



## A Mathematical Model for the Transmission Dynamics of Diarrhea with Treatment Intervention



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### ABSTRACT

Diarrhea remains a major public health challenge, particularly in developing regions where poor sanitation and limited access to clean water contribute to its rapid spread. This study presents a deterministic compartmental model to better understand the transmission dynamics of the disease. The model categorizes the population into six groups: susceptible, exposed, asymptomatic infected, symptomatic infected, treated, and recovered individuals. Key factors such as human-to-human and waterborne transmission, treatment effectiveness, and recovery rates are incorporated to provide a comprehensive analysis of disease spread and control. A mathematical analysis is conducted to examine both local and global stability, determining the conditions under which the disease can either persist or be eradicated. Sensitivity analysis identifies the most influential factors driving transmission, highlighting the crucial role of reducing contact rate and improving treatment accessibility. Numerical simulations further demonstrate that timely medical intervention and improved sanitation significantly reduce infection rate and disease prevalence.

### Keywords:

Diarrhea transmission,  
Disease control,  
Mathematical  
Modeling,  
Local stability analysis,  
Global stability  
Analysis,  
Sensitivity analysis,  
Numerical simulation.

### INTRODUCTION

Diarrheal disease continues to pose a major public health challenge worldwide, with children under the age of five in low- and middle-income countries bearing the greatest burden. In 2020, the African region recorded an estimated 1.01 billion cases of diarrhea and more than 515,000 associated deaths, highlighting the severity of the problem on the continent (Thystrup, et al., 2024). This considerable disease burden emphasizes the need for strengthened prevention, control, and treatment strategies aimed at reducing diarrheal morbidity and mortality. Diarrhea arises from a complex interplay of causative agents, including bacterial, viral, and parasitic pathogens.

Research conducted in Ile-Ife, Nigeria, revealed that rotavirus was the leading cause of acute diarrhea among children under five, with diarrheagenic *Escherichia coli* ranking next in prevalence (Omotade et al., 2023). These findings underscore the prominent contribution of viral and bacterial pathogens to pediatric diarrheal infections in the region.

Environmental and socioeconomic conditions play a pivotal role in shaping diarrhea incidence. A large-scale systematic review covering studies published between 2013 and 2023 identified poor access to potable water, inadequate sanitation infrastructure, and low socioeconomic status as major contributors to diarrheal disease among African children under five (Shane et al., 2017).

Mitigating these underlying inequities is therefore crucial for lowering disease prevalence and improving child survival outcomes. Preventive interventions, particularly vaccination, have demonstrated significant potential in curbing diarrheal disease incidence. Although rotavirus vaccines are available, their incorporation into routine national immunization programs remains insufficient in several African countries, including Nigeria (Omotade et al., 2023). Expanding rotavirus vaccine coverage through routine immunization schedules could markedly reduce cases of rotavirus-related diarrhea.

Effective diarrhea management relies heavily on early intervention, especially rehydration therapy and, where appropriate, antimicrobial treatment. The Infectious Diseases Society of America recommends reduced-osmolarity oral rehydration solutions as the primary treatment for mild to moderate dehydration resulting from acute diarrhea (Shane et al., 2017). Furthermore, antimicrobial therapy should be pathogen-specific to maximize treatment effectiveness and minimize the development of antimicrobial resistance. Mathematical modeling has become an essential approach for analyzing and controlling infectious diseases, including diarrhea. Through the use of mathematical frameworks, researchers can examine disease transmission mechanisms, assess the effectiveness of intervention strategies, and forecast outbreak scenarios. Compartmental models such as the Susceptible–Infectious–Recovered (SIR) model are commonly employed to describe transitions between disease states, enabling detailed analysis of infection spread and recovery dynamics. These models offer several advantages, including the estimation of critical epidemiological parameters, improved planning for resource distribution during outbreaks, and support for evidence-based public health decision-making. By incorporating factors such as environmental conditions and behavioral patterns, mathematical models provide a more comprehensive understanding of disease dynamics. In diarrheal disease research, modeling techniques have been used to evaluate vaccination and treatment strategies, demonstrating their effectiveness in lowering transmission rates and informing public health interventions (Olutimo et al., 2024).

Several studies have applied mathematical modeling to the study of infectious diseases. Olutimo et al. (2024) proposed a compartmental framework to investigate diarrhea transmission through both direct person-to-person contact and indirect exposure via contaminated water sources. The model differentiates between infants and adults, reflecting the increased susceptibility of infants to diarrheal infections. By incorporating vaccination and treatment components, the study assesses how these interventions influence disease prevalence. The results indicate that focused vaccination efforts and efficient treatment strategies can substantially reduce

disease transmission, especially in settings with high infant mortality. Smith et al. (2023) explored the integration of social vulnerability indicators into infectious disease models. Their findings reveal that communities with limited healthcare access, inadequate sanitation, and low socioeconomic standing experience a disproportionate burden of infectious diseases, including diarrhea. The authors emphasize the importance of embedding social determinants into modeling frameworks to improve predictive accuracy and to design interventions that are both equitable and effective.

Johnson et al. (2023) developed a mathematical model to examine the transmission dynamics of giardiasis, a protozoan infection associated with diarrheal illness. The model accounts for asymptomatic carriers and evaluates intervention strategies such as screening and treatment. Sensitivity analysis identified key parameters driving transmission, offering valuable insights into effective outbreak control measures. Lee et al. (2023) introduced an innovative modeling approach using a piecewise modified ABC (ABC) fractional derivative to describe the spread of acute diarrhea. This method captures both classical and fractional dynamics of disease transmission, allowing for a more detailed representation of disease progression. Analysis of local and global stability around the disease-free equilibrium provides important guidance for implementing effective control strategies. Thompson et al. (2024) highlighted the critical role of mathematical models in outbreak preparedness and response. Their study demonstrates how models can characterize complex transmission patterns and simulate various intervention scenarios. When applied to diarrheal diseases, such models enable public health authorities to anticipate outbreak trends, allocate resources efficiently, and ultimately reduce disease-related morbidity and mortality.

The primary aim of this study is to construct a deterministic compartmental model that accurately represents the transmission dynamics of diarrhea in environments characterized by poor sanitation and limited access to safe water. The study seeks to conduct both local and global stability analyses to determine conditions under which the disease persists or is eliminated, perform sensitivity analysis to identify key parameters influencing transmission—such as contact rate and treatment effectiveness—and apply numerical simulations to assess the impact of intervention strategies, including sanitation improvements and timely treatment, on reducing infection rates and controlling disease spread

The novelty of this model lies in its detailed compartmentalization of diarrhea transmission dynamics, distinguishing between *exposed*, *asymptomatic infected*, *symptomatic infected*, *treated*, and *recovered* individuals. Unlike simpler models, it accounts for asymptomatic carriers and their role in disease spread. It also

incorporates treatment as a dynamic process with separate rates for symptomatic and asymptomatic infections, allowing for better evaluation of intervention strategies. Additionally, it includes key epidemiological parameters such as *progression rates*, *effective contact rates*, *recovery rates*, and *disease-induced mortality*, providing a more realistic representation of transmission.

## MATERIALS AND METHODS

### Model Formulation

In this section, a deterministic compartmental model on the transmission dynamics of diarrhea is been formulated. The total human population  $N_H(t)$ , is subdivide into six (6) epidemiological classes of susceptible humans  $S$ , exposed humans to diarrhea infection  $E$ , asymptomatic infected humans with diarrhea  $I_A$ , symptomatic infected humans with diarrhea  $I_S$ , treatment class of diarrhea  $T$ , and recovered

individuals  $R$  from this diarrhea disease. The constant recruitment rate of individuals into the susceptible compartment is at the rate  $\Lambda_H$  so that  $\beta$  denotes the effective contact rate with the probability of infection per contact with infected human with diarrhea disease and the rate progression rate from exposed class to symptomatic class is  $\theta(1-\omega)$ . The rate at which remaining exposed individuals move to asymptomatic class is given as  $\theta\omega$  and  $\alpha_1$  is the symptom gain rate of asymptomatic infected individuals.  $\alpha_2$  and  $\alpha_3$  are the treatment rates of  $I_S$  and  $I_A$  respectively whereas  $\phi_1$  and  $\phi_2$  are the wining and recovery rates respectively. The natural death rate of human in any compartment is given as  $\mu_H$  and the disease induced death rate of humans due to diarrhea infection is  $\sigma$

Table 1. Variable/Parameter and Descriptions

| Variable           | Description   |
|--------------------|---|
| $S$                | Susceptible   |
| $E$                | Exposed   |
| $I_A$              | Asymptomatic infected                                   |
| $I_S$              | Symptomatic infected                                    |
| $T$                | Treatment class   |
| $R$                | Recovered   |
| Parameter          | Description   |
| $\Lambda$          | Recruitment rate  |
| $\beta$            | Contact rate  |
| $\theta\omega$     | Progression rate from $E$ to $I_A$                      |
| $\theta(1-\omega)$ | Progression rate from $E$ to $I_S$                      |
| $\alpha_1$         | Symptom gain rate                                       |
| $\alpha_2$         | Treatment rate of $I_S$                                 |
| $\alpha_3$         | Treatment rate of $I_A$                                 |
| $\mu$              | Natural death rate                                      |
| $\sigma$           | Disease induced death rate                              |
| $\phi_2$           | Recovery rate   |
| $\phi_1$           | Rate at which recovered individuals becomes susceptible |

Table 1 presents descriptions of parameters and variables used in the model. The transition of individuals from one compartment to another is dependent on the rates described in the above table 1.

### Model Assumptions

The model is formulated based on the following mathematical assumptions.

1. The population mixture is homogeneous, age, social status or gender does not affect the probability of an individual been infected.
2. The model considered both birth and death rates
3. The recovered individuals can also be susceptible to diarrhea disease

4. The mode of transmission considered in this model is direct (from human to human)
5. There is no inherited immunity

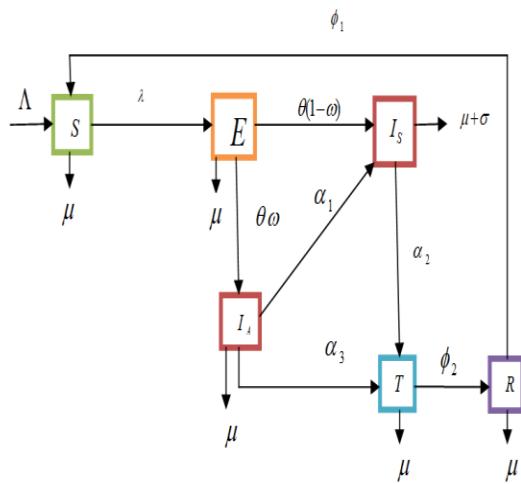


Figure 1. Schematic diagram for the model

#### Model equations

From the parameters described in table 1 and the above schematic we have the following system of differential equations.

$$\frac{dS}{dt} = \Lambda + \phi_1 R - (\lambda + \mu)S$$

$$\frac{dE}{dt} = \lambda S - (\theta + \mu)E$$

$$\frac{dI_A}{dt} = \theta\omega E - (\alpha_1 + \alpha_3 + \mu)I_A$$

$$\frac{dI_S}{dt} = \theta(1-\omega)E + \alpha_1 I_A - (\mu + \sigma + \alpha_2)I_S$$

$$\frac{dT}{dt} = \alpha_2 I_S + \alpha_3 I_A - (\mu + \phi_2)T$$

$$\frac{dR}{dt} = \phi_2 T - (\mu + \phi_1)R$$

$$\lambda = \frac{\beta(I_S + I_A)}{N}$$

## RESULTS AND DISCUSSION

#### Model Analysis

Let  $N(t)$  be the total population at time  $t$ . Hence, the total population is expressed as:

$$N(t) = S(t) + E(t) + I_A(t) + I_S(t) + T(t) + R(t)$$

Where the state variables have their usual meaning as defined in table 1.

The differential equation yields

$$\frac{dN(t)}{dt} = \Lambda - \mu N \quad (2)$$

#### Lemma 1

Let  $(S, E, I_A, I_S, T, R)$  be the solution of the model (1) with initial conditions in a epidemiological feasible region  $D$  with:

$$D = S, E, I_A, I_S, T, R \in R_+^6 : N \leq \frac{\Lambda}{\mu} \quad (3)$$

Then  $D$  is non-negative invariant

From the result of Somma et al (2019), we obtain

$$0 \leq N(t) \leq N_h(0)e^{-\mu(t)} + \frac{\Lambda}{\mu} \left(1 - e^{-\mu(t)}\right) \quad (4)$$

Therefore, the set  $D$  is positively invariant for all  $t$

#### Asymptotic Stability of the Disease Free Equilibrium of the Diarrhea Model

The disease-free equilibrium (DFE) describes a condition in which an infectious disease is entirely eliminated from a population, leaving no individuals affected. This scenario signifies either the successful eradication of the disease or its inability to propagate within the community (Van den Driessche & Watmough, 2002). When minor disturbances in the system fail to reintroduce the infection, the DFE is deemed stable, suggesting that long-term disease elimination is achievable. The disease free equilibrium of our model is given by

$$\eta_0 = \left\{ S^*, E^*, I_A^*, I_S^*, T^*, R^* \right\} = \left\{ \frac{\Lambda}{\mu}, 0, 0, 0, 0, 0 \right\}$$

#### Basic Reproduction Number of the Model

The basic reproduction number is obtained as follows

$$R_0 = \rho(FV^{-1}), \quad \rho \text{ is given by dominant eigenvalue of}$$

$FV^{-1}$  where  $F$  and  $V$  matrices are the new infection terms and the remaining transfer terms respectively. Following the result obtained in (Van den Driessche & Watmough, 2002), we have

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

$$V = \begin{bmatrix} P_1 & 0 & 0 & 0 \\ -\theta\omega & P_2 & 0 & 0 \\ -\theta(1-\omega) & -\alpha_1 & P_3 & 0 \\ 0 & -\alpha_3 & -\alpha_2 & P_4 \end{bmatrix}$$

$$F.V^{-1} = \begin{bmatrix} -\frac{\theta((P_2 - P_3 - \alpha_1)\omega - P_2)\beta}{P_2 P_1 P_3} & \frac{\beta(P_3 + \alpha_1)}{P_3 P_2} & \frac{\beta}{P_3} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} J(\varepsilon_0) =$$

$$\begin{aligned} & \lambda^4 + (P_4 + P_3 + P_2 + P_1)\lambda^3 + (\beta\omega\theta + P_1 P_2 + P_1 P_3 + P_1 P_4 + P_3 P_2 + P_2 P_4 + P_3 P_4 - \beta\theta)\lambda^2 \\ & + \left( P_2 \beta\omega\theta + P_4 \beta\omega\theta - \beta\omega\theta\alpha_1 + \beta\omega\theta\alpha_2 - \beta\omega\theta\alpha_3 + P_3 P_2 P_1 + P_1 P_2 P_4 \right. \\ & \left. + P_1 P_3 P_4 + P_2 P_3 P_4 - P_2 \beta\theta - P_4 \beta\theta - \beta\theta\alpha_2 \right. \\ & \left. + P_1 P_2 P_3 (1 - R_0) \right) \end{aligned}$$

Applying the Routh Hurwitz criterion, we have that

$$(1 - R_0) > 0$$

$\Rightarrow R_0 < 1$ , thus the model locally asymptotically stable.

Where  $P_1 = (\theta + \mu)$ ,  $P_2 = (\alpha_1 + \alpha_3 + \mu)$ ,  
 $P_3 = (\mu + \sigma + \alpha_2)$ ,  $P_4 = (\mu + \phi_2)$ , s

#### Global Asymptotic Stability of the Disease-Free Equilibrium Point of the Diarrhea Model.

To investigate the global stability of the disease free equilibrium, we use the technique implemented by Castillo-Chavez and song (2004).

To do this, we write the equation in the uninfected class as

$$\frac{dX}{dt} = F(X, Z)$$

And we re-write the equation in the infected class as

$$\frac{dZ}{dt} = G(X, Z)$$

Where  $X = (S, R) \in R^2_+$  denotes the uninfected population and

$$R_0 = -\frac{\beta\theta((P_2 - P_3 - \alpha_1)\omega - P_2)}{P_2 P_1 P_3}$$

$$J(\varepsilon_0) = \begin{bmatrix} -\mu & 0 & 0 & -\beta & -\beta & \phi_1 \\ 0 & -P_1 & 0 & \beta & \beta & 0 \\ 0 & \theta\omega & -P_2 & 0 & 0 & 0 \\ 0 & 0 & \alpha_3 & \alpha_2 & -P_4 & 0 \\ 0 & 0 & 0 & 0 & \phi_2 & -P_5 \end{bmatrix}$$

The reduced Jacobian matrix becomes

$$J(\varepsilon_0) = \begin{bmatrix} -P_1 & 0 & \beta & \beta \\ \theta\omega & -P_2 & 0 & 0 \\ \theta(1-\omega) & \alpha_1 & -P_3 & 0 \\ 0 & \alpha_3 & \alpha_2 & -P_4 \end{bmatrix}$$

The eigenvalues of the reduced Jacobian matrix becomes

$$Z = (E, I_A, I_S, T) \in R^4_+$$

denotes the infected population

$$\varepsilon_0 = (X^*, 0)$$

represent the disease-free equilibrium of the system, and it globally asymptotically stable if it satisfies the following conditions:

$$H_1: \frac{dX}{dt} = F(X^*, 0), X^* \text{ is globally}$$

asymptotically stable

$$H_2: \frac{dZ}{dt} = D_Z G(X^*, 0)Z - \hat{G}(X, Z)$$

$\hat{G}(X, Z) \geq 0$  for all  $(X, Z) \in D$  and where

$D_Z G(X^*, 0)$  is an M-matrix (i.e the diagonal elements are no-negative and it is also the Jacobian of  $\hat{G}(X, Z)$  evaluated at  $(X^*, 0)$ ).

If the system satisfies the above condition, then the theorem below holds.

#### Theorem 1

The equilibrium point  $\varepsilon_0 = (X^*, 0)$  is globally asymptotically stable if  $R_0 \leq 1$

$$F(X, Z) = \begin{bmatrix} \Lambda + \phi_1 R - (\lambda + \mu) S \\ \phi_2 T - P_5 R \end{bmatrix} \quad , \quad \hat{G}(X, Z) = D_z G(X^*, 0) Z - G(X, Z)$$

$$G(X, Z) = \begin{bmatrix} \lambda S - P_1 E \\ \theta \omega E - P_2 I_A \\ \theta(1 - \omega) E + \alpha_1 I_A - P_3 I_s \\ \alpha_2 I_s + \alpha_3 I_A - P_4 T \end{bmatrix}$$

$$\hat{G}(X, Z) = \begin{bmatrix} \beta(I_s + I_A) \left(1 - \frac{S}{N}\right) \\ 0 \\ 0 \\ 0 \end{bmatrix}$$

At disease free equilibrium,

$H_1$ :

$$\frac{dS}{dt} = \Lambda - \mu S$$

$$\frac{dR}{dt} = 0$$

$H_2$ :

$$D_z G(X^*, 0) Z = \begin{bmatrix} \frac{\beta(I_s + I_A) S}{N} - P_1 E \\ \theta \omega E - P_2 I_A \\ \theta(1 - \omega) E + \alpha_1 I_A - P_3 I_s \\ \alpha_2 I_s + \alpha_3 I_A - P_4 T \end{bmatrix}$$

$$S^{**} = \frac{\Lambda P_1 P_2 P_3 P_4 P_5}{(-(-\alpha_2(\omega-1)P_2 + \omega(P_3\alpha_3 + \alpha_1\alpha_2))\theta\phi_2\phi_1 + P_1 P_2 P_3 P_4 P_5)\lambda + P_1 P_2 P_3 P_4 P_5\mu}$$

$$E^{**} = \frac{\Lambda P_2 P_3 P_4 P_5 \lambda}{(-(-\alpha_2(\omega-1)P_2 + \omega(P_3\alpha_3 + \alpha_1\alpha_2))\theta\phi_2\phi_1 + P_1 P_2 P_3 P_4 P_5)\lambda + P_1 P_2 P_3 P_4 P_5\mu}$$

$$I_A^{**} = \frac{\Lambda P_3 P_4 P_5 \lambda \omega \theta}{(-(-\alpha_2(\omega-1)P_2 + \omega(P_3\alpha_3 + \alpha_1\alpha_2))\theta\phi_2\phi_1 + P_1 P_2 P_3 P_4 P_5)\lambda + P_1 P_2 P_3 P_4 P_5\mu}$$

$$I_s^{**} = \frac{\Lambda((1-\omega)P_2 + \alpha_1\omega)\theta P_4 P_5 \lambda}{((\omega\theta\alpha_2\phi_1\phi_2 + P_1 P_3 P_4 P_5 - \theta\alpha_2\phi_1\phi_2)P_2 - \phi_1\phi_2\omega\theta(P_3\alpha_3 + \alpha_1\alpha_2))\lambda + P_1 P_2 P_3 P_4 P_5\mu}$$

$$T^{**} = \frac{(((\alpha_1 - P_2)\omega + P_2)\alpha_2 + \omega\alpha_3 P_3)\Lambda\theta P_5 \lambda}{-\phi_1((\alpha_1 - P_2)\omega + P_2)\theta\phi_2\lambda\alpha_2 + P_3((-\omega\theta\alpha_3\phi_1\phi_2 + P_1 P_2 P_4 P_5)\lambda + \mu P_1 P_2 P_4 P_5)}$$

$$R^{**} = \frac{(((\alpha_1 - P_2)\omega + P_2)\alpha_2 + \omega\alpha_3 P_3)\Lambda\theta\phi_2\lambda}{-\phi_1((\alpha_1 - P_2)\omega + P_2)\theta\phi_2\lambda\alpha_2 + P_3((-\omega\theta\alpha_3\phi_1\phi_2 + P_1 P_2 P_4 P_5)\lambda + \mu P_1 P_2 P_4 P_5)}$$

Substituting into the force of infection for the diarrhea model

Clearly,  $1 \geq \frac{S}{N}$  this implies that  $\hat{G}(X, Z) \geq 0$ .

Therefore the disease free equilibrium of the diarrhea model is globally asymptotically stable.

#### Endemic Equilibrium Point of the Diarrhea Model

The endemic equilibrium is the state where disease persist in the population. The endemic equilibrium of our model is given by  $\eta_1 = \{S^{**}, E^{**}, I_A^{**}, I_s^{**}, T^{**}, R^{**}\}$

$$\lambda = \frac{\beta(I_s + I_A)}{N}$$

We obtained

$$\lambda \left\{ \left( \left( \left( \left( -\alpha_2 - P_4 \right) P_2 + \left( \alpha_1 + P_3 \right) P_4 + \alpha_1 \alpha_2 + P_3 \alpha_3 \right) P_5 - \phi_2 \left( P_2 \alpha_2 - P_3 \alpha_3 - \alpha_1 \alpha_2 \right) \right) \omega \right)_{\theta + P_2 P_3 P_4 P_5} \right)_{\lambda} \right\} = 0$$

$$\lambda \neq 0$$

$$\Rightarrow \lambda = \frac{P_1 P_2 P_3 P_4 P_5 (1 - R_0)}{\left( \left( \left( \left( -\alpha_2 - P_4 \right) P_2 + \left( \alpha_1 + P_3 \right) P_4 + \alpha_1 \alpha_2 + P_3 \alpha_3 \right) P_5 - \phi_2 \left( P_2 \alpha_2 - P_3 \alpha_3 - \alpha_1 \alpha_2 \right) \right) \omega \right)_{\theta + P_2 P_3 P_4 P_5} \right)}$$

$$(R_0 - 1) > 0$$

$$\Rightarrow R_0 > 1$$

Thus, the endemic equilibrium point of the model is stable.

#### Sensitivity Analysis of the Model

In infectious disease modeling, sensitivity analysis is particularly useful for evaluating key epidemiological parameters such as transmission rates, recovery rates, and intervention effectiveness (Agbata et al, 2025). The sensitivity index of the reproduction number of the model with respect to any parameter say  $x$  is given by:

$$\mathfrak{S}_x^{R_0} = \frac{\partial R_0}{\partial x} \times \frac{x}{R_0}$$

$$\text{Given that } R_0 = -\frac{\beta \theta ((P_2 - P_3 - \alpha_1) \omega - P_2)}{P_2 P_1 P_3}$$

5. For  $\alpha_3$ :

$$\mathfrak{S}_{\alpha_3}^{R_0} = \frac{-\beta \theta \omega}{(\alpha_1 + \alpha_3 + \mu)(\mu + \sigma + \alpha_2)}$$

6. For  $\mu$ :

$$\mathfrak{S}_{\mu}^{R_0} = \frac{-\beta \theta (\omega(\mu + \sigma + \alpha_2))}{(\alpha_1 + \alpha_3 + \mu)(\theta + \mu)(\mu + \sigma + \alpha_2)}$$

7. For  $\sigma$ :

$$\mathfrak{S}_{\sigma}^{R_0} = \frac{-\beta \theta}{(\mu + \sigma + \alpha_2)}$$

1. For  $\beta$ :

$$\mathfrak{S}_{\beta}^{R_0} = 1$$

8. For  $\omega$ :

2. For  $\theta$ :

$$\mathfrak{S}_{\theta}^{R_0} = \frac{((\alpha_1 + \alpha_3 + \mu) - (\mu + \sigma + \alpha_2) - \alpha_1) \omega - (\alpha_1 + \alpha_3 + \mu)}{(\alpha_1 + \alpha_3 + \mu)(\theta + \mu)}$$

1. For  $\beta$ :

$$\mathfrak{S}_{\beta}^{R_0} = 1$$

3. For  $\alpha_1$ :

$$\mathfrak{S}_{\alpha_1}^{R_0} = \frac{-\omega(\mu + \sigma + \alpha_2)}{(\alpha_1 + \alpha_3 + \mu)(\mu + \sigma + \alpha_2)}$$

2. For  $\theta$ :

$$\mathfrak{S}_{\theta}^{R_0} = -4.2944$$

4. For  $\alpha_2$ :

3. For  $\alpha_1$ :

$$\mathfrak{J}_{\alpha_1}^{R_0} = -1.9231$$

4. For  $\alpha_2$ :

$$\mathfrak{J}_{\alpha_2}^{R_0} = 0.0014$$

5. For  $\alpha_3$ :

$$\mathfrak{J}_{\alpha_3}^{R_0} = -0.6154$$

6. For  $\mu$ :

$$\mathfrak{J}_{\mu}^{R_0} = -4.5792$$

7. For  $\sigma$ :

$$\mathfrak{J}_{\sigma}^{R_0} = -0.5186$$

8. For  $\omega$ :

$$\mathfrak{J}_{\omega}^{R_0} = -0.00001$$

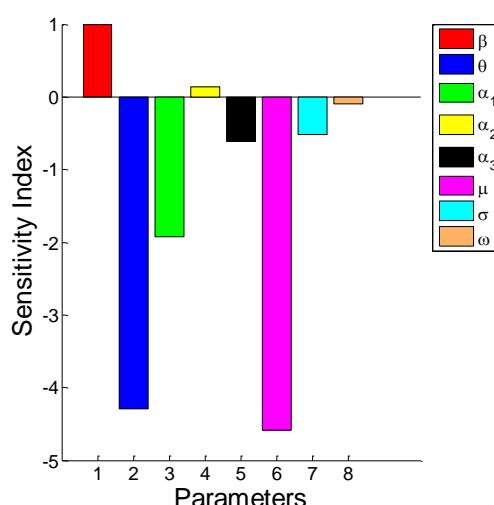


Figure 2. Bar chart of diarrhea sensitivity Indices

### Numerical Simulations

The sensitivity analysis highlights key parameters that influence the spread of diarrhea, with positive sensitivity indices indicating factors that increase transmission, such as human-to-human and waterborne contact. Controlling these factors through improved sanitation and reduced exposure can help mitigate outbreaks (Agbata et al, 2024). Conversely, parameters with negative sensitivity indices, like treatment rate, showed that timely and effective medical interventions significantly reduce disease prevalence. Strengthening healthcare systems, enhancing treatment accessibility, and implementing proactive public health measures, such as sanitation improvements and early diagnosis, are essential in controlling and preventing diarrhea outbreaks.

Numerical simulation is a crucial step in analyzing the behavior of mathematical models, particularly in infectious disease modeling, where complex differential equations govern disease dynamics. In this study, MATLAB is used to perform numerical simulations to understand the real-life behavior of the formulated model equations for diarrhea transmission (Acheneje et al 2024). MATLAB is a powerful computational tool that provides robust numerical solvers for solving systems of ordinary differential equations (ODEs), allowing researchers to visualize disease progression under various conditions. MATLAB-based numerical simulations allow for a comprehensive analysis of the formulated model equations, offering valuable predictions about diarrhea transmission and control.

Table 2. Parameter values used for Simulation

| Parameters | Values  | Source                |
|------------|---------|-----------------------|
| $\Lambda$  | 2000    | Olutimo et al, 2024   |
| $\beta$    | 0.500   | Olutimo et al, 2024   |
| $\theta$   | 0.700   | Olutimo et al, 2024   |
| $\omega$   | 0.100   | Olutimo et al, 2024   |
| $\alpha_1$ | 1.00    | Assumed               |
| $\alpha_2$ | 0.98    | Odeh et al, 2024      |
| $\alpha_3$ | 0.0003  | Olutimo et al, 2024   |
| $\mu$      | 0.200   | Olutimo et al, 2024   |
| $\sigma$   | 0.100   | Agbata et al, 2024    |
| $\phi_2$   | 0.05    | Bolarinwa et al, 2024 |
| $\phi_1$   | 0.00045 | Assumed               |

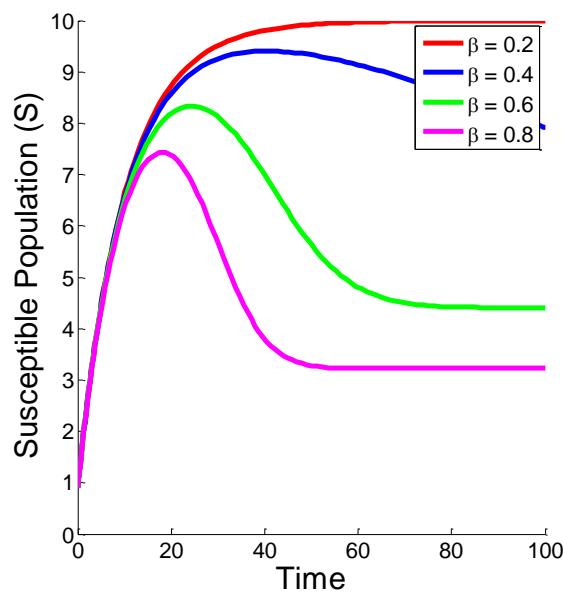


Figure 3a. Graph of susceptible humans

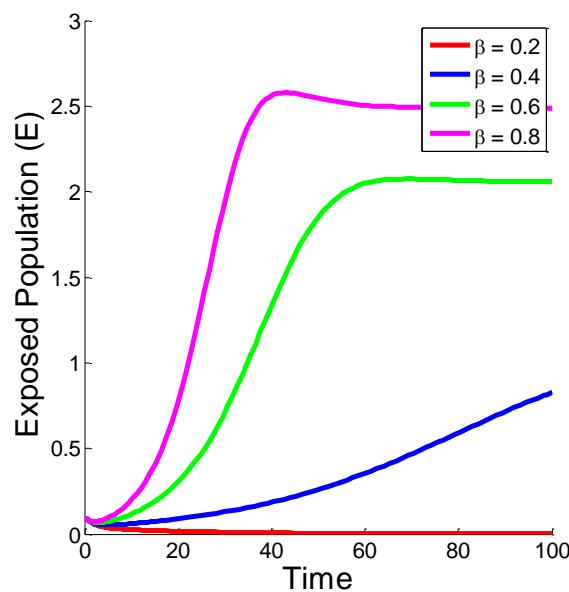
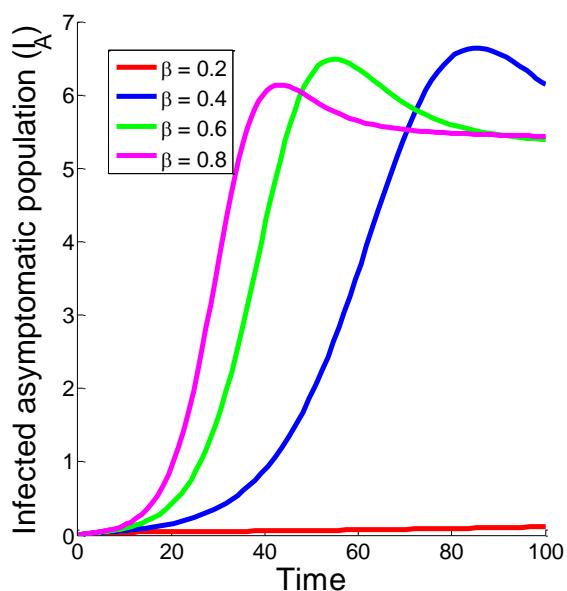
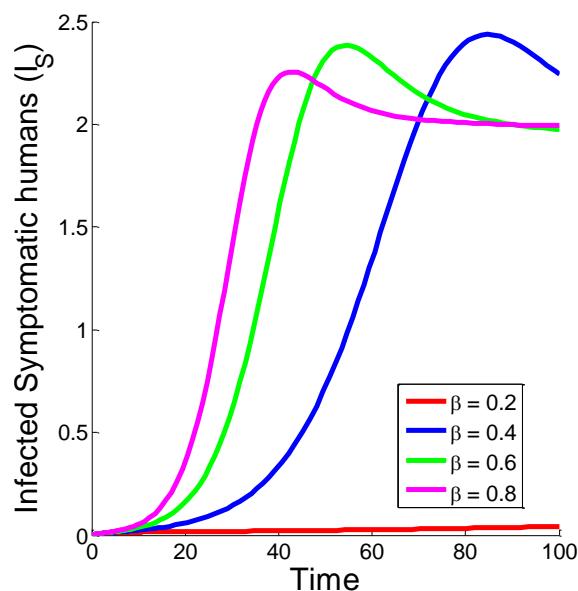


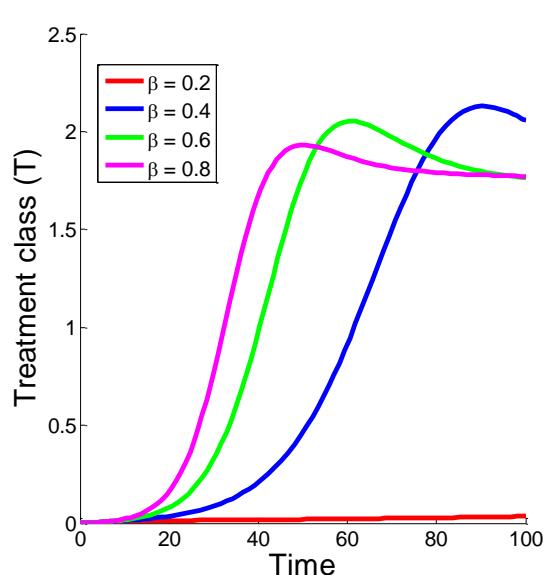
Figure 3b. Graph of exposed humans



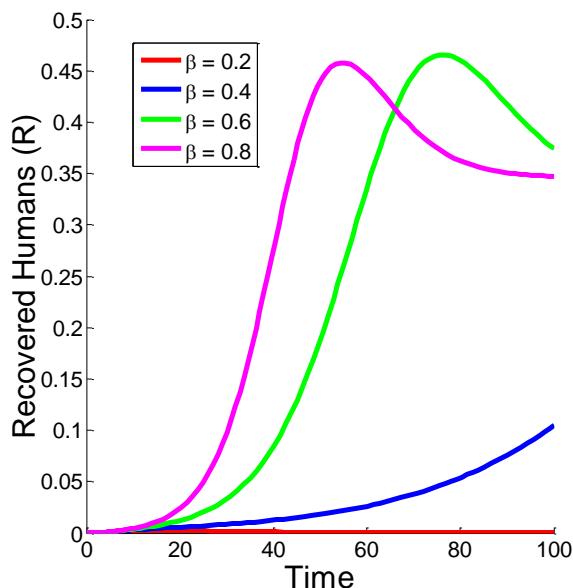
**Figure 3c. Graph infected asymptomatic humans**



**Figure 3d. Graph infected symptomatic humans**



**Figure 3e. Graph of treated humans**



**Figure 3f. Graph of recovered humans**

**Figure 3a** illustrates the graph of susceptible humans over time. Initially, the number of susceptible individuals increases rapidly; however, it later declines towards zero. A similar trend is observed in **Figure 3b**, where the number of exposed individuals sharply increases before gradually decreasing to zero. This pattern suggests effective disease control, as the population of both susceptible and exposed individuals diminishes over time. In **Figures 3c and 3d**, an increase in the contact rate between susceptible and infected individuals leads to a rise in the number of infected asymptomatic and

symptomatic individuals, respectively. This observation indicates that higher contact rates contribute to the spread of the infection. Consequently, reducing the contact rate between susceptible and infected individuals could significantly curb the transmission of the disease. **Figure 3e** highlights the impact of a high treatment rate for infected individuals. The data suggest that effective treatment plays a crucial role in controlling diarrhea infections by reducing the duration and severity of the illness. This increased treatment rate directly contributes to a higher recovery rate, as depicted in **Figure 3f**.

Overall, the figures emphasize two key strategies for controlling the spread of the disease: reducing contact rates between susceptible and infected individuals and ensuring prompt and effective treatment of infected persons.

## CONCLUSION

This study provides a detailed analysis of diarrhea transmission dynamics, emphasizing key factors that influence its spread and control. The findings highlight that reducing contact between susceptible and infected individuals is essential in limiting disease transmission. An increase in contact rates significantly raises the number of infections, both symptomatic and asymptomatic, reinforcing the need for strict public health measures. Additionally, effective treatment plays a crucial role in reducing disease prevalence by shortening infection duration and increasing recovery rates. A higher treatment rate directly leads to better disease outcomes, demonstrating the importance of strengthening healthcare systems and ensuring timely medical intervention. Sensitivity analysis further confirms that transmission-related factors, such as human-to-human contact and exposure to contaminated water, contribute to disease persistence, while improved sanitation and medical treatment significantly reduce its impact. Numerical simulations support these findings, showing that a combination of reduced exposure, enhanced sanitation, and prompt treatment can effectively curb diarrhea outbreaks. This study underscores the importance of early diagnosis, access to healthcare, and proactive public health interventions in managing and preventing the disease. Strengthening sanitation infrastructure, promoting hygiene awareness, and ensuring effective medical care are critical steps toward long-term disease control. These insights provide a valuable foundation for policymakers and health authorities in designing targeted strategies to reduce the burden of diarrhea and improve public health outcomes.

## Recommendations

- 1. Implement Public Health Measures to Reduce Contact Rates:** Authorities should enforce and promote interventions that minimize contact between susceptible and infected individuals such as isolation of symptomatic patients, public education on hygiene, and safe community practices to effectively lower transmission rates.
- 2. Strengthen Healthcare Systems for Timely and Effective Treatment:** Investments should be made in healthcare infrastructure to ensure rapid diagnosis and prompt treatment of diarrhea

cases, which can significantly reduce the duration of infection and improve recovery rates.

- 3. Enhance Sanitation and Clean Water Access:** Government and non-governmental organizations should prioritize the development and maintenance of clean water sources and sanitation facilities to reduce exposure to waterborne pathogens.
- 4. Promote Hygiene Education and Community Awareness:** Public health campaigns must be expanded to educate communities on personal hygiene practices, safe food and water consumption, and the importance of seeking early medical care to prevent the spread of diarrhea.

**Adopt Data-Driven Strategies for Targeted Interventions:** Policymakers should utilize sensitivity and simulation analysis from models like this study to identify high-impact intervention points, enabling the efficient allocation of resources and the development of tailored responses in vulnerable regions.

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