ${R_o}^D=\frac{\beta S_0}{(\mu+\eta+\alpha\ )}$ Where. $S_0=\frac{\pi(\theta+c+\mu)+\theta K}{(\theta+c+\mu)(a+\mu)-\theta a}$

3.4.2. Stochastic basic reproduction number ($\boldsymbol{R_o^S}$)

From model system (2) we take the forth equation i.e

$$dI=[\beta SI-(\mu+\eta+\alpha)I]dt+\rho_4 IdW_4$$

The stochastic model basic reproductive number can be determined by using Ito's formula for twice differentiable function on [0, T], $f(I)=ln(I)$ its expansion in Taylor series is $df(t,I(t))=\frac{\partial f}{\partial t}dt+\frac{\partial f}{\partial \mathrm{I}}dI+\frac{1}{2}\frac{\partial^2 f}{\partial I^2}(dI)^2+\frac{\partial^2 f}{\partial \mathrm{t}\partial \mathrm{I}}dtdI+\frac{1}{2}\frac{\partial^2 f}{\partial t^2}(dt)^2$

$$dI\Big)\Big(dI\Big)(dI)^2=\Big(\beta SI-\Big(\mu+\eta+\alpha\Big)I\Big)^2(dt)^2+2(\beta SI-(\mu+\eta+\alpha)I)(\rho_4 I)dtdW_4+(\rho_4 I)^2(dW_4)^2$$

$$\textit{The partial derivatives are}:\ \frac{\partial f}{\partial t}=0\ ,\frac{\partial^2 f}{\partial t^2}=0\ ,\ \frac{\partial^2 f}{\partial t\partial \mathrm{I}}=0,\frac{\partial f}{\partial \mathrm{I}}=\frac{1}{\mathrm{I}}\ ,\ \frac{\partial^2 f}{\partial I^2}=-\frac{1}{I^2}$$

$$\Rightarrow df(t,I(t))=\frac{\partial f}{\partial t}dt+\frac{\partial f}{\partial \mathrm{I}}dI+\frac{1}{2}\frac{\partial^2 f}{\partial I^2}(dI)^2+\frac{\partial^2 f}{\partial t\partial \mathrm{I}}dt(dI)+\frac{1}{2}\frac{\partial^2 f}{\partial t^2}(dt)^2$$

$$\Rightarrow df(t,I(t))=(0)dt+\frac{1}{\mathrm{I}}([\beta SI-(\mu+\eta+\alpha)I]dt+\rho_4 IdW_4)-\frac{1}{2I^2}\Big((\beta SI-\quad \textit{tde is a pivotal concept in}\ (\mu+\eta+\alpha)I)^2(dt)^2$$

$$+2(\beta SI-(\mu+\eta+\alpha)I)(\rho_4 I)dtdW_4+(\rho_4 I)^2(dW_4)^2\Big)+(0)dt([\beta SI-(\mu+\eta+\alpha)I]dt+\quad \rho_4 IdW_4)+\frac{1}{2}(0)(dt)^2$$

$$\Rightarrow df(t,I(t))=([\beta S-(\mu+\eta+\alpha)]dt+\rho_4 dW_4)-\frac{1}{2}\Big({\rho_4}^2(dW_4)^2\Big)$$

The differentials of higher order (dt, dW) become fast zero; $(dt)^2\to 0$ *and* $dtdW(t)\to 0$. the stochastic term $\mathrm{dW}^2(t)$ according to the rules of Brownian motion is given as $\mathrm{dW}^2(t)=dt$.where for computing $(dI(t))^2$ we use the following properties

$$dt\Big)\Big(dt\Big)(dt)^2=0\ ;\ \ dtdW(t)=dW(t)dt=0;\quad \Big(dW\Big(t\Big)\Big)^2=dWdW=dt$$

$$dt\ \textit{for}\ (dW(t))^2\ \Big\{\textit{due to variance of a wiener process}\Big\}$$

$$\Rightarrow df(t,I(t))=\left(\left[\beta S-(\mu+\eta+\alpha)-\frac{1}{2}\Big({\rho_4}^2\Big)\right]dt+\rho_4 dW_4(t)\right)\tag{7}$$

$$\Rightarrow df(t,I(t))=\left[\beta S-\frac{1}{2}{\rho_4}^2-(\mu+\eta+\alpha)\right]dt+\rho_4 dW(t)$$

Using next generation matrix

$$DFE=(S_0,V_1^*,V_2^*,0,0)\quad where\ \ S_0=\frac{\pi(\theta+c+\mu)+\theta K}{(\theta+c+\mu)(a+\mu)-\theta a}$$

$$\mathrm{f}=\beta S-\frac{1}{2}{\rho_4}^2\qquad ,\mathrm{f}_{\mathrm{DFE}}=\beta S_0-\frac{1}{2}{\rho_4}^2$$

$$\nu=(\mu+\eta+\alpha)\qquad \nu^{-1}=\frac{1}{(\mu+\eta+\alpha)}$$

Hence.$R_o{}^S=f\nu^{-1}=\left(\beta S_0-\frac{{\rho_4}^2}{2}\right)*\left(\frac{1}{(\mu+\eta+\alpha)}\right)=\frac{\beta S_0}{(\mu+\eta+\alpha)}-\frac{{\rho_4}^2}{2(\mu+\eta+\alpha)}$

Therefore.$\mathrm{R_o}^{\mathrm{S}}=\mathrm{R_o}^{\mathrm{D}}-\frac{{\rho_4}^2}{2(\mu+\eta+\alpha)}$, since $\mathrm{R_o}^{\mathrm{D}}=\frac{\beta S_0}{(\mu+\eta+\alpha)}$

Note: $\mathrm{R_o}^{\mathrm{S}}<\mathrm{R_o}^{\mathrm{D}}$ The stochastic basic reproduction number is less than deterministic basic reproduction number, because the stochastic model version approaches to reality than deterministic model.

3.5. Local stability of disease-free equilibrium

3.5.1. Local stability of disease-free equilibrium of the deterministic model

The number of infected class I will tend to zero in the long run provided that $R_o{}^D<1$. If $R_o{}^D<1$ then disease-free equilibrium of the deterministic model is locally asymptotically stable.

Theorem 3. *The disease-free equilibrium* $DFE=(S_0,V_1^*,V_2^*,0,0)$ *of the system* (1) *is locally asymptotically stable if* $R_o{}^D<1$ *and unstable if* $R_o{}^D>1$.

Proof. We investigate the stability of the disease-free equilibrium $DFE=(S_0,V_1^*,V_2^*,0,0)$ by considering the system (1), taking the Jacobian matrix and obtained

$$J=\begin{bmatrix} \frac{\partial f_1}{\partial S} & \frac{\partial f_1}{\partial V_1} & \frac{\partial f_1}{\partial V_2} & \frac{\partial f_1}{\partial I} & \frac{\partial f_1}{\partial R} \\ \frac{\partial f_2}{\partial S} & \frac{\partial f_2}{\partial V_1} & \frac{\partial f_2}{\partial V_2} & \frac{\partial f_2}{\partial I} & \frac{\partial f_2}{\partial R} \\ \frac{\partial f_3}{\partial S} & \frac{\partial f_3}{\partial V_1} & \frac{\partial f_3}{\partial V_2} & \frac{\partial f_3}{\partial I} & \frac{\partial f_3}{\partial R} \\ \frac{\partial f_4}{\partial S} & \frac{\partial f_4}{\partial V_1} & \frac{\partial f_4}{\partial V_2} & \frac{\partial f_4}{\partial I} & \frac{\partial f_4}{\partial R} \\ \frac{\partial f_5}{\partial S} & \frac{\partial f_5}{\partial V_1} & \frac{\partial f_5}{\partial V_2} & \frac{\partial f_5}{\partial I} & \frac{\partial f_5}{\partial R} \end{bmatrix}, \text{ where } \begin{aligned} f_1 &= \pi+\theta V_1-\beta SI-aS-\mu S \\ f_2 &= K+aS-\theta V_1-cV_1-\mu V_1 \\ f_3 &= cV_1-bV_2-\mu V_2 \\ f_4 &= \beta SI-\alpha I-\eta \mathrm{I}-\mu \mathrm{I} \\ f_5 &= \alpha I+bV_2-\mu R \end{aligned}$$

$$J=\begin{bmatrix} -(\beta I+a+\mu) & \theta & 0 & -\beta S & 0 \\ a & -(\theta+c+\mu) & 0 & 0 & 0 \\ 0 & c & -(b+\mu) & 0 & 0 \\ \beta I & 0 & 0 & \beta S-(\alpha+\eta+\mu) & 0 \\ 0 & 0 & b & \alpha & -\mu \end{bmatrix}$$

The Jacobean matrix of the system (1) at disease free equilibrium is

$$J(S_0,V_1^*,V_2^*,0,0)=\begin{bmatrix} -(a+\mu) & \theta & 0 & -\beta S_0 & 0 \\ a & -(\theta+c+\mu) & 0 & 0 & 0 \\ 0 & c & -(b+\mu) & 0 & 0 \\ 0 & 0 & 0 & \beta S_0-(\alpha+\eta+\mu) & 0 \\ 0 & 0 & b & \alpha & -\mu \end{bmatrix}$$

The determinant is given by: $\left|J(S_0,V_1^*,V_2^*,0,0)-\lambda In\right|=0$

$$\Rightarrow \begin{vmatrix} -(a+\mu)-\lambda & \theta & 0 & -\beta S_0 & 0 \\ a & -(\theta+c+\mu)-\lambda & 0 & 0 & 0 \\ 0 & c & -(b+\mu)-\lambda & 0 & 0 \\ 0 & 0 & 0 & \beta S_0-(\alpha+\eta+\mu)-\lambda & 0 \\ 0 & 0 & b & \alpha & -\mu-\lambda \end{vmatrix}=0$$

$$\Rightarrow(-(a+\mu)-\lambda) \times \begin{vmatrix} -(\theta+c+\mu)-\lambda & 0 & 0 & 0 \\ c & -(b+\mu)-\lambda & 0 & 0 \\ 0 & 0 & \beta S_0-(\alpha+\eta+\mu)-\lambda & 0 \\ 0 & b & \alpha & -\mu-\lambda \end{vmatrix} - a \begin{vmatrix} \theta & 0 & -\beta S_0 & 0 \\ c & -(b+\mu)-\lambda & 0 & 0 \\ 0 & 0 & \beta S_0-(\alpha+\eta+\mu)-\lambda & 0 \\ 0 & b & \alpha & -\mu-\lambda \end{vmatrix}=0$$

$$\Rightarrow(-(a+\mu)-\lambda)\Bigg[-(\theta+c+\mu)-\lambda \begin{vmatrix} -(b+\mu)-\lambda & 0 & 0 \\ 0 & \beta S_0-(\alpha+\eta+\mu)-\lambda & 0 \\ b & \alpha & -\mu-\lambda \end{vmatrix} - a\theta \begin{vmatrix} -(b+\mu)-\lambda & 0 & 0 \\ 0 & \beta S_0-(\alpha+\eta+\mu)-\lambda & 0 \\ b & \alpha & -\mu-\lambda \end{vmatrix} \Bigg]=0$$

$$\Rightarrow(-(a+\mu)-\lambda)\left[(-(\theta+c+\mu)-\lambda)(-(b+\mu)-\lambda)\begin{vmatrix}\beta S_0-(\alpha+\eta+\mu)-\lambda & 0\\ \alpha & -\mu-\lambda\end{vmatrix}-a\theta(-(b+\mu)-\lambda)\right.$$
$$\left.\times\begin{vmatrix}\beta S_0-(\alpha+\eta+\mu)-\lambda & 0\\ \alpha & -\mu-\lambda\end{vmatrix}\right]=0$$

$$\Rightarrow(-(a+\mu)-\lambda)[(-(\theta+c+\mu)-\lambda)(-(b+\mu)-\lambda)-a\theta(-(b+\mu)-\lambda)]=0 \quad (8)$$

$$\text{Or}\begin{vmatrix}\beta S_0-(\alpha+\eta+\mu)-\lambda & 0\\ \alpha & -\mu-\lambda\end{vmatrix}=0 \quad (9)$$

when equation (8) it becomes

$$\Rightarrow(-(a+\mu)-\lambda)[(-(\theta+c+\mu)-\lambda)(-(b+\mu)-\lambda)-a\theta(-(b+\mu)-\lambda)]=0$$

$$(-(a+\mu)-\lambda)=0\Rightarrow\lambda_1=-(a+\mu)<0 \quad or$$

$$(-(b+\mu)-\lambda)[(-(\theta+c+\mu)-\lambda)-a\theta]=0$$

$$(-(b+\mu)-\lambda)=0\Rightarrow\lambda_2=-(b+\mu)<0 \quad or$$

$$(-(\theta+c+\mu)-\lambda)-a\theta=0\Rightarrow\lambda_3=-(\theta+c+\mu+a\theta)<0$$

$$\lambda_1<0,\lambda_2<0 \text{ and } \lambda_3<0$$

Then, equation (8) has strictly negative root and stable.
The determinant of equation (9) can be obtained,

$$\Rightarrow\begin{vmatrix}\beta S_0-(\alpha+\eta+\mu)-\lambda & 0\\ \alpha & -\mu-\lambda)\end{vmatrix}=0$$

$$\Rightarrow(\beta S_0-(\alpha+\eta+\mu)-\lambda)(-\mu-\lambda))=0$$

$$\Rightarrow\lambda^2+(2\mu+\alpha+\eta-\beta S_0)\lambda+\mu(\alpha+\eta+\mu-\beta S_0)=0$$

By using quadratic formula: $\lambda=\frac{-b\pm\sqrt{b^2-4ac}}{2a}$

Where, $a=1$, $b=2\mu+\alpha+\eta-\beta S_0$, $c=\mu(\alpha+\eta+\mu-\beta S_0)$

$$\Rightarrow\lambda=\frac{-(2\mu+\alpha+\eta-\beta S_0)\pm\sqrt{(2\mu+\alpha+\eta-\beta S_0)^2-4(\mu(\alpha+\eta+\mu-\beta S_0))}}{2}$$

$$\Rightarrow\lambda_4=\frac{-(2\mu+\alpha+\eta-\beta S_0)-\sqrt{(2\mu+\alpha+\eta-\beta S_0)^2-4(\mu(\alpha+\eta+\mu-\beta S_0))}}{2}<0 \text{ is stable}$$

$$\Rightarrow\lambda_5=\frac{-(2\mu+\alpha+\eta-\beta S_0)+\sqrt{(2\mu+\alpha+\eta-\beta S_0)^2-4(\mu(\alpha+\eta+\mu-\beta S_0))}}{2}$$

DFE to be stable, $b^2-4ac<0$ *and and* $ac>0$

$$ac>0\Rightarrow(1)(\mu(\alpha+\eta+\mu-\beta S_0))>0$$

$$(\mu+\eta+\alpha)>\beta S_0$$

$$\frac{(\mu+\eta+\alpha)}{(\mu+\eta+\alpha)}>\frac{\beta S_0}{(\mu+\eta+\alpha)}$$

$$\frac{\beta S_0}{(\mu+\eta+\alpha)}<1,$$

$$\Rightarrow {R_o}^D < 1$$

Therefore, If ${R_o}^D < 1$ then the DFEP of the deterministic model is locally asymptotically stable and also the eigen values of the Jacobian matrix evaluated at DFEP are strictly negative. This is the theorem.

3.5.2. Local stability of disease-free equilibrium point of the stochastic model

We provide a similar the stochastic extinction of infection for the stochastic model. The number of infected class I will tend to zero in the long run provided that ${R_o}^S < 1$. If ${R_o}^S < 1$ then disease-free equilibrium of the stochastic model is locally asymptotically stable.

Theorem 4. *If* ${R_o}^S < 1$, then for any initial value$(S(0), V_1(0), V_2(0), I(0), R(0)) = (S_0, V_1^*, V_2^*, 0, 0) \in R_+^5$, I (t) will tend to zero almost surely exponentially stable (i.e the disease dies out with probability one) of the system (2) is locally asymptotically stable and unstable if ${R_o}^S > 1$. That is, $\limsup_{t\to\infty} \frac{\ln I(t)}{t} < 0$ *or* $\lim_{t\to\infty} I(t) = 0$ as

Proof. By using equation (7)

$$\Rightarrow d\ln(I) = \left[\beta S - (\mu+\eta+\alpha) - \tfrac{1}{2}{\rho_4}^2\right]dt + \rho_4 dW_4(t)$$ *Integrating both sides on* [0,t] *we get*

$$\Rightarrow \ln(I) = \ln(I_0) + \int_0^t \left[\beta S - (\mu+\eta+\alpha) - \frac{1}{2}{\rho_4}^2\right]dt + \int_0^t \rho_4 dW_4(t)$$

$$\Rightarrow \ln(I) = \ln(I_0) + \left[\beta S - (\mu+\eta+\alpha) - \frac{1}{2}{\rho_4}^2\right]t + \int_0^t \rho_4 dW_4(t)$$

$$\Rightarrow \ln(I) \le \ln(I_0) + \left[\beta S - (\mu+\eta+\alpha) - \frac{1}{2}{\rho_4}^2\right]t + \int_0^t \rho_4 dW_4(t)$$

$$\Rightarrow \ln(I) \le \ln(I_0) + \left[\beta S - (\mu+\eta+\alpha) - \frac{1}{2}{\rho_4}^2\right]t + M(t)$$

$$\Rightarrow \frac{\ln(I(t)) - \ln(I(0))}{t} \le \left(\beta S - \mu - \eta - \alpha - \frac{1}{2}\rho_4^2\right) + \frac{M(t)}{t}$$

Let $M(t) = \int_0^t \rho_4 dW_4(s)$ *Then, M is a martingale* (Mao & Yuan, 2006), *with a quadratic variation given by*

$$< M, M >_t = \int_0^t \rho_4^2 ds = \rho_4^2 t$$

Since $\limsup_{t\to\infty} \frac{< M,M >_t}{t} = \rho_4^2 < \infty$ *by the strong law of large numbers, it follows that*

$$\limsup_{t\to\infty} \frac{M(t)}{t} = 0.$$

Thus, $\limsup_{t\to\infty} \frac{\ln I(t)}{t} = \left(\beta S - \mu - \eta - \alpha - \frac{1}{2}\rho_4^2\right)$

Now if ${R_o}^S < 1$ *we have* $\left(\beta S - \mu - \eta - \alpha - \frac{1}{2}\rho_4^2\right) < 0$

Thus, $\limsup_{t\to\infty} \frac{\ln I(t)}{t} = \left(\beta S - \frac{1}{2}\rho_4^2 - (\mu+\eta+\alpha)\right) < 0$ *since DFE* $= (S_0, V_1^*, V_2^*, 0, 0)$

$$= \left(\beta S_0 - \frac{1}{2}\rho_4^2 - (\mu+\eta+\alpha)\right) < 0$$

$$= (\mu+\eta+\alpha)\left(\frac{\beta S_0}{(\mu+\eta+\alpha)} - \frac{\rho_4^2}{2(\mu+\eta+\alpha)} - \frac{(\mu+\eta+\alpha)}{(\mu+\eta+\alpha)}\right) < 0$$

$$= \left(\mu+\eta+\alpha\right)\left(R_o{}^D - \frac{\rho_4^2}{2(\mu+\eta+\alpha)} - 1\right) < 0$$

$$= (\mu+\eta+\alpha)\left(R_o{}^S - 1\right) < 0$$

$$\leq \left(R_o{}^S - 1\right) < 0$$

Therefore.$R_o{}^S < 1$

3.6. Endemic equilibrium point (EEP)

In this sub-section we obtain the equilibrium point at which the disease persists in the community. The endemic equilibrium point (EEP) of system (1) is obtained by equating all equations of the model to be zero.

$$\begin{cases} \pi + \theta V_1 - \beta SI - aS - \mu S = 0 \\ K + aS - \theta V_1 - cV_1 - \mu V_1 = 0 \\ cV_1 - bV_2 - \mu V_2 = 0 \\ \beta SI - \alpha I - \eta I - \mu I = 0 \\ \alpha I + bV_2 - \mu R = 0 \end{cases} \tag{10}$$

By adding all the equation of system (10) we obtain

$$\pi + K - \mu N - \eta I = 0 \text{ where } N = S + V_1 + V_2 + I + R$$

$$\text{Hence}, I^* = \frac{\pi + K - \mu N}{\eta} \tag{11}$$

From the fourth equation in (10), we have

$$\beta SI - \alpha I - \eta I - \mu I = 0$$

$$(\beta S - \alpha - \eta - \mu) I = 0$$

$$\beta S = (\alpha + \eta + \mu)$$

$$\text{Hence}, S^* = \frac{(\alpha+\eta+\mu)}{\beta} \tag{12}$$

From the second equation in (10), we have

$$K + aS - \theta V_1 - cV_1 - \mu V_1 = 0$$

$$K + aS = (\theta + c + \mu)V_1, \text{ from (12)}$$

$$\text{Hence}, \ V_1^* = \frac{\beta K + a(\alpha+\eta+\mu)}{\beta(\theta+c+\mu)} \tag{13}$$

From the third equation in (10), we have

$$cV_1 - bV_2 - \mu V_2 = 0$$

$$cV_1 = (b+\mu)V_2, \text{ from (13)}$$

$$\text{Hence}, \ V_2^* = \frac{c\beta K + ca(\alpha+\eta+\mu)}{\beta(b+\mu)(\theta+c+\mu)} \tag{14}$$

From the fifth equation in (10), we have

$$\alpha I + bV_2 - \mu R = 0$$

$$\alpha I + bV_2 = \mu R, \text{ from (11) and (14)}$$

$$\text{Hence, } R^* = \alpha\left(\frac{\pi + K - \mu N}{\eta}\right) + b\left(\frac{c\beta K + ca(\alpha+\eta+\mu)}{\beta(b+\mu)(\theta+c+\mu)}\right) \tag{15}$$

Therefore, by (11),(12),(13),(14) and (15) system (1) has endemic equilibrium point. EEP $= \left(S^*, V_1^*, V_2^*, I^*, R^*\right) = \left(\frac{(\alpha+\eta+\mu)}{\beta}, \frac{\beta K + a(\alpha+\eta+\mu)}{\beta(\theta+c+\mu)}, \frac{c\beta K + ca(\alpha+\eta+\mu)}{\beta(b+\mu)(\theta+c+\mu)}, \frac{\pi+K-\mu N}{\eta}, \frac{\alpha(\pi+K-\mu N)}{\eta} + \frac{b(c\beta K + ca(\alpha+\eta+\mu))}{\beta(b+\mu)(\theta+c+\mu)}\right)$

Theorem 5. *If* $R_o^S > 1$, then for any initial value $(S(0), V_1(0), V_2(0), I(0), R(0)) \in \Omega$ I(t) will tend to zero exponentially almost surely. That is, $\lim_{t\to\infty} sup \frac{\ln I(t)}{t} > \phi$ almost surly. Where ϕ is a positive root of equation (7).

3.7. Sensitivity analysis of the model parameters

Sensitivity analysis is performed to determine the importance of each parameter to the transmission dynamics of measles disease. The analysis helps to measure the relative change in a variable when a parameter changes. Such information is very important to study transmission dynamics of the disease. The sensitivity index with respect to a parameter p is given by

$$P_{x_i} = \frac{\partial R_o^s}{\partial x_i} * \frac{x_i}{R_o^s}, \text{where is any parameter in } R_o^s, i = 1, 2, \ldots, 9$$

$$R_o^s = R_o^D - \frac{{\rho_4}^2}{2(\mu+\eta+\alpha)} = \frac{\beta S_0}{(\mu+\eta+\alpha)} - \frac{\rho_4^2}{2(\mu+\eta+\alpha)} = \frac{2\beta S_0 - \rho_4^2}{2(\mu+\eta+\alpha)} \text{ where } S_0 = \frac{\pi(\theta+c+\mu)+\theta K}{(\theta+c+\mu)(a+\mu)-\theta a}$$

$$= \frac{2\beta(\theta\pi + c\pi + \mu\pi + \theta K) - {\rho_4}^2(\mu^2 + \mu\theta + \mu c + \mu a + ca)}{2(\mu+\eta+\alpha)(\mu^2 + \mu\theta + \mu c + \mu a + ca)}$$

$$P_\beta = \frac{\partial {R_o}^S}{\partial \beta} * \frac{\beta}{{R_o}^S} = \frac{2\beta(\theta\pi + c\pi + \mu\pi + \theta K)}{2\beta(\theta\pi + c\pi + \mu\pi + \theta K) - {\rho_4}^2(\mu^2 + \mu\theta + \mu c + \mu a + ca)} > 0$$

$$P_\pi = \frac{\partial {R_o}^S}{\partial \pi} * \frac{\pi}{{R_o}^S} = \frac{2\pi\beta(\theta + c + \mu)}{2\beta(\theta\pi + c\pi + \mu\pi + \theta K) - {\rho_4}^2(\mu^2 + \mu\theta + \mu c + \mu a + ca)} > 0$$

$$P_K = \frac{\partial {R_o}^S}{\partial K} * \frac{K}{{R_o}^S} = \frac{\beta\theta K}{(\mu+\eta+\alpha)(\mu^2 + \mu\theta + \mu c + \mu a + ca)\left[2\beta(\theta\pi + c\pi + \mu\pi + \theta K) - {\rho_4}^2(\mu^2 + \mu\theta + \mu c + \mu a + ca)\right]} > 0$$

$$P_\theta = \frac{\partial R_o^S}{\partial \theta} * \frac{\theta}{R_o^S} =$$

$$\frac{4\theta(\mu+\eta+\alpha)\left[\beta\left[(c\pi + \mu\pi + K\pi)\left(\mu^2 + \mu\theta + \mu c + \mu a + ca\right) + (\theta\pi + c\pi + \mu\pi + \theta K)\left(\mu^2 + \mu c + \mu a + ca - a\right)\right]\right.}{\left. -{\rho_4}^2(\mu^2 + \mu c + \mu a + ca - a)(\mu^2 + \mu\theta + \mu c + \mu a + ca)\right]}$$
$$2(\mu+\eta+\alpha)(\mu^2 + \mu\theta + \mu c + \mu a + ca)\left[2\beta\pi(\theta + c + \mu) + 2\beta\theta K - {\rho_4}^2(\mu^2 + \mu\theta + \mu c + \mu a + ca)\right] > 0$$

$$P_{\mu}=\frac{\partial R_o^S o^S}{\partial \mu}*\frac{\mu}{{R_o}^S}=-\frac{2\rho_4^2\mu(2\mu+\theta+c+a)\left[(\mu+\eta+\alpha)(\mu^2+\mu\theta+\mu c+\mu a+ca)\right]+4\beta\pi\mu}{2(\mu+\eta+\alpha)(\mu^2+\mu\theta+\mu c+\mu a+ca)\left[2\beta\pi(\theta+c+\mu)+2\beta\theta K-\rho_4^2[(\theta+c+\mu)(a+\mu)-\theta a]\right]}+$$

$$\frac{2\mu\left[3\mu^2+\mu(\theta+c+a+\eta+\alpha)+(\eta+\alpha)(\theta+c+a+ca)\right]\left[2\beta(\theta\pi+c\pi+\mu\pi+\theta K)-\rho_4^2(\mu^2+\mu\theta+\mu c+\mu a+ca)\right]}{2(\mu+\eta+\alpha)(\mu^2+\mu\theta+\mu c+\mu a+ca)\left[2\beta\pi(\theta+c+\mu)+2\beta\theta K-\rho_4^2[(\theta+c+\mu)(a+\mu)-\theta a]\right]}<0$$

$$P_{\eta}=\frac{\partial R_o^S}{\partial \eta}*\frac{\eta}{R_o^S}=-\frac{\eta}{(\mu+\eta+\alpha)}<0$$

$$P_{\alpha}=\frac{\partial R_o^S}{\partial \alpha}*\frac{\alpha}{{R_o}^S}=-\frac{\alpha}{(\mu+\eta+\alpha)}<0$$

$$P_{\rho_4}=\frac{\partial R_o^S}{\partial \rho_3}*\frac{\rho_4}{R_o^S}=-\frac{2\rho_4^2(\mu^2+\mu\theta+\mu c+\mu a+ca)}{2\beta(\theta\pi+c\pi+\mu\pi+\theta K)-\rho_4^2(\mu^2+\mu\theta+\mu c+\mu a+ca)}<0$$

Sensitivity index of measles disease.

Parameter	Description	Index
β	contact rate	+ve
π	recruitment of newborn not vaccinated rate	+ve
K	recruitment of newborn vaccinated rate	+ve
θ	waning for first dose of vaccine rate	+ve
a	receive first dose of vaccine to susceptible rate	+ve
μ	natural death rate	-ve
η	disease death rate	-ve
α	recovered from the infected rate	-ve
ρ_4	Intensity of infected	-ve

The above analysis and table shows the sensitivity indices of to the parameters for measles disease model. We summarize the interpretation of sensitivity indices:

The sensitivity indices of the basic reproductive number with respect to the main parameters are discussed above and summarized in Table 1. Accordingly the parameters β, π, K, θ *and* a have positive sensitivity indices which show that they have great impact on expanding the disease in the community if their values get increased. On the other hand the parameters μ, η, α *and* ρ_4 have negative sensitivity indices, so decreasing their value will result in minimizing the expansion of the disease.

4. Numerical simulations results and discussion

In this section, we present and analyze the parameter values of the model along with numerical simulations. For simulation of the developed model we have used parameter values in Table 1 below. Numerical analysis of the model is presented in the form of graphs generated by using MATLAB. This is conducted to find out the transmission dynamics of the measles disease in population.

4.1. Trend of the model with deterministic and stochastic approaches

In Fig. 2 for deterministic case we observe that the number of population in all compartments decreases as time increases, except the number in the recovered population in which case the number of individuals recovered rises steadily as time increases. The stochastic case in the same figure shows that susceptible, infected ones are decreasing from the beginning till a certain time t, where it starts decreasing to be zero. This means that vaccinating first and second dose (double dose) contributes in controlling the measles disease and helps to eradicate the disease from the population after a certain period of time, while keeping the recovered population increasing. We can observe that the solutions of the deterministic model equations are smooth in nature whereas that of the stochastic model exhibit randomness behavior and more effectively.

4.2. Effect of contact rate on infected population

In Fig. 3 we tried to demonstrate the impact of the contact rate β on the number of infected population. The numerical results were obtained by varying the value of contact rate β while keeping other parameters constant. In the deterministic model (left), when the value of contact rate β increased from 0.09091 to 0.3, there is a significant and regular increase in the

Table 1
Parameter values of the model.

Parameter	Value	Source
β	0.09091	Ethiopian health and nutr (2012)
π	0.02755	(Ethiopian health and nutr, 2012; Federal Democratic Republic of Ethiopia, 2008)
K	0.03755	(Ethiopian health and nutr, 2012; Federal Democratic Republic of Ethiopia, 2008)
θ	0.167	Raymond (2016)
μ	0.00875	(Ethiopian health and nutr, 2012; Witbooi, 2013)
a	0.6	Edward et al. (2015)
B	0.8	Edward et al. (2015)
C	0.7	Edward et al. (2015)
η	0.125	Ethiopian health and nutr (2012)
α	0.14286	(Ethiopian health and nutr, 2012; KermackMcKendrick, 1927)
ρ_1	0.02	Witbooi (2013)
ρ_2	0.2	Moss and Griffin (2012)
ρ_3	0.3	Witbooi (2013)
ρ_4	0.4	Moss and Griffin (2012)
ρ_5	0.5	Moss and Griffin (2012)

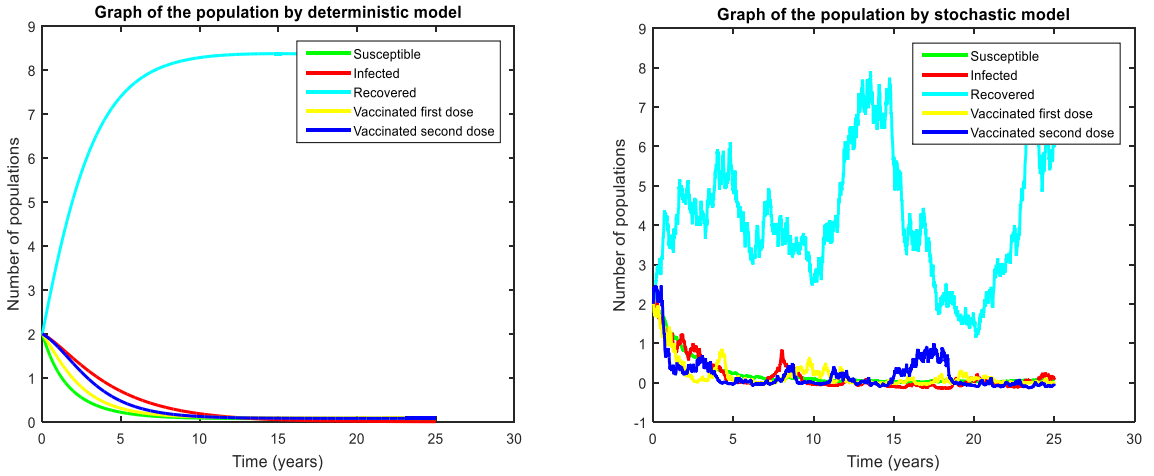

Fig. 2. Graph of the population by deterministic (left) and stochastic (right) measles model.

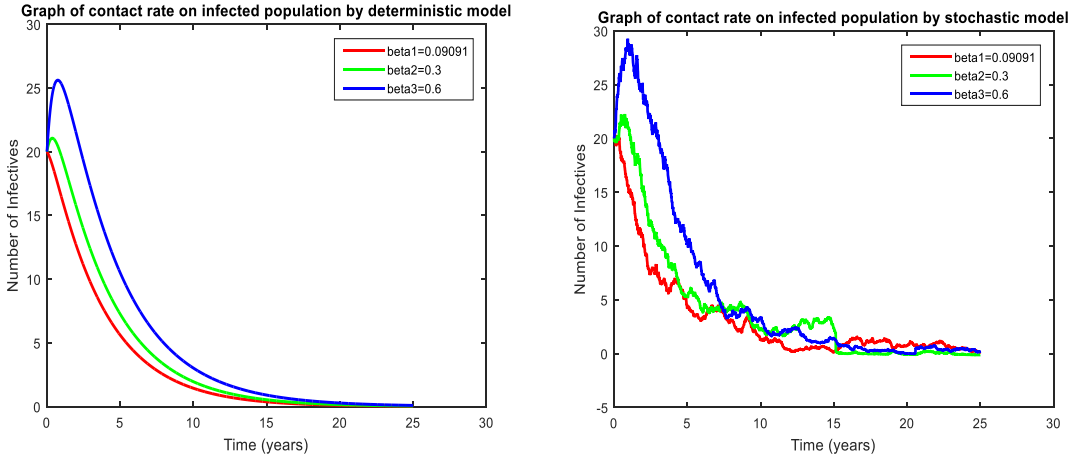

Fig. 3. Graph of the contact rate on infected population for deterministic (left) stochastic (right) measles model.

number of infected population. Moreover, when $\beta = 0.6$ the number of infected people quickly increases above 25 and start to go down a bit, but still manages to be higher than the previous two cases. The results from the stochastic model (right) are also increasing maintaining their perturbing property due to the randomness behavior. However, the overall outcome is that

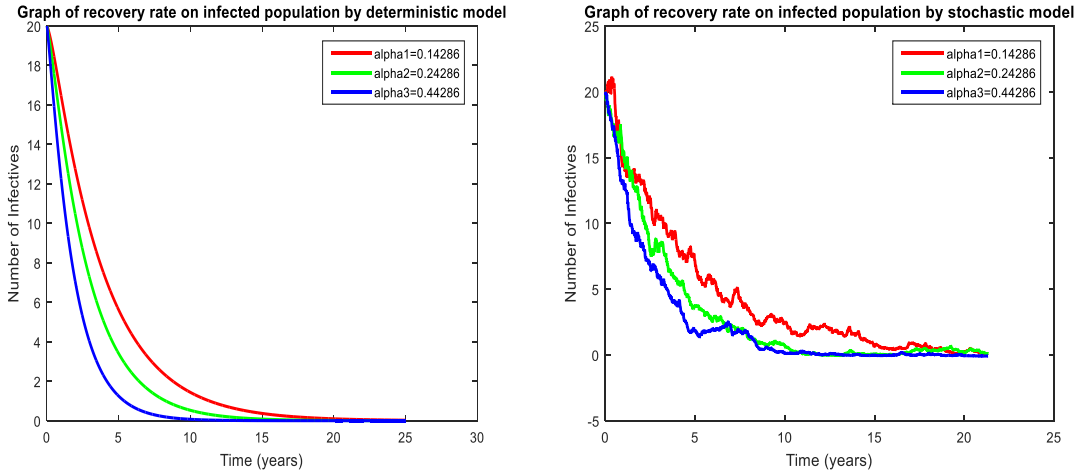

Fig. 4. Graph of the recovery rate on infected population for deterministic (left) stochastic (right) measles model.

the number of infected population still increases significantly with increasing the value of contact rate β. Therefore, we can infer that, when the contact rate β is increasing and other parameters are kept constant, the measles disease transmission expands in the community.

4.3. *Effect of recovery rate on infected population*

In Fig. 4, we investigate the impact of recovery rate α on the size of infected population. The simulation results were obtained by varying the value of recovery rate and keeping the other parameters constant. The numerical results revealed that the number of infected population decreases with increasing the value of recovery rate α. When the value of recovery rate increased from 0.14286 to 0.24286; there is a significant and regular decrease in the number of infected population. Moreover, for the case of $\alpha = 0.44286$ the number of infected people rapidly decreases. It is also observed in the figure that for the deterministic model (left) the infected population decreases smoothly but in the stochastic model (right) it decreases irregularly. Hence, we can analyze that when the value of the recovery rate increase and other parameters are kept constant, the measles disease transmission eliminate from the community.

4.4. *Effect of receiving second dose(double dose) of vaccine rate on vaccinated second dose population*

Fig. 5 displays the plot of the number in the population who got vaccinated second dose against time by varying the value of receiving second dose of vaccine rate b and keeping the other parameters constant. When the value of receiving second dose of vaccine rate b increased from 0.8 to 1.8; there is a significant and regular increase in the number of vaccinated second population. Moreover, when $b = 2.8$ the number of vaccinated second population quickly increases. It is also observed in the figure that for deterministic model (left) the vaccinated (second dose) population decreases smoothly but for the stochastic

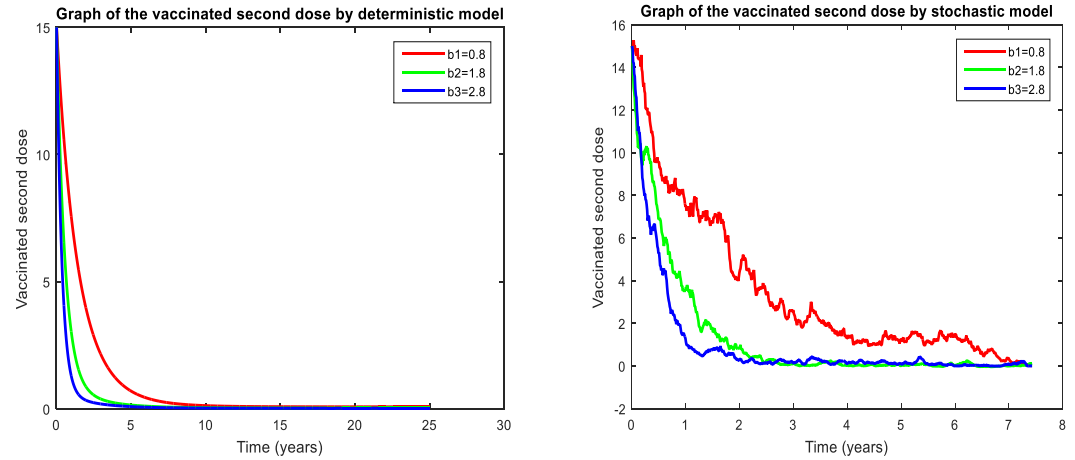

Fig. 5. Graph of receiving first dose of vaccine rate on vaccinated second dose population for deterministic (left) stochastic (right) measles model.

model (right) it decreases irregularly. Therefore, receiving second dose (double dose) of vaccine the target population has a significant contribution in eliminating the measles disease from the community.

5. Summary and conclusion

In this paper we have developed a stochastic model of measles transmission dynamics with double dose vaccination. The governing equations for both deterministic and stochastic models have been formulated. The basic qualitative behaviors of the two models have been analyzed. We have determined a closed form expressions for the basic reproductive numbers of the models and showed that the disease-free equilibrium point is locally asymptotically stable if the basic reproductive number is less than one in each case. Numerical simulations have been also carried out to examine the increase or decrease of the size of the population over time and investigate the effect of basic parameters like the rate of contact and recovery on the infected population. According to the numerical results, it is clear that real world problems such as disease are not deterministic in nature so including random effects to the model gives us a more realistic way of modeling measles epidemics diseases. So, stochastic model analysis is more effective compared to deterministic model analysis in studying the measles transmission dynamics with double dose vaccination. Therefore, we advice the use of stochastic analysis in studying dynamics of infectious diseases, decrease in contact between susceptible and infective population, increase receiving double dose vaccination coverage, combination of awareness and treatment to elimination measles in the community.

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Availability of data and materials

The data we used for this paper is from respective published articles that are cited.

Authors' contributions

All authors have equal contribution for this paper. All authors read and approved the manuscript.

Declaration of competing interest

The authors declare that they have no conflict of interests.

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