MODELLING CHOLERA TRANSMISSION INCORPORATING MEDIA COVERAGE

BY

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BA with ED

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Declaration

Declaration by the Candidate

This thesis is my original work and has not been presented for a degree in any other University. No part of this thesis may be reproduced without the prior written permission of the author and/or Moi University.

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Dedication

I dedicate this work to my parents, Chrispinus and Jean and my siblings Christine, Sebastian and Reagan.

Abstract

Cholera is a gastrointestinal disease caused by a bacterium called *Vibrio cholerae*. The spread of the disease depends largely on social and environmental factors such as, eating food or drinking water contaminated with feaces from an infected person. Cholera outbreaks, for instance, in Kenya have led to deaths and hospitalisation. Since cholera spreads and kills very fast, the effectiveness of control strategies such as vaccination, water chlorination and therapeutic treatment may be enhanced by the use of media alert and awareness campaigns. This aspect has not been considered in existing cholera models. In this study, the impact of media coverage on the spread of cholera was investigated using a mathematical model. The specific objectives of the study were to construct a mathematical model for cholera transmission incorporating media coverage, to analyse the stability of equilibrium points of the model and to evaluate the role of media coverage as a disease control strategy. Positivity and boundedness of solutions was established to ensure that the model was biologically meaningful. The model was thereafter analysed using the stability theory of differential equations. The basic reproduction number R_0, was derived using the next generation matrix approach. The existence of equilibrium points of the model was determined. The results of stability analysis showed that the disease free equilibrium was both locally and globally asymptotically stable when $R_0 < 1$ while the endemic equilibrium was locally asymptotically stable when $R_0 > 1$. Simulation analysis done using existing epidemiological data graphically confirmed the validity of the analytic results. The simulation results also showed that when media coverage was efficient ($\rho = 0.9$, where $0 < \rho < 1$ measures the efficacy of media coverage) the number of cholera infectives decreased faster, while inefficient media reporting ($\rho = 0.4$) on an outbreak and the preventive measures led to increased cases of infection, implying that, media alert and awareness campaigns are vital in controlling the spread of cholera. Based on this study, it is recommended that health practictioners embrace the use of efficacious means of media coverage to publicise awareness campaigns of an outbreak and the preventive measures.

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Acronyms

SIR	**S**usceptible **I**nfected **R**ecovered
SEIR	**S**usceptible **I**nfected **E**xposed **R**ecovered
DFE	**D**isease **F**ree **E**quilibrium
WHO	**W**orld **H**ealth **O**rganisation
MATLAB	**MAT**rix **LAB**oratory

Chapter 1

Introduction

Infectious diseases are still the leading cause of death and morbidity in the world. New infections such as Ebola, Zika, SARS and Swine flu emerge everyday leading to unprecedented numbers of death. Mathematical modelling is an important tool used in analysing the dynamics of these diseases. Mathematical models help in understanding transmission of infections and assessing the potential impact of control strategies in reducing death and morbidity. They use the most recent information to extrapolate the state and progress of an outbreak and make predictions. Subsequently, results drawn from these models help in determining optimal control strategies, predicting the impact of control strategies against infections and quantifying uncertainties in these predictions (Keeling & Danon, 2009). In recent times mathematical models have been used to explain the dynamics of various infectious diseases including cholera, where our study is based. These models give a greater understanding on the underlying biological mechanisms of a disease and enables the precise prediction of its behaviour in the human population (Johnson, 2006).

1.1 Background of the Study

Cholera is a highly infectious disease which is endemic in many parts of Africa and Asia. The disease is extremely virulent and kills very fast. Its spread depends on social and environmental factors and therefore control measures should be based on these factors.

1.1.1 History of Cholera

Cholera has been in existence for a long time. Records of the Greek physician Hippocrates of around $460-377$ B.C, describe a disease thought to be cholera (Handa,

2016). However, it is German bacteriologist Robert Koch who is credited with the discovery of the cholera organism during an outbreak in Egypt (Finkelstein, 1996). Cholera as a disease gained global importance in 1817, with the first cholera pandemic occurring in India after the bacteria spread from its original reservoir in the Ganges Delta. Since then, six other cholera pandemics have occurred in the world (World Health Organization, 2010). The second pandemic began in 1829 in Russia, spreading into Europe (Colwell, 1996), and by 1834, it had reached most of the major cities in Canada and the United States. The third and most deadly pandemic began in India in 1852 spreading to Europe, United States, Mexico and West indies (Pollitzer, 1954). The worst single year of this pandemic was in 1854 when $23,000$ people died due to cholera in Great Britain alone (Mariam & Ronald, n.d.). The fourth and fifth pandemics occurred in 1863 and 1881 respectively, but were less lethal than the previous outbreaks. Here the disease further spread to China, Japan, and South America. The sixth pandemic started in $1899-1923$ and greatly affected India, the North African coast and Arabia. During this pandemic, an estimated $34,000$ people died in Egypt in a period of three months (Mariam & Ronald, n.d.), while 4,000 other muslim pilgrims died during Hajj in Mecca in 1902. After this period, cholera receded in most parts of the world though it remained endemic in the Indian subcontinent. The seventh cholera pandemic which was the most extensive started in 1961 in Indonesia spreading through Asia, the Middle East and Africa (Faruque, Albert, & Mekalanos, 1998). The disease had not re-appeared in Africa for over a very long time and it struck harder than before with 90 percent of the cholera cases reported to the WHO in 1990 being from Africa (Mariam & Ronald, n.d.). Since then, the cholera incidence has significantly decreased in developed countries but still remains prevalent in Africa. The most recent severe epidemic in Africa occurred in Zimbabwe between 2008 and 2009 and caused $4,287$ deaths coupled with an economic collapse (Mukandavire et al., 2011). Haiti also suffered from one of the most explosive cholera outbreaks in mordern history from 2010 to 2011 after a devastating earthquake hit the country (Mintz & Tauxe, 2013). The epidemic occured because the Artibonite River; which was the major source of drinking water, was contaminated by faecal mat-

ter. By October of the same year, 473,649 cholera cases and 6,631 deaths had been recorded by the WHO (Mariam & Ronald, n.d.). Cholera is now endemic in many countries of the world especially in Africa, China and India (Skanchy, Chantry, & Nielsen, 2009) where provision of safe drinking water and sanitation is challenging. In countries like India and Bangladesh, cholera occurs seasonally and this is associated with the flactuation in water temperatures, monsoon cycles, El nino rains and Zoo plankton levels (Johnson, 2006).

1.1.2 Cholera and *Vibrio cholerae*

Cholera is caused by infection from *Vibrio Cholerae*. *Vibrio cholerae* is a comma shaped, highly motile, gram negative bacterium as shown in Figure 1.1. and 1.2.

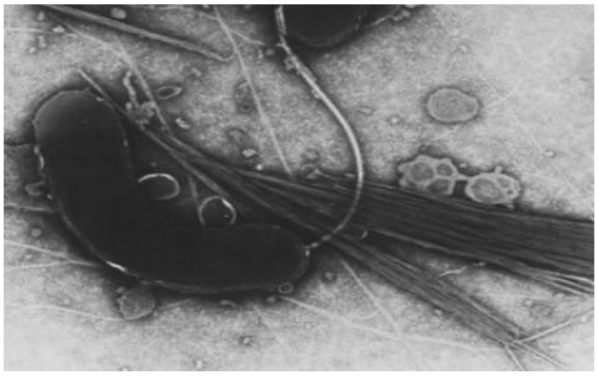

FIGURE 1.1: Electron microscope image of Vibrio cholerae bacteria
(Johnson, 2006)

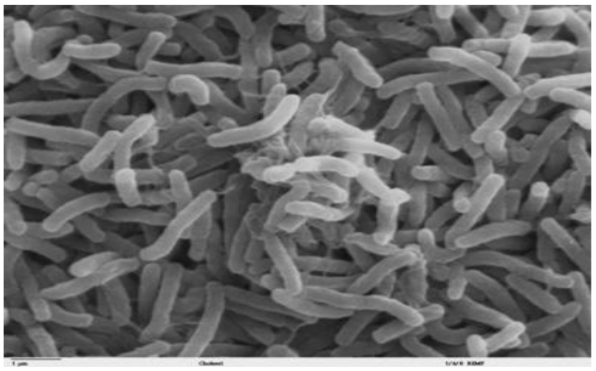

FIGURE 1.2: Scanning electron microscope image of Vibrio cholera during early infection
(Finkelstein, 1996)

The classification of the bacterium into serogroups is done on the basis of its somatic antigens (O antigens). There are over 200 O serogroups, but only two have

been known to cause the disease namely, O1 and O139 which is also known as Bengal (Reidl & Klose, 2002). The O1 serogroup causes majority of the outbreaks while the O139 serogroup is confined to SouthEast Asia (World Health Organization, 2015). *V. cholerae* O1 serogroup has two biotypes, the classical and the El Tor (Nair et al., 2006). The infection caused by the classical biotype is usually more severe than that of the El Tor biotype because the El Tor produces less cholera enterotoxin (Colwell, 1996). A classic biotype attack creates antibodies which, protect the body against recurrent infection by either of the two biotypes (Handa, 2016). The O1 serogroup further contains three serotypes namely Inaba , Ogawa and Hikojima which are found in both the El Tor and the Classical biotypes (Finkelstein, 1996). The Non O1 and Non O139 serogroups do not generate epidemics (Begum et al., 2006) though they can cause mild cases of diarrhoea . The cholera bacterium has two reservoirs: humans and the aquatic environment (Nelson, Harris, Morris, Calderwood, & Camilli, 2009). In the acquatic environment, it occurs naturally (Colwell, 1996) attaching itself to surfaces provided by plankton especially copepods, green algae and crustaceans. The *Vibrios* can spread from the coastal areas where they exist naturally to inland areas through waterways and the river network. In the inland areas, epidemic outbursts cause the bacteria to spread further from one area to another (Bertuzzo et al., 2007). Studies conducted in the recent years have also shown that global warming creates a conducive environment for the bacteria to thrive (World Health Organization, 2015).

1.1.3 Transmission, Symptoms, Diagnosis and Treatment

Cholera is a waterborne disease. Humans acquire the bacterium through ingestion developing mild or no symptoms and shed the bacterium back to environment through their stool and vomitus (Mari et al., 2011). Viable *Vibrios* can be found in faecal matter for upto 50 days, on glass surfaces for upto a month, on coins for seven days, in the soil for close to 16 days and on fingertips for 1 to 2 hours (Center for Food Security and Public Health, 2004). The bacteria also survive well in water and they may remain viable in shellfish and planktons in aquatic

regions. Spread of the bacterium is through the sanitary route, that is, eating food or drinking water contaminated with *Vibrio cholerae* and human-to- human contact through the fecal oral route (Bakhtiar, 2016). The incubation period of the disease is two hours to five days (World Health Organization, 2015). Once ingested, the bacteria pass through the stomach's gastric barrier and penetrate the mucus lining of the intestinal epithelial cells. They then adhere, multiply and colonize the intestinal epithelial cells producing cholera toxins (enterotoxin) (Reidl & Klose, 2002). The infection cycle of the disease is shown in Figure 1.3 .

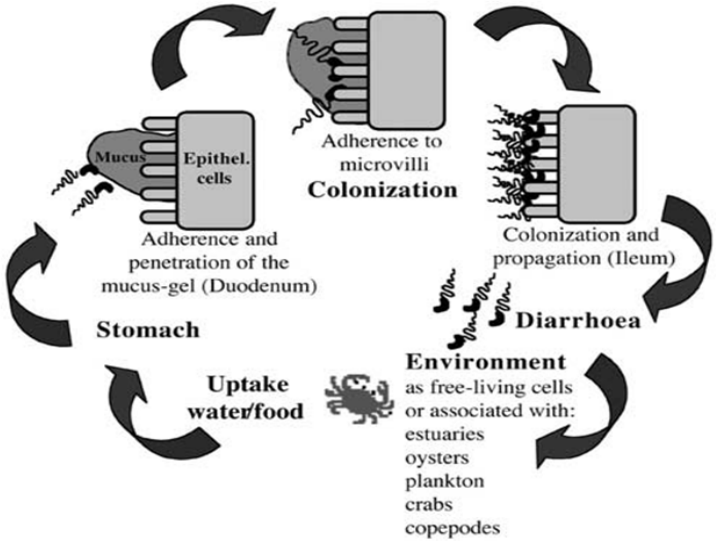

FIGURE 1.3: Infection cycle of *Vibrio cholerae*
(Reidl & Klose, 2002)

These toxins cause the intestines to secrete large amounts of fluids leading to loss of water and salts and lead to the onset of the cholera symptoms (Faruque et al., 1998). Signs and symptoms of the disease include profuse watery diarrhoea, vomiting, acidiosis and muscular cramps (Keen & Bujalski, 1992). These can lead to extreme dehydration as one can lose upto one litre of fluids per hour (Nelson et al., 2009). If untreated, severe dehydration leads to death in just a few hours. Unlike other diarrhoeal diseases, cholera is extremely virulent and can kill healthy adults in a matter of hours.

Diagnosis of the disease is done by laboratory testing through culture of the stool. In places with limited or no laboratory testing, Rapid Diagnostic Tests (RDT) are used to alert public officials on the presence of the disease. However, the sensitiv-

ity and specificity of these tests is not the best (Centers for Disease Control and Prevention, n.d.).

Treatment of cholera is based on the severity of dehydration of the patient. Simple oral rehydration solutions containing salts and glucose are used to treat mild to moderate cases, while for severe cases, treatment is based on aggressive intravenous rehydration solutions like the Ringer's lactate solution (World Health Organization, 2015). Antibiotics can also be used to treat severe dehydration cases though their mass use is discouraged due to development of resistance. People with lower immunity such as those suffering from malnutrition, HIV and those with blood group O are more susceptible to cholera attacks (Finkelstein, 1996).

1.1.4 Prevention and Control

Public health goals that can help prevent the spread of the disease are improved food safety, provision of clean drinking water and proper disposal of human waste. Health education is another important factor in prevention as it raises public awareness on preventive measures. This reminds the communities to practice good hygiene measures such as washing hands with soap after visiting the toilets and before handling or eating food, safe preparation of food, safe storage of food, chlorination of water and breastfeeding. Modern communication gadgets like mobile phones, radios, newspapers and televisions should be used in disseminating health education messages and carrying out awareness campaigns (World Health Organization, 2010). Both religious and community leaders should be included in the social mobilization process. Apart from the above, strengthening surveillance and early warning can greatly help in detecting initial cases and putting in place control measures.

The tools for cholera control include: Proper and timely management of cases in treatment centres, sufficient medical supplies to manage cases, improved public information and communication, specific training of personnel for proper management of cases, proper management of waste, vector control, improved access to water and sanitation and improved hygiene and food practices (World Health Or-

ganisation, n.d.). Immunization by two oral cholera vaccines Dukoral and Shanchol can also be done to control cholera in high risk areas (World Health Orginization, 2004)

Diarrhoeal diseases like cholera cause most global death in children under the age of five. An estimated 2,195 children die daily due to these diseases; this is more than the deaths caused by AIDS, malaria and measles combined (Center for Disease Control and Prevention, 2013). Cholera affects millions of people worldwide with an estimated 1.4 - 4.3 million cases and 28,000 - 142,000 deaths reported per year (World Health Organization, 2015). The disease is more common in developing countries especially Africa, parts of Asia and South and Central America where there is inadequate sanitation and lack of clean water. In 2012 , 25 African nations reported 94,553 cholera cases with 1,834 deaths due to Cholera over the same period (Mintz & Tauxe, 2013). In Kenya, Cholera has been endemic with periodic outbreaks of the disease occurring especially during the rainy season. In February 2015, a cholera outbreak alert was issued by the Director of medical services after an increase in cases of acute watery diarrhoea (International Federation of the Red Cross and Red Crescent Societies, 2015). This followed the outbreak that had affected 21 of the 47 counties since December 2014. During this period 7,000 cases had been reported with 100 deaths caused due to the disease (European Commission Humanitarian Aid Office, 2015). In November 2015, the cholera outbreak that had been spreading reached the Daadab refugee complex causing 1,000 cases and 10 deaths (United Nations High Commision for Refugees, 2015). By May 2016, 30 out of the 47 counties had been affected by the disease with 23 counties managing to successfully control the outbreaks. However, there is still a high risk of outbreak waves. The most active counties associated with the sporadic outbreaks of Cholera are Mandera, Wajir, Garissa, Tana river, Tharaka Nithi and Narok. Currently, Mandera county is the most affected as it is experiencing a dual outbreak of Cholera and Chikungunya that started in April 2016 (Medecins Sans Frontieres, 2016). Nationally, Kenya has had a total of 14, 878 cholera cases with 234 deaths recorded between December 2014 and May 2016

(United Nations Children's Emergency Fund, 2016).

1.2 Statement of the Problem

Developing countries carry the highest burden of childhood mortality with an estimated 9.2 million deaths reported each year. Diarrhoeal diseases are the leading cause of preventable death in children under the age of five years. Cholera, a highly infectious and fatal disease, is one of the bacterial caused diarrhoeal diseases. Failure to communicate about an outbreak of such a highly infectious disease can lead to very high proportions of infections in a population leading to hospitalisation or even death. Existing cholera models have centered on vaccination, therapeutic treatment and water sanitation as control strategies whose effectiveness may be greatly enhanced by use of print and electronic media which reaches many people faster and is cost effective. We therefore model the impact of media coverage on the spread of cholera.

1.3 Objectives of the Study

The main objective of this study was to investigate the impact of media coverage on the spread of cholera.

The specific objectives of this study were to:

(i) Construct a mathematical model for cholera transmission incorporating media coverage.

(ii) Analyse the stability of the equilibrium points of the model.

(iii) Evaluate the role of media coverage as a disease control strategy

1.4 Significance of the Study

Cholera is a disease which is highly infectious and endemic in kenya. This study will therefore assist health practitioners and policy makers to understand the dy-

namics of cholera transmission and come up with effective ways of relaying information about the disease and its preventive measures so as to reduce the cases of infection. It will also contribute to the intervention strategies for control of cholera and form a foundation for more research about cholera in Kenya.

1.5 Justification of the Study

Many Kenyan households still find it challenging to access safe drinking water and proper sanitation, conditions which, provide a favourable environment for transmission of cholera. Poor hygiene practices associated with poverty (42 percent of the kenyan population live below the poverty line) enables the disease to spread very fast. Reduction of these cholera cases is important in achieving global sustainable development goals number two and six, that is, ensuring healthy lives and promoting well being for all at all ages and ensuring availability and sustainable management of water and sanitation for all respectively. Last but not least, the causative agent of cholera is extremely virulent and has a short incubation period and thus it's more likely to lead to a high mortality rate if immediate medical attention is not provided. Since existing cholera models have focused solely on vaccination, treatment and water sanitation we add the component of media to our system of differential equations to investigate its effect in control of the disease.

Chapter 2

Literature Review

2.1 Introduction

The focus of this chapter is on literature in mathematical modelling of infectious diseases, mathematical models for cholera and epidemic models incorporating media coverage.

Cholera is an acute infectious disease. Susceptible individuals are exposed to the bacterium that causes cholera from infected individuals or from the environment. It is characterized by severe diarrhoea and vomiting. The disease usually results from poor sanitation, poor food hygiene and untreated water. The cholera bacteria produces a toxin which prevents the body from absorbing liquids hence leading to dehydration. If left untreated, infected people may die from severe dehydration within two to three hours (Ochoche, 2013). Treatment is based on the severity of dehydration of the patient. Simple oral rehydration solutions are used to treat mild to moderate cases while severe cases use aggressive intravenous rehydration. Public health goals that can help prevent and control the spread of the disease are improved food safety, provision of safe drinking water, proper sanitation, and strengthening surveillance. Health education is also very important in raising public awareness on preventive measures. The media can be used in disseminating health education messages (World Health Orginization, 2004).

2.2 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 (Marion, 2008). 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 (Murray, 2002). Although this was among the earliest models, deterministic modelling of infectious diseases is said to have started in the 20^{th} century (Hethcote, 2000). In 1906, Hamar developed a discrete time model which was deterministic in nature, to explore the repeated occurrence of the measles epidemic (Hethcote, 2000). In 1911, Ross developed a simple epidemic model for malaria (Bubniakova, 2007). Kermack and McKendrick published papers on epidemic models in 1927, 1932 and 1933, which greatly influenced the development of compartmental mathematical modelling. These models are still widely used in some epidemic situations (Murray, 2002). Modelling of infectious diseases grew drastically in the middle of the 20^{th} 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, whooping cough, measles, gonorrhea, syphilis and chickenpox.

2.3 Mathematical Models for Cholera

The dynamics of the cholera disease involve multiple interactions between the human host, the pathogen and the environment (Nelson et al., 2009). A number of models have been formulated to understand the complex dynamics of this disease. A simple deterministic model was developed by Codeço (2001) 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 communication which can greatly influence social and environmental factors therefore changing the dynamics of the disease.

Codeço's model is modified by Hartley, Glen, and Smith (2006) to include a hyperinfectious state of the bacterium. This is based on laboratory observations which suggest that the passage of the O1 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 media which can effectively disseminate public health education on good hygiene practices to minimize the risk of infection.

A model is developed by Mukandavire et al. (2011) to study the 2008-2009 cholera outbreak in Zimbabwe. This is a simplified version of the model by Hartley et al. (2006). 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 media as a tool for disseminating information on the availability of these vaccines.

A mathematical model to investigate the role of human mobility in long range spread of cholera is developed by Mari et al. (2011). 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. Peo-

ple 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, a media alert on an outbreak would greatly alter human movement and 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 J. Wang and Modnak (2011). 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 communicated via the media which is not incorporated in the model.

A model is developed by X. Wang, Gao, and Wang (2015) 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 reaction-convection- 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. 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 through media and other sources 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 Bertuzzo et al. (2007) 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 media, as a means to convey information on water sanitation and chlorination which reduces the amount of *Vibrios* in the environment and consequently limits the spread of *Vibrios* through the waterways.

A mathematical model for cholera transmission is developed and fitted for incidence data reported in Haiti by Andrews and Basu (2011). 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 use of media in mass education on these preventative and control measures.

A compartmental model that allows for person- to-person and waterborne transmission of cholera is developed by Tulte et al. (2011) 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 $2.06 - 2.78$ and that public heath interventions and vaccination substantially affect the disease transmission. 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 Bakhtiar (2016), 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 that the media plays in edu-

cating the masses on how to 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 media in creating awareness on these control strategies. We therefore develop a cholera model that includes media as a control strategy for the disease.

2.4 Mathematical Models Incorporating Media Coverage

Media coverage has been known to greatly influence an individuals behaviour as well as government policies on prevention and control of infectious diseases (Misra, Sharma, & Shukla, 2011).

Tchuenche and Bauch (2012) developed a model on the dynamics of an infectious disease where media coverage influences transmission. A signal function capturing the media coverage over time was incorporated into the model using an exponentially decreasing function. The authors observed that media coverage (which encompasses designed programs that take efforts to a critical breadth and depth of effort) does not eradicate the disease because the media signal fades when the prevalence and incidence decline to small values, but it contributes in the control process or strategy via information dissemination, which can help to a greater extent to reverse the escalation of an epidemic.

Cui, Sun, and Zhu (2007) formulated a Susceptible Exposed Infected (SEI) model to analyse the impact of media on the control of infectious diseases. Numerical simulations done on this model show that the presence of media alert shortens the time of the secondary peak in the transmission of a disease while lack of media alert can lead to multiple outbreaks of a disease.

Analysis of the impact of media on influenza is done by Tchuenche, Dube, Bhunu, Smith, and Bauch (2011) on a deterministic transmission and vaccination model. Optimal control theory is used to investigate the relative impact of costs on vaccination and media coverage. Simulations done on the model show that more people get access to media and vaccination if the costs are kept mimimal while an increase in costs reduces the degree of vaccination and media coverage and thus increases the number of infectives. The simulations also suggest that though

media can encourage vaccination among people, it can also trigger an epidemic if it promotes overconfidence in the idea that a vaccine can fully protect a person from a disease.

Zuo and Liu (2014) formulate and analyse a model on the effect of awareness programs on an epidemic with time delay. Numerical simulations of the model suggest that increasing the implementation and dissemination rates of awareness programs reduces the number of people infected with a disease in the population.

An influenza model that includes the dynamics of twitter is developed by Huo and Zhang (2016). Since twitter is popular in providing both positive and negative information, its influence on people is discussed. Sensitivity analysis done on the model shows that negative information on twitter about influenza is less significant than positive information about the disease. Therefore, increasing the infuence of positive information by twitter about influenza and keeping the rates of transmission low is essential in the control of influenza.

Zhao and Zhao (2016) perform bifurcation analysis on an SIR model incorporating media coverage. Their analysis of the stability of the disease free equilibrium shows that it cannot be affected by a delay in media coverage while the endemic equilibrium can be affected by a time delay. Numerical simulations done on this model conclude that communication about an epidemic should be done swiftly in order to contain it.

A Susceptible Infected Recovered Susceptible (SIRS) epidemic model incorporating media coverage with time delay is developed and analysed by Zhao, Lin, and Dai (2014). The analysis shows that if the delay of information about and appraisal of an epidemic on media coverage is too large, it will lead to repeated episodes of epidemic which is unfavourable for the containment of the epidemic. It is, therefore, helpful for the control of an epidemic to communicate about the outbreaks as soon as possible. It is also important to formulate a mathematical model for cholera that will incorporate media coverage as a disease control startegy.

Chapter 3

Methodology

3.1 Introduction

In this chapter we construct a mathematical model by developing a system of ordinary differential equations. Positivity and boundedness of solutions is checked by integrating the system of equations at time $t \geq 0$. The next generation matrix approach is used to calculate the basic reproduction number of the system. The local stability of the disease free equilibrium is analysed by using the techniques by Van den Driessche and Watmough (2002). The global stability of the disease free equilibrium is analysed by using the Castillo - Chavez theorem. The existence of the endemic equilibrium is checked using the Descartes' rule of signs. The local stability of the endemic equilibrium is analysed by evaluating the Jacobian matrix, and finally sensitivity analyses of the various parameters on the basic reproduction number is carried out using the normalized forward sensitivity index.

3.2 Model Description and Formulation

To achieve the objectives of this study a mathematical model based on a system of ordinary differential equations for the dynamics of cholera incorporating media coverage was formulated.

3.2.1 Model Assumptions

The model assumes that

(i) Infected people develop a transient immunity after they recover.

(ii) The rate of transfer from one compartment to another is constant.

(iii) The population is homogenous.

(iv) The population of the susceptible is replenished at a constant rate Λ.

3.2.2 Model Parameters and Variables

The model subdivides the human population into classes of susceptible S, infected I and recovered R with the total population

$$N(t) = S(t) + I(t) + R(t) \tag{3.1}$$

The concentration of *Vibrios* in the environment (contaminated water) is denoted by B. Susceptible individuals acquire cholera infection through ingesting environmental *Vibrios* from contaminated water reservoirs or through human to human transmission after ingestion of hyperinfectious *Vibrios* at the rates

$$\lambda_e = \frac{\beta_e B}{\kappa + B} \quad and \quad \lambda_h = \beta_h I$$

respectively, with the subscripts e and h representing the environmental-to-human and human-to-human transmissions as proposed in the model by Mukandavire et al. (2011). We extend the model by Mukandavire et al. (2011) to incorporate the effects of media coverage in the transmission dynamics of the infection. μ denotes the natural death rate, Λ is the rate at which individuals are recruited into the susceptible population, β_e is the rate of ingestion of *Vibrios* from the environment, $\rho\beta_e$ $(0 < \rho < 1)$ is the reduced rate of ingestion of *Vibrios* from the environment due to media coverage, where ρ measures the efficacy of media coverage. This means that when ρ is close to 1 the media is very effective and the environmental transmission is close to zero and when ρ is near zero the media is not as effective and the environmental transmission is high. β_h is the rate of human to human transmission, $\frac{\rho\beta_h I}{m+I}$ is the reduced rate of contact with infected persons due to media alert where the function $\frac{I}{m+I}$ is a continuous bounded function which takes into account disease saturation or psychological effects, κ is the pathogen concentration that yields 50% chance of contracting cholera, γ is the rate of recovery from cholera,

δ is the death rate due to cholera, m is the effect of media coverage, σ is the rate of human contribution to *Vibrio cholerae* and ξ is the death rate of *Vibrio cholerae*.

3.2.3 The flow chart diagram

The resulting flow chart diagram is shown in Figure 3.1

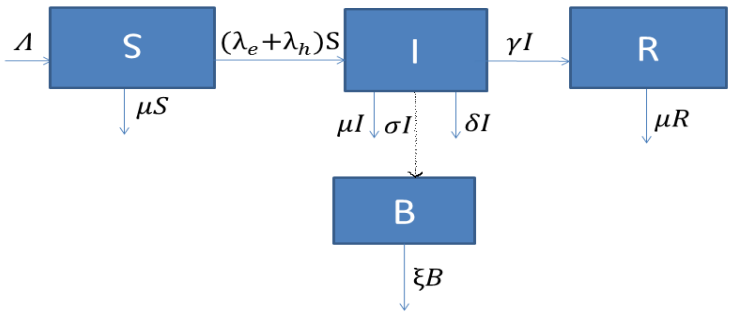

FIGURE 3.1: Flow chart diagram

3.2.4 Model Equations

By including the effects of media coverage on cholera transmission the rates of λ_e and λ_h now become,

$$\lambda_e = \beta_e - \frac{\rho\beta_e B}{\kappa + B} \quad and \quad \lambda_h = \beta_h - \frac{\rho\beta_h I}{m + I}$$

The flow diagram then gives rise to the following system of ordinary differential equations.

$$\begin{aligned}
\frac{dS(t)}{dt} &= \Lambda - (\beta_e - \rho\beta_e)\frac{S(t)B(t)}{\kappa + B(t)} - (\beta_h - \frac{\rho\beta_h I(t)}{m + I(t)})S(t)I(t) - \mu S(t) \\
\frac{dI(t)}{dt} &= (\beta_e - \rho\beta_e)\frac{S(t)B(t)}{\kappa + B(t)} + (\beta_h - \frac{\rho\beta_h I(t)}{m + I(t)})S(t)I(t) - (\gamma + \mu + \delta)I(t) \\
\frac{dB(t)}{dt} &= \sigma I(t) - \xi B(t) \\
\frac{dR(t)}{dt} &= \gamma I(t) - \mu R(t)
\end{aligned} \tag{3.2}$$

These equations form the model system. For simplicity in working out we let $S(t) = S, B(t) = B, I(t) = I, R(t) = R$, so that our new system becomes.

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - (\beta_e - \rho\beta_e)\frac{SB}{\kappa + B} - (\beta_h - \frac{\rho\beta_h I}{m + I})SI - \mu S \\
\frac{dI}{dt} &= (\beta_e - \rho\beta_e)\frac{SB}{\kappa + B} + (\beta_h - \frac{\rho\beta_h I}{m + I})SI - (\gamma + \mu + \delta)I \\
\frac{dB}{dt} &= \sigma I - \xi B \\
\frac{dR}{dt} &= \gamma I - \mu R
\end{aligned} \tag{3.3}$$

3.3 Model Analysis

We establish the well posedness of the model by showing that its solutions are positive and bounded.

3.3.1 Boundedness of Solutions

We show that our solutions are bounded in the invariant region Ω where $\Omega = \{(S, I, R, B) : N \leq \frac{\Lambda}{\mu}\}$.

Theorem 3.1. *The solutions of the model are contained in the feasible region* Ω.

Proof. Taking the time derivative of N(t) from (3.3) we have

$$\frac{dN}{dt} = \Lambda - \mu S - (\gamma + \mu + \delta)I + \gamma I - \mu R \tag{3.4}$$

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

$$\frac{dN}{dt} \leq \Lambda - \mu N$$

$$\frac{dN}{dt} + \mu N \leq \Lambda \tag{3.5}$$

Using the integrating factor $e^{\mu t}$ to solve (3.5) we get

$$N \leq \frac{\Lambda}{\mu} + e^{-\mu t}C$$

At $t = 0$

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

$$N \leq \frac{\Lambda}{\mu} + (N(0) - \frac{\Lambda}{\mu})e^{-\mu t}$$

where N(0) is the initial population. As t$\rightarrow \infty$

$$\lim_{t\to\infty} N(t) \leq \frac{\Lambda}{\mu}$$

which implies

$$0 \leq N \leq \frac{\Lambda}{\mu}.$$

The solutions are bounded in the invariant region Ω. □

3.3.2 Positivity of Solutions

This model monitors a population, therefore, we assume that all associated parameters are non-negative at all times for all $t \geq 0$. We show that all state variables in the equations above will remain non-negative and that all the solutions with positive initial data, will remain positive for $t \geq 0$.

Theorem 3.2. *Let the initial conditions be* $\{S(0), (B(0), I(0), R(0)) \geq 0\} \in \Omega$, *then the solution set* $\{S(t), I(t), B(t), R(t)\}$ *of the model system is positive for all* $t > 0$.

Proof. From the first equation of (3.3)

$$\frac{dS}{dt} = \Lambda - (\beta_e - \rho\beta_e)\frac{SB}{\kappa + B} - (\beta_h - \frac{\rho\beta_h I}{m + I})SI - \mu S$$

$$\frac{dS}{dt} \geq -(\beta_e - \rho\beta_e)\frac{SB}{\kappa + B} - (\beta_h - \frac{\rho\beta_h I}{m + I})SI - \mu S$$

$$= -[(\beta_e - \rho\beta_e)\frac{B}{\kappa + B} + (\beta_h - \frac{\rho\beta_h I}{m + I})I + \mu]S.$$

Separating by variables and integrating both sides gives

$$\int \frac{dS}{S} \geq -\int [(\beta_e - \rho\beta_e)\frac{B}{\kappa + B} + (\beta_h - \frac{\rho\beta_h I}{m + I})I + \mu]dt$$

$$\ln S \geq -[(\beta_e - \rho\beta_e)\frac{B}{\kappa + B} + (\beta_h - \frac{\rho\beta_h I}{m + I})I + \mu]t + c_1$$

$$S(t) \geq e^{-[(\beta_e - \rho\beta_e)\frac{B}{\kappa+B} + (\beta_h - \frac{\rho\beta_h I}{m+I})I + \mu]t} \times e^{c_1}$$

$$S(t) \geq e^{-[(\beta_e - \rho\beta_e)\frac{B}{\kappa+B} + (\beta_h - \frac{\rho\beta_h I}{m+I})I + \mu]t} \times K$$

where $K = e^{c_1}$

$$S(t) \geq Ke^{-[(\beta_e - \rho\beta_e)\frac{B}{\kappa+B} + (\beta_h - \frac{\rho\beta_h I}{m+I})I + \mu]t}.$$

Using the initial condition $t = 0$ we have $S(0) = K$ which implies that

$$S(t) \geq S(0)e^{-[(\beta_e - \rho\beta_e)\frac{B}{\kappa+B} + (\beta_h - \frac{\rho\beta_h I}{m+I})I + \mu]t}.$$

Therefore $S(t) \geq 0$ for all $t \geq 0$.

From the second equation of (3.3)

$$\frac{dI}{dt} = (\beta_e - \rho\beta_e)\frac{SB}{\kappa + B} + (\beta_h - \frac{\rho\beta_h I}{m + I})SI - (\gamma + \mu + \delta)I$$

we have,

$$\frac{dI}{dt} \geq -(\gamma + \mu + \delta)I.$$

We integrate by separation of variables to get;

$$\int \frac{dI}{I} \geq \int -(\gamma + \mu + \delta)dt$$

$$lnI(t) \geq -(\gamma + \mu + \delta)t + c_2$$

$$I(t) \geq e^{-(\gamma+\mu+\delta)t+c_2}$$

$$I(t) \geq e^{-(\gamma+\mu+\delta)t} \times e^{c_2}$$

$$I(t) \geq e^{-(\gamma+\mu+\delta)t} \times K_1$$

where $K_1 = e^{c_2}$. Therefore,

$$I(t) \geq K_1 e^{-(\gamma+\mu+\delta)t}.$$

At $t = 0$ we have $I(0) = K_1$, hence

$$I(t) \geq I(0)e^{-(\gamma+\mu+\delta)t}$$

Which implies that;

I(t)$\geq$ 0 for all $t \geq 0$

From the third equation of (3.3),

$$\frac{dB}{dt} = \sigma I - \xi B$$

$$\frac{dB}{dt} \geq -\xi B$$

Separation of variables and integration gives

$$\int \frac{dB}{B} \geq -\int \xi dt$$

$$lnB(t) \geq -\xi t + c_3$$

$$B(t) \geq e^{-\xi t} \times e^{c_3}$$

$$B(t) \geq K_2 e^{-\xi t}$$

where $K_2 = e^{c_3}$, at $t = 0$ we have $B(0) = K_2$ which implies that

$$B(t) \geq B(0)e^{-\xi t}$$

and therefore $B(t) \geq 0$ for all $t \geq 0$.

From the fourth equation of (3.3)

$$\frac{dR}{dt} = \gamma I - \mu R$$

we have

$$\frac{dR}{dt} \geq -\mu R$$

Separating by variables and integrating gives

$$\int \frac{dR}{R} \geq \int -\mu dt$$

$$\ln R(t) \geq -\mu(t) + c_4$$

$$R(t) \geq e^{-\mu(t)+c_4}$$

$$R(t) \geq e^{-\mu(t)} \times e^{c_4}$$

$$R(t) \geq e^{-\mu(t)} \times K_3$$

$$R(t) \geq K_3 e^{-\mu(t)}$$

where $K_3 = e^{c_4}$. At $t = 0$ we have $R(0) = K_3$, hence

$$R(t) \geq R(0)e^{-\mu(t)}$$

which implies that;
$R(t) \geq 0$ for all $t \geq 0$.
All the state variables are positive for all time t. The solutions are therefore non negative for t$\geq$ 0 and are bounded in the invariant region Ω and thus the model is mathematically well posed and biologically meaningful in the feasible region Ω. □

3.4 Stability Analysis of Equilibrium Points

An equilibrium is defined as a constant solution of a model system. The equilibrium points of the model system are obtained by setting the right hand side of the differential equations to zero and solving each to get a constant solution. These points are also reffered to as steady state solutions. Epidemiological models usually have two equilibrium points, namely, the disease free equilibrium and the endemic equilibrium.

The existence of the equilibrium points of the model is determined with respect to the basic reproduction number, derived using the next generation matrix ap-

proach. Stability analysis of the model is done to determine the conditions for the spread of the disease in a given population. Since $R(t) = N(t) - S(t) - I(t)$, we will leave it out of the stability analysis of equilibrium points.

3.4.1 Disease Free Equilibrium Point(DFE)

The disease-free equilibrium (E^0) is a point where the disease is not present in the population. This means that cholera is absent in the human population and the environment and therefore I, B and $R = 0$ because there is no disease to recover from. From our model equations, we get

$$\begin{aligned} S'(t) &= \Lambda - \mu S \\ I'(t) &= 0 \\ B'(t) &= 0 \end{aligned}$$

Thus, the disease free equilibrium of the system (E^0) is given by $(\frac{\Lambda}{\mu}, 0, 0)$. The basic reproduction number R_0 is the threshold parameter that is used in determining the dynamics of a model. When $R_0 < 1$ the disease-free equilibrium is said to be locally asymptotically stable while when $R_0 > 1$ it is said to be unstable.

3.4.2 Basic Reproduction Number

The basic reproduction number; commonly denoted as R_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 (Van den Driessche & Watmough, 2002). R_0 measures the potential of the bacteria to spread within the human population. This number is very important because it is directly related to the effort required to eliminate an infection. If $R_0 < 1$, then 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.

The basic reproduction number depends mainly on the definition of the infected and uninfected compartments. We determine R_0 using the next generation matrix approach (Van den Driessche & Watmough, 2002). Consider the next generation matrix G made up of two $m \times m$ matrices F and V, such that

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

and

$$F = \left[\begin{array}{c} \frac{\partial \mathcal{F}_i(x_0)}{\partial x_j} \end{array} \right]$$

$$V = \left[\begin{array}{c} \frac{\partial \mathcal{V}_i(x_0)}{\partial x_j} \end{array} \right]$$

Here, F is defined as the Jacobian of $\mathcal{F}_i$, such that f_i is the rate of appearance of new infections in compartment i. V is the Jacobian of $\mathcal{V}_i$, such that v_i is the rate of transfer of individuals from compartment i by all other means and x_o is the disease free equilibrium. The basic reproduction number R_0 is given as the dominant eigen value or the spectral radius of matrix G.

$$R_0 = \rho F V^{-1} \tag{3.6}$$

We use the the second and third equation of system (3.3) to compute R_0

$$\begin{aligned} \frac{dI}{dt} &= (\beta_e - \rho\beta_e)\frac{SB}{\kappa + B} + (\beta_h - \frac{\rho\beta_h I}{m + I})SI - (\gamma + \mu + \delta)I \\ \frac{dB}{dt} &= \sigma I - \xi B \end{aligned}$$

From the two equations we get our $\mathcal{F}$ and $\mathcal{V}$ which are given by

$$\mathcal{F} = \left[\begin{array}{c} (\beta_e - \rho\beta_e)\frac{SB}{\kappa + B} + (\beta_h - \frac{\rho\beta_h I}{m+I})SI \\ 0 \end{array} \right]$$

$$\mathcal{V} = \begin{bmatrix} (\gamma + \mu + \delta)I \\ -\sigma I + \xi B \end{bmatrix}$$

Calculating the Jacobian matrix at the disease free equilibrium gives us

$$F = \begin{bmatrix} \beta_h \frac{\Lambda}{\mu} & (\beta_e - \rho\beta_e)\frac{\Lambda}{\mu\kappa} \\ 0 & 0 \end{bmatrix}$$

And

$$V = \begin{bmatrix} \gamma + \mu + \delta & 0 \\ -\sigma & \xi \end{bmatrix}$$

On solving for the inverse of the matrix V, get

$$V^{-1} = \begin{bmatrix} \frac{1}{(\gamma+\mu+\delta)} & 0 \\ \frac{\sigma}{\xi(\gamma+\mu+\delta)} & \frac{1}{\xi} \end{bmatrix}$$

Therefore

$$FV^{-1} = \begin{bmatrix} \frac{\beta_h\Lambda}{\mu(\gamma+\mu+\delta)} + (\beta_e - \rho\beta_e)\frac{\Lambda\sigma}{\mu\kappa\xi(\gamma+\mu+\delta)} & (\beta_e - \rho\beta_e)\frac{\Lambda}{\mu\kappa\xi} \\ 0 & 0 \end{bmatrix}$$

$$R_0 = \frac{\Lambda}{\mu(\gamma + \delta + \mu)}(\beta_h + \frac{(\beta_e - \rho\beta_e)\sigma}{\kappa\xi})$$

3.4.3 Local Stability of the Disease Free Equiibrium

The following result is a proof of local stability of the disease free equilibrium and the proof applies techniques used in (Van den Driessche & Watmough, 2002).

Theorem 3.3. *: The disease free equilibrium is locally asymptotically stable if* $R_0 < 1$ *and is unstable if* $R_0 > 1$

Proof. To show this we first evaluate the Jacobian matrix of system at the DFE.

$$J = \begin{bmatrix} -\mu & -\beta_h\frac{\Lambda}{\mu} & -(\beta_e - \rho\beta_e)\frac{\Lambda}{\mu\kappa} \\ 0 & \beta_h\frac{\Lambda}{\mu} - (\gamma + \mu + \delta) & (\beta_e - \rho\beta_e)\frac{\Lambda}{\mu\kappa} \\ 0 & \sigma & -\xi \end{bmatrix}$$

One of the eigen values is $-\mu$, we find the other eigen values by checking the signs of the eigen values of the reduced block matrix given by

$$\begin{bmatrix} \beta_h \frac{\Lambda}{\mu} - (\gamma + \mu + \delta) & (\beta_e - \rho\beta_e)\frac{\Lambda}{\mu\kappa} \\ \sigma & -\xi \end{bmatrix}$$

Now let Tr be the Trace of A and α be the Determinant of A and consider the linear system $x'(t) = Ax(t)$ where

$$A = \begin{bmatrix} a & b \\ c & d \end{bmatrix}$$

The following conditions can be shown.

(a) If $\alpha < 0$, the characteristic roots of A will have opposite signs.

(b) If $\alpha > 0$ and $\Delta = Tr^2 - 4\alpha \geq 0$, the characteristic roots of matrix A will have the same sign. The roots will be negative if $Tr < 0$ and positive if $Tr > 0$.

(c) If $\alpha > 0$, $\Delta < 0$ and $Tr \neq 0$,then the characteristic roots of A will be imaginary with negative real part if $Tr < 0$ and a positive real part if $Tr > 0$.

(d) If $\alpha > 0$ and $Tr = 0$, matrix A will have purely imaginary roots.

The eigen values of Matrix A are obtained from the characteristic equation

$$\lambda^2 - (a + d)\lambda + (ad - bc) = 0$$

$$\lambda^2 - Tr\lambda + \alpha = 0$$

$$\lambda = \frac{Tr \pm \sqrt{Tr^2 - 4\alpha}}{2}$$

Thus

(a*) If $\alpha < 0$, there exists two real eigen values of opposite signs.

(b*) If $\alpha > 0$ and $\Delta \geq 0$, there exists two real eigen values of the same sign as the Trace.

(c*) If $\alpha > 0$, $\Delta < 0$ and $Tr \neq 0$,there exists two complex conjugate eigen values $\lambda = p \pm ir$.

(d*) If $\alpha > 0$ and $Tr = 0$, there exists two purely imaginary complex conjugate eigen values.

Using conditions (b) we can now determine the signs of the other eigen values. For the two remaining eigen values to be negative, then $\alpha > 0$ and $Tr < 0$. We now find the conditions that make the determinant positive and the trace negative. From the reduced block matrix the determinant is given by

$$-\xi\left[\beta_h\frac{\Lambda}{\mu} - (\gamma + \mu + \delta)\right] - (\beta_e - \rho\beta_e)\frac{\sigma\Lambda}{\mu\kappa} \tag{3.7}$$

If we let $(\beta_e - \rho\beta_e) = Q$ we will have,

$$-\xi\left[\beta_h\frac{\Lambda}{\mu} - (\gamma + \mu + \delta)\right] - \frac{\sigma Q\Lambda}{\mu\kappa}$$

For the determinant to be positive

$$\xi\beta_h\frac{\Lambda}{\mu} + \frac{\sigma Q\Lambda}{\mu\kappa} < \xi(\gamma + \mu + \delta) \tag{3.8}$$

Dividing both sides of (3.8) by $\xi(\gamma + \mu + \delta)$ gives

$$\frac{\Lambda}{\mu\xi(\gamma + \delta + \mu)}(\xi\beta_h + \frac{\sigma Q}{\kappa}) < 1$$

$$\frac{\Lambda}{\mu(\gamma + \delta + \mu)}(\beta_h + \frac{(\beta_e - \rho\beta_e)\sigma}{\kappa\xi}) < 1 \tag{3.9}$$

Thus $R_o < 1$.

The trace of the reduced block matrix is given by

$$\beta_h \frac{\Lambda}{\mu} - \xi - (\gamma + \mu + \delta). \tag{3.10}$$

If we make $\gamma + \mu + \delta$ be the subject of the formula and $(\beta_e - \rho\beta_e) = Q$ from the basic reproduction number, we get

$$\gamma + \mu + \delta = \frac{\Lambda}{\mu R_0}\left[\beta_h + \frac{Q\sigma}{\kappa\xi}\right]. \tag{3.11}$$

Substituting (3.11) in (3.10) gives us

$$\beta_h \frac{\Lambda}{\mu} - \left[\frac{\Lambda}{\mu R_0}(\beta_h + \frac{Q\sigma}{\kappa\xi})\right] - \xi. \tag{3.12}$$

The trace needs to be negative for us to have negative eigen values. Since all the other parameters are negative, we find the conditions that make

$$\beta_h \frac{\Lambda}{\mu} - \frac{\Lambda}{\mu R_0}\beta_h \tag{3.13}$$

negative.

We can simplify (3.13) to get

$$\beta_h \frac{\Lambda}{\mu}\left[1 - \frac{1}{R_0}\right] < 0. \tag{3.14}$$

Equation 3.14 can only be negative if $R_0 < 1$.

It can be seen that the Jacobian matrix of the disease free equilibrium has negative eigen values only when $R_0 < 1$ and therefore, the DFE is locally asymptotically stable. The results of the theorem therefore confirm the conditions for local stability as outlined in (Van den Driessche & Watmough, 2002). □

In terms of the disease spread, this means that, if there is a small pertubation on the system, the system will still return to the disease free equilibrium.

3.4.4 Global Stability of the DFE

We use the Castillo-Chavez theorem (Castillo-Chávez, Feng, & Huang, 2002) to investigate the global asymptotic stability of the disease free state. For the theorem to work, we rewrite (3.3) in the form

$$\begin{aligned} \frac{dX}{dt} &= H(X,Z) \\ \frac{dZ}{dt} &= G(X,Z),\ G(X,0)=0 \end{aligned} \tag{3.15}$$

where $X = S$ and $Z = (I, B)$. Here the components of $X \in \mathbb{R}$ denote the uninfected individuals and the components of $Z \in \mathbb{R}^2$ denote the infected individuals. The disease free equilibrium of the system now becomes $E^0 = (X^*, 0)$, $X^* = \frac{\Lambda}{\mu}$. To guarantee local asymptotic stability, the following two conditions must be met.

1. $\frac{dX}{dt} = H(X,0)$, X^* is globally asymptotically stable (GAS)
2. $G(X,Z) = PZ - \widehat{G}(X,Z)$, $\widehat{G}(X,Z) \geq 0$ for $(X,Z) \in \Omega$

where $P = D_Z G(X^*, 0)$ is an M matrix (the off diagonal elements of P are non-negative) and Ω is the region where the model is biologically meaningful. If the system (3.15) satisfies conditions 1 and 2 then the following theorem holds.

Theorem 3.4. *The fixed point $E^0 = (X^*, 0)$ is a globally asymptotic stable equilibrium of (3.15) provided that $R_0 < 1$ and the assumptions 1 and 2 are satisfied.*

Proof. Since $X = S$ and $Z = (I, B)$, then

$$H(X,0) = \left[\ \Lambda - \mu S\ \right]$$

$$G(X,Z) \quad = \quad = PZ - \widehat{G}(X,Z) \tag{3.16}$$

where

$$P = \begin{bmatrix} \beta_h S - (\gamma + \mu + \delta) & (\beta_e - \rho\beta_e)\frac{S}{\kappa} \\ \sigma & -\xi \end{bmatrix}$$

and the set $Z = (I, B)$ is expressed as a column matrix to get

$$PZ = \begin{bmatrix} \beta_h SI - (\gamma + \mu + \delta)I + (\beta_e - \rho\beta_e)\frac{SB}{\kappa} \\ \sigma I - \xi B \end{bmatrix}$$

$$G(X,Z) = \begin{bmatrix} (\beta_e - \rho\beta_e)\frac{SB}{\kappa+B} + (\beta_h - \frac{\rho\beta_h I}{m+I})SI - (\gamma + \mu + \delta)I \\ \sigma I - \xi B \end{bmatrix}$$

and

$$\widehat{G}(X,Z) = \begin{bmatrix} (\frac{\rho\beta_h I}{m+I})SI + (\beta_e - \rho\beta_e)\frac{SB^2}{\kappa(\kappa+B)} \\ 0 \end{bmatrix}$$

Since $0 < \rho < 1$, then $\widehat{G}(X,Z) \geq 0$. The conditions 1 and 2 have been met and therefore E^0 is globally asymptotically stable. □

This means that if there is a large perturbation on the system, it will still return to the disease free equilibrium.

3.4.5 Endemic Equilibrium Point

This is where the basic reproduction number becomes greater than one and therefore, the disease spreads in the susceptible population. We denote our endemic equilibrium point as $E^* = (S^*, I^*, B^*)$. To find this equilibrium point we equate the right hand side of (3.3) to zero to get.

$$\begin{aligned} \Lambda - (\beta_e - \rho\beta_e)\frac{SB}{\kappa + B} - (\beta_h - \frac{\rho\beta_h I}{m+I})SI - \mu S &= 0 \\ (\beta_e - \rho\beta_e)\frac{SB}{\kappa + B} + (\beta_h - \frac{\rho\beta_h I}{m+I})SI - (\gamma + \mu + \delta)I &= 0 \\ \sigma I - \xi B &= 0 \end{aligned} \tag{3.17}$$

From this we get

$$B^* = \frac{\sigma}{\xi} I^* \tag{3.18}$$

$$S^* = \frac{\Lambda}{\mu} - \frac{(\gamma + \mu + \delta) I^*}{\mu} \tag{3.19}$$

Substituting (3.18) and (3.19) in the second equation of (3.17) and solving gives us $I^* = 0$ and

$$AI^{*3} + BI^{*2} + CI^* + D = 0 \tag{3.20}$$

where

$$A = (\rho - 1)(\gamma + \mu + \delta)\beta_h \sigma$$

$$B = (1 - \rho)\beta_h \Lambda \sigma - (\gamma + \mu + \delta)[(\beta_e - \rho\beta_e)\sigma + m\beta_h \sigma + \beta_h \kappa\xi - \rho\beta_h \kappa\xi + \mu\sigma]$$

$$C = (\beta_e - \rho\beta_e)\Lambda\sigma + [m\Lambda\sigma + \Lambda\kappa\xi - \rho\Lambda\kappa\xi]\beta_h - (\gamma + \mu + \delta)[m(\beta_e - \rho\beta_e)\sigma + m\beta_h \kappa\xi + m\mu\sigma + \mu\kappa\xi]$$

$$D = m(\beta_e - \rho\beta_e)\Lambda\sigma + m\beta_h \Lambda\kappa\xi - m(\gamma + \mu + \delta)\mu\kappa\xi$$

The endemic equilibrium of the system exists if the roots of $AI^{*3}+BI^{*2}+CI^*+D = 0$ are real and positive. We use the Descartes' rule of signs to check the possible number of real roots of our polynomial.

Theorem 3.5. *The number of positive real roots of a polynomial is equal to the number of sign changes in the coefficients of the terms.*

Proof. We first check the sign of A which is the coefficient of I^3. Since all the parameters used are positive and $0 < \rho < 1$, them $\rho - 1$ is negative and the sign becomes negative.
Next we check the sign of D.

$$D = m(\beta_e - \rho\beta_e)\Lambda\sigma + m\beta_h \Lambda\kappa\xi - m(\gamma + \mu + \delta)\mu\kappa\xi \tag{3.21}$$

Dividing through (3.21) by

$$\frac{m(\gamma+\mu+\delta)\mu\kappa\xi}{m(\gamma+\mu+\delta)\mu\kappa\xi}$$

gives us

$$\left[\left[\frac{\Lambda}{\mu(\gamma+\delta+\mu)}(\beta_h+\frac{(\beta_e-\rho\beta_e)\sigma}{\kappa\xi})\right]-1\right]m(\gamma+\mu+\delta)\mu\kappa\xi \tag{3.22}$$

Since R_0 is

$$\left[\frac{\Lambda}{\mu(\gamma+\delta+\mu)}(\beta_h+\frac{(\beta_e-\rho\beta_e)\sigma}{\kappa\xi})\right]$$

(3.22) becomes

$$(R_0-1)[m(\gamma+\mu+\delta)\mu\kappa\xi] \tag{3.23}$$

The endemic equilibrium exists when $R_0 > 1$ therefore, D is positive. Now that A is negative and D is positive, we can conclude that there will be a sign change regardless of the sign of B and C. Equation (3.20) has atleast one positive real root and hence the endemic equilibrium exists. □

3.4.6 Local Stability of the Endemic Equilibrium

Theorem 3.6. *The endemic equilibrium E^* of our system is locally asymptotically stable when $R_0 > 1$.*

Proof. For the endemic equilibrium to be asympotically stable, we show that the eigen values of the Jacobian matrix will have negative real parts. The Jacobian matrix of the system evaluated at the endemic equilibrium is given by

$$\begin{bmatrix} -\frac{(\beta_e-\rho\beta_e)B^*}{\kappa+B^*}-(\beta_h-\frac{\rho\beta_h I^*}{m+I^*})I^*-\mu & -[\beta_h-\frac{\rho\beta_h I^*(2m+I^*)}{(m+I^*)^2}]S^* & -\frac{(\beta_e-\rho\beta_e)s^*\kappa}{(\kappa+B^*)^2} \\ \frac{(\beta_e-\rho\beta_e)B^*}{\kappa+B^*}+(\beta_h-\frac{\rho\beta_h I^*}{m+I^*})I^* & [\beta_h-\frac{\rho\beta_h I^*(2m+I^*)}{(m+I^*)^2}]S^*-(\gamma+\mu+\delta) & \frac{(\beta_e-\rho\beta_e)s^*\kappa}{(\kappa+B^*)^2} \\ 0 & \sigma & -\xi \end{bmatrix}$$

if we take

$$X = \frac{(\beta_e - \rho\beta_e)B^*}{\kappa + B^*} + (\beta_h - \frac{\rho\beta_h I^*}{m + I^*})I^* \tag{3.24}$$

$$Y = [\beta_h - \frac{\rho\beta_h I^*(2m + I^*)}{(m + I^*)^2}]S^* \tag{3.25}$$

$$Z = \frac{(\beta_e - \rho\beta_e)S^*\kappa}{(\kappa + B^*)^2} \tag{3.26}$$

Then the matrix is simplified to

$$J(E^*) = \begin{bmatrix} -X - \mu & -Y & -Z \\ X & Y - (\gamma + \mu + \delta) & Z \\ 0 & \sigma & -\xi \end{bmatrix}$$

The characteristic equation of the Jacobian matrix at $J(E^*)$ is given by

$$a_0\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0$$

where

$$\begin{aligned} a_0 &= 1 \\ a_1 &= -Y + (\gamma + \mu + \delta) + \xi + (X + \mu) \\ a_2 &= (X + \mu)[-Y + (\gamma + \mu + \delta) + \xi] - Y\xi + (\gamma + \mu + \delta)\xi - \sigma Z + XY \\ a_3 &= (X + \mu)[-Y\xi + (\gamma + \mu + \delta)\xi - \sigma Z] - X\sigma Z + XY\xi \end{aligned}$$

We use the Routh Hurwitz stability criterion to show that the roots of the characteristic equation are negative. As per the criterion, the following is true.

All the roots of a polynomial with real coefficients have negative real parts when all the coeffients are positive and $a_1a_2 - a_3 > 0$.
We show that $a_1 > 0, a_2 > 0, a_3 > 0$. At the endemic equilibrium the right hand side of the second and third equation of (3.3) becomes zero, giving us

$$(\gamma+\mu+\delta)I^* = (\beta_e - \rho\beta_e)\frac{S^*B^*}{\kappa+B^*} + (\beta_h - \frac{\rho\beta_h I^*}{m+I^*})S^*I^* \tag{3.27}$$

Substituting (3.18) in (3.27) and dividing through by I^* yields

$$(\gamma+\mu+\delta) = (\beta_e - \rho\beta_e)\frac{S^*\sigma}{\kappa\xi+\sigma I^*} + (\beta_h - \frac{\rho\beta_h I^*}{m+I^*})S^*. \tag{3.28}$$

We also substitute (3.18) in (3.24) and (3.26) to get

$$X = \frac{(\beta_e - \rho\beta_e)\sigma I^*}{\kappa\xi+\sigma I^*} + (\beta_h - \frac{\rho\beta_h I^*}{m+I^*})I^* \tag{3.29}$$

$$Y = \left[\beta_h - \frac{\rho\beta_h I^*(2m+I^*)}{(m+I^*)^2}\right]S^* \tag{3.30}$$

$$Z = \frac{(\beta_e - \rho\beta_e)S^*\kappa\xi}{(\kappa\xi+\sigma I^*)^2} \tag{3.31}$$

X, Y and Z are all positive, therefore, we show that all the other coefficients are also positive. For a_1 to be positive we need to show that

$$-Y + (\gamma+\mu+\delta) + \xi > 0 \tag{3.32}$$

Substituting (3.28) and (3.30) in (3.32) gives us

$$-\left[\beta_h - \frac{\rho\beta_h I^*(2m+I^*)}{(m+I^*)^2}\right]S^* + (\beta_e - \rho\beta_e)\frac{S^*\sigma}{\kappa\xi+\sigma I^*} + (\beta_h - \frac{\rho\beta_h I^*}{m+I^*})S^* + \xi. \tag{3.33}$$

If

$$\left[\beta_h - \frac{\rho\beta_h I^*(2m+I^*)}{(m+I^*)^2}\right] < \left[\beta_h - \frac{\rho\beta_h I^*}{m+I^*}\right] \tag{3.34}$$

then (3.32) will be positive. (3.34) can be reduced to

$$\frac{2m+I^*}{(m+I^*)^2} > \frac{1}{m+I^*}. \tag{3.35}$$

This shows that (3.32) is positive and therefore $a_1 > 0$.

Next we show that $a_2 > 0$. Since we have already proved that $-Y+(\gamma+\mu+\delta)+\xi >$

0, it suffices to show that $-Y\xi + (\gamma + \mu + \delta)\xi - \sigma Z > 0$, that is,

$$Y\xi + \sigma z < (\gamma + \mu + \delta)\xi \tag{3.36}$$

Substituting (3.30) and (3.31) in (3.36) gives us

$$\left[\beta_h - \frac{\rho\beta_h I^*(2m + I^*)}{(m + I^*)^2}\right] S^*\xi + (\beta_e - \rho\beta_e)\frac{S^*\sigma\kappa\xi}{(\kappa\xi + \sigma I^*)^2} < (\beta_e - \rho\beta_e)\frac{S^*\sigma\xi}{\kappa\xi + \sigma I^*} + (\beta_h - \frac{\rho\beta_h I^*}{m + I^*})S^*\xi \tag{3.37}$$

Since $S^*\xi$ is common, we divide through by it to get

$$\left[\beta_h - \frac{\rho\beta_h I^*(2m + I^*)}{(m + I^*)^2}\right] + (\beta_e - \rho\beta_e)\frac{\sigma\kappa}{(\kappa\xi + \sigma I^*)^2} < (\beta_e - \rho\beta_e)\frac{\sigma}{\kappa\xi + \sigma I^*} + \left[\beta_h - \frac{\rho\beta_h I^*}{m + I^*}\right] \tag{3.38}$$

Considering the proof of (3.34) we are left to show that

$$\left[(\beta_e - \rho\beta_e)\frac{\sigma\kappa}{(\kappa\xi + \sigma I^*)^2}\right] < \left[(\beta_e - \rho\beta_e)\frac{\sigma\xi}{\kappa\xi + \sigma I^*}\right] \tag{3.39}$$

We can simplify (3.39) to

$$\frac{\kappa}{(\kappa\xi + \sigma I^*)^2} < \frac{1}{\kappa\xi + \sigma I^*} \tag{3.40}$$

which holds hence $a_2 > 0$. Using the same method we can show in a similar way that $a_3 > 0$. We now show that $a_1 a_2 - a_3 > 0$. let

$$\begin{aligned} a &= -Y + (\gamma + \mu + \delta) + \xi \\ b &= X + m \\ c &= -Y\xi + (\gamma + \mu + \delta)\xi - \sigma Z \end{aligned}$$

Substituting this in $a_1 a_2 - a_3 > 0$, we get

$$(a + b)ba + c(a + b) + (a + b)XY - (bc - X\sigma Z + XY\xi) > 0 \tag{3.41}$$

since all other parameters are positive we simplify and show that

$$[(a + b) - \xi]XY > 0 \tag{3.42}$$

This is true if $(a+b)-\xi$ is positive. Resubstituting the values of a and b in (3.42) gives,

$$\Big[-Y+(\gamma+\mu+\delta)+\xi+X+\mu-\xi\Big] \tag{3.43}$$

which is positive since $X > Y$ and therefore, $a_1a_2 - a_3 > 0$ holds.

It can now be seen that all the coefficients are positive and therefore the eigen values of the characteristic polynomial have negative real parts and the endemic equilibrium is locally asymptotically stable. □

3.5 Sensitivity Analysis

The basic reproduction number is very important in the effort required to eradicate a disease. We carry out sensitivity analysis of the Basic reproduction number with respect to the model parameters to access the relative impact of each of the parameters in the transmission and prevalence of the disease. This will enable us to determine which intervention strategy is most effective in the control of cholera transmission. The normalized forward sensitivity index is used to calculate sensitivity.

We define the normalized forward sensitivity index of the basic reproduction number with respect to a parameter A (Sirajo, Niniuola, Omotayo, & Usman, 2013) as

$$S_A^{R_0} = \frac{\partial R_0}{\partial A} \times \frac{A}{R_0}$$

Therefore, the sensitivity index of R_0 on parameter B_h is given by

$$S_{\beta_h}^{R_0} = \frac{\partial R_0}{\partial \beta_h} \times \frac{\beta_h}{R_0}$$

where

$$\frac{\beta_h}{R_0} = \frac{\beta_h \kappa \xi \mu(\gamma+\delta+\mu)}{\Lambda[\kappa\xi\beta_h + (\beta_e - \rho\beta_e)]}$$

$$\frac{\partial R_0}{\partial \beta_h} = \frac{\Lambda}{\mu(\gamma+\delta+\mu)}$$

thus

$$S_{\beta_h}^{R_0} = \frac{\Lambda}{\mu(\gamma+\delta+\mu)} \times \frac{\beta_h\kappa\xi\mu(\gamma+\delta+\mu)}{\Lambda[\kappa\xi\beta_h+(\beta_e-\rho\beta_e)]}$$

$$= \frac{\beta_h\kappa\xi}{\kappa\xi\beta_h+(\beta_e-\rho\beta_e)\sigma}$$

Likewise, the sensitivity index of ρ is given by

$$S_{\rho}^{R_0} = \frac{\partial R_0}{\partial \rho} \times \frac{\rho}{R_0}$$

where

$$\frac{\rho}{R_0} = \frac{\rho\kappa\xi\mu(\gamma+\delta+\mu)}{\Lambda[\kappa\xi\beta_h+(\beta_e-\rho\beta_e)\sigma]}$$

$$\frac{\partial R_0}{\partial \rho} = \frac{-\Lambda\sigma\beta_e}{\kappa\xi\mu(\gamma+\delta+\mu)}$$

Therefore

$$S_{\rho}^{R_0} = \frac{-\Lambda\sigma\beta_e}{\kappa\xi\mu(\gamma+\delta+\mu)} \times \frac{\rho\kappa\xi\mu(\gamma+\delta+\mu)}{\Lambda[\kappa\xi\beta_h+(\beta_e-\rho\beta_e)\sigma]}$$

$$= \frac{-\rho\beta_e\sigma}{\kappa\xi\beta_h+(\beta_e-\rho\beta_e)\sigma}$$

Similarly, we have the following sensitivity indices for the other parameters

$$S_{\beta_e}^{R_0} = \frac{(\beta_e-\rho\beta_e)\sigma}{\kappa\xi\beta_h+(\beta_e-\rho\beta_e)\sigma}$$

$$S_{\sigma}^{R_0} = \frac{(\beta_e-\rho\beta_e)\sigma}{\kappa\xi\beta_h+(\beta_e-\rho\beta_e)\sigma}$$

$$S_{\Lambda}^{R_0} = 1$$

$$S_{\kappa}^{R_0} = \frac{-(\beta_e-\rho\beta_e)\sigma}{\kappa\xi\beta_h+(\beta_e-\rho\beta_e)\sigma}$$

$$S_{\xi}^{R_0} = \frac{-(\beta_e-\rho\beta_e)\sigma}{\kappa\xi\beta_h+(\beta_e-\rho\beta_e)\sigma}$$

$$S_{\mu}^{R_0} = \frac{-(\gamma+\delta+2\mu)}{(\gamma+\delta+\mu)}$$

$$S_{\gamma}^{R_0} = \frac{-\gamma}{\gamma+\delta+\mu}$$

$$S_{\delta}^{R_0} = \frac{-\delta}{\gamma+\delta+\mu}$$

Remark 3.7

From the results of Sirajo et al. (2013), it follows that, a positive index sign indicates that an increase in the parameter's value will result in an increase in the value of the reproduction number and a reduction in the parameter's value will reduce the value of the reproduction number. A negative index sign indicates that an increase in the parameter's value will result in a reduction in the value of the reproduction number and a reduction in the parameter's value will result in an increase in the value of the reproduction number.

Chapter 4

Results and Discussion

4.1 Introduction

In this chapter we give the results and carry out numerical simulations to confirm the validity of these results and to illustrate the long term behaviour of the system.

4.2 Results

All state variables were found to be positive and bounded meaning that the model was biologically meaningful.

The disease free equilibrium (DFE) at (S,I,B) existed and was given by $(\frac{\Lambda}{\mu}, 0, 0)$, while the endemic equilibrium point was shown to exist with $B^* = \frac{\sigma}{\xi} I^*$ and $S^* = \frac{\Lambda}{\mu} - \frac{(\gamma+\mu+\delta)I^*}{\mu}$.

The Basic reproduction number R_0, which was the threshold parameter of the model was found to be $R_0 = \frac{\Lambda}{\mu(\gamma+\delta+\mu)}(\beta_h + \frac{(\beta_e-\rho\beta_e)\sigma}{\kappa\xi})$.

Analysis of the DFE showed that it was both locally and globally stable when $R_0 < 1$ which means that if the basic reproduction number is kept below unity, the number of people infected reduces and thus the disease spread reduces.

The endemic equilibrium was locally stable when $R_0 > 1$ which means that if the disease exists, it can be kept at managable levels and therefore prevent an epidemic from occuring.

The parameters β_e, β_h, σ and Λ were found to have a positive sensitivity index which means that their levels should be reduced inorder to reduce the basic reproduction number and the disease spread.

The parameters ρ, κ, γ and ξ had a negative sensitivity index which means that their levels should be increased inorder to reduce the basic reproduction number and the disease spread.

ρ which is the efficacy of media coverage has an inverse relationship with the spread of the disease, such that whenever it is high the disease spread reduces and when it is low the disease spread increases.

4.3 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 which are compatible with cholera have been obtained from literature while others have been estimated. The parameter values are shown in Table 4.1.

TABLE 4.1: Model Parameters and Values

Symbol	**Value**	**Source**
Λ	9.6274×10^{-5}/day	(Lawi, Mugisha, & Omolo, 2011)
μ	2.537×10^{-5}/day	(Lawi et al., 2011)
β_e	0.75/day	Estimate
β_h	1/day	Estimate
κ	10^6cells/ml	(Codeço, 2001)
γ	5people/day	(Hartley et al., 2006)
δ	4.0×10^{-4}/day	(Mari et al., 2011)
m	0.00001	Varies
σ	10cells/ml-day	(J. Wang & Modnak, 2011)
ξ	0.23/day	(Mari et al., 2011)
ρ	0.6	Varies

4.4 Simulation and Intepretation

To be able to illustrate the behaviour of solutions with time, there must be a susceptible human population and *Vibrios* in the environment therefore, $S(t) > 0$, $B(t) > 0$, $I(t) \geq 0$ and $R(t) \geq 0$. With these conditions in mind, we consider the following initial values $S(t) = 10$, $I(t) = 1$, $R(t) = 1$ and $B(t) = 100$. When we calculate R_0 from the values shown in Table 4.1, we get its value to be $0.7589 < 1$.

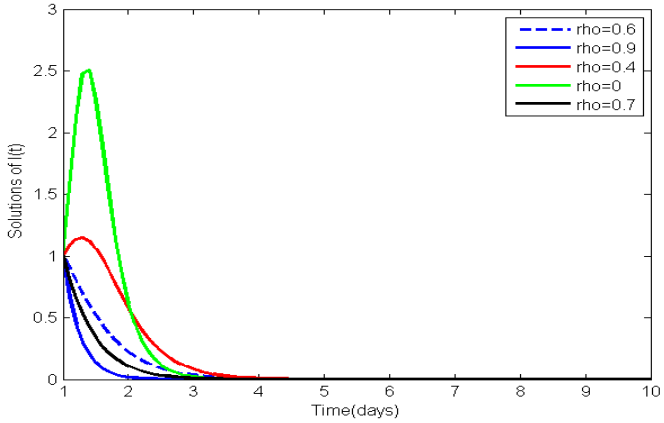

FIGURE 4.1: Numerical solutions when $R_0 < 1$

Figure 4.1 shows that when $R_0 < 1$ all the trajectories of infectives converge to zero regardless of the values of ρ. Consequently, our cholera free state can only be asymptotically stable supporting Theorem 3.3.

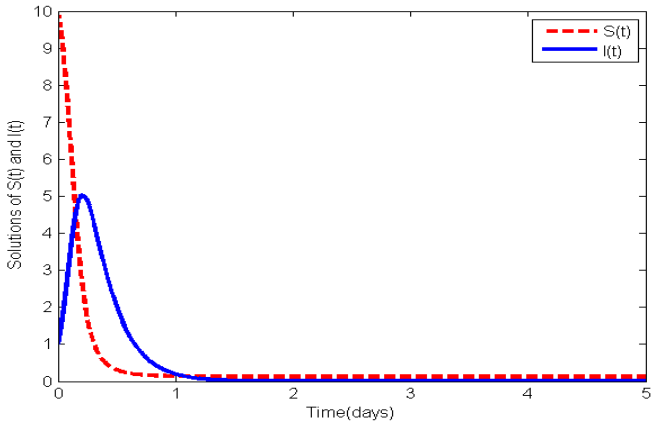

FIGURE 4.2: Numerical solutions when $R_0 > 1$

Using the values in Table 4.1 and changing the value of β_h to 5, to get $R_0 > 1$, we compute the value of $R_0 = 3.794 > 1$. When $R_0 > 1$, the cholera free equilibrium becomes unstable and the endemic equilibrium becomes stable. Consequently, the endemic equilibrium is asymptotically stable. Figure 4.2 shows that all solutions of S(t) and I(t) converge to E^* when $R_0 > 1$ supporting Theorem 3.6.

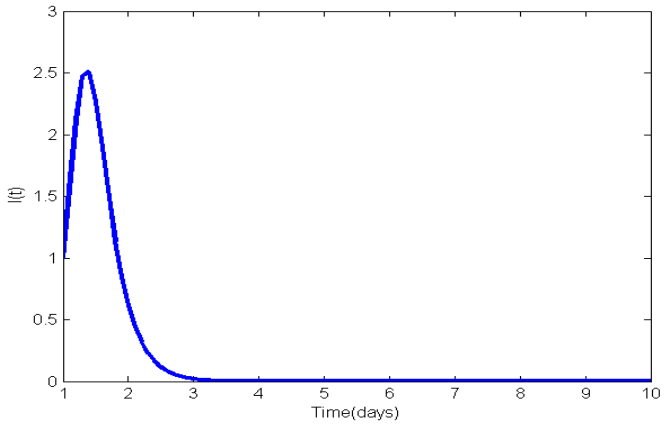

FIGURE 4.3: Cholera infectives in the absence of media coverage ($m = 0$ and $\rho = 0$)

Lack of media coverage about an outbreak of the disease causes the number of infectives to first rise steadily as many people are not yet aware about the outbreak and the preventive measures before it starts droping as the susceptibles are depleted from the population as shown in Figure 4.3.

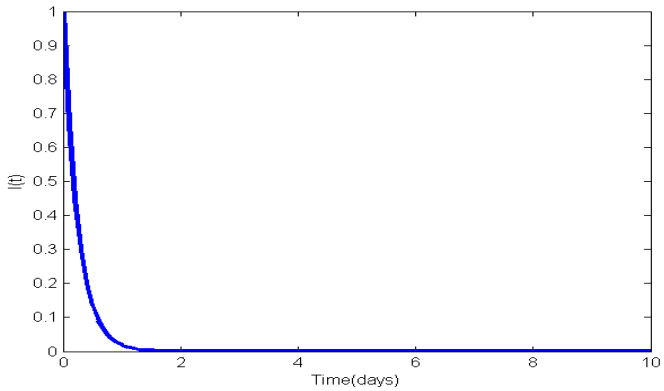

FIGURE 4.4: Cholera infectives in the presence of media coverage ($m = 0.00001$ and $\rho = 0.6$)

In the presence of the media coverage the number of infectives decreases sharply as many people are aware of the outbreak and take precautionary measures to prevent infection as shown in Figure 4.4.

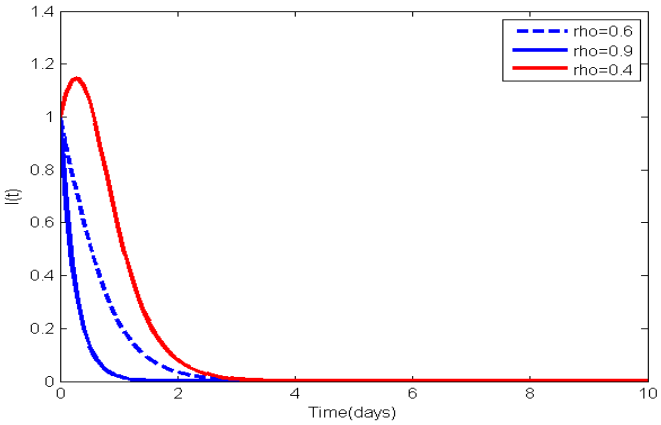

FIGURE 4.5: Cholera infectives with different values of ρ

When the media is very effective in reporting about the outbreak of the disease and the preventive measures, the infectious individuals are eliminated faster from the population, while if there is ineffective or no media coverage then the infection spreads for a longer time in the population as shown in Figure 4.5.

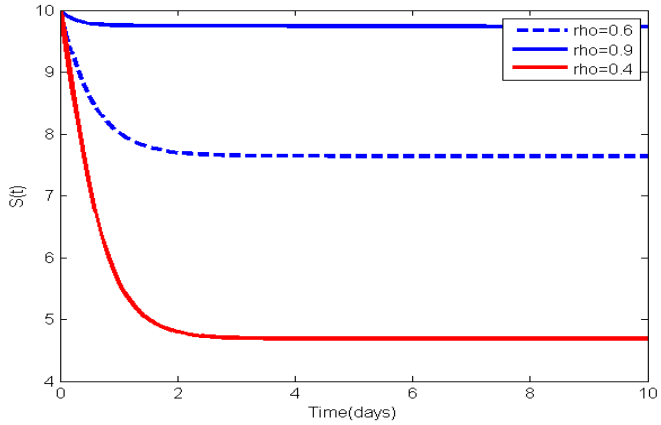

FIGURE 4.6: Cholera Susceptibles with different values of ρ

When the media is very effective in reporting about an outbreak, many people take precautionary measures and the rate of infection reduces, this causes the number of susceptibles to remain high in the population, while if there is ineffective media coverage, the infection spreads and the number of susceptible individuals are depleted from the population as shown in Figure 4.6.

Chapter 5

Conclusions and Recomendations

5.1 Conclusion

In this study, a mathematical model based on a system of ordinary differential equations incorporating media coverage has been formulated and analysed with an aim of investigating the effect of media as a disease control strategy.

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, which was the threshold parameter, 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 when the basic reproduction number is less than unity. Ideally, this means that keeping R_0 less than unity is a possible strategy for curbing the spread of the disease. Analysis of the endemic equilibrium shows its existence when $R_0 > 1$. Furthemore, the endemic equilibrium is also locally asymptotically stable. This shows that when $R_0 > 1$, the disease persists and spreads in the population.

Sensitivity analysis of the model shows that a reduction in the rate of human to human transmission, environment to human transmission, rate of recruitment of susceptibles and the rate of human shedding of *Vibrio cholerae* to the environment is required to reduce the basic reproduction number and the disease spread. On the other hand, an increase in the rate of media efficacy, pathogen concentration required for someone to catch the disease, rate of recovery, and the rate of death of *Vibrio cholerae* is required to reduce the basic reproduction number and to lower

the disease spread.

Numerical Analysis of the model supports the fact that both the disease free and endemic equilibriun are stable. It also shows that lack of effective media reporting on the presence of the disease and preventive measures greatly increases the number of people infected by cholera.

5.2 Recommendation

Cholera still remains endemic in many countries with most African countries experiencing sporadic outbreaks. The findings of this study show that lack of or inefficient media reporting of an outbreak of the disease and the preventive measures leads to increased cases of infection. Therefore, we recommend that policy makers and health practitioners should embrace the use of efficacious means of media coverage to carry out massive awareness campaigns on preventive measures whether there is an epidemic or not.

5.3 Future Work

The model has some limitations. We did not carry out optimal control and cost effective analysis of different cholera intervention strategies, which can be explored in future to find out which strategy is the best in the control of cholera.

The model can also be extended to incorporate environmental factors such as climate change, sea surface temperature, zooplankton levels and human mobility which have been known to greatly influence the occurence of cholera.

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Appendix

Appendix A

Matlab Codes Used for Simulating the Cholera Model

```
clear
options=odeset('RelTol',1e-4,'AbsTol',[1e-4 1e-4 1e-4 1e-4]);
[T1,Y1]=ode45('be1',[1:0.1:10],[10,1,100,1],options);
[T2,Y2]=ode45('be2',[1:0.1:10],[10,1,100,1],options);
[T3,Y3]=ode45('be3',[1:0.1:10],[10,1,100,1],options);
[T4,Y4]=ode45('be4',[1:0.1:10],[10,1,100,1],options);
[T5,Y5]=ode45('be5',[1:0.1:10],[10,1,100,1],options);
plot(T1,Y1(:,2),'--',T2,Y2(:,2),'-b',T3,Y3(:,2),'-r',
T4,Y4(:,2),'g',T5,Y5(:,2),'k', 'LineWidth',2);
xlabel 'Time(days)';
ylabel ' Solutions of I(t)';
h=legend('rho=0.6','rho=0.9','rho=0.4','rho=0','rho=0.7',1);
\includegraphics [width=4in]{Figure4.1}

clear
options=odeset('RelTol',1e-4,'AbsTol',[1e-4 1e-4 1e-4 1e-4]);
[T1,Y1]=ode45('be1',[0:0.1:10],[10,1,100,1],options);
[T2,Y2]=ode45('be2',[0:0.1:10],[10,1,100,1],options);
[T3,Y3]=ode45('be3',[0:0.1:10],[10,1,100,1],options);
plot(T1,Y1(:,2),'--',T2,Y2(:,2),'-b',T3,Y3(:,2),'-r','LineWidth',3);
xlabel 'Time(days)';
ylabel 'I(t)';
h=legend('rho=0.6','rho=0.9','rho=0.4',1);
\includegraphics [width=4in]{Figure4.5}

clear
```

```
options=odeset('RelTol',1e-4,'AbsTol',[1e-4 1e-4 1e-4 1e-4]);
[T1,Y1]=ode45('be1',[0:0.1:10],[10,1,100,1],options);
[T2,Y2]=ode45('be2',[0:0.1:10],[10,1,100,1],options);
[T3,Y3]=ode45('be3',[0:0.1:10],[10,1,100,1],options);
plot(T1,Y1(:,1),'--',T2,Y2(:,1),'-b',T3,Y3(:,1),'-r','LineWidth',3);
xlabel 'Time(days)';
ylabel 'S(t)';
h=legend('rho=0.6','rho=0.9','rho=0.4',1);
\includegraphics [width=4in]{Figure4.6.png}

clear
options=odeset('RelTol',1e-4,'AbsTol',[1e-4 1e-4 1e-4 1e-4]);
[T2,Y2]=ode45('be2',[0:0.1:10],[10,1,100,1],options);
plot(T2,Y2(:,2),'-b','Linewidth',3);
xlabel 'Time(days)';
ylabel 'I(t)';
\includegraphics [width=4in]{Figure4.4}

clear
options=odeset('RelTol',1e-4,'AbsTol',[1e-4 1e-4 1e-4 1e-4]);
[T4,Y4]=ode45('be4',[1:0.1:10],[10,1,100,1],options);
plot(T4,Y4(:,2),'-b','Linewidth',3);
xlabel 'Time(days)';
ylabel ' Solutions of I(t)';
\includegraphics [width=4in]{Figure4.3}

function dy=be1(t,y)
lam=9.6274*10e-5;
mu=2.537*10e-5;
betae=0.75;
alpha=1;
kappa=1000000;
gamma=5;
delta=0.0004;
```

```
m=0.00001;
sigma=10;
xi=0.23;
rho=0.6;
dy=[0 0 0 0]';
dy(1)=lam-(betae-rho*betae)*((y(1)*y(3))/(kappa+y(3)))-
(alpha-(rho*alpha*y(2)/(m+y(2))))*y(1)*y(2)-mu*y(1);
dy(2)=(betae-rho*betae)*((y(1)*y(3))/(kappa+y(3)))+
(alpha-(rho*alpha*y(2)/(m+y(2))))*y(1)*y(2)-(gamma+mu+delta)*y(2);
dy(3)=sigma*y(2)-xi*y(3);
dy(4)=gamma*y(2)-mu*y(4);

function dy=be2(t,y)
lam=9.6274*10e-5;
mu=2.537*10e-5;
betae=0.75;
alpha=1;
kappa=1000000;
gamma=5;
delta=0.0004;
m=0.0001;
sigma=10;
xi=0.23;
rho=0.9;
dy=[0 0 0 0]';
dy(1)=lam-(betae-rho*betae)*((y(1)*y(3))/(kappa+y(3)))-
(alpha-(rho*alpha*y(2)/(m+y(2))))*y(1)*y(2)-mu*y(1);
dy(2)=(betae-rho*betae)*((y(1)*y(3))/(kappa+y(3)))+
(alpha-(rho*alpha*y(2)/(m+y(2))))*y(1)*y(2)-(gamma+mu+delta)*y(2);
dy(3)=sigma*y(2)-xi*y(3);
dy(4)=gamma*y(2)-mu*y(4);

function dy=be3(t,y)
lam=9.6274*10e-5;
mu=2.537*10e-5;
```

```
betae=0.75;
alpha=1;
kappa=1000000;
gamma=5;
delta=0.0004;
m=0.000001;
sigma=10;
xi=0.23;
rho=0.4;
dy=[0 0 0 0]';
dy(1)=lam-(betae-rho*betae)*((y(1)*y(3))/(kappa+y(3)))-
(alpha-(rho*alpha*y(2)/(m+y(2))))*y(1)*y(2)-mu*y(1);
dy(2)=(betae-rho*betae)*((y(1)*y(3))/(kappa+y(3)))+
(alpha-(rho*alpha*y(2)/(m+y(2))))*y(1)*y(2)-(gamma+mu+delta)*y(2);
dy(3)=sigma*y(2)-xi*y(3);
dy(4)=gamma*y(2)-mu*y(4);

function dy=be4(t,y)
lam=9.6274*10e-5;
mu=2.537*10e-5;
betae=0.75;
alpha=1;
kappa=1000000;
gamma=5;
delta=0.0004;
m=0;
sigma=10;
xi=0.23;
rho=0;
dy=[0 0 0 0]';
dy(1)=lam-(betae-rho*betae)*((y(1)*y(3))/(kappa+y(3)))-
(alpha-(rho*alpha*y(2)/(m+y(2))))*y(1)*y(2)-mu*y(1);
dy(2)=(betae-rho*betae)*((y(1)*y(3))/(kappa+y(3)))+
(alpha-(rho*alpha*y(2)/(m+y(2))))*y(1)*y(2)-(gamma+mu+delta)*y(2);
dy(3)=sigma*y(2)-xi*y(3);
```

```
dy(4)=gamma*y(2)-mu*y(4);

function dy=be5(t,y)
lam=9.6274*10e-5;
mu=2.537*10e-5;
betae=0.75;
alpha=1;
kappa=1000000;
gamma=5;
delta=0.0004;
m=0.000015;
sigma=10;
xi=0.23;
rho=0.7;
dy=[0 0 0 0]';
dy(1)=lam-(betae-rho*betae)*((y(1)*y(3))/(kappa+y(3)))-
(alpha-(rho*alpha*y(2)/(m+y(2))))*y(1)*y(2)-mu*y(1);
dy(2)=(betae-rho*betae)*((y(1)*y(3))/(kappa+y(3)))+
(alpha-(rho*alpha*y(2)/(m+y(2))))*y(1)*y(2)-(gamma+mu+delta)*y(2);
dy(3)=sigma*y(2)-xi*y(3);
dy(4)=gamma*y(2)-mu*y(4);

clear options=odeset('RelTol',1e-4,'AbsTol',[1e-4 1e-4 1e-4 1e-4]);
[T1,Y1]=ode45('bee1',[0:0.001:5],[10,1,100,1],options);
[T2,Y2]=ode45('bee2',[0:0.001:5],[10,1,100,1],options);
[T3,Y3]=ode45('bee3',[0:0.001:5],[10,1,100,1],options);
[T4,Y4]=ode45('bee4',[0:0.001:5],[10,1,100,1],options);
plot(T1,Y1(:,1),'--r',T1,Y1(:,2),'-b','LineWidth',3);
xlabel 'Time(days)';
ylabel 'Solutions of S(t) and I(t)';
h=legend('S(t)','I(t)',1);
\includegraphics [width=4in]{Figure4.2}

function dy=bee1(t,y)
```

```
lam=9.6274*10e-5;
mu=2.537*10e-5;
betae=0.75;
alpha=5;
kappa=1000000;
gamma=5;
delta=0.0004;
m=0.00001;
sigma=10;
xi=0.23;
rho=0.6;
dy=[0 0 0 0]';
dy(1)=lam-(betae-rho*betae)*((y(1)*y(3))/(kappa+y(3)))-
(alpha-(rho*alpha*y(2)/(m+y(2))))*y(1)*y(2)-mu*y(1);
dy(2)=(betae-rho*betae)*((y(1)*y(3))/(kappa+y(3)))+
(alpha-(rho*alpha*y(2)/(m+y(2))))*y(1)*y(2)-(gamma+mu+delta)*y(2);
dy(3)=sigma*y(2)-xi*y(3);
dy(4)=gamma*y(2)-mu*y(4);

function dy=bee2(t,y)
lam=9.6274*10e-5;
mu=2.537*10e-5;
betae=0.75;
alpha=5;
kappa=1000000;
gamma=5;
delta=0.0004;
m=0.0001;
sigma=10;
xi=0.23;
rho=0.9;
dy=[0 0 0 0]';
dy(1)=lam-(betae-rho*betae)*((y(1)*y(3))/(kappa+y(3)))-
(alpha-(rho*alpha*y(2)/(m+y(2))))*y(1)*y(2)-mu*y(1);
dy(2)=(betae-rho*betae)*((y(1)*y(3))/(kappa+y(3)))+
```

```
(alpha-(rho*alpha*y(2)/(m+y(2))))*y(1)*y(2)-(gamma+mu+delta)*y(2);
dy(3)=sigma*y(2)-xi*y(3);
dy(4)=gamma*y(2)-mu*y(4);

function dy=bee3(t,y)
lam=9.6274*10e-5;
mu=2.537*10e-5;
betae=0.75;
alpha=5;
kappa=1000000;
gamma=5;
delta=0.0004;
m=0.000001;
sigma=10;
xi=0.23;
rho=0.4;
dy=[0 0 0 0]';
dy(1)=lam-(betae-rho*betae)*((y(1)*y(3))/(kappa+y(3)))-
(alpha-(rho*alpha*y(2)/(m+y(2))))*y(1)*y(2)-mu*y(1);
dy(2)=(betae-rho*betae)*((y(1)*y(3))/(kappa+y(3)))+
(alpha-(rho*alpha*y(2)/(m+y(2))))*y(1)*y(2)-(gamma+mu+delta)*y(2);
dy(3)=sigma*y(2)-xi*y(3);
dy(4)=gamma*y(2)-mu*y(4);

function dy=bee4(t,y)
lam=9.6274*10e-5;
mu=2.537*10e-5;
betae=0.75;
alpha=5;
kappa=1000000;
gamma=5;
delta=0.0004;
m=0;
sigma=10;
xi=0.23;
```

```
rho=0;
dy=[0 0 0 0]';
dy(1)=lam-(betae-rho*betae)*((y(1)*y(3))/(kappa+y(3)))-
(alpha-(rho*alpha*y(2)/(m+y(2))))*y(1)*y(2)-mu*y(1);
dy(2)=(betae-rho*betae)*((y(1)*y(3))/(kappa+y(3)))+
(alpha-(rho*alpha*y(2)/(m+y(2))))*y(1)*y(2)-(gamma+mu+delta)*y(2);
dy(3)=sigma*y(2)-xi*y(3);
dy(4)=gamma*y(2)-mu*y(4);
```

Appendix

Appendix B

List of Publications

(1) **Musundi, B.O., Lawi, G.O., and Nyamwala, F.O. (2016). Mathematical Analysis of a Cholera Transmission Model Incorporating Media Coverage. *International Journal of Pure and Applied Mathematics*, *111*(2), 219-231.**