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Modelling the Impact of Isolating Infected Population on the Dynamics of Diarrhea Epidemics: Applying Systems of Ordinary Differential Equations

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Abstract: This paper provides a mathematical study to characterize the impact of isolating infected population in the dynamics of diarrhea epidemic. System of non-linear differential equation (consists five human compartments S, I, E, I_h, R human compartment) is used to determine a certain threshold value (known as the basic reproductive number R_0 that represents the epidemic indicator obtained from the Eigen value of the next-generation matrix) to model the impact of isolating infected population in the dynamics of diarrhea epidemic. The equilibrium points of the model are calculated and the stability analysis of the numerical simulation has been shown. We investigate the local asymptotic stability of the deterministic epidemic model and similar properties in terms of the basic reproduction number. If at least one of the partial reproduction numbers is greater than unity then the disease will persist in the population. The disease free equilibrium point is locally and globally asymptotically stable when $R_0 < 1$ and unstable when $R_0 > 1$. Numerical simulation of the model is carried to assess or supplement the impact of isolation on the dynamics of diarrhea disease. Numerical simulation results show that as the rate of isolation is increases, then the recovered populations also increase. According to sensitivity analysis of the model, we presented numerical simulation results that confirm theoretical findings and the work has been illustrated through figures for different values of sensitive parameters.

Keywords: Modeling, Isolation, Basic Reproductive Number, Stability, Sensitivity Analysis, Diarrhea, Numerical Simulation

1. Introduction

Diarrhea is the passage of three or more loose or liquid stools per day in a period not exceeding 14 days [1]. It is commonly a sign of an infection in the intestinal tract that is caused by different bacteria, virus and parasitic entities [1]. In low resource areas, Rota-virus and Escherichia coli bacteria cause the highest incidents of diarrhea [2]. These microorganisms spread throughout unclean water and contaminated food or from one person to another, and are most widespread in settings with poor hygiene and absence of access to clean drinking water and sanitation [2]. Diarrhea continues to be one of the leading causes of child mortality, mostly in children less than 5 years of age living in low and middle-income countries [3]. In 2015, 5.9 million children globally died before reaching their fifth birthday where diarrhea was responsible for 9 percent of these deaths [5]. An estimated 1.7 million cases of diarrheal diseases arise each year killing around 760,000 children under the age of 5 years[1, 5]. The majority of deaths take place in children less than 2 years of age living in South Asia and sub- Saharan Africa [6]. In 2013, 6.3 million children under 5 year died in which 2.9 million of them in the African Region, about 473000 from diarrhea [7]. Diarrhea disease is the second leading cause of death in children under five years old. In 2008, 16 percent of death was caused by infectious disease worldwide [2]. When an infective individual or external vector is introduced into a close population, the infectious disease tends to spread within the population [4]. Diarrhea is responsible for killing around 76,000 children globally, there are nearly 1.7 billion cases of diarrheal disease [1]. In developing countries, the annual incidence rate of diarrhea disease episodes in children less than five years old is 3.2 episodes per child [3]. It kills more young children than HIV,

malaria and measles combined [2]. Diarrhea illness alone causes more than 1.5 million deaths annually, thereby making it a worse health threat than cancer or AIDS in terms of death toll [7]. Diarrhea is an abnormal looseness of the stool, changes in stool frequency, consistency, urgency and continence. Sub-Sahara Africa is the most vulnerable region of infectious disease [1], this is due to the fact that the region is greatly affected by climate change which makes it more vulnerable to infectious diseases. Diarrhea outbreaks are associated with periods of rainfall and runoff when subsequent turbidity compromises the efficiency of the drinking water treatment plants [1] found out that heavy rainfall increases diarrhea outbreak due to water contaminated distribution. Many waterborne disease outbreaks occur following a period of intense rainfall [2]. Diarrhea could be acute which lasts for 2 weeks and chronic which lasts for more than 2 weeks [2]. It is one of the most common diseases that is transferred through contaminated food and water [4]. There are two types of diarrhea which are infectious and non-infectious diarrhea. Infectious diarrhea is caused by virus, parasite or bacterium, which could be can pylobacteria, shiga -toxin producing E. Coli, giardiasis, salmonellosis, shigellosis, Rotavirus, yersinia, cryptosphoridiosis etc. Non-infections is caused by toxins (e.g. food poisoning). This type of diarrhea does not spread from person to person [12]. original infection [4]. However, diarrhea is preventable and can be treated. Diarrhea disease can be prevented by taking safe clean drinking water, by using improved sanitation, washing hands with soap regularly, exclusive breast feeding for the first six months and taking of rotavirus vaccination. Various studies have been conducted to investigate diarrheal disease transmission dynamics. Lopman, B. analyzed the dynamic transmission model of nor virus infection disease and immunity [1]. The immunity after infection is temporary and the infection tend to be less severe than the It was found that asymptomatic prevalence of norovirus can change dramatically with small changes in the basic reproduction number R_0. Chaturvedi, O. et. al [1] formulated a continuous mathematical model for shigella outbreaks. They designed the model as an SIRS system comprising of a non-constant population. It was proved that as long as the value of basic reproduction number R_0 is kept minimal, the disease can be eradicated. The model shows that the higher the value of R_0 the more likely an epidemic will spread at higher rate. In this present work, we incorporate the impact of a isolation and treatment in the control of the disease. We show the efficacy of isolation and treatment of infected individuals in the control of the disease. It is important to establish the consequence of multi-intervention campaigns for the spread of diarrhea in order to understand and predict it. Diarrheal disease affects rich and poor, old and young, and those in developed and developing countries alike, yet a strong relationship exists between poverty, an unhygienic environment, and the number and severity of diarrheal episodes especially for children under five [13]. Poverty is associated with poor housing, crowding, dirt floors, lack of access to sufficient clean water or to sanitary disposal of fecal waste, cohabitation with domestic animals that may carry human pathogens, and a lack of refrigerated storage for food-all of which increase the frequency of diarrhea. Poverty also restricts the ability to provide age-appropriate, nutritionally balanced diets or to modify diets when diarrhea develops so as to mitigate and repair nutrient losses. The impact is exacerbated by the lack of adequate, available, and affordable medical care. Thus, the young suffer from an apparently never-ending sequence of infections, rarely receive appropriate preventive care, and too often encounter the health care system when they are already severely ill [13]. Although the presence of blood in the stool is a recognized danger signal, prompting more urgent care seeking, even these patients either are not treated early or receive poor medical care. Ironically, the poor spend considerable amounts on inappropriate care and useless drugs purchased from local shops and untrained practitioners. If antibiotics are properly prescribed, poverty often limits the purchase of a full course of treatment or leads to cessation of treatment as soon as symptoms improve, even though the infection has not been cured [13]. Diarrhea 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 clinical cases of diarrhea 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 diarrhea 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,45 percent of child mortality is caused by rotavirus diarrhea [8]. It can be generally misunderstood that the hazards of water-borne disease like diarrhea will be of minimal levels. Due to the presence of various water reservoirs in the country, Botswana also suffers significantly due to water-borne diarrheal 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 [5] with only a limited portion of the population being subject to adequate sanitation which is 53 in the urban and 18 percent in the rural areas [6]. When a high percentage of the population lives and depends on open water sources, the risk of diarrhea 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 diarrhea in 2012 [7]. This shows that apart from the numerous treatment methods available, necessary prevention and precaution methods need to be employed so as to avoid diarrheal hazards in Botswana [13]. Diarrhea 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 diarrhea is the rotavirus. Rotavirus is classified into several serotypes which can cause viral gastroenteritis. Gastroenteritis is the inflammation of the gastrointestinal tract and has common symptoms of diarrhea, vomiting, fever and abdominal pains [8]. Rotavirus is the leading cause of diarrhea 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 diarrhea infections in Botswana too [9, 10]. Although the pathogen usually infects the immune suppressed 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.

2. Formulation of the Model

2.1. Existing Mathematical Model

The model considered four (4) compartments to gain insight into the effect of vaccine on the dynamical spread of diarrhea disease in a community. The model comprises of susceptible individuals S(t), vaccinated individuals V(t), exposed individuals E(t) and infected individuals I(t) so that N(t) = S(t) + V (t) + E(t) + I (t). The susceptible population is increased by the recruitment of individuals into the population at rate λ, the population decrease by fraction of recruitment for vaccinated individuals at the rate ρ and by susceptible individuals who acquire diarrhea infection with effective contact with people infected with diarrhea, where β is the effective contact rate. The population increased by recovered individuals that has been treated and vaccinated individuals who lost vaccine due to vaccine wanes off at the rate τ and ω respectively. The population of susceptible individuals further reduced by natural death at the rate μ.

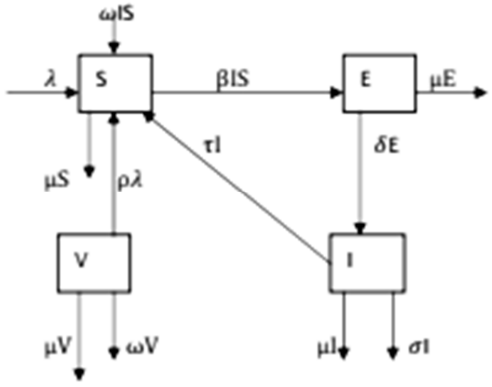


Figure 1. *The flow chart of existing model.*

2.2. The Present Model Formulation

The model maintains the basic structure of the SV EI models, but the vaccinated (V) compartment is replaced by a compartment of recovered individuals (R) and the new compartment infected isolation is added.

a) Model Assumptions

The model will be maintaining the basic structure of the SV EI models. The general model will be developed based on the following assumptions:

1) Susceptible populations are recruited by birth at a constant rate ∧.
2) Individuals in each group have the same natural death rate μ.
3) Human populations are divided in to five groups.
4) Susceptible human can be infected by the infected humans.
5) Infected human can die due to the infection.
6) Infected human can recover due to some treatment.
7) All new born-once are susceptible to infection.
8) All the parameters which are used in this model are positive.
9) Treatment given only for active diarrhea class (isolating class).
10) Sex structure is not considered.
11) Age structure is not considered.
12) $\theta \approx \mu + \omega + \alpha$.

b) Flow chart of proposed model

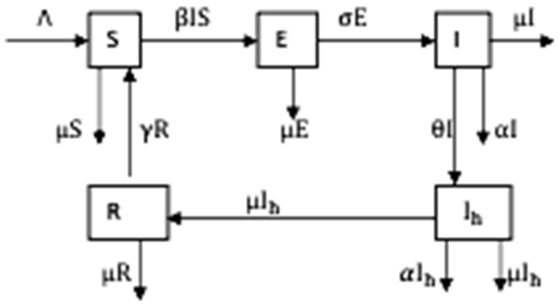


Figure 2. *The flow chart of present model.*

Table 1. *Description of variables of the model.*

Parameters	Description
S(t)	Human population size in susceptible compartment at any time t
E(t)	Humans population size in exposed compartment at any time t
I(t)	Human population size in infected compartment at any time t
$I_h(t)$	Infected but isolated human population size at any time t
R (t)	Recovered human population at any time t

The population under this study is heterogeneous and varying with time which is represented

$$P(t) = S(t) + E(t) + I(t) + I_h(t) + R(t) \quad (1)$$

Based on the assumptions and flow chart in figure 2 leads to the following system of ordinary differential equations (ODE)

$$\left.\begin{aligned} \frac{dS}{dt} &= \Lambda + \gamma R - \beta SI + \mu S \\ \frac{dE}{dt} &= \beta SI - \mu E - \sigma E \\ \frac{dI}{dt} &= \sigma E - \mu I - \theta I - \alpha I \\ \frac{dI_h}{dt} &= \theta I - \mu I_h - \alpha I_h - \omega I_h \\ \frac{dR}{dt} &= \omega I_h - \gamma\mu - R\mu \end{aligned}\right\} \quad (2)$$

All the parameters are nonnegative real numbers, and their descriptions are explained in Table 2.

Table 2. *Description of parameters of the model.*

Parameters	Description
∧	Recruitment rate of humans
β	Effective contact rate
γ	Human recover rate from disease by immunity loss
ω	Treatment rates given for infectious individuals
μ	Natural death rate for humans population
α	Human death rate due to diarrhea disease
θ	Isolation rate from infected human population
σ	Infected rate

2.3. Positivity of Solutions

Theorem 1 If the initial data $(S(0), E(0), I(0), I_h(0), R(0)) \in \Omega$ then the solution set S(t), E(t), I(t), I_h(t), R(t) of the system (2) is positive for all $t \geq 0$.
Proof: From each of the equation of the model system (2) we have the following system in (3) hence one can solve the following system from system (2)

$$\left.\begin{aligned} \frac{dS}{dt} &\geq (-\beta I + \mu)S \\ \frac{dE}{dt} &\geq -(\sigma + \mu)E \\ \frac{dI}{dt} &\geq -(\mu + \theta + \alpha)I \\ \frac{dI_h}{dt} &\geq -(\alpha + \mu + \omega)Ih \\ \frac{dR}{dt} &\geq -(\omega + \mu)R \end{aligned}\right\} \quad (3)$$

By integrating each of the equation in (3) and use separation of variables in each we have the following corresponding system in (4).

$$\left.\begin{aligned} S(t) &\geq S(0)e^{-\int \beta I dt + \mu t} \geq 0 \\ E(t) &\geq E(0)e^{-(\sigma+\mu)t} \geq 0 \\ I(t) &\geq I(0)e^{-(\mu+\theta+\alpha)t} \geq 0 \\ I_h(t) &\geq I_h(0)e^{-(\alpha+\mu+\omega)t} \geq 0 \\ R(t) &\geq R(0)e^{-(\alpha+\mu+\omega)t} \geq 0 \end{aligned}\right\} \quad (4)$$

Hence the solution set S(t), E(t), I(t),$I_h(t)$, R(t) of the system (2) is positive for all $t \geq 0$.

Consider the total human population $N_H(t) = S(t) + E(t) + I(t) + I_h(t) + R(t)$. By taking the derivative of $N_H(t)$ with respect to the time along with the solution of system (2), it is obtained that

$$\frac{dN_H}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dI_h}{dt} + \frac{dR}{dt}.$$

Then, using direct computation gives that

$$\left.\begin{aligned} \frac{N_H}{dt} &= \Lambda - \mu S - \mu E - \mu I - \alpha I - \mu I_h - \alpha I_h - \mu R \\ &= \Lambda - \mu N_H - \alpha(I + I_h) \end{aligned}\right\} \quad (5)$$

In the absence of diarrhea, there is no death from diarrhea, that is, $\alpha = 0$, then

$$\frac{N_H}{dt} \leq \Lambda - \mu N_H \quad (6)$$

Applying Birkhoff and Rota's theorem on a differential inequality (5), we get

$$\frac{N_H}{\Lambda - \mu N_H} \leq dt \quad (7)$$

Integrating the equation (7) on both sides and applying the initial conditions we obtain

$$N_H \leq \frac{\Lambda}{\mu} \leq \left(\frac{\Lambda - \mu N_0}{\mu}\right) e^{-\mu t} \quad (8)$$

which implies that $N_H \leq \frac{\Lambda}{\mu}$, as $t \to \infty$. Hence, all the solutions of system (1) are uniformly bounded, and therefore we have finished the proof.

According to system (1), the feasible region of it can be written as follows:

$$\Omega_H = \left\{(S, E, I, I_h, R) \in \Re^5{}_+, N_H \leq \frac{\Lambda}{\mu}\right\} \quad (9)$$

3. Model Analysis

3.1. Existence of the Equilibrium Points and Basic Reproduction Number

The equilibrium points are obtained by setting the right-hand sides of the model system (2) to zero, that is

$$\frac{dS}{dt} = 0, \frac{dE}{dt} = 0, \frac{dI}{dt} = 0, \frac{dI_h}{dt} = 0, \frac{dR}{dt} = 0$$

Therefore the system of equations (2) becomes

$$\left.\begin{aligned} \Lambda + \gamma R - \beta SI + \mu S &= 0 \\ \beta SI - \mu E - \sigma E &= 0 \\ \sigma E - \mu I - \theta I - \alpha I &= 0 \\ \theta I - \mu I_h - \alpha I_h - \omega\, I_h &= 0 \\ \omega\, I_h - \gamma\mu - R\mu &= 0 \end{aligned}\right\} \quad (10)$$

Then$X_0 = (S^*, E^*, I^*, I_h{}^*, R^*)$ is the equilibrium point of the model system (2).

3.2. The Disease-Free Equilibrium Point

Since the basic reproduction number is computed at this equilibrium point, hence in the following the computation of this number is carried out, and then the endemic equilibrium point is determined. Now, in order to determine the basic reproduction number, the "next-generation method" or "Spectral Radius method" is used [10, 11]. Consider an epidemic model having n different compartments from which compartments contained infected individuals with the disease, then the next-generation matrix (operator) is given by$(FV)^{-1}$,

Where

$$F = \left[\frac{\partial F_i(X_0)}{\partial x_j}\right] \text{ and } V = \left[\frac{\partial V_i(X_0)}{\partial x_j}\right] \quad (11)$$

where i, j = 1, 2,...,m, X_0 is the disease-free equilibrium

point, while $x_i$denotes the number of individuals in the i^{th} infected compartment. However, $F_i(x_i)$ is the rate of appearance of new infections in the compartment i, while$V_i(x_i) = V_i^{-1}(x_i) - V_i^{+1}(x_i)$where $V_i^{+1}(x_i)$ represents the rate of the shift of individuals into i^{th}compartment by all other means and denotes the rate of the shift of individuals out of the i^{th} compartment. The difference$F_i(x_i) - V_i(x_i)$gives the rate of change of x_i. Note that $F_i(x_i)$ shouldinclude only infections that are newly arising but does not include terms that describe the shift of infectious individuals from one infected compartment to another. Finally, the basic reproduction number, that is denoted by R_0, is given by the spectral radius (dominant eigen value) of the matrix FV^{-1}. It is well known that the basicreproduction number R_0 is oneof the most crucial quantities in infectious diseases as R_0, measures how contagious a disease is. For$R_0 < 1$, the diseaseis expected to stop spreading, but for $R_0 = 1$, an infectedindividual can infect on an average 1 person; that is, the spread of the disease is stable. The disease can spread and become epidemic if $R_0 > 1$. Accordingly, regarding system (7), it is obtained that

$$V = \begin{bmatrix} (\sigma+\mu) & 0 & 0 \\ 0 & \mu+\alpha+\theta & 0 \\ 0 & -\theta & \mu+\alpha+\omega \end{bmatrix} \quad (12)$$

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

Then we have to find the inverse of the Jacobian matrix of V, which is given by

$$V^{-1} = \begin{bmatrix} \frac{1}{\sigma+\mu} & 0 & 0 \\ \frac{\theta\sigma}{(\sigma+\mu)(\mu+\alpha+\omega)} & \frac{1}{\mu+\alpha+\theta} & 0 \\ \frac{\mu+\alpha}{(\sigma+\mu)(\theta+\mu+\alpha)(\mu+\alpha+\omega)} & \frac{\theta}{(\theta+\mu+\alpha)(\mu+\alpha+\omega)} & \frac{1}{\mu+\alpha+\omega} \end{bmatrix}$$

Therefore,

$$FV^{-1} = \begin{bmatrix} \frac{\sigma\beta\theta S}{(\sigma+\mu)(\theta+\mu+\alpha)(\mu+\alpha+\omega)} & \frac{\beta\theta S}{(\theta+\mu+\alpha)(\mu+\alpha+\omega)} & \frac{\theta S}{\mu+\alpha+\omega} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

Consequently, the basic reproduction number of system (2) is determined as

$$R_0 = \frac{\sigma\beta\theta\Lambda}{\mu(\sigma+\mu)(\theta+\mu+\alpha)(\mu+\alpha+\omega)}$$

3.3. Local Stability Analysis

This section treats the local stability of system (2) using the linearization technique. The Jacobian matrix for system (2) at the point (S, E, I, I_h) can be written as follows:

$$J = \begin{bmatrix} -(\sigma+\mu) & \beta\frac{\Lambda}{\mu} & 0 & 0 \\ \sigma & -(\theta+\mu+\alpha) & 0 & 0 \\ 0 & \theta & -(\omega+\mu+\alpha) & 0 \\ 0 & 0 & \omega & -(\mu+\gamma) \end{bmatrix}$$

Theorem 2. The disease free equilibrium point,$X_0 = \left(\frac{\Lambda}{\mu}, 0,0,0,0\right)$ is locally asymptoticallystable if $R_0 < 1$ and $R_0 < \frac{\theta}{(\mu+\alpha+\omega)}$ otherwise unstable. The system is then re-defined as

$$\left.\begin{aligned} f_1(S,E,I,I_h,R) &= \Lambda + \gamma R - \beta SI - \mu S \\ f_2(S,E,I,I_h,R) &= \beta SI - \mu E - \sigma E \\ f_3(S,E,I,I_h,R) &= \sigma E - \mu I - \theta I - \alpha I \\ f_4(S,E,I,I_h,R) &= \theta I - \mu I_h - \alpha I_h - \omega I_h \\ f_5(S,E,I,I_h,R) &= \omega I_h - \gamma\mu - R\mu \end{aligned}\right\} \quad (13)$$

The Jacobian of the system (13) at$X_0 = \left(\frac{\Lambda}{\mu}, 0,0,0\right)$ is given by

$$J(X_0) = \begin{bmatrix} -(\sigma+\mu) & \beta\frac{\Lambda}{\mu} & 0 & 0 \\ \sigma & -(\theta+\mu+\alpha) & 0 & 0 \\ 0 & \theta & -(\omega+\mu+\alpha) & 0 \\ 0 & 0 & \omega & -(\mu+\gamma) \end{bmatrix} \tag{14}$$

Consider the matrix (14) and let k be the eigenvalue. Then we have $|J(X_0) - kI| = 0$ where I is a 5×5 identity matrix. Thus, we have

$$|J(X_0) - kI| = \begin{vmatrix} -(\sigma+\mu)-k & \beta\frac{\Lambda}{\mu} & 0 & 0 \\ \sigma & -(\theta+\mu+\alpha)-k & 0 & 0 \\ 0 & \theta & -(\omega+\mu+\alpha)-k & 0 \\ 0 & 0 & \omega & -(\mu+\gamma)-k \end{vmatrix} \tag{15}$$

Direct computations show that this Jacobian matrix has the following characteristic equation:

$$(\sigma+\mu+k)(\mu+\theta+\alpha+k)(\mu+\omega+\alpha+k)(\mu+\gamma+k) - \frac{\beta\Lambda\sigma}{\mu}(\mu+\omega+\alpha+k)(\mu+\gamma+k) = 0$$

$$Ak^4 + Bk^3 + Ck^2 + Dk + E = 0$$

Where

$$A = 1$$

$$B = (\sigma + 4\mu + \theta + \omega + \gamma + 2\alpha)$$

$$C = (\mu+\theta+\alpha)(\sigma+\mu) + (\sigma+2\mu+\theta+\alpha)(2\mu+\omega+\alpha+\gamma) + (\mu+\omega+\alpha)(\mu+\gamma) - \frac{\sigma\beta\Lambda}{\mu}$$

$$D = (\mu+\theta+\alpha)(\sigma+\mu)(\gamma+2\mu+\omega+\alpha) + (2\mu+\sigma+\theta+\alpha)(\mu+\omega+\alpha)(\mu+\gamma) - \frac{\sigma\beta\Lambda}{\mu}(2\mu+\omega+\alpha+\gamma)$$

$$E = (\mu+\theta+\alpha)(\sigma+\mu)(\mu+\omega+\alpha)(\mu+\gamma) - \frac{\sigma\beta\Lambda}{\mu}(\mu+\omega+\alpha)(\mu+\gamma)$$

Due to the complexity in determining the signs of the remaining eigenvalues, we employ Routh-Hurwitz conditions for stability. The Routh-Hurwitz conditions to ensure that all roots of (15) have negative real partsare $A > 0$, $B > 0$, $E > 0$ and $BC > AD$, $BCD > AD^2 + B^2$Eclearly A and B are positive. For C, D and E are to be positive, set

$$(\mu+\theta+\alpha)(\sigma+\mu) + (\sigma+2\mu+\theta+\alpha)(2\mu+\omega+\alpha+\gamma) + (\mu+\omega+\alpha)(\mu+\gamma) - \frac{\sigma\beta\Lambda}{\mu} > 0$$

$$(\mu+\theta+\alpha)(\sigma+\mu) + (\sigma+2\mu+\theta+\alpha)(2\mu+\omega+\alpha+\gamma) + (\mu+\omega+\alpha)(\mu+\gamma) > \frac{\sigma\beta\Lambda}{\mu}$$

For Dto be positive, set

$$(\mu+\theta+\alpha)(\sigma+\mu)(\gamma+2\mu+\omega+\alpha) + (2\mu+\sigma+\theta+\alpha)(\mu+\omega+\alpha)(\mu+\gamma) - \frac{\sigma\beta\Lambda}{\mu}(2\mu+\omega+\alpha+\gamma) > 0$$

$$(\mu+\theta+\alpha)(\sigma+\mu)(\gamma+2\mu+\omega+\alpha) + (2\mu+\sigma+\theta+\alpha)(\mu+\omega+\alpha)(\mu+\gamma) > \frac{\sigma\beta\Lambda}{\mu}(2\mu+\omega+\alpha+\gamma)$$

For E to be positive, set

$$(\mu+\theta+\alpha)(\sigma+\mu)(\mu+\omega+\alpha)(\mu+\gamma) - \frac{\sigma\beta\Lambda}{\mu}(\mu+\omega+\alpha)(\mu+\gamma) > 0$$

This leads to

$$1 - R_0 \frac{(\mu+\omega+\alpha)}{\theta} > 0$$

From the assumption we have $\theta \approx \mu + \omega + \alpha$ so that $1 - R_0 > 0$, since$R_0 = \frac{\sigma\beta\theta\Lambda}{\mu(\sigma+\mu)(\theta+\mu+\alpha)(\mu+\alpha+\omega)}$

This can be true if and only if $R_0 < 1$. Hence, by Routh-Hurwitz criterion, all the eigenvalues have negative real parts.

This shows that X_0 locally asymptotically stable if $R_0 < 1$, and unstable if $R_0 > 1$.

3.4. Global Stability of the Disease-Free Equilibrium Point

In this section, we study the global properties of the disease-free equilibrium. The following theorem provides the global property of the disease-free equilibrium:

Theorem: If $R_0 < 1$, then the disease free equilibrium of the model is globally asymptotically stable in the feasible domain. Proof By the comparison theorem, the rate of change of the variables representing the infected components of model system (2) can be re-written as

$$\begin{bmatrix} E'(t) \\ I'(t) \\ I_h'(t) \end{bmatrix} = (F - V) \begin{bmatrix} E \\ I \\ I_h \end{bmatrix} - \begin{bmatrix} \beta I(1 - S) \\ 0 \\ 0 \end{bmatrix}$$

where the matrices F and V are defined by the expressions (12) respectively. But we also note that $S \leq \frac{\Lambda}{\mu}$for all $t \geq 0$ in Ω›. Thus

$$\begin{bmatrix} E'(t) \\ I'(t) \\ I_h'(t) \end{bmatrix} \leq \begin{bmatrix} E \\ I \\ I_h \end{bmatrix} \quad (16)$$

Using the fact that the eigenvalues of the matrix (F - V) all have negative real parts, it follows that the linearised differential inequality system (16), is stable whenever $R_0 < 1$. Consequently, $(E, I, I_h) = (0, 0, 0)$ as $t \to \infty$and evaluating system (2) at $E = I = I_h = 0$ gives $S \to \frac{\Lambda}{\mu}$, for $R_0 < 1$. Hence, the disease-free equilibrium, X_0, is globally asymptotically stable for $R_0 < 1$.

3.5. The Endemic Equilibrium Point

We shall now study the existence of the endemic equilibrium state of the modified model. Endemic equilibrium pointX_1 is a steady-state solution, where the disease persists inthe population. For the existence and uniqueness of endemic equilibrium $X_1 = (S^*, E^*, I^*, I_h^*, R^*)$, its coordinates should satisfy the conditions: $X_1 = (S^*, E^*, I^*, I_h^*, R^*) > 0$.

From the system of equation (10) the endemic equilibrium point is

$$S^* = \left(\frac{\mu+\sigma}{\beta\sigma}\right)(\mu + \theta + \alpha)$$

$$E^* = \left(\frac{(\mu+\theta+\alpha)\mu(\mu+\sigma)-\lambda\beta\sigma}{\beta\sigma}\right)\left(\frac{(\mu+\theta+\alpha)(\gamma+\mu)}{\gamma\omega\sigma-(\mu+\theta+\alpha)(\mu+\gamma)(\mu+\sigma)}\right)$$

$$I^* = \left(\frac{(\mu+\theta+\alpha)\mu(\mu+\sigma)-\lambda\beta\sigma}{\beta\sigma}\right)\left(\frac{(\gamma+\mu)}{\gamma\omega\sigma-(\mu+\theta+\alpha)(\mu+\gamma)(\mu+\sigma)}\right)$$

$$I_h^* = \left(\frac{\theta}{\mu+\alpha}\right)\left(\frac{a\mu(\mu+\sigma)-\lambda\beta\sigma}{\beta}\right)\left(\frac{(\gamma+\mu)}{\gamma\omega\sigma-(\mu+\theta+\alpha)(\mu+\gamma)(\mu+\sigma)}\right)$$

$$R^* = \left(\frac{\omega\mu a(\mu+\sigma)-\omega\lambda\beta\sigma}{\beta\gamma\omega\sigma-\beta(\mu+\theta+\alpha)(\mu+\gamma)(\mu+\sigma)}\right)$$

The result shows us endemic equilibrium point is exists and it is unique.

3.6. Local Stability of Endemic Equilibrium Point

The endemic equilibrium can expressed in terms of R_0. For the existence of endemic equilibrium X_1 all state variables are non-negative. We analyze the stability of the endemic equilibrium by linearizing the above system of differential equations (2) to give the Jacobian matrix. The Jacobian matrix is computed by differentiating each equation in the system equation (2) with respect to the state variables. and solve at endemic equilibrium point. Endemic equilibrium points are steady-state solutions where there is diarrhea infection and this equilibrium points are obtained by setting the right hand sides of the model equations (2) equals to zero. The local stability of the endemic equilibrium point X_1 is decided by considering the sign of the eigenvalues of the Jacobian matrix of the system (2).

Theorem The positive equilibrium X_1 of system (2) is locally asymptotically stable if$R_0 > 1$ and unstable if$R_0 < 1$.

3.7. Sensitivity Analysis

Given the explicit formula for R_0 we can easily derive analytical expression for the sensitivity of R_0 with respect to each parameter that comprisesR_0. In order to study the effect of this parameter on R_0 we performed a sensitivity analysis on R_0 with respect to this parameter. The normalized index $S_k$is defined as:

$$S_k = \frac{\partial R_0}{\partial h}\frac{h}{R_0}(*)$$

where h is the parameter of interest. The larger the magnitude of the sensitivity index leads to more sensitivity R_0 with respect to that parameters.

$$\frac{\partial R_0}{\partial \sigma} = 1 - \frac{\sigma}{\sigma+\mu} > 0$$

$$\frac{\partial R_0}{\partial \beta} = 1 > 0$$

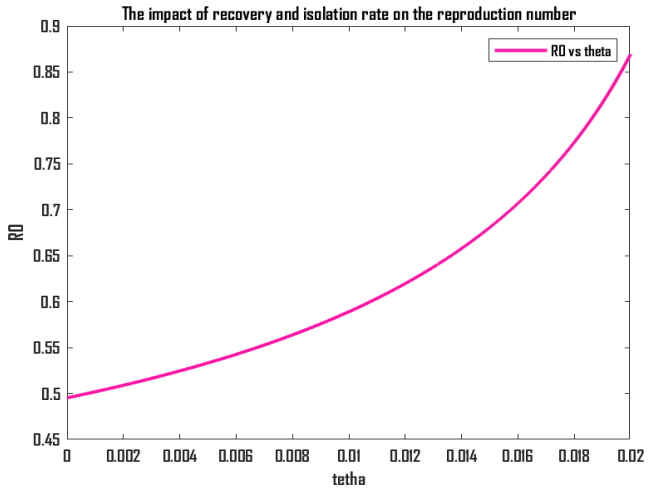
$$\frac{\partial R_0}{\partial \theta} = 1 - \frac{\theta}{\sigma+\mu+\alpha} > 0$$

$$\frac{\partial R_0}{\partial \Lambda} = 1 > 0$$

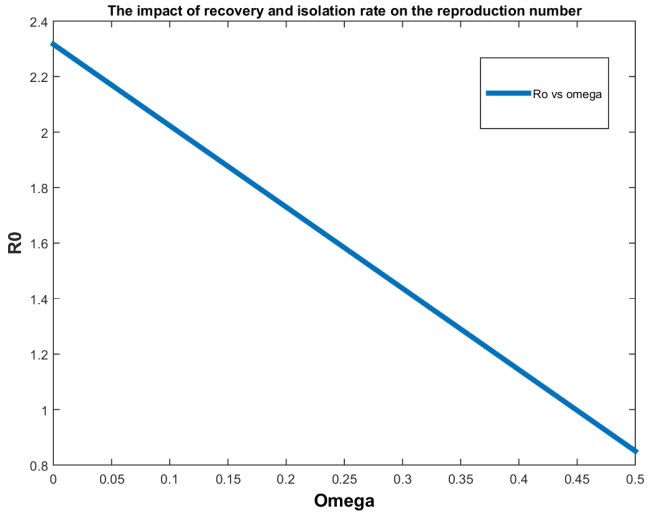
$$\frac{\partial R_0}{\partial \omega} = -\frac{\omega^2}{\omega+\mu+\alpha} < 0$$

$$\frac{\partial R_0}{\partial \mu} = -\frac{\mu^3}{\mu+\alpha+\sigma} < 0$$

$$\frac{\partial R_0}{\partial \alpha} = -\frac{\mu^3}{\alpha+\mu+\omega} < 0$$



(a) The reproduction number decreases when treatment



(b) The reproduction number increases as isolation rate is increases rate is increases

Figure 3. *Sensitivity analysis of reproduction number with respect to treatment rate and isolation rate.*

From figure 3, figure 3a shows that when the treatment rate of increasing then Reproduction number is also decreasing, and figure 3b the isolating rate increase and Reproduction number also increases. So increasing the treatment rate of exposed of human population have positive impact on the reduction of the disease.

4. Numerical Simulation

Numerical Simulations of the dynamic model were carried out by MATLAB function ode 45, using the Rurnge-Kutta of order four. The set of parameter values in table we were used to investigate the effect of isolating people in the control of the spread of diarrhea. This parameter values whose sources are from literature and assumptions. Four hypothetical cases were considered and in each case, the probability that individuals who are exposed to the diseases will progress to infectious class depends on the level of immunity individual has. It is prominent to note here that when series diarrhea patient are isolated from infected people and kept in a separate place, and it is assumed that they will have herd immunity, (i.e. the level of immunity in a population which prevents epidemics). some of the parameter values used:

Natural mortality rate of individuals, (μ): The time unit is set at year and the constant natural mortality rate, μ is assumed to be inversely related to life expectancy at birth which is approximately 50 years.

$$\mu = \frac{1}{50} = 0.02 \text{ per year.}$$

Recruitment rate, (Λ): The recruitment rate, (Λ) controls the total population sizes because the asymptotic carrying capacity of the population is $\frac{\Lambda}{\mu}$. For purposes of this study, we shall set the recruitment rate at 24 individuals per year.

Contact rate (β) in this case the contact rate assumed to be constant it is 0.35.

Human death rate due to diarrhea disease α, α varies from country to country. It is as low as 0.07 in developed countries but reaches 0.365 per year in some African countries (Snideretal [15]. Therefore, we take α =0.365 [15].

Table 3. *The parameter values of the model.*

Parameters	Case1	Case2	Case3	Case4	Reference
Λ	24	24	24	24	estimated
μ	0.02	0.02	0.02	0.02	[14]
β	0.35	0.35	0.35	0.35	estimated
σ	0.4	0.4	0.4	0.4	estimated
α	0.365	0.365	0.365	0.365	[15]
ω	0.4	0.4	0.4	0.4	estimated
θ	0.2	0.2	0.6	0.6	estimated
γ	0.98	0.98	0.98	0.98	estimated

And the following initial conditions have been considered; $S[0] = 1200;\ E[0] = 800;\ I[0] = 500;\ I_h[0] = 200;\ R[0] = 100$ at time $t_0 = 0$ and $t_f = 15$.

5. Result and Discussion

This has been done to show the dynamics of the disease in the population when there are no interventions. The numerical results should examine the effect of parameters on the transmission of diarrhea disease which are used in the present model. Let us discuss on the following some numerical outputs.

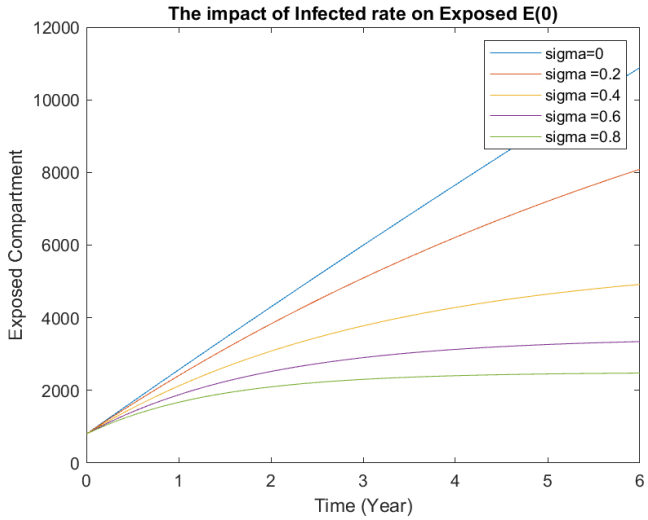


Figure 4. *Effect of infected rate σ on Exposed compartment.*

Figure 4: Numerical simulation on E(t) that shows the impact of infected rate σ on ex-posed human population. From this figure we observe that the infected rate σ is inversely proportional with the exposed human population. i.e. whenever the rate infection is increases then exposed human compartment decreases, inversely if the rate infected decreases the exposed human population increases through a time. In figure 5 we can observe that, if the isolation rate is increased throughout a time proportionally the infected isolated human population is also increases. And as the number of series infected is decreases, the number of infected human population becomes low.

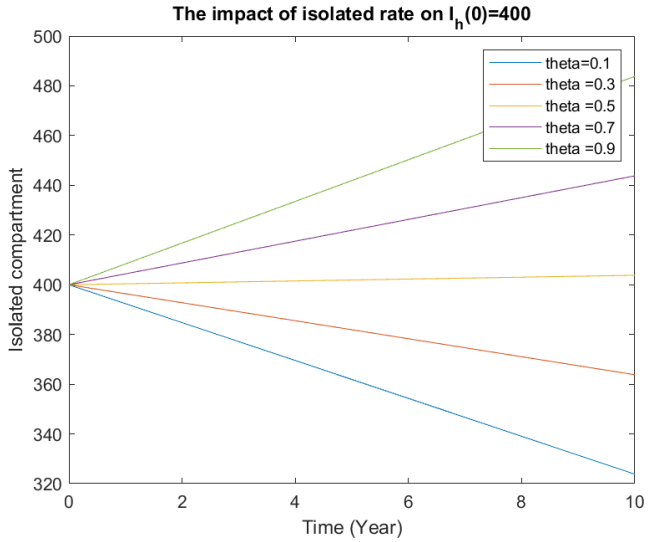


Figure 5. *Effect of isolation rate θ on isolated compartment.*

In figure 6 we can observe that, if some treatment rate is increased throughout a time proportionally there covered human population is also increases.

Figures 7 and 8 shows that as the isolation rate θ decreases from 0.4 to 0.2 the reproduction number is also decreases from 0.91 to the 0.055. This indicates that the chance of expose rate is very rear and the infected becomes zero.

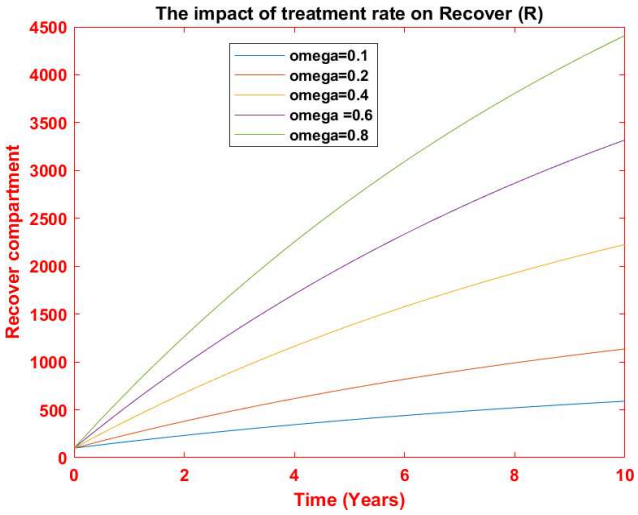


Figure 6. *Effect of treatment rate ω on Recover compartment.*

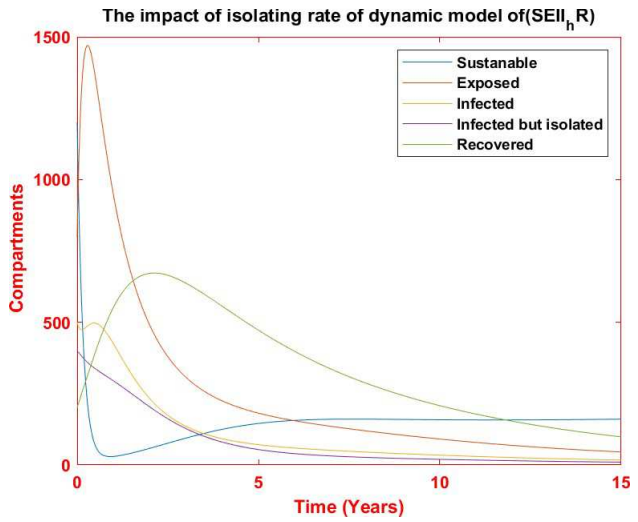


Figure 7. *Reproduction number* $R_0 = 0.91$ *and isolation rate* $\theta = 0.4$.

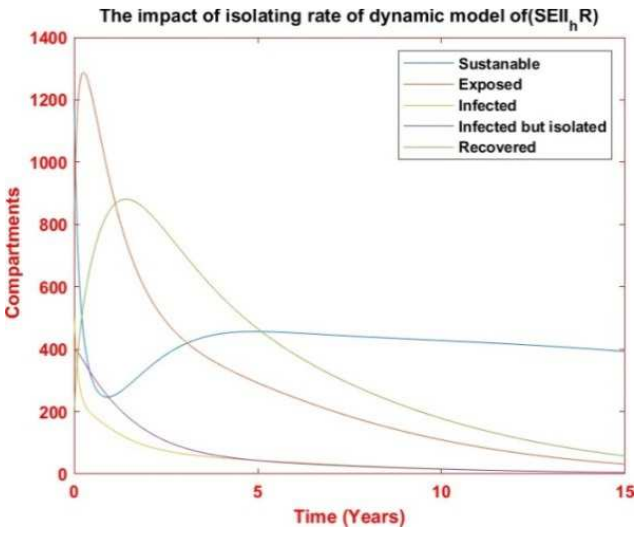


Figure 8. *Reproduction number* $R_0 = 0.055$ *and isolation rate* $\theta = 0.2$.

6. Conclusion

In this study is to formulate and analyze the deterministic

compartmental mathematical model on impact of isolating infected human population for the transmission of diarrhea dynamics. The 5 dimensional system of ordinary differential equations were formulated. This model has shown importance of isolation in preventing transmission of diarrhea disease with in human population. We first showed that there exists a domain where the model is epidemiological and mathematically well-posed. The disease free and endemic equilibrium points are calculated. The basic reproduction number has been computed using next generation matrix method. By using the principle of linearized stability and Routh Hurwitz conditions, we proofed that the stability of the disease free and endemic equilibrium points are controlled by the basic reproduction ratio,R_0. If $R_0 < 1$then the disease-free equilibrium point X_0, is locally asymptotically stable; and if $R_0 > 1$, then X_0 is unstable. We also proved that an endemic equilibrium point X_1 exists and locally asymptotically stable for all $R_0 > 1$. From the reproduction numberR_0, we conclude that; when $R_0 < 1$ the diarrhea disease becomes decrease from the society over a period of time. When $R_0 > 1$then the diarrhea disease becomes endemic. The analysis and the numerical simulations showed that the disease decrease if the isolation of the infected human population increases and the recovery rate of infected human population is also increases. Conversely diarrhea disease increases if the isolation of the infected human population decreases and this decreases the recovery rate of infected human population. The sensitivity analysis of the basic reproduction number shows that isolation rate is the most sensitive parameter, next to it is treatment rate followed by recover rate. Therefore, isolating active diarrhea class and giving treatment for them will be reducing diarrhea infection.

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