# Analysis of Mathematical Model for Diarrhea in the Presence of Vaccine and Treatment


## Abstract

Diarrhea is the second leading cause of death in children under five years old. It is responsible for killing thousands of children globally. It kills more young children than other childhood infectious diseases. Diarrhea illness alone causes more than 1.5 million deaths annually, thereby making it a worse health threat than infectious diseases in terms of death roll. Nonetheless, diarrhea is avoidable and manageable with appropriate treatment. Therefore, this research studied the analysis of a mathematical model of diarrhea dynamics in the presence of vaccination and treatment. To do this, a compartmental mathematical model of (S, V, E, I, R) was considered to investigate the effect of vaccine and treatment in the dynamic spread of diarrhea in the community. The mathematical analysis showed that the disease-free equilibrium point and endemic point of the model exist. Also the basic reproduction $R_o$ was determined through the Next Generation Matrix. The model has a disease-free equilibrium point which is locally asymptotically stable and globally stable over time. Numerical simulations are given to demonstrate the effects of vaccine and treatment on the spread of diarrhea and the result presented showed that vaccine and treatment have a pronounced effect of reducing diarrhea infection.


*Keywords: Stability; Basic Reproduction Number; Diarrhea Model*

2010 Mathematics Subject Classification: 92D30;92B05;34D20

## 1 Introduction

Diarrhea is a medical condition characterized by frequent loose and watery bowel movements, accompanied by abdominal bloating and pressure. These conditions can be categorized as acute, persistent, or chronic. Typically, acute diarrhea lasts for only one day or two and subsides without any treatment.

Persistent diarrhea lasts for over two weeks but less than four weeks, while chronic diarrhea lasts for at least four weeks. The symptoms of chronic diarrhea may be ongoing or may come and go. Although immunity after infection is temporary, subsequent infections are usually less severe than the initial ones. However, diarrhea can be prevented and effectively treated with appropriate measures.
The use of differential equations to model biological, ecological, and medical systems has a long history dating back to Verhulst, Malthus, Lotka, and Volterra, [(8)]. Differential equations are known to be useful for modeling natural phenomena. Ordinary differential equations, for instance, are known to be very useful in modeling population behavior, transmission of infectious diseases, interaction between two or more species, and other biological processes, see ([(6)], [(7)], [(10)], [(11)] and [(12)]). Loopman *et al*.[(9)] analyzed the dynamic transmission model of nor virus infection disease and immunity. It was found that the asymptomatic prevalence of norovirus can change dramatically with small changes in the basic reproduction number $R_o$. Adewale *et al*.[(1)] worked on mathematical analysis of diarrhea in the presence of a vaccine. They computed $R_o$ in cases where $R_o > 1$, the disease became endemic, meaning the disease remained in the population at a consistent rate, as one infected individual transmits the disease to one susceptible. Ardkaew and Tongkumchum,[(2)] also worked on the epidemiological model of diarrhea diseases and its application in prevention and control. The model was able to mimic the observed epidemiology patterns of infantile diarrhea diseases associated mainly with enterotoxigenic Escherichia coli or with rotavirus. The proposed mathematical model predicted a plausible pattern of the serological profile of an enteric infection. Bonyah *et al*.[(3)] investigated a mathematical model of (SITR) to investigate the effect of saturation treatment in the dynamic spread of diarrhea in the community. Cherry *et al*.[(4)] worked on the Assessment of bovine viral diarrhea virus management utilizing a mathematical model depicting infection dynamics. The model architecture was a development of the traditional model framework using susceptible, infectious, and removed animals (the SIR model). The model forecasted a 1.2% rate of persistent infection (falling within the fields's estimated range) and showed limited sensitivity to changes in structures or parameter values. This model drew important conclusions regarding the control of Bovine Viral Diarrhea (BVD), particularly concerning the importance of persistently infected (PI) animals in maintaining BVD as an endemic entity in the herd. A model of dysentery diarrhea was proposed to investigate the criteria for stability of the disease free-equilibrium which makes the reproduction number the most sensitive to the control of the effective rate of transmission of dysentery diarrhea.[(5)]
Despite various measures taken, eradicating diarrhea has proven to be a challenging task due to persistent infection despite the presence of a vaccine. A deterministic epidemic model (SVEIR) is considered in this study to gain more insight into the effect of vaccines and treatment of infected individuals on the dynamic spread of diarrhea in the population. Results established indicate that the vaccine plays a vital role in the control of the spread of diarrhea disease, the increase in susceptible individuals is dependent on the effectiveness of the vaccine given against diarrhea and the rate of treatment decreases the number of infected individuals.

## 1.1 Model Diagram

The model comprises of Susceptible (S), Vaccinated (V), Exposed (E), Infected (I), and Recovered (R), i.e SVEIR. Figure 1 illustrates the relationship between the human compartments within the population as well depicts the movement of individuals within the compartment and in and out of population. At time t, the total human population is,

$$N = S + V + E + I + R$$

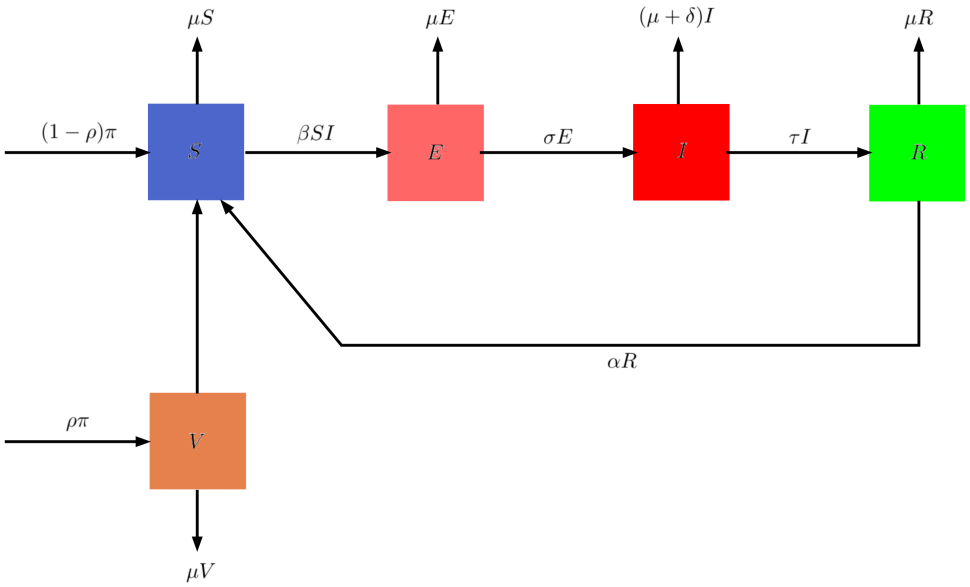


Figure 1: Diagram of the S, V, E, I, R Model.

## 1.2 Model Equation

Here we consider five classes of individuals which are: susceptible (S), vaccinated (V), exposed (E), infected (I), and recovered (R) which is SVEIR. This is an appropriate model for a disease where there is a considerable post-infected incubation period in which the exposed person is not yet infectious. The SVEIR model consists of a set of five differential equations,

$$\begin{aligned}
\frac{dS}{dt} &= (1-\rho)\pi - \beta SI + \omega V - \mu S + \alpha R \\
\frac{dV}{dt} &= \rho\pi - (\mu+\omega)V \\
\frac{dE}{dt} &= \beta SI - (\mu+\sigma)E \\
\frac{dI}{dt} &= \sigma E - (\mu+\tau+\delta)I \\
\frac{dR}{dt} &= \tau I - (\mu+\alpha)R
\end{aligned} \tag{1.1}$$

### 1.3 Description of Parameters of the Model

List 1. Description of Parameters

| Parameter | Description |
|---|---|
| $\pi$ | Recruitment rate |
| $\rho$ | Vaccine rate |
| $\beta$ | Contact rate |
| $\omega$ | Rate at which vaccine wanes off |
| $\mu$ | Natural death |
| $\sigma$ | Rate at which the exposed individuals becomes infected |
| $\tau$ | Rate at which infected individual are treated |
| $\delta$ | Induced diseases death rate |
| $\alpha$ | Rate at which recovered individuals move to susceptible class |

## 2 Disease Free Equilibrium

The steady state, also known as disease-free equilibrium, occurs when there is no infection, meaning that both the exposed and infected classes are at zero. That is, putting $E = I = 0$, the model equation Eq. (1.1) becomes;

$$\begin{aligned}\frac{dS}{dt} &= (1-\rho)\pi - \beta SI + \omega V - \mu S + \alpha R \\ \frac{dV}{dt} &= \rho\pi - \mu V - \omega V \\ \frac{dR}{dt} &= \tau I - \mu R - \alpha R\end{aligned}$$

Solving for $S$, $V$, and $R$, gives the disease free equilibrium as

$$E_o = (S_o, V_o, E_o, I_o, R_o) = \left(\frac{(\mu+\omega)(1-\rho)\pi + \omega\rho\pi}{\mu(\mu+\omega)}, \frac{\rho\pi}{\mu+\omega}, 0, 0, 0\right). \tag{2.1}$$

## 3 Endemic Equilibrium

At endemic equilibrium, there is presence of infection in the host population i.e $E, I \neq 0$.
To obtain an endemic equilibrium, we set each equations in the model formulated to zero in Eq. (1.1) to get,

$$\begin{aligned}(1-\rho)\pi - \beta SI + \omega V - \mu S + \alpha R &= 0 \\ \rho\pi - \mu V - \omega V &= 0 \\ \beta SI - \mu E - \sigma E &= 0 \\ \sigma E - \mu I - \tau I - \delta I &= 0 \\ \tau I - \mu R - \alpha R &= 0\end{aligned}$$

and Solving for $S, V, E, I, R$, we have

$$V^* = \frac{\rho\pi}{\mu+\omega},$$

$$S^* = \frac{(\mu+\sigma)(\mu+\tau+\delta)}{\sigma\beta},$$

$$I^* = \frac{\alpha\sigma(\mu+\alpha)}{(\mu+\tau+\delta)(\mu+\sigma)(\mu+\alpha)} - \tau\alpha\sigma\left(\frac{(1-\rho)\pi}{\alpha} + \frac{\omega\rho\pi}{\alpha(\mu+\omega)} - \frac{\mu(\mu+\tau+\delta)(\mu+\sigma)}{\alpha\sigma\beta}\right),$$

$$E^* = \frac{1}{(\mu+\sigma)-\tau\sigma}\left(\frac{(1-\rho)\pi}{\alpha} + \frac{\omega\rho\pi}{\alpha(\mu+\omega)} - \frac{\mu(\mu+\tau+\delta)(\mu+\sigma)}{\alpha\sigma\beta}\right),$$

$$R^* = \frac{1}{(\mu+\tau+\delta)(\mu+\sigma)(\mu+\alpha)}\left(\frac{(1-\rho)\pi}{\alpha} + \frac{\omega\rho\pi}{\alpha(\mu+\omega)} - \frac{\mu(\mu+\tau+\delta)(\mu+\sigma)}{\alpha\sigma\beta}\right).$$

# 4 Basic Reproduction Number $R_o$

The basic reproduction number $R_o$ of this model is calculated by using the next generation matrix

$$\frac{dE}{dt} = \beta SI - \mu E - \sigma E = F_1$$
$$\frac{dI}{dt} = \sigma E - \mu I - \tau I - \delta I = F_2$$
$$F = \begin{pmatrix} \frac{\partial F_1}{\partial I} & \frac{\partial F_1}{\partial E} \\ \frac{\partial F_2}{\partial I} & \frac{\partial F_2}{\partial E} \end{pmatrix} = \begin{pmatrix} \beta S_o & 0 \\ 0 & 0 \end{pmatrix}_E^I$$
$$V = \begin{pmatrix} \frac{\partial F_1}{\partial I} & \frac{\partial F_1}{\partial E} \\ \frac{\partial F_2}{\partial I} & \frac{\partial F_2}{\partial E} \end{pmatrix} = \begin{pmatrix} 0 & (\mu+\sigma) \\ (\mu+\tau+\delta) & -\sigma \end{pmatrix}_E^I$$

which implies

$$V^{-1} = -\left(\frac{1}{(\mu+\sigma)(\mu+\tau+\delta)}\right)\begin{pmatrix} -\sigma & -(\mu+\sigma) \\ -(\mu+\tau+\delta) & 0 \end{pmatrix}$$
$$FV^{-1} = \begin{pmatrix} \beta S_0 & 0 \\ 0 & 0 \end{pmatrix}\begin{pmatrix} \frac{\sigma}{(\mu+\sigma)(\mu+\tau+\delta)} & \frac{1}{\mu+\tau+\delta} \\ \frac{1}{\mu+\sigma} & 0 \end{pmatrix}$$
$$= \begin{pmatrix} \frac{\beta S_0\sigma}{(\mu+\sigma)(\mu+\tau+\delta)} & \frac{\beta S_0}{\mu+\tau+\delta} \\ 0 & 0 \end{pmatrix}$$
$$|FV^{-1} - I\lambda| = 0$$

$$\left|\begin{pmatrix} \frac{\beta S_o\sigma}{(\mu+\sigma)(\mu+\tau+\delta)} & \frac{\beta S_o}{\mu+\tau+\delta} \\ 0 & 0 \end{pmatrix} - \begin{pmatrix} \lambda & 0 \\ 0 & \lambda \end{pmatrix}\right| = 0$$
$$\left|\begin{pmatrix} \frac{\beta S_0\sigma}{(\mu+\sigma)(\mu+\tau+\delta)} - \lambda & \frac{\beta S_0}{\mu+\tau+\delta} \\ 0 & 0-\lambda \end{pmatrix}\right| = 0.$$

At disease free equilibrium $E_o$ in Eq. (2.1), we have

$$\lambda_1 = \sigma\beta\left(\frac{(1-\rho)\pi(\mu+\omega)+\omega\rho\pi}{(\mu+\sigma)(\mu+\tau+\delta)\mu(\mu+\omega)}\right)$$
$$\lambda_2 = 0$$

So,

$$R_0 = \sigma\beta\left(\frac{(1-\rho)\pi(\mu+\omega)+\omega\rho\pi}{\mu(\mu+\omega)[(\mu(\mu+\tau+\delta+\sigma)+\sigma(\tau+\delta)]}\right).$$

# 5 Stability Analysis of The Disease Free Equilibrium

Theorem 1: The disease-free equilibrium $E_0 = \left(\frac{(\mu+\omega)(1-\rho)\pi+\omega\rho\pi}{\mu(\mu+\omega)}, \frac{\rho\pi}{\mu+\omega}, 0, 0, 0\right)$, exists for all non-negative values of its parameters and it is locally asymptotically stable when $R_o \leq 1$ and it is unstable when $R_o > 1$.

Proof: From equation Eq. (1.1), we have that

$$\begin{aligned}
F_1 &= (1-\rho)\pi - \beta SI + \omega V - \mu S + \alpha R = 0 \\
F_2 &= \rho\pi - \mu V - \omega V = 0 \\
F_3 &= \beta SI - \mu E - \sigma E = 0 \\
F_4 &= \sigma E - \mu I - \tau I - \delta I = 0 \\
F_5 &= \tau I - \mu R - \alpha R = 0
\end{aligned}$$

The Jacobian matrix of system of equation Eq. (1.1) at disease free equilibrium $E_o$ in Eq. (2.1) is given by

$$J = \begin{pmatrix}
-\mu - \beta I_0 & \omega & 0 & -\beta S_0 & \alpha \\
0 & -\mu - \omega & 0 & 0 & 0 \\
\beta I_0 & 0 & -\mu - \omega & \beta S_0 & 0 \\
0 & 0 & \sigma & -(\mu + \tau + \delta) & 0 \\
0 & 0 & 0 & \tau & -(\mu + \alpha)
\end{pmatrix}$$

$$A = \begin{pmatrix}
-\mu & \omega & 0 & -\beta S_0 & \alpha \\
0 & -\mu - \omega & 0 & 0 & 0 \\
0 & 0 & -\mu - \omega & \beta S_0 & 0 \\
0 & 0 & \sigma & -(\mu + \tau + \delta) & 0 \\
0 & 0 & 0 & \tau & -(\mu + \alpha)
\end{pmatrix}$$

Solving

$$|A - I\lambda| = 0$$

that is,

$$\Longrightarrow \begin{vmatrix}
-(\mu+\lambda) & \omega & 0 & -\beta\left(\frac{(\mu+\omega)(1-\rho)\pi+\omega\rho\pi}{\mu(\mu+\omega)}\right) & \alpha \\
0 & -(\mu+\omega+\lambda) & 0 & 0 & 0 \\
0 & 0 & -(\mu+\sigma+\lambda) & \beta\left(\frac{(\mu+\omega)(1-\rho)\pi+\omega\rho\pi}{\mu(\mu+\omega)}\right) & 0 \\
0 & 0 & \sigma & -(\mu+\tau+\delta+\lambda) & 0 \\
0 & 0 & 0 & \tau & -(\mu+\alpha+\lambda)
\end{vmatrix} = 0.$$

Evaluating the determinant gives,

$$(\mu+\lambda)(\mu+\omega+\lambda)(\mu+\alpha+\lambda)[-(\mu+\sigma+\lambda)(\mu+\tau+\delta+\lambda)+\beta S_o] = 0$$

Clearly,

$$\begin{aligned}
\lambda_1 &= -\mu \\
\lambda_2 &= -(\mu+\omega) \\
\lambda_3 &= -(\mu+\alpha)
\end{aligned}$$

Also,

$$\begin{aligned}
&[-(\mu+\sigma+\lambda)(\mu+\tau+\delta+\lambda)+\beta S_o] = 0 \\
\Longrightarrow\ &(\mu+\sigma+\lambda)(\mu+\delta+\tau+\lambda) - \sigma\beta S^o = 0 \\
\Longrightarrow\ &\lambda^2 + [(\mu+\sigma)+(\mu+\delta+\tau)]\lambda + (\mu+\sigma)(\mu+\delta+\tau) - \sigma\beta S^o = 0
\end{aligned}$$

substituting $S^o$, we have

$$\lambda^2 + [(\mu+\sigma)+(\mu+\delta+\tau)]\,\lambda + (\mu+\sigma)(\mu+\delta+\tau)\left[1-\sigma\beta\left(\frac{(\mu+\omega)(1-\rho)\pi+\omega\rho\pi}{\mu(\mu+\omega)(\mu+\sigma)(\mu+\delta+\tau)}\right)\right]=0$$

$$\implies \lambda^2 + [(\mu+\sigma)+(\mu+\delta+\tau)]\,\lambda + (\mu+\sigma)(\mu+\delta+\tau)\,[1-R_o]=0 \tag{5.1}$$

By Descartes's rule of sign, the polynomial equation (5.1) has no sign change if $R_o<1$, and so there are no positive roots for the equation (5.1). This implies that all roots of (5.1) are purely imaginary or complex with negative real parts. Hence the DFE is locally asymptotically stable.
This completes the proof.

# 6 Global Stability

Theorem 2: If $R_0 \leq 1$, then the disease-free equilibrium is globally asymptotically stable, and unstable otherwise.
Proof
Let $L$ be a candidate Lyapunov function such that

$$L=\left(S-S^o-S^o\ln\frac{S}{S^o}\right)+\frac{\sigma E}{(\mu+\sigma)(\mu+\delta+\tau)}+\frac{I}{(\mu+\delta+\tau)} \tag{6.1}$$

where $S^o=\frac{(\mu+\omega)(1-\rho)\pi+\omega\rho\pi}{\mu(\mu+\omega)}$ is the value $SV$ at DFE.
Obviously, the second and third terms on the RHS of 6.1 are positive for the first term, $S^o \leq S$ (since $S^o$ is an equilibrium point of $S$). Then $S-S^o-S\ln\frac{S}{S^o}$ is also positive. Therefore, $L(S,E,I)$ is positive definite.
Now, for the time derivative of $L$ along the solution of the model equation 6.1, we have.

$$\frac{dL}{dt}=\left(1-\frac{S^o}{S}\right)\frac{dS}{dt}+\frac{\sigma}{(\mu+\sigma)(\mu+\delta+\tau)}\frac{dE}{dt}+\frac{1}{\mu+\delta+\tau}\frac{dI}{dt}$$

substituting $\frac{dS}{dt}$, $\frac{dE}{dt}$ and $\frac{dI}{dt}$ from (1.1) gives

$$\frac{dL}{dt}=\left(1-\frac{S^o}{S}\right)[(1-\rho)\pi-\beta SI-\omega V-\mu S+\alpha R]$$
$$+\frac{\sigma}{(\mu+\sigma)(\mu+\delta+\tau)}[\beta SI-(\mu+\sigma)E]+\frac{1}{\mu+\delta+\tau}[\sigma E-(\mu+\delta+\tau)I]$$

At disease free equilibrium:

$$\left.\begin{aligned}(1-\rho)\pi &= \beta SI^o+\mu S^o-(\omega V^o+\alpha R^o)\\ \frac{\beta SI^o}{\mu+\sigma} &= E^o\\ \sigma E^o &= (\mu+\delta+\tau)I^o\end{aligned}\right\} \tag{6.2}$$

substituting (6.2) into $\frac{dL}{dt}$, gives

$$\frac{dL}{dt}=\left(1-\frac{S^o}{S}\right)[(\beta SI^o-\beta SI)+\omega(V^o-V)+\mu(S^o-S)+\alpha(R^o-R)]$$
$$+\frac{\sigma}{(\mu+\sigma)(\mu+\delta+\tau)}\left[\beta SI-\frac{(\mu+\sigma)\beta S^oI^o}{\mu+\sigma}\right]+\frac{1}{\mu+\delta+\tau}[(\mu+\delta+\tau)(I^o-I)]$$

$$\Longrightarrow$$

$$-\beta SI\left(1-\frac{S^o}{S}\right)-\mu(S-S^o)-\omega(V-V^o)-\alpha R+\left[\frac{\sigma\beta S}{(\mu+\sigma)(\mu+\delta+\tau)}-1\right]I$$

At disease-free equilibrium,

$$\begin{aligned}E_o&=(S^o,V^o,E^o,I^o,R^o)\\&=\left(\frac{(\mu+\omega)(1-\rho)\pi+\omega\beta\tau}{\mu(\mu+\omega)},\frac{\rho\pi}{\mu+\omega},0,0,0\right)\end{aligned}$$

$$\frac{dL}{dt}=-\beta SI\left(\frac{S-S^o}{S}\right)-\omega(V-V^o)-\alpha R+(R_o-1)I \tag{6.3}$$

obviously from (6.3), $\frac{dL}{dt}<0$ if $R_o\leq 1$

$$\text{where } R_o=\sigma\beta\left[\frac{(\mu+\omega)(1-\rho)\pi+\omega\rho\pi}{\mu(\mu+\omega)(\mu+\sigma)(\mu+\delta+\tau)}\right] \tag{6.4}$$

$\frac{dL}{dt}=0$ if and only if $S=S^o, V=V^o$ and $I=0$.
Thus

$$(S,V,E,I,R)\longrightarrow\left(\frac{(\mu+\omega)(1-\rho)\pi+\omega\pi}{\mu(\mu+\omega)},\frac{\rho\pi}{\mu+\omega},0,0,0\right)\text{ as } t\to\infty$$

and the largest compact invariant set is the singleton $\{E_o\}$. So, by lasalle's invariant principle (Lasalle, 1996), every solution of the model system (1.1) with initial conditions in approaches $E_o$ as $t\to\infty$. whenever $R_o\leq 1$. Then the disease-free equilibrium is globally asymptotically stable whenever $R_o\leq 1$ and unstable otherwise.
This completes the proof.

# 7 Stability Analysis of the Endemic Equilibrium

Theorem 3: The endemic equilibrium $E^*=(S^*,V^*,E^*,I^*,R^*)$ is stable if $R_o>1$
Proof: If the disease is persistent (i.e endemic) in the community, then $\frac{dI}{dt}>0$ by [(14)]

$$\begin{aligned}&\text{i.e } \sigma E^*-(\mu+\delta+\tau)I^*>0\\&\Longrightarrow\ \sigma E^*>(\mu+\delta+\tau)I^*\\&\Longrightarrow\ (\mu+\delta+\tau)I^*<\sigma E^*\\&\Longrightarrow\ 1<\frac{\sigma E^*}{(\mu+\delta+\tau)I^*}\end{aligned}$$

Stability $E^*$ and $I^*$ from the endemic equilibrium and simplifying gives

$$1<R_o$$

$$\text{i.e } R_o>1$$

Hence, the endemic equilibrium is stable whenever $R_o>1$ and unstable otherwise.

# 8 Numerical Simulations and Results

The evaluation of the model involved a numerical analysis. Through simulations, it was possible to observe the impact of the parameters. The software used for the simulations is Wolfram Mathematica. Some values for the parameters of the SVEIR model were obtained from [(1)].

Table 1: Values of the Parameters for Fig. 2, Fig. 3, Fig. 4, Fig. 5, and Fig. 6

| Parameter | Description | Value | Source |
|---|---|---|---|
| $\pi$ | Recruitment rate | 2000 | [(1)] |
| $\rho$ | Vaccine rate | 0.5 | [(1)] |
| $\beta$ | Contact rate | 0.0003 | Estimated |
| $\omega$ | Rate at which vaccine wanes off | 0.1 | [(1)] |
| $\mu$ | Natural death | 0.2 | [(1)] |
| $\sigma$ | Rate at which the exposed individuals becomes infected | 0.7 | [(1)] |
| $\tau$ | Rate at which infected individuals are treated | 0.1 | [(1)] |
| $\delta$ | Induced diseases death rate | 0.1 | [(1)] |
| $\alpha$ | Rate at which recovered individuals move to susceptible class | 0.2 | [(1)] |
| $S(0)$ | Susceptible class | 1000 | Estimated |
| $V(0)$ | Vaccinated class | 800 | Estimated |
| $E(0)$ | Exposed class | 600 | Estimated |
| $I(0)$ | Infected class | 500 | Estimated |
| $R(0)$ | Recovered class | 700 | Estimated |

# 9 Sensitivity Analysis

The sensitivity index of each parameter can be seen in the table below, inputting the value of each parameter into the differential equations and solving them using

$$X_x^{R_o} = \frac{\partial R_o}{\partial x} \cdot \frac{x}{R_o}$$

where $X^{R_o}$ denote the sensitivity of $R_o$ then sensitivity index $R_o$ with respect to any parameter.

Table 2: Parameter sensitivity index

| Parameter | Index | Sensitivity index |
|---|---|---|
| $\pi$ | $X_\pi^{R_o}$ | 0.090909091 |
| $\sigma$ | $X_\sigma^{R_o}$ | 0.9623655914 |
| $\mu$ | $X_\mu^{R_o}$ | -0.06989247312 |
| $\omega$ | $X_\omega^{R_o}$ | 0.09090909091 |
| $\tau$ | $X_\tau^{R_o}$ | -0.005376344086 |
| $\beta$ | $X_\beta^{R_o}$ | 1 |
| $\rho$ | $X_\rho^{R_o}$ | -0.8181818182 |
| $\delta$ | $X_\delta^{R_o}$ | -0.005376344086 |

The parameter sensitivity index using the derivative-based local method is as shown in Table 2 which indicates that the parameters $\beta, \sigma, \omega$ and $\pi$ have direct relationship with the reproduction

number $R_o$ and parameters $\rho, \mu, \tau$ and $\delta$ have inverse relationship with $R_o$. Hence, reducing the contact rate between the infected human and susceptible individuals as well as restricting direct access to public food and water by the infected individual could significantly reduce the $R_o$. Other factors like increase in vaccination rate and ensuring reduction in the rate if waning of immunity as well as increasing the rate of treatment of infected individuals will eventually and effectively reduce the value of $R_o$.

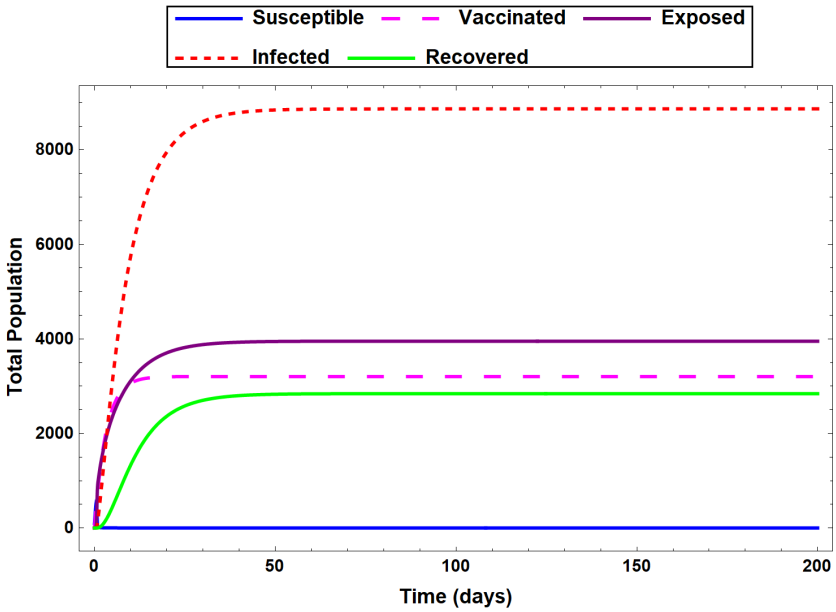


Figure 2: Shows that the higher the rate at which the vaccine wanes off the higher the number infected population

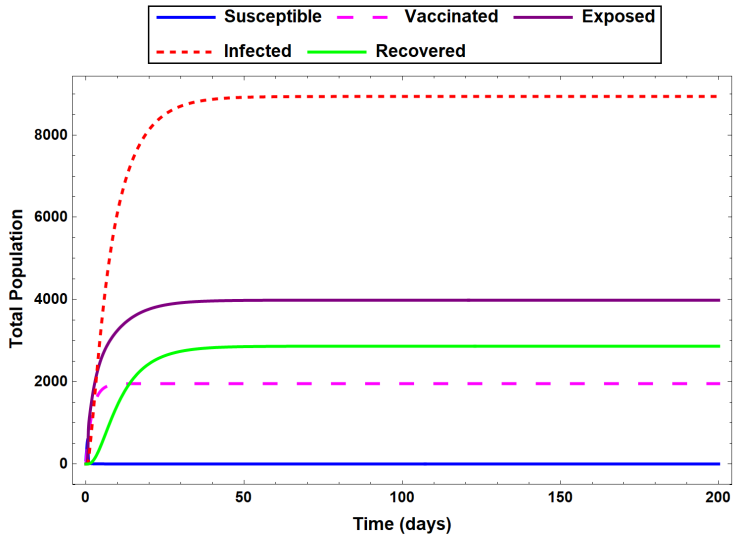


Figure 3: Shows that the higher the rate at which the vaccine wanes off the higher the number of exposed population

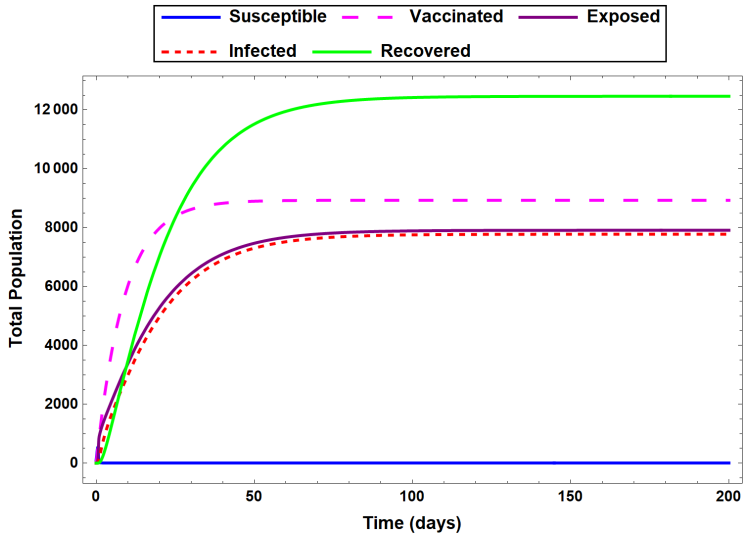


Figure 4: Shows that the higher the rate of treatment the lower the vaccinated population and the higher the number of exposed and infected population indicating that the rate at which the vaccine wanes off is rapid.

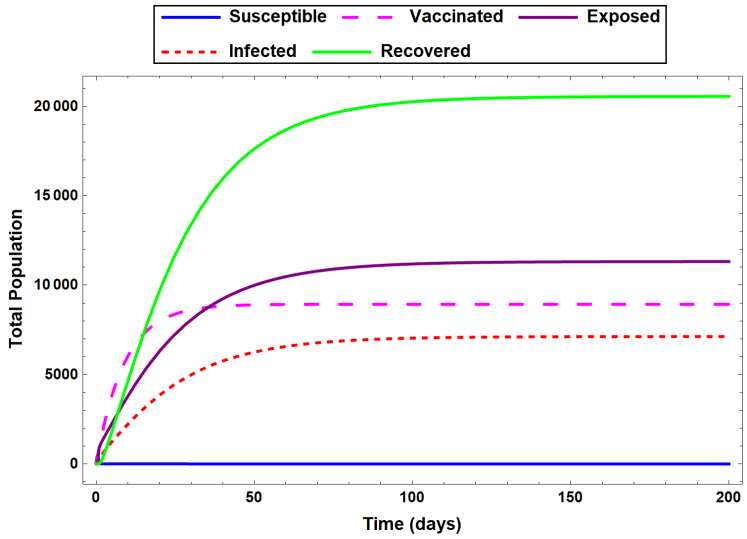


Figure 5: Shows that the higher the rate of treatment the higher the recovered population as the vaccine wanes off.

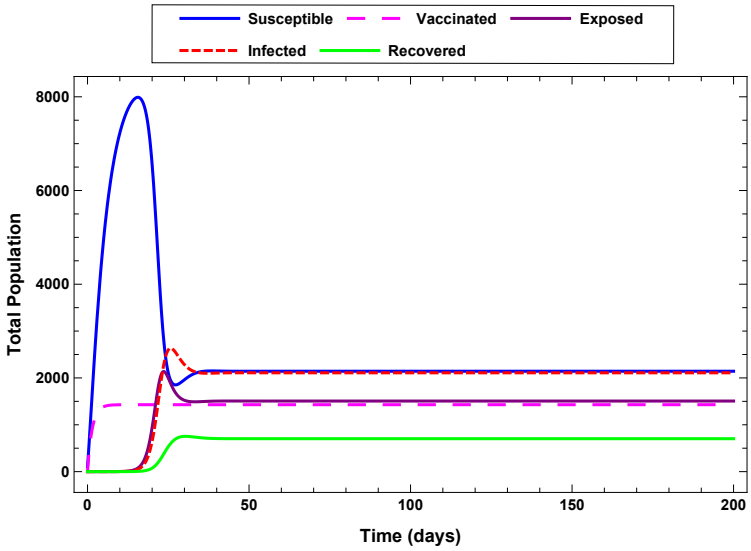


Figure 6: Shows that as the rate at which the vaccine wanes off increases the number of the susceptible class increases at a decreasing rate as the rate of treatment increases.

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