Science Journal of Applied Mathematics and Statistics
2022; 10(5): 90-97
http://www.sciencepublishinggroup.com/j/sjams
doi: 10.11648/j.sjams.20221005.12
ISSN: 2376-9491 (Print); ISSN: 2376-9513 (Online)

Mathematical Modeling and Treatment Impacts of Water Borne Disease Spread

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To cite this article:
Mideksa Tola Jiru. Mathematical Modelling and Treatment Impacts of Water Borne Disease Spread. *Science Journal of Applied Mathematics and Statistics*. Vol. 10, No. 5, 2022, pp. 90-97. doi: 10.11648/j.sjams.20221005.12

Received: August 19, 2022, 2022; **Accepted**: September 14, 2022, 2022; **Published**: November 11, 2022

Abstract: In this work, the treatment impacts of water borne disease is modeled and analyzed from a mathematical perspective via a deterministic SEIR model. The total human population is partitioned into four sub-classes namely susceptible individuals, exposed individuals, infected individuals and recovered individuals. The stability theory of non-linear differential equations and the basic reproductive number represents the epidemic indicator which is obtained from the largest eigen value of the next-generation matrix. The model explored invariant region, equilibrium condition, basic reproduction number, and stability analysis. The invariant region was proved to be positive and bounded that confirm the feasible model solution. It is also observed that the water borne disease is free equilibrium is locally asymptotically stable if the basic reproduction number is less than one. In this situation it is found that the disease is controlled whenever the treatment is allowable in the community. The disease is endemic equilibrium and globally asymptotically stable in the invariant region if the basic reproduction number is greater than one. The sensitivity analysis revealed that the rate of transmission and the rate at which exposed individuals become infectious are the most sensitive parameters. The numeric results have been illustrated through figures for different values of sensitive parameters by use of MATLAB simulation method. The findings indicate that effective treatment is adequate in eradicating and controlling water borne disease.

Keywords: Water Borne, Basic Reproduction Number, Mathematical Model, Numerical Simulation

1. Introduction

Commonly, most water borne disease is a sign of an infection in the intestinal tract that is caused by different bacteria, virus and parasitic entities. It is also described as an increase in the frequency of bowel movements or decrease in the consistency of stools that cause the discharge of watery, loose stools. The severity of diarrhea is determined by the size and number of stools passed within a period of time. Severe diarrhea means having more than 10 loose, watery stools in a single day [1]. Diarrhea is preventable and can be treated by taking safe clean drinking water by using improved sanitation, washing hands with soap regularly, exclusive breast feeding for the first six months [3]. Diarrhea is transmitted throughout unclean water and contaminated food or from an infected person to another, and is most widespread in settings with poor hygiene and drinking unclean water and sanitation. Diarrhea could be acute which lasts for 2 weeks and chronic which leads to severe. Severe diarrhea is the second leading cause of death in children under five years old [4]. In 2008, 16 percent of death was caused by infectious disease worldwide [2]. In 2015, among 5.9 million children globally deaths before reaching their fifth birthday 9 percent were died due to diarrhea infection [6]. 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 [5]. 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 [8]. 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 [14]. When a high percentage of the population lives and depends on open

water sources, the risk of diarrhea increases appreciably [7]. 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 [13]. Gastroenteritis is the inflammation of the gastrointestinal tract and has common symptoms of diarrhea, vomiting, fever and abdominal pains [9]. 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 [10].

2. Methods

This study subdivided the human population into four compartmental model; namely: susceptible individuals represent the number of people susceptible to the disease at the time t. Exposed individuals denote individuals who are effective contact with infected individuals who are probably infected. Infected individuals are the numbers of people who have been infected with the disease and are able to spread the disease to the susceptible individuals. Recovered individuals is the compartment used for those who infected and have recovered from the disease so that the total population will be $N(t) = S(t) + E(t) + I(t) + R(t)$. The model assumes direct transmission of diarrhea from infected individuals to susceptible individuals. However, water borne disease is largely contacted from environmental bacteria through contaminated water [2]. All human populations experience natural death at the rateμ. And the infected individuals die from diarrhea disease at the rateα. The susceptible population is increased by the rate of recruitment Λ, either by immigration or birth rate and also increased by recovered individuals' that has been recovered from infected individuals due to some rate of treatmentω. We also assume that susceptible individuals reduced to exposed individual at a rate of effective contactβ. Exposed individuals are probably reduced into infected individuals at rate of infection σ due to infection. Due to infection rate α of infected individual reduced by death and some treatment ω rate of infected individuals are probably recovered.

2.1. Model Assumptions

The following are the assumptions of the model:

a. Susceptible populations are recruited by birth at a constant rate Λ.
b. Individuals in each group have the same natural death rate μ.
c. Susceptible human can be infected by the infected humans.
d. Infected human can die due to the infection.
e. Infected human can recover due to some treatment.
f. All new born-once are susceptible to infection.
g. All the parameters which are used in this model are positive.
h. $\omega \approx \mu + \alpha$.

2.2. Model Flowchart and Equations Model Flow Chart

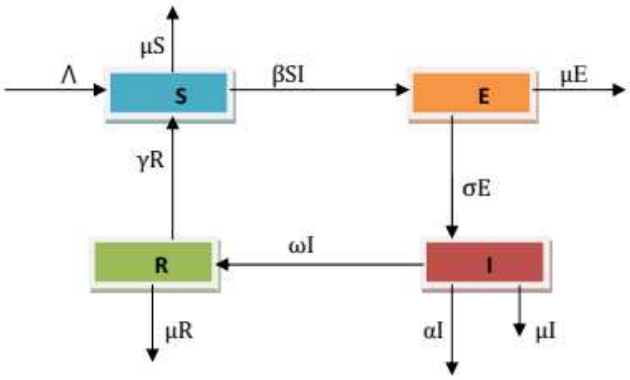

Figure 1. *The flow chart of the model.*

2.3. Model Equations

Dynamic systems are set of equations which describes an event in nature that further describes primarily a time changing process [11]. The properties which characterize these dynamical equations are either finite or infinite dimensions or being non-deterministic or deterministic in nature. The description of these systems is by use of differential equations. Differential equations are defined as equations which contain a single or more derivatives which are of unknown functions. From figure 1 we have obtained the following ordinary differential equations (ODE)

$$\left.\begin{aligned} \frac{dS}{dt} &= \Lambda + \gamma R - \beta SI - \mu S \\ \frac{dE}{dt} &= \beta SI - \sigma E - \mu E \\ \frac{dI}{dt} &= \sigma E - \alpha I - \omega I - \mu I \\ \frac{dR}{dt} &= \omega I - \gamma R - \mu R \end{aligned}\right\} \quad (1)$$

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

Variables	Description
S(t)	Human population size in susceptible compartment at any time t
E(t)	Human population size in exposed compartment at any time t
I(t)	Human population size in infected compartment at any time t
R (t)	Recovered human population at any time t

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

Parameters	Interpretation
Λ	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 diarrhoea disease
σ	Infected rate

2.4. The Invariant Region

This is the region which the model solution lies positively. Consider the total human population $N_H(t) = S(t) + E(t) + I(t) + R(t)$. Taking the derivative of $N_H(t)$ with respect to time we have

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

$$= \Lambda - \mu S - \mu E - \mu I - \alpha I - \mu R$$

$$= \Lambda - (S + E + I + R)\mu - \alpha I$$

$$= \Lambda - \mu N_H - \alpha I$$

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

$$\frac{dN_H}{dt} \leq \Lambda - \mu N_H$$

Applying Birkhoff and Rota's theorem on a differential inequality, we have

$$\frac{dN_H}{\Lambda - \mu N_H} \leq dt - - - - - - - - - - - - - - - - (*)$$

Integrating $(*)$ on both sides and applying the initial conditions we obtain $N_H \leq \frac{\Lambda}{\mu} \leq \left(\frac{\Lambda - \mu N_0}{\mu}\right) e^{-\mu t}$, which implies that, $N_H \leq \frac{\Lambda}{\mu}$, as $t \to \infty$. Hence, all the solutions of system (1) are uniformly bounded. According to system (1), the feasible region of it can be written as:

$\Omega_H = \left\{(S, E, I, R) \in \Re^4_+, N_H \leq \frac{\Lambda}{\mu}\right\}$. This is positively invariant and bounded.

Hence the system is biologically meaningful and mathematically well-posed in the domain.

3. Model Analysis

3.1. Existence of the Equilibrium Points and Basic Reproduction Number

The disease free equilibrium points of the model are its steady state solutions in the absence of infection or disease. To obtain the equilibrium points for the model we set the right hand side of (1) to zero, that is

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

Therefore the system of equations (1) becomes

$$\left.\begin{array}{c} \Lambda + \gamma R - \beta SI - \mu S = 0 \\ \beta SI - \mu E - \sigma E = 0 \\ \sigma E - \mu I - \alpha I - \omega I = 0 \\ \omega I - \gamma R - \mu R = 0 \end{array}\right\} \quad (2)$$

Then $X_0 = (S^*, E^*, I^*, R^*)$ is the equilibrium point of the model system (1).

3.2. The Disease-Free Equilibrium Point

Disease-free equilibrium points (D.F.E) are steady-state solutions where there is no diarrhea disease. We define the diarrhea infected classes as the human population that is either exposed, or infected.

Hence, in the absence of infection, $E^* = 0, I^* = 0,$ and $R^* = 0$.

Thus, the system (2) is reduced into $\Lambda - \mu S = 0$ which implies that $S^* = \frac{\Lambda}{\mu}$.

So, the disease-free equilibrium point $X_0 = (S^*, 0, 0, 0)$.

Hence, $X_0 = \left(\frac{\Lambda}{\mu}, 0,0,0\right)$.

3.3. The Basic Reproduction Number, R_0

The basic reproduction number is the average number of secondary infection generated by infectious individual in his or her infectiousness. It is important in that it is directly related to the effort required to eliminate infection. Using the next generation matrix approach [15]. R_0 is given by $\rho(FV^{-1})$ (the spectral radius of the matrix FV^{-1}). The matrices F and V are given by

$$V = \begin{bmatrix} (\sigma + \mu) & 0 \\ 0 & (\mu + \alpha + \omega) \end{bmatrix} \text{ and } F = \begin{bmatrix} 0 & \beta S \\ 0 & 0 \end{bmatrix}$$

Then we can find the inverse of the matrix V, which is given by

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

Therefore, *t*he basic reproduction number, R_0 is :

$$R_0(\rho(FV^{-1}) = \frac{\sigma\beta\Lambda}{\mu(\sigma+\mu)(\mu+\alpha)(\mu+\alpha+\omega)}$$

3.4. Local Stability Analysis

This section treats the local stability of system (1) using the linearization technique.

Let the system is re-defined as

$$\left.\begin{array}{c} f_1(S, E, I, R) = \Lambda + \gamma R - \beta SI - \mu S \\ f_2(S, E, I, R) = \beta SI - \sigma E - \mu E \\ f_3(S, E, I, R) = \sigma E - \mu I - \alpha I - \omega I \\ f_4(S, E, I, R) = \omega I - \gamma R - \mu R \end{array}\right\} \quad (3)$$

Then, the Jacobian matrix for system (1 and 3) at the point (S, E, I, R) can be written as

$$J=\begin{bmatrix}\frac{\partial f_1}{\partial S} & \frac{\partial f_1}{\partial E} & \frac{\partial f_1}{\partial I} & \frac{\partial f_1}{\partial R}\\ \frac{\partial f_2}{\partial S} & \frac{\partial f_2}{\partial E} & \frac{\partial f_2}{\partial I} & \frac{\partial f_2}{\partial R}\\ \frac{\partial f_3}{\partial S} & \frac{\partial f_3}{\partial E} & \frac{\partial f_3}{\partial I} & \frac{\partial f_3}{\partial R}\\ \frac{\partial f_4}{\partial S} & \frac{\partial f_4}{\partial E} & \frac{\partial f_4}{\partial I} & \frac{\partial f_4}{\partial R}\end{bmatrix}=\begin{bmatrix}-\mu & 0 & -\beta S & \gamma\\ 0 & -(\sigma+\mu) & \beta S & 0\\ 0 & \sigma & -(\alpha+\mu+\omega) & 0\\ 0 & 0 & \omega & -(\mu+\gamma)\end{bmatrix} \quad (4)$$

Theorem 1 The disease free equilibrium point, $X_0=\left(\frac{\Lambda}{\mu},0,0,0\right)$ is locally asymptotically stable if $R_0<1$ and $R_0<\frac{\omega}{(\mu+\gamma)}$ otherwise unstable.

The Jacobian of the system (4) at $X_0=\left(\frac{\Lambda}{\mu},0,0,0\right)$ is given by

$$J(X_0)=\begin{bmatrix}-\mu & 0 & -\beta\frac{\Lambda}{\mu} & \gamma\\ 0 & -(\sigma+\mu) & \beta\frac{\Lambda}{\mu} & 0\\ 0 & \sigma & -(\alpha+\mu+\omega) & 0\\ 0 & 0 & \omega & -(\mu+\gamma)\end{bmatrix} \quad (5)$$

Let k be the Eigen value. Then we have $|J(X_0)-kI|=0$ where I is a 4×4 identity matrix. Thus, we have

$$|J(X_0)-kI|=\begin{vmatrix}-\mu-k & 0 & -\beta\frac{\Lambda}{\mu} & \gamma\\ 0 & -(\sigma+\mu)-k & \beta\frac{\Lambda}{\mu} & 0\\ 0 & \sigma & -(\alpha+\mu+\omega)-k & 0\\ 0 & 0 & \omega & -(\mu+\gamma)-k\end{vmatrix}=0 \quad (6)$$

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

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

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

Where

$$A=1$$

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

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

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

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

Due to the complexity in determining the signs of the remaining Eigen values, we employ Routh-Hurwitz conditions for stability. The Routh-Hurwitz conditions to ensure that all roots of (5) have negative real parts are $A>0$, $B>0$, $E>0$ and $BC>AD, BCD>AD^2+B^2$ Eclearly A and B are positive. For C, D and E are to be positive, set

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

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

For D to be positive, set

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

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

For E to be positive, set

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

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

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

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

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

3.5. Global Stability of the Disease-Free Equilibrium Point

We study the global asymptotic stability of the endemic equilibrium using LaSalle's invariance principle [11].

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

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

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

Thus

$$\begin{bmatrix}E'(t)\\I'(t)\end{bmatrix}\leq\begin{bmatrix}E\\I\end{bmatrix}\qquad(7)$$

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

3.6. The Endemic Equilibrium Point

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

$$X_1=(S^*, E^*, I^*, R^*)>0.$$

From the system of equation the endemic equilibrium point is

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

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

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

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

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

3.7. Local Stability of Endemic Equilibrium Point

We analyze the stability of the endemic equilibrium by linearizing the above system of differential equations (1) to give the Jacobian matrix. The Jacobian matrix is computed by differentiating each system equation (1) with respect to the state variables. 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 (1) 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 (1).

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

3.8. Sensitivity Analysis

Sensitivity analysis is the study carried out to determine the input parameters which affect the output of a model most. Let $S_h$denote the sensitivity ofR_0, and then the sensitivity index R_0 with respect to any parameter h is:

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

The larger the magnitude of the sensitivity index leads to more sensitivity $R_0$with respect to that parameters.

$$\frac{\partial R_0}{\partial\sigma}=1-\frac{\sigma}{\sigma+\mu}>0,\frac{\partial R_0}{\partial\beta}=1>0,\frac{\partial R_0}{\partial\Lambda}=1>0,\frac{\partial R_0}{\partial\omega}=-\frac{\omega^2}{\omega+\mu+\alpha}<0,\frac{\partial R_0}{\partial\mu}=-\frac{\mu^3}{\mu+\alpha+\sigma}<0\text{ and }\frac{\partial R_0}{\partial\alpha}=-\frac{\mu^3}{\alpha+\mu+\omega}<0$$

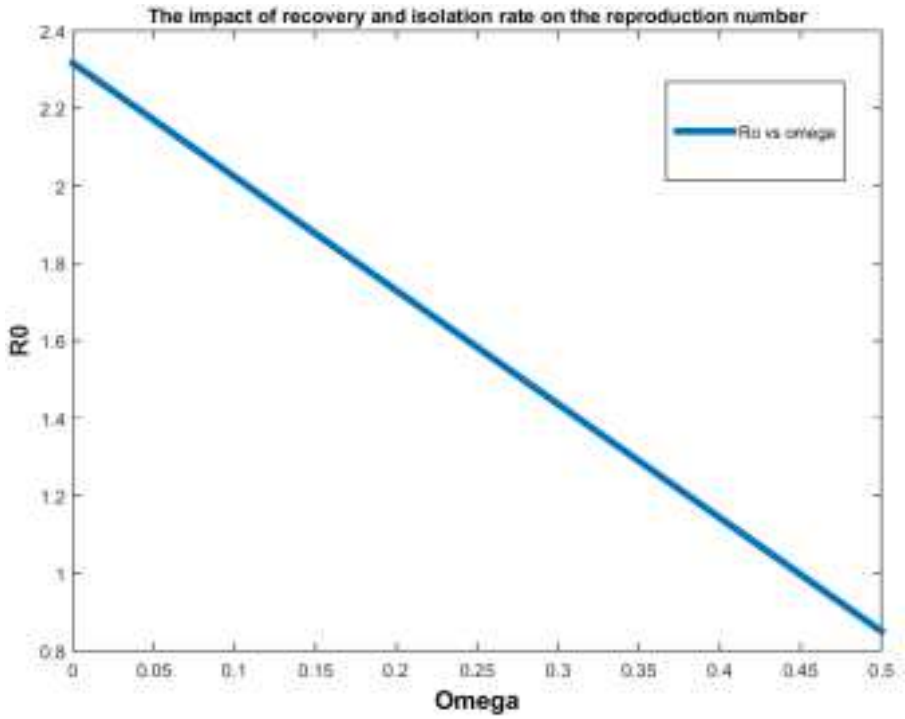

Figure 2. *Sensitivity analysis of reproduction number with respect to treatment rate.*

Figure 2 show that when the treatment rate is increasing the reproduction number is decreasing. Hence, treatment of exposed human population has negative impact of disease transmission.

4. Numerical Simulation

Numerical Simulations of the dynamic model were carried out by MATLAB functionode 45, using the Runge-Kutta of order four. The set of parameter values in table we have used to investigate the effect of treatment in the control of severe water borne disease like diarrhea 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.

Some of the parameter values used:

i. 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 birth which is approximately 50 years. $\mu = \frac{1}{50} = 0.02$ per year.
ii. 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.
iii. Contact rate (β): In this case the contact rate assumed to be constant. It is 0.35.
iv. Human death rate due to diarrhea disease α, va Modeling the rises from country to country. It is as low as 0.07 in developed countries but reaches 0.365 per year in some African countries. Thus we take α =0.365 [12].

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	[12]
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	[17]
ω	0.4	0.4	0.4	0.4	estimated
γ	0.98	0.98	0.98	0.98	estimated

And the following initial conditions have been considered;

$$S[0] = 1150; E[0] = 750; I[0] = 450; R[0] = 95 \text{ at time } t_0 = 0 \text{ and } t_f = 10$$

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 is used in the present model. Let us discuss on the following some numerical outputs.

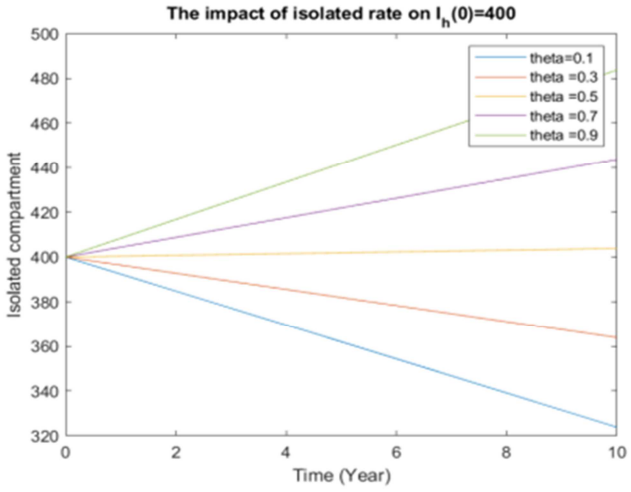

Figure 3. *Effect of infected rate σon exposed compartment.*

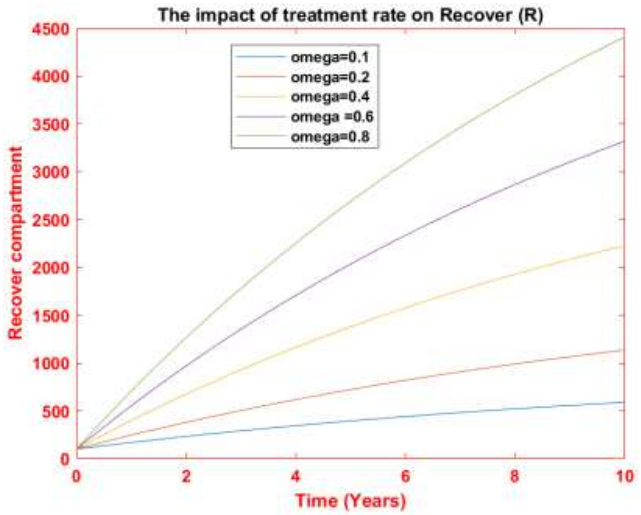

Figure 4. *Effect of treatment rate ω on Recover compartment.*

From figure 3, we observe that the infected rate σ is inversely proportional with the exposed human population. i.e. whenever the rate of infection increases the exposed human compartment decreases through the time.

In figure 4 we can observe that whenever treatment rate is increased throughout the time proportionally the recovered human population increases.

6. Conclusion

In this study, we have formulated a mathematical model describing the transmission of diarrhea disease with treatment as a control. This model has shown significance of treatment in preventing transmission of diarrhea disease in human population. The basic reproduction number has been evaluated using next generation matrix method. 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. Over all, the findings of the numerical simulation shown that effective treatment is sufficient in eradicating diarrhea disease.. It is also indicated that the recovered human population increases as effective treatment is delivered.

Data Availability

The data, which is available with the author, will be made available upon reasonable request.

Authors' Contributions

MT enthusiastically designed methodology, drafted data, analyzed, and interpreted the results.

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