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MODELING SOCIAL FACTOR AND FAULTY HEALTH SYSTEM ON THE DYNAMIC OF CHILDHOOD DIARRHEA IN MAJENGO NYERI COUNTY KENYA.

Abstract: Mathematical modeling of infectious diseases offers insights into the core processes of disease propagation and transmission. The main way that diarrhea, an illness symptom caused by parasite, viral, or bacterial pathogens, spreads is through fecal matter-contaminated water. Stress, whether experienced in childhood or adulthood, can significantly influence the development of bowel disease. Wellness facility-based surveillance studies may understate the disease burden when it is impossible to count the percentage of cases that do not seek care, as in resource-poor environments where access to care is limited or in communities where frequently visited healthcare providers are not included in the surveillance system. This study developed a mathematical model of social factor and faulty health system on the dynamic of childhood diarrhea in Kenya. The model was developed from a system described by first-order equations nonlinear ordinary differential equations in which the disease dies out and the disease-free equilibrium was attained when the basic reproduction number $R_0 < 1$, The basic reproduction number was shown to be $R_0 = 0.008278$ that proved with proper care on under five children during diarrhea outbreak the disease can be contained. Whereas the disease could persists and the endemic equilibrium is reached when $R_0 > 1$. Simulation studies using the model parameters was calculated to show how social factor (stress) and faulty health system propagate childhood diarrhea as seen in figure 2 and 5. The results of the study will provide valuable information to stakeholders, informed laboratory technicians and field experts by demonstrating the effect of stress and faulty health system that will aid in development of new intervention strategies.

Key Words: Childhood diarrhea, Social factor, faulty health system,

1. Introduction

Diarrhea is a condition caused by infections from parasites, viruses, or bacteria, with the main mode of transmission being water contaminated by fecal matter [1]. The fecal-oral pathway is the primary means of transmission for diarrheal infections [2]. Even though being prevalent in all economic contexts, diarrheal illness has the greatest potential to have serious repercussions when resources and medical attention are scarce or when co-morbidity is present. While chronic gastrointestinal infections are now believed to be associated with environmental enteric disorder (EED), which causes lower immunity, stunted growth, and a chronically damaged gut, acute episodes of sickness more swiftly result in fatal dehydration [3].

In addition to limiting access to healthcare, low socioeconomic level have been shown to have an impact on housing conditions, food and other elements that raise the risk of contracting infectious diseases or weaken resistance to them. Children from lower-income families are less likely to receive oral re-hydration therapy a treatment that involves giving fluids by mouth to prevent or manage dehydration compared to those from higher-income families [4].

Mathematical models are essential in shaping public health strategies for disease management and prevention, as they convert real-world cases into epidemiological representations and help predict the spread of infectious diseases [5]. These models are essential frameworks for comprehending the transmission of diseases, forecasting possible outbreaks, and evaluating the results of different therapies [6]. However, few studies have employed mathematical models to analyze the dynamics of diarrhea among children in Kenya and to propose potential preventive measures. Therefore, this study suggested creating a new deterministic model to examine how societal factors and a flawed healthcare system affect the trends in childhood diarrhea in Kenya. The findings of this study will be vital for achieving Sustainable Development Goal 3, target 3.2, which seeks to reduce under-five mortality to 25 or fewer deaths per 1,000 live births by 2030 [7].

2. Model formulation

The model was adapted from the classical SIS framework and explains how social factors and a flawed healthcare system influence the dynamics of childhood diarrhea in Nyeri County, Kenya. T_C: Diarrhea treatment of under five children in faulty health system; T_K: Diarrhea treatment of under five children in normal health system; (I_S):Stress-related diarrhea infections in children under five; (I_N):Incidence of diarrhea among unstressed children under five; and (S): susceptible children. The model's primary characteristic is that mass-mixing of people within a population yields the force of infection λ. The fundamental reproduction number for a compartmental model of infectious disease propagation was obtained using the next-generation matrix. It was utilized in population dynamics to calculate the fundamental reproduction number for organized population models.

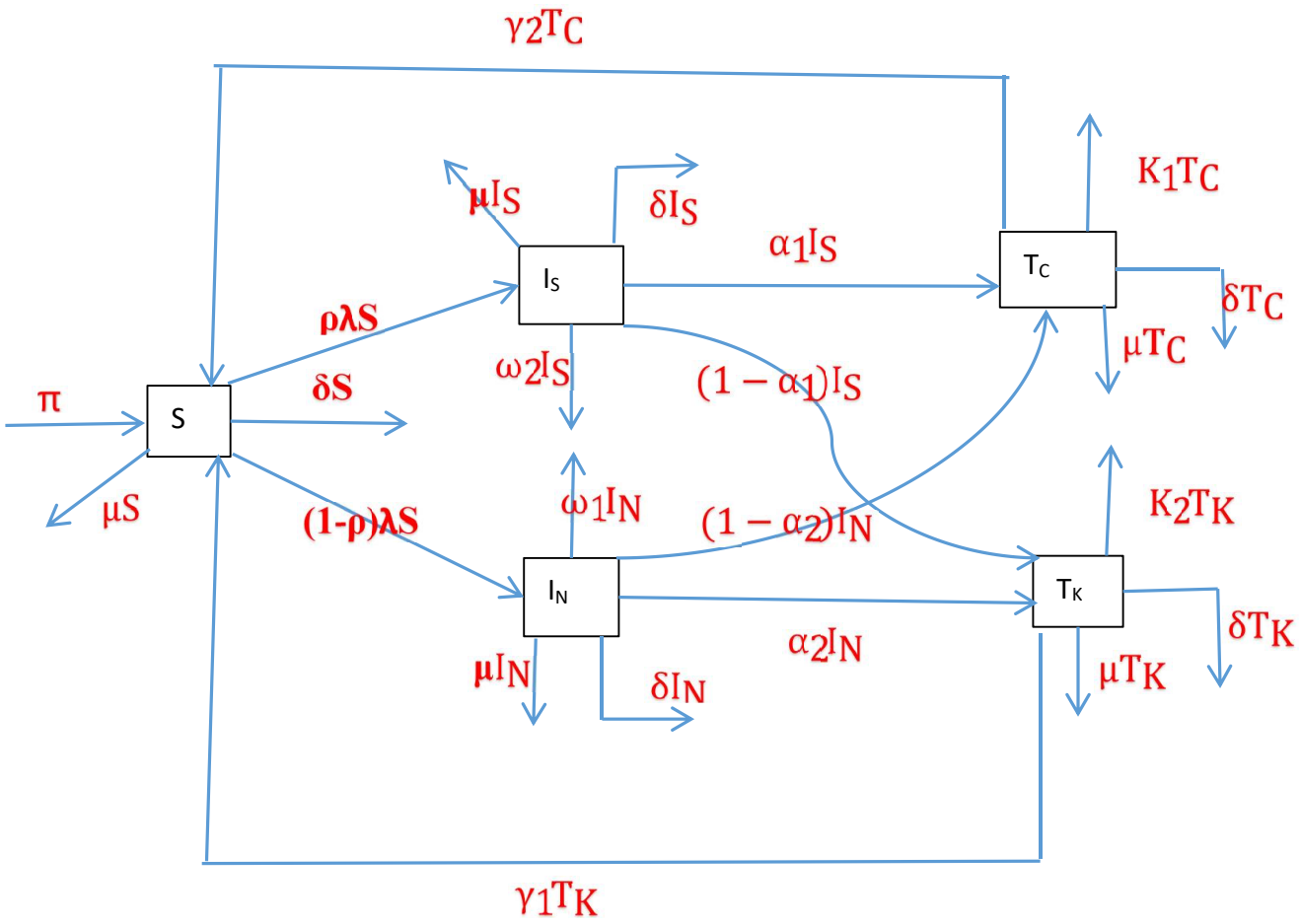
Variables	description
S(t)	The population's number of susceptible under five children at time t
$I_S(t)$	Stress-associated diarrhea in children under five
$I_N(t)$	Under-five children suffering from diarrhea without stress
$T_C(t)$	Diarrhea treatment of under five children in faulty health system
$T_K(t)$	Diarrhea treatment of under five children in normal health system

Table 1. *Model variables*

Parameters	Description
π	Recruiting rate for the susceptible group (S)
μ	Net natural death rate for under five children
γ_1	Recovery rate for treatment under faulty health system
γ_2	Recovery rate for for treatment under normal health system
δ	The rate at which children less than five years old depart the five-year-old age range
ω_1	Diarrhea-related mortality rate among sick children without stress.
ω_2	Death rate from diarrhea in children under stress.
α_1	The fraction of infected with stress children receive treatment.
α_2	Rate at which infected without stress children receive treatment.
$(1-\alpha_1)$	The fraction of infected with stress children receiving treatment.
$(1-\alpha_2)$	Rate at which infected with stress children receive treatment.
k_1	Death rate from diarrhea in the class with a flawed health system
k_2	Diarrhea-related mortality rate in the typical health system class
ρ	The percentage of vulnerable children enrolled in stress-infected classes
$(1-\rho)$	The percentage of vulnerable children that enroll in the stress-free, infected class
λ	Force of infection/mass action
β	Rate of infection
N	Total population

Table 2. *Model parameters*

FIG 1. **Model Flow Chart**



3.1. Model Equations

$$\frac{dS}{dt} = \pi + \gamma_1 T_K + \gamma_2 T_C - \mu S - \delta S - \rho\lambda S - (1-\rho)\lambda S, \tag{1}$$
$$\frac{dI_S}{dt} = \rho\lambda S - \mu I_S - \rho I_S - \omega_2 I_S - \alpha_1 I_S - (1-\alpha_1) I_S, \tag{2}$$
$$\frac{dI_N}{dt} = (1-\rho)\lambda S - \mu I_N - \omega_1 I_N - \delta I_N - \alpha_2 I_N - (1-\alpha_2) I_N, \tag{3}$$
$$\frac{dT_C}{dt} = \alpha_1 I_S - \gamma_2 T_C - K_1 T_C - \mu T_C - \delta T_C + (1-\alpha_2) I_N, \tag{4}$$
$$\frac{dT_K}{dt} = \alpha_2 I_N + (1-\alpha_1) I_S - K_2 T_K - \mu T_K - \delta T_K - \gamma_1 T_K. \tag{5}$$

Where the force of infection is given by;

$$\lambda = \frac{\beta(I_S + \eta_1 I_N + \eta_2 T_C + \eta_3 T_K)}{N} \tag{6}$$

with η_1 being proportion for childhood diarrhea infected without stress and contribute more to the force of infection and η_2 being proportion for children at treatment at faulty health system and contribute less to the force of infection and η_3 being proportion for children at treatment in normal health system and contribute more less to the force of infection as treatment significantly lowers transmission and mortality that is $\eta_1 \geq \eta_2 \geq \eta_3$.

4. Model analysis

4.1. Existence and positive in variance

Theorem 1: Solutions of the model equations (1-5) together with the initial conditions $S(0) \geq 0$, $I_S \geq 0$, $I_N \geq 0$, $T_K \geq 0$ are always positive or the model variables S,I_S,I_N,T_C,T_K all positive for all t and will remain in $\mathbb{R}_5^+$

proof: For the sake of analysis

$$\frac{dS}{dt} = \pi + \gamma_1 T_K + \gamma_2 T_C - \Omega_1 S \tag{7}$$
$$\frac{dI_S}{dt} = \rho\lambda S - \Omega_2 I_S \tag{8}$$
$$\frac{dI_N}{dt} = (1-\rho)\lambda S - \Omega_3 I_N \tag{9}$$
$$\frac{dT_C}{dt} = \alpha_1 I_S + (1-\alpha_2) I_N - \Omega_4 T_C \tag{10}$$
$$\frac{dT_K}{dt} = \alpha_2 I_N + (1-\alpha_1) I_S - \Omega_5 T_K \tag{11}$$

where
$-\mu - \delta - \lambda = \Omega_1, -\mu - \rho - \omega_2 - 1 = \Omega_2, -\mu - \omega_1 - \delta - 1 = \Omega_3, -\gamma_2 - k_1 - \mu - \delta + 1 - \alpha_2 = \Omega_4, -k_2 - \mu - \delta - \gamma_1 = \Omega_5$, to reduce the number of parameters, therefore; For t¿0, let W=((s(t),$i_s(t)$,$i_n(t)$,$t_c(t)$,$t_k(t)$)^T and F(W)=$(F1(W), F2(W), F3(W), F4(W), F5(W))^T$, where F1(W)=$\pi + \gamma_1 T_K + \gamma_2 T_C - \Omega_1 S$, F2(W)=$\rho\lambda S - \Omega I_S$, F3(W)= $(1-\rho)\lambda S - \Omega_3 I_N$, F4(W)= $\alpha_1 I_S - \Omega_4 T_C$, F5(W)= $\alpha_2 I_N + (1-\alpha_2) I_S - \Omega_5 T_K$. Then, system (1-5) can be written as $\frac{dQ}{dt} = F(W)$ where F:$C_+ \longrightarrow (\mathbb{R}_+^5$ with W(0)= $W_0 \in (R)_+^5$. Thus, the function W is locally Lipschitzian and completely stable on $\mathbb{R}_+^5$. Therefore, the solution of the system with non-negative initial conditions exists and is unique. It is also evident that these solutions exist for all $t > 0$ and are non-negative, hence the region $\mathbb{R}_+^5$ is an invariant domain of the system [8].

4.2. Boundedness of the system

Theorem 2: The positive solutions of the system of model equations (1-5) are bounded. That is, the model variables S,I_S, I_N, T_C and T_K are bound for all t [9]. *Proof*:

$$N(t) = S(t) + I_S(t) + I_N(t) + T_C(t) + T_K(t) \tag{12}$$

By differentiating equation (12) gives,

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI_S}{dt} + \frac{dI_N}{dt} + \frac{dT_C}{dt} + \frac{dT_K}{dt} \tag{13}$$

$$\frac{dS}{dt} = \pi + \gamma_1 T_K + \gamma_2 T_C - \mu S - \delta S - \rho\lambda S - (1-\rho)\lambda S \tag{14}$$

$$\tag{15}$$

In the absence of mortality due to diarrhea illness equation (14) becomes

$$\frac{dS}{dt} = \pi - (\delta + \mu)S. \tag{16}$$

We integrate inequality (15) by the method of separation of variables and then apply initial conditions to obtain

$$N \leq \frac{\pi}{\delta + \mu} + (N_0 - \frac{\pi}{\delta + \mu})e^{-(\delta+\mu)t} \tag{17}$$

We analyze the behavior of N in inequality (17) by considering two possible cases. First, we consider $(N_0 > \frac{\pi}{\delta+\mu})$ so that, at t=0, the right-hand side of inequality (17) experiences the largest possible value of N_0.Thus $N \leq N_0, \forall > 0$. Next, we consider $N_0 < \frac{\pi}{\delta+\mu})$ so that the largest possible value of the right-hand side of inequality (17) approaches $\frac{\pi}{\delta+\mu})$ as $t \longrightarrow \infty$ that is, $N \leq \frac{\pi}{\delta+\mu}), \forall t > 0$. Hence, we conclude that $N \leq (max(N_0, \frac{\pi}{\delta+\mu}))$ for all $t > 0$ and therefore the feasible solution set of the model enters and remains in the region $\Omega = S(t), I_S(t), I_N(t), T_C(t), T_K(t)\mathbb{R}^5_+ : 0 \leq N \leq N \leq (max(N_0, \frac{\pi}{\delta+\mu}))$. This result indicates that the entire diarrhea population is bounded above by $N \leq (max(N_0, \frac{\pi}{\delta+\mu}))$ for all $t > 0$. Furthermore, we conclude that model equation (1-5) is well-posed biologically since all the solutions are non-negative for all $t > 0$. Hence, it is adequate to study the dynamics of the model in the region Ω.

4.3. Disease free equilibrium

To find the disease-free equilibrium point, we set the lefthand side of (1-5) to zero. In the absence of diarrhea illness, we let $I_S = I_N = T_C = T_N = 0$. On solving the resultant equations for the noninfected state variable, we obtain $S^0 = \frac{\pi}{\delta+\mu}$. Thus, the disease-free equilibrium point of the model is given by

$E^0 = (S^0, I_N^0, I_N^0, T_C^0, T_K^0) = [\frac{\pi}{\delta+\mu}, 0, 0, 0, 0]$.
It shows that in the absence of diarrhea, the system of equation (1-5) will consist of one compartment (the susceptible class). At this equilibrium point, the disease is eradicated from the population.

Parameters	values	Source
π	1000	Estimated
μ	0.063	[10]
γ_1	0.0012795	[11]
γ_2	0.0009403	[11]
δ	0.0006088	[11]
β	0.008034177	Estimated
ω_1	0.001	Assumed
ω_2	0.06575	Assumed
ρ	0.54795	Estimated
α_1	0.1	Assumed
α_2	0.009	Estimated
β	0.008034177	Estimated
k_1	0.0000054795	[12]
k_2	0.0001	Assumed
η_1	0.0009	Assumed
η_2	0.0007	Assumed
η_3	0.0005	Assumed
Initial conditions	values	Source
S	1000	Assumed
I_S	800	Assumed
I_N	600	Assumed
T_C	500	Assumed
T_K	400	Assumed

Table 3. *Parameter values and initial conditions for the model estimated from Kenya*

4.4. Effective reproduction number (R_{eff})

We derive the effective reproductive number $(R_{eff}$) which is defined as the average number of secondary cases produced by a single infectious case when introduced in a population where intervention strategies are in place. To obtain it, we use the technique applied by the authors in [11]. We rewrite the model equation (1-5) starting with newly infective classes as

$$\frac{dI_S}{dt} = \rho\lambda S - (\mu + \rho + \omega_2 + 1)I_S \tag{18}$$

$$\frac{dI_N}{dt} = (1-\rho)\lambda S - (\mu + \omega_1 + \delta + 1)I_N \tag{19}$$

$$\frac{dT_C}{dt} = \alpha_1 I_S - (\gamma_2 + K_1 + \mu + \delta + (1-\alpha_2))I_N \tag{20}$$

$$\frac{dT_K}{dt} = \alpha_2 I_N + (1-\alpha_1)I_S - (K_2 + \mu + \delta + \gamma_1)T_K \tag{21}$$

By the principle of the next-generation matrix, we use the notation f to represent the newly infected and the notation v to represent the secondary infected. Thus, we have

$$f = \begin{pmatrix} \rho\lambda S \\ (1-\rho)\lambda S \\ 0 \\ 0 \end{pmatrix} \tag{22}$$

$$v = \begin{pmatrix} \Omega_2 I_S \\ \Omega_3 I_N \\ -\alpha_1 I_S + \Omega_4 T_C \\ -\alpha_2 I_N + (-1+\alpha_1)I_S + \Omega_5 T_K \end{pmatrix} \tag{23}$$

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We obtain the matrices F and V by finding the Jacobian matrices of f and v evaluated at DFE respectively to obtain,

$$F = \begin{pmatrix} \rho\beta & \rho\beta\eta_1 & \rho\beta\eta_2 & \rho\beta\eta_3 \\ (1-\rho)\beta & (1-\rho)\beta\eta_1 & (1-\rho)\beta\eta_2 & (1-\rho)\beta\eta_3 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \tag{24}$$

$$V = \begin{pmatrix} \Omega_2 & 0 & 0 & 0 \\ 0 & \Omega_3 & 0 & 0 \\ -\alpha_1 & 0 & \Omega_4 & 0 \\ (-1+\alpha_1 & -\alpha_2 & 0 & \Omega_5 \end{pmatrix} \tag{25}$$

$$FV^1 = \begin{pmatrix} \frac{\beta\rho}{\Omega_2} + \frac{\beta\rho\alpha_1\eta_2}{\Omega_2\Omega_3} + \frac{\beta\rho\eta_3(\Omega_3\Omega_4-\alpha_2\Omega_3\Omega_4)}{\Omega_2\Omega_3\Omega_4\Omega_5}, \frac{\beta\rho\eta_1}{\Omega_3} + \frac{\beta\rho\alpha_2\eta_3}{\Omega_3\Omega_5}, \frac{\beta\rho\eta_2}{\Omega_4}, \frac{\beta\eta_3\rho}{\Omega_5}, \frac{\beta(1-\rho)}{\Omega_2} \\ +\frac{\beta(1-\rho)\alpha_1\eta_2}{\Omega_2\Omega_4} + \frac{\beta(1-\rho)\eta_3(\Omega_3\Omega_4-\alpha_2\Omega_3\Omega_4)}{\Omega_2\Omega_3\Omega_4\Omega_5}, \frac{\beta(1-\rho)\eta_1}{\Omega_3} + \frac{\beta(1-\rho)\alpha_2\eta_3}{\Omega_3\Omega_5}, \frac{\beta(1-\rho)\eta_2}{\Omega_4}, \frac{\beta(1-\rho)\eta_3}{\Omega_5}, \\ (0,0,0,0), (0,0,0,0) \end{pmatrix} \tag{26}$$

Finding the eigen values of FV^1 we obtain; $X_1 = 0$
$X_2 = 0$
$X_3 = 0$
$X_4 = R_{eff} = \frac{\Omega_2\Omega_3\Omega_4\Omega_5}{-\alpha_2\eta_3\Omega_2\Omega_4+\rho\eta_3\alpha_2\Omega_2\Omega_4-\rho\eta_3\Omega_3\Omega_4+\rho\alpha_2\eta_3\Omega_3\Omega_5-\eta_1\Omega_2\Omega_4\Omega_5+\rho\eta_1\Omega_2\Omega_4\Omega_5-\rho\Omega_3\Omega_4\Omega_25}$
The effective reproduction number is thus given by the spectral radius (the dominant or largest eigenvalue) of the matrix FV^1 denoted by ςFV^1, hence we have
$R_0 = -\frac{\Omega_2\Omega_3\Omega_4\Omega_5}{\alpha_2\eta_3\Omega_2\Omega_4+\rho\eta_3\alpha_2\Omega_2\Omega_4-\rho\eta_3\Omega_3\Omega_4+\rho\alpha_2\eta_3\Omega_3\Omega_5-\eta_1\Omega_2\Omega_4\Omega_5+\rho\eta_1\Omega_2\Omega_4\Omega_5-\rho\Omega_3\Omega_4\Omega_25}$

4.5. Existence of the Endemic Equilibrium Point (EE) of the Model

Theorem 4.1. A positive endemic equilibrium point of the model exists if $R_{eff} > 1$

Proof. In this section, we use the technique applied by the authors in [13]

We let $E^* = S^* + I_S^* + I_N^* + T_C^* + T_K^*$ be the endemic equilibrium point. This point is obtained by equating the left-hand side of the model equation (1-5) to zero. Thus, we have

$$0 = \pi + \gamma_1 T_K + \gamma_2 T_C - \Omega_1 S \tag{27}$$
$$0 = \rho\lambda S - \Omega_2 I_S \tag{28}$$
$$0 = (1-\rho)\lambda S - \Omega_3 I_N \tag{29}$$
$$0 = \alpha_1 I_S - \Omega_4 T_C \tag{30}$$
$$0 = \alpha_2 I_N + (1-\alpha_1) I_S - \Omega_5 T_K \tag{31}$$

where at the endemic equilibrium point, the force of infection is given by

$$\lambda^* = \frac{\beta(I_S^* + \eta_1 I_N^* + \eta_2 T_C^* + \eta_3 T_K^*)}{N} \tag{32}$$

We simplify system (27-36)

$$I_S = \frac{\rho\lambda S}{\Omega_2} \tag{33}$$
$$I_N = \frac{(1-\rho)\lambda S}{\Omega_3} \tag{34}$$
$$T_C = \frac{\alpha_1 I_S}{\Omega_4} \tag{35}$$
$$T_K = \frac{\alpha_2 I_N + (1-\alpha_2) I_S}{\Omega_5} \tag{36}$$

We substitute (27-36) into equation (32) in terms of λ^* obtain the following cases: Case 1: λ_1^* = 0, which makes no sense in this context because it corresponds to the disease-free equilibrium point of the model.
Case 2: $\lambda_2^* = \frac{s\beta I_N\Omega_4(\alpha_2\eta_3+\eta_1\Omega_5)+I_S(-S\beta(-1+\alpha_2)\eta_3\Omega_4+(S\beta\alpha_1\eta_2+(S\beta-\Omega_2)\Omega_4)\Omega_5)}{\Omega_4\Omega_5}$, which is the solution we are interested in. For disease to exist, $\lambda_2^* > 0$. If $\Omega_1, \Omega_2, \Omega_3, \Omega_4, \Omega_5 > \lambda_2^* = \frac{s\beta I_N\Omega_4(\alpha_2\eta_3+\eta_1\Omega_5)+I_S(-S\beta(-1+\alpha_2)\eta_3\Omega_4+(S\beta\alpha_1\eta_2+(S\beta-\Omega_2)\Omega_4)\Omega_5)}{\Omega_4\Omega_5}$ and therefore the model has a unique endemic equilibrium point. This result indicates that diarrhea would persist whenever $R_{eff} > 1$.

4.6. The Local Stability of DFE

We state and prove the following theorem in order to establish the local stability of the disease-free equilibrium.
Theorem 4.2. The disease-free equilibrium is locally asymptotically stable if $R_{eff} < 1$ and unstable if $R_{eff} > 1$

Proof. We use the technique applied by the authors in [13]. We first rewrite the model equation (1-5) as

Where $\Omega_1 = -(\mu + \rho + \omega_2 + 1)$, $\Omega_2 = -(\mu + \omega_1 + \delta + 1)$, $\Omega_3 = \gamma_2 + K_1 + \mu + \delta)$,$\Omega_4 = K_2 + \mu + \delta + \gamma_1$

$$F_1 = \pi + \gamma_1 T_K + \gamma_2 T_C - (\mu + \delta)S - \lambda S \tag{37}$$
$$F_2 = \rho\lambda S - \Omega_1 I_S \tag{38}$$
$$F_3 = (1-\rho)\lambda S - \Omega_2 I_N \tag{39}$$
$$F_4 = \alpha_1 I_S - \Omega_3 T_C + (1-\alpha_2) I_N \tag{40}$$
$$F_5 = \alpha_2 I_N + (1-\alpha_1) I_S - \Omega_4 T_K \tag{41}$$

$$J(E^0) = \begin{pmatrix} -\mu-\delta & -\beta & -\eta_1\beta & -\eta_2\beta+\gamma_2 & -\eta_3\beta+\gamma_1 \\ 0 & \rho\beta-\Omega_1 & \rho\eta_1\beta & \rho\eta_2\beta & \rho\eta_3\beta \\ 0 & (1-\rho)\beta & (1-\rho)\eta_1\beta & (1-\rho)\eta_2\beta & (1-\rho)\eta_3\beta \\ 0 & \alpha_1 & (1-\alpha_2) & -\Omega_3 & 0 \\ 0 & (1-\alpha_1) & \alpha_2 & 0 & -\Omega_4 \end{pmatrix} \tag{42}$$

we shall determine signs of the remaining eigen values using Routh-Hurwitz criterion. The characteristic function $|A - X_i I| = 0$ with i=1,2,3,4,5. By Routh-Harwitz criterion for determining the negatives real signs of the eigen values of the cubic polynomial are; $\lambda_3 + a_1\lambda_2 + a_2\lambda + a_3$ with conditions $a_1 > 0, a_1a_2 > a_3$
a_1 = -1
$a_2 = \delta - \mu + \beta\rho + \beta\eta_1\beta\rho\eta_1 - \Omega_1 - \Omega_2 - \Omega_3 - \Omega_4$. The remaining eigen values will be given in the appendix 1

4.7. The Global Stability of DFE.

To analyze the global stability of the disease-free equilibrium point, we use the technique employed by [14]. We first rewrite our model equation (1-5) in the form

$$\frac{dX}{dt} = H(X,Z), \frac{dZ}{dt} = G(X,Z), G(X,0) = 0 \tag{43}$$

Here X=S and Z=I_S, I_N, T_C, T_K the components of $X \in R$ denote the uninfected classes, while the components $Z = \in R^4$ denote the infected classes. The disease-free equilibrium of (43) now becomes $E^0 = (X^*, 0) = [\frac{\pi}{\delta+\mu}, 0, 0, 0, 0]$, where $X^* = (\frac{\pi}{\delta+\mu})$. To guarantee the global asymptotic stability, the following two conditions below must be satisfied:

i. (dX/dt)= H(X,0),X^* is globally asymptotically stable

ii. G(X,Z)=AZ-$\widehat{G}(X,Z), \widehat{G}(X,Z) \geq 0$ for $(X,Z) \in \Omega$

Where A=$D_Z G(X^*, 0)$ is a Metzler (M) matrix (the off- diagonal entries of A are non-negative) and Ω is the region where the model is biologically feasible. If (43) satisfies conditions i and ii, then the following theorem holds.
Theorem 4.3. The disease-free equilibrium $E^* = (X^*, 0)$ is a globally asymptotically stable equilibrium point of (42) provided that $R_{eff} < 1$ and conditions i and ii are met.

Proof. We begin by providing condition i, that is,

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$$\frac{dX}{dt} = H(X,0) = [\pi - (\delta + \mu)]. \tag{44}$$

From equation (44), we obtain the following ordinary differential equation:

$$\frac{dS}{dt} = \pi - (\delta + \mu). \tag{45}$$

Solving equation (45) and applying initial condition $S0 = S_0$ at t=0, we obtain

$$S = \frac{\pi}{(\delta + \mu)} + (S^0 - \frac{\pi}{\delta + \mu})e^{-(\delta+\mu)t} \tag{46}$$

Hence,S $\longrightarrow \frac{\pi}{\delta+\mu}$ as t$\Longrightarrow \infty$, irrespective of the values of the initial conditions. Thus, X^* is globally asymptotically stable. We then prove condition ii as

$$\frac{dZ}{dt} = G(X,Z) = \begin{pmatrix} \rho\lambda S - \Omega_1 I_S \\ (1-\rho)\lambda S - \Omega_2 I_N \\ \alpha_1 I_S - \Omega_3 T_C + (1-\alpha_2) I_N \\ \alpha_2 I_N + (1-\alpha_1) I_S - \Omega_4 T_K \end{pmatrix} \tag{47}$$

From vector (47), we have the Metzler matrix A given as

$$A = \begin{pmatrix} \beta - \Omega_1 & \eta_1\beta & \eta_2\beta & \eta_3\beta \\ (1-\rho)\beta & (1-\rho)\eta_1\beta - \Omega_2 & (1-\rho)\eta_2\beta & (1-\rho)\eta_3\beta \\ \alpha_1 & (1-\alpha_2) & -\Omega_3 & 0 \\ (1-\alpha_1) & \alpha_2 & 0 & -\Omega_4 \end{pmatrix} \tag{48}$$

Multiplying A and Z we get that;

$$\widehat{G}(X,Z) = \begin{pmatrix} (S^0 - S)(\beta\rho(I_S, I_N, T_C, T_K) \\ (S^0 - S)(1-\rho)(I_S, I_N, T_C, T_K) \\ 0 \\ 0 \end{pmatrix} \tag{49}$$

We note that $0 < \rho < 1$, and the susceptible are bounded as $S^0 \leq S$.Thus $\widehat{G_1}(X,Z), \widehat{G_2}(X,Z), \widehat{G_3}(X,Z)$ and $\widehat{G_4}(X,Z)$ are greater or equal to zero, hence $\widehat{G}(X,Z) \geq 0$. Conditions i and ii have been satisfied and therefore the disease-free equilibrium point is globally asymptotically stable provided that $R_{eff} < 1$. This result indicates that the disease would die out whenever $R_{eff} < 1$ irrespective of the initial condition.

4.8. Local Stability of the EE

In this case, the linearization method at the endemic equilibrium point is somehow mathematically complicated. We therefore analyze the local stability of the endemic equilibrium point using the method. This method is applied to check the existence of forward and backward bifurcation.

Theorem 4.4. The model exhibits a forward bifurcation at $R_{eff} = 1$. Hence, the endemic equilibrium point E^* is locally asymptotically stable for $R_{eff} > 1$ but close to 1.

Proof. To apply the center manifold theory, we consider the following $S = x_1, I_S = x_2, I_N = x_3, T_C = x_4$ and $T_K = x_5$change of variables. Let $N = x_1 + x_2 + x_3 + x_4 + x_5$ By introducing the vector notation $x = (x_1, x_2, x_3, x_4, x_5)^5$

system (1-5) can be written in the form, $(\frac{dx}{dt}) = F(x)$ with $F = (f_1, f_2, f_3, f_4, f_5)^T$ as follows:

$$\frac{dx_1}{dt} = f_1 = \pi + \gamma_1 T_K + \gamma_2 T_C - \mu S - \delta S - \rho\lambda S - (1-\rho)\lambda S \tag{50}$$

$$\frac{dx_2}{dt} = f_2 = \rho\lambda S - \mu I_S - \rho I_S - \omega_2 I_S - \alpha_1 I_S - (1-\alpha_1) I_S \tag{51}$$

$$\frac{dx_3}{dt} = f_3 = (1-\rho)\lambda S - \mu I_N - \omega_1 I_N - \delta I_N - \alpha_2 I_N - (1-\alpha_2) I_N \tag{52}$$

$$\frac{dx_4}{dt} = f_4 = \alpha_1 I_S - \gamma_2 T_C - K_1 T_C - \mu T_C - \delta T_C + (1-\alpha_2) I_N \tag{53}$$

$$\frac{dx_5}{dt} = f_5 = \alpha_2 I_N + (1-\alpha_1) I_S - K_2 T_K - \mu T_K - \delta T_K - \gamma_1 T_K \tag{54}$$

with

$$\lambda = \frac{\beta(I_S + \eta_1 I_N + \eta_2 T_C + \eta_3 T_K)}{N} \tag{55}$$

The Jacobian of system (50-54) at disease-free equilibrium point with $\beta = \beta^*$ is obtained as

$$J(E^0, \beta^*) = \begin{pmatrix} -\mu-\delta & -\beta & -\eta_1\beta & -\eta_2\beta+\gamma_2 & -\eta_3\beta+\gamma_1 \\ 0 & \rho\beta-\Omega_1 & \rho\eta_1\beta & \rho\eta_2\beta & \rho\eta_3\beta \\ 0 & (1-\rho)\beta & (1-\rho)\eta_1\beta & (1-\rho)\eta_2\beta & (1-\rho)\eta_3\beta \\ 0 & \alpha_1 & (1-\alpha_2) & -\Omega_3 & 0 \\ 0 & (1-\alpha_1) & \alpha_2 & 0 & -\Omega_4 \end{pmatrix} \tag{56}$$

We consider a case when $R_{eff} = 1$ and we let $\beta = \beta^*$ be a bifurcation parameter. Then, solving β from $R_{eff} = 1$, we get $\beta = \beta^* = -\frac{\Omega_2\Omega_3\Omega_4\Omega_5}{-\alpha_2\eta_3\Omega_2\Omega_4+\rho\alpha_2\eta_3\Omega_2\Omega_4-\rho\eta_3\Omega_2\Omega_4+\rho\alpha_2\eta_3\Omega_2\Omega_4-\rho\alpha_1\eta_2\Omega_3\Omega_5-\eta_1\Omega_2\Omega_4\Omega_5+\rho\alpha_1\Omega_2\Omega_4\Omega_5-\rho\Omega_3\Omega_4\Omega_5}$ It follows that $J(E^*)$ with $\beta = \beta^*$ has a simple zero eigenvalue. Thus, the center manifold theory is applied to analyze the dynamics of the model around $\beta = \beta^*$. $J(E^*)$ near $\beta = \beta^*$ has a right eigenvector and a left eigenvector associated with the zero eigenvalue given by $w = (w_1, w_2, w_3, w_4, w_5)^T$ and $v = (v_1, v_2, v_3, v_4, v_5)^T$ respectively. We multiply the right eigenvector w with $J(E^0, \beta^*)$ and then equate to zero. On solving, we obtain

$$w_1 = \frac{-\eta_1\beta w_2 + (-\eta_2\beta+\gamma_2) + (-\eta_3\beta+\gamma_1)w_4}{\mu+\delta} \tag{57}$$

$$w_2 = \frac{-(\rho\eta_1\beta w_3 + \rho\eta_2 w_4 + \rho\,\eta_3\beta w_5)}{\rho\beta-\Omega_1} \tag{58}$$

$$w_3 = \frac{-(1-\rho)\beta w_2 - (1-\rho)\eta_2\beta w_4 - (1-\rho)\eta_3\beta w_5}{(1-\rho)\beta} \tag{59}$$

$$w_4 = \frac{\alpha_1 w_2 + (1-\alpha_2)w_3}{\Omega_3} \tag{60}$$

$$w_5 = \frac{(1-\alpha_1)w_2 + \alpha_2 w_3}{\Omega_4} \tag{61}$$

The transpose of $J(E^0, \beta^*)$ is obtained as

$$J(E^0, \beta^*) = \begin{pmatrix} -\mu-\delta & 0 & 0 & 0 & 0 \\ -\beta & \rho\beta-\Omega_1 & (1-\rho)\beta & \alpha_1 & (1-\alpha_1) \\ -\eta_1\beta+\gamma_2 & \rho\eta_1\beta & (1-\rho)\eta_1\beta & (1-\alpha_2) & \alpha_2 \\ -\eta_2\beta+\gamma_2 & \rho\eta_2\beta & (1-\rho)\eta_2\beta & -\Omega_3 & 0 \\ -\eta_3\beta+\gamma_1 & \rho\eta_3\beta & (1-\rho)\eta_3\beta & 0 & -\Omega_4 \end{pmatrix} \tag{62}$$

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$$v_1 = 0 \tag{63}$$

$$v_2 = -\frac{(1-\rho)\beta v_3 + \alpha v_4 + (1-\alpha_1)v_5}{\rho\beta - \Omega_1} \tag{64}$$

$$v_3 = -\frac{\rho\eta_1\beta v_2 + (1-\alpha_2)v_4 + \alpha_2 v_5}{(1-\rho)\eta_1\beta} \tag{65}$$

$$v_4 = -\frac{\rho\eta_2\beta v_2 + (1-\rho)\eta_2\beta v_3}{\Omega_3} \tag{66}$$

$$v_5 = \frac{\rho\eta_3\beta v_2 + (1-\rho)\eta_3\beta v_3}{\Omega_4} \tag{67}$$

We now compute for the bifurcation coefficients a and b. Since $v_1 = 0$, we will compute the partial derivatives of f_2, f_3, f_4 and f_5 we will compute the partial derivatives of f_2, f_3, f_4 and f_5 at disease-free equilibrium point is given by

$$\frac{\partial^2 x_2}{\partial x_1 \partial x_2} = \rho\beta \tag{68}$$

$$\frac{\partial^2 x_2}{\partial x_1 \partial x_3} = \rho\eta_1\beta \tag{69}$$

$$\frac{\partial^2 x_2}{\partial x_1 \partial x_4} = \rho\eta_2\beta \tag{70}$$

$$\frac{\partial^2 x_2}{\partial x_1 \partial x_5} = \rho\eta_3\beta \tag{71}$$

$$\frac{\partial^2 x_2}{\partial x_1 \partial x_2} = (1-\rho)\beta \tag{72}$$

$$\frac{\partial^2 x_2}{\partial x_1 \partial x_3} = (1-\rho)\eta_1\beta \tag{73}$$

$$\frac{\partial^2 x_2}{\partial x_1 \partial x_4} = (1-\rho)\eta_2\beta \tag{74}$$

$$\frac{\partial^2 x_2}{\partial x_1 \partial x_5} = (1-\rho)\eta_3\beta \tag{75}$$

The bifurcation coefficients a and b are thus computed as follows: $a = [2w_1w_2(\rho\beta) + 2w_1w_3(\rho\eta_1\beta) + 2w_1w_4(\rho\eta_2\beta) + w_1w_5(\rho\eta_3\beta)] + [v_1v_2(1-\rho)\beta + v_1v_3(1-\rho)\eta_1\beta + v_1v_4(1-\rho)\eta_2\beta + v_1v_5(1-\rho)\eta_3\beta]$ Where Q=S^0.If $w_3 > 0, w_5 > 0, v_3 > 0$ and $w_1 < 0$, then $a < 0$ and $b > 0$, hence the model exhibits a forward bifurcation at $R_{eff} = 1$. Therefore, the endemic equilibrium E^* is locally asymptotically stable for $R_{eff} > 1$ but close to 1. This result indicates that the disease would persist whenever $R_{eff} > 1$ if the initial condition of the disease dynamics starts close to 1. If $w_3 > 0, w_5 > 0, v_3 > 0$ and $w_1 > 0$, then $a > 0$ and $b > 0$ hence the model exhibits a backward bifurcation at $R_{eff} = 1$. Therefore, a stable endemic equilibrium may exist even when $R_{eff} < 1$. Thus, this indicates that $R_{eff} < 1$ is not sufficient to control the spread of the disease.

4.9. The Global Stability of EE

We establish the global stability of the endemic equilibrium point in this section.

Theorem 4.5. If $P < Q$, the endemic equilibrium point E^* of the model is globally asymptotically stable.

Proof. We apply the technique employed by [13] We propose the following Lyapunov function:

$$L(S, I_S, I_N, T_C, T_K) = (S - S^* - S^* log(\frac{S^*}{S})) + (I_S - I_S^* - I_S^* log(\frac{S^*}{S})) \tag{76}$$

$$+(I_N - I_N^* - I_N^* log(\frac{I_N^*}{I_N})) + (T_C - T_C^* - T_C^* log(\frac{T_C^*}{T_C})) + (T_K - T_K^* - T_K^* log(\frac{T_K^*}{T_K})) \tag{77}$$

We differentiate equation (76-77) with respect to time and then substitute (dS/dt), $(dI_S/dt), (dI_N/dt), (dT_C/dt)$ and (dT_K/dt) from (1-5) to obtain

$$\frac{dL}{dt} = (\frac{S-S^*}{S})\pi + \gamma_1(T_K - T_K^*) + \gamma_2(T_C - T_C^*) - (\mu + \delta - \lambda)(S - S^*) + \tag{78}$$

$$(\frac{I_S - I_S^*}{I_S})\rho\lambda(S - S^*) - (\mu + \rho + \omega_2 + 1)(I_S - I_S^*) + \tag{79}$$

$$(\frac{I_N - I_N^*}{I_N})(1-\rho)\lambda(S - S^*) - (\mu + \omega_1 + \delta + 1)(I_N - I_N^*) + \tag{80}$$

$$(\frac{T_C - T_C^*}{T_C})\alpha_1(I_S - I_S^*) - (\gamma_2 + K_1 + \mu + \delta + (1-\alpha_2))(I_N - I_N^*) + \tag{81}$$

$$(\frac{T_K - T_K^*}{T_K})\alpha_2 I_N + (1-\alpha_1)(I_S - I_S^*) - (K_2 + \mu + \delta + \gamma_1)(T_K - T_K^*) \tag{82}$$

We then expand equation (78-82) and collect the positive and negative terms, respectively, to obtain where

$$\frac{dL}{dt} = P - Q, \tag{83}$$

$P = \gamma_1 T_K + \gamma_2 T_C + \rho\lambda S + (1-\rho)\lambda S + \frac{T_C I_S}{T_C} + \frac{T_K I_N}{T_K}\alpha_2 + (1-\alpha_1)I_S + \pi + \lambda S$

$Q = \gamma_1 T_K^* + \gamma_2 T_C^* + (\mu + \delta)S^* + \frac{\rho\lambda I^* S^*}{I_S} + (\mu + \rho + \omega_2 + 1)I_S^* +$
$(1-\rho)\lambda\frac{I_N^* S^*}{I_N} + (\mu + \omega_1 + \delta + 1)I_N^* + \frac{T_C^* I_S}{T_C}\alpha_1 + (\gamma_2 + k_1 + \mu + \delta +$
$(1-\alpha_2))\frac{I_N^* T_C^*}{T_C} + \frac{T_K^* I_S^*}{T_K}\alpha_1 + (k_2 + \mu + \delta + \gamma_1)T_K^* + \alpha_1 I_S$ If $P < Q$, then we obtain $dL/dt \leq 0$ noting that $dL/dt = 0$ if and only if $S = S^*, I_S = I_S^*, I_N = I_N^*, T_C = T_C^*$ and $T_K = T_K^*$. Thus, the largest compact invariant set in $(S^*, I_S^*, I_N^* T_C^*, T_K^*) \in \Omega : (dL/dt) = 0$ is the singleton E^*, where E^* is the endemic equilibrium point of the model. Therefore, by LaSalleâs invariance principle [15], E^* is globally asymptotically stable in E^* if $P < Q$. This result indicates that the disease would persist whenever $P < Q$ irrespective of the initial condition.

4.10. Normalized sensitivity analysis of basic reproduction numbers

Sensitivity analysis of parameters is carried out using the differential calculus. The analysis involves examining the parameter which affects the basic reproduction number most. It tells us how each model parameter is important to childhood diarrhea infection transmission. It is used to discover parameters that have high impact on R_0 and should be targeted by intervention strategy. Sensitivity indices allow us to measure the relative change in a variable when a parameter changes.

Parameters	Sensitivity indices
π	-0.00377942
μ	-0.000031224
ρ	0.528113
ω_1	$-4.58265 * 10^-7$
ω_2	-0.0412177
δ	0.00031516
β	0.000266559
k_1	0.000196475
α_2	-0.0000108841
k_2	$3.03942 * 10^-6$
ρ	-0.00389483
η_3	0.124437
α_1	$1.92379 * 10^-6$
η_2	$3.70097 * 10^-11$
η_1	0.0000328237

Table 4. *Sensitivity indices of parameters*

5. Model parameters used for simulation

In order to examine the dynamic Runge-Kutta analysis of the framework's state variables' dynamics when the model specifications are present technique is applied to the model equations in this section and then utilized to do numerical simulations using the fourth order Runge-Kutta method in MatlabR2015a. An ordinary differential equation's initial value problem can be solved numerically using the Runge-Kutta method. The initial conditions and parameters values listed in table 2 are used to carry out the numerical simulations and a graphical presentation of the numerical results is made.

5.1. Susceptible population for under five children

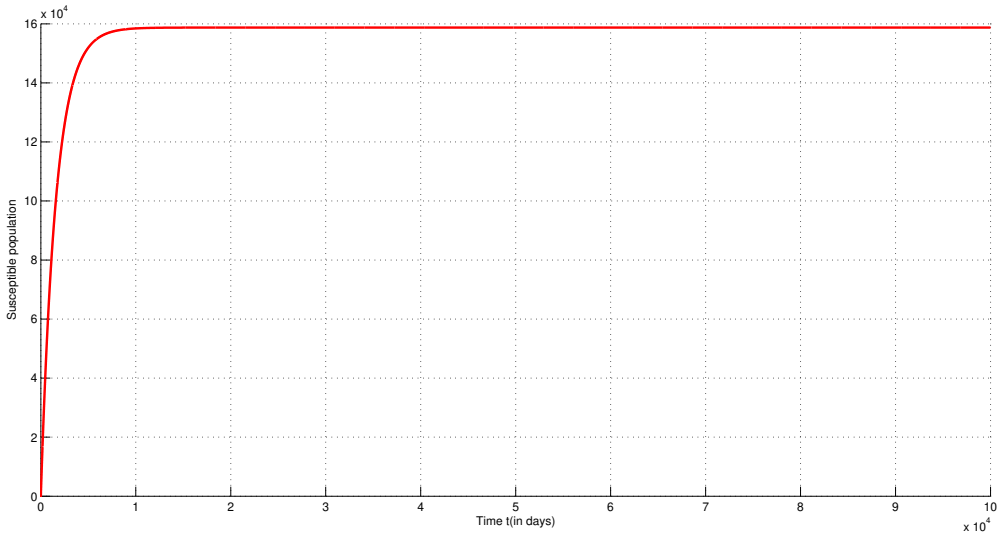

Figure 2. *Total population for under five children with time*

The susceptible population particularly in Kenyan population is expected to double after every ten years according to data published in literature, however in the occurrence of disease the population will rise and then stabilize at a point like in this case the population of children rise until it start to stabilize as some of them become infected and leave that class also when they leave the class of five years as observed in fig 2. The population will remain like this as long as the childhood diarrhea persist in the population but if proper health care services are given in time it could save more live that would otherwise be lost.

5.2. Diarrhea infected under five children under stress

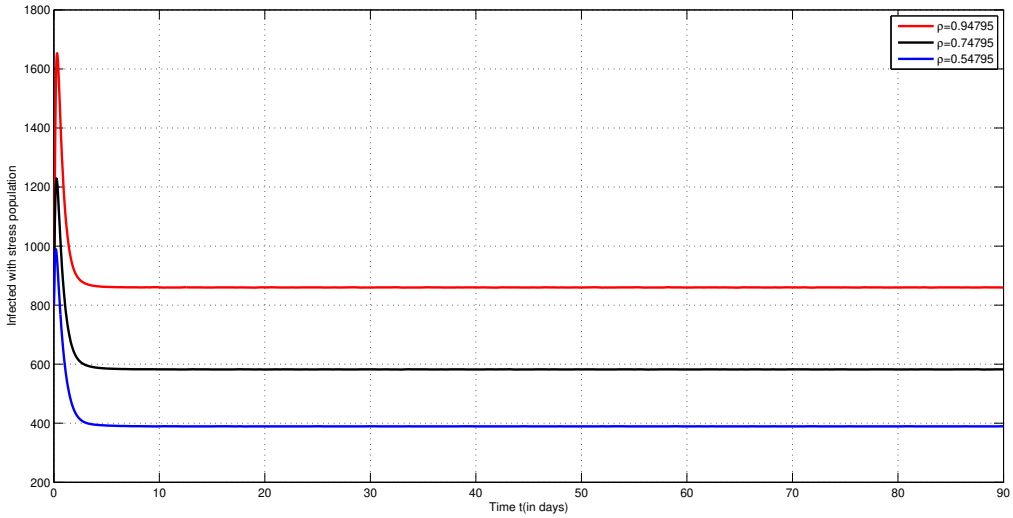

Figure 3. *Diarrhea infected under five children under stress*

This class begin by gaining from the susceptible who acquire diarrhea, its characterized by acute diarrhea as these children are also subjected to stress and it have been known to increase bowel movement particularly in under five children. This class is characterized by under five children subjected to poor condition in Majengo Nyeri county and these kids are more prone to stress and and poor house holds where by most of them find it difficult to access good health condition and this necessitate high retention in disease and high mortality and morbidity. In fig 3 the higher the force of infection the higher the infection and this lead to an increase in infected population, when $\rho = 0.94795$ the higher the population as more becomes infected as even stress fuel diarrhea, $\rho = 0.74795$ as the force of infection decrease the lower the infected population, $\rho = 0.54795$ this shows a lower transmission and lower infected class due to decreased force of infection as more children seek good health care services

5.3. Diarrhea infected under five children free of stress

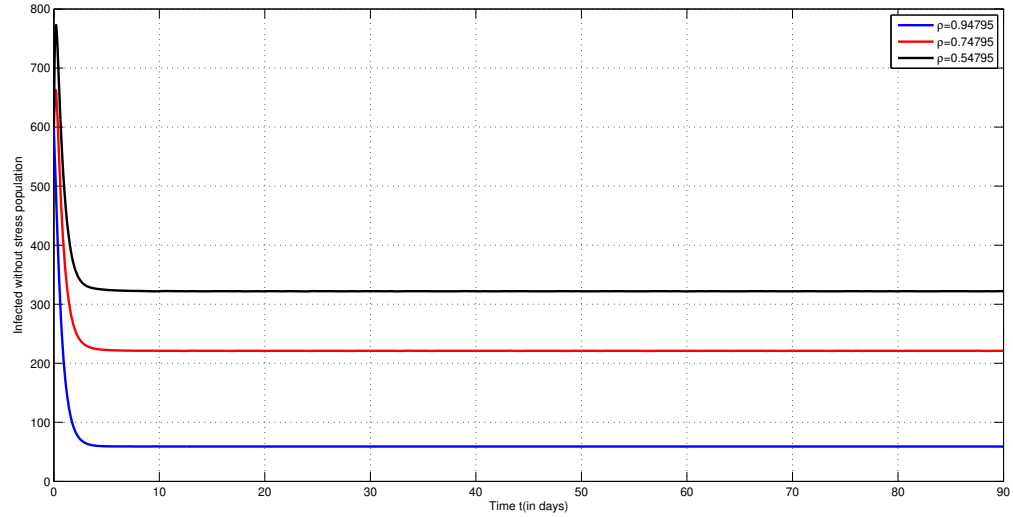

Figure 4. *Diarrhea infected under five children under stress*

This class is characterized by a bit low population of diarrhea infected as compared with the above class with stress, though mortality and morbidity is a bit high but cant be compared to the class with stress. These include under five children outside Majengo area in Nyeri county where by they can access better health care and good house holds. When the force of infection was compared it showed variation, as in fig 4 $\rho = 0.94795$ at this rate there was more infected as rate of infection was also high, $\rho = 0.74795$ as the force of infection decrease there's a decrease in infection and when $\rho = 0.54795$ at this rate there was minimal infection which show diarrhea can be contained in the population through increase good health service and proper house holds for the under five children.

5.4. Treatment for under five children in faulty health system

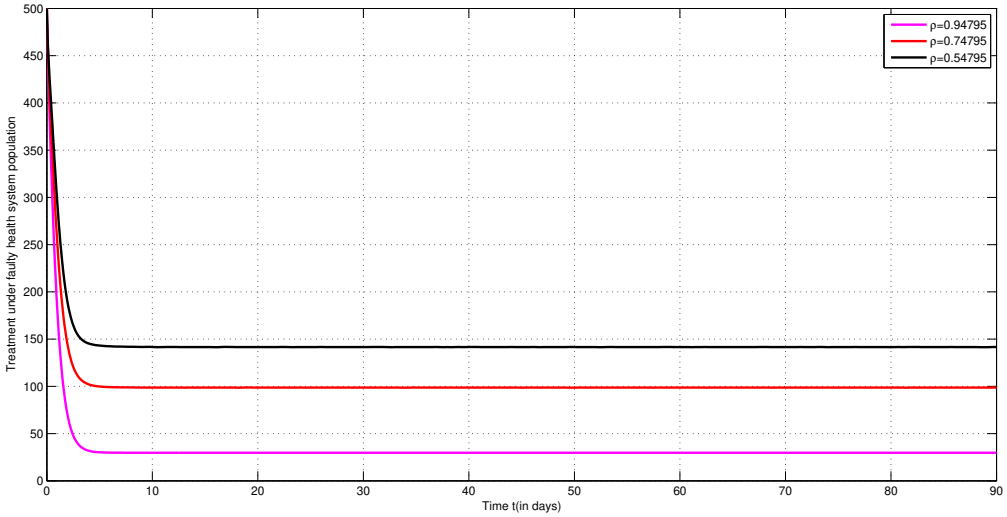

Figure 5. *Treatment for under five in faulty health system with time*

This class is characterized by low turn out for those kids seeking medical attention as these kids come from poor house holds and also the services offered are poor also the means to reach theses services is a problem the personnel and necessary facilities in these health system is also a problem. This class may also receive under five children from infected without stress class who may unknowingly seek health services. There is high mortality rate in this class as most children don't get adequate medical attention and only those who may opt to look for medical services else where can get help. In fig 5 The variation in the force of infection show difference in population the higher the force of infection the higher rate of under five children seeking treatment.

5.5. Treatment in normal health system for under five children at varying force of infection

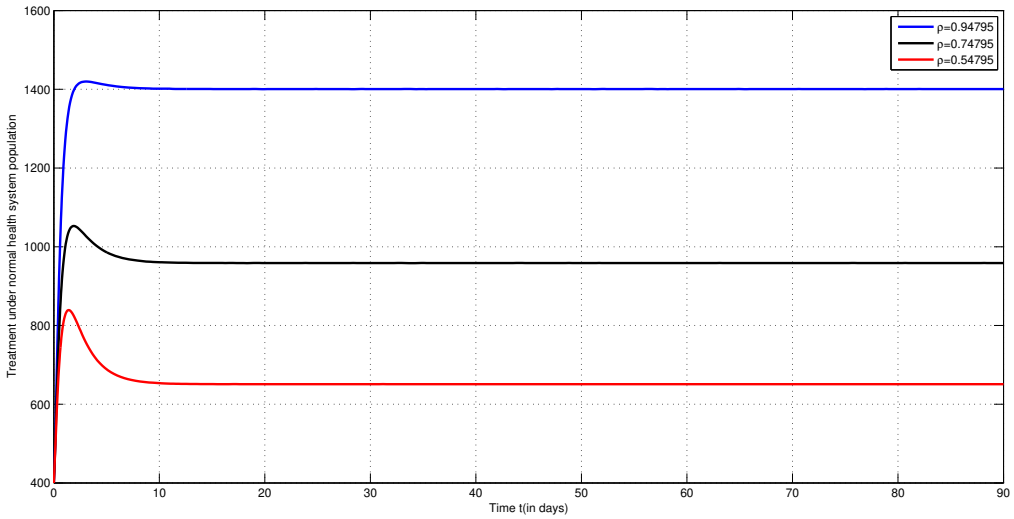

Figure 6. *Treatment for under five in normal health system with time*

This class is characterized by high population of under five children seeking treatment, these children though have diarrhea though its not under influence of others factor as compared to those under stress. Here the health facilities resources and personnel is in good working condition, children can be able to acquire these services with ease at varying force of infection mean varying population seeking treatment. The higher the force of infection the higher the population, some children under stress can can access these services if they have resources to pay for the service as in fig 6.

5.6. Treatment for under five children at varying treatment rate under stress

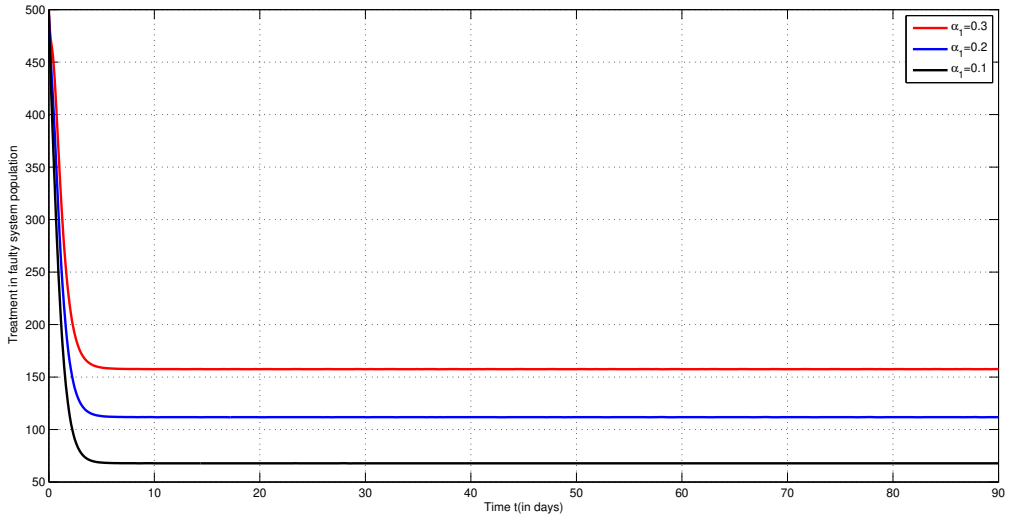

Figure 7. *treatment for under five children with time*

This class is characterized by immense low turn out as these facilities usually are known to have faulty health system and also poor medical services. This population is also characterized by children living in poor house holds like Majengo in Nyeri county

where the case of social factors(stress) is paramount and access to good services is the only problem though some families can be able to afford good services but are minimal. An increase in the rate of seeking treatment increase the population of those seeking treatment decreasing the mortality rate. From the graph fig 7 its evident that an increase in rate seeking treatment have significant positive impact on the population of children.

5.7. Treatment for under five children at varying rate of recovery

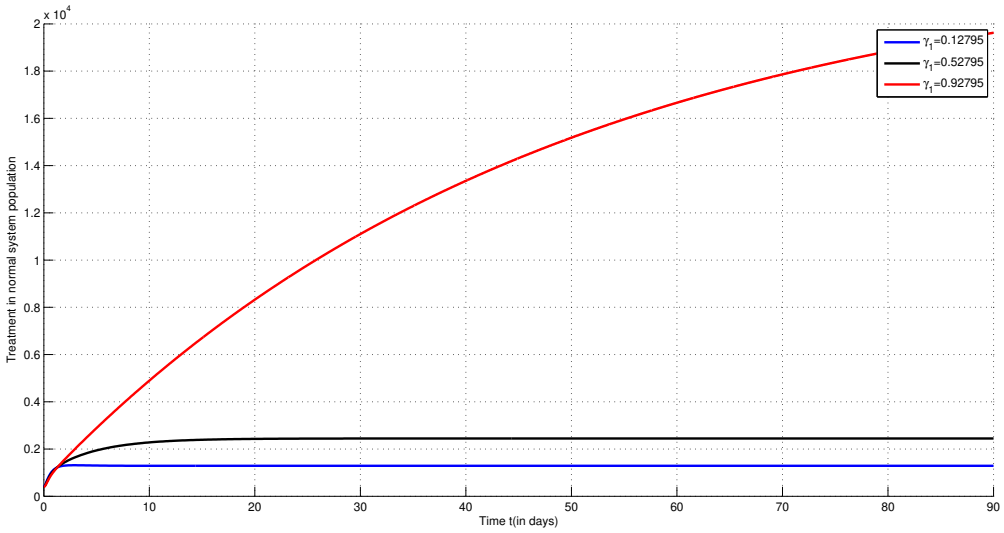

Figure 8. *traetment for under five children with time*

In this class under five children are subjected to good health system and proper house holds where access to health care is not a problem and social factor like stress is not a problem. When the rate of recovery increases that means more will be leaving and joining the susceptible class the low the recover the lower the recovered population according to fig 8.

5.8. Treatment for under five children in faulty health system at varying rate of recovery

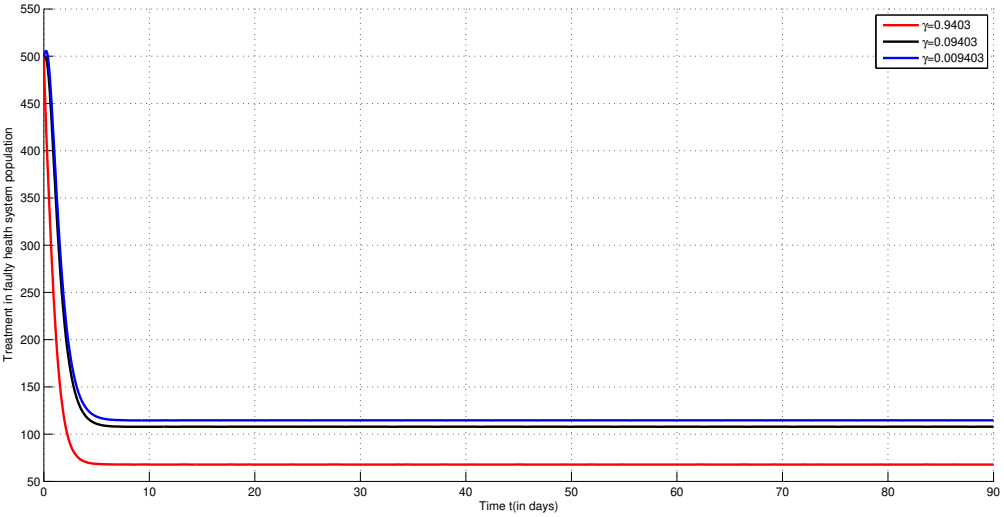

Figure 9. *treatment for under five children with time*

As compared to treatment under normal health system this class usually have few children as the suffer from social health facilities and poor services in the health system and house holds. An increase in rate recovery increase the out put population and a decrease will lead to a decrease as observed in the in the figure 9.

5.9. Comparison between infected under stress and without stress at varying force of infection

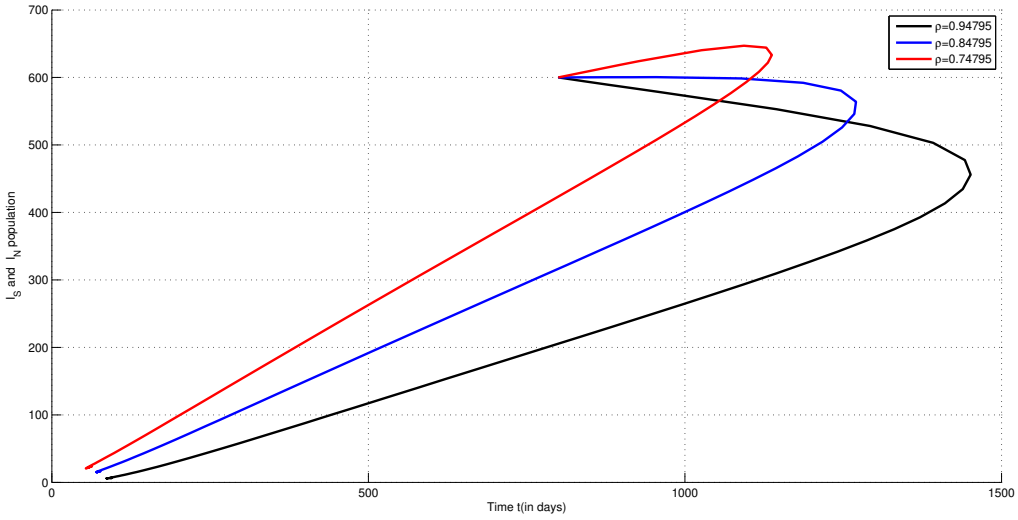

Figure 10. *Comparison between infected under stress and without stress with time*

In this we compare between infected with stress I_N and infected with stress I_N and I_S at varying force of infection. If the parameters increasing the force of infection are reduce this means that children will not be acute as compared when its high also when social factors like stress is contained in children the effect of diarrhea is maintained. In fig 10 as ρ decrease as seen in the red graph the population will near zero point that means the diarrhea disease will be maintained at baseline level.

5.10. Comparison between infected under stress and treatment in faulty health system varying rate of seeking treatment

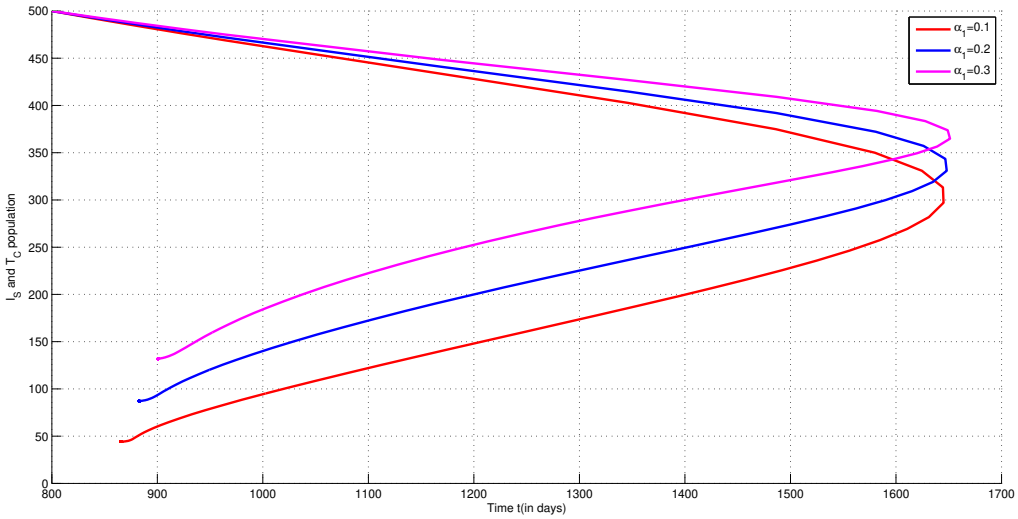

Figure 11. *A graph of I_S and T_C with time*

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The comparison between infected with stress I_S and treatment under faulty health system T_C the two classes suffer two main problem where one is under social factor (stress) and the other is faulty health system under diarrhea there seem to be an increase in the population when α_1 increase this may be characterized by the increase in problem of social factor and poor health system and house holds as observed in fig 11.

5.11. Comparison between infected without stress and treatment in normal health system at varying rate of seeking treatment

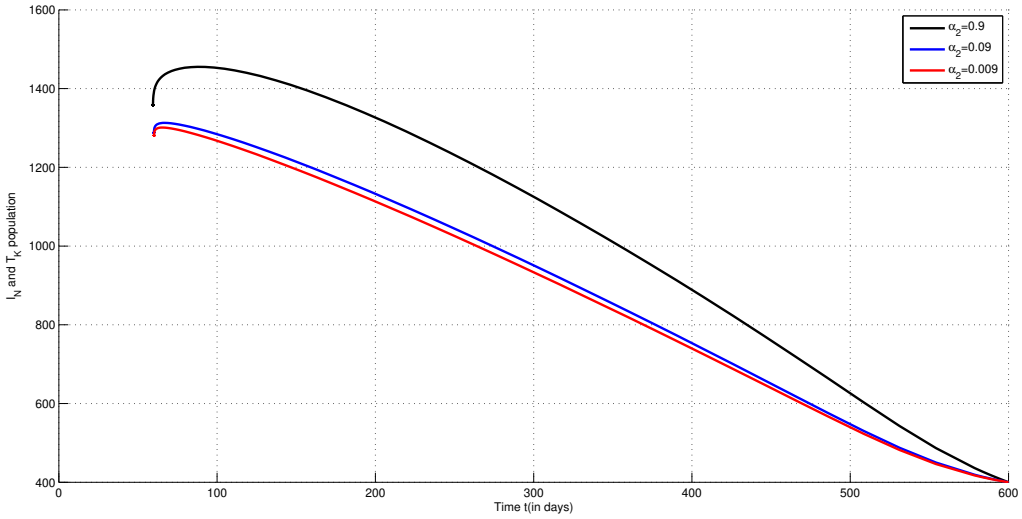

Figure 12. *Comparison between infected without stress and treatment in normal health system at varying rate of seeking treatment with time*

The comparison between under five children seeking treatment without stress and treatment in normal health facilities show that a decrease in rate of seeking treatment will lead to decrease in both class as less people will be treated and less will recover as observed in figure 12. Hence more emphasis should be on increasing treatment rate and ensuring that health facilities are well equipped to ensure better health facilities.

5.12. Conclusion

In this study, we developed and analyzed a deterministic under five childhood diarrhea model incorporating social factor (stress) and faulty health system and their effect on under five children during diarrhea outbreak. The model structure comprised of five compartments of human populations, namely; susceptible individuals, diarrhea infected with stress, diarrhea infected without stress, treated in faulty health system and treated in normal health system. The model assumed social factor (stress) and faulty health system as the agents of diarrhea transmission. The model is a first order ordinary differential equation. The state variables were proved to be positive and lie in the feasible region. The model reproduction number, was determined as the largest eigen value of the Jacobian matrix using the Next Generation Matrix Method. Using the Jacobian Matrix, the local stability of the disease free equilibrium point was established to be locally asymptotically stable whenever $R_0^* < 1$. The analytical results were obtained for the basic reproduction number. Normalized sensitivity indices for the basic reproduction number with respect to control parameters were determined. The initial conditions of the state variables and model parameters were obtained from the secondary data available in the literature. The numerical simulations were carried out in MATLAB which has an inbuilt numerical method-fourth and fifth order Runge-Kutta method. Computational mathematics, integral and differential calculus was used for mathematical analysis. The numerical results are presented graphically. An increase in the force of infection lead to increase in diarrhea infection which will also lead to increase in diarrhea on under five children with social factor problem (stress) leading to higher fueling of diarrhea. This infers that under five with stress condition contributes significantly to the spread of childhood diarrhea as seen in figure 3. Faulty health system and poor house holds usually hinder good health services to a child whereby they cant get good health services or it become a hindrance thus most of the may suffer severely and high mortality rate in prevalence as observed in figure 5.

5.13. Conflict of interest

The writers state that they have no personal or professional ties to individuals or groups that could improperly influence their work. There isn't any personal or professional relationship with any firm, product, or service that would be thought to have an impact on the evaluation or the opinion stated in the article.

5.15. Compliance with Ethical Standards

Informed consent is not required for this type of study.

5.16. Ethical Conduct

The required norms while carrying out research was adhered to.

5.17. Data availability statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

COMPETING INTERESTS DISCLAIMER:
Authors have declared that they have no known competing financial interests OR non-financial interests OR personal relationships that could have appeared to influence the work reported in this paper.

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