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Mathematical Modeling of Cholera Mitigation Incorporating Handwashing

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ABSTRACT

Cholera is a bacterial infection caused by Vibrio cholerae. This bacterium produces a toxin that leads to severe diarrhea and dehydration. Cholera is often associated with areas with poor sanitation, limited access to clean water and overcrowded conditions. Cholera remains a significant global health concern, with outbreaks occurring frequently in areas with poor sanitation and hygiene practices. This study aims to investigate the role of handwashing in mitigating cholera. considering the key factors such as mode of transmission, incubation period, signs and symptoms, treatment measures, risk factors and the main causes of cholera. The study tends to assess the effectiveness of handwashing as a preventive measure against cholera transmission.. The research addresses the critical gap in understanding the specific contribution of handwashing in preventing cholera, considering its complex transmission dynamics. The expected results of this study are an association between increased handwashing and a decrease in cholera occurrence. Results from this study will inform evidence-based strategies for disease prevention and contribute to the broader field of infectious disease prevention modeling. The study seeks to elucidate the dynamics of cholera transmission, considering the interplay between susceptible, infectious and recovered population. Analysis of the model shows that there exists a region where the model is mathematically and epidemiologically well posed because its solutions were positive and bounded. Computation of the basic reproduction number, was done using the next generation matrix approach. It was determined that when $R_0 < 1$, cholera does not spread. Stability analysis of the cholera model showed that the disease free equilibrium is both locally and globally asymptotically stable. Ideally, this means that keeping $R_0 < 1$ is a possible strategy for curbing the spread of the disease. Analysis of the endemic equilibrium shows its existence when $R_0 > 1$. Furthermore, the endemic equilibrium is also locally asymptotically stable. This shows that when $R_0 > 1$, the disease persists and spreads in the population.Numerical simulation was done using MATLAB software to show the effectiveness of handwashing on cholera mitigation.
Keywords: Cholera, handwashing, endemic equilibrium

1 Introduction

Cholera remains a significant global public health concern, particularly in regions with inadequate sanitation and limited access to clean water. The causative agent, Vibrio cholerae is primarily transmitted through contaminated water and food sources leading to severe diarrhoea illness and dehydration. Efforts to control cholera outbreak have traditionally focused on improving water and sanitation infrastructure, yet the role of a simple yet fundamental practice-hand washing-cannot be understated. Cholera outbreaks are often exacerbated by poor hygiene practices and insufficient sanitation. Factors such as crowded living conditions, lack of access to clean water, and inadequate waste disposal contribute to the persistent spread of cholera. Understanding the epidemiological landscape is crucial for devising effective strategies to mitigate cholera transmission.

Handwashing is crucial in preventing cholera. Proper hygiene, especially hand washing with soap and clean water, helps reduce the spread of the bacteria. This simple practice is essential in interrupting the fecal-oral transmission cycle. The incubation period varies but is generally short, ranging from a few hours to five days. This period reflects the time it takes for an individual to show symptoms after being exposed to the bacteria.

The mode of cholera transmission primarily involves the fecal-oral route, emphasizing the importance of interrupting this cycle. Contaminated hands can serve as vectors for the bacteria, particularly in settings where sanitation is compromised. Exploring the dynamics of how hand hygiene influences cholera transmission is fundamental to our research. Cholera manifests with rapid and severe dehydration, often leading to life-threatening complications. While treatment focuses on rehydration, the critical role of preventing initial exposure through hand hygiene should not be underestimated [15]. Investigating the impact of hand washing on reducing cholera incidence can contribute valuable insights to clinical management strategies.

Certain populations are more vulnerable to cholera due to socio-economic factors, geographical location and healthcare accessibility. Identifying these at-risk groups and understanding the interplay of hand hygiene in mitigating cholera within these populations is essential for targeted interventions. Despite advances in public health, challenges persist in implementing effective hand hygiene practices, particularly in resource constrained settings.
Identifying these challenges and addressing knowledge gaps in the understanding of the behavioral aspects of hand washing in cholera mitigation will be integral to the success of these study. Our research aims to comprehensively investigate the role of hand washing in the mitigation of cholera by assessing its impact on transmission dynamics, clinical outcomes and vulnerable populations [16]. Through a multi-faceted approach, we seek to bridge the gaps, inform public health policies

and contribute to the global efforts in cholera prevention and control.

The main objective of this study is therefore to develop and analyze a mathematical model on the transmission dynamics of cholera incorporating hand washing. This will entail formulating the model using system of Ordinary Differential Equations, carrying out stability analysis in order to determine the solutions around the equilibrium points and performing simulation in order to establish the impacts of handwashing in the control and management of cholera.

2 History of Modelling of Infectious Diseases

Mathematical modelling is the representation of the behaviour of an object in terms of mathematical terminologies. These models are used to aid policy makers in decision making, test the effects of introducing changes in a system and develop scientific understanding of systems [17]. Mathematical models can be classified as deterministic or stochastic according to the type of outcome it predicts. Deterministic models assume certainty of parameters in all its aspects while stochastic models allow for probabilistic variation of events by random processes. In mathematical epidemiology, these mathematical models have three aims: to understand the mechanisms of the spread of a disease, to forecast the future course of the disease and to come up with control strategies for the disease.One of the earliest mathematical models was formulated in 1760 by David Bernoulli to evaluate the effectiveness of vaccination in the control of the small pox virus [4]. Although this was among the earliest models, deterministic modelling of infectious diseases is said to have started in the 20th century . In 1906, Hamar developed a discrete time model which was deterministic in nature, to explore the repeated occurrence of the measles epidemic. [3] developed a simple epidemic model for malaria . The model is still widely used in some epidemic situations. Modelling of infectious diseases grew drastically in the middle of the 20th century and since then a variety of models have been formulated, analysed and applied to infectious diseases. Special models have been developed for diseases like HIV, malaria, cholera, smallpox, whoopingcough,measles,gonorrhea,syphilis and chickenpox.

3 Mathematical Models For Cholera

The dynamics of the cholera disease involve multiple interactions between the human host, the pathogen and the environment [7]. A number of models have been formulated to understand the complex dynamics of this disease.A simple deterministic model was developed by [5] to examine the role of aquatic reservoirs in the persistence of endemic cholera. This is done by use of a Susceptible -Infected -Recovered (SIR) model incorporating aquatic population of Vibrio cholerae. Three hypothetical communities are used to illustrate the dynamics, these are the endemic, epidemic and cholera free populations. Qualitative results of the cholera free population shows that the disease can be minimized by preventing water contamination, drinking of untreated water and by diluting cholera diarrhea using large quantities of water. The results of the model show that the importance of the aquatic reservoir is dependent on the sanitary conditions of a community and that the rate of cholera reproduction is a product of social and environmental factors. However, this model does not incorporate washing of hands which can greatly influence hygienic and environmental factors therefore changing the dynamics of the disease.Codeco's model is modified by [6] to include a hyperinfectious state of the bacterium. This is based on laboratory observations which suggest that the passage of the Ol Inaba El Tor cholera bacterium through the gastrointestinal tract results in a short lived, hyperinfectious stage of the bacterium which decays in a matter of hours to a state of lower infectiousness. The model results show that interventions should target to minimize the risk of transmission of the short lived hyperinfectious state of toxic Vibrio cholerae in order to limit the spread of cholera. The model does not incorporate handwashing which can enhance good hygiene practices to minimize the risk of infection. A model is developed by [6] to study the 2008-2009 cholera outbreak in Zimbabwe. This is a simplified version of the model by [8]. In his model, he explores the ”fast” human-to-human and ”slow” environment-to-human transmission modes of cholera. His results show that both modes of transmission contributed in sustaining cholera outbreaks in Zimbabwe and that prevention of the outbreaks can be done through mass vaccination with a cholera vaccine that has moderate uptake. However, the model does not include the use of handwashing as a way of reducing the force of cholera infection.

A mathematical model to investigate the role of human mobility in long range spread of cholera is developed by [7]. The model is applied in Kwazulu Natal province in South Africa. It explains that infected persons spread the bacteria to other water reservoirs that are far away through movements. People can also be exposed from other destinations and bring the bacteria back to the community. Model simulation and analysis of the basic reproduction number shows that although availability of clean water and toilets plays a big role in cholera incidence, human mobility is key in spread of the disease outside its hydrological catchments. However, the model does not incorporate any control strategy.Besides, practising handwashing during human movement reduces the force of infection thus impact on the spread of the infection. A model that investigates the effects of control measures like vaccination,therapeutic treatment and water sanitation on the dynamics of cholera is developed by [12]. Numerical simulations done on the model show that the various control measures are closely interrelated and that the strength of one measure as an optimal strategy depends on its relative cost and the population setting. The implementation of these measures can be greatly enhanced by public health education on the role of handwashing which is not incorporated in the model.

A model is developed by [15] to study the impact of human behaviour on cholera dynamics. It uses a system of ordinary differential equations that incorporates human behaviour and includes dependent contact rates and the rate of host shedding. The model is then extended to a reactionconve diffusion partial differential equation. This is done to investigate the interaction among human behaviour, the host, the pathogen and the disease transmission dynamics. It assumes that the population is well aware of the development and severity of the disease. Analysis of the quantitative results shows that human behaviour changes after knowledge of the outbreak with people reducing their contact with the infected persons, eating well cooked food and improving their human waste disposal [13]. The outcome of these changes the rate at which the disease spreads, the risk of infection in the environment and the epidemic and endemic levels. The significant contribution of the knowledge of the development and severity of the disease has not been incorporated in the model instead it is assumed that people will be aware of the presence of the disease.

A model that explicitly accounts for the role of the river networks in transportation and distribution of Vibrio cholerae between several human communities is developed by [3] and applied to the Kwa-Zulu Natal province of South Africa. The model concludes that waterways and river networks play a significant role in transportation and redistribution of free living Vibrios and thus hydrological controls should be based on this. However, the model does not include the role of handwashing, as a means to improve on hygiene and sanitation which reduces the amount of Vibrios in the environment and consequently limits the spread of Vibrios through .

A mathematical model for cholera transmission is developed and fitted for incidence data reported in Haiti by Andrews and Basu [1]. The model is used to provide projections of future morbidity and mortality due to cholera and to produce comparative estimates of the effects of proposed interventions. The model findings show that reduced consumption of contaminated water, vaccination and expanded use of antibiotics will avert thousands of death due to cholera. The model does not input the role of handwashing as one of these preventative and control measures.

A compartmental model that allows for person- to-person and waterborne transmission of cholera is developed to predict the sequence and timing of cholera epidemics in Haiti and to explore the potential effects of disease intervention strategies. The results show that the basic reproduction number for cholera is between 2.06 and 2.78 and that public heath interventions and vaccination substantially affect the disease transmission [11]. The media plays a great role in relaying public health messages, yet its importance has been understated in this model.

A model is formulated in the framework of optimal control by [2], to discuss the optimal intervention strategies for cholera by education and chlorination. Education is divided into human-to-human related education and human-to-environment related education. It is found that direct education is the best control strategy in the control of cholera as compared to chlorination. However,the model does not put into account the great role of handwashing on how it control the spread of cholera. All these models talk about control measures that can be used to reduce the spread of cholera, yet they do not include the use of handwashing as one of these control strategies. We therefore develop a cholera model that includes handwashing as a control strategy for the disease.

4 Hand Hygiene Impact

Studies published in 2022 cover various topics on hand hygiene impact. Most studies focus on assessing the effectiveness of different interventions in preventing and controlling diseases such as diarrheal disease, healthcare-associated infections, and parasitic infections. Several studies evaluated the effectiveness of hand hygiene practices in preventing or controlling diseases among children. A systematic review and meta-analysis by [9] reviewed and synthesized evidence on the effectiveness of interventions to improve drinking water, sanitation, and handwashing with soap on the risk of diarrheal disease in children in low- and middle-income settings. The study found that interventions that promoted hand hygiene were effective in reducing the risk of diarrheal disease among children under 5 years old by 30 percent. Findings from this study are consistent with previous studies that concluded handwashing with soap can reduce the risk of endemic diarrhea up to 30 to 48 percent. [17] investigated the prevalence of diarrhea and handwashing practices among children attending early childhood development centers in KwaZulu-Natal, South Africa. The study revealed a high prevalence of childhood diarrhea among children in early childhood development centers, with prevalence associated with the number of children in a household and handwashing practices among children and their parents or guardians.

A cross-sectional study by [4] assessed the frequency of intestinal helminth infections and their related risk factors among school children in Adola Town, Ethiopia. The prevalence of intestinal helminth infections among school children was reported as 33.91 percent, with the rate of double infection noted as 2.72 percent. The authors found significant associations with risk factors such as gender, education level, toilet usage and handwashing habits before feeding and after defecation. [1] examined the association between school water, sanitation, and hygiene and diarrhea, malnutrition, and dehydration among children in Metro Manila, Philippines. The study found that over 28 percent of students had diarrhea and 68 percent were dehydrated. Diarrhea was associated with poor handwashing behavior, while dehydration was associated with the lack of water in school restrooms. Other publications focused on other impacts of hand hygiene, including healthcare-associated infections.[15] focused on controlling healthcare-associated carbapen resistant Acinetobacter baumannii (CRAB). Through enhanced infection control measures, with emphasis on directly observed hand hygiene, hospital-onset CRAB infections decreased by 9.8 percent each year of the 5-year study period. Furthermore, a study by [3]evaluated the cost-effectiveness of a multimodal hand hygiene intervention involving alcohol-based hand rub for the prevention of neonatal bloodstream infections (BSI) in a neonatalintensive care unit in Ghana. Their analysis showed that the alcohol-based handrub program was effective in reducing patient cost of neonatal BSI by 41.7 percent and BSI-attributable hospital cost by 48.5 percent. Neonatal BSI-attributable deaths and length of hospital stay also decreased by 73 percent and 50 percent respectively, highlighting the significant potential cost-savings benefits from hand hygiene intervention While individual study results may vary, these studies show the breadth of hand hygiene impact across several health and development outcomes.

5 Vibrio Cholerae In The Environment

Until the late 1970's Vibrio cholerae was believed to be a highly host adapted bacterium and incapable of surviving longer than a few hours outside the host. More recent studies have however shown that Vibrio cholerae can survive for long periods in laboratory microcosms-water. Data accumulated over the past decade shows that V. cholerae is an autonomous inhabitant of brackish water and estuarine systems[5]. Colwell et al. [5]found that Vibrio cholerae can be more readily isolated from aquatic systems when the water temperature is higher than 17 degrees C and the salinity between 0.2 and 2.00percent [5]. Several other factors influencing survival including the level of water pollution,water pH,the association with zooplankton and Chironomid egg masses as reservoirs have been reported. It was proposed that Vibrio cholerae might utilise the nutrient rich egg masses as growth substrate.The factors influencing active propagation of Vibrio cholerae in the environment is still a popular research topic, and new knowledge is gained on a continuous basis.

Vibrio cholerae can survive in nutrient deprived environments in a dormant or viable but non-culturable (VNBC) state in environments of nutrient deprivation. The bacteria thus do not necessarily die within the aquatic environment, but survive in a manner that allows it to be infectious under favourable conditions. These aquatic Vibrio cholerae strains can not be isolated and cultured, but when inoculated into rabbit ileal loops they do cause fluid accumulation. The bacteria survive better in aquatic systems where the temperature is above 10 degrees C. The bacteria undergo physical changes associated with conversion to the VNBC state, such as becoming ovoid and reduced in size. These cells do not grow on standard laboratorium media but have been shown to be metabolically active.Experiments showed that under nutrient deprivation conditions Vibrio cholerae cells will become spherical and smaller within 20 days, these cells can then survive in a semi-dormant state for long periods. With addition of nutrients these cells can regain their culturable state within two hours.The change to the VNBC state is also accompanied by a decrease in lipid, carbohydrate, protein and DNA content at a macromolecular level[5].

6 Model Formulation and Analysis

6.1 Model Formulation

This model subdivides the human population into 3 classes that is S(t)-I(t)-R(t) where

S(t)- susceptible

I(t)- infectious

R(t)- recovered

Thus the total human population is given by N(t)=S(t)+I(t)+R(t)

6.2 Assumptions of the model

(1) The model assumes that individuals in a population mix uniformly, meaning that everyone has an equal chance of coming in contact with each other.

(2) The model assumes that parameters remain constant throughout the course of the epidemic.

(3) The model assumes that there is no latency period between the time an individual gets infected and when they become infectious.

6.3 The force of infection

The rate at which individuals are recruited into the susceptible population is α, the force of infection is defined as $\alpha = (1 - \tau)\theta\epsilon I/N$

Where $(1-\tau)\theta$ is the rate of transmission and ϵI is the contact rate between the infected. We extend the model of [6] to incorporate the effects of handwashing with clean water and soap in the transmission dynamics of the infection. $(0 < \tau < 1)$ is the reduced rate of transmission of Vibrios from the infected person due to handwashing with clean water and soap, where τ measures the efficacy of handwashing. This means that when τ is close to 1 the handwashing is very effective and the transmission is close to zero and when τ is near zero the handwashing is not effective and the infection transmission is high.

6.4 Flow diagram

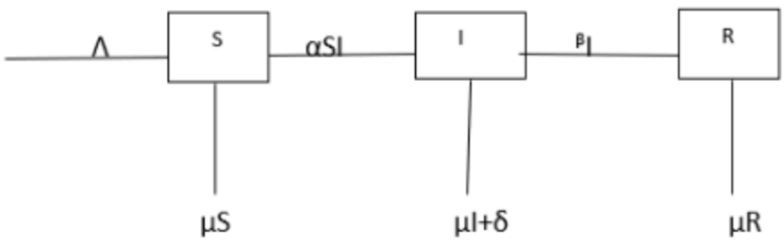

Figure 1: SIR diagram with parameters

The model equations derived from the SIR diagram are;

$$\frac{dS}{dt} = \Lambda - \alpha IS - \mu S \tag{1}$$

$$\frac{dI}{dt} = \alpha IS - (\beta + \mu + \sigma)I \tag{2}$$

$$\frac{dR}{dt} = \beta I - \mu R \tag{3}$$

Where;
$\Lambda-$ per capita rate of susceptible

$\alpha-$ the per capita rate of infection

$\beta -$ the per capita rate of recovery

$\mu -$ mortality rate

$\sigma -$ death due to cholera

6.5 Model Analysis

6.6 Positivity Of Model

Consider the first equation of the model; Proposing all the solutions of the model are positive in η

$$\frac{dS}{dt} = \lambda - \alpha IS - \mu S \tag{4}$$

$$\frac{dS}{dt} > -\alpha SI - \mu S \tag{5}$$

$$\frac{dS}{dt} > -(\alpha I + \mu)S \tag{6}$$

$$\int \frac{dS}{S} > -\int (\alpha I + \mu)dt \tag{7}$$

$$S(t) = S_0 \exp^{-\int_0^t (\alpha I(t) + \mu)dt} \tag{8}$$

6.7 Boundedness

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} \tag{9}$$

$$= \Lambda - \alpha SI - \mu S + \alpha IS - \beta I - \mu I + \beta I - \mu R \tag{10}$$

$$= \Lambda - \mu S - \mu I - \mu R \tag{11}$$

$$= \Lambda - \mu(S + I + R) \tag{12}$$

(13)

$$But; S + I + R = N \tag{14}$$

$$Thus; \Lambda - \mu N = \frac{dN}{dt} \tag{15}$$

(16)

$$\int (\frac{dN}{dt}) = \int (\Lambda - \mu N) \tag{17}$$

$$N(t) = \frac{\Lambda}{\mu} - \frac{\Lambda - \mu N0}{\mu} \exp^{\mu t} \tag{18}$$

$$\lim_{t \longrightarrow 0} N(t) = \frac{\lambda}{\mu} \tag{19}$$

$$0 \leq N(t) \leq \frac{\Lambda}{\mu} \tag{20}$$

Hence the model is bounded above and below.

6.7.1 Equilibrium points

At first we assume no disease in the population
That is $S \neq 0$, I=0 , R= 0

$$\lambda - \alpha SI - \mu S = 0 \lambda - \mu S = 0 which implies S = \frac{\lambda}{\mu} \tag{21}$$

$$DFE = E_0 = [\frac{\lambda}{\mu}, 0, 0] \tag{22}$$

6.7.2 The basic Reproduction number

The basic reproduction number; commonly denoted asR_0, in a given population is the average number of secondary infections caused by a single infectious individual during his or her entire life time as an infective when introduced into a totally or purely susceptible population [11] R_0 measures the potential of the bacteria to spread within the human population [10]. This number is very important because it is directly related to the effort required to eliminate an infection.λ can only be negative if $R_0 < 1$, meaning each infected individual in his entire life time as an infective will produce less than one infected individual on average and so the disease will die out of the population. On the other hand, if $R_0 > 1$, then each infected individual in his entire lifetime as an infective will produce more than one infected individual, and thus the disease will spread or the pathogen will be able to invade a susceptible population. When a disease is endemic, we can provide useful guidance for public health policies by determining the most appropriate control measures that will effectively reduce the basic reproduction number to less than one [9]. The basic reprodction number depends mainly on the definition of the infected and uninfected compartments. We determine R_0 using the next generation matrix approach[11]. Consider the next generation matrix G made up of two $m \times m$ matrices F and V, such that

$$G = FV^{-1}$$

where F is the Jacobian of fj , and fj is the rate of new infections in compartment j and V is the Jacobian of vj where vj is the rate of transfer of infections from one compartment to another [14]. The basic reproduction number R_0 is given as the dominant eigen value or the spectral radius of matrix G.

$$R_0 = \tau(FV - 1)$$

$F = (\alpha\varepsilon(1-\tau)IS/N)$

taking the derivative with respect to the disease compartment I, we have
$F = (\alpha\varepsilon(1-\tau)S/N)$
At DFE, S = N then $F = \alpha\varepsilon(1-\tau)$
and the matrix V is given by
$V = ((\mu + \sigma + \beta)I)$
Taking the derivative with respect to I we get
$V = (\mu + \sigma + \beta)$
On computing V^{-1}
we have $V = 1/V$ = $1/(\mu + \sigma + \beta)$
thus $F(V^{-1})$ = $\alpha\varepsilon(1-\tau)/(\mu + \sigma + \beta)$
The basic reproduction number R_0 is given by
$\tau(F()V^{-1})$ =$\alpha\varepsilon(1-\tau)/(\mu + \sigma + \beta)$
Therefore
R_0 = $\alpha\varepsilon(1-\tau)/(\mu + \sigma + \beta)$
which is the measure of the severity of an epidemic.

7 Local stability of the DFE

Using the matrix of linearization (Jacobian matrix)
Let;

$$\frac{dS}{dt} = \lambda - \alpha IS - \mu S........f1 \tag{23}$$

$$\frac{dI}{dt} = \alpha IS - \beta I - \mu I........f2 \tag{24}$$

$$\frac{dR}{dt} = \tau I - \mu R........f3 \tag{25}$$

J = $\begin{bmatrix} \frac{\partial f1}{\partial s} & \frac{\partial f1}{\partial I} & \frac{\partial f1}{\partial R} \\ \frac{\partial f2}{\partial s} & \frac{\partial f2}{\partial I} & \frac{\partial f2}{\partial R} \\ \frac{\partial f3}{\partial s} & \frac{\partial f3}{\partial I} & \frac{\partial f3}{\partial R} \end{bmatrix}$ =

$$\begin{bmatrix} -\alpha I - \mu & -\alpha S & 0 \\ \alpha I & -\alpha S(\mu + \tau) & 0 \\ 0 & \tau & -\mu \end{bmatrix}$$

Evaluating the Jacobian matrix at DFE $[\frac{\lambda}{\mu} 0, 0]$

J $[\frac{\lambda}{\mu} 0, 0]$ = $\begin{bmatrix} -\alpha I - \mu & -\alpha S & 0 \\ \alpha I & -\alpha S(\mu + \tau) & 0 \\ 0 & \tau & -\mu \end{bmatrix}$

J $[\frac{\lambda}{\mu}0, 0]$ = $\begin{bmatrix} -\mu & \frac{-\alpha\lambda}{\mu}m & 0 \\ 0 & R0-1 & 0 \\ 0 & \tau & -\mu \end{bmatrix}$

Therefore;

$\lambda_1 = -\mu$

$\lambda_2 = R0 - 1$

$\lambda_3 = -\mu$

Thus it implies that all eigen values are negative provided that $R_0 > 1$. The equilibrium point is stable which implies that the DFE is asymptotically stable suggesting that if there is a small outbreak ,the system will naturally converge back to the disease free state,indicating control of the infection over time.

7.1 Existence of Endemic Equilibrium

$S \neq 0$, $I \neq 0$ and $R \neq 0$

Λ - αSI - $\mu S = 0$

αSI - $(\mu + \beta + \sigma I) = 0$

βI - $\mu R = 0$

αSI - $(\mu + \beta + \sigma)I = 0$
αS - $\mu + \beta = 0$
αS = $\mu + \beta$

$S^* = \mu\frac{\beta}{\alpha}$

$I^* = (\frac{\Lambda}{\mu+\beta}) - \frac{\mu}{\alpha}$

7.2 Local stability of Endemic Equilibrium

$$J = \begin{bmatrix} \frac{\partial f1}{\partial s} & \frac{\partial f1}{\partial I} & \frac{\partial f1}{\partial R} \\ \frac{\partial f2}{\partial s} & \frac{\partial f2}{\partial I} & \frac{\partial f2}{\partial R} \\ \frac{\partial f3}{\partial s} & \frac{\partial f3}{\partial I} & \frac{\partial f3}{\partial R} \end{bmatrix} = \begin{bmatrix} -\alpha\mu(R0-1) - \frac{\mu}{\alpha} & \frac{-\alpha\Lambda}{\mu RO} & 0 \\ \alpha\mu(R0-1) & \frac{-\alpha\Lambda}{\mu RO} - (\mu+\beta) & 0 \\ 0 & \beta & -\mu \end{bmatrix}$$

$$J_E^* = \begin{bmatrix} -\alpha(R0-1) - \mu & \frac{-\alpha\Lambda}{\mu RO} & 0 \\ \alpha\mu(R0-1) & \frac{-\alpha\Lambda}{\mu RO} - (\mu+\beta) & 0 \\ 0 & \beta & -\mu \end{bmatrix}$$

Therefore;

$$\lambda_1 = -\mu(R0-1) - \mu$$

$$\lambda_2 = \frac{-\alpha\Lambda}{\mu RO} - (\mu+\beta)$$

$$\lambda_3 = -\mu$$

Clearly the eigen values are negative provided that $R_0 > 1$, thus the modelis locally asymptotically stable.

8 Global stability of DFE

For global stability of DFE, the technique by [4] is used. There are two conditions that if met guarantee the global asymptotic stability of the diseases free state. Equation (3.4 to 3.6) maybe written in the form of

$$\frac{dX}{dt} = H(X,Z) \quad (26)$$

$$\frac{dZ}{dt} = G(X,Z), G(X,0) = 0 \quad (27)$$

where $X \in \mathbb{R}^2$ and X=S(t),R(t) denotes the number of uninfected individuals. $Z \in \mathbb{R}^1$ where Z=I(t) denotes the number of infected individuals,E_0= $(\lambda/\mu, 0, 0)$ denotes the disease free equilibrium point of the system where $X^* = (\lambda/\mu)$. Conditions in (1) must be met to guarantee a local asymptotic stability;

$$\frac{dX}{dt} = H(X,0) \quad (28)$$

X^* is globally asymptotically stable
G(X,Z) = PZ - $\hat{G}(X,Z) \geq 0$ for (X,Z) $\in \Omega$ ....(1) Where P = DZG (X^*,0) is a M-matrix i.e (the off diagonal elements of P are non negative and Ω is the region where the model makes biological sense.

Theorem 8.1. *If system (3.29 and 3.30) satisfies condition (1), then the fixed point* $E_0 = (X^*, 0, 0)$ *is globally asymptotically stable of equilibrium of system (3.29 and 3.30) provided that* $R_0 < 1$ *and assumptions in (1) are satisfied.*

Proof. Consider H(X,0)= $\Lambda - \mu S$ and G(X,Z)= PZ-$\hat{G}(X,Z)$ where P = $\begin{bmatrix} -\beta + \mu + \sigma & 0 \\ \beta & -\mu \end{bmatrix}$

but $\hat{G}(X,Z) = -\frac{1-\tau\theta\epsilon I}{N}$

G(X,Z) is given by $\hat{G}1(X,Z) = -1 + \tau\theta\epsilon I$
$\hat{G}2(X,Z) = 0$

Considering Jacobian matrix and replacing S(t)= λ/μ, I(t)=0, R(t)=0 we obtain G(X,Z)=0 and so the condition in(1) is met. So E_0 is globally asymptotically stable when $R_0 < 1$. This implies that we do not expect the disease outbreak for life. Thus this Epidemic will die out or will not develop in the population. □

9 Global stability of Endemic Equilibrium (EE)

The global stability of the endemic is obtained by means of Lyapunov's direct method and Lasalle's invariance principle(11). Consider the non-linear Lyapunov's function V:(SIR) ∈omega⊂ $\mathbb{R}^3 : SIR > 0$ defined as V=S-S*lnS+ I-I*lnI- R*lnR ....(2) . where V is the interior of the region Ω. E^* is the global minimum of V on Ω and $\forall$ V:(S,I,R)=0 The time derivative of the equation (2) is given by

$$\frac{dV}{dt} = \dot{V} = \dot{S}(1 - S^*/S) + \dot{I}(1 - I^*/I) + \dot{R}(1 - R^*/R)$$

$$\dot{V} = (\lambda - \alpha IS - \mu S)(1 - S^*/S) + (\alpha IS - (\beta + \mu + \sigma)I)(1 - I^*/I) + (\beta I - \mu R)(1 - R^*/R)$$

$$\dot{V} = \lambda - \alpha IS - \mu S - (S^*/S)\lambda + \alpha IS^* + \mu S^* + \alpha IS - (\beta + \mu + \sigma)I - \alpha I^*S + (\beta + \mu + \sigma)I^* + \beta I - \mu R - \beta I(R^*/R) + \mu R^*$$

$$\dot{V} = \lambda - \mu S - S^*/S\lambda + \mu S^* - (\beta + \mu + \sigma)I + (\beta + \mu + \sigma)I^* + \beta I - \mu R - \beta I(R^*/R) + \mu R^* \leq 0$$

Hence $V < 0$. We see that V=0 iff S=S*, I=I*, R=R* and thus the largest compact invariant set in S,I,R $\in \Omega$:V=0 is the singleton E^* where E^* is the endemic equilibrium. Thus E^* is globally asymptotically stable in interior region of Ω. This implies that the disease transmission levels can be kept quite low or manageable with minimal deaths at the peak times of the re-occurrence.

10 Results and Discussions

10.1 Results

All state variables were found to be positive and bounded meaning that the model was biologically meaningful. In this work, we formulated a model for the dynamics of cholera infections incorporating handwashing. The existence of the disease free equilibrium and endemic equilibrium was established and the stability of the same was analysed and were found to be both globally and locally asymptoticall stable. From the numerical simulation, we observe that handwashing has effect of mitigating the disease prevalence.

10.2 Model Parameters and Values

Numerical simulations were carried out using MATLAB software to illustrate the behaviour of our system for different values of the model parameters. Some of the parameters have been obtained from literature will others are being estimated. The parameter values are shown in the table below;

Parameter	Description	Value	Source
S(t)	Susceptible individuals	10×10^5	[19]
I(t)	Infected individuals	7.082×10^5	[27]
R(t)	Recovered individuals	0.2	Estimated
εI	Contact rate between infected	1000	Estimated
Λ	Rate of susceptible	9.6274×10^{-5}	[12]
μ	Motality rate	2.537×10^{-5}	[12]
Tau	Efficacy of handwashing	0 < Tau < 1	Assumed
ϴ	Rate of transmission	1	Estimated
δ	Death due to cholera	10	[19]
β	Rate of recovery	1.825×10^{-3}	[12]

Figure 2: Model Parameters and Values

10.3 Numerical Simulations and Interpretation

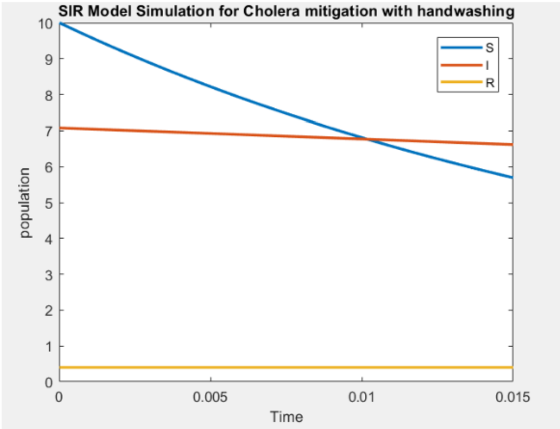

Figure 3: numerical solutions when $\tau < 1$

In the presence of handwashing,the number of infectives decreases as time tends to infinity as many people practice handawshing and hygiene to prevent infections.This causes the number of susceptibles to remain high in the population. As people continues to practice handwashing the number of recovered individuals increases.

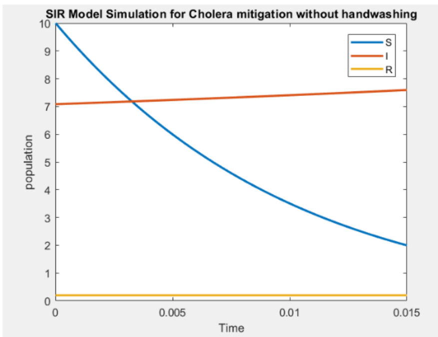

Figure 4: numerical solutions when $\tau < 1$

With a low probability of successs of handwashing,the number of susceptibles depletes sharply with time due to increase infections.The recovered individuals are less since precautionary measures like handwashing are not practised.

11 Conclusion

In this study, a mathematical model based on a system of ordinary differential equations incorporating handwashing has been formulated and analysed with an aim of investigating the effect of handwashing as a disease prevention strategy. Numerical Analysis of the model supports the fact that both the disease free and endemic equilibrium are stable. It also shows that lack of effective handwashing greatly increases the number of people infected by cholera.

12 Recommendation

Cholera still remains endemic in many countries with most African countries experiencing sporadic outbreaks. The findings of this study show that lack of handwashing leads to increased cases of infection. Therefore, we recommend that policy makers and health practitioners should embrace the practice of handwashing whether there is an epidemic or not.

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