MATHEMATICAL MODELING AND ANALYSIS OF DIARRHEA TRANSMISSION WITH CAMPAIGN-BASED CONTROL MEASURE AND TREATMENT

ADESOLA, O. I.[1*] – UWAUNYIN, N. G.[1] – OLADAPO, O. P.[1]

[1] *Department of Mathematics and Statistics, Federal University Wukari, Taraba State, Nigeria.*

**Corresponding author*
e-mail: isaac.olopade[at]fuwukari.edu.ng

(Received 30th December 2024; revised 20th March 2025; accepted 28th March 2025)

Abstract. The SEIR (Susceptible-Exposed-Infectious-Recovered) model is a well-established framework for examining the spread of infectious diseases, including diarrhea. This study enhances the traditional SEIR model by incorporating a campaign parameter including hygiene practices, environmental cleanliness, and waste management, as a control measure to assess its effectiveness in managing diarrheal disease outbreaks. The model's stability was analyzed, and the basic reproduction number (R_0) was derived using the next-generation matrix method. Findings indicate that the disease-free equilibrium is locally asymptotically stable when $R_0<1$ suggesting that the infection can be controlled within the population. Conversely, when $R_0<1$ the equilibrium is unstable, indicating continued spread of the infection. Results emphasize that improving hygiene practices, environmental cleanliness, avoiding contaminated food product and waste management significantly reduces exposure among susceptible individuals, thereby decreasing the infection rate and enhancing recovery. Forward sensitivity analysis highlights the critical factors affecting disease transmission, underscoring the need for targeted interventions in high-risk areas. Numerical simulations demonstrate the effectiveness of public health campaigns and other epidemiological measures in controlling diarrheal outbreaks, offering valuable insights for public health policy and strategic resource allocation.
Keywords: *campaign, equilibrium point, reproduction number, sensitivity and stability*

Introduction

A state in which an individual continuously or frequently passes watery stool is referred to as diarrheal (WHO, 2020). Diarrhea occurs in the intestine as a result of different bacteria, viruses or parasites from contaminated food and water (especially in sittings with poor hygiene), contact with contaminated surfaces or object, digestive disorder, reaction to medication (WHO, 1995). It can be considered in two stages, the first stage is called acute diarrhea (for a short while as few days), the second stage is called chronic diarrhea (lasting for a week or longer) (WHO, 2020). Diarrhea comes with passing stool, abdominal pain, nausea and dehydration. An individual infected with diarrhea should drink Oral rehydration solution (ORS), electrolyte-rich beverages like coconut water or sports drinks, eating of nutrient rich food, taking of balance diet which includes banana, rice, apples (WHO, 2020). These balance diet and easily digestible food can help firm up stools and when it is chronic the percent should be given antibiotics (De Bruyn et al., 1996; WHO, 1995). However, there are other effective alternatives in preventing and controlling diarrhea. Diarrhea remains a major public health challenge globally, particularly in low- and middle-income countries (Hoyle et al., 2023). The ability to model its transmission and control through vaccination and other strategies has attracted significant attention from both medical and mathematical researchers (Tao et al., 2021). Many researchers have utilized mathematical models as powerful tools to study the epidemiology of diseases across diverse populations. These

models serve as critical frameworks for understanding how diseases spread, predict potential outbreaks, and assess the impact of various interventions (Adesola et al., 2024a; 2024b; Ajao et al., 2023; Philemon et al., 2023; Musibau et al., 2022; Akinwumi et al., 2021; Olopade et al., 2021a; 2021b; Heesterbeek et al., 2015).

According to Siettos and Russo (2013), these mathematical frameworks provide valuable insights into understanding and managing outbreaks. They facilitate the exploration of disease dynamics, allowing for the prediction of transmission patterns and the impact of interventions. Ki (2006) provided a comprehensive review of simulation models used to analyze the dynamics of infectious diseases. The authors explore various modeling approaches that have been developed to better understand disease transmission and inform public health interventions. Pitzer et al. (2014) investigated the role of vaccination in controlling diarrheal diseases, demonstrating that vaccinating susceptible individuals can significantly reduce the transmission rate. This research emphasizes the importance of immunization as a public health strategy, highlighting its dual benefit: not only does vaccination protect individuals, but it also contributes to herd immunity, subsequently decreasing the overall incidence of disease within the community (Rasmussen, 2020; Dembek et al., 2018). Canga and Bidegain (2022) advanced this research by employing systems of differential equations to analyze the interactions between vaccinated, unvaccinated, and initially infected populations. Their findings illustrate how mathematical modeling can predict the duration and dynamics of disease outbreaks, enabling health authorities to tailor interventions more effectively. Das et al. (2013) further underscored the significance of controlling diarrhea through vaccination and treatment. They advocated for comprehensive strategies that combine vaccination with effective treatment options to manage outbreaks effectively. In addition to vaccination, other preventive measures are critical for controlling diarrhea. In WHO (1995) recommends strategies that include ensuring access to clean water, promoting personal hygiene, and improving environmental sanitation. Hoyle et al. (2023) highlight that regular hand washing with soap significantly reduces the incidence of diarrheal diseases, emphasizing behavior change as a vital component of public health initiatives. Ki (2006) allows researchers to simulate various scenarios based on different vaccination strategies, contact rates, and public health interventions.

Exclusive breastfeeding for the first six months of life is also noted to provide protective effects against infections, including diarrhea (Victora et al., 2016). These non-vaccine interventions are essential in a multi-faceted approach to controlling diarrheal diseases. Recent advancements, particularly the introduction of rotavirus vaccines, have been groundbreaking in preventing severe diarrheal diseases among children. Research has shown that these vaccines reduce hospitalization rates and overall mortality associated with diarrhea. Ongoing studies from 2020 to 2024 reinforce the efficacy of these vaccines and their critical role in public health strategies aimed at reducing the burden of diarrheal disease. The integration of mathematical modeling and preventive strategies is crucial for effectively managing and controlling diarrhea. Continued research in this field can enhance the understanding of disease dynamics and inform public health interventions. By combining treatment efforts with improved sanitation and hygiene practices, communities can significantly reduce the burden of diarrheal diseases. This work examines the influence of campaign parameters combined with treatment impact on controlling diarrhea disease.

Materials and Methods

The model focuses on four compartmental models to acquire insight into the effect of campaign and treatment rate on the transmission of diarrhea disease in a community. The model includes the Susceptible individuals $S(t)$ Exposed individuals $E(t)$, Infected individuals, Recovered $I(t)$ individuals $R(t)$ (Eq. (1), so that the susceptible population increases by birth at a constant rate λ and all individuals in the recovered class returns to the susceptible due to lose of immunity at a rate ϕ ,where β is the effective contact rate. The population of the susceptible decreases due to the campaign parameter at a rate $(1-z)$ and natural death at the rate μ (Eq. (2).

$$N(t) = S + E + I + R \qquad \text{Eq. (1)}$$

$$\frac{dS}{dt} = \lambda - (1-z)\beta SI + \phi R - \mu S \qquad \text{Eq. (2)}$$

The exposed individuals are the ones that carry the bacteria but there are not capable of infecting the susceptible class (*Table 1* and *Table 2*), it increases by new infected susceptible individuals who acquired diarrhea with effective contact rate β. The population decreases due the rate at which exposed individuals progresses infected class at the rate σ and through natural death rate μ (Eq. (3) (*Figure 1*). The infected individuals increase by the progression of the exposed individuals we began to show case signs and symptoms of diarrhea at a rate σ and decreases due to the treatment rate τ, they also reduce as a result of natural death rate μ and induced disease death rate δ (Eq. (4). The recovered individuals grow by the number treated individuals and those who recovered due to their immune system from the infected compartment at a rate τ and θ respectively. The population reduces due to natural death rate μ and recovered individuals returning to the susceptible compartment due to loss of immunity at a rate ϕ (Eq. (5), Eq. (6), Eq. (7). For simplicity, where $A_1 = (\mu + \sigma),\ A_2 = (\tau + \mu + \delta + \theta),\ A_3 = (\mu + \phi)$.

$$\frac{dE}{dt} = (1-z)\beta SI - (\mu + \sigma)E \qquad \text{Eq. (3)}$$

$$\frac{dI}{dt} = \sigma E - (\tau + \mu + \delta + \theta)I \qquad \text{Eq. (4)}$$

$$\frac{dR}{dt} = (\tau + \theta)I - (\mu + \phi)R \qquad \text{Eq. (5)}$$

$$\left.\begin{aligned}\frac{dS}{dt} &= \lambda - (1-z)\beta SI + \phi R - \mu S \\ \frac{dE}{dt} &= (1-z)\beta SI - (\mu+\sigma)E \\ \frac{dI}{dt} &= \sigma E - (\tau+\mu+\delta+\theta)I \\ \frac{dR}{dt} &= (\tau+\theta)I - (\mu+\phi)R\end{aligned}\right\} \quad \text{Eq. (6)}$$

$$\left.\begin{aligned}\frac{dS}{dt} &= \lambda - (1-z)\beta SI + \phi R - \mu S \\ \frac{dE}{dt} &= (1-z)\beta SI - A_1 E \\ \frac{dI}{dt} &= \sigma E - A_2 I \\ \frac{dR}{dt} &= (\tau+\theta)I - A_3 R\end{aligned}\right\} \quad \text{Eq. (7)}$$

Table 1. *Descriptive of variables.*

Variables	Description
S	Susceptible
E	Exposed
I	Infected
R	Recovered

Table 2. *Description of parameters and values.*

Parameter	Description	Value	Source
λ	The recruitment rate	1,000	Assumed
z	Campaign rate	0.2	Varied
β	Effective contact rate	0.0003	Das et al. (2013)
ϕ	Loss of immunity	0.2	Das et al. (2013)
μ	Natural death rate	0.2	Das et al. (2013)
θ	Recovery rate	0.1	Assumed
τ	Treatment rate	0.3	Pitzer et al. (2014)
δ	Induced disease death rate	0.4	Venkatesan and Van de Verg (2015)
σ	Progression rate	0.7	Chavda et al. (2024)
R_0	Basic reproduction number	0.933	Estimated
$S(0)$	Susceptible class	1,000	Estimated
$E(0)$	Exposed class	800	Estimated
$I(0)$	Infected class	500	Estimated
$R(0)$	Recovered class	700	Estimated

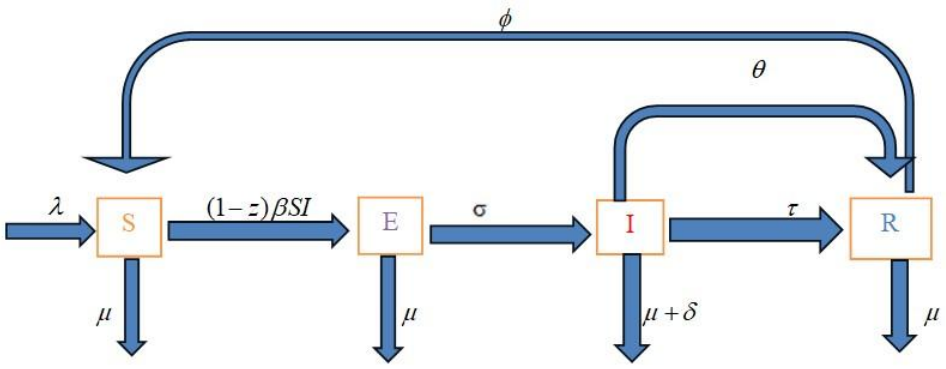

Figure 1. *Flow diagram of SEIR Epidemic Model.*

Analysis of model equations for existence of solution

Theorem 1

Let:

$$x_1 = f_1\left(x_1, x_2, \ldots, x_n, t\right), x_1\left(t_0\right) = x_{10}$$

$$x_2 = f_2\left(x_1, x_2, \ldots, x_n, t\right), x_2\left(t_0\right) = x_{20}$$

$$x_n = f_n\left(x_1, x_2, \ldots, x_n, t\right), x_n\left(t_0\right) = x_{n0}$$

Let D denote the region in $\left(n+1\right)$ dimensional space one dimension for t and n dimension for the vector x

$$D = \left\{(x,t), /t - t_0/ \le a, /x - x_0/ \le b\right\}$$

Then there is a constant $\delta > 0$ such that there exists a unique continuous vector solution.

$$\underline{x} = [x_1(t), x_2(t), \ldots, x_n(t)]$$

In these interval $/t - t_0/ \le \delta$, let:

$$\frac{dS}{dt} = \lambda - \left(1-z\right)\beta SI + \phi R - \mu S \;\; S\left(t_0\right) = S_0$$

$$\frac{dE}{dt} = \left(1-z\right)\beta SI - \left(\mu + \sigma\right)E \;\; E\left(t_0\right) = E_0$$

$$\frac{dI}{dt} = \sigma E - \left(\tau + \mu + \delta + \theta\right)I \;\; I\left(t_0\right) = I_0$$

$$\frac{dR}{dt} = (\tau + \theta) I - (\mu + \phi) R \; R(t_0) = R_0$$

$$D = \{(S, E, I, R,) / S - S_0 / \le a, / E - E_0 / \le b, / I - I_0 / \le c, / R - R_0 / \le d, / t - t_0 / \le e\}$$

Then the equation has a unique solution, proof:

$$\left.\begin{aligned} \left.\frac{df_1}{dS}\right|_{0,0,0,0} &= |(1-z)\beta I - \mu| = (1-z) + \mu < \infty \\ \left.\frac{df_1}{dE}\right|_{0,0,0,0} &= 0 < \infty \\ \left.\frac{df_1}{dI}\right|_{0,0,0,0} &= |-(1-z)| = (1-z)\beta S < \infty \\ \left.\frac{df_1}{dR}\right|_{0,0,0,0} &= |\phi| = \phi < \infty \end{aligned}\right\}$$

$$\left.\begin{aligned} \left.\frac{df_2}{dS}\right|_{0,0,0,0} &= |(1-z)\beta I| = (1-z)\beta I < \infty \\ \left.\frac{df_2}{dE}\right|_{0,0,0,0} &= |-(\mu + \sigma)| = \mu + \sigma < \infty \\ \left.\frac{df_2}{dI}\right|_{0,0,0,0} &= |(1-z)\beta S| = (1-z)\beta S < \infty \\ \left.\frac{df_2}{dR}\right|_{0,0,0,0} &= 0 < \infty \end{aligned}\right\}$$

$$\left.\begin{aligned} \left.\frac{df_3}{dS}\right|_{0,0,0,0} &= 0 < \infty \\ \left.\frac{df_3}{dE}\right|_{0,0,0,0} &= |\sigma| = \sigma < \infty \\ \left.\frac{df_3}{dI}\right|_{0,0,0,0} &= |-(\tau + \mu + \delta + \theta)| = \tau + \mu + \delta + \theta < \infty \\ \left.\frac{df_3}{dR}\right|_{0,0,0,0} &= 0 < \infty \end{aligned}\right\}$$

$$\left.\begin{aligned}
&\left.\frac{df_4}{dS}\right|_{0,0,0,0} = 0 < \infty \\
&\left.\frac{df_4}{dE}\right|_{0,0,0,0} = 0 < \infty \\
&\left.\frac{df_4}{dI}\right|_{0,0,0,0} = |(\tau+\theta)| = \tau+\theta < \infty \\
&\left.\frac{df_4}{dR}\right|_{0,0,0,0} = |-(\mu+\phi)| = \mu+\phi < \infty
\end{aligned}\right\}$$

Therefore, $\left|\frac{df_i}{dx_j}\right| i, j = 1,2$ are continuous and bounded, then the model has a unique solution. Hence, the problem has a unique solution and the model is mathematically and epidemiologically well posed.

Theorem 2

Let the initial data $\mathrm{S}(0)$, $\mathrm{E}(0)$, $\mathrm{I}(0)$ and $\mathrm{R}(0)$ be non-negative, then the solution $(\mathrm{S},\mathrm{E},\mathrm{I},\mathrm{R})$ of the model are non-negative for all $\mathrm{t} > 0$. Proof:

Consider the biological feasibility region $D = \{(S,E,I,R) \in \mathbb{R}^4 + N \leq \frac{\lambda}{\mu}\}$

It will be shown that D is positive invariance (i.e. all solutions remain in D for all time $t > 0$). The rate of change of total population.

$$\frac{dN}{dt} = \lambda - \mu(S+E+I+R)$$

$$\mathrm{N} = \mathrm{S}+\mathrm{E}+\mathrm{I}+\mathrm{R}$$

$$\frac{dN}{dt} = \lambda - \mu N \Rightarrow \frac{dN}{dt} \leq \lambda - \mu N$$

Using integrating factor

$$N(t) \leq e^{-\int \mu dt}[\int \lambda e^{\int \mu dt}\, dt]$$

$$N(t) \leq e^{-\mu t}[\frac{\lambda}{\mu} e^{\mu t} + C]$$

$$N(t) \leq \frac{\lambda}{\mu} + Ce^{-\mu t} \quad \mathrm{t} = 0$$

$$N(0) \leq \frac{\lambda}{\mu} + C$$

$$N(0) - \frac{\lambda}{\mu} \leq C$$

Therefore, $N(t) \leq \frac{\lambda}{\mu} + e^{-\mu t}(N(0) - \frac{\lambda}{\mu})$ Eq. (8)

If $N(0) - \frac{\lambda}{\mu}$, therefore all solutions of the model with initial conditions in D remain in the region for $t > 0$, this implies that D is positively invariant. In this region, the model can be considered as been epidemiologically and mathematically well posed.

Disease free equilibrium

At disease free equilibrium, when there is no infection. That is $E = I = R = 0$ and at equilibrium point, the normalized model is obtained by setting.

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

Hence, the disease-free equilibrium is given by:

$$E_0 = \left(\frac{\lambda}{\mu}, 0, 0, 0 \right)$$ Eq. (9)

The endemic equilibrium points are:

$$\left.\begin{aligned} S^* &= \frac{-A_2 A_1}{\beta\sigma(1-z)} \\ E^* &= \frac{A_3 A_2 (\beta\lambda\sigma z - \beta\lambda\sigma + \mu A_1 A_2) A_2}{\beta\sigma(\phi\sigma\tau z + \phi\sigma\theta z - z A_1 A_2 A_3 - \phi\sigma\tau - \phi\sigma\theta + A_1 A_2 A_3)} \\ I^* &= \frac{A_3 (\beta\lambda\sigma z - \beta\lambda\sigma + \mu A_1 A_2)}{\beta(\phi\sigma\tau z + \phi\sigma\theta z - z A_1 A_2 A_3 - \phi\sigma\tau - \phi\sigma\theta + A_1 A_2 A_3)} \\ R^* &= \frac{(\tau+\theta)(\beta\lambda\sigma z - \beta\lambda\sigma + \mu A_1 A_2)}{\beta(\phi\sigma\tau z + \phi\sigma\theta z - z A_1 A_2 A_3 - \phi\sigma\tau - \phi\sigma\theta + A_1 A_2 A_3)} \end{aligned}\right\}$$ Eq. (10)

The endemic equilibrium points can be written in terms of (R_0) hence;

$$S^* = \frac{-A_2 A_1}{\beta\sigma(1-z)}$$

$$E^* = \frac{A_3 A_2 (-A_1 A_2 \mu (R_0 - 1)) A_2}{\beta \sigma M}$$

$$I^* = \frac{A_3 (-A_1 A_2 \mu (R_0 - 1))}{\beta M}$$

$$R^* = \frac{(\tau + \theta)(-A_1 A_2 \mu (R_0 - 1))}{\beta M}$$

Where;

$$M = (\phi \sigma \tau z + \phi \sigma \theta z - z A_1 A_2 A_3 - \phi \sigma \tau - \phi \sigma \theta + A_1 A_2 A_3)$$

A positive equilibrium in the domain is achieved when the endemic equilibrium point of the diarrhea model Eq. (6) exists, which occurs whenever the basic reproduction number exceeds one.

The basic reproduction number (R_0)

The basic reproduction number is the average number of secondary infections generated from a single infectious source found only in the susceptible class. it is obtained by taking the longest eigenvalue (Adesola et al., 2024b; Ajao et al., 2023).

$$F = \left(\frac{\partial F(E_0)}{\partial x_j} \right) \left(\frac{\partial V(E_0)}{\partial x_J} \right) \qquad \text{Eq. (11)}$$

$$F = \begin{bmatrix} (1-z)\beta SI \\ 0 \end{bmatrix} \; V = \begin{bmatrix} (\mu + \sigma)E \\ -\sigma E + (\tau + \mu + \delta + \theta)I \end{bmatrix}$$

$$F = \begin{bmatrix} 0 & (1-z)\beta S \\ 0 & 0 \end{bmatrix} \; V = \begin{bmatrix} \mu + \sigma & 0 \\ -\sigma & \tau + \mu + \delta + \theta \end{bmatrix}$$

$$R_0 = \rho(FV^{-1}) = \begin{bmatrix} \frac{(1-z)\beta\lambda\sigma}{\mu(\mu+\sigma)(\tau+\mu+\delta+\theta)} - \lambda & \frac{(1-z)\beta\lambda}{\mu(\tau+\mu+\delta+\theta)} \\ 0 & 0 - \lambda \end{bmatrix}$$

$$R_0 = \frac{(1-z)\beta\lambda\sigma}{\mu(\mu+\sigma)(\tau+\mu+\delta+\theta)} < 1 \qquad \text{Eq. (12)}$$

Local stability of Disease-Free Equilibrium (LDFE)

Theorem 3

The Disease-Free equilibrium of model equation is locally asymptotically stable whenever the basic reproduction number R_0 is less than one ($(R_0 < 1)$ and when R_0 is greater than one, i.e., $(R_0 > 1)$, it is locally asymptomatically unstable. Proof:

The Jacobian matrix of the system will be computed at

$$E_0 = (S_0, E_0, I_0, R_0) = \left(\frac{\lambda}{\mu}, 0, 0, 0 \right)$$

$$J = \begin{bmatrix} -\mu & 0 & \frac{-(1-z)\beta\lambda}{\mu} & 0 \\ 0 & -(\mu+\sigma) & \frac{(1-z)\beta\lambda}{\mu} & 0 \\ 0 & \sigma & -(\tau+\mu+\delta+\theta) & 0 \\ 0 & 0 & (\tau+\theta) & -(\mu+\phi) \end{bmatrix} \quad \text{Eq. (13)}$$

Then the characteristics equation is obtained as $|J - \lambda I| = 0$ where λ is the eigenvalues;

$$J = \begin{bmatrix} -\mu-\lambda & 0 & \frac{-(1-z)\beta\lambda}{\mu} & 0 \\ 0 & -(\mu+\sigma)-\lambda & \frac{(1-z)\beta\lambda}{\mu} & 0 \\ 0 & \sigma & -(\tau+\mu+\delta+\theta)-\lambda & 0 \\ 0 & 0 & (\tau+\theta) & -(\mu+\phi)-\lambda \end{bmatrix} \quad \text{Eq. (14)}$$

$\lambda = -\mu$ and $\lambda = -(\mu+\phi)$ and the remaining 2*2 matrix leads to Eq. (15).

$$\lambda^2 + ((\mu+\sigma)(\tau+\mu+\delta+\theta))\lambda - \frac{(1-z)\beta\lambda\sigma}{\mu} + (\mu+\sigma)(\tau+\mu+\delta+\theta) \quad \text{Eq. (15)}$$

$$\frac{-(1-z)\beta\lambda\sigma}{\mu} > -(\mu+\sigma)(\tau+\mu+\delta+\theta) - \lambda^2 - ((\mu+\sigma)(\tau+\mu+\delta+\theta))\lambda$$

$$\frac{-(1-z)\beta\lambda\sigma}{\mu} < 1 \quad \text{Eq. (16)}$$

Implying that $R_0 < 1$ This is locally asymptomatically stable.

Global Stability of Disease-Free Equilibtium (GSDFE)

Theorem 4

The disease-free equilibrium (DFE) is globally asymptotically stable if all trajectories of the system converge to the (DFE) as time approaches infinity, provided the basic reproduction number R_0 is less than one $(R_0 < 1)$ and when R_0 is greater than one $(R_0 > 1)$ it is Globally asymptomatically unstable. Proof:

Using Lyapunov function for Eq. (6):

$$R_0 = \frac{(1-z)\beta\lambda\sigma}{\mu(\mu+\sigma)(\tau+\mu+\delta+\theta)}$$

$$\frac{R_0}{1} = \frac{(1-z)\beta\lambda\sigma}{\mu(\mu+\sigma)(\tau+\mu+\delta+\theta)}$$

$$(1-z)\beta\sigma = \frac{R_0\mu(\mu+\sigma)(\tau+\mu+\delta+\theta)}{\lambda}$$

Using infected compartment for DFE, Let L denote the Lyapunov function which is given by:

$$L = AE + BI \qquad \text{Eq. (17)}$$

$$L' = AE' + BI' \qquad \text{Eq. (18)}$$

$$L' = A[(1-z)\beta SI - (\mu+\sigma)E] + B[\sigma E - (\tau+\mu+\delta+\theta)I]$$

$$L' = A(1-z)\beta SI - A(\mu+\sigma)E + B\sigma E - B(\tau+\mu+\delta+\theta)I$$

$$E' = -A(\mu+\sigma) + \beta\sigma$$

$$I' = A(1-z)\beta S - B(\tau+\mu+\delta+\theta)$$

$$A = \tau+\mu+\delta+\theta$$

$$B = (1-z)\beta S$$

Putting A and B into Eq. (18):

$$L' = (\tau+\mu+\delta+\theta)[(1-z)\beta SI - (\mu+\sigma)E] + (1-z)\beta S[\sigma E - (\tau+\mu+\delta+\theta)I]$$

$$L' = \left[\frac{\lambda(1-z)\beta\sigma}{\mu} - (\tau+\mu+\delta+\theta)(\mu+\sigma)\right]E$$

$$L' = \left[\frac{\lambda R_0\mu(\mu+\sigma)(\tau+\mu+\delta+\theta)}{\lambda\mu} - (\tau+\mu+\delta+\theta)(\mu+\sigma)\right]E$$

$$L' = [R_0 A - A]E$$

$$L' = (R_0 - 1)AE \qquad \text{Eq. (19)}$$

$$L' < 0$$

Implying that $R_0 < 0$

Local stability of endemic equilibrium

Theorem 5

If $R_0 > 1$ the endemic equilibrium E^* of the system is locally asymptotically stable. Proof: Linearizing the Jacobian matrix of system of E^*;

$$J(E^*) = \begin{bmatrix} -(1-z)\beta I^* - \mu & 0 & -(1-z)\beta S^* & 0 \\ (1-z)\beta I^* & -A_1 & (1-z)\beta S^* & 0 \\ 0 & \sigma & -A_2 & 0 \\ 0 & 0 & (\tau+\theta) & -A_3 \end{bmatrix} \qquad \text{Eq. (20)}$$

$$J(E^*) = \begin{bmatrix} (-(1-z)\beta I^* - \mu) - \lambda & 0 & -(1-z)\beta S^* & 0 \\ (1-z)\beta I^* & -A_1 - \lambda & (1-z)\beta S^* & 0 \\ 0 & \sigma & -A_2 - \lambda & 0 \\ 0 & 0 & (\tau+\theta) & -A_3 - \lambda \end{bmatrix}$$

$$\lambda^4 + (A_3 + A_2 + A_1 + Q + mu)\lambda^3 + (QA_1 + QA_2 + QA_3 - X\sigma + \mu A_1 + \mu^* A_2 + \mu A_3 + A_1A_2 + A_1A_3 + A_2A_3)\lambda^2$$
$$+(QA_1A_2 + QA_1A_3 + QA_2A_3 - X\mu\sigma - X\sigma A_3 + \mu A_1A_2 + \mu A_1A_3 + \mu A_2A_3 + A_1A_2A_3)\lambda$$
$$-Q\phi\sigma\tau - Q\phi\sigma\theta + QA_1A_2A_3 - X\mu\sigma A_3 + \mu A_1A_2A_3$$

$$C_0 = 1$$

$$C_1 = (A_3 + A_2 + A_1 + Q + \mu)$$

$$C_2 = Q(A_1 + A_2 + A_3) + \mu(A_1 + A_2 + A_3) - \sigma X + A_1(A_2 + A_3) + A_3A_2$$

$$C_3 = Q(A_2A_1 + A_1A_3 + A_2A_3) - \sigma X(\mu + A_3) + \mu(A_2A_1 + A_1A_3 + A_2A_3) + A_1A_2A_3$$

$$C_4 = \sigma Q\phi(\tau + \theta) + QA_1A_2A_3 - \mu(X\sigma A_3 + A_1A_2A_3)$$

Where $Q = (1-z)\beta I^*$ and $X = (1-z)\beta S^*$

According to Hurwitz Criterion, when $R_0 > 1$ the endemic equilibrium $E*$ of system is locally asymptotically stable if $C_2C_1 - C_3C_0 > 0$ and $C_3C_2C_1 - C_4C_1^2 - C_3^2 C_0 > 0$

Global stability of endemic equilibrium

Theorem 6

Let $En*$ be the unique positive equilibrium point of the system (6), if $R_0 > 1$ then the endemic equilibrium $En*$ of the system (6) is globally asymptotically stable. Proof: using the Lyapunov function;

$$L(S*E*I*R) = \left\{\left(S - S* - S*\ln\left(\frac{S*}{S}\right)\right) + \left(E - E* - E*\ln\left(\frac{E*}{E}\right)\right) + \left(I - I* - I*\ln\left(\frac{I*}{I}\right)\right) + \left(R - R* - R*\ln\left(\frac{R*}{R}\right)\right)\right.$$

The derivative of L along the solution of the system is direct;

$$\frac{dL}{dt} = \left(\frac{S - S*}{S}\right)\frac{dS}{dt} + \left(\frac{E - E*}{E}\right)\frac{dE}{dt} + \left(\frac{I - I*}{I}\right)\frac{dI}{dt} + \left(\frac{R - R*}{R}\right)\frac{dR}{dt} \qquad \text{Eq. (21)}$$

$$\frac{dL}{dt} = \left\{\begin{array}{l} \left(\frac{S - S*}{S}\right)[\lambda - (1-z)\beta SI - \mu S + \phi R] \\ + \left(\frac{E - E*}{E}\right)[(1-z)\beta SI - (\mu + \sigma)E] \\ + \left(\frac{I - I*}{I}\right)[\sigma E - (\tau + \mu + \delta + \theta)I] + \left(\frac{R - R*}{R}\right)[(\tau + \theta)I - (\mu + \phi)R] \end{array}\right.$$

By expansion:

$$\frac{dL}{dt} = \left\{\begin{array}{l} \lambda - (1-z)\beta SI - \mu S + \phi R - \frac{\lambda S*}{S} + (1-z)\beta IS* + \mu S* - \frac{\phi RS*}{S} \\ + (1-z)\beta SI - \mu E - \sigma E - \frac{(1-z)\beta SIE*}{E} + (\mu + \sigma)E* \\ + \sigma E - \tau I - \mu I - \delta I - \theta I - \frac{\sigma EI*}{I} + (\tau + \mu + \delta + \theta)I* \\ + \tau I + \theta I - \mu R - \phi R - \frac{(\tau + \theta)IR*}{R} + (\mu + \phi)R* \end{array}\right.$$

By simplification;

$$\frac{dL}{dt}=\begin{cases}\lambda-\mu S-\dfrac{\lambda S^*}{S}+(1-z)\beta IS^*+\mu S^*-\dfrac{\phi RS^*}{S}\\ -\mu E-\dfrac{(1-z)\beta SIE^*}{E}+(\mu+\sigma)E^*\\ -(\mu+\delta)I-\dfrac{\sigma EI^*}{I}+(\tau+\mu+\delta+\theta)I^*\\ -\mu R-\dfrac{(\tau+\theta)IR^*}{R}+(\mu+\phi)R^*\end{cases}$$

Suppose, $\frac{dL}{dt}=P-M$ Eq. (22)

Where, *p* denotes the positive terms and *M* denotes the negative terms so that:

$$P=\lambda+(1-z)\beta IS^*+\mu S^*+(\mu+\sigma)E^*+(\tau+\mu+\delta+\theta)I^*+(\mu+\phi)R^*$$

The largest invariant set is $\left\{\left(S^*,E^*,I^*,<R^*\right)\in\theta:\frac{dL}{dt}=0\right\}$ a unit set of En^*

Where En^* is the endemic equilibrium signifying that the endemic is globally asymptotically stable.

Sensitivity analysis

To evaluate the sensitivity of the basic reproduction number R_0 with respect to each of the following parameters $\lambda,\beta,z,\phi,\mu,\theta,\tau,\sigma$ and δ by calculating each value applying the derivative-based method, which reflects the bond between every parameter and the basic reproduction number R_0. Inserting the value of each parameter into equation and solving them using;

$$X_x^{R_0}=\frac{\partial R_0}{\partial x}\cdot\frac{x}{R_0}$$ Eq. (23)

The parameter sensitivity index uses the derivative-based local method as described in *Table 3* which displays that the parameter λ, β and σ have direct relationship with the basic reproduction number R_0 and it can increase the rate at which diarrhea progresses in the population (*Figure 2*). While, z, μ, θ, τ and δ have inverse relationship with R_0, here this parameters decreases the rate at which diarrhea spreads. However, reducing effective contact rate between infected human and susceptible individuals as well as educating them on the importance of personal hygiene and keeping their environment clean and also restricting infected individual direct access to public food and water this could significantly reduce the R_0. The sensitivity analysis

findings indicate that campaign and treatment can completely lower the basic reproduction number; they effectively help in disease control.

Table 3. *Parameter sensitivity index.*

Parameter	Sensitivity expression	Sensitivity value	Sensitivity index
λ	1	1	+
z	$\frac{z}{(-1+z)}$	-0.3	-
β	1	1	+
μ	$-\mu^5-2\mu^4\sigma-\mu^3\sigma^2+\frac{2(-1+z)\beta\lambda\sigma\mu+\beta\sigma^2\lambda(-1+z)}{\mu^2A_1^2A_2}$	-5.9	-
θ	$\frac{-\theta}{A_2}$	-0.1	-
τ	$\frac{-\tau}{A_2}$	-0.3	-
δ	$\frac{-\delta}{A_2}$	-0.4	-
σ	$\frac{-\sigma\mu^2-\sigma\mu(\tau+\delta+\theta)-(-1+z)\beta\lambda}{\mu A_1A_2}$	0.5	+

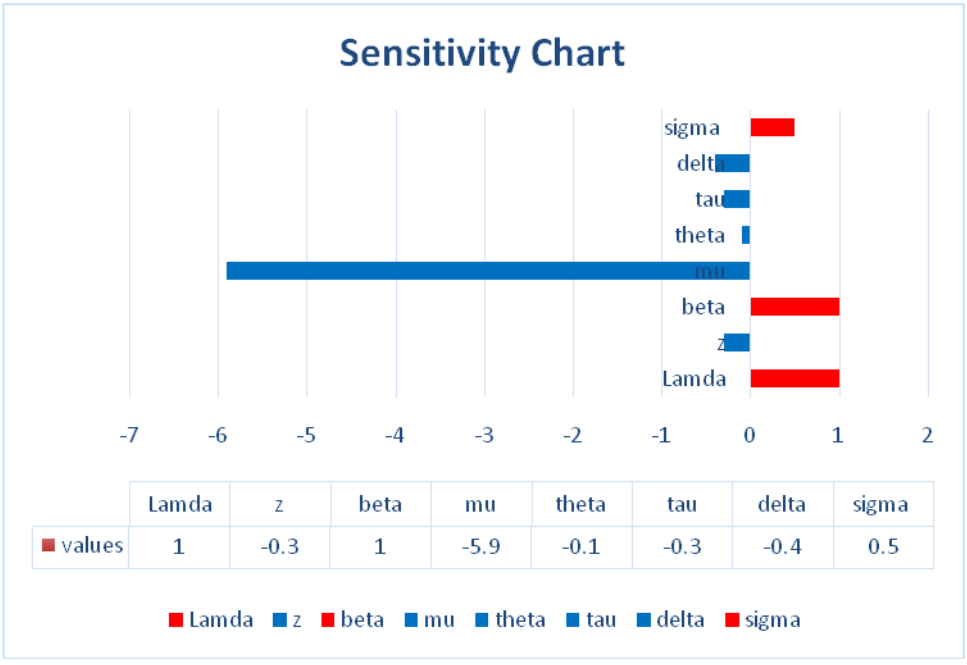

Figure 2. *The sensitivity analysis graph.*

Numerical simulations

To evaluate the theoretical calculation of the model, the numerical simulation of the model (6) is carried out by differential transformation method using a set of parameter values given in *Table 2*.

Results and Discussion

An epidemic model of four compartmental models (S, E, I, R) was considered to gain more insight into the effect of campaign and treatment of infected individuals on the progression rate of diarrhea in a population. The disease free and endemic equilibrium were obtained and the basic reproduction number R_0 was computed. The result of the quantitative analyses showed that the disease-free equilibrium is both locally and globally asymptotically stable $R_0 < 1$ and unstable otherwise. On the other hand, the endemic equilibrium is stable whenever $R_0 > 1$. This means that: Diarrhea can be controlled via campaign and treatment if the basic reproduction number is below unity, irrespective of the initial number of infections in the population. However, if the basic reproduction goes beyond unity, then diarrhea will persist in the population. The result of the sensitivity analysis shows that the recruitment rate λ, effective contact rate β and progression rate σ are the sensitive parameter of the basic reproduction number with positive index, this means that λ , β and σ have great impact in the basic reproduction number and also on the prevalence of the disease in the population.

The result of the numerical simulation is shown graphically in figures form. *Figure 3*, *Figure 4*, *Figure 5* and *Figure 6* demonstrate a clear trend that as individuals become increasingly aware of the benefits of maintaining personal hygiene, practicing proper environmental sanitation, consuming healthy food, and accessing clean water, the number of exposed and infected individuals significantly declines. In tandem, the number of susceptible individuals grows, signifying a lack of new infection cases. This positive result is well pronounced in *Figure 6* by showing that when a community fully adopts and complies with public health campaigns, the susceptible population increases, and no new cases of infection emerge. This finding illustrates the profound impact of collective action and adherence to health-promoting behaviors on disease prevention. In *Figure 7* demonstrates the effect of public health campaigns on the susceptible population. When the campaign reaches full effectiveness (z=1), all susceptible individuals are fully informed about the risks and preventive measures for diarrhea disease, including hygiene, clean water, and sanitation practices. This increased awareness decreases the rate at which susceptible individuals become infected, significantly reducing the likelihood of new cases. In *Figure 8* shows public health campaigns are vital in preventing diarrhea among susceptible individuals by increasing awareness, promoting hygiene and sanitation, and encouraging early intervention, thereby preventing progression to the exposed class. These efforts help curb infections, enhance community health, and empower individuals to adopt preventive measures, leading to a reduced incidence of diarrhea and long-term health benefits.

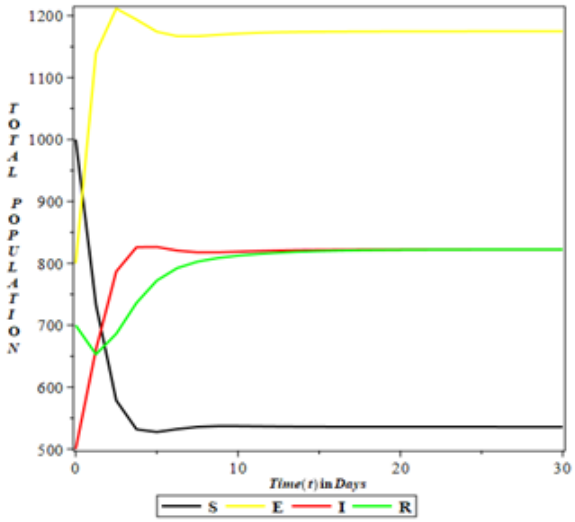

Figure 3. *Total population of SEIR with z=0.2.*

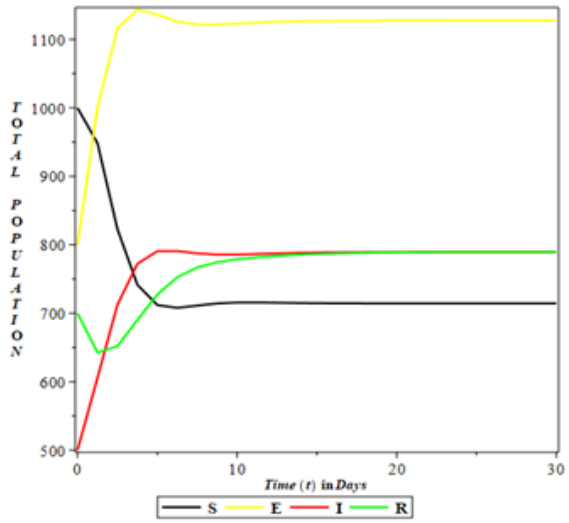

Figure 4. *Total population of SEIR with z=0.4.*

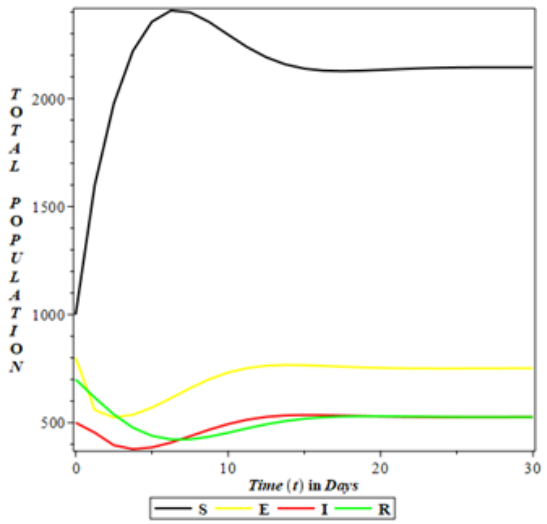

Figure 5. *Total population of SEIR with z=0.8.*

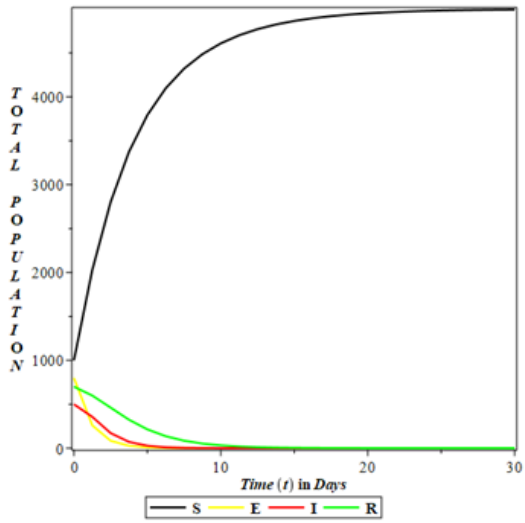

Figure 6. *Total population of SEIR with z=1.*

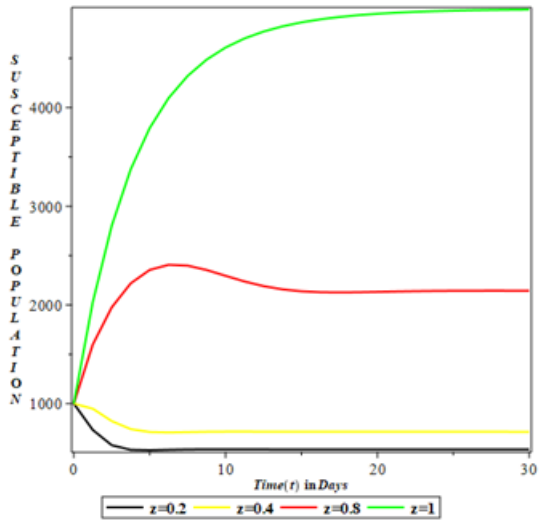

Figure 7. *Total population of susceptible individuals with z=0.2; 0.4; 0.8 and 1.*

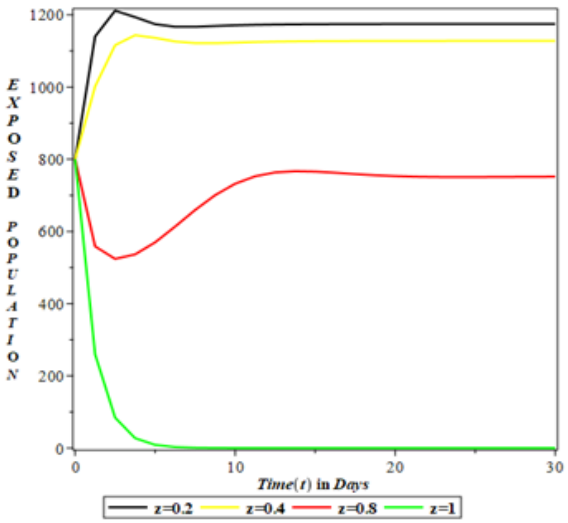

Figure 8. *Total population of infected with z=0.2; 0.4; 0.8 and 1.*

Figure 9 illustrates the vital role of effective treatment for infected individuals. It shows that timely and appropriate medical care increases the likelihood of recovery, allowing these individuals to return to the susceptible class. This highlights the importance of access to quality treatment in reducing disease spread and promoting overall community health. In *Figure 10* illustrates the natural recovery process from diarrhea, demonstrating how the body gradually heals on its own, without the need for medical intervention. The graph emphasizes the body's innate ability to combat infection through the immune system, showcasing the gradual decline in infection rates over time. In *Figure 11* highlights a critical scenario where there are no public health campaigns, no functional immune system responses, and no available treatments, illustrating the severe impact of these absences on disease dynamics, particularly in the case of diarrhea. The graph presents a grim picture of what occurs when these essential elements are missing, showing how quickly the situation deteriorates, increased rate of infection and exposure, decline in susceptible individuals and minimal or no recovery. In *Figure 12* demonstrates the important role that public health campaigns, immune system effectiveness, and medical treatment play in the control and eventual eradication of diarrhea. The figure illustrates how these three key factors, when functioning together, create a comprehensive strategy for reducing infections and preventing the spread of the disease.

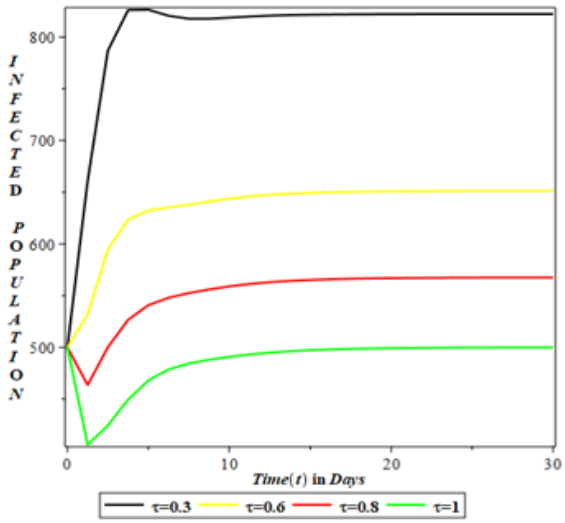

Figure 9. *Total population of infected individuals with τ=0.3; 0.6; 0.8 and 1.*

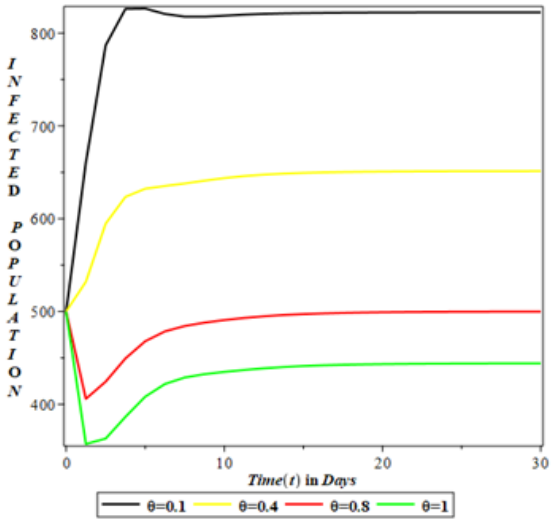

Figure 10. *Total population of infected individuals with θ=0.1; 0.4; 0.8 and 1.*

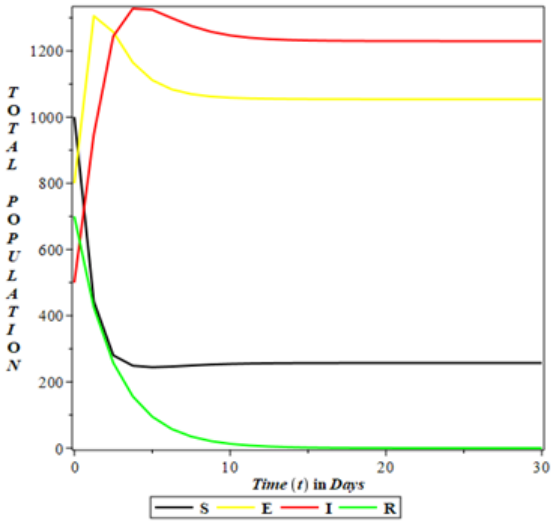

Figure 11. *Total population of SEIR at a point where z=0; τ=0; θ=0.*

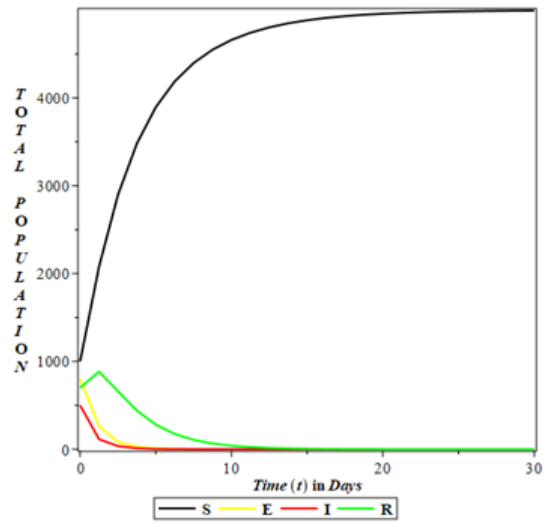

Figure 12. *Total population of SEIR at a point where z=1; τ=1; θ=1.*

Conclusion

In conclusion, effectively combating diarrhea requires concerted efforts and sacrifices from policymakers, healthcare practitioners, and the general population. To successfully reduce the spread of the disease, it is essential to bring the basic reproduction number R_0 below unity, which signifies that each infected individual transmits the disease to fewer than one other person on average. Achieving this goal involves several critical measures, including lowering the effective contact rate through improved hygiene practices and public health interventions. Furthermore, expanding awareness campaigns, boosting immunity through clean and rich food, and providing timely and effective treatment are vital strategies, as highlighted by the sensitivity analysis conducted in this research. Furthermore, the outcomes of the numerical simulations reinforce the importance of these combined efforts in controlling and eventually eradicating the disease. A sustained and unified approach can significantly reduce the incidence of diarrhea, improving public health outcomes and saving lives.

Acknowledgement

This research is self-funded.

Conflict of interest

The authors confirm that there is no conflict of interest involve with any parties in this research study.

REFERENCES

[1] Adesola, O.I., Oloruntoyin, S.S., Emmanuel, P.M., Temilade, M.I., Adeyemi, A.G., Oladele, A.S., Mamman, A.U., Kareem, A.A. (2024a): Mathematical modelling and analyzing the dynamics of condom efficacy and compliance in the spread of HIV/AIDS. – Asian Research Journal of Current Science 11p.

[2] Adesola, O.I., Temilade, M.I., Emmanuel, P.M., Oladele, A.S., Adeyemi, A.G., Sunday, S., Olumuyiwa, A.S. (2024b): Mathematical Analysis of Optimal Control of Human Immunodeficiency Virus (HIV) Co-infection with Tuberculosis (TB). – Asian Research Journal of Current Science 30p.

[3] Ajao, S., Olopade, I., Akinwumi, T., Adewale, S., Adesanya, A. (2023): Understanding the transmission dynamics and control of HIV infection: A mathematical model approach. – Journal of the Nigerian Society of Physical Sciences 1p.

[4] Akinwumi, T.O., Olopade, I.A., Adesanya, A.O., Alabi, M.O. (2021): A mathematical model for the transmission of HIV/AIDS with early treatment. – Journal of Advances in Mathematics and Computer Science 36(5): 35-51.

[5] Canga, A., Bidegain, G. (2022): Modelling the Effect of the Interaction between Vaccination and Nonpharmaceutical Measures on COVID-19 Incidence. – Global Health, Epidemiology and Genomics 15p.

[6] Chavda, V.P., Vuppu, S., Mishra, T., Kamaraj, S., Sharma, N., Punetha, S., Sairam, A., Vaghela, D., Dargahi, N., Apostolopoulos, V. (2024): Combatting infectious diarrhea: innovations in treatment and vaccination strategies. – Expert Review of Vaccines 23(1): 246-265.

[7] Das, J.K., Tripathi, A., Ali, A., Hassan, A., Dojosoeandy, C., Bhutta, Z.A. (2013): Vaccines for the prevention of diarrhea due to cholera, shigella, ETEC and rotavirus. – BMC Public Health 13: 1-11.
[8] De Bruyn, G., Hahn, S., Borwick, A., Cochrane Infectious Diseases Group (1996): Antibiotic treatment for travellers' diarrhoea. – Cochrane Database of Systematic Reviews 2019(5): 29p.
[9] Dembek, Z.F., Chekol, T., Wu, A. (2018): Best practice assessment of disease modelling for infectious disease outbreaks. – Epidemiology & Infection 146(10): 1207-1215.
[10] Heesterbeek, H., Anderson, R.M., Andreasen, V., Bansal, S., De Angelis, D., Dye, C., Eames, K.T., Edmunds, W.J., Frost, S.D., Funk, S., Hollingsworth, T.D. (2015): Modeling infectious disease dynamics in the complex landscape of global health. – Science 347(6227): 24p.
[11] Hoyle, E., Davies, H., Bourhill, J., Roberts, N., Lee, J.J., Albury, C. (2023): Effectiveness of hand-hygiene interventions in reducing illness-related absence in educational settings in high income countries: systematic review and behavioural analysis. – Journal of Public Health 12p.
[12] Ki, M.R. (2006): Modeling and Simulations for Infectious Diseases. – Korean Journal of Epidemiology 4p.
[13] Musibau, O., Muritala, A.A., Isaac, A.O., Adelani, O.A., Akeem, O.Y. (2022): Mathematical analysis of sensitive parameters due to dynamic transmission of Ebola virus disease. – Comprehensive Research and Reviews in Multidisciplinary Studies 1(01): 001-016.
[14] Olopade, A.I., Adesanya, A.O., Akinwumi, T.O. (2021a): Mathematical transmission of SEIR epidemic model with natural immunity. – Asian Journal of Pure and Applied Mathematics 10p.
[15] Olopade, I.A., Adewale, S.O., Muhammed, I.T., Adeniran, G.A., Ajao, S., Ogunsola, A.W. (2021b): Effect of effective contact tracing in curtaining the spread of Covid-19. – Asian Journal of Research in Biosciences 3(2): 118-134.
[16] Philemon, M.E., Olopade, I.A., Ogbaji, E.O. (2023): Mathematical Analysis of the Effect of Quarantine on the Dynamical Transmission of Monkey Pox. – Asian Journal of Pure and Applied Mathematics 19p.
[17] Pitzer, V.E., Bowles, C.C., Baker, S., Kang, G., Balaji, V., Farrar, J.J., Grenfell, B.T. (2014): Predicting the impact of vaccination on the transmission dynamics of typhoid in South Asia: a mathematical modeling study. – PLoS Neglected Tropical Diseases 8(1): 12p.
[18] Rasmussen, A.L. (2020): Vaccination is the only acceptable path to herd immunity. – Med 1(1): 21-23.
[19] Siettos, C.I., Russo, L. (2013): Mathematical modeling of infectious disease dynamics. – Virulence 4(4): 295-306.
[20] Tao, Y., Hite, J.L., Lafferty, K.D., Earn, D.J., Bharti, N. (2021): Transient disease dynamics across ecological scales. – Theoretical Ecology 14(4): 625-640.
[21] Venkatesan, M.M., Van de Verg, L.L. (2015): Combination vaccines against diarrheal diseases. – Human Vaccines & Immunotherapeutics 11(6): 1434-1448.
[22] Victora, C.G., Bahl, R., Barros, A.J., França, G.V., Horton, S., Krasevec, J., Murch, S., Sankar, M.J., Walker, N., Rollins, N.C. (2016): Breastfeeding in the 21st century: epidemiology, mechanisms, and lifelong effect. – The Lancet 387(10017): 475-490.
[23] World Health Organization (WHO) (2020): Diarrhoeal disease. – WHO 4p.
[24] World Health Organization (WHO) (1995): The treatment of diarrhoe. – WHO 50p.