Dynamical Analysis of a Rotavirus Infection Model with Vaccination and Saturation Incidence Rate

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Abstract. In this paper, we present and analyze a SVIR epidemic mathematical model for rotavirus infection with vaccination and saturated incidence rate. Dynamical analysis of this model is done by determining the equilibrium point and stability of the equilibrium point. The model exhibits two equilibrium points, i.e. disease free and endemic equilibrium. The basic reproduction number $R_v$ and $R_0$ has been obtained. The stability of the disease free and endemic equilibrium exists when the basic reproduction number less or greater than unity, respectively. Analytical result shows that those equilibrium points are locally asymptotically stable under certain condition. It is proved that the disease free equilibrium is globally stable when the value of basic reproduction number $1 < R_v$ and $R_0 < 1$, respectively. Numerical simulations are presented to support and complement the theoretical results.

1. Introduction

Rotavirus is considered a directly transmitted disease due to its high infectivity. It has been recognized for about 40 years as the most important cause gastroenteritis and diarrhea in infants and young children in both industrialized and developing countries. Rotaviruses are transmitted primarily by the fecal-oral route both through close person-to-person contact and through contaminated environment-to-person [1-2]. Rotavirus generally infects unvaccinated children initially between 6 and 24 months of age, and nearly always before 5 years of age. The incubation period of rotavirus infection is about two days [3]. Rotavirus has infected millions of children worldwide. In high-income countries, rotavirus infections result in few deaths but still constitute a substantial healthcare burden and can cause severe morbidity. The primary mode of transmission is the passage of the virus in the stool of an infected individual to the mouth of another individual (via contact with contaminated hands, surfaces, or objects) [4-5].

Mathematical model is one of the most important tools in analyzing the epidemiological characteristics of infectious disease. It can provide some useful insights about the dynamics of the disease. Regarding mathematical modelling and dynamic analysis of the spread of Rotavirus disease, several researchers have conducted studies with different studies. In 2006, Shim et al. have introduced the model of rotavirus infection that includes the impact of breastfeeding, seasonality, and the possibility of control by vaccination with standard incidence rate [6]. In 2015, Namawejje et al.[7] proposed the model of rotavirus infection incorporate three doses vaccination and treatment with bilinear incidence rate. A new mathematical model for rotavirus infection that incorporate vaccination with bilinear incidence rate has developed and comprehensively analyzed by Omondi et al.[8].
In this paper, we will study the mathematical model for rotavirus infection model that incorporates vaccination with saturated incidence rate. The model is a generalization of the SVIR epidemic model proposed by Purnomo and Darti [9]. The generalization is performed by including the saturated incidence rate when infected individuals make contact with vaccinated individuals. The saturated incidence rate can interpret the physiological effect from the behavioral change of the susceptible individuals when their number increase or from the crowding effect of the infective individuals [10-11]. Moreover, the saturated incidence rate is more reasonable than bilinear incidence rate because it includes the effect of behavioral change and crowding effect of the infective individuals. Beside of it, by choosing suitable parameters using this rate can prevent the unboundedness of the contact rate [12].

2. Mathematical Model
In this section, we investigate the basic model formulation by dividing the total population into four compartments, namely susceptible $S(t)$, vaccinated $V(t)$, infected $I(t)$ and recovered individuals $R(t)$. Thus, at time $t$, the total population: $N(t) = S(t) + V(t) + I(t) + R(t)$. The movement between the classes is shown in Figure 1. The recruitment rate into susceptible class is $(1 - \rho)\Lambda$ and recruitment rate vaccinated class is $\rho\Lambda$. Parameter $\delta$ and $\omega$ denote the transmission susceptible to vaccinated and the vaccine efficacy wanes, respectively. The expected decrease in the risk of infection as a result of vaccination at the rate $\varepsilon \in (0,1)$. Here, $\mu$ and $\tau$ respectively denote the natural mortality rate of human and rotavirus-induced mortality rate of infected human. Infected individuals may recover at rate $\gamma$. The effective contact rate for disease transmission is given by $\beta$. The saturation constant of incidence transmission $S$ and $I$ is given by $\alpha_1$, while the saturation constant of incidence transmission $V$ and $I$ is given $\alpha_2$.

![Figure 1. Compartmental flow of a mathematical model for Rotavirus infection](image)

Keeping and view the definitions and variables discussed and the transition diagram shown in Figure 1, our proposed model is governed by the following system of ordinary differential equations:

$$\frac{dS}{dt} = (1 - \rho)\Lambda - \frac{\beta SI}{1 + \alpha_1 I} - \delta S + \omega V - \mu S,$$

$$\frac{dV}{dt} = \rho\Lambda + \delta S - \frac{\varepsilon \beta VI}{1 + \alpha_2 I} - (\omega + \mu)V,$$

$$\frac{dI}{dt} = \frac{\beta SI}{1 + \alpha_1 I} - (\tau + \mu)I,$$

$$\frac{dR}{dt} = \gamma I.$$

(1)
\[
\frac{dI}{dt} = \frac{\beta SI}{1 + \alpha_1 I} + \frac{\epsilon \beta VI}{1 + \alpha_2 I} - (\tau + \gamma + \mu)I,
\]

\[
\frac{dR}{dt} = \gamma I - \mu R,
\]

with initial condition \( S(0) \geq 0, V(0) \geq 0, I(0) \geq 0 \) and \( R(0) \geq 0 \). Here, all parameter values of the model must be non-negative.

Since \( N = S + I + V + R \), we have
\[
\frac{dN}{dt} = \Lambda - \mu N - \tau I. \tag{2}
\]

In the absence of infection, from equation (2) we get
\[
\frac{dN}{dt} = \Lambda - \mu N.
\]

so that \( N \) would approach carrying capacity \( \frac{\Lambda}{\mu} \).

Model (1) is mathematically well posed and its dynamics can be considered in a proper region \( \Omega = \{(S, V, I, R) \in \mathbb{R}^4_+; N \leq \frac{\Lambda}{\mu}\} \).

3. Existence of Equilibrium Points and Basic Reproduction Number

Since the equation for the variable \( R \) in model (1) is independent of the other equations, their dynamical behaviors are determined by the following system:
\[
\frac{dS}{dt} = (1 - \rho)\Lambda - \frac{\beta SI}{1 + \alpha_1 I} - \delta S + \omega V - \mu S,
\]
\[
\frac{dV}{dt} = \rho \Lambda + \delta S - \frac{\epsilon \beta VI}{1 + \alpha_2 I} - (\omega + \mu)V,
\]
\[
\frac{dI}{dt} = \frac{\beta SI}{1 + \alpha_1 I} + \frac{\epsilon \beta VI}{1 + \alpha_2 I} - (\tau + \gamma + \mu)I. \tag{3}
\]

The equilibrium points of (3) are obtained by equating the derivatives to zero and solving for the variables. When \( I = 0 \), a solution of (3) namely the disease-free equilibrium (DFE) of system (3) is obtained as
\[
E_0 = (S_0, V_0, I_0) = \left( \frac{\Lambda(1 - \rho) + \omega}{\mu(\delta + \omega + \mu)}, \frac{\Lambda(\rho \mu + \delta)}{\mu(\delta + \omega + \mu)}, 0 \right).
\]

The other equilibrium point of (3) is called endemic equilibrium and it is as follows:
\[
E^* = (S^*, V^*, I^*)
\]

where
\[
S^* = \frac{(\Lambda - (\tau + \gamma + \mu)I)(\omega + \mu)(1 + \alpha_2 I) + \epsilon \beta I)}{\mu((\delta + \omega + \mu)(1 + \alpha_2 I) + \epsilon \beta I)}
\]
\[
V^* = \frac{\mu \rho \Lambda + \delta (\Lambda - (\tau + \gamma + \mu)I)(1 + \alpha_2 I)}{\mu((\delta + \omega + \mu)(1 + \alpha_2 I) + \epsilon \beta I)}
\]
The value of $l^*$ is the root of polynomial $q_4 l^{*2} + q_2 l^{*} + q_3 = 0$, where

$$q_1 = \delta \alpha_1 (\tau + \gamma + \mu) \left( \omega \alpha_2 + \epsilon (\beta + \alpha_1 (\delta + \mu)) \right) - \left( \epsilon (\beta + \alpha_1 \delta) + \alpha_2 (\omega + \mu) \right)$$

$$+ \left( \beta (\tau + \gamma + \mu) + \alpha_1 (\delta + \mu) \right)$$

$$q_2 = \delta \alpha_1 (\tau + \gamma + \mu) (\omega + \epsilon (\delta + \mu)) + \left[ \rho \Lambda \beta + \delta (\tau + \gamma + \mu) \right] \left[ \alpha_2 \omega + \epsilon (\beta + \alpha_1 (\delta + \mu)) \right]$$

$$- \left[ \beta \epsilon + \alpha_2 (\omega + \mu) + \alpha_1 + \delta \epsilon \right] \left[ (\tau + \gamma + \mu) (\delta + \mu) - (1 - \rho) \Lambda \beta \right]$$

$$+ \left[ \beta \epsilon + \omega + \mu \right] \left[ \beta (\tau + \gamma + \mu) + \alpha_1 (\delta + \mu) \right]$$

$$q_3 = \left[ \rho \Lambda \beta + \delta (\tau + \gamma + \mu) \right] \left[ \omega + \epsilon (\delta + \mu) \right] - \left[ \omega + \mu + \delta \epsilon \right] \left[ (\tau + \delta + \mu) (\delta + \mu) - (1 - \rho) \Lambda \beta \right].$$

The endemic equilibrium exists when

i. $q_2^2 - 4q_1 q_3 > 0$ and $\frac{q_3}{q_1} < 0$ or

ii. $q_2^2 - 4q_1 q_3 = 0$ and $-\frac{q_2}{q_1} > 0$.

In epidemiological, determining the basic reproduction number is important to analyze the spread of disease. The basic reproduction number is defined as the expected number of secondary cases produced by a single (typical) infection in a completely susceptible population [13]. In this paper, specifically we define the reproduction number $R_v$ of the model (3) as the basic reproduction number in the presence of vaccination. If no such vaccination is employed, the basic reproduction number is defined as $R_0$. We calculate the basic reproduction number using the method of van Driessche and Watmough, similarly as in [13], which yields $R_v$ and $R_0$ as following:

$$R_v = \frac{\beta \Lambda}{\mu (\tau + \gamma + \mu)} \left[ \frac{\mu (1 - \rho + \rho e) + \omega + \epsilon \delta}{\mu + \omega + \delta} \right], \quad (4)$$

$$R_0 = \frac{\beta \Lambda}{\mu (\tau + \gamma + \mu)}. \quad (5)$$

Equation (4) can be expressed in (5) as following:

$$R_v = R_0 \left[ \frac{\mu (1 - \rho + \rho e) + \omega + \epsilon \delta}{\mu + \omega + \gamma} \right]. \quad (6)$$

From the expression of $R_v$, we can see that the vaccination from birth as well as vaccination of susceptibles both have positive impact on the reduction of new infections. If $\epsilon = 1$ then $R_v = R_0$ which implies that vaccination is not important and this should not be the case in this study, as we assumed $\epsilon \in (0, 1)$ . From this fact, the value of $\frac{\mu (1 - \rho + \rho e) + \omega + \epsilon \delta}{\mu + \omega + \delta} < 1$, so $R_v < R_0$. It means that vaccination can reduce the spread of rotavirus infection.

4. Stability Analysis of Equilibria

4.1 Local stability of the disease-free equilibrium.

**Theorem 1.** The disease-free equilibrium (DFE) is locally asymptotically stable if $R_v < 1$. 

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Proof:
The Jacobian matrix at DFE is given by
\[
J(E_0) = \begin{bmatrix}
-(\delta + \mu) & \omega & -\beta S \\
\delta & -(\omega + \mu) & -\varepsilon BV \\
0 & 0 & -(R_v - 1)(\tau + \gamma + \mu)
\end{bmatrix}.
\]
The characteristic equation of system (3) at \(E_0\) is of the following form \(J(E_0) - \lambda I = 0\) or
\[
\lambda^3 + P_1\lambda^2 + P_2\lambda + P_3 = 0
\]
where the coefficients are given by
\[
P_1 = (1 - R_v)(\tau + \gamma + \mu) + \delta + \omega + 2\mu,
P_2 = (1 - R_v)(\tau\delta + 2\tau(\mu + \omega) + \gamma(\delta + 2\mu + \omega) + \mu(\omega + \delta + 2\mu) + 2\mu(\omega + \delta + \mu),
P_3 = (1 - R_v)(\tau\mu(\delta + \omega) + \gamma\mu(\delta + \omega) + \mu^2(\gamma + \delta + \omega + \tau + \mu)).
\]
We observe that \(P_1 > 0\) automatically satisfies if \(R_v < 0\). Both conditions \(P_3 > 0\) and \(P_2 - P_3 > 0\) satisfies if \(R_v < 0\). Hence by Routh-Hurwitz criterion [14], all the eigenvalues of the system (3) has negative real part and the disease free equilibrium point at \(E_0\) is locally asymptotically stable.

4.2 Global stability of the disease free equilibrium

The global stability of the disease-equilibrium \(E^0\) is proved by using Lyapunov function.

Theorem 2. The disease-free equilibrium of (1) is globally asymptotically stable in \(\Omega\) if \(R_v < 1\), where \(\Omega\) is feasible region of equation (1).

Proof:
We construct the following Lyapunov function:
\[
L : \{(S,V,I,R) \in \Omega\} \rightarrow \mathbb{R}
\]
by
\[
L(S,V,I,R) = (\omega + \mu)I
\]
(8)
Then, differentiating equation (8) with respect to time \(t\), we obtain
\[
L' = (\omega + \mu) \left[ \frac{\beta SI}{1 + \alpha I} + \frac{s\beta I}{1 + \alpha R} - (\tau + \gamma + \mu)I \right]
\]
\[
= (\omega + \mu) \left[ \beta \left( \frac{S}{1 + \alpha I} + \frac{\varepsilon V}{1 + \alpha I} \right) - (\tau + \gamma + \mu)I \right]
\]
\[
\leq (\omega + \mu) \left[ \beta S + eV - (\tau + \gamma + \mu)I \right]
\]
\[
\leq (\omega + \mu) \left[ \beta S + V - (\tau + \gamma + \mu)I \right]
\]
\[
< (\omega + \mu) \left[ \beta S + V - (\tau + \gamma + \mu)I \right]
\]
\[
= (\omega + \mu) \left[ \beta \frac{\lambda_{max}}{\mu} - (\tau + \gamma + \mu)I \right]
\]
\[
= (R_v - 1)(\omega + \mu)(\tau + \gamma + \mu)I.
\]
Since \(R_v < R_0\) in \(\varepsilon \in (0,1)\) then we have \(L' < (R_v - 1)(\omega + \mu)(\tau + \gamma + \mu)I\).

The value of \(L' < 0\) if \(R_v < 1\), then the disease-free equilibrium of (3) is globally asymptotically stable in \(\Omega\).

4.3 Local stability of the endemic equilibrium

The Jacobian matrix \(J(E^*)\) at endemic equilibrium point \(E^*\) is given by
The characteristic equation is
\[ \lambda^3 + A_1 \lambda^2 + A_2 \lambda + A_3 = 0 \]  
(9)
where
\[ A_1 = -j_{11} - j_{22} - j_{33} \]
\[ A_2 = j_{11}j_{22} + j_{11}j_{33} - j_{22}j_{33} + j_{23}j_{32} - j_{12}j_{21} - j_{13}j_{31} \]
\[ A_2 = -j_{11}j_{22}j_{33} + j_{11}j_{33}j_{32} + j_{12}j_{21}j_{33} - j_{12}j_{23}j_{31} - j_{13}j_{21}j_{32} \].

The Routh-Hurwitz criterion [14] requires \( A_1 > 0, A_2 > 0, A_3 > 0, \) and \( A_1A_2 - A_3 > 0 \) as the necessary and sufficient conditions for the locally asymptotical stability, i.e., all roots of the polynomial (8) have negative real parts.

5. Numerical Simulations
In this section to demonstrate the theoretical results obtained in this paper, we present some numerical simulations. The dynamical behaviors of the model are observed by considering a set parameter values and initial conditions. All the parameter value in this paper are chosen hypothetically due to unavailability the real data work. In Table 1 we give the detailed explanation of the parameter and values used in the model. The numerical results are shown in Figures 2 to 4.

| Parameters | Description | Value |
|------------|-------------|-------|
| \( \Lambda \) | Recruitment rate of humans | 0.9 |
| \( \rho \) | Recruitment of vaccinated individuals | 0.5 |
| \( \mu \) | Natural death of humans | 0.09 |
| \( \tau \) | Rotavirus-induced deaths | 0.01 |
| \( \beta \) | Disease contact rate | 0.002 |
| | | 0.2 |
| \( \gamma \) | Recovery rate | 0.001 |
| \( \varepsilon \) | Expected decrease in the risk of infection | 0.001 |
| \( \omega \) | Vaccine efficacy waning rate | 0.01 |
| \( \delta \) | Vaccination rate | 0.08 |
| \( \alpha_1 \) | Saturation constant (S-I) | 0.03 |
| \( \alpha_2 \) | Saturation constant (V-I) | 0.2 |
Figure 2 provides the phase portraits of the dynamic rotavirus infection. It can be seen that when \( \beta = 0.002 \) the value of \( R_0 = 0.0198 < 1 \) and \( R_v = 0.0606 < 1 \), the disease free equilibrium is stable at \( E_0 = (3.05, 6.94, 0) \) locally as well as globally asymptotically stable as seen in Figure 2(a). From epidemiological point of view the disease dies out from the population. If the basic reproduction number \( R_0 = 19.8020 > 1 \) and \( R_v = 6.0644 > 1 \) (in this case \( \beta = 0.2 \) ), as depicted in Figure 2(b), we can say that the unique endemic equilibrium exist and is locally asymptotically stable at \( E^* = (0.509, 4.87, 4.11) \). From biological point of view, it means that the disease will persist in the population.

Figure 3 illustrates the dynamical behavior of infected class for different values of \( \alpha_1 \) (saturation constant \( S-I \)) and \( \alpha_2 \) (saturation constant \( V-I \)). From this figure, we see that the saturation constant rate ultimately affecting the dynamics of the model system.

Figure 4(a) is plotted with \( \alpha_2 = 0.2 \) for varying value for \( \alpha_1 (= 0.003; 0.3, 3) \) , while figure 4(b) is plotted for varying value \( \alpha_2 (= 0.001; 0.1; 10) \). From figure 4(a), we see the effect of saturation constant between susceptible and infected individuals, that when \( \alpha_1 \) increases, the population of rotavirus infected individuals decreases. Moreover, it is observed from figure 4(b) that when \( \alpha_2 \) increases the size of infected class decreases, however the effect very slight. Hence the saturation constant between
vaccinated and infected individuals given insignificant effect for the dynamical behavior of the model. In this fact, minimizing contacts between infected and susceptibles are highly recommended. Furthermore, our numerical simulations have performed the effectiveness of vaccination and self-protect susceptible and vaccinated class as the effort to reduce the spread of rotavirus infection.

![Figure 4](image.png)

**Figure 4.** Effect of saturation constant. (a) Size of infected class for different values of $\alpha_1$ when $\alpha_2 = 0.2$. (b) Size of infected class for different values of $\alpha_2$ when $\alpha_1 = 0.03$

6. Conclusions
We have formulated an rotavirus infection model incorporating a vaccination and saturation incidence rate and investigated their dynamical behaviors. Based on analysis and numerical simulation, it has been proved that disease-free equilibrium points are locally and globally asymptotically stable. The endemic equilibrium is locally asymptotically stable under certain conditions. Numerical results for the model carried out, in order to illustrate the theoretical results. For future research, suggested to investigate the dynamics of this model with re-infection.

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