

**MODELING SOCIAL FACTOR AND FAULTY HEALTH SYSTEM ON THE
DYNAMIC OF CHILDHOOD DIARRHOEA IN LOW INCOME POPULATION**

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**A Research Project Submitted in Partial Fulfillment of the Requirements for the
Award of the Degree of Master of Science in Applied Mathematics to the School of
Pure and Applied Sciences, Kirinyaga University**

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DECLARATION

This research project is my original work and has not been presented for a degree or any other award in any other university.

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DEDICATION

To my mother, father, brothers, my husband Charles, and my friends. I appreciate your unwavering support.

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I would like to express my thanks to the Almighty GOD most sincerely for His continued faithfulness and enabling to the hallmark of this research. In addition, my honest gratitude goes to my supervisors Dr Rachel Atieno and Dr Moffat Chamuchi. I extend my gratitude to the Kariara Junior School community for providing a supportive environment that facilitated my research and academic development.

ABSTRACT

Mathematical modeling of infectious diseases offers insights into the core processes of disease propagation and transmission and assesses the potential severity of an epidemic. The main way that diarrhoea, 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. Surveillance studies conducted in wellness facilities may underestimate the true burden of disease, especially in settings with limited resources where many individuals do not seek medical care. In addition to limiting access to healthcare, low socioeconomic level can have an impact on housing conditions, food, and other elements that raise the risk of contracting infectious diseases. Proper management of childhood stress and improvements in healthcare systems significantly reduce diarrhoea incidence and associated complications. Numerous models have been suggested in scholarly works to control childhood diarrhoea disease, however there is scanty information about modeling social factor and faulty health system regarding the trends and factors influencing diarrhea in children in Kenya. This study developed a mathematical model of social factor and faulty health system on the dynamic of childhood diarrhoea in Kenya. The model was based on a system of nonlinear first-order ordinary differential equations. It demonstrated using the Jacobian matrix that the local and global stability analysis of the disease dies out and reaches a disease-free equilibrium when the basic reproduction number (R_0) is less than 1. Specifically, R_0 was calculated to be 0.008278, indicating that with appropriate care for children under five during a diarrhoea outbreak, the disease can be effectively controlled. Conversely, if R_0 is greater than 1, the disease may persist, leading to the establishment of an endemic equilibrium. Using center manifold theorem to calculate bifurcation analysis demonstrated a forward bifurcation showing the disease will die out at sometime. Simulation studies using the model parameters was calculated to show how social factor (stress) and faulty health system propagate childhood diarrhoea as seen in figure 2 and 5. Under five children with stress and also subjected to faulty health system usually suffer severely as compared to those without stress and in normal health facilities. The findings of this study will offer important insights to relevant 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 which will help to reduce the spread of under five childhood diarrhea during an outbreak that will otherwise remain unknown leading to better designs for future development.

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SYMBOLS AND INDEX OF NOTATIONS

$S(t)$	The population's number of vulnerable kids at time t
$I_S(t)$	Diarrhoea among under-five children linked to stress
$I_N(t)$	Diarrhoea infection in children under five without the influence of stress
$T_C(t)$	Diarrhoea treatment of under five children in faulty health system
T_K	Diarrhoea treatment of under five children in normal health system
π	Recruiting rate for the susceptible class (S)
μ	Children's net natural death rate
λ	The pace of infectious transmission or force
γ_1	Recovery rate for therapy under faulty health system
γ_2	Recovery rate for for therapy under normal health system
δ	The rate at which children less than five years old leave the five-year-old age range
ω_1	Diarrhoea-related mortality rate among infected without stress.
ω_2	Diarrhoea-related mortality rate among infected with 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	Mortality rate from diarrhoea within the compromised health system group
K_2	Mortality rate from diarrhoea in a functioning health system

ρ	The percentage of vulnerable kids that join the stress-infected
$(1 - \rho)$	The percentage of vulnerable kids who join the infected group stress-free
N	Total population

ABBREVIATION AND ACRONYMS

SIR	Susceptible Infected Recovered
CRC	China Red Cross
MATLAB	Matrix Laboratory
R_0	Basic reproduction Number
DFE	Disease-free steady state
EEP	Equilibrium Free of Disease
WHO	World Health Organisation
SDG	Sustainable Development Goals
NACOSTI	National Commission for Science, Technology, and Innovation

CHAPTER ONE

INTRODUCTION

1.1 Contextual Framework

Diarrhoea is a condition caused by infections from parasites, viruses, or bacteria, with the main mode of transmission being water contaminated by fecal matter (Hailu *et al.*, 2006). It is indicative of a digestive tract infection and is typically accompanied by symptoms such as cramp, stomach pain, and bloating (Alam *et al.*, 2003). The fecal-oral pathway is the primary means of transmission for diarrhoeal infections (Boschi *et al.*, 2009). It can be divided into two phases: acute diarrhoea, which lasts for a few days, and chronic diarrhoea, which lasts for a week or more (WHO, 2020).

These infections spread from person to person through the environmental media such as contaminated food and water, poor sanitation, and vectors like flies and unclean hands (Julian and Timothy, 2016). Oral re-hydration therapy (ORT), vaccinations, zinc supplements, access to and quality of healthcare facilities, and exclusive breastfeeding are some methods for preventing and treating diarrhoea (Boschi *et al.*, 2009). In addition, proper sanitation, maintaining proper personal and household hygiene at home, and clean water can lessen the prevalence of diarrhoea (Boschi *et al.*, 2009). After an illness, the protection to diarrhoea is temporary, and the infection is usually milder than it was initially (Qureshi *et al.*, 2019).

Diarrhoea can be classified as either infectious or non-infectious and may be caused by viruses, parasites, or bacteria. Infectious diarrhoea is typically the result of pathogens such as *Campylobacter*, Shiga toxin-producing *E. coli*, *Giardia lamblia* (giardiasis), *Salmonella* (salmonellosis), *Shigella* (shigellosis), Rotavirus, *Yersinia*, or *Cryptosporidium* (cryptosporidiosis). Non-infectious diarrhea, on the other hand, is often caused by toxins, such as those responsible for food poisoning, and does not spread from person to person. Immunity following an infection is usually short-lived, and any subsequent infections are generally milder than the first. Despite its causes, diarrhea is both treatable and preventable (marino, 2013). Diarrhoea ranks among the top causes of death in children under the age of five globally, accounting for more than 1.5 million deaths annually—approximately one in every nine deaths in this age group (ogwel, 2024).

Each year, approximately 1.7 billion cases of diarrhoea are reported among children worldwide, and low-income nations account for a large share of diarrheal morbidity and death because of a lack of resources and infrastructure in their healthcare systems (mills, 2014). Africa's Sub-Saharan region is the most susceptible to infectious diseases. This is because of the region's significant climate change impact, this increases the risk of outbreaks during periods of rainfall and runoff, as the resulting turbidity can reduce the effectiveness of drinking water treatment facilities (Black, 1993). These illnesses are usually transmitted via tainted food, drinking water, or animals to humans or between people due to inadequate personal hygiene (WHO, 2005). It is thought to kill more young children than the combined effects of HIV/AIDS, measles, and malaria (Bonyah *et al.*,2018).

Over 60% of diarrhea-related deaths take place in Sub-Saharan Africa, where it stands as the fourth leading cause of death among children under five globally (Winter *et al.*,2019). Among children under five in Africa, diarrhea is the third leading cause of death. In 2015 alone, it was responsible for approximately 30 million severe cases and 330,000 deaths (Reiner *et al.*,2018). In Kenya, diarrhea is a major cause of death among children under five, ranking third after pneumonia and neonatal conditions (Mutama *et al.*, 2019). In 2018, Kenya recorded 1,499,146 cases of diarrhoea in children under the age of five.

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 (Headey, 2016). In addition to affecting the way visceral organs, particularly the digestive system, work, stress can lead to irregularities in behavior and/or thought, including anxiety and depression. Patients with depression may have mental issues linked to oxidation and oxidative stress, as seen by elevated levels of the fatty acid peroxide malondialdehyde in their serum (Maes *et al.*, 2013).

In the meantime, depression has also been linked to bacterial translocations and immunological alterations (Ohlsson *e al.*, 2019). Therefore, there appears to be a shared connection

between depression and bowel disorders (diarrhea) that explains how the two conditions interact. A rising body of research in recent years has shown that bowel illness patients, both adults and children, have a higher prevalence of mental disorders than the general population (mikočka *et al.*, 2016).

Additionally, the development of bowel disease can be somewhat impacted by stress at any time, whether it occurs in childhood or maturity. Health facility-based surveillance studies may underestimate the true disease burden when it's not possible to account for cases that do not seek medical care—particularly in under-resourced areas with limited access to healthcare or in communities where commonly visited healthcare providers are excluded from the surveillance system. Moreover, characteristics including age, sex, socioeconomic status (SES), the severity of the condition, and the household's closeness to the health facility can all affect care-seeking behavior, which can lead to confounding or interaction and skew the results (Bigogo *et al.*, 2010).

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 (Forsberg *et al.*, 2009).

However, in poor nations, the behavior of seeking medical attention is influenced by certain conditions, and determinants that impact the delay in obtaining the right medical care may result in household-level self-care practices, use of home remedies, and visits to traditional healers (Nyamongo, 2002). The result of transportation issues brought on by inadequate road infrastructure and a lack of transit to the medical institution represents a significant obstacle to proper access to healthcare services (Alom, 2012). Financial accessibility issues could make it more difficult to pay for care seeking out of pocket.

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 forecast the transmission of infectious diseases (Hartemink *et al.*, 2011). These models are essential frameworks for comprehending the transmission of diseases, forecasting possible outbreaks, and evaluating the results of different therapies (Adesola *et al.*, 2024).

Nevertheless, limited research has utilized mathematical modeling to examine the patterns of diarrhea among children in Kenya and to suggest possible prevention strategies. 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 Target 3.2 of sustainable Development Goal 3 aims to lower under-five mortality rates to no more than 25 deaths per 1,000 live births by the year 2030 (Bora *et al.*, 2018).

Adewale, 2015 To better understand how vaccination and treatment of infected individuals affect the transmission of diarrhoea within a population, a deterministic epidemic model consisting of four compartments—Susceptible (S), Vaccinated (V), Exposed (E), and Infected (I)—was developed. This model examines how vaccination influences the spread of diarrhoeal disease in the community. The total population is represented as

$$N(t) = S + V + E + I$$

Individuals enter the population at a rate λ , increasing the number of susceptibles. However, the susceptible group decreases as a portion ρ of new individuals are vaccinated, and as susceptibles become infected through contact with infectious individuals, with β denoting the effective contact rate.

Overall, the model highlights the significant role vaccination plays in controlling the spread of diarrhoeal infections. The number of susceptible people increases based on how well the vaccine works to prevent diarrhea; the fewer susceptible people there are, the less the disease spreads; however, if the vaccine wears off quickly, the number of infected people increases. Accordingly, vaccinations should be administered as soon as feasible after delivery, before the risk of contracting a diarrhoeal illness, and they should be efficient to reduce the disease's spread and associated expenses. To help reduce the spread of diarrhoea in the community, medications should be made more accessible by lowering their cost, and

the impact of vaccination should be carefully considered. However, the approach did not take into account a fractional diarrhoea model incorporating the Mittag-Leffler kernel.

Bonyah *et al.*, 2019 The study analyzed the endemic equilibrium (E_1), the disease-free equilibrium (E_0), and the basic reproduction number (R_0). The stability of the disease-free equilibrium was examined both locally and globally. When R_0 is greater than 1, the endemic equilibrium is locally asymptotically stable, indicating that diarrhoeal disease will continue to exist within the population. Conversely, if R_0 is less than 1, the disease will eventually be eliminated. Numerical simulations, performed using Python, showed that the dynamics of diarrheal disease are significantly influenced by the maximum achievable treatment level, referred to as the saturation treatment rate. As more individuals receive this treatment, the disease increasingly disappears from the community. The treatments discussed include anti-diarrhoeal medications such as Imodium (loperamide) and Pepto-Bismol or Kaopectate (bismuth subsalicylate).

These are particularly effective in treating cases like traveler's diarrhoea, which is often caused by consuming contaminated food or water. Although the study incorporated an analysis of a fractional diarrhoea model using the Mittag-Leffler kernel, it did not address the traditional infectious diarrhea model. The researchers recommend that health policymakers ensure widespread availability of anti-diarrheal medications, aiming for a 99% saturation treatment rate at an affordable cost to effectively curb the spread of diarrhoea and prevent future outbreaks.

Iqbal *et al.*, 2022 examined a traditional model of infectious diarrhoea and transformed it into a fractional order model. Fractional differential operators in Caputo-sense, developed by Atangana Baleanu, were used for the conversion. The Mittag-Leffler Kernel approach was used to solve the expanded model once it had been modified. However, the existence and uniqueness of the solution are established before solving the underlying model. To examine the behavior of the suggested approach, computer-aided numerical graphs are plotted. Depending on the parameter ν , each graph shows a distinct rate of convergence towards the precise steady state. The order of the fractional derivative, denoted by ν , is directly proportional to the rate of convergence. Both the endemic and disease-free steady

states have been shown to be stable locally and globally.

Furthermore, the stability of these equilibrium states and the role of the basic reproduction number (R_0) in disease dynamics were analyzed, revealing that the equilibrium points' stability depends on the value of R_0 . Stability is achieved locally and globally if R_0 is smaller than one, and this is also true for endemic states. In the future, this technique could be used to solve and change different nonlinear systems. However, they did not include optimal control of certain techniques, such as X, in their study.

(Naveed *et al.* ,2023) They provided epidemiological models based on ordinary differential equations in their work to comprehend the dynamics of diarrheal infections while taking into account multiple carriers. To determine the interaction and dynamics between flies and humans during a disease outbreak, mathematical studies and numerical simulation were conducted. The best condition was then achieved by using each control approach and combining them in various ways. Scenario X, a simulation that integrates all control strategies and effectively minimizes disease transmission, was identified as the most successful approach for reducing both the fly population and the number of affected individuals.

They suggested that further research be done in the future on more general topics, like how temperature affects the spread of disease, because houseflies' numbers peak in warmer climates. Taking into account numerous carriers, epidemiological dynamics, and pertinent characteristics, the suggested framework was deemed an efficient disease control method. However, they did not employ a mathematical model to analyze the dynamics of diarrhoeal disease in relation to preventive vaccination and treatment.

(Olutimo *et al.*, 2024) A mathematical model was employed to examine how preventive vaccination and treatment affect the spread of diarrhoeal disease. The analysis involved determining the disease-free and endemic equilibrium points, followed by the computation of the basic reproduction number (R_0). The results showed that the disease-free equilibrium is unstable unless $R_0 < 1$, in which case it becomes locally asymptotically stable, and globally asymptotically stable when $R_0 \leq 1$. In contrast, the endemic equilibrium is stable when $R_0 > 1$. This implies that effective vaccination and treatment can control the spread

of diarrhoea, regardless of the initial number of cases, as long as the basic reproduction number remains below one.

Further analysis confirmed that when $R_0 < 1$, the disease-free state is both locally and globally stable, whereas if R_0 exceeds one, diarrhoea will continue to circulate in the population. Sensitivity analysis identified the contact rate (β) as the most critical parameter influencing R_0 , with a positive sensitivity index. This suggests that the system will remain asymptotically stable when $R_0 < 1$, and that β has the greatest effect on the number of new infections and, consequently, the overall prevalence of the disease.

Motivated by these findings, a deterministic model exploring how social factor(stress) and faulty health system affect diarrhoea transmission in under five years is formulated and rigorously analyzed. The study has explored by creating a model on the relationship between social health factor and a dysfunctional healthcare system and the prevalence of childhood diarrhea, and will demonstrate how stress and these factors may exacerbate the condition in children under five.

1.1.1 Basic Mathematical Concepts

This section presents the essential mathematical concepts necessary for understanding this study. Epidemic models are typically used to represent short-term disease outbreaks lasting less than a year, while endemic models are applied to longer-term scenarios where new susceptible individuals are introduced into the population through births or loss of temporary immunity. The foundational Susceptible-Infectious-Recovered (SIR) models offer a straightforward framework for grasping more advanced results in epidemiological modeling (Allman and Rhodes, 2004).

1.1.2 Formulating Epidemiology Models

Let $S(t)$ denote the number of individuals susceptible to infection at time t , $I(t)$ represent the number of infected individuals at that time, and $R(t)$ indicate the number of individuals who have recovered. The total population is represented by N . $S(t) = \frac{S(t)}{N}$ and $i(t) = \frac{I(t)}{N}$ they correspond to the susceptible and infected portions of the population, respectively. If β Represents the average number of effective contacts (i.e., those capable of causing transmission) an individual has per unit of time, then $\frac{\beta I}{N} = \beta i$ indicates the average rate at which susceptible individuals come into contact with infected persons over a given time

period as a result of the $S = N_s$ individuals who are at risk of infection. This type of horizontal transmission is referred to as standard incidence.

1.1.3 The Classic Epidemic Model

The traditional epidemic model, known as the SIR model, is defined by the following initial value problem.

$$\frac{dS}{dt} = \frac{-\beta IS}{N} \quad (1.1.1)$$

$$\frac{dI}{dt} = \frac{\beta IS}{N} - \gamma I \quad (1.1.2)$$

$$\frac{dR}{dt} = \gamma I \quad (1.1.3)$$

Where $S(0) > 0, I(0) > 0$ and $R(0) > 0$ $S(t)$, $I(t)$, and $R(t)$ are the numbers in these classes so that $S(t) + I(t) + R(t) = N$ This model applies standard incidence and assumes recovery occurs at a rate of γI which corresponds to an exponential waiting time of $e^{-\gamma t}$. Due to the short time frame considered, the model does not incorporate vital dynamics such as births or deaths. Additionally, since the recovered class R does not influence the first two equations, the system can be simplified to two equations by reducing its dimensions. By dividing the equations.

$$\frac{ds}{dt} = -\beta i s \quad (1.1.4)$$

$$\frac{di}{dt} = \beta i \left(s - \frac{\beta}{\beta} \right) \quad (1.1.5)$$

Obviously any point $(s; i) = (c; 0)$ is a fixed point, that is we have a line of fixed points. Also the i - axis is a separatrix because

$$\frac{di}{ds} = \frac{\beta s - \gamma}{-\beta s} \quad (1.1.6)$$

To examine the nature of one of the fixed points $(c, 0)$, denote

$$y = s - c \quad (1.1.7)$$

$$x = i \quad (1.1.8)$$

then

$$\frac{dy}{dt} = -\beta(y + c)x \quad (1.1.9)$$

$$\frac{dx}{dt} = \beta y(x + c - p) \quad (1.1.10)$$

where $\frac{\gamma}{\beta} = \rho$ the linearized equations at $(c, 0)$ are

$$\begin{pmatrix} \dot{y} \\ \dot{x} \end{pmatrix} = \begin{pmatrix} 0 & -\beta c \\ 0 & \beta(c - \rho) \end{pmatrix} \begin{pmatrix} y \\ x \end{pmatrix} \quad (1.1.11)$$

The eigenvalues are $\lambda_1 = 0, \lambda_2 = \beta(c - \rho)$. The solution in the neighborhood of $y = 0$ and $x = 0$ is therefore given by

$$y = k_1 + k_2 e^{\lambda_2 t} \quad (1.1.12)$$

$$x = -\frac{\lambda_2}{\beta c} k_2 e^{\lambda_2 t} \quad (1.1.13)$$

1.1.4 Special Orbits.

When $k_2 = 0, y = k_1$ and $x=0$. This is not an orbit. Consider a case when $k_1 = 0,$
 $y = k_1 + k_2 e^{\lambda_2 t} = -\frac{\lambda_2}{\beta c} k_2 e^{\lambda_2 t} = -\frac{\lambda_2}{\beta c} y = (\frac{c-\rho}{c})y$. This is a family of straight lines with
slope $-(\frac{c-\rho}{c})$ The peak value of i occurs when $i=0$, which happens when $s=p$. When $s > p$,
the straight-line trajectories have negative slopes, and when $c < p$, the slopes become
positive. The solution presented above is an approximation that provides insight into the
behavior of the system's trajectories. The equation

$$\frac{di}{ds} = \frac{\beta s - \gamma}{-\beta s} \quad (1.1.14)$$

is solved as follows

$$di = (-1 + \frac{\rho}{s}) ds \quad (1.1.15)$$

integrating this gives $i = -s + \rho \ln s + k$. At $t=0, s_1(0) = n = s_0$ and $i_2(0) = a$.
 $i = -s + \rho \ln(\frac{s}{s_0}) + n + a$. Since i represents a family of trajectories, its maximum value
occurs when $s=p$. Suppose an initial number of infected individuals $i(0)$ is introduced into

a population with s_0 susceptible individuals. If $S_0 > p$, the number of infected individuals will rise until the susceptible population falls below ρ , after which the infections will begin to decline. If $S_0 < p$, the number of infected individuals will steadily decrease to zero, meaning no epidemic occurs.

Thus, ρ acts as a threshold value of susceptibles necessary for an epidemic to take place (known as the threshold effect). When $i(0)=\infty$, the number of infected individuals must eventually drop to zero at some positive s value. This indicates that the epidemic ends due to a lack of infected individuals rather than a shortage of susceptibles, allowing some people to completely avoid the disease (referred to as the escape effect). In cases where $x_1(0) > \rho$ $x_1(0) - \rho$ is small, the epidemic curves tend to be nearly symmetrical around the point $x_1 = \rho$. This means that throughout the epidemic, the susceptible population ultimately decreases to a level roughly as far below the threshold ρ as it was initially above it (known as the symmetry effect).

1.2 Statement of the Problem

Frequent watery bowel movements in children of under five years poses a significant challenge mostly to developing countries where poor sanitation and faulty health system is a problem. The mortality rate caused by diarrhoea in under five year is quiet alarming mostly in sub-Saharan countries. Different mathematical scholars have developed different mathematical models on childhood diarrhoea trying to come up with possible solution like (Adewale, 2015, Iqbal *et al.*, 2022) but childhood diarrhoea remains a persistent public health concern despite previous modeling efforts. Bowel disease development can be some what impacted by stress at any time, whether it be in childhood or maturity. In addition to affecting housing conditions, food and other factors that raise the risk of exposure to infectious organisms or lower resistance to infectious diseases, low socioeconomic level can restrict access to health treatment. In our study we will show how social factor like stress and faulty healthy system significantly help fuels under five childhood diarrhoea.

1.3 Objectives

1.3.1 General Objective

The main objective of this research was to develop a mathematical model for the spread of childhood diarrhoea that incorporated social factor and weaknesses in the healthcare system in low income population.

1.3.2 Specific Objectives

The specific aims of this study were to:

- i. Develop a diarrhoea model with characteristics unique to low income population for children under five years.
- ii. Perform stability and sensitivity analysis of the results obtained.
- iii. Simulate the model under different scenarios to validate analytical outcomes.

1.4 Justification of the Study

Diarrhoea affects an estimated 1.7 billion children worldwide each year, and low-income nations account for a large share of diarrhoeal morbidity and mortality because of the health system's resource and infrastructure constraints (mills ,2014). Additionally, bacterial translocations and autoimmune alterations have been noted in depression (Ohlsson *et al.*, 2019). Therefore, there appears to be a shared connection between depression and bowel disorders (diarrhoea) that explains how the two conditions interact. A rising body of research in recent years has shown that bowel illness patients, both adults and children, have a higher prevalence of mental disorders than the general population (Mikocka *et al.*, 2016). This study has examined diarrhea in children under five and explore how stress and weaknesses in the healthcare system contribute to the spread of the disease mostly in kenyan low population settings. Moreover, the findings of this study support the goals of SDG 3.2, which seeks to lower the under-five mortality rate to 25 deaths per 1,000 live births by the year 2030 (Bora *et al.*, 2018).

1.5 Scope of the study

This research examined how social factors, especially stress and shortcomings in the healthcare system, affect the transmission of diarrhoea among young children. The aim was to

demonstrate how stress in children under five, combined with weaknesses in healthcare services, contributes to the spread of childhood diarrhoea in low income population. The study specifically focused on children under five in Kenya, exploring the role of stress and healthcare system challenges in influencing diarrhoea transmission.

1.6 Limitation of the study

In spite of the insights gained from modeling the interplay between social factors and the health system in the dynamics of childhood diarrhoea in Kenya, several limitations must be acknowledged. First, data limitations, including under reporting and inconsistent record-keeping, may have an impact on the precision of the model. Second, the model relies on assumptions that may oversimplify the complex interactions between health service delivery and social determinants. Third, while the study identifies correlations, it cannot fully establish causality between observed factors. Additionally, the diversity within Kenya's population and healthcare infrastructure limits the generalization of the findings to the national level. Lastly, certain cultural and behavioral factors influencing childhood health were not fully accounted for due to their qualitative nature and limited data availability.

1.7 Basic Assumptions

In addition to fundamental assumptions of mathematical epidemiological model, this study is based on the following assumptions:

- i. Children under five years with stress are more prone to severe infection as compared to those without stress but still infected with diarrhoea.
- ii. Faulty healthy system can propagate the enhancement of the disease as most of the patient may not receive the intended medical attention.
- iii. Faulty health system may involve problems in the health care like lack of enough personnel, drugs, lab equipment or even lack of resources by the poor children to make it to the health system.

1.8 Expected Output

By the end of this study, the following output are expected:

- i. One publications published in peer reviewed journal in order to add to the corpus of scientific knowledge, aid in the development of innovative diarrhoea control in-

intervention strategies, guide field and laboratory research, and provide stakeholders with pertinent information.

- ii. A deterministic model of diarrhoea in children under five that takes into account social influences and a dysfunctional healthcare system.

1.9 Operational Definition of Terms

Susceptible: Susceptible individuals may catch the infection if exposed to it(easily influenced or harmed by something).

Basic reproduction metric (R_0): It is the average number of secondary cases generated by a single primary case in a fully susceptible population.

SIR model: Susceptible-Infected-Recovered.

Mortality rate:It's the proportion of a population who die of defined cause.

Morbidity rate: It measures how often a disease or condition occurs within a population during a specific time period.

Faulty health system: It refer to a situation where a health care system is not functioning effectively leading to a negative consequences for patients and the overall health care infrastructure.

Social factor: these are characteristics of individuals or groups that influence thoughts behavior and outcomes within a social context.

Stress: it's a natural bodily response to challenges or demands often described as a feeling of emotional or physical tension.

CHAPTER TWO

LITERATURE REVIEW

2.1 Classical epidemiological models

In dynamic system modeling, a population of size N at time t , with a continuous influx of susceptible individuals at rates β and π , is divided into compartments based on disease status. These compartmental frameworks form the basis of most dynamic models for infectious diseases. Initially introduced by Kermack and McKendrick in 1927, these models were further developed by other biomathematicians in 1932. For viral infections such as chikungunya, swine flu, influenza, and measles where individuals gain immunity after recovery the SIR (Susceptible-Infected-Recovered) model is an appropriate modeling approach.

For bacterial infections such as gonorrhea, bubonic plague, tuberculosis, and syphilis, individuals who recover do not develop lasting immunity and can be reinfected. To model such diseases, the SIS (Susceptible-Infectious-Susceptible) framework is often employed. The concept of compartmental models in infectious disease dynamics was originally introduced by Kermack and McKendrick in 1927. Traditional epidemiological models categorize individuals based on their disease status typically as susceptible, infectious, recovered, immune, or exposed (but not yet infectious). The structure and number of compartments used in a model depend on the nature of the disease being studied and the desired level of detail. Variables in the system typically represent different population groups, while parameters reflect fundamental rates such as birth, transmission, and mortality, along with other adjustable constants that influence disease progression.

2.1.1 The SIS Model

Many epidemiological models are derived from this basic framework, which is one of the simplest compartmental structures. The model includes three key compartments: S for susceptible individuals, I for infectious individuals, and S again for those who recover without lasting immunity and return to the susceptible pool. The SIS model is especially applicable to diseases where reinfection is possible. It uses time dependent functions $S(t)$ for the number of susceptible and $I(t)$ for the number of infectious individuals to track how these populations evolve over time, assuming a constant total population. For infectious

diseases like measles, mumps, and rubella, where transmission occurs between people and recovery leads to long-term immunity, such models particularly the SIR model are highly effective at forecasting outbreaks (Moreno and Amira Yaazim, 2012). The SIR model's strength lies in its ability to dynamically represent disease progression through its time-based variables, which is particularly useful for understanding endemic diseases with short infectious periods. For instance, before the introduction of the measles vaccine in the UK in 1968, the susceptible population $S(t)$ would rise and fall over time, resulting in recurring outbreaks. As people moved from being susceptible to infected and then to the removed (immune or recovered) group, the number of susceptible would drop sharply. The disease could not reemerge until the susceptible group was replenished, typically through births adding new individuals to that compartment.

2.1.2 Approaches to mathematical disease modeling

Mathematical modeling of infectious diseases typically follows two main approaches. The first focuses on simulating how diseases spread over time through interactions between infectious and susceptible individuals. The second approach examines disease transmission at the cellular or molecular level within a single infected host. The earliest known epidemiological model was developed by Bernoulli in 1766, who used mathematical life table analysis to study the impact of smallpox variolation a precursor to vaccination on life expectancy (Dietz and Heesterbeek, 2000). Although Bernoulli's model addressed smallpox, modern deterministic models in epidemiology largely emerged in the 20th century, when the nonlinear dynamics of disease transmission began to be fully understood. At the start of the 20th century, researchers debated why epidemics often ended before all susceptible individuals were infected. Some hypothesized that changes in the pathogen's virulence played a role. In 1906, Hamer introduced a discrete time model to explain the cyclical nature of measles outbreaks. His model was the first to propose that disease incidence the number of new cases per unit time depends on the product of the densities of susceptible and infectious individuals. Later, in 1927, Kermack and McKendrick advanced the field significantly by publishing a series of papers on epidemic modeling. They introduced the concept of an epidemic threshold, showing that an outbreak can only occur if the density of susceptible individuals exceeds a critical level.

2.2 Model on Childhood diarrhoea stability and sensitivity analysis

Adewale *et al.*, 2015 Created a methodology for analyzing diarrhea mathematically when a vaccination is present. Their study considered a deterministic epidemic model (S, V, E, I) to better understand how vaccination and treatment of infected individuals influence the dynamic spread of diarrhoea within a population. The model's endemic and the system exhibits disease-free equilibrium points, according to the mathematical study. To sum up, vaccination is essential for preventing the spread of diarrheal illness. The number of susceptible people increases based on how well the vaccine works to prevent diarrhoea; the fewer susceptible people there are, the less the disease spreads; however, if the vaccine wears off quickly, the number of infected people increases. This implies that the vaccine should be administered as soon as possible after delivery, before the risk of contracting a diarrhoeal infection, and that it must be effective to reduce the disease's spread and associated expenses. The effectiveness of the vaccination should be taken into consideration to lessen the evolving spread of diarrhoea in a population, and the medications should be made available to customers at a significantly lower cost. But they failed to take into consideration in a Nairobi urban slum, children's diarrhoea is linked to soil consumption.

Bauza *et al.*, 2017 In a Nairobi, Kenya, urban slum, children's diarrhoea is linked to soil consumption. In a Nairobi, Kenya, urban slum, children's diarrhoea is linked to soil ingestion. In an urban slum setting, this study showed a link between children's diarrhoea and soil consumption. This contributes to the expanding body of research showing that current WASH initiatives that focus on increasing Hand washing, access to toilets or latrines, and water availability may not fully eliminate children's exposure to fecal contamination in the home. Children aged 6 to 24 months exhibited the highest levels of dirt ingestion, consistent with other studies on children's mouthing behavior and geophagy. In contrast, no soil ingestion was reported in children under six months old, likely due to their limited mobility at that age.

Compared to a previous study conducted in rural Kenya, which found a moderate correlation between soil ingestion and diarrhoea (Pearson's $r=0.306$, $r=0.306$), this study observed a stronger association (Pearson's $r=0.46$, $r=0.46$) between soil ingestion and diarrhoea in urban slum areas. This may suggest that soil ingestion poses a higher risk in urban slums,

although the relatively small sample sizes in both studies could influence the results. Interestingly, our research found a link between diarrhoea and dirt ingestion even in homes without earth floors (only one household in our sample had an earth floor). Exposure to soil is still likely since many homes in the slum are surrounded by soil, and children are often not confined indoors. However, the study did not incorporate a mathematical model to analyze the saturation incidence rate of diarrhoea.

Bonyah *et al.*, 2018 Developed an analytical mathematical model that incorporated a saturated incidence rate to study the transmission dynamics of diarrheal disease. Their analysis focused on the disease free equilibrium (E_0), the endemic equilibrium (E_1), and the basic reproduction number (R_0). They demonstrated both local and global stability of the disease-free equilibrium. Additionally, they showed that when $R_0 > 1$, the endemic equilibrium becomes locally asymptotically stable, suggesting that the disease will persist within the population. In contrast, if $R_0 < 1$, the disease will eventually die out. Numerical simulations, conducted using Python, further illustrated that the dynamics of diarrhoeal disease are influenced by the saturation level of treatment. The results indicated that increasing access to treatment significantly reduces disease prevalence in the community. The study emphasized the importance of widely available anti diarrheal medications such as loperamide (Imodium) and bismuth subsalicylate (Pepto-Bismol or Kaopectate) which are effective not only in treating diarrhea but also in preventing traveler's diarrhea caused by consuming contaminated food or water. To curb outbreaks and slow the spread of diarrhea, the researchers advised health policymakers to ensure these medications are affordable and readily accessible, aiming for a treatment coverage rate of 99%. However, the study did not explore the specific risk factors affecting children under five in Nairobi County's Kawangware slum.

Mutama *et al.*, 2019 Examined the risk factors associated with diarrhoeal illness in children under five living in the Kawangware slum of Nairobi County. It revealed that diarrhea is highly prevalent in this community, particularly among young children. Although infants aged 0 to 5 months exhibited notable infection rates, the highest incidence was recorded in the 6 to 11 month age group. Interviews with caregivers showed that many mothers introduced liquids and solid foods to their infants before the recommended age of six months. The study also identified a significant association between a child's gender and the likeli-

hood of contracting diarrhea. Girls were found to be more vulnerable to diarrheal infections compared to boys in the study population. Based on these findings, the researchers recommended that health promotion efforts focus more on educating caregivers about key preventive measures, including water treatment, handwashing with soap, safe food handling, and proper disposal of children's feces. They also highlighted the need for further research into behavioral risk factors for diarrhoea within this population and called for deeper exploration of cultural and traditional beliefs held by mothers and caregivers about disease prevention. Insights from such studies could help design targeted communication strategies that promote better hygiene, sanitation, and water treatment practices among caregivers of young children. However, this study did not examine water, sanitation, and hygiene (WASH) related risk factors for diarrhea among children under five in Nairobi County's Kasarani area.

Kimani *et al.*, 2019 Investigated the water, sanitation, and hygiene (WASH)-related risk factors for diarrheal illness among children under five in Kasarani, Nairobi County. The primary aim was to examine the link between childhood diarrhoea and WASH conditions. To capture variation across the area, Kasarani was divided into four zones based on population density and income levels: low-density high-income, medium-density middle-income, high-density low-income, and informal low-income settlements. Data collection was conducted through structured questionnaires and sanitation checklists. Statistical analyses, including chi-square tests and inferential methods, were used to explore associations between variables, with ANOVA applied to compare quantitative data. The study found that the quality of drinking water significantly influenced diarrhoea risk in children ($p = 0.019$). Notably, tap water was more contaminated with *T. coli* bacteria (13.7%) than water stored in household containers (7.2%). Daily household water consumption was also a strong predictor of diarrhoeal illness ($p = 0.001$). Other significant factors included the child's age ($p = 0.046$), water treatment methods ($p = 0.002$), how solid waste was stored ($p < 0.001$), and both the amount and frequency of water supply ($p < 0.001$). Overall, the study concluded that inadequate WASH conditions in Kasarani are closely linked to higher rates of diarrhea among young children. It highlighted the importance of further research into the role of solid waste storage practices and the impact of household income levels on diarrhea prevalence across different residential zones. However, the study did not incorpo-

rate a deterministic compartmental model to analyze the dynamics of dysentery or diarrhea.

Berhe *et al.*, 2019 Proposed a deterministic compartmental model for diarrhoeal disease. They showed that the disease-free equilibrium is globally stable. As established earlier, the global stability of this equilibrium depends entirely on the basic reproduction number (R_0), which serves as a critical threshold: if ($R_0 > 1$), the disease will eventually die out. Therefore, to eliminate the disease from the population, policymakers must aim to reduce R_0 to below one, to validate their model, the researchers applied it to replicate reported cases of dysentery-related diarrhoea in Ethiopia in 2017 and achieved a good fit. The optimization problem was tackled by solving a system of nonlinear differential equations using the Runge-Kutta method (ODE45), combined with the Trust-Region-Reflective algorithm and the lsqnonlin function to minimize the sum of squared residuals and estimate parameter values. Their results confirmed that the model successfully simulated the 2017 outbreak data. They estimated the basic reproduction number for dysentery transmission in Ethiopia to be $R_0 = 1.1208$, indicating that the disease had the potential to persist in the population.

Guillaume *et al.*, 2020 Explored the factors contributing to diarrhea prevalence among children under five in Mathare, an informal settlement in Nairobi, Kenya. The main goal was to identify key variables associated with diarrhoeal illness in this population. Using a cross-sectional survey approach, data were collected between July 1 and August 1, 2019, from primary caregivers (PCGs) of children under five living in Mathare. The area was deliberately selected due to its urban informal setting. Households and participants were randomly selected, and a p-value below 0.05 was considered statistically significant. A total of 324 primary caregivers were surveyed, each representing a household. Of the respondents, 56.17% were aged between 25 and 31. The overall prevalence of diarrhea among children was 18.7%. Several significant associations were identified between diarrhea occurrence and various household and caregiver factors, including the child's gender, the caregiver's relationship to the child, household size, and the number of children present ($p = 0.008$, $p < 0.001$, $p < 0.001$, and $p < 0.001$, respectively). Notably, 52.78% of caregivers had no formal education, and in 38.89% of households, children's feces were disposed of in garbage bins. Other factors that showed significant links to diarrhoea prevalence included the caregiver's education level ($p < 0.001$), residing in rental housing ($p = 0.024$), and

poor environmental hygiene—specifically the presence of flies, visible feces, and open garbage within or near the household compound (all $p < 0.001$). These findings highlight multiple contributors to the high rates of diarrhea in children under five in informal settlements. Globally, diarrhoea remains the second leading cause of death among children in this age group, with around 1.7 billion cases and approximately 525,000 deaths reported annually, according to the World Health Organization (WHO). In Kenya alone, 1,499,146 cases of diarrhoea in children under five were reported in 2018, with Nairobi accounting for 136,028 of those cases. A 2017 study further reported that 25.6% of children living in Nairobi’s informal settlements suffered from diarrhea, underscoring the significant public health burden faced by these communities.

Ikua *et al.*, 2020 A case study conducted in Korogocho, Nairobi County, Kenya, investigated environmental risk factors influencing the incidence of diarrhoea among children under five living in informal urban settlements. Their primary goal is to determine how environmental risk factors for childhood diarrhoea relate to its prevalence in Nairobi County’s Korogocho slum. The hypotheses that needed to be tested were that there was no meaningful connection between the Korogocho slum’s surroundings and outside risk factors and the occurrence of childhood diarrhoea in children under five. Accessibility, the kind and cleanliness of restroom facilities, and the sources and treatment of drinking water were among the study variables. However, for the sample group, sharing restrooms and having access to them did not significantly increase the risk of getting diarrhea. According to a two-week recall, 36.4% of mothers said that diarrhea had been experienced by their children within the age range. The study made a number of suggestions and recommended topics for more research based on the findings. The main suggestions are to institutionalize intentional actions to give slum inhabitants access to safe and efficient fecal waste disposal, as well as clean and high-quality drinking water. However, the Ef, Lf, Uf, and Cf fly models as well as the Sh, Ih, and Rh human models were not taken into consideration.

2.3 Numerical simulation of childhood diarrhoea

2.3.1 Ef,Lf,Uf, Cf flies model and Sh,Ih,Rh human model

Rahmadani *et al.*, 2020 Developed a dynamic model for diarrhoea epidemiology and simulation that takes into account several disease carriers. The human and fly compartments were included in their model. A collection of mathematical models based on differential

equations that forecast potential future outcomes for the efficacy of disease control measures that aim to reduce the number of sick people as well as the number of flies, which are the primary disease carriers. The human population is represented by $N = S_h, I_h, R_h$, and the house fly population is represented by $N_f = E_f, L_f, U_f, C_f$. To determine the link and condition of flies and people during disease outbreaks, mathematical studies and numerical simulation were conducted. In order to achieve the ideal situation, each control approach was then used and integrated in various ways. Because it reduced the disease's spread, Scenario X, which incorporates all control strategies, was found to be the most successful approach in lowering the number of flies and the afflicted population under a particular simulated scenario. Further research on more general topics, such as how temperature affects disease transmission, is necessary in the future because housefly populations peak at higher temperatures. Taking into account numerous carriers, epidemiological dynamics, and pertinent characteristics, the suggested framework is regarded as an efficient disease control method. Nevertheless, they neglected to take into account the use of sensitivity analysis in the Analytical modeling of diarrhoea in the context of vaccination and therapeutic interventions.

Akinola *et al.*, 2021 Used sensitivity analysis to examine a model on the mathematical modeling of diarrhea dynamics with vaccination and periodic treatment interventions. The diarrhoea model in this work was created using fundamental mathematical modeling approaches, which resulted in a five compartment model represented by a system of ordinary differential equations (ODEs). The region in which the model is epidemiologically viable is then identified by analyzing the model's presence and uniqueness. The reproduction number was also used to determine the model's equilibrium points and the stability of the disease-free state. After that, we conducted a global sensitivity study on the reproduction number in relation to each of its factors, and we discovered that four (4) of them were sensitive. They were able to demonstrate that the approach that encourages susceptible people to get vaccinated should be prioritized when treating diarrhea because it lessens the disease's dynamic spread more effectively in the susceptible population than in the unvaccinated population. To ensure a diarrhea-free environment, medical professionals and policymakers should also monitor parameters that raise the fundamental reproduction number R_0 . The use of a fractal fractional operator in analyzing the fractional order diarrhea

model was not explored.

Yao *et al.*, 2022 Used a fractional operator model was developed to study diarrhoea dynamics using a fractional-order approach. The model was structured around four compartments: Susceptible (S), Infected (I), Treated (T), and Recovered (R), representing the progression of individuals affected by diarrhoeal illness. The system was analyzed using the fractal–fractional derivative operator (FFO) with an extended Mittag–Leffler kernel to solve the proposed equations. The researchers conducted a qualitative analysis and confirmed the existence of a unique, non-negative solution to the model. They used fixed-point theory to prove the existence and uniqueness of the solution under the Atangana–Baleanu fractal–fractional operator. An error analysis was also carried out to evaluate the model’s accuracy. To understand the behavior of the diarrhea outbreak and support potential control strategies, numerical methods tailored for fractional-order systems were applied. Simulations with different fractional orders and dimensions revealed notable variations in outcomes. However, while modeling the dynamics of acute diarrhoeal infection, the study did not incorporate critical aspects such as best practices for disease control or the influence of poor sanitation.

Lasisi *et al.*, 2020 Created a model to simulate an acute diarrhoea infection, taking into account the effects of inadequate sanitation and optimum control. The study analyzed two models and proposed an improved framework for understanding the transmission dynamics of acute diarrhoeal infections. The first model assessed the impact of adherence to good hygiene practices on infection rates. The second, an optimal control model, incorporated two intervention strategies: one aimed at increasing the number of individuals receiving treatment, and the other focused on reducing contact between susceptible individuals and infectious agents. The analysis showed that the endemic equilibrium is globally asymptotically stable when $R_0 > 0$, while the disease-free equilibrium is globally asymptotically stable if $R_0 \leq 0$. The control strategy, defined by $\alpha_1 \neq \alpha_2 \neq 0$ and $\alpha_1 = \alpha_2 = 0$, demonstrated that a combined prevention and treatment approach significantly reduces the basic reproduction number, as shown graphically, and could be effective in controlling or eradicating acute diarrhoeal infections. However, the study did not consider the development

of a modified Atangana–Baleanu–Caputo (pmABC) order model.

Affandi *et al.*, 2021 Explored a model for the best possible management of diarrhoeal illness using immunization and therapy. A deterministic epidemic model (SIR-VT) was taken into consideration for their investigation in order to better understand how vaccination and infected person treatment affect the progression of diarrhea outbreaks in a community. Vaccines play a crucial role in preventing the spread of diarrhoeal disease. The number of susceptible people increases in proportion to the effectiveness of the vaccine; the lower the vaccine, the fewer susceptible people and the slower the disease spreads. However, if the vaccine drops off rapidly, the number of infected people increases. This implies that the vaccine should be administered as soon as possible after delivery, before the risk of contracting a diarrheal illness, and that it must be effective to reduce the disease's spread and associated expenses. Nevertheless, the analysis of the fractional diarrhea model incorporating the Mittag-Leffler kernel was not considered.

Iqbal *et al.*, 2022 Developed a fractional-order diarrhea model incorporating the Mittag-Leffler kernel was developed to enable deeper analysis, replacing the traditional infectious diarrhoea model. In this framework, $T(t)$ denotes the treated population, $R(t)$ the recovered group, $S(t)$ the susceptible individuals most likely to contract the disease, $I(t)$ the infected population, and $N(t)$ the total population, such that

$$N(t)=S(t)+I(t)+T(t)+R(t).$$

The model's equations were transformed into fractional form using the Atangana-Baleanu fractional differential operators in the Caputo sense. After this conversion, the model was expanded and solved employing the Mittag-Leffler kernel approach. Prior to solving, the existence and uniqueness of the solution were established. Numerical simulations were conducted and visualized to assess the model's behavior. The graphs illustrated convergence toward a steady state at rates dependent on the fractional derivative order ν ; specifically, the convergence speed is directly proportional to ν . Both the endemic and disease-free steady states were shown to be stable locally and globally. The analysis of the basic reproduction number (R_0) demonstrated its critical role in disease dynamics and stability of equilibrium points. Stability both local and global is achieved when $R_0 < 1$, indicating disease elimination akin to an endemic scenario. This modeling technique offers potential

for addressing and modifying other nonlinear systems in the future. However, the study did not explore strategies for optimal management of diarrhoeal disease through vaccination and treatment, nor did it include the development of a modified ABC order (pmABC) model.

Madani *et al.*, 2025 By employing a modified ABC order (pmABC) model, they were able to investigate the crossover effect and pinpoint efficient management methods that were suited to the difficulties encountered in some communities with potentially restricted access to resources. Their results emphasize the need of personalized treatment and reducing contact between vulnerable and infected people as crucial disease control measures. Additionally, they discovered that the diarrhoea model's DFE was globally asymptotically stable inside the feasible area Ω for $R_0 \leq 1$ by determining the fundamental reproduction number and determining the DFE's local and worldwide stability. According to their findings, a larger reproduction number is correlated with an increase in the effective contact rate, highlighting the significance of preventative measures in lowering the spread of illness. Additionally, their data showed that by lowering the contact rate, increasing adherence to proper hygiene measures might result in fewer reproductions. It was demonstrated that the use of tactics pertaining to the controls α_1 and α_2 was highly successful in halting the spread of the diarrheal illness. The fractional pmABC derivative, they deduced from the simulations, is an effective tool for illustrating abrupt or rapid changes in the prevalence of diarrhoeal sickness, enabling a closer match to known disease patterns. These results indicated that the best strategy for managing acute diarrhea outbreaks in environments with limited resources is a mix of preventative actions, such as enhancing sanitation and hygiene, and the focused treatment of affected patients. In order to enhance local public health initiatives, future research might build on this work by adding other elements including regional heterogeneity, the effects of climate change, and the role of particular bacterial strains. But they failed to take into consideration Using the Mittag-Leffler kernel to analyze the fractional diarrhoea model.

CHAPTER THREE

METHODOLOGY

3.1 Introduction

This chapter begins by reviewing traditional epidemiological models previously employed to simulate diarrhea dynamics, laying the foundation for the proposed model. A deterministic model, grounded in the standard SIS framework, was developed to assess how social factors and deficiencies in the healthcare system influence the progression of childhood diarrhoea in Kenya. The chapter first outlines the model framework, drawing from established classical models, and then details the assumptions, variables, and parameters involved. By the chapter's conclusion, a system of ordinary differential equations derived from this framework is presented.

3.1.1 Model Formulation

The model, adapted from the classical SIS framework, describes how social factors and deficiencies in the healthcare system affect the dynamics of childhood diarrhea in Nyeri County, Kenya. The variables include T_C , representing diarrhea treatment for children under five in a faulty healthcare system; T_K , treatment in a functioning healthcare system; I_S , stress-related diarrhoea infections among children under five; I_N , diarrhea cases in unstressed children under five; and S, the susceptible children. A key feature of the model is that the force of infection λ arises from mass mixing within the population. The basic reproduction number for this infectious disease compartmental model was derived using the next-generation matrix method, which is commonly applied in population dynamics to calculate this fundamental threshold in structured population models.

3.1.2 Model Assumptions

- i. Since therapy was thought to greatly reduce the transmission and death rate of childhood diarrhea, we assumed that those receiving treatment have reduced infectivity and mortality rates.
- ii. Diarrhea was equally likely to affect all under five children.
- iii. It was believed that the population of children was homogeneous.

- iv. The force of infection was higher in children who are stressed and have diarrhea than in those who are only sick and attending therapy.
- v Treatment of under five with stress in normal health facility increases the recovery rate.

Table 1: Model variables

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 2: Model parameters

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

3.2 Model Flow Chart

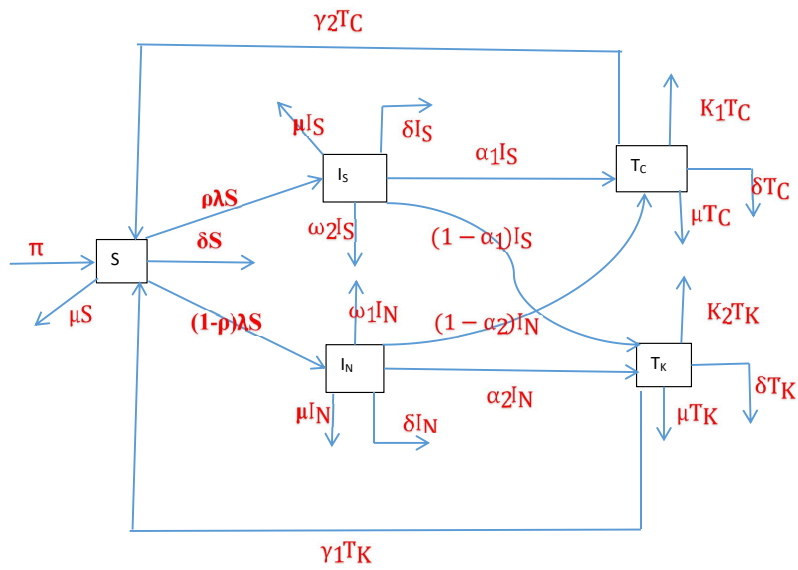


Figure 1: Flow chart for the dynamic of childhood diarrhoea with social factor and faulty health system

3.2.1 Model Equations

$$\frac{dS}{dt} = \pi + \gamma_1 T_K + \gamma_2 T_C - \mu S - \delta S - \rho \lambda S - (1 - \rho) \lambda S, \quad (3.2.1)$$

$$\frac{dI_S}{dt} = \rho \lambda S - \mu I_S - \rho I_S - \omega_2 I_S - \alpha_1 I_S - (1 - \alpha_1) I_S, \quad (3.2.2)$$

$$\frac{dI_N}{dt} = (1 - \rho) \lambda S - \mu I_N - \omega_1 I_N - \delta I_N - \alpha_2 I_N - (1 - \alpha_2) I_N, \quad (3.2.3)$$

$$\frac{dT_C}{dt} = \alpha_1 I_S - \gamma_2 T_C - K_1 T_C - \mu T_C - \delta T_C + (1 - \alpha_2) I_N, \quad (3.2.4)$$

$$\frac{dT_K}{dt} = \alpha_2 I_N + (1 - \alpha_1) I_S - K_2 T_K - \mu T_K - \delta T_K - \gamma_1 T_K. \quad (3.2.5)$$

Where the force of infection is given by;

$$\lambda = \frac{\beta(I_S + \eta_1 I_N + \eta_2 T_C + \eta_3 T_K)}{N} \quad (3.2.6)$$

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 $\eta_1 \geq \eta_2 \geq \eta_3$.

3.3 Model Analysis

3.3.1 Basic reproduction number R_0

A key parameter influencing the model's behavior is the basic reproduction number. In epidemiology, assessing the potential severity of current or future infectious disease outbreaks heavily relies on this measure. Calculating the basic reproduction number, R_0 , which indicates the probability that an infection will result in an epidemic, is a common practice. As noted by May *et al.*, (2000), R_0 represents the average number of secondary cases caused by one infected individual in a completely susceptible population. In this study, R_0 was determined using the next-generation matrix method, which involves analyzing the Jacobian matrix of the system evaluated at the disease-free equilibrium (D.F.E.). The eigenvalues of this matrix were then computed using mathematical software.

3.4 Numerical simulation

MATLAB software with inbuilt ODE solver based on Runge Kutta order five will be used.

3.5 Ethical Considerations

All contributions to the research were properly acknowledged and given credit. Plagiarism and other false claims about study findings were avoided, and recommendations were kept discreet and informed. Before commencing any research, approval had to be obtained from the National Commission for Science, Technology, and Innovation (NACOSTI). The study's findings were thoroughly and critically examined to ensure their credibility and that they were reported honestly and ethically. The results were transparently released to aid in the growth of knowledge in applied mathematics fields.

CHAPTER FOUR

RESULTS AND DISCUSSIONS

4.1 Overview

This chapter conducted an epidemiological analysis of the existence and positivity of the state variables. A theorem was presented to establish the conditions for positivity and boundedness. The system's threshold parameter, including the basic reproduction number and the disease-free equilibrium point, was identified. Subsequently, the local and global stability of the disease-free equilibrium was examined. The existence of an endemic equilibrium was also explored, and this analysis helped determine the threshold conditions that govern whether the infection persists or is eradicated..

4.1.1 Existence and positive in variance

Theorem 1: Analytical results of the model equations (3.2.1-3.2.5) together with the initial conditions $S(0) \geq 0, I_S \geq 0, I_N \geq 0, T_K \geq 0$ the variables of the model are always non-negative. S, I_S, I_N, T_C, T_K maintain positivity for all t and continue to lie within \mathbb{R}_5^+

proof: To facilitate analysis

$$\frac{dS}{dt} = \pi + \gamma_1 T_K + \gamma_2 T_C - \Omega_1 S \quad (4.1.1)$$

$$\frac{dI_S}{dt} = \rho \lambda S - \Omega_2 I_S \quad (4.1.2)$$

$$\frac{dI_N}{dt} = (1 - \rho) \lambda S - \Omega_3 I_N \quad (4.1.3)$$

$$\frac{dT_C}{dt} = \alpha_1 I_S + (1 - \alpha_2) I_N - \Omega_4 T_C \quad (4.1.4)$$

$$\frac{dT_K}{dt} = \alpha_2 I_N + (1 - \alpha_1) I_S - \Omega_5 T_K \quad (4.1.5)$$

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 $\mathbf{W} = (s(t), i_s(t), i_n(t), t_c(t), t_k(t))^T$ and $\mathbf{F}(\mathbf{W}) = (F_1(W), F_2(W), F_3(W), F_4(W), F_5(W))^T$, where $F_1(\mathbf{W}) = \pi + \gamma_1 T_K + \gamma_2 T_C - \Omega_1 S$, $F_2(\mathbf{W}) = \rho \lambda S - \Omega_2 I_S$, $F_3(\mathbf{W}) = (1 - \rho) \lambda S - \Omega_3 I_N$, $F_4(\mathbf{W}) = \alpha_1 I_S - \Omega_4 T_C$, $F_5(\mathbf{W}) = \alpha_2 I_N + (1 - \alpha_1) I_S - \Omega_5 T_K$. Then, system (3.2.1-3.2.5)

can be written as $\frac{dQ}{dt} = F(W)$ where $F: C_+ \rightarrow (\mathbb{R}_+^5)$ with $W(0) = W_0 \in (R)_+^5$. Hence, the function W exhibits local Lipschitz continuity and is fully stable on \mathbb{R}_+^5 . Thus, the system's solution with non-negative initial conditions is both existent and unique. Moreover, it is clear that these solutions persist for all $t > 0$ and remain non-negative, ensuring that the region. \mathbb{R}_+^5 is an invariant domain of the system (Belew *et al.*, 2022).

4.1.2 Boundedness of the solution

Theorem 2: The non-negative solutions of the model's system of equations (3.2.1-3.2.5) Remain within limits, meaning the model variables S, I_S, I_N, T_C and T_K are bound for all t (Gurmu *et al.*, 2019). *Proof:*

$$N(t) = S(t) + I_S(t) + I_N(t) + T_C(t) + T_K(t) \quad (4.1.6)$$

By differentiating equation (4.1.6) gives,

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI_S}{dt} + \frac{dI_N}{dt} + \frac{dT_C}{dt} + \frac{dT_K}{dt} \quad (4.1.7)$$

$$\frac{dS}{dt} = \pi + \gamma_1 T_K + \gamma_2 T_C - \mu S - \delta S - \rho \lambda S - (1 - \rho) \lambda S \quad (4.1.8)$$

$$(4.1.9)$$

In the absence of mortality due to diarrhea illness equation (4.1.9) becomes

$$\frac{dS}{dt} = \pi - (\delta + \mu)S. \quad (4.1.10)$$

We integrate inequality (4.1.10) by the method of separation of variables and then apply initial conditions to obtain

$$N \leq \frac{\pi}{\delta + \mu} + (N_0 - \frac{\pi}{\delta + \mu})e^{-(\delta + \mu)t} \quad (4.1.11)$$

We analyze the behavior of N in inequality (4.1.11) 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 (4.1.11) experiences the largest possible value of N_0 . Thus $N \leq N_0, \forall t > 0$. Next, we consider

$N_0 < \frac{\pi}{\delta+\mu}$) so that the largest possible value of the right-hand side of inequality (4.1.11) approaches $\frac{\pi}{\delta+\mu}$ as $t \rightarrow \infty$ 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 (3.2.1-3.2.5) 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 .

4.1.3 Disease-free equilibrium point (DFE)

To determine the disease-free equilibrium point, we set the left-hand side of (3.2.1-3.2.5) 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 (3.2.1-3.2.5) will consist of one compartment (the susceptible class). At this equilibrium point, the disease is eradicated from the population.

Table 3: Parameter values and initial conditions for the model derived from Kenya

Parameters	values	Source
π	1000	Estimated
μ	0.063	bonyahet <i>al.</i> ,2018
γ_1	0.0012795	Gitonga, 2017
γ_2	0.0009403	Gitonga, 2017
δ	0.0006088	Gitonga, 2017
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	Kigenet <i>al.</i> ,2021
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

4.1.4 Effective reproduction number (R_{eff})

We compute the effective reproduction number (R_{eff}) defined as the average number of secondary infections caused by one infectious individual in a population where control measures are implemented (Ega *et al.*, 2015). To derive it, we employ the method used by the authors in (Gitonga, 2017). We rewrite the model equation (3.2.1-3.2.5) starting with newly infective classes as

$$\frac{dI_S}{dt} = \rho\lambda S - (\mu + \rho + \omega_2 + 1)I_S \quad (4.1.12)$$

$$\frac{dI_N}{dt} = (1 - \rho)\lambda S - (\mu + \omega_1 + \delta + 1)I_N \quad (4.1.13)$$

$$\frac{dT_C}{dt} = \alpha_1 I_S - (\gamma_2 + K_1 + \mu + \delta + (1 - \alpha_2))I_N \quad (4.1.14)$$

$$\frac{dT_K}{dt} = \alpha_2 I_N + (1 - \alpha_1)I_S - (K_2 + \mu + \delta + \gamma_1)T_K \quad (4.1.15)$$

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

$$f = \begin{pmatrix} \rho\lambda S \\ (1-\rho)\lambda S \\ 0 \\ 0 \end{pmatrix} \quad (4.1.16)$$

$$v = \begin{pmatrix} \Omega_2 I_S \\ \Omega_3 I_N \\ -\alpha_1 I_S + \Omega_4 T_C \\ -\alpha_2 I_N + (-1 + \alpha_1) I_S + \Omega_5 T_K \end{pmatrix} \quad (4.1.17)$$

We derive the matrices F and V by computing the Jacobian matrices of f and v , respectively, evaluated at the disease-free equilibrium (DFE), resulting in:

$$F = \begin{pmatrix} \rho\beta & \rho\beta\eta_1 & \rho\beta\eta_2 & \rho\beta\eta_3 \\ (1-\rho)\beta & (1-\rho)\beta\eta_1 & (1-\rho)\beta\eta_2 & (1-\rho)\beta\eta_3 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \quad (4.1.18)$$

$$V = \begin{pmatrix} \Omega_2 & 0 & 0 & 0 \\ 0 & \Omega_3 & 0 & 0 \\ -\alpha_1 & 0 & \Omega_4 & 0 \\ (-1 + \alpha_1) & -\alpha_2 & 0 & \Omega_5 \end{pmatrix} \quad (4.1.19)$$

$$FV^{-1} = \begin{pmatrix} \frac{\beta\rho}{\Omega_2} + \frac{\beta\rho\alpha_1\eta_2}{\Omega_2\Omega_3} + \frac{\beta\rho\eta_3(\Omega_3\Omega_4 - \alpha_2\Omega_3\Omega_4)}{\Omega_2\Omega_3\Omega_4\Omega_5}, \frac{\beta\rho\eta_1}{\Omega_3} + \frac{\beta\rho\alpha_2\eta_3}{\Omega_3\Omega_5}, \frac{\beta\rho\eta_2}{\Omega_4}, \frac{\beta\eta_3\rho}{\Omega_5}, \frac{\beta(1-\rho)}{\Omega_2} \\ + \frac{\beta(1-\rho)\alpha_1\eta_2}{\Omega_2\Omega_4} + \frac{\beta(1-\rho)\eta_3(\Omega_3\Omega_4 - \alpha_2\Omega_3\Omega_4)}{\Omega_2\Omega_3\Omega_4\Omega_5}, \frac{\beta(1-\rho)\eta_1}{\Omega_3} + \frac{\beta(1-\rho)\alpha_2\eta_3}{\Omega_3\Omega_5}, \frac{\beta(1-\rho)\eta_2}{\Omega_4}, \frac{\beta(1-\rho)\eta_3}{\Omega_5}, \\ (0, 0, 0, 0), (0, 0, 0, 0) \end{pmatrix} \quad (4.1.20)$$

Finding the eigen values of FV^1 we obtain; $X_1 = 0$

$$X_2 = 0$$

$$X_3 = 0$$

$$X_4 = R_{eff} = \frac{\Omega_2\Omega_3\Omega_4\Omega_5}{-\alpha_2\eta_3\Omega_2\Omega_4 + \rho\eta_3\alpha_2\Omega_2\Omega_4 - \rho\eta_3\Omega_3\Omega_4 + \rho\alpha_2\eta_3\Omega_3\Omega_5 - \eta_1\Omega_2\Omega_4\Omega_5 + \rho\eta_1\Omega_2\Omega_4\Omega_5 - \rho\Omega_3\Omega_4\Omega_2\Omega_5}$$

The effective reproduction number is therefore defined as the spectral radius—that is, the largest (dominant) eigenvalue—of the matrix FV^1 denoted by ςFV^1 , hence we have

$$R_0 = -\frac{\Omega_2\Omega_3\Omega_4\Omega_5}{\alpha_2\eta_3\Omega_2\Omega_4 + \rho\eta_3\alpha_2\Omega_2\Omega_4 - \rho\eta_3\Omega_3\Omega_4 + \rho\alpha_2\eta_3\Omega_3\Omega_5 - \eta_1\Omega_2\Omega_4\Omega_5 + \rho\eta_1\Omega_2\Omega_4\Omega_5 - \rho\Omega_3\Omega_4\Omega_2\Omega_5}$$

4.1.5 Existence of the Endemic Equilibrium Point (EE) of the Model

Theorem 4.1.1. *A positive endemic equilibrium point of the model exists if $R_{eff} > 1$*

Proof. We utilize the technique proposed by the authors in this section (Ngari *et al.*, 2020) ■

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 (3.2.1-3.2.5) to zero. Thus, we have

$$0 = \pi + \gamma_1 T_K + \gamma_2 T_C - \Omega_1 S \quad (4.1.21)$$

$$0 = \rho \lambda S - \Omega_2 I_S \quad (4.1.22)$$

$$0 = (1 - \rho) \lambda S - \Omega_3 I_N \quad (4.1.23)$$

$$0 = \alpha_1 I_S - \Omega_4 T_C \quad (4.1.24)$$

$$0 = \alpha_2 I_N + (1 - \alpha_1) I_S - \Omega_5 T_K \quad (4.1.25)$$

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

$$\lambda^* = \frac{\beta(I_S^* + \eta_1 I_N^* + \eta_2 T_C^* + \eta_3 T_K^*)}{N} \quad (4.1.26)$$

We simplify system (4.1.21 - 4.1.25)

$$I_S = \frac{\rho\lambda S}{\Omega_2} \quad (4.1.27)$$

$$I_N = \frac{(1-\rho)\lambda S}{\Omega_3} \quad (4.1.28)$$

$$T_C = \frac{\alpha_1 I_S}{\Omega_4} \quad (4.1.29)$$

$$T_K = \frac{\alpha_2 I_N + (1-\alpha_2)I_S}{\Omega_5} \quad (4.1.30)$$

We substitute (4.1.27-4.1.30) into equation (4.1.26) in terms of λ^* obtain the following cases: Case 1: $\lambda_1^* = 0$, 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 $R_{eff} > 1$.

4.1.6 The Local Stability of DFE

To establish the local stability of the disease-free equilibrium, we present and prove the following theorem.

Theorem 4.1.2. *The system is locally asymptotically stable around the disease-free equilibrium stable if $R_{eff} < 1$ and unstable if $R_{eff} > 1$*

Proof. We use the technique applied by the authors in [Ngari *et al.*,2020]. We first rewrite the model equation (3.2.1-3.2.5) as ■

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

$$F_1 = \pi + \gamma_1 T_K + \gamma_2 T_C - (\mu + \delta)S - \lambda S \quad (4.1.31)$$

$$F_2 = \rho\lambda S - \Omega_1 I_S \quad (4.1.32)$$

$$F_3 = (1 - \rho)\lambda S - \Omega_2 I_N \quad (4.1.33)$$

$$F_4 = \alpha_1 I_S - \Omega_3 T_C + (1 - \alpha_2) I_N \quad (4.1.34)$$

$$F_5 = \alpha_2 I_N + (1 - \alpha_1) I_S - \Omega_4 T_K \quad (4.1.35)$$

$$J(E^0) = \begin{pmatrix} -\mu - \delta & -\beta & -\eta_1\beta & -\eta_2\beta + \gamma_2 & -\eta_3\beta + \gamma_1 \\ 0 & \rho\beta - \Omega_1 & \rho\eta_1\beta & \rho\eta_2\beta & \rho\eta_3\beta \\ 0 & (1 - \rho)\beta & (1 - \rho)\eta_1\beta & (1 - \rho)\eta_2\beta & (1 - \rho)\eta_3\beta \\ 0 & \alpha_1 & (1 - \alpha_2) & -\Omega_3 & 0 \\ 0 & (1 - \alpha_1) & \alpha_2 & 0 & -\Omega_4 \end{pmatrix} \quad (4.1.36)$$

We will use the Routh-Hurwitz criterion to determine the signs of the remaining eigenvalues. The characteristic equation $|A - X_i I| = 0$ with $i=1,2,3,4,5$. Based on the Routh-Hurwitz criterion, the signs of the eigenvalues' real parts for a cubic polynomial are determined as follows; $\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3$ 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 eigenvalues are provided in the appendix.

4.1.7 The Global Stability of DFE.

The global stability of the disease-free equilibrium point is analyzed using the approach implemented by (Bery *et al.*,2016). We first rewrite our model equation (3.2.1-3.2.5) in the form

$$\frac{dX}{dt} = H(X, Z), \frac{dZ}{dt} = G(X, Z), G(X, 0) = 0 \quad (4.1.37)$$

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 (4.1.37) 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 $A = D_Z G(X^*, 0)$ Matrix A is a Metzler (M) matrix, meaning its off-diagonal elements are non-negative, and Ω represents the region where the model is biologically feasible. If equation (4.1.37) meets conditions (i) and (ii), then the subsequent theorem applies.

Theorem 4.1.3. *The disease-free equilibrium $E^* = (X^*, 0)$ is a globally asymptotically stable equilibrium point of (4.1.36) provided that $R_{eff} < 1$ and conditions i and ii are met.*

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

$$\frac{dX}{dt} = H(X, 0) = [\pi - (\delta + \mu)]. \quad (4.1.38)$$

From equation (4.1.38), we obtain the following ordinary differential equation:

$$\frac{dS}{dt} = \pi - (\delta + \mu). \quad (4.1.39)$$

Solving equation (4.1.39) and applying initial condition $S_0 = S_0$ at $t=0$, we obtain

$$S = \frac{\pi}{(\delta + \mu)} + (S^0 - \frac{\pi}{\delta + \mu})e^{-(\delta+\mu)t} \quad (4.1.40)$$

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

$$\frac{dZ}{dt} = G(X, Z) = \begin{pmatrix} \rho\lambda S - \Omega_1 I_S \\ (1 - \rho)\lambda S - \Omega_2 I_N \\ \alpha_1 I_S - \Omega_3 T_C + (1 - \alpha_2) I_N \\ \alpha_2 I_N + (1 - \alpha_1) I_S - \Omega_4 T_K \end{pmatrix} \quad (4.1.41)$$

From vector (4.1.41), we have the Metzler matrix A given as

$$A = \begin{pmatrix} \beta - \Omega_1 & \eta_1\beta & \eta_2\beta & \eta_3\beta \\ (1 - \rho)\beta & (1 - \rho)\eta_1\beta - \Omega_2 & (1 - \rho)\eta_2\beta & (1 - \rho)\eta_3\beta \\ \alpha_1 & (1 - \alpha_2) & -\Omega_3 & 0 \\ (1 - \alpha_1) & \alpha_2 & 0 & -\Omega_4 \end{pmatrix} \quad (4.1.42)$$

Multiplying A and Z we get that;

$$\widehat{G}(X, Z) = \begin{pmatrix} (S^0 - S)(\beta\rho(I_S, I_N, T_C, T_K)) \\ (S^0 - S)(1 - \rho)(I_S, I_N, T_C, T_K) \\ 0 \\ 0 \end{pmatrix} \quad (4.1.43)$$

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.

4.1.8 Local Stability of the EE

In this scenario, linearizing the system at the endemic equilibrium point proves to be mathematically complex. Therefore, we assess the local stability of the endemic equilibrium using the approach described in [4.1.21]. This method is also employed to examine the presence of forward and backward bifurcations.

Theorem 4.1.4. *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 (3.2.1-3.2.5) 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:

$$\frac{dx_1}{dt} = f_1 = \pi + \gamma_1 T_K + \gamma_2 T_C - \mu S - \delta S - \rho \lambda S - (1 - \rho) \lambda S \quad (4.1.44)$$

$$\frac{dx_2}{dt} = f_2 = \rho \lambda S - \mu I_S - \rho I_S - \omega_2 I_S - \alpha_1 I_S - (1 - \alpha_1) I_S \quad (4.1.45)$$

$$\frac{dx_3}{dt} = f_3 = (1 - \rho) \lambda S - \mu I_N - \omega_1 I_N - \delta I_N - \alpha_2 I_N - (1 - \alpha_2) I_N \quad (4.1.46)$$

$$\frac{dx_4}{dt} = f_4 = \alpha_1 I_S - \gamma_2 T_C - K_1 T_C - \mu T_C - \delta T_C + (1 - \alpha_2) I_N \quad (4.1.47)$$

$$\frac{dx_5}{dt} = f_5 = \alpha_2 I_N + (1 - \alpha_1) I_S - K_2 T_K - \mu T_K - \delta T_K - \gamma_1 T_K \quad (4.1.48)$$

with

$$\lambda = \frac{\beta(I_S + \eta_1 I_N + \eta_2 T_C + \eta_3 T_K)}{N} \quad (4.1.49)$$

The Jacobian of system (4.1.44-4.1.48) at disease-free equilibrium point with $\beta = \beta^*$ is obtained as

$$J(E^0, \beta^*) = \begin{pmatrix} -\mu - \delta & -\beta & -\eta_1 \beta & -\eta_2 \beta + \gamma_2 & -\eta_3 \beta + \gamma_1 \\ 0 & \rho \beta - \Omega_1 & \rho \eta_1 \beta & \rho \eta_2 \beta & \rho \eta_3 \beta \\ 0 & (1 - \rho) \beta & (1 - \rho) \eta_1 \beta & (1 - \rho) \eta_2 \beta & (1 - \rho) \eta_3 \beta \\ 0 & \alpha_1 & (1 - \alpha_2) & -\Omega_3 & 0 \\ 0 & (1 - \alpha_1) & \alpha_2 & 0 & -\Omega_4 \end{pmatrix} \quad (4.1.50)$$

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

$$w_1 = \frac{-\eta_1\beta w_2 + (-\eta_2\beta + \gamma_2) + (-\eta_3\beta + \gamma_1)w_4}{\mu + \delta} \quad (4.1.51)$$

$$w_2 = \frac{-(\rho\eta_1\beta w_3 + \rho\eta_2 w_4 + \rho\eta_3\beta w_5)}{\rho\beta - \Omega_1} \quad (4.1.52)$$

$$w_3 = \frac{-(1 - \rho)\beta w_2 - (1 - \rho)\eta_2\beta w_4 - (1 - \rho)\eta_3\beta w_5}{(1 - \rho)\beta} \quad (4.1.53)$$

$$w_4 = \frac{\alpha_1 w_2 + (1 - \alpha_2)w_3}{\Omega_3} \quad (4.1.54)$$

$$w_5 = \frac{(1 - \alpha_1)w_2 + \alpha_2 w_3}{\Omega_4} \quad (4.1.55)$$

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

$$J(E^0, \beta^*) = \begin{pmatrix} -\mu - \delta & 0 & 0 & 0 & 0 \\ -\beta & \rho\beta - \Omega_1 & (1 - \rho)\beta & \alpha_1 & (1 - \alpha_1) \\ -\eta_1\beta + \gamma_2 & \rho\eta_1\beta & (1 - \rho)\eta_1\beta & (1 - \alpha_2) & \alpha_2 \\ -\eta_2\beta + \gamma_2 & \rho\eta_2\beta & (1 - \rho)\eta_2\beta & -\Omega_3 & 0 \\ -\eta_3\beta + \gamma_1 & \rho\eta_3\beta & (1 - \rho)\eta_3\beta & 0 & -\Omega_4 \end{pmatrix} \quad (4.1.56)$$

$$v_1 = 0 \quad (4.1.57)$$

$$v_2 = -\frac{(1 - \rho)\beta v_3 + \alpha v_4 + (1 - \alpha_1)v_5}{\rho\beta - \Omega_1} \quad (4.1.58)$$

$$v_3 = -\frac{\rho\eta_1\beta v_2 + (1 - \alpha_2)v_4 + \alpha_2 v_5}{(1 - \rho)\eta_1\beta} \quad (4.1.59)$$

$$v_4 = -\frac{\rho\eta_2\beta v_2 + (1 - \rho)\eta_2\beta v_3}{\Omega_3} \quad (4.1.60)$$

$$v_5 = \frac{\rho\eta_3\beta v_2 + (1 - \rho)\eta_3\beta v_3}{\Omega_4} \quad (4.1.61)$$

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

$$\frac{\partial^2 x_2}{\partial x_1 \partial x_2} = \rho\beta \quad (4.1.62)$$

$$\frac{\partial^2 x_2}{\partial x_1 \partial x_3} = \rho\eta_1\beta \quad (4.1.63)$$

$$\frac{\partial^2 x_2}{\partial x_1 \partial x_4} = \rho\eta_2\beta \quad (4.1.64)$$

$$\frac{\partial^2 x_2}{\partial x_1 \partial x_5} = \rho\eta_3\beta \quad (4.1.65)$$

$$\frac{\partial^2 x_2}{\partial x_1 \partial x_2} = (1 - \rho)\beta \quad (4.1.66)$$

$$\frac{\partial^2 x_2}{\partial x_1 \partial x_3} = (1 - \rho)\eta_1\beta \quad (4.1.67)$$

$$\frac{\partial^2 x_2}{\partial x_1 \partial x_4} = (1 - \rho)\eta_2\beta \quad (4.1.68)$$

$$\frac{\partial^2 x_2}{\partial x_1 \partial x_5} = (1 - \rho)\eta_3\beta \quad (4.1.69)$$

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.

4.1.9 The Global Stability of EE

In this section, we demonstrate the global stability of the endemic equilibrium point.

Theorem 4.1.5. *If $P < Q$, The endemic equilibrium point E^* of the model is globally asymptotically stable.*

Proof. Using the approach applied by Ngari *et al.*, (2020), we introduce the following Lyapunov function: ■

$$L(S, I_S, I_N, T_C, T_K) = (S - S^* - S^* \log(\frac{S^*}{S})) + (I_S - I_S^* - I_S^* \log(\frac{S^*}{S})) \quad (4.1.70)$$

$$+ (I_N - I_N^* - I_N^* \log(\frac{I_N^*}{I_N})) + (T_C - T_C^* - T_C^* \log(\frac{T_C^*}{T_C})) + (T_K - T_K^* - T_K^* \log(\frac{T_K^*}{T_K})) \quad (4.1.71)$$

We differentiate equation (4.1.70-4.1.71) with respect to time and then substitute (dS/dt) , (dI_S/dt) , (dI_N/dt) , (dT_C/dt) and (dT_K/dt) from (3.4.1-3.4.5) to obtain

$$\frac{dL}{dt} = (\frac{S - S^*}{S})\pi + \gamma_1(T_K - T_K^*) + \gamma_2(T_C - T_C^*) - (\mu + \delta - \lambda)(S - S^*) + \quad (4.1.72)$$

$$(\frac{I_S - I_S^*}{I_S})\rho\lambda(S - S^*) - (\mu + \rho + \omega_2 + 1)(I_S - I_S^*) + \quad (4.1.73)$$

$$(\frac{I_N - I_N^*}{I_N})(1 - \rho)\lambda(S - S^*) - (\mu + \omega_1 + \delta + 1)(I_N - I_N^*) + \quad (4.1.74)$$

$$(\frac{T_C - T_C^*}{T_C})\alpha_1(I_S - I_S^*) - (\gamma_2 + K_1 + \mu + \delta + (1 - \alpha_2))(I_N - I_N^*) + \quad (4.1.75)$$

$$(\frac{T_K - T_K^*}{T_K})\alpha_2 I_N + (1 - \alpha_1)(I_S - I_S^*) - (K_2 + \mu + \delta + \gamma_1)(T_K - T_K^*) \quad (4.1.76)$$

We then expand equation (4.1.72-4.1.76) and collect the positive and negative terms, respectively, to obtain where

$$\frac{dL}{dt} = P - Q, \quad (4.1.77)$$

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

$$Q = \gamma_1 T_K^* + \gamma_2 T_C^* + (\mu + \delta) S^* + \frac{\rho \lambda I_S^* S^*}{I_S} + (\mu + \rho + \omega_2 + 1) I_S^* +$$

$$(1 - \rho) \lambda \frac{I_N^* S^*}{I_N} + (\mu + \omega_1 + \delta + 1) I_N^* + \frac{T_C^* I_S}{T_C} \alpha_1 + (\gamma_2 + k_1 + \mu + \delta +$$

$$(1 - \alpha_2)) \frac{I_N^* T_C^*}{T_C} + \frac{T_K^* I_S^*}{T_K} \alpha_1 + (k_2 + \mu + \delta + \gamma_1) T_K^* + \alpha_1 I_S$$

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^* corresponds to the endemic equilibrium point in the model. Con-

sequently, by applying LaSalle's invariance principle (Lissale, 1968), E^* is stable globally and asymptotically in E^* if $P < Q$. This finding suggests that the disease will continue to persist whenever $P < Q$, regardless of the initial conditions.

4.1.10 Normalized sensitivity analysis of basic reproduction numbers

Sensitivity analysis of parameters is performed using differential calculus. This analysis focuses on identifying which parameters have the greatest impact on the basic reproduction number, R_0 . It helps determine the importance of each model parameter in the transmission of childhood diarrhea infection. The goal is to identify parameters that significantly influence R_0 and should therefore be prioritized in intervention strategies. Sensitivity indices quantify the relative change in a variable resulting from changes in a parameter. The normalized forward sensitivity index of a variable with respect to a parameter is defined as the ratio of their relative changes. When the variable is a differentiable function of the parameter, this index can alternatively be expressed using partial derivatives (Rodrigues *et al.*, 2013). According to Ngari and Koech, (2017), the normalized forward sensitivity index of a variable \emptyset , which depends differentially on a parameter ϑ , is given by:

$$R_{\vartheta}^{\emptyset} = \frac{\partial \emptyset}{\partial \vartheta} * \frac{\vartheta}{\emptyset} \quad (4.1.78)$$

On differentiating partially the expressions of R_0 and their initial conditions the sensitivity indices for all the parameters at EEP. The normalized sensitivity analysis was evaluated using equation 4.3.45 by substituting parameters from table 3 and 4. The following values were obtained.

Table 4: Sensitivity indices of parameters

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

From the above sensitivity analysis, it is observed that the values for $\rho, \delta, \beta, k_1, k_2, \alpha_1, \eta_1, \eta_2$ and η_3 are positive for each reproduction number and negative for $\pi, \mu, \omega_1, \omega_2, \alpha_2,$ and $\rho,$. The implication of these analysis is that the parameters $\pi, \mu, \omega_1, \omega_2, \alpha_2,$ and $\rho,$ are inversely proportional to the basic reproduction number while the infection rate β is directly proportional to the basic reproduction number. This suggests that to reduce the spread of childhood diarrhea infection further efforts and measures should be focused in reducing $\rho, \delta, k_1, k_2, \alpha_1, \eta_1, \eta_2$ and η_3 that increases development of stress and faulty health system.

4.2 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 3 are used to carry out the numerical simulations and a graphical presentation of the numerical results is made.

4.2.1 Susceptible population for under five children

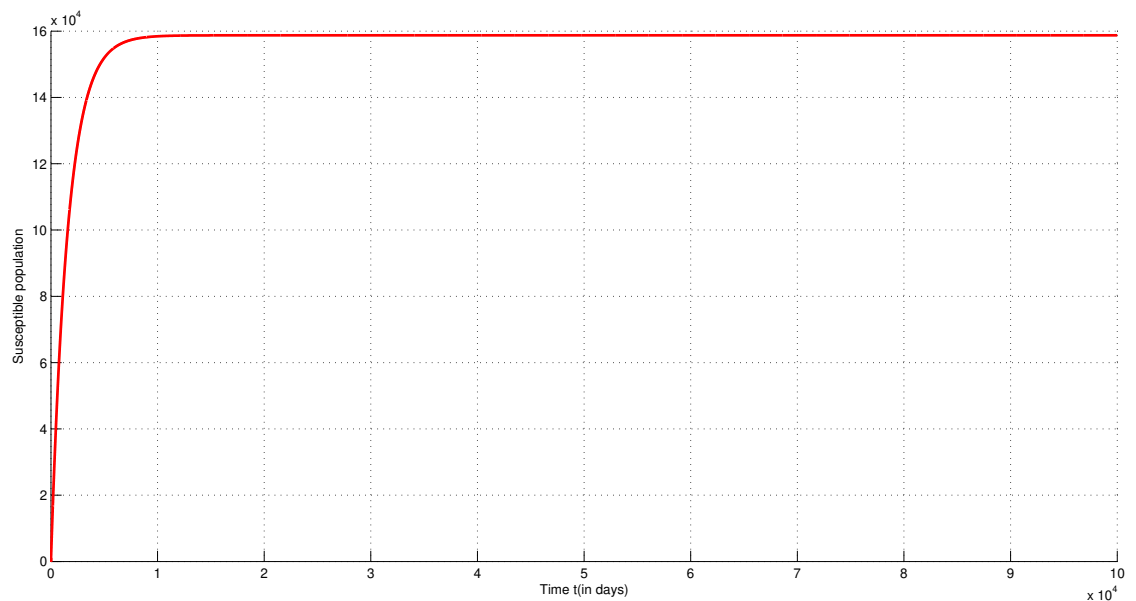


Figure 2: Total population for under five children with time

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 diarrhoea persist in the population but if proper health care services are given in time it could save more live that would otherwise be lost.

4.2.2 Diarrhea infected under five children under stress

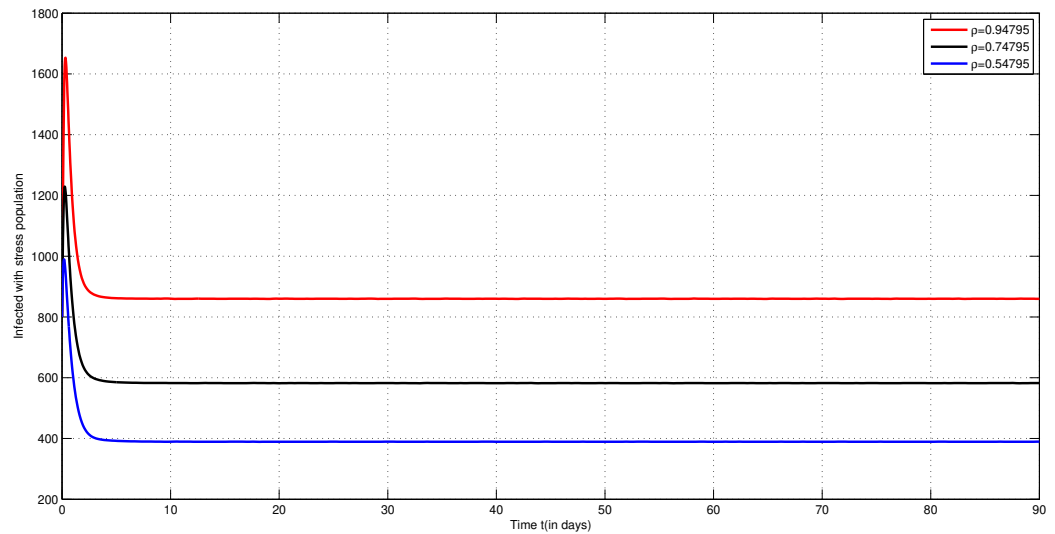


Figure 3: Diarrhoea infected under five children under stress

This class begin by gaining from the susceptible who acquire diarrhoea, 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 low income population 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

4.2.3 Diarrhea infected under five children free of stress

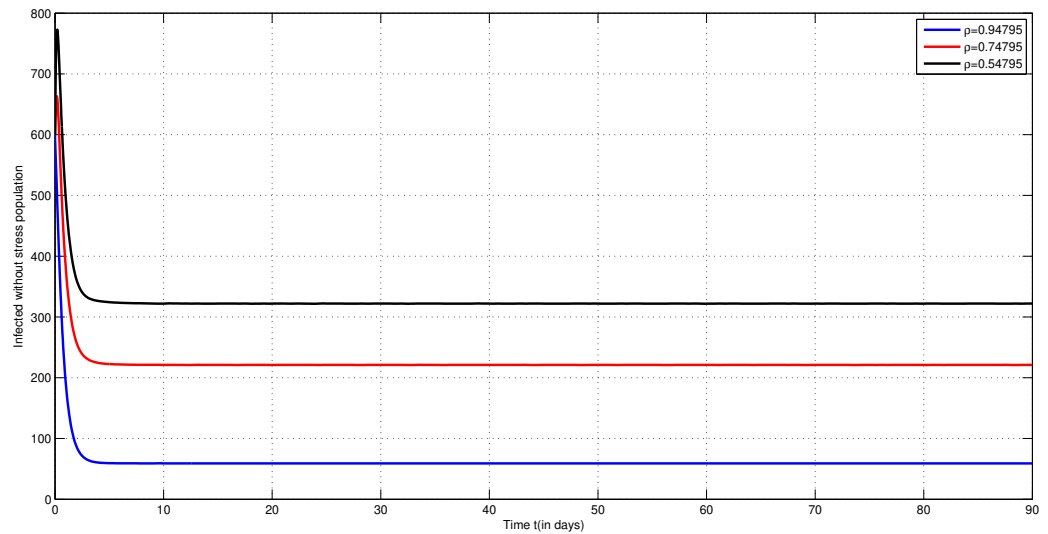


Figure 4: Diarrhoea infected under five children under stress

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 low income population 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 diarrhoea can be contained in the population through increase good health service and proper house holds for the under five children.

4.2.4 Treatment for under five children in faulty health system

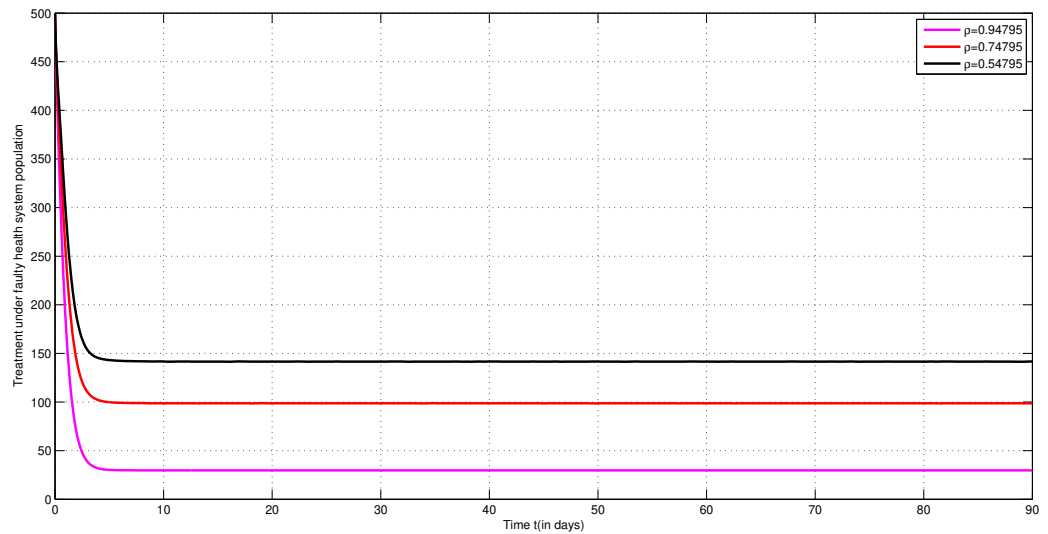


Figure 5: Treatment for under five in faulty health system with time

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.

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

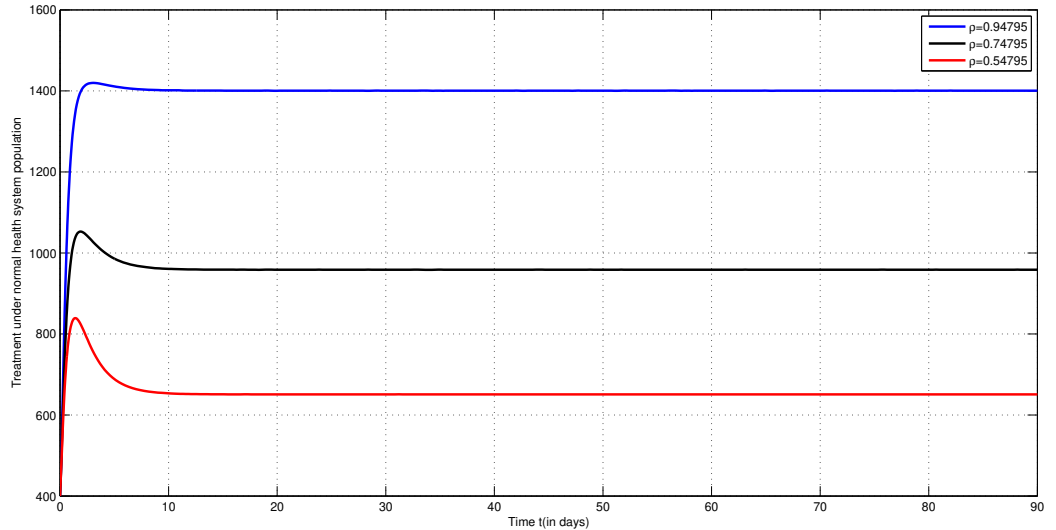


Figure 6: Treatment for under five in normal health system with time

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 were 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.

4.2.6 Treatment for under five children at varying treatment rate under stress

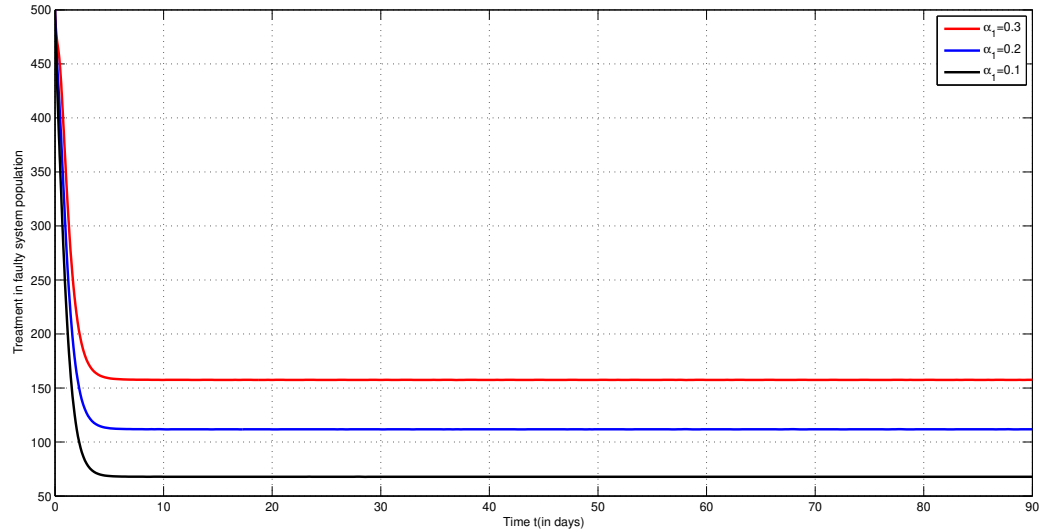


Figure 7: treatment for under five children with time

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 in low income population 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.

4.2.7 Treatment for under five children at varying rate of recovery

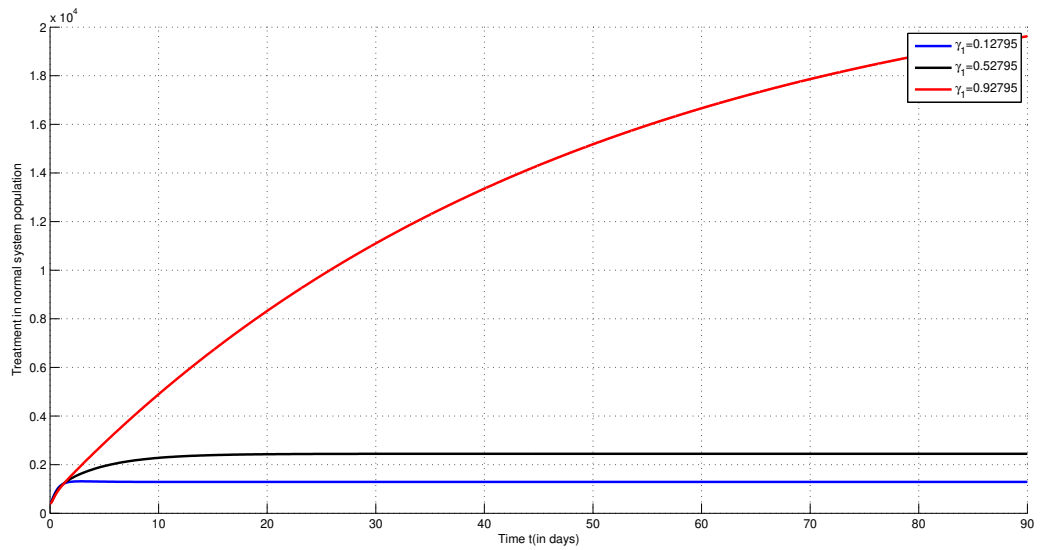


Figure 8: traetment for under five children with time

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.

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

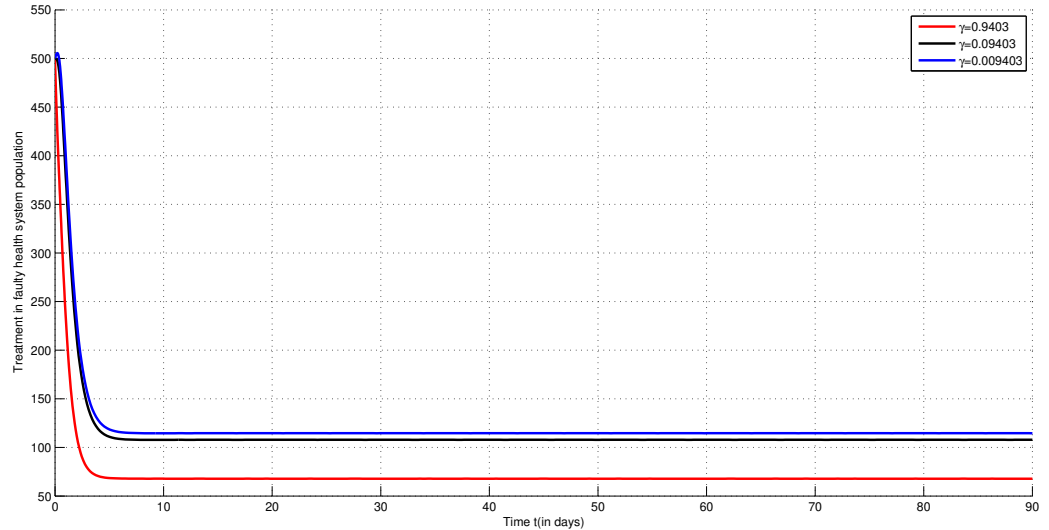


Figure 9: treatment for under five children with time

As compared to treatment under normal health system this class usually had few children as they suffer from social health facilities and poor services in the health system and households. An increase in the rate of recovery increases the output population, and a decrease will lead to a decrease as observed in the figure 9.

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

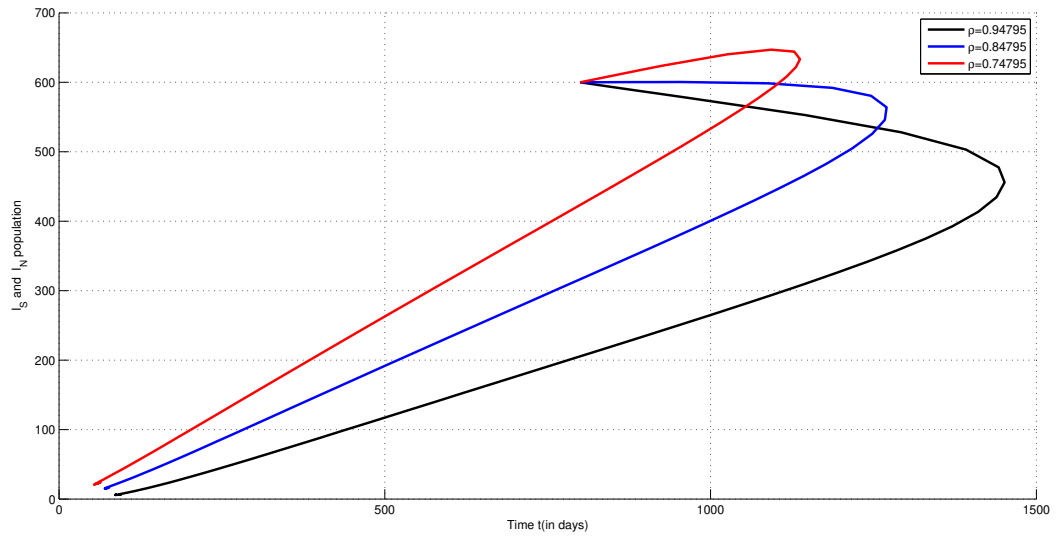


Figure 10: Comparison between infected under stress and without stress with time

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 diarrhoea 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.

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

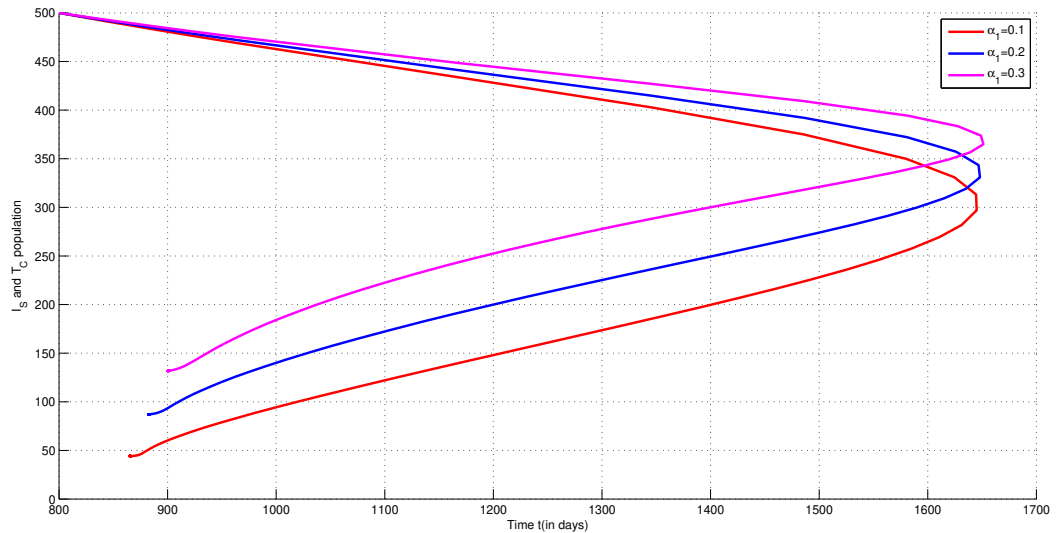


Figure 11: A graph of I_S and T_C with time

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 diarrhoea 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.

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

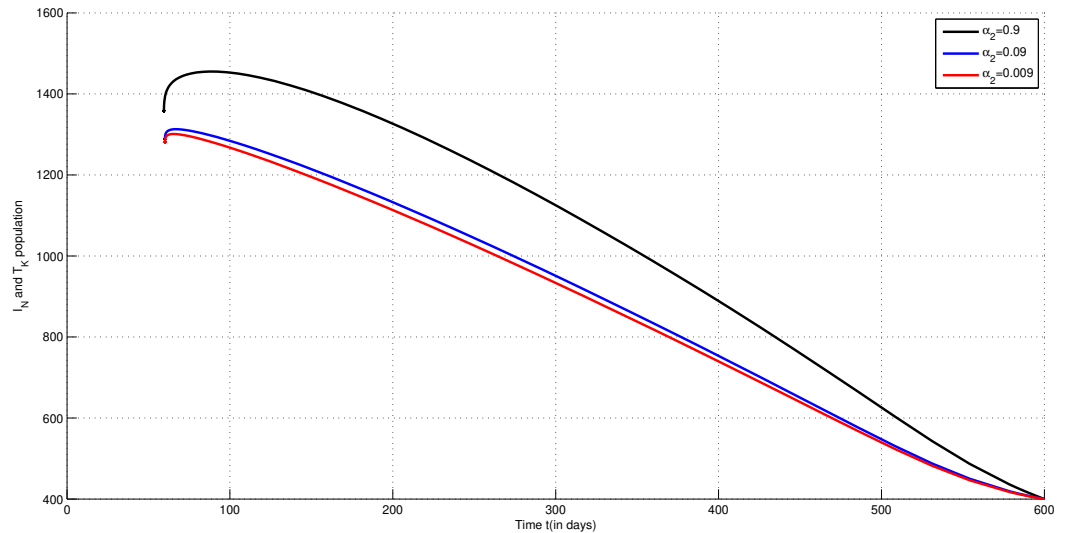


Figure 12: Comparison between infected without stress and treatment in normal health system at varying rate of seeking treatment with time

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.

4.2.12 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 cholera 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. The normalized forward sensitivity index of a variable with respect to a parameter is the ratio of the relative change in the parameter. When the variable is a differentiable function of the parameter the sensitivity index may be alternatively defined using partial derivatives (Rodrigues *et al.*, 2013). According to (Ngari and Koech, 2017),

the normalized forward sensitivity index of a variable, \emptyset that depends differentially on a parameter, ϑ , is defined as

$$R_{\vartheta}^{\emptyset} = \frac{\partial \emptyset}{\partial \vartheta} * \frac{\vartheta}{\emptyset} \quad (4.2.1)$$

On differentiating partially the expressions of R_O and their initial conditions the sensitivity indices for all the parameters at EEP. The normalized sensitivity analysis was evaluated using equation 4.3.45 by substituting parameters from table 3 and 4. The following values were obtained.

Parameters	Sensitivity indices
π	-0.00377942
μ	-0.000031224
ρ	0.528113
ω_1	$-4.58265 * 10^{-7} \omega_2$
δ	-0.0412177
β	0.00031516
k_1	0.000266559
α_2	0.000196475
k_2	-0.0000108841
ρ	$3.03942 * 10^{-6}$
η_3	-0.00389483
α_1	0.124437
η_2	$1.92379 * 10^{-6}$
η_1	$3.70097 * 10^{-11}$
ϵ	0.0000328237

Table 5: Sensitivity indices of parameters

From the above sensitivity analysis, it is observed that the values for β, η_1, η_2 and ϵ are positive for each reproduction number and negative for $\Sigma, \tau, \alpha, \mu, \theta, \delta, \chi, \omega, \delta_2$. The implication of these analysis is that the parameters $\Sigma, \eta_1, \tau, \alpha, \mu, \theta, \delta, \chi, \omega, \delta_2$ are inversely proportional to the basic reproduction number while the infection rate β is directly proportional to the basic reproduction number. This suggests that to reduce the spread of cholera infection further efforts and measures should be focused in reducing θ and δ_1 that increases development of achlorhydria condition.

4.3 Simulations parameters of the model

In this section, the Runge-Kutta method is applied in the model equations and there after used to carry out numerical simulations by fourth order Runge-Kutta method in MatlabR2015a to study the dynamical behavior of the model state variables in the presence of

model parameters. The Runge-Kutta method is a numerical method of solving an initial value problem of ordinary differential equations. The numerical simulations are performed using the initial conditions and parameters in tables 3 and 4 and the numerical results are presented graphically.

4.3.1 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 cholera 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. The normalized forward sensitivity index of a variable with respect to a parameter is the ratio of the relative change in the parameter. When the variable is a differentiable function of the parameter the sensitivity index may be alternatively defined using partial derivatives (Rodrigues *et al.*, 2013). According to (Ngari and Koech, 2017), the normalized forward sensitivity index of a variable, \emptyset that depends differentially on a parameter, ϑ , is defined as

$$R_{\vartheta}^{\emptyset} = \frac{\partial \emptyset}{\partial \vartheta} * \frac{\vartheta}{\emptyset} \quad (4.3.1)$$

On differentiating partially the expressions of R_0 and their initial conditions the sensitivity indices for all the parameters at EEP. The normalized sensitivity analysis was evaluated using equation 4.3.45 by substituting parameters from table 3 and 4. The following values were obtained.

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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	$3.70097 * 10^{-11}$	
ϵ	0.0000328237	

Table 6: Sensitivity indices of parameters

From the above sensitivity analysis, it is observed that the values for β, η_1, η_2 and ϵ are positive for each reproduction number and negative for $\Sigma, \tau, \alpha, \mu, \theta, \delta, \chi, \omega, \delta_2$. The implication of these analysis is that the parameters $\Sigma, \eta_1, \tau, \alpha, \mu, \theta, \delta, \chi, \omega, \delta_2$ are inversely proportional to the basic reproduction number while the infection rate β is directly proportional to the basic reproduction number. This suggests that to reduce the spread of cholera infection further efforts and measures should be focused in reducing θ and δ_1 that increases development of achlorhydria condition.

$$R_{\vartheta}^{\vartheta} = \frac{\partial \vartheta}{\partial \vartheta} * \frac{\vartheta}{\vartheta} \quad (4.3.2)$$

On differentiating partially the expressions of R_O and their initial conditions the sensitivity indices for all the parameters at EEP. The normalized sensitivity analysis was evaluated using equation 4.3.45 by substituting parameters from table 3 and 4. The following values were obtained.

Parameters	Sensitivity indices	
π	-0.00377942	
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ρ	0.528113	
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ρ	-0.00389483	
η_3	0.124437	
α_1	$1.92379 * 10^{-6}$	
η_2	$3.70097 * 10^{-11}$	
ϵ	0.0000328237	

Table 7: Sensitivity indices of parameters

From the above sensitivity analysis, it is observed that the values for β, η_1, η_2 and ϵ are positive for each reproduction number and negative for $\Sigma, \tau, \alpha, \mu, \theta, \delta, \chi, \omega, \delta_2$. The implication of these analysis is that the parameters $\Sigma, \eta_1, \tau, \alpha, \mu, \theta, \delta, \chi, \omega, \delta_2$ are inversely proportional to the basic reproduction number while the infection rate β is directly proportional to the basic reproduction number. This suggests that to reduce the spread of cholera infection further efforts and measures should be focused in reducing θ and δ_1 that increases development of achlorhydria condition.

4.4 Simulations parameters of the model

In this section, the Runge-Kutta method is applied in the model equations and there after used to carry out numerical simulations by fourth order Runge-Kutta method in MatlabR2015a to study the dynamical behavior of the model state variables in the presence of model parameters. The Runge-Kutta method is a numerical method of solving an initial value problem of ordinary differential equations. The numerical simulations are performed using the initial conditions and parameters in tables 3 and 4 and the numerical results are presented graphically.

CHAPTER FIVE

SUMMARY, CONCLUSION AND RECOMMENDATIONS

5.1 SUMMARY

In this study, we developed and analyzed a deterministic under five childhood diarrhoea model incorporating social factor (stress) and faulty health system and their effect on under five children during diarrhoea outbreak. The model structure comprised of five compartments of human populations, namely; susceptible individuals, diarrhoea infected with stress, diarrhea infected without stress, treated in faulty health system and treated in normal health system. The model considered social factors, specifically stress, and a faulty health-care system as key drivers of diarrhea transmission. It is formulated as a first-order ordinary differential equation. The state variables were shown to remain positive and within a biologically feasible region. The model's reproduction number was calculated as the largest eigenvalue of the Jacobian matrix using the Next Generation Matrix Method. Local stability analysis of the disease-free equilibrium revealed that it is locally asymptotically stable when $R_0^* < 1$. Analytical expressions were derived for the basic reproduction number, and normalized sensitivity indices were computed to assess the impact of control parameters on this reproduction number. Initial conditions for the state variables and model parameters were sourced from 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. In this study, there is no error analysis or validation done since there was no observed data used to fit the model. Instead, the model outcomes were derived using secondary data.

5.2 CONCLUSION

From the study's results, the following conclusions were drawn;

1. An increase in the force of infection lead to increase in diarrhoea 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 diarrhoea as seen in figure 3.
2. 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.

3. Sensitivity analysis revealed that the infection rate is inversely proportional to the rate of seeking treatment and recovery, increasing treatment and recovery rate will reduce the force of infection and thus childhood diarrhoea can be maintained at baseline level.

5.3 RECOMMENDATIONS

Based on the conclusions above, the following recommendations are proposed;

1. The basic reproduction number should be kept below unity $R_0 = 0.008278$ so as to control the spread of under five diarrhoea.
2. Children under five living in low income settings under stress should be given special attention during diarrhea outbreak.
3. There should be public awareness on social factors like stress and faulty health system and how they fuels diarrhoea so that the mass will be educated and take necessary precaution in case of diarrhea outbreak.
4. The government should put up measure on these faulty health system and maintain free medical services for all and this will reduces mortality and morbidity during disease outbreak.
5. Treatment of under five children with stress in normal health facility reduced the morbidity of diarrhea.

5.4 FURTHER RESEARCH WORK

The following areas are recommended for further research;

1. A model incorporating under five children, children with compromised immune system and malnourished children.
2. Focus on childhood diarrhoea transmission model using stochastic approach.
3. Develop a data fitting model.

Appendix A

PUBLICATION CERTIFICATE



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Journal of Advances in Mathematics and Computer Science

Letter No. ACCJAMCS142964CAR

Date: 26-08-2025

Subject: Acceptance Letter for (Manuscript Number: 2025/JAMCS/142964) submitted in Journal of Advances in Mathematics and Computer Science

Dear Colleague,

We are pleased to inform you that the peer review process and editorial review process have been completed for your following manuscript.

Manuscript Number: 2025/JAMCS/142964

Title: Modeling Social Factor and Faulty Health System on the Dynamic of Childhood Diarrhea In Majengo Nyeri County, Kenya

Author(s): Caroline Wambui Muiiri, Rachel Atieno, Moffat Chamuchi

We are ready with the final decision. We are happy to inform you that your manuscript is officially accepted for publication in the **Journal of Advances in Mathematics and Computer Science**. This journal is Peer Reviewed and Referred journal.

Once your manuscript is moved to publishing, our production editor will keep you informed of your article's progress in the production process. You will also receive a galley proof of your manuscript for final review. We're excited to move forward with your submission. Please feel free to email me with any questions.

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Appendix B

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Appendix C

$$\begin{aligned}
 a_3 = & -\beta\delta\rho + \beta\mu\rho + -\beta\delta\eta_1 + \beta\mu\eta_1 + \beta\delta\rho\eta_1 + \beta\eta_2 - \beta\eta\rho + \beta\rho\alpha_1\eta_2 - \beta\alpha_2\eta_2 + \beta\rho\alpha_1\eta_2 - \\
 & \beta\alpha_2\eta_2 + \beta\rho\alpha_2\eta_2 + \beta\rho\eta_3 - \beta\rho\alpha_1\eta_3 + \beta\alpha_2\eta_3 - \beta\rho\alpha_2\eta_3 + \delta\Omega_1 - \mu\Omega_1 + \beta\eta_1\Omega_1 - \beta\rho\eta_1\Omega_1 + \\
 & \delta\Omega_2 - \mu\Omega_2 + \beta\rho\Omega_2 - \Omega_1\Omega_2 + \delta\Omega_3 - \mu\Omega_3 + \beta\rho\Omega_3 + \beta\eta_1\Omega_3 - \beta\rho\eta_1\Omega_3 - \Omega_1\Omega_3 - \Omega_2\Omega_3 + \\
 & \delta\Omega_4 + \delta\Omega_4 - \mu\Omega_4 - \mu\Omega_4 + \beta\rho\Omega_4 + \beta\eta_1\Omega_4 - \beta\rho\eta_1\Omega_4 - \Omega_1\Omega_4 - \Omega_2\Omega_4 - \Omega_3\Omega_4.
 \end{aligned}$$

$$\begin{aligned}
 a_4 = & -\beta\delta\eta_2 + \beta\mu\beta_2 + \beta\delta\rho\eta_2 - \beta\mu\rho\eta_2 - \beta\delta\rho\alpha_1\eta_2 + \beta\mu\rho\alpha_1\eta_2 + \beta\delta\alpha_2\eta_2 - \beta\mu\alpha_2\eta_2 - \\
 & \beta\delta\rho\alpha_2\eta_2 + \beta\mu\rho\alpha_2\eta_2 - \beta\delta\rho\eta_3 + \beta\mu\rho\eta_3 + \beta\delta\rho\alpha_1\eta_3 - \beta\mu\rho\alpha_1\eta_3 - \beta\delta\alpha_2\eta_3 + \beta\mu\alpha_2\eta_3 + \\
 & \eta\delta\rho\alpha_2\eta_3 - \beta\mu\rho\alpha_2\eta_3 - \beta\delta\eta_1\Omega_1 + \beta\mu\eta_1\Omega_1 + \beta\delta\rho\eta_1\Omega_1 - \beta\mu\rho\eta_1\Omega_1 + \beta\eta_2\Omega_1 - \beta\rho\eta_2\Omega_1 - \\
 & \beta_2\eta_2\Omega_1 + \beta\rho\alpha_2\eta_2\Omega_1 + \beta\alpha_2\eta_3\Omega_1 - \beta\rho\alpha_2\eta_3\Omega_1 - \beta\delta\rho\Omega_2 + \beta\mu\rho\Omega_2 + \beta\rho\alpha_1\eta_2\Omega_2 + \beta\rho\eta_3\Omega_2 - \\
 & \beta\rho\alpha_1\eta_3\Omega_2 + \delta\Omega_1\Omega_2 - \mu\Omega_1\Omega_2 - \beta\delta\rho\Omega_3 + \beta\mu\rho\Omega_3 - \beta\delta\eta_1\Omega_3 + \beta\mu\eta_1\Omega_3 + \beta\delta\rho\eta_1\Omega_3 - \beta\mu\rho_1\Omega_3 + \\
 & \beta\rho\eta_3\Omega_3 - \beta\rho\alpha_1\eta_3\Omega_3 + \beta\alpha_2\eta_3\Omega_3 - \beta\rho\alpha_2\eta_3\Omega_3 + \delta\Omega_1\Omega_3 - \mu\Omega_1\Omega_3 + \beta\eta_1\Omega_1\Omega_3 - \beta\rho\eta_1\Omega_1\Omega_3 + \\
 & \delta\Omega_2\Omega_3 - \mu\Omega_2\Omega_3 + \rho\Omega_2\Omega_3 - \Omega_1\Omega_2\Omega_3 - \beta\delta\rho\Omega_4 + \beta\mu\rho\Omega_4 - \delta\eta_1\Omega_4 + \beta\mu\eta_1\Omega_4 + \beta\delta\rho\eta_1\Omega_4 - \\
 & \beta\mu\rho\eta_1\Omega_4 + \beta\eta_2\Omega_4 - \beta\rho\eta_2\Omega_4 + \beta\rho\alpha_1\eta_2\Omega_4 - \beta\alpha_2\eta_2\Omega_4 + \beta\rho\alpha_2\eta_2\Omega_4 + \delta\Omega_1\Omega_4 - \mu\Omega_1\Omega_4 + \\
 & \beta\eta_1\Omega_1\Omega_4 - \beta\rho\eta_1\Omega_1\Omega_4 + \delta\Omega_2\Omega_4 - \mu\Omega_2\Omega_4 + \beta\rho\Omega_2\Omega_4 - \Omega_1\Omega_2\Omega_4 + \delta\Omega_3\Omega_4 - \mu\Omega_3\Omega_4 + \\
 & \beta\rho\Omega_3\Omega_4 + \beta\eta_1\Omega_3\Omega_4 - \beta\rho\eta_1\Omega_3\Omega_4 - \Omega_1\Omega_3\Omega_4 - \Omega_2\Omega_3\Omega_4.
 \end{aligned}$$

$$\begin{aligned}
 a_5 = & -\beta\delta\eta_2\Omega_1 + \beta\mu\eta_2\Omega_1 + \beta\delta\rho\eta_2\Omega_1 - \beta\mu\rho\eta_2\Omega_1 + \beta\delta\alpha_2\eta_2\Omega_1 - \beta\mu\alpha_2\eta_2\Omega_1 - \beta\delta\rho\alpha_2\eta_2\Omega_1 + \\
 & \beta\mu\rho\alpha_2\eta_2\Omega_1 - \beta\delta\alpha_2\eta_3\Omega_1 + \beta\mu\alpha_2\eta_3\Omega_1 + \beta\delta\rho\alpha_2\eta_3\Omega_1 - \beta\mu\rho\alpha_2\eta_3\Omega_1 - \beta\delta\rho\alpha_1\eta_2\Omega_2 + \beta\mu\rho\alpha_1\eta_2\Omega_2 - \\
 & \beta\delta\rho\eta_3\Omega_2 + \beta\mu\rho\eta_3\Omega_2 + \beta\delta\rho\alpha_1\eta_3\Omega_2 - \beta\mu\rho\alpha_1\eta_3\Omega_2 - \beta\delta\rho\eta_3\Omega_3 + \beta\mu\rho\eta_3\Omega_3 + \delta\delta\rho_1\eta_3\Omega_3 - \\
 & \beta\mu\rho\alpha_1\eta_3\Omega_3 - \beta\delta\alpha_2\eta_3\Omega_3 + \beta\mu\alpha_2\eta_3\Omega_3 + \beta\delta\rho\alpha_2\eta_3\Omega_3 - \beta\mu\rho\alpha_2\eta_3\Omega_3 - \beta\delta\eta_1\Omega_1\Omega_3 + \beta\mu\eta_1\Omega_1\Omega_3 + \\
 & \beta\delta\rho\eta_1\Omega_1\Omega_3 - \beta\mu\rho\eta_1\Omega_1\Omega_3 + \beta\alpha_2\eta_3\Omega_1\Omega_3 - \beta\rho\alpha_2\eta_3\Omega_1\Omega_3 - \beta\delta\rho\Omega_2\Omega_3 + \beta\mu\rho\Omega_2\Omega_3 + \beta\rho\eta_3\Omega_2\Omega_3 - \\
 & \beta\rho\alpha_1\eta_3\Omega_2\Omega_3 + \delta\Omega_1\Omega_2\Omega_3 - \mu\Omega_1\Omega_2\Omega_3 - \beta\delta\eta_2\Omega_4 + \beta\mu\eta_2\Omega_4 + \beta\delta\rho\eta_2\Omega_4 - \beta\mu\rho\eta_2\Omega_4 - \\
 & \beta\delta\rho\alpha_1\eta_2\Omega_4 + \beta\mu\rho\alpha_1\eta_2\Omega_4 + \beta\delta\alpha_2\eta_2\Omega_4 - \beta\mu\alpha_2\eta_2\Omega_4 - \beta\delta\rho\alpha_2\eta_2\Omega_4 + \beta\mu\rho\alpha_2\eta_2\Omega_4 - \beta\delta\eta_1\Omega_1\Omega_4 + \\
 & \beta\mu\eta_1\Omega_1\Omega_4 + \beta\delta\rho\eta_1\Omega_1\Omega_4 - \beta\mu\rho\eta_1\Omega_1\Omega_4 + \beta\eta_2\Omega_1\Omega_4 - \beta\rho\eta_2\Omega_1\Omega_4 - \beta\alpha_2\eta_2\Omega_1\Omega_4 + \beta\rho\alpha_2\eta_2\Omega_1\Omega_4 - \\
 & \beta\delta\rho\Omega_2\Omega_4 + \beta\mu\rho\Omega_2\Omega_4 + \beta\rho\alpha_1\eta_2\Omega_2\Omega_4 + \delta\Omega_1\Omega_2\Omega_4 - \mu\Omega_1\Omega_2\Omega_4 - \beta\delta\rho\Omega_3\Omega_4 + \beta\mu\rho\Omega_3\Omega_4 - \\
 & \beta\delta\eta_1\Omega_3\Omega_4 + \beta\mu\eta_1\Omega_3\Omega_4 + \beta\delta\rho\eta_1\Omega_3\Omega_4 - \beta\mu\rho\eta_1\Omega_3\Omega_4 + \delta\Omega_1\Omega_3\Omega_4 - \mu\Omega_1\Omega_3\Omega_4 + \beta\eta_1\Omega_1\Omega_3\Omega_4 - \\
 & \beta\rho\eta_1\Omega_1\Omega_3\Omega_4 + \delta\Omega_2\Omega_3\Omega_4 - \mu\Omega_2\Omega_3\Omega_4 + \beta\rho\Omega_2\Omega_3\Omega_4 - \Omega_1\Omega_2\Omega_3\Omega_4.
 \end{aligned}$$