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# Epidemic Model Formulation, Analysis and Simulation of Rotavirus Diarrhea for Prevention

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***Authors' contributions***

*This work was carried out in collaboration between all authors. Author SM designed the study and laid out the methodology of the project. Author EL guided the major mathematical analysis for the study. Author OC carried out the study, literature review, statistical and mathematical analyses, worked out the results and wrote the manuscript. All authors read and approved the final manuscript.*

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## Abstract

**Aims:** Practical employment of epidemic models for rotavirus diarrhea is the aim of the study so that prevention is attained more swiftly thus reducing global disease burdens, mortality rates and financial burdens due to treatment.
**Study Design:** The design of this study is purely mathematical.
**Place and Duration of Study:** Data for this study was collected from the National Health Laboratories, Botswana and the study was completed at the University of Botswana during the duration from August 2012 to October 2014.
**Methodology:** Using pathogenesis study of Rotavirus from literature, the epidemic model was built in the continuous mode, taking reference from the basic SIR model. The model was mathematically analysed and simulated in MATLAB.
**Results:** Simulation results were in line with the epidemic theory and showed that as long as the value of

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the basic reproduction number ($R_0$) is kept to be lower than one, an epidemic can be avoided. If this value goes above one, then an epidemic is likely to break out.
**Conclusion:** The model was successfully built, analysed and simulated showing results that matched the epidemic theory. From this stage, the model can be easily implemented on different platforms for prevention mechanisms to be exploited.

*Keywords: Epidemic modelling; rotavirus; modelling and simulation; diarrhoea.*

# 1 Introduction

Transmissible diseases repeatedly produce significant world threats due to the diffusive nature of being transferred from human to human. Such transmissible diseases have accounted for 16% of world-wide deaths in 2008 [1]. In general, such diseases are introduced into a closed population by the insertion of an infective individual or by an external vector [1]. Examples of external vectors include water, air, body fluids, insects such as mosquitoes or biological vectors which carry infectious agents such as protozoa, bacteria or virus. Being essential to life, water is has a major contribution to the diffusion of infectious diseases. Water related diseases are spread through different uses of water including bathing, agriculture, washing, food preparation and drinking. Outbreaks of these diseases primarily result from the direct or indirect ingestion of contaminated water which contains the pathogenic micro-organisms.

## 1.1 Diarrhoea – Global and national burden

Diarrhoea is a disease that is characterized by the unusual passage of fluid stool three or more times in a day and is transferred via contaminated food and water. There are up to 1.7 billion clinicalcases of diarrhoea annually across the globe [2]. The disease is known to cause severe illness in children under five and is listed as the second leading cause of mortality in children under the age of five causing around 700000 child deaths per year [2].

Countries in the United Kingdom report about 13000 clinical cases of rotavirus diarrhoea annually in children [3]. Australia has had high numbers of rotavirus infections of up to 32000 clinical cases every year [4]. In the whole of Africa, 15% of child mortality is caused by rotavirus diarrhoea [5].

It can be generally misunderstood that the hazards of a water-borne disease like diarrhoea will be of minimal levels. Due to the presence of various water reservoirs in the country, Botswana also suffers significantly due to water-borne diarrhoeal diseases. These water reservoirs contribute to the transmission of infectious diseases to a noteworthy extent mainly because their vicinity acts as habitats for a large percentage of the country population. More to this, the water sanitation systems have only been properly allocated in the middle and upper income residences [6] with only a limited portion of the population being subject to adequate sanitation which is 53% in the urban and 18% in the rural areas [7]. When a high percentage of the population lives and depends on open water sources, the risk of diarrhoea also increases appreciably. This possible association was implied in the International Disease Surveillance and Response Center in Botswana that reported about 15000 cases and 200 deaths due to diarrhoea in 2012 [8]. This shows that apart from the numerous treatment methods available, necessary prevention and precaution methods need to be employed so as to avoid diarrhoeal hazards in Botswana.

Diarrhoea can be caused by a variation of pathogens including many types of virus, bacteria and protozoa. One of the most perilous pathogen in relation to diarrhoea is the rotavirus. Rotavirus is classified intoseveral serotypes which can cause viral gastroenteritis. Gastroenteritis is the inflammation of the gastrointestinal tract and has common symptoms of diarrhoea, vomiting, fever and abdominal pains [9]. Rotavirus is the leading cause of diarrhoea around the world and results in approximately 527000 deaths annually. One of the most hazardous diseases causing about 900000 deaths annually happens to be malaria. The figures produced by rotavirus can easily be compared to this mortality rate, hence proving the importance of the study and

prevention of the disease. On the national level, Rotavirus remains the leading pathogen for diarrhoea infections in Botswana too [10].

Although the pathogen usually infects the immunosuppressed individuals like small children and older people, adults and youth are also at high risk of infection. Transmission of the virus occurs mainly through the fecal-oral route but indirect transmission through any object that is touched with contaminated hands, e.g. toys, furniture, door knobs and sink surfaces is also common. Rotavirus is stable in the environment thus if sanitation is poor, the contaminated surfaces can continue to spread the pathogen.

### 1.2 Overview of the model

As the viral burden of rotavirus is quite high globally, a system that incorporates the dynamics and transmission of rotavirus diarrhoea will prove to be of great advantage. The necessary prevention strategies can be based on the results provided by the system, thus the transmission of the virus can be controlled.

Disease dynamics can be well understood scientifically using Epidemic modelling. As the risk of infectious diseases is increasing throughout the world, models depicting the respective transmission are becoming more functional. These models are simply tools that are used to predict the infections mechanisms and future outbreaks of diseases. Although many treatment methods are employed across the globe to combat epidemics such as the diarrhoea, many individuals fail to receive or respond to these methods. Thus, the epidemic models focus more on the prevention mechanisms. Prevention is exceptionally necessary because in the context of diarrhoea, treatment methodologies may not be very helpful in eradicating the disease and its transmission. The most common treatments given for diarrhoea are mainly supportive through the use of Oral Rehydration Therapies (ORT) using Oral Rehydration Salts (ORS). Antibiotics are not effective in case of viral infections and ORT is not readily available to everyone and due to lack of knowledge or poor administration and reconstitution of the ORS. A recent study in Botswana showed that only a particular portion of the population was able to receive the ORS and out of this population, 74% did not have the knowledge to correctly administer the treatment [11]. Therefore, despite increasing the availability of treatment methods like ORS, the expected result of reduction in the number of diarrhoeal cases was not achieved. For such situations, a prevention mechanism is highly recommended which can be achieved through the model like the one proposed in this study. Apart from avoiding untimely deaths across the globe, prevention is essential to lighten the economic burden of numerous countries. Diseases like diarrhoea are perceived not to have any significant financial or economic burden due to the simplicity of treatment methods. But due to heavy burdens of the pathogens like rotavirus, significant financial amounts are being used for treating diarrhoea. The estimated medical costs due to rotavirus range up to $264 million in the US [12].

In order to minimize the effects of the rotavirus, a proposed system is presented herewith that designs the dynamics of rotavirus diarrhoea. The model is of predictive nature that shows the possibility of future outbreaks thus enhancing prevention. An active map is integrated with the model such that the predictions of the model can be directly related to the various areas of the country. A communication platform is also included in the model which presents an automated alert service to the general public. Any information regarding the disease can be communicated easily and fast enough to the public using this facility. In this present study, the main focus is on building the initial stage of the system. This stage consists of the continuous model which is analyzed and simulated to show fidelity. The continuous model acts as basis on which the above described system can be built up.

## 2 Background

### 2.1 Other models

Epidemic modelling begun in 1927 when Karmack and McKendrick introduced the first compartmental epidemic model consisting of three compartments - the Susceptible-Infected-Recovered (SIR) model [6]. In its simplest form, the SIR model can be shown as follows:

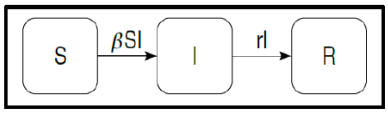


**Fig. 1. The basic SIR model**

The above schematic diagram can be described using a set of ordinary differential equations and some defined parameters. β represents the infectivity parameter and r is the rate of recovery.

$$\frac{dS}{dt} = -\beta SI \tag{1}$$

$$\frac{dI}{dt} = \beta SI - rI \tag{2}$$

$$\frac{dR}{dt} = rI \tag{3}$$

Utilizing the above traditional model as a base, many other models have been formulated in the continuous perspective. The Biomedical Modelling Centre, LA has modeled influenza using the SIR model [13]. Simple extensions and modifications have been used here to include factors such as demographics – in/out flow of individuals from the population. These alterations in the model make it possible to calculate the incidence rate of influenza for different transmission rates [14]. Pathogens like the Hepatitis C virus give rise to liver diseases. The University of New South Wales, Australia, has created an agent based model to describe the dynamics of this virus. This model uses inputs such as age, sex and immigrant status and delivers outputs such as infections, cure and death rates related to the disease [15].

Data needed for modelling epidemics is usually collected at discrete times [16], thus discrete models prove to be equally informative for epidemics. Evaluation in discrete models is done for the next time interval. This property discloses the predictive ability of the model and thus proves to be very advantageous.

Some examples of discrete models in epidemics are as follows. Severe Acute Respiratory Syndrome (SARS) in China has been modelled using discrete modelling by adding three more compartments to the SIR model, namely the exposed, quarantined and diagnosed individuals [17]. In this model, the importance of the quarantine for SARS was established. Certain diseases can be modelled using only two compartments, the susceptible and infected classes. Discrete SI and SIS models have been found to be very useful in determining the dynamical behaviour of diseases such as malaria or gonorrhoea [18]. An extended study to these models has been done by adding the effects of seasonality in the model. This work uses the fact the seasonal factors have an important relation with the various species in the environment [19].

## 2.2 Disease epidemiology and pathogenesis

Copious efforts are being employed worldwide to eradicate the cause and effects of infectious diseases. Water-related diseases occupy a significant position in the context of infectious disease hazards. Among the numerous water–related diseases, diarrhoea remains one of the diseases that causes alarming morbidity rates annually. Major prevention of the disease can be done through proper sanitation but according to the non-profit organization water.org, globally, only 10% of the discarded water is treated and the rest goes into water bodies for disposal. This largely increases the possibility of disease spread. Diarrhoea is caused by bacteria, virus or other parasitic organisms and is predominantly transmitted through contaminated food and water. In order to reduce the speed of the transmission of this disease, it is necessary to recognize the disease characteristics and pathogenesis as this forms the basis of the methods that can be implemented to fight the diffusion of the pathogens.

Diarrhoea is a condition that involves an increment in the frequency of bowel movements and fluidity of stool. Apart from this major symptom, diarrhoea is associated with symptoms such as abdominal pain, fever and most important of all dehydration [20]. Dehydration results in the loss of many necessary salts and chemicals along with water. This condition of loss can become dreadfully harsh resulting in serious circumstances ranging from heavy malnutrition to even death. Immuno-suppressed individuals are at higher risks of severity.

Diarrhoea is caused by a variation of pathogens and manifests as a disease on its own or as a symptom of another disease. As per the national records, the four main pathogens that are attributed to diarrhoea in Botswana are the rotavirus, the protozoan cryptosporidium and the bacteria shigella and salmonella. Out of the above list, rotavirus occupies the highest position majorly causing diarrhoea in Botswana.

The recorded infective dose of rotavirus is 10-100 viral particles [21] and trillions of virions are released from an infected individual during one diarrhoeal episode. Rotavirus involves an incubation period of about two to three days. During this time, the person remains asymptomatic but due to its high contagiousness, rotavirus is spread during this period too. Transmission of rotavirus occurs through contaminated food, water or any object that is touched with contaminated hands, e.g. toys. An infected person remains highly contagious up to ten days after the onset of symptoms and may continue to shed the virus at slower rates after this period [22]. The main treatment given to the infected people with rotavirus is rehydration. As of date, there are no particular medicines that can be used to treat the rotavirus. The mainstay of treatment is supportive through oral rehydration.

# 3 Methodology – Model Development

## 3.1 Description

The model considered herewith is based on the SIR model with a few extensions and additions. There are five compartments included in the system, which can be described as follows:

1. Susceptible (S) – this represents the individuals of the whole human population that can catch the disease
2. Asymptomatic Infected ($I_A$) – these are the individuals who have the pathogen within them but so not show any symptoms of the disease, yet they continue to contribute to the pathogen population
3. Symptomatic Infected ($I_S$) – the individuals who have the pathogen and show symptoms of the disease. This group also contributes to the pathogen population
4. Recovered (R) – this class consists of the individuals who have recovered from the disease

A schematic illustration of the model is given in Fig. 2 and all the associated parameters are described in Table 1.

In a group of susceptible people, a certain proportion can be infected to either become symptomatic or asymptomatic infectious. Both these classes contribute to the infection at different rates. After a certain period of time, depending on the dose of the virus in the body, the asymptomatic individuals become symptomatic. With the help of medication and rehydration therapies or body immunity, the infectious people become recovered. For some time, the body maintains an immunity level but gradually, this level drops and the recovered population again becomes susceptible to the disease.

In the described system, the following assumptions are considered:

1. The shedding rate of virus by the infected population consists of both direct and indirect shedding.
2. All individuals are born as susceptible.

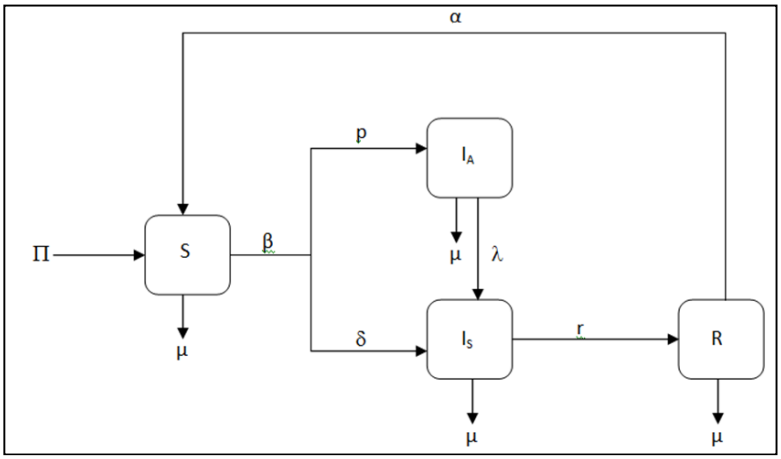


**Fig. 2. Schematic representation of model**

**Table 1. Description of parameters**

| Parameter | Description |
|---|---|
| β | Infectivity rate |
| α | Immunity loss rate |
| μ | Natural death rate |
| p | Probability of asymptomatic infection |
| r | Recovery rate |
| δ | Probability of symptomatic infection |
| Π | Population renewal |
| λ | Symptom gaining rate |

Using the above mentioned parameters and assumptions, the extended compartmental model can be defines using the following equations:

$$\frac{dS}{dt} = \Pi - \mu S - \beta S(pI_A + \delta I_S) + \alpha R \tag{4}$$

The rate of change of the susceptible population can be described using equation 4 above. A constant recruitment/birth rate $\pi$ provides new individuals in the class and a constant birth rate μ reduces the susceptible population. A certain proportion of the susceptible get infected at an infectivity rate β. Recovered individuals gradually join the susceptible class at an immunity loss rate α.

$$\frac{dI_A}{dt} = \beta pSI_A - \mu I_A - \lambda I_A \tag{5}$$

Equation 5 describes the change in the asymptomatic infectious population. A portion of the susceptible class at becomes asymptomatic infectious depending on the infectivity rate β and probability p. The above class gets reduced by a constant death rate μ and symptom gain rate λ.

$$\frac{dI_S}{dt} = \beta \delta SI_S - \mu I_S - rI_S + \lambda I_A \tag{6}$$

The above equation defines the symptomatic infectious population. This compartment is fed in by the susceptible population that becomes symptomatic at an infectivity rate β and probability δ. A constant death

rate μ reduces the symptomatic population and a certain percentage of the class becomes recovered at a recovery rate r.

$$\frac{dR}{dt} = rI_S - \mu R - \alpha R \tag{7}$$

The recovered compartment of the model can be described using the equation 7. As the symptomatic population recovers at the rate r, they get added to the recovered class. People die at a constant death rate μ from the recovered class at time t. A certain part of this compartment gradually loses its immunity at a rate α and moves on again to the susceptible class.

## 3.2 Calculations and analysis

The Virus-free equilibrium (VFE) is the state at which there are no infections in the population. For the population to be deprived of the pathogens, the infected states will be assumed to be zero.

$$\Rightarrow I_A = I_S = 0$$

Since there are is infectious population, it implies that there will be no recovered population either. Therefore R = 0. At this state, the only non-zero class is the susceptible class. At the VFE, all the classes will be denoted with an asterisk.

In order to get the asymptotic state, the right hand side of equation (4) will be equated to zero.

$$\Pi - \mu S^* = 0$$
$$S^* = \frac{\Pi}{\mu}$$

Thus, the VFE can be described as follows:

$$VFE = \left(\frac{\Pi}{\mu}, 0,0,0\right) \tag{8}$$

Firstly, for the above calculation, the classes that contain new infection parameters need to be considered. The susceptible and recovered classes are not related to new infections, thus, the Asymptomatic infectious and the Symptomatic infectious classes will be taken into consideration.

The new infections in the three classes mentioned above can be denoted in the matrix below, using equations (2) and (3).

$$\mathcal{F} = \begin{bmatrix} \beta p S I_A \\ \beta \delta S I_S \end{bmatrix}$$

The other changes in the same classes can also be shown in a matrix. Note that the signs of the respective terms will change in this case.

$$\mathbb{V} = \begin{bmatrix} (\mu + \lambda) I_A \\ (\mu + r) I_S \end{bmatrix}$$

Calculating the Jacobian Matrix of each of the above matrices, we get:

$$F = \begin{bmatrix} \beta p S & 0 \\ 0 & \beta \delta S \end{bmatrix}$$

$$V = \begin{bmatrix} (\mu + \lambda) & 0 \\ 0 & (\mu + r) \end{bmatrix}$$

At the VFE, the replacement $S^* = \frac{\Pi}{\mu}$ can be used and the above matrices can be written as:

$$F = \begin{bmatrix} \frac{\beta p \Pi}{\mu} & 0 \\ 0 & \frac{\beta \delta \Pi}{\mu} \end{bmatrix}$$

The Basic reproduction number can be calculated using the Jacobian matrix and next generation method. The expression of $R_0$ is found out to be:

$$R_0 = \frac{\beta p \Pi}{\mu(\mu + \lambda)} \tag{9}$$

The Routh Hurwitz stability test examines the system by ensuring that all the roots of a given polynomial are in the left half pane of the axis i.e. negative values (real or imaginary). For any physical system, the variations within are described using equations. In epidemic modelling, ODEs are used to describe the motion of the population in the prescribed compartments. Solutions of these equations are stable and this can be confirmed using the Routh Hurwitz method.

**Theorem 1:** If the roots have negative values, this will indicate a stable system.

To work out the stability analysis of the rotavirus system, firstly we consider the set of equations that describe the model. For this step we assume that the immunity loss rate, α is approximately 0, for calculation purposes.

$$f1 = \Pi - \mu S - \beta S(pI_A + \delta I_S)$$
$$f2 = \beta p S I_A - \mu I_A - l I_A$$
$$f3 = \beta \delta S I_S - \mu I_S - r I_S + l I_A$$
$$f4 = r I_S - \mu R$$

The Jacobian matrix of the system above is evaluated as:

$$J = \begin{bmatrix} -\mu - \beta(pI_A + \delta I_s) & -\beta \delta S & -\beta p S & 0 \\ \beta \delta I_S & \beta S - \mu - r & k & 0 \\ \beta p I_A & 0 & \beta S - \mu - \lambda & 0 \\ 0 & r & 0 & -\mu \end{bmatrix} \tag{10}$$

To work out the stability of the DFE, we substitute the above Jacobian matrix with the DFE expression for rotavirus. Doing this we get,

$$E_0 = \left(\frac{\Pi}{\mu}, 0,0,0\right)$$

$$J(E_0) = \begin{bmatrix} -\mu & \frac{-\beta \Pi \delta}{\mu} & \frac{-\beta \Pi p}{\mu} & 0 \\ 0 & -\mu - r & k & 0 \\ 0 & 0 & \frac{\beta \Pi}{\mu} - \mu - \lambda & 0 \\ 0 & r & 0 & -\mu \end{bmatrix}$$

Subtracting the index matrix multiplied by the operating parameter γ, we get:

$$J(E_0) - \gamma I = \begin{bmatrix} -\mu-\gamma & \frac{-\beta\Pi\delta}{\mu} & \frac{-\beta\Pi p}{\mu} & 0 \\ 0 & \frac{\beta\Pi}{\mu}-\mu-r-\gamma & k & 0 \\ 0 & 0 & \frac{\beta\Pi}{\mu}-\mu-k-\gamma & 0 \\ 0 & r & 0 & -\mu-\gamma \end{bmatrix}$$

We use this matrix do derive the characteristic equation which is defined as:

$$E_\lambda = (-\mu-\gamma)\times(-\mu-\gamma)\times\left(\frac{\beta\Pi}{\mu}-\mu-r-\gamma\right)\times\left(\frac{\beta\Pi}{\mu}-\mu-\lambda-\gamma\right) \quad (11)$$

The roots of this equation are:

$$\gamma_1 = -\mu$$

$$\gamma_2 = -\mu$$

$$\gamma_3 = \frac{\beta\Pi}{\mu}-\mu-r = R_0\left(\frac{\mu+k}{\Pi}\right)-\mu-r$$

Using theorem 1, the results deducedare describe as:

The VFE of the rotavirus system is locally asymptotically stable when $R_0$ <1 and is instable if $R_0$> 1.

# 4 Results – Simulation and Discussion

The simulation of the above continuous model was done in order to establish the fidelity and stability of the model.

The value of the basic reproduction number is controlled using the infectivity rate. A calculated value of the infectivity rate when the basic reproduction number is one is used to manipulate an epidemic or non-epidemic state of the population.

For $R_0$ = 1,

$$\beta p\Pi = \mu(\mu+\lambda)$$

$$\beta = \frac{\mu(\mu+\lambda)}{p\Pi} = \frac{0.012(0.012+0.25)}{0.9\times 22} = 0.000159$$

It can be followed that when β< 0.000159, it indicates that the value of $R_0$< 1 and there is no epidemic in the population. Similarly, when β> 0.000159; $R_0$> 1 and this shows the presence of an epidemic in the population. The following simulations of the model show the changes in the compartments of the population for different values of β, which in turn reflect the variations in the value of the basic reproduction number, $R_0$. In this way, the model is simulated for various epidemic situations in the population and the resulting curves are discussed.

**Table 2. Parameter values**

| Parameter | Value |
|---|---|
| α | 0.25 |
| μ | 0.012 [23] |
| p | 0.9 |
| r | 0.14 |
| δ | 0.2 |
| Π | 22 [23] |
| λ | 0.25 |

The figures show the variations of the population for different infectivity rates. Fig. 3 shows a non-epidemic situation as the values of β are less than 0.000159. It can be seen from the graphs that the susceptible population grows while the other compartments show a decline. In case of no epidemic, the susceptible population is not infected, thus it displays an increment. As no individuals are being fed into the infected classes from the susceptible class (due to low infectivity rate and no epidemic), the infected classes decrease in number. Following this, it is obvious that if there are no infected persons in the population, there will be no recovered people too. Thus the other classes show a decline. It can be deduced that as long as the value of $R_0$ is kept to be lower than 1, there will be no epidemic, regardless of the exact value.

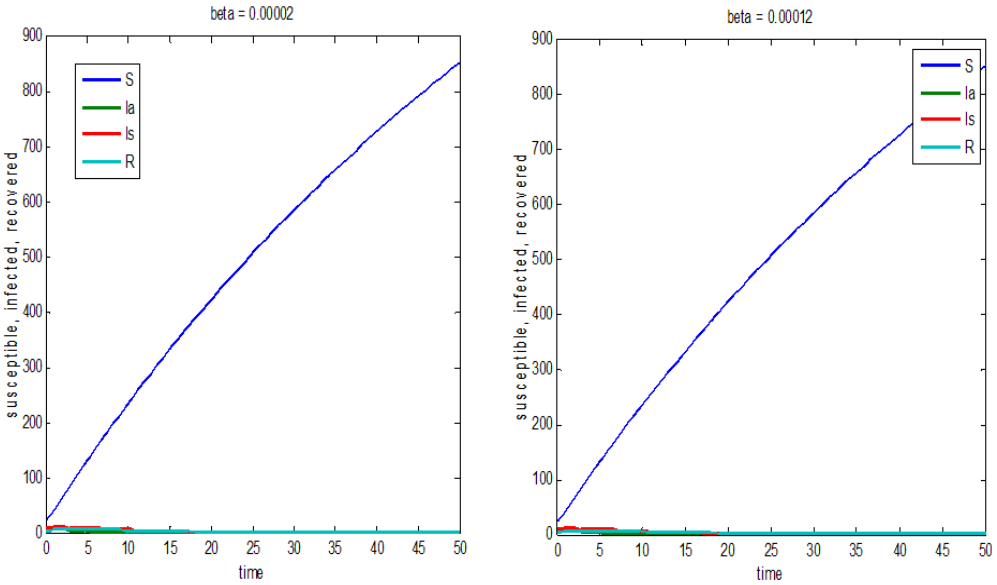


**Fig. 3. Simulations for values of low $R_0$**

Slightly increasing the infectivity rate introduces an epidemic in the population and the susceptible population begins to reduce as people get infected. This is shown in Fig. 4. Since the value of $R_0$ is increased by a small amount, the decline in the susceptible class only begins after a certain period of time. Because the susceptible class feeds into the infected classes, it reduces while the infected classes increase. As a result, the recovered population also increases. As the infectivity rate is increased, the value of $R_0$ also increases, thus presenting higher levels of epidemic in the population. This can be depicted in Figs. 5 and 6. It can be seen from the figures that as the basic reproduction number increases, the infection spreads at higher rates, thus causing the susceptible class to show a decline at faster rates and reduce to lower values. For β = 0.0007, the decline begins at 45 time intervals, as the value of β is increased to 0.001, the decline in the susceptible population begins at 35 time intervals and there after the population reduces to significant levels. As the

infectivity rate increases, the infected populations increase too. If the value of $R_0$ increases to higher values, the susceptible population immediately decreases and gradually diminishes. This is shown in Fig. 6.

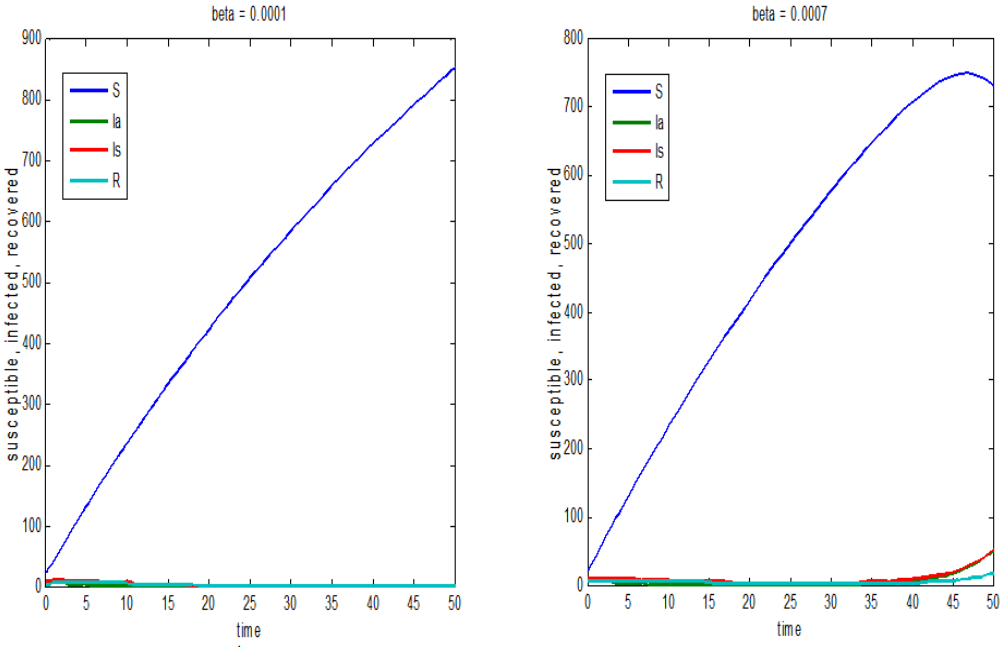


**Fig. 4. Simulations for values of slightly higher $R_0$**

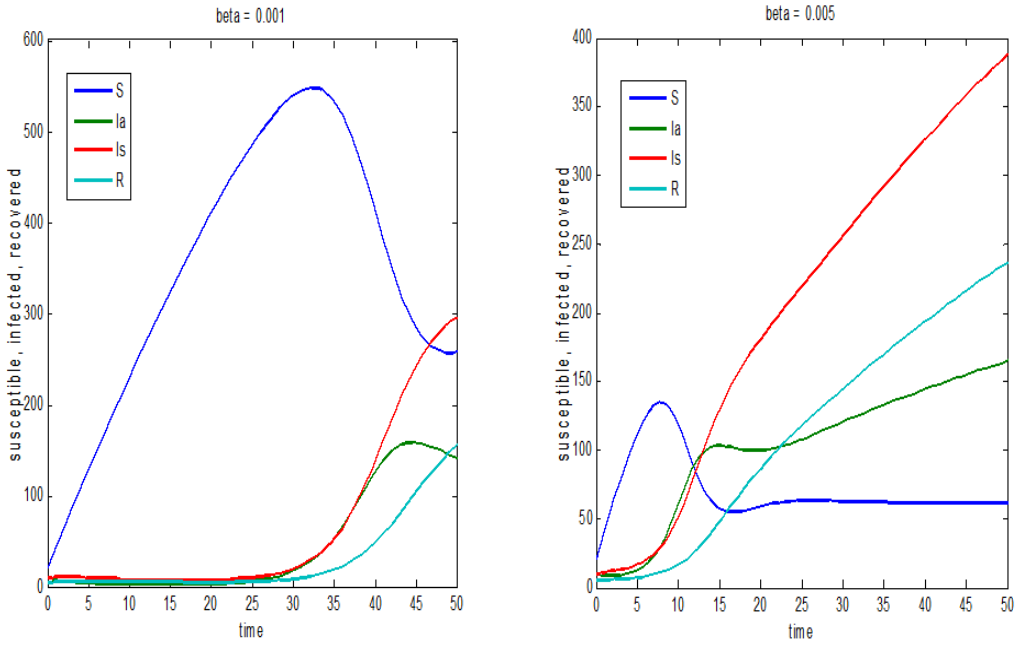


**Fig. 5. Simulations for values of high $R_0$**

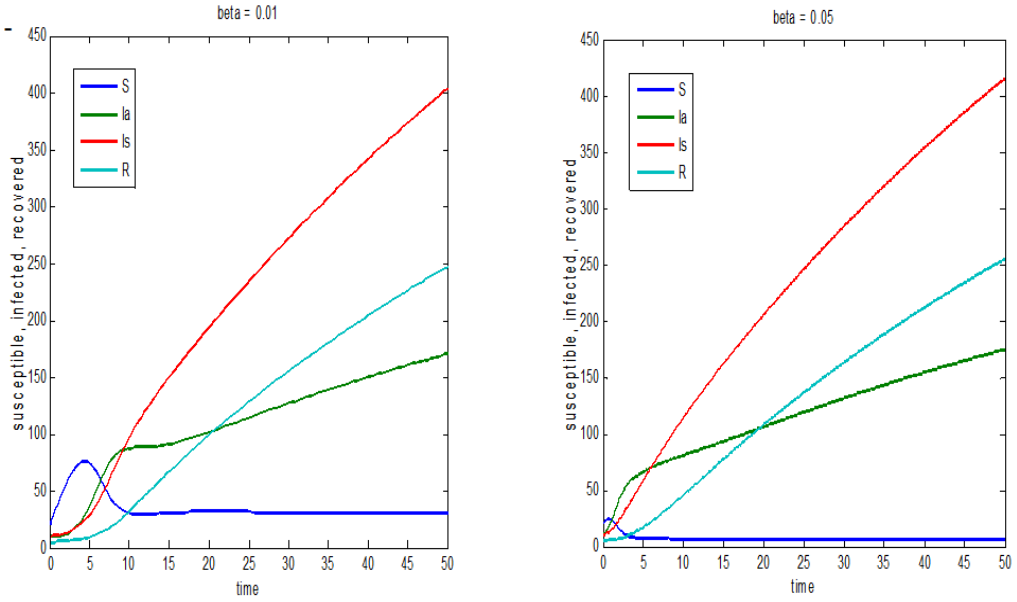


**Fig. 6. Simulations for values of very high $R_0$**

## 5 Conclusions and Future Works

This paper presented a continuous model on diarrhoea caused by rotavirus thus provided an increased understanding of the dynamics of the disease in a country like Botswana. Analysis of the system was done by evaluating the basic reproduction number and the model was simulated using this evaluated parameter. It was proved that as long as the value of $R_0$ is kept minimal, the disease can be eradicated from the population. The model shows that the higher the value of $R_0$, the more likely an epidemic will spread at higher rates. $R_0$ can be kept low by employing various policies such as increasing knowledge of public in terms of prevention and treatment, increased hygiene conditions at work places and better water treatment facilities. This is a very basic project which can be elaborated for form a very robust system which is successful in disease prevention. The model of rotavirus can be implemented onto a system that acts as an interface for the public health sectors showing the current status, predictive nature and strength of the epidemic. Similar models can be built for other pathogens [24] causing diarrhea and be implemented as well on the system. The system can be further elaborated by including other diseases too. Practical implementation of epidemic models can thus be achieved. Another useful criterion to be included is integration of the interface with communication systems to provide a direct alert to the population at large and in general.

## Competing Interests

Authors have declared that no competing interests exist.

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