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Mathematical Modeling of Diarrhea with Vaccination and Treatment Factors

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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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.

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The model also has stability of the endemic equilibrium which is stable when $R_o > 1$. 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. Moreover, combined with sensitivity analysis, we observe that even though vaccination is adequate but not sufficient in reducing the basic reproduction number, it effectively manages the disease.

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, [1]. 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 ([2], [3], [4], [5] and [6]). Loopman *et al.*[7] 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.*[8] 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. Akinola *et al.*[9] also studied similar model with vaccine and found out that vaccination of susceptible individuals will reduce the spread of diarrhea disease compared to when there is no vaccination. Ardkaew and Tongkumchum, [10] 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.*[11] 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.*[12] 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.[13]. Other similar investigations on the endemic diseases using similar model to estimate the active cases, deaths, recoveries in order to control the disease in the presence of vaccine and treatment were carried out by ([14],[15],[16] and [17]).

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. Fig. 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,

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

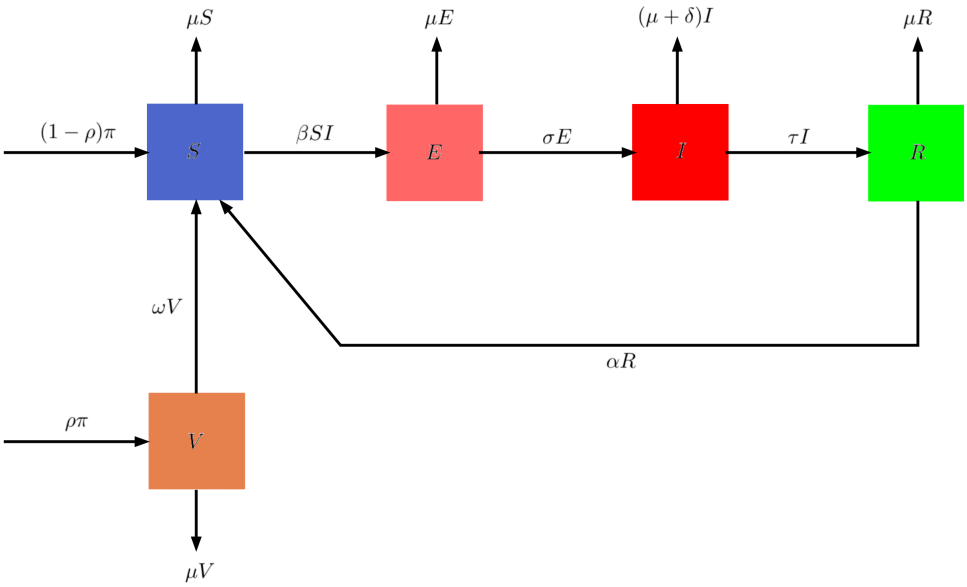

Fig. 1. Diagram of the S, V, E, I, R Model

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. From the model diagram in Fig. 1, the susceptible population increases due to individual recruitment at rate π. This population decreases due to vaccination, with a fraction ρ of vaccinated individuals leaving, and susceptible individuals acquiring diarrhea infection through effective contact with infected people at rate β. The susceptible population increases from recovered individuals returning and vaccinated individuals experiencing waning immunity at rates α and ω respectively, and decreases at rate μ. The vaccinated class increases at rate $\rho\pi$ and decreases due to waning immunity and natural death at rates ω and μ respectively. The exposed class increases from new infections among susceptible individuals at rate β and decreases due to natural death at rate μ and individuals becoming infected at rate σ. Infected individuals increase from the exposed class at rate σ and decrease due to treatment, natural death, and induced death at rates τ, μ, and δ respectively. The recovered class increases from treated infected individuals at rate τ and decreases due to natural death and individuals returning to the susceptible class at rates μ and α respectively.

Thus, 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

Parameter	Description
π	Recruitment rate
ρ	Vaccine rate
β	Contact rate
ω	Rate at which vaccine wanes off
μ	Natural death
σ	Rate at which the exposed individuals becomes infected
τ	Rate at which infected individual are treated
δ	Induced diseases death rate
α	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

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

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,

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

substituting S^o, we have

$$\begin{aligned}&\lambda^2+[(\mu+\sigma)+(\mu+\delta+\tau)]\,\lambda+(\mu+\sigma)(\mu+\delta+\tau)\left[1-\sigma\beta\left(\frac{(\mu+\omega)(1-\rho)\pi+\omega\rho\pi}{\mu(\mu+\omega)(\mu+\sigma)(\mu+\delta+\tau)}\right)\right]=0\\ \implies &\lambda^2+[(\mu+\sigma)+(\mu+\delta+\tau)]\,\lambda+(\mu+\sigma)(\mu+\delta+\tau)\,[1-R_o]=0 \qquad (5.1)\end{aligned}$$

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 $R_0 \le 1$, then the disease-free equilibrium is globally asymptotically stable, and unstable otherwise.

Proof

Let L be a candidate Lyapunov function such that

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

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, $S^o \le S$ (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

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

At disease free equilibrium:

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

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

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

$\Longrightarrow$

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

At disease-free equilibrium,

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

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

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, every solution of the model system (1.1) with initial conditions 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 [18]

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

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 [8].

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

Parameter	Description	Value	Source
π	Recruitment rate	2000	[8]
ρ	Vaccine rate	0.5	[8]
β	Contact rate	0.0003	Estimated
ω	Rate at which vaccine wanes off	0.1	[8]
μ	Natural death	0.2	[8]
σ	Rate at which the exposed individuals becomes infected	0.7	[8]
τ	Rate at which infected individuals are treated	0.1	[8]
δ	Induced diseases death rate	0.1	[8]
α	Rate at which recovered individuals move to susceptible class	0.2	[8]
$S(0)$	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

To investigate the sensitivity of the basic reproduction number R_o with respect to parameters β, σ, ω, π, ρ, μ, τ and δ, we calculate each value using the derivative-based method, which reflects the relationship between each parameter and R_o. 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
π	$X_\pi^{R_o}$	0.090909091
σ	$X_\sigma^{R_o}$	0.9623655914
μ	$X_\mu^{R_o}$	-0.06989247312
ω	$X_\omega^{R_o}$	0.09090909091
τ	$X_\tau^{R_o}$	-0.005376344086
β	$X_\beta^{R_o}$	1
ρ	$X_\rho^{R_o}$	-0.8181818182
δ	$X_\delta^{R_o}$	-0.005376344086

The parameter sensitivity index using the derivative-based local method is as shown in Table 2 which indicates that the parameters β, σ, ω and π have direct relationship with the reproduction number R_o and parameters ρ, μ, τ and δ 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 of waning of immunity as well as increasing the rate of treatment of infected individuals will eventually and effectively reduce the value of R_o. The sensitivity analysis findings indicate that while the vaccination doesn't significantly lower the basic reproduction number, it effectively aids in disease control.

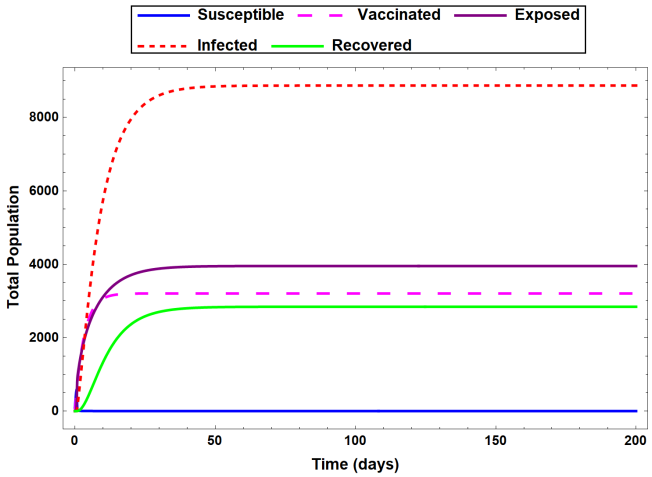

Fig. 2. Shows that the higher the rate at which the vaccine wanes off the higher the number of infected population

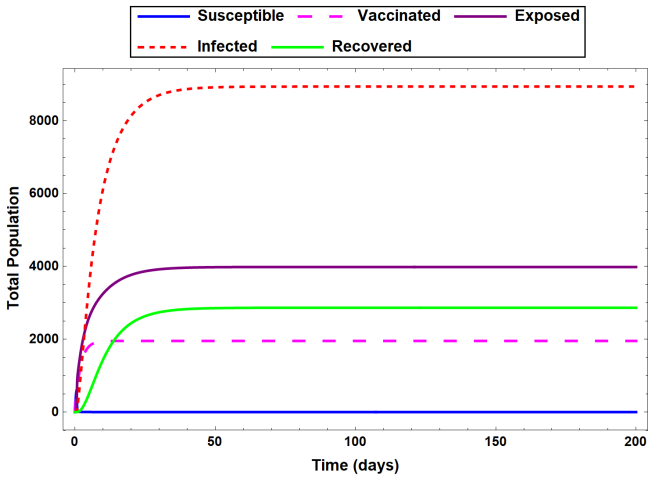

Fig. 3. Shows that the higher the rate at which the vaccine wanes off the higher the number of exposed population

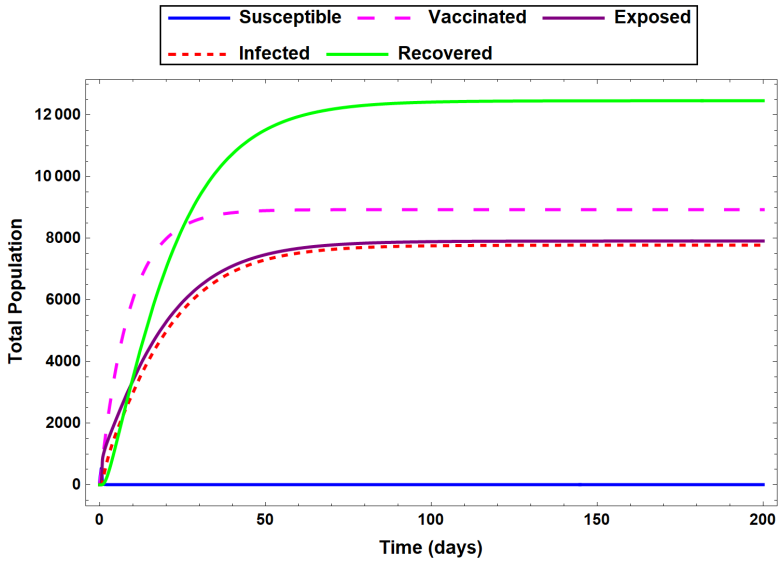

Fig. 4. Shows that the higher the rate of treatment the lower the vaccinated population and the higher the number of exposed and infected population indicating that the rate at which the vaccine wanes off is rapid.

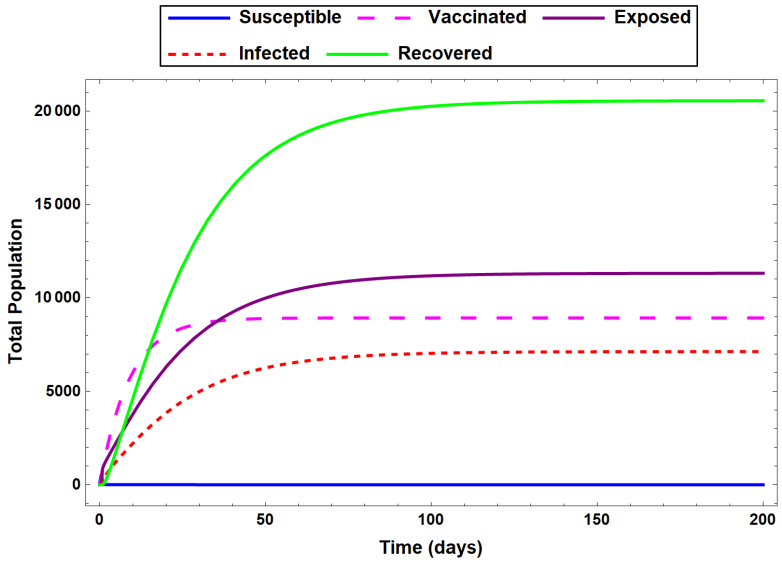

Fig. 5. Shows that the higher the rate of treatment the higher the recovered population as the vaccine wanes off.

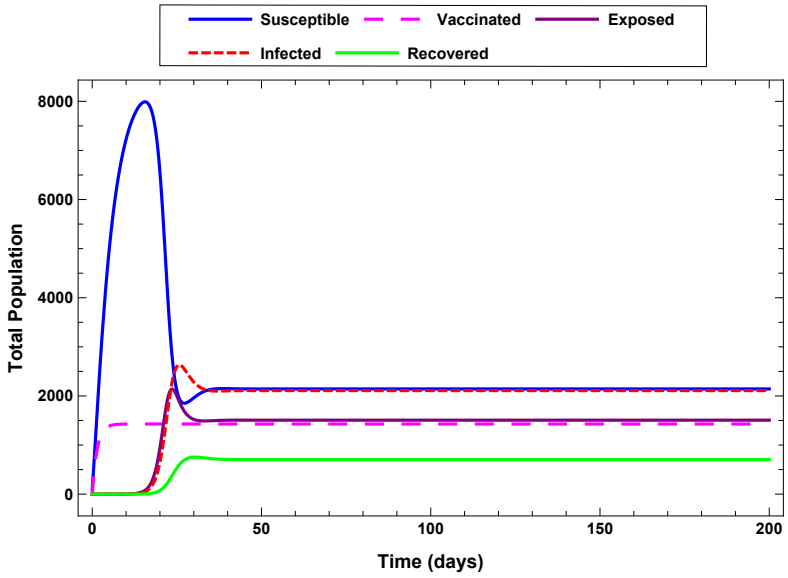

Fig. 6. Shows that as the rate at which the vaccine wanes off increases, the number of the susceptible class increases at a decreasing rate as the rate of treatment increases.

10 Discussion and Conclusion

In this work, we studied the impact of preventive vaccination and treatment on the dynamics of diarrhea disease using a mathematical model. The disease free and endemic equilibria were obtained and the basic reproduction number R_o computed. The result of the quantitative analyses showed that the disease-free equilibrium is both locally and globally asymptotically stable if $R_o < 1$ and $R_o \leq 1$ respectively and unstable otherwise. On the other hand, the endemic equilibrium is stable whenever $R_o > 1$. The implication of this is: Diarrhea can be controlled via the use of vaccination and treatment if the basic reproduction number is below unity, irrespective of the initial number of infection in the population. However, if the reproduction number exceeds unity, then diarrhea will persist in the population. The result of the sensitivity analysis revealed that the contact rate β is the most sensitive parameter of the basic reproduction number with positive index i.e. the value of β has the greatest effect on the reproduction number, and hence the prevalence of the disease in the population. The result $X_{\beta}^{R_o} = 1.0$ implies that if β is increased (decreased) by 10%, then R_o will also increase (decrease) by 10%. Also very sensitive are the infectivity rate of the exposed individuals σ and the vaccination rate ρ with positive and negative indices respectively. The result $X_{\rho}^{R_o} = -0.8182$ implies that if ρ is increased (decreased) by 10% then the R_o will decrease (increase) by 8.182%. The sensitivity indices of other parameters can be interpreted in similar manner. The results of the numerical simulations, were shown graphically in Figure 2 to 6. In figure 2, 3, 4 and 6 the effect of vaccine waning rate ω were shown. Both figures 2 and 3 showed that increment in the rate of vaccine waning results in increment in the population of both infected and exposed individuals respectively. Also, as shown in figure 6, this increment in the rate of vaccine waning leads to increase in the number of susceptible individuals. This implies that the more the rate of waning of vaccine, the more the number of those that are prone to diarrhea disease in the population. Thus if the waning rate can be reduced, then the number of those that get exposed and infected with diarrhea can be reduced. Also, the number of individuals that are prone to the disease would be reduced and more people can be protected. Furthermore, the effect of treatment rate τ on the dynamics of diarrhea disease were investigated and the results shown depicted in figures 4 and 5. Figure 4 showed that the higher the rate of treatment the lower the infected population. This implies that

increasing the rate of treatment decreases the number of infective in the population. Also, this increase in the rate of treatment leads to a corresponding increase in the number of recovered individuals as depicted in figure 5. In conclusion, in order to have a successful combat against diarrhea disease in the population, efforts have to be made by policy makers, health practitioners and the entire populace to bring down the threshold value, R_o (the basic reproduction number) below unity. This can be achieved through lowering the contact rate and increasing the rate and coverage of vaccines and vaccination programs, as indicated by the results of the sensitivity analysis conducted in this study. Also, as suggested by the results of the numerical simulations, efforts has to be made to come by vaccines whose waning rates are very reduced and also to target treating more infected individual. To future studies, we shall work on which of these control measures is both optimal and cost-effective.

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Competing Interests

Authors have declared that no competing interests exist.

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