

Mathematical Modeling of Cholera Transmission Dynamics with Vaccination and Pathogen Mutation: Model Development, Equilibrium Analysis and Basic Reproduction Number

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Abstract: Cholera is a disease caused by the bacterium *Vibrio cholerae* which is transmitted through contaminated water and food sources mainly in poorly sanitised areas. Cholera continues to pose a significant public health challenge in Kenya, with recurrent outbreaks complicating control measures such as the introduction of vaccination and sanitisation campaigns. Although there have been successful oral cholera vaccination programmes in controlling transmission, factors such as waning immunity, low vaccination levels and mutation of pathogens continue to threaten the eradication of the disease. Emergence of mutant strains that limit the efficiency of the vaccine poses a serious problem in the control of the disease and calls for a deeper study on the relationship between the vaccine and mutation of the disease. A deterministic compartmental model for describing the transmission dynamics of cholera with vaccination, pathogen mutation, environmental contamination, sanitization intervention and compliance with good hygiene practices was formulated in this study. The population in the study was separated into susceptible, vaccinated, wild-type infected, mutant infected and recovered compartments together with an environment bacteria compartment. Direct and environmental transmission, waning immunity, bacteria shedding, sanitization process and bi-directional mutation between the wild-type and the mutants were included in the model. The basic reproduction number, R_0 , was calculated using the next generation matrix approach. The positivity and boundedness of solutions were discussed and disease free equilibrium obtained. Local stability analysis of the disease free equilibrium showed that the equilibrium point is locally asymptotically stable whenever $R_0 < 1$ and unstable when $R_0 > 1$. Endemic equilibrium was obtained and its existence proven whenever $R_0 > 1$. Analysis of sensitivity was carried out to assess the effect of the model parameters on the transmission of the disease. It was found out that efficacy of the vaccine, hygiene compliance, recovery rate, sanitation effectiveness and bacteria shedding have the highest impact in reducing cholera transmission. Numerical simulations using the software package MATLAB showed that increasing the level of vaccine coverage, efficacy of the vaccine, hygiene compliance and sanitization helps in reducing the prevalence of the disease and the level of bacteria in the environment. Increase in transmission rates, bacteria shedding and mutations promotes the disease. It has been concluded from the study that integrated intervention strategies including vaccination, effective treatment, sanitization, good hygiene practices and environmental contamination control can help in reducing cholera transmission in order to prevent future outbreaks of the disease in Kenya.

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I. INTRODUCTION

Cholera is a serious acute diarrheal illness that results from eating food or drinking water that contains the organisms which trigger the disease in humans, typically

Vibrio cholerae bacteria [30, 1]. However, the disease remains a significant public health problem in many developing nations, especially in Sub-Saharan Africa and Asia, due to poor hygiene and sanitation systems, lack of access to clean water and a poor health infrastructure which

allows reoccurrence to happen easily [30]. Cholera is endemic in Kenya and outbreaks have been reported in various regions, such as urban informal settlements, refugee camps, flood-prone localised parts, and arid and semi-arid regions, where clean water and sanitation is limited [30]. Occasional rainfall, environmental pollution and inefficient disposal of waste have worsened the situation creating a continuing threat from which cholera control is a continued challenge [30].

The direct human-to-human contact or indirect transmission via contaminated water sources in the environment is the primary way for cholera to be transmitted to humans, as mentioned by Wang et al. [20]. The ability of *Vibrio cholerae* to survive in aquatic milieu is also responsible for the reactivation of this epidemic disease, because presence of the bacteria in the aquatic environment allows it to remain in the aquatic environment for long periods of time, allowed by suitable conditions [13]. The survival time of the bacteria, sanitation procedures, shedding by infected people, water treatment interventions have therefore been identified as playing important roles in understanding the dynamics of transmission [13].

The recent availability of effective oral cholera vaccines (OCVs) has great potential to serve as a tool to prevent and control outbreaks of cholera in endemic areas, especially those with frequent outbreaks [4]. Epidemiological studies have showed that vaccination provides a powerful mitigation tool in conjunction with sanitation and enhanced hygiene [8]. However, vaccine immunity is usually short-lived, with susceptible individuals who previously had vaccinations becoming vulnerable once again as immunity fades [8, 4]. This is an important factor to consider in longterm transmission.

In recent years a growing number of epidemiological and microbiological survey has identified genetically distinct and multidrug-resistant strains of *Vibrio cholerae* that differ in specific ways [12]. As for transmissibility, virulence, multidrug resistance and the ability of the pathogen to stay infectious despite repeated infections [12], pathogen mutation and bacterial evolution are involved. Bacterial strains that are capable of overcoming vaccine-induced immunity and developing antimicrobial resistance might emerge because dynamic changes in mutation may affect the process of disease control [12]. Dense populations, frequent outbreaks and environmental contamination are a common feature in endemic areas of cholera, complicating transmission within the population with the presence of multiple bacterial strains [12].

Mathematical modelling has been useful for understanding the dynamics of infectious disease

transmission and planning interventions [29]. The compartmental epidemic models, which include the SIR model, SEIR model and SIWR model, have been widely used to analyse the cholera outbreak in recent years [13]. Such models simplify the intricate epidemiologic dynamics to a set of differential equations which can be mathematically studied [13].

Since 2020, multiple cholera mathematical models have been developed that include environmental bacterial reservoirs and/or vaccination, sanitation, seasonality, stochastic effects, and/or antimicrobial resistance [4, 12, 3, 8, 10]. Mixed up in repeating outbreaks has been recognised as a function of polluted water resources by environmental reservoir models [13]. Oral cholera vaccines are shown to be effective in reducing disease prevalence and mortality in vaccination models [4, 8]. Finally, the significance of hydrological conditions, rain variability, flooding and disease intensity in cholera outbreak was demonstrated using the cholera models of the season [8]. For pathogen evolution, cases were included in both AMC and mutation models, which also demonstrated its significance in the transmission dynamics of pathogens [12].

Although there have been a number of models that have emerged over the last few years, most current cholera mathematical models have only introduced one or a few factors independently including: vaccination, environmental contamination, seasonality or antimicrobial resistance for instance [13, 4]. Also, most of the models already in use rely on the assumption of only one bacterial strain that does not have the ability to evolve or adapt [12, 3]. The union of the dynamics of the vaccination program, interstrain mutations, bacterial reservoirs in the environment, sanitation and the force of infection in a deterministic compartmental model has been studied rarely, with only a few work, e.g. [8, 10].

To close this important gap, this study therefore aimed to create a comprehensive deterministic cholera transmission model that explicitly includes the dynamics of vaccination, that takes account of the possibility of mutations of both wild and mutant strains of cholera-causing bacteria, the environmental bacterial reservoir, sanitation interventions, and hygiene compliance. The model is calibrated to the Kenya context using values for the parameters.

II. MODEL FORMULATION AND ANALYSIS

In this section, the model is formulated. Positivity and boundedness of model solutions are shown. The equilibrium points of the model are obtained. The basic reproduction number is computed. The stability analysis for DFE and EE are presented.

➤ Model Flow Diagram

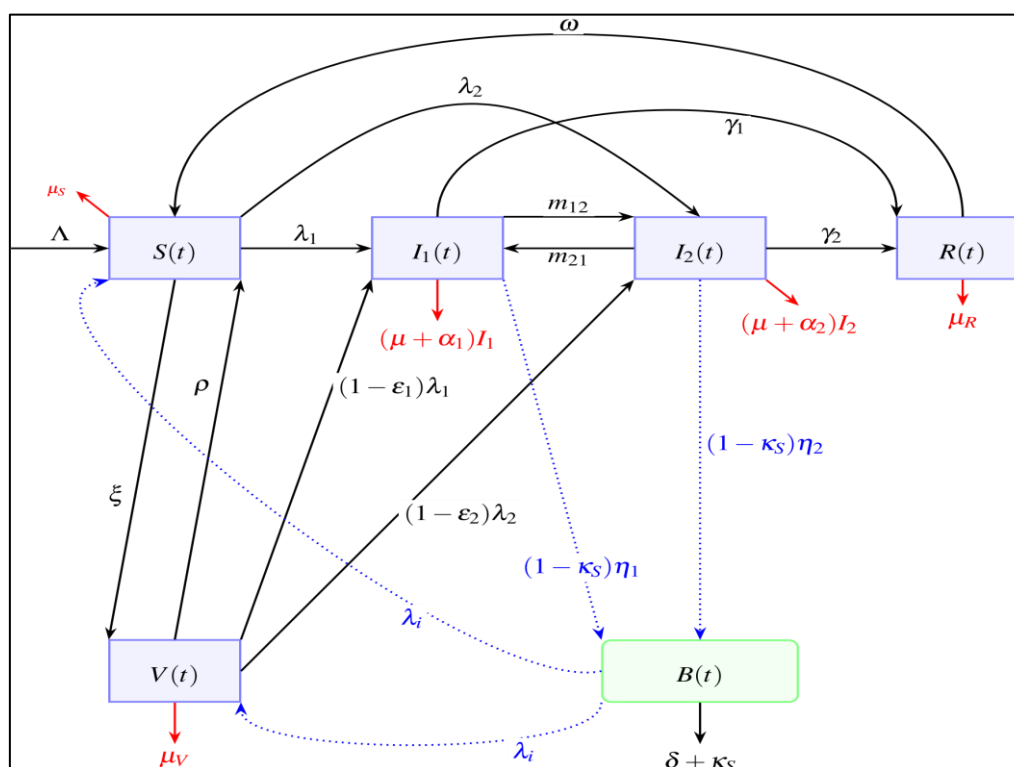


Fig 1 Cholera Transmission Flowchart Model with Vaccination and Mutation Dynamics.

➤ Model Description

A cholera transmission model based on basic compartmental framework was developed for this study, which would also include genetic mutations of the pathogen, environmental contamination, sanitation interventions, and establishment of good hygiene practices. A set of susceptible, vaccinated, wild-type infected, mutant infected and recovered bacteria behavioral compartments and an environmental bacteria compartment were used to partition the population studied. In the model direct and environmental transmission, from the waning immunity, shedding of bacteria and the sanitation process, as well as bidirectional mutations between the wild-type and mutants were taken into account.

The total human population is taken to be N which is subdivided into five compartments namely: $S(t)$ the susceptible individuals, $V(t)$ the vaccinated individuals, $I_1(t)$ the individuals infected with wild-type strain, $I_2(t)$ the individuals infected with mutant strain, and $R(t)$ the recovered individuals. $B(t)$ is the concentration of bacteria that are in the environment.

The susceptible individuals $S(t)$ are added to the population at the constant rate Λ . The rate at which the infection spreads to the susceptible population is β_1 for "wildtype" and β_2 for "mutant" strain of infection, or there is an "environmental water source" of infection from which the infection is spread to the susceptible population at rate $\beta_{E,1}$ and $\beta_{E,2}$, respectively. Susceptible people are assumed to be vaccinated at rate ξ while mutant vaccines are assumed to

have vaccine efficacy of ε_2 and wild-type vaccine of ε_1 . The immune system has a natural decay rate ω and vaccine-induced immune system has a decay rate ρ . The recovery percentage for wild-type and mutant infections is by γ_1 and γ_2 respectively. The mortality due to diseases is α_1, α_2 . The two infections can each infect the other and the rate is m_{12} or m_{21} , respectively. The rate of Bidirectional Mutation of the two infections are m_{12} and m_{21} respectively.

The amount of shed bacteria from an infected person is η_1 and η_2 , and $B(t)$ is the bacterial concentration in the environment which decreases at rate δ due to various bacterial natural decay processes and also κ_S which is the rate of bacteria removal from the environment due to the disinfecting measure taken. Reduction in direct transmission, $\kappa_H =$ outcomes of hygiene compliance.

The forces of infection for strains $i = (1,2)$ both include direct and environmental pathways:

$$\lambda_i = (1 - \kappa_H) \beta_i \frac{I_i}{N} + \beta_{E,i} \frac{B}{K + B}, \quad (1)$$

Where $N = S + V + I_1 + I_2 + R$. The term $(1 - \kappa_H) \beta_i \frac{I_i}{N}$ is direct human-to-human transmission modified by the compliance with hygiene measures, κ_H ; $\beta_{E,i} \frac{B}{K + B}$ is the environmental transmission through contaminated water. The parameter K is the half-saturation constant.

The associated parameters are summarized in Table 1.

Table 1 Model Parameters, Descriptions, Baseline Values and Sources

Symbol	Description	Baseline Value	Source
Λ	Recruitment rate into susceptible population	0.55 day^{-1}	[11]
μ	Natural mortality rate	$4.5 \times 10^{-5} \text{ day}^{-1}$	[11]
ξ	Vaccination rate	0.005 day^{-1}	[4]
ε_1	Vaccine efficacy against wild strain	0.85	[4]
ε_2	Vaccine efficacy against mutant strain	0.70	Assumed
ρ	Vaccine waning rate	0.0011 day^{-1}	[5, 15]
ω	Natural immunity waning rate	0.0005 day^{-1}	[5]
β_1	Human-to-human transmission coefficient for strain 1	0.25 day^{-1}	[11, 25]
β_2	Human-to-human transmission coefficient strain 2	for 0.30 day^{-1}	Assumed
$\beta_{E,1}$	Environmental transmission coefficient strain 1	for 0.15 day^{-1}	[6, 18]
$\beta_{E,2}$	Environmental transmission coefficient strain 2	for 0.18 day^{-1}	Assumed
K	Half-saturation constant	$1.0 \times 10^6 \text{ cells/ml}$	[6, 11]
γ_1	Recovery rate from strain 1 infection	0.20 day^{-1}	[25]
γ_2	Recovery rate from strain 2 infection	0.18 day^{-1}	Assumed
α_1	Disease-induced mortality rate for strain 1	0.010 day^{-1}	[11]
α_2	Disease-induced mortality rate for strain 2	0.015 day^{-1}	Assumed
m_{12}	Mutation rate from strain 1 to strain 2	0.002 day^{-1}	[12]
m_{21}	Mutation rate from strain 2 to strain 1	0.001 day^{-1}	[12]
η_1	Bacterial shedding rate by strain 1 infected individuals	$10 \text{ cells ml}^{-1} \text{ day}^{-1}$	[9, 10]
η_2	Bacterial shedding rate by strain 2 infected individuals	$12 \text{ cells ml}^{-1} \text{ day}^{-1}$	Assumed
δ	Natural bacterial decay rate	0.23 day^{-1}	[6, 11]
κ_S	Sanitation effort parameter	0.10	Assumed
κ_H	Hygiene compliance parameter	0.10	Assumed

The system's dynamics depicted in Figure 1 are regulated by the following system of ordinary differential equations (ODEs):

$$\begin{aligned}
 \frac{dS}{dt} &= \Lambda - (\lambda_1 + \lambda_2)S - \xi S + \rho V + \omega R - \mu S, \\
 \frac{dV}{dt} &= \xi S - (1 - \varepsilon_1)\lambda_1 V - (1 - \varepsilon_2)\lambda_2 V - \rho V - \mu V, \\
 \frac{dI_1}{dt} &= \lambda_1(S + (1 - \varepsilon_1)V) + m_{21}I_2 - (\gamma_1 + \alpha_1 + \mu + m_{12})I_1, \\
 \frac{dI_2}{dt} &= \lambda_2(S + (1 - \varepsilon_2)V) + m_{12}I_1 - (\gamma_2 + \alpha_2 + \mu + m_{21})I_2, \\
 \frac{dR}{dt} &= \gamma_1 I_1 + \gamma_2 I_2 - (\omega + \mu)R, \\
 \frac{dB}{dt} &= (1 - \kappa_S)\eta_1 I_1 + (1 - \kappa_S)\eta_2 I_2 - (\delta + \kappa_S)B.
 \end{aligned} \quad (2)$$

III. ANALYSIS OF THE MODEL

➤ Positivity of Solutions

Since the model describes human population and environmental bacterial concentration, the solutions remain positive and bounded. The state variables $S(t), V(t), I_1(t), I_2(t), R(t), B(t)$ are always positive for every time $t \geq 0$.

Theorem 1. All solution sets $\{S(t), V(t), I_1(t), I_2(t), R(t), B(t)\}$ of the model (2) with non-negative initial conditions are positive for all $t > 0$.

Proof. Considering the first equation in model (2):

$$\frac{dS}{dt} = \Lambda - (\lambda_1 + \lambda_2)S - \xi S + \rho V + \omega R - \mu S. \quad (3)$$

Since all parameters are positive and $\Lambda, \rho V, \omega R \geq 0$, we can write the inequality:

$$\frac{dS}{dt} \geq -(\lambda_1 + \lambda_2 + \xi + \mu)S. \quad (4)$$

Integrating by variable separation, we get:

$$\int \frac{dS}{S} \geq - \int (\lambda_1 + \lambda_2 + \xi + \mu) dt. \quad (5)$$

Thus:

$$S(t) \geq S(0) \exp \left(- \int_0^t [(\lambda_1 + \lambda_2 + \xi + \mu)] d\zeta \right). \quad (6)$$

This implies that $S(t) \geq 0$ for all $t \geq 0$.

Taking the second equation that is vaccinated population equation V:

$$\frac{dV}{dt} = \xi S - (1 - \varepsilon_1)\lambda_1 V - (1 - \varepsilon_2)\lambda_2 V - \rho V - \mu V. \quad (7)$$

Since all parameters are positive and $\xi S \geq 0$, we can write:

$$\frac{dV}{dt} \geq -((1 - \varepsilon_1)\lambda_1 + (1 - \varepsilon_2)\lambda_2 + \rho + \mu)V. \quad (8)$$

Integrating by variable separation:

$$\int \frac{dV}{V} \geq - \int ((1 - \varepsilon_1)\lambda_1 + (1 - \varepsilon_2)\lambda_2 + \rho + \mu) dt. \quad (9)$$

Thus:

$$V(t) \geq V(0) \exp \left(- \int_0^t ((1 - \varepsilon_1)\lambda_1 + (1 - \varepsilon_2)\lambda_2 + \rho + \mu) d\zeta \right) \geq 0. \quad (10)$$

In a similar way, all other variables I_1, I_2, R, B can be shown to be positive in the region Ω . $\square$

Theorem 2. For all time $t \geq 0$, the solutions of model (2) are bounded in the region Ω .

Model (2) is analyzed on a suitable feasible region:

$$\Omega = \left\{ (S, V, I_1, I_2, R, B) \in \mathbb{R}_+^6 : S(t) + V(t) + I_1(t) + I_2(t) + R(t) + B(t) \leq \frac{(1 - \kappa_S)\eta_1 \Lambda}{\mu(\delta + \kappa_S)} \right\}. \quad (11)$$

Proof. The rate of change of the human population $N(t)$ is given by:

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dV}{dt} + \frac{dI_1}{dt} + \frac{dI_2}{dt} + \frac{dR}{dt}. \quad (12)$$

Substituting the right-hand sides and simplifying:

$$\frac{dN}{dt} = \Lambda - \mu N - \alpha_1 I_1 - \alpha_2 I_2. \quad (13)$$

Since $\alpha_1, \alpha_2 \geq 0$ and $I_1, I_2 \geq 0$, we obtain the inequality:

$$\frac{dN}{dt} \leq \Lambda - \mu N. \quad (14)$$

By variation of constant formula:

$$N(t) \leq N(0)e^{-\mu t} + \frac{\Lambda}{\mu}(1 - e^{-\mu t}). \quad (15)$$

Taking the limit superior as $t \rightarrow \infty$:

$$\limsup_{t \rightarrow \infty} N(t) \leq \frac{\Lambda}{\mu}. \quad (16)$$

Similarly, for the environmental bacterial concentration:

$$B(t) \leq \frac{(1 - \kappa_S)\eta_1 \Lambda}{\mu(\delta + \kappa_S)}. \quad (17)$$

Thus, all solutions of the model (2) are bounded in the region Ω . $\square$

Clearly from theorems 1 and 2, all solutions of the model (2) are positive and bounded in the invariant region Ω . Thus, model (2) is mathematically and epidemiologically well-posed in a biologically feasible region Ω .

➤ Disease-Free Equilibrium Point (DFE)

The disease-free equilibrium (DFE) is defined as the state which nothing infected can be found. If the community has not yet experienced a cholera infection, then it is termed as being free from the disease. That is, the whole community is at risk for the infection. Initial conditions at the DFE are obtained by setting the concentration of all infected compartments of the system to zero, and the concentration of the outside bacteria in the environment to zero. That is, $I_1 = 0, I_2 = 0, R = 0, B = 0$.

Theorem 3. A correction matrix which results in a DFE of model (2) is given by:

$$E_0 = (S^0, V^0, 0, 0, 0, 0) = \left(\frac{\Lambda(\rho + \mu)}{\mu(\xi + \rho + \mu)}, \frac{\Lambda\xi}{\mu(\xi + \rho + \mu)}, 0, 0, 0, 0 \right). \quad (18)$$

Proof. At the disease-free equilibrium state (DFE) there is no cholera outbreak happening uninhibited in a population. Therefore, $I_1 = I_2 = R = B = 0$. Putting these conditions into the system (2) and setting all the time-derivatives to zero gives:

Using the equation from vaccinated compartment:

$$\xi S^0 - (\rho + \mu)V^0 = 0 \implies V^0 = \frac{\xi}{\rho + \mu} S^0. \quad (19)$$

The equation above can be rearranged as follows:

$$\Lambda - \xi S^0 + \rho \left(\frac{\xi}{\rho + \mu} S^0 \right) - \mu S^0 = 0. \quad (20)$$

Simplifying:

$$\Lambda = S^0 \left(\xi + \mu - \frac{\rho\xi}{\rho + \mu} \right). \quad (21)$$

Thus:

$$S^0 = \frac{\Lambda(\rho + \mu)}{\mu(\xi + \rho + \mu)}. \quad (22)$$

Employing (22) in (19) we obtain the expression:

$$V^0 = \frac{\Lambda \xi}{\mu(\xi + \rho + \mu)}. \quad (23)$$

Knowing this, the disease-free equilibrium is clearly specified in equation (18). $\square$

➤ Basic Reproduction Number

The basic reproduction number, R_0 , is the geometric mean of the number of infections produced by a single infected individual injected into a totally susceptible population. It is one of the necessary threshold values that will decide whether the disease is able to enter the population or not. The basic reproduction number (quite important) signifies whether cholera can enter a population or not. Epidemiologically, when $R_0 < 1$ then the infection can't spread in the population. However, when $R_0 > 1$ then the infection is transmitted in a population and can lead to an epidemic.

Here, R_0 is calculated following the method of van den Driessche and Watmough 2002 (vdDW) [?] which is based on the next generation matrix method defined there.

The compartments that are infected in the model are:

$$I_1, \quad I_2, \quad B. \quad (24)$$

Then let the column vector of infected variables be:

$$\mathbf{x} = (I_1, I_2, B). \quad (25)$$

Equations can be recorded in the following form:

$$\frac{d\mathbf{x}}{dt} = \mathcal{F}(\mathbf{x}) - \mathcal{V}(\mathbf{x}), \quad (26)$$

V denotes the rate at which people move from one compartment to another and F denotes the appearance of new infections.

The new infection terms are gleaned from the model equations:

$$\mathcal{F} = \begin{pmatrix} \lambda_1 S + (1 - \varepsilon_1) \lambda_1 V \\ \lambda_2 S + (1 - \varepsilon_2) \lambda_2 V \\ 0 \end{pmatrix}. \quad (27)$$

The rest of the equation is equal to:

$$\mathcal{V} = \begin{pmatrix} (\gamma_1 + \alpha_1 + \mu + m_{12}) I_1 - m_{21} I_2 \\ (\gamma_2 + \alpha_2 + \mu + m_{21}) I_2 - m_{12} I_1 \\ -(1 - \kappa_S)(\eta_1 I_1 + \eta_2 I_2) + (\delta + \kappa_S) B \end{pmatrix}. \quad (28)$$

Evaluating at the disease-free equilibrium $E_0 = (S^0, V^0, 0, 0, 0, 0)$, we find the Jacobian matrices F and V at E_0 :

$$F = \begin{pmatrix} (1 - \kappa_H) \beta_1 q_1 & 0 & \beta_{E,1} r_1 \\ 0 & (1 - \kappa_H) \beta_2 q_2 & \beta_{E,2} r_2 \\ 0 & 0 & 0 \end{pmatrix}, \quad (29)$$

$$V = \begin{pmatrix} A_1 & -m_{21} & 0 \\ -m_{12} & A_2 & 0 \\ -(1 - \kappa_S) \eta_1 & -(1 - \kappa_S) \eta_2 & C \end{pmatrix}, \quad (30)$$

Where:

$$q_i = \frac{\rho + \mu + (1 - \varepsilon_i) \xi}{\xi + \rho + \mu}, \quad r_i = \frac{\Lambda q_i}{K \mu}, \quad A_i = \gamma_i + \alpha_i + \mu + m_{ij}, \quad C = \delta + \kappa_S. \quad (31)$$

Taking:

$$\beta_1^* = (1 - \kappa_H) \beta_1, \quad \beta_2^* = (1 - \kappa_H) \beta_2, \quad \Delta = A_1 A_2 - m_{12} m_{21}. \quad (32)$$

The reciprocal of V is:

$$V^{-1} = \begin{pmatrix} \frac{A_2}{\Delta} & \frac{m_{21}}{\Delta} & 0 \\ \frac{m_{12}}{\Delta} & \frac{A_1}{\Delta} & 0 \\ \frac{(1 - \kappa_S)(\eta_1 A_2 + \eta_2 m_{12})}{C \Delta} & \frac{(1 - \kappa_S)(\eta_1 m_{21} + \eta_2 A_1)}{C \Delta} & \frac{1}{C} \end{pmatrix}. \quad (33)$$

The next generation matrix is $A = FV^{-1} \equiv IV = I$, so we refer it to the initial matrix A . The basic reproduction number R_0 is equal to the spectral radius (dominant eigenvalue) of A .

The actual transition model for the two strains accounting for environmental feedback is:

$$\bar{A} = \begin{pmatrix} \bar{A}_{11} & \bar{A}_{12} \\ \bar{A}_{21} & \bar{A}_{22} \end{pmatrix}, \quad (34)$$

Where:

$$\bar{A}_{11} = \frac{1}{\Delta} \left(\beta_1^* q_1 A_2 + \beta_{E,1} r_1 \frac{(1 - \kappa_S)(\eta_1 A_2 + \eta_2 m_{12})}{C} \right), \quad (35)$$

$$\bar{A}_{12} = \frac{1}{\Delta} \left(\beta_1^* q_1 m_{21} + \beta_{E,1} r_1 \frac{(1 - \kappa_S)(\eta_1 m_{21} + \eta_2 A_1)}{C} \right), \quad (36)$$

$$\bar{A}_{21} = \frac{1}{\Delta} \left(\beta_2^* q_2 m_{12} + \beta_{E,2} r_2 \frac{(1 - \kappa_S)(\eta_1 A_2 + \eta_2 m_{12})}{C} \right), \quad (37)$$

$$\bar{A}_{22} = \frac{1}{\Delta} \left(\beta_2^* q_2 A_1 + \beta_{E,2} r_2 \frac{(1 - \kappa_S)(\eta_1 m_{21} + \eta_2 A_1)}{C} \right). \quad (38)$$

Then:

$$R_0 = \frac{\tilde{A}11 + \tilde{A}22 + \sqrt{(\tilde{A}11 - \tilde{A}22)^2 + 4\tilde{A}12\tilde{A}21}}{2}. \quad (39)$$

The basic reproduction number R_0 was computed using the following parameter values given in Table 1:

$$R_0 = 0.78398. \quad (40)$$

➤ Local Stability of Disease-Free Equilibrium

The disease-free equilibrium (DFE) is the state at which there is no infection in a certain population (absence of infection) [?].

$$J(E_0) = \begin{pmatrix} -(\xi + \mu) & \rho & -\beta_1^* q_1 & -\beta_2^* q_2 & \omega & -\beta_{E,1} r_1 - \beta_{E,2} r_2 \\ \xi & -(\rho + \mu) & -(1 - \varepsilon_1) \beta_1^* q_1 & -(1 - \varepsilon_2) \beta_2^* q_2 & 0 & -(1 - \varepsilon_1) \beta_{E,1} r_1 - (1 - \varepsilon_2) \beta_{E,2} r_2 \\ 0 & 0 & \beta_1^* q_1 - A_1 & m_{21} & 0 & \beta_{E,1} r_1 \\ 0 & 0 & m_{12} & \beta_2^* q_2 - A_2 & 0 & \beta_{E,2} r_2 \\ 0 & 0 & \gamma_1 & \gamma_2 & -(\omega + \mu) & 0 \\ 0 & 0 & (1 - \kappa_S) \eta_1 & (1 - \kappa_S) \eta_2 & 0 & -C \end{pmatrix}. \quad (41)$$

The Jacobian matrix can be written in block form:

$$J(E_0) = \begin{pmatrix} J_{11} & J_{12} \\ 0 & J_{22} \end{pmatrix}, \quad (42)$$

Where:

$$J_{11} = \begin{pmatrix} -(\xi + \mu) & \rho \\ \xi & -(\rho + \mu) \end{pmatrix}, \quad (43)$$

$$J_{22} = \begin{pmatrix} \beta_1^* q_1 - A_1 & m_{21} & \beta_{E,1} r_1 \\ m_{12} & \beta_2^* q_2 - A_2 & \beta_{E,2} r_2 \\ (1 - \kappa_S) \eta_1 & (1 - \kappa_S) \eta_2 & -C \end{pmatrix}. \quad (44)$$

The eigenvalues of J_{11} are $-\mu$ and $-(\xi + \rho + \mu)$, both of which have negative real parts.

According to the theorem of van den Driessche and Watmough (2002), the eigenvalues of the matrix J_{22} have negative real parts whenever $R_0 < 1$. Thus, if $R_0 < 1$, all eigenvalues of J_{22} have negative real parts.

Since all eigenvalues of both J_{11} and J_{22} are negative, it follows that all eigenvalues of the full Jacobian matrix $J(E_0)$ have negative real parts.

Therefore, the disease-free equilibrium E_0 is locally asymptotically stable if $R_0 < 1$, and unstable if $R_0 > 1$. □

Theorem 4. The disease-free equilibrium E_0 is locally asymptotically stable whenever $R_0 < 1$ and unstable whenever $R_0 > 1$.

Proof. The Jacobian matrix of model (2), evaluated at the DFE E_0 , has the form:

➤ Endemic Equilibrium (EE)

The model state of (2) where the cholera disease persists is known as the endemic equilibrium (EE). The location where the cholera disease is continuing to affect the population studied. The EE of the model (2) is given by setting $(S \cdot V \cdot I_1 \cdot I_2 \cdot R \cdot B) \neq 0$, thus

$$E^* = (S \cdot V \cdot I_1 \cdot I_2 \cdot R \cdot B) \in \mathbb{R}_+^6.$$

Theorem 5. There exists an endemic equilibrium (EE) state of model (2) if $(I_1 \cdot I_2 \cdot B^*) > 0$ whenever $R_0 > 1$.

Proof. The forces of infection are at equilibrium at:

$$\lambda_1^* = (1 - \kappa_H) \beta_1 \frac{I_1^*}{N^*} + \beta_{E,1} \frac{B^*}{K + B^*}, \quad (45)$$

And

$$\lambda_2^* = (1 - \kappa_H) \beta_2 \frac{I_2^*}{N^*} + \beta_{E,2} \frac{B^*}{K + B^*}, \quad (46)$$

Where:

$$^* = S^* + V^* + I_1^* + I_2^* + R^*. \quad (47)$$

Eliminating n and q from the model equations at equilibrium:

$$R^* = \frac{\gamma_1 I_1^* + \gamma_2 I_2^*}{\omega + \mu}. \quad (48)$$

$$B^* = \frac{(1 - \kappa_S)\eta_1 I_1^* + (1 - \kappa_S)\eta_2 I_2^*}{\delta + \kappa_S}. \quad (49)$$

$$V^* = \frac{\xi S^*}{(1 - \varepsilon_1)\lambda_1^* + (1 - \varepsilon_2)\lambda_2^* + \rho + \mu}. \quad (50)$$

Using the expressions of R^* and V^* in the susceptible equation:

$$\Lambda - (\lambda_1^* + \lambda_2^* + \xi + \mu)S^* + \rho V^* + \omega R^* = 0. \quad (51)$$

This gives:

$$S^* = \frac{\Lambda + \rho \frac{\xi S^*}{(1 - \varepsilon_1)\lambda_1^* + (1 - \varepsilon_2)\lambda_2^* + \rho + \mu} + \omega \frac{\gamma_1 I_1^* + \gamma_2 I_2^*}{\omega + \mu}}{\lambda_1^* + \lambda_2^* + \xi + \mu}. \quad (52)$$

When $R_0 > 1$ the following simultaneous equations, (48)-(52), have positive solutions for I_1^*, I_2^*, B^* .

The endemic equilibrium occurs when $R_0 > 1$ which means that cholera is present and maintained in the population. $\square$

➤ Local Stability of Endemic Equilibrium

The local stability of the endemic equilibrium is analysed by computing the Jacobian matrix of the model (2) at the point E^* . The equations of the model are:

$$\begin{aligned} \frac{dS}{dt} &= \Lambda - (\lambda_1 + \lambda_2)S - \xi S + \rho V + \omega R - \mu S, \\ \frac{dV}{dt} &= \xi S - (1 - \varepsilon_1)\lambda_1 V - (1 - \varepsilon_2)\lambda_2 V - \rho V - \mu V, \\ \frac{dI_1}{dt} &= \lambda_1(S + (1 - \varepsilon_1)V) + m_{21}I_2 - A_1 I_1, \\ \frac{dI_2}{dt} &= \lambda_2(S + (1 - \varepsilon_2)V) + m_{12}I_1 - A_2 I_2, \\ \frac{dR}{dt} &= \gamma_1 I_1 + \gamma_2 I_2 - (\omega + \mu)R, \\ \frac{dB}{dt} &= (1 - \kappa_S)\eta_1 I_1 + (1 - \kappa_S)\eta_2 I_2 - (\delta + \kappa_S)B, \end{aligned} \quad (53)$$

Where:

$$A_1 = \gamma_1 + \alpha_1 + \mu + m_{12}, \quad A_2 = \gamma_2 + \alpha_2 + \mu + m_{21}. \quad (54)$$

The Jacobian matrix of the system at E^* is:

$$J(E^*) = \begin{pmatrix} a_{11} & \rho & -S \frac{\partial \lambda_1}{\partial I_1} & -S \frac{\partial \lambda_2}{\partial I_2} & \omega & -S^* \frac{\partial \lambda}{\partial B} \\ \xi & a_{22} & -(1 - \varepsilon_1)V \frac{\partial \lambda_1}{\partial I_1} & -(1 - \varepsilon_2)V \frac{\partial \lambda_2}{\partial I_2} & 0 & -V^* \frac{\partial \lambda}{\partial B} \\ \lambda_1^* & (1 - \varepsilon_1)\lambda_1^* & a_{33} & m_{21} & 0 & a_{36} \\ \lambda_2^* & (1 - \varepsilon_2)\lambda_2^* & m_{12} & a_{44} & 0 & a_{46} \\ 0 & 0 & \gamma_1 & \gamma_2 & -(\omega + \mu) & 0 \\ 0 & 0 & (1 - \kappa_S)\eta_1 & (1 - \kappa_S)\eta_2 & 0 & -(\delta + \kappa_S) \end{pmatrix}, \quad (55)$$

Where:

$$a_{11} = -(\lambda_1^* + \lambda_2^* + \xi + \mu), \quad (56)$$

$$a_{22} = -[(1 - \varepsilon_1)\lambda_1^* + (1 - \varepsilon_2)\lambda_2^* + \rho + \mu], \quad (57)$$

$$a_{33} = \frac{\partial}{\partial I_1} [\lambda_1(S^* + (1 - \varepsilon_1)V^*)] - A_1, \quad (58)$$

$$a_{44} = \frac{\partial}{\partial I_2} [\lambda_2(S^* + (1 - \varepsilon_2)V^*)] - A_2, \quad (59)$$

$$a_{36} = \frac{\partial}{\partial B} [\lambda_1(S^* + (1 - \varepsilon_1)V^*)], \quad (60)$$

$$a_{46} = \frac{\partial}{\partial B} [\lambda_2(S^* + (1 - \varepsilon_2)V^*)]. \quad (61)$$

The characteristic equation of the Jacobian matrix is obtained from:

$$\det(J(E^*) - \lambda I) = 0, \quad (62)$$

Where:

$$J(E^*) = \begin{pmatrix} J_{11} & J_{12} \\ J_{21} & J_{22} \end{pmatrix}. \quad (63)$$

If the determinant is expanded, then the characteristic polynomial is:

$$\lambda^6 + c_1 \lambda^5 + c_2 \lambda^4 + c_3 \lambda^3 + c_4 \lambda^2 + c_5 \lambda + c_6 = 0, \quad (64)$$

The $c_i, i = 1, 2, \dots, 6$ are elements of the Jacobian of E^* .

Given the characteristic polynomial:

$$P(\lambda) = \lambda^n + a_1 \lambda^{n-1} + a_2 \lambda^{n-2} + \dots + a_{n-1} \lambda + a_n, \quad (65)$$

Where $a_i > 0$, for $i = 1, 2, \dots, n$.

According to the Routh-Hurwitz criterion, the Routh matrix is positive or if all the Hurwitz determinants are positive, then all the roots of the polynomial have negative real parts.

From the above characteristic polynomial, using Routh Hurwitz criterion, the endemic equilibrium point E^* is locally asymptotically stable if $b_0 b_1 > 0$ and the sign patterns of the remaining coefficients are as follows:

$$c_1 > 0, \quad c_2 > 0, \quad c_3 > 0, \quad c_4 > 0, \quad c_5 > 0, \quad c_6 > 0, \quad (66)$$

$$c_1 c_2 - c_3 > 0, \quad (67)$$

$$c_1 c_2 c_3 - c_1^2 c_4 - c_3^2 > 0, \quad (68)$$

With all the other Hurwitz determinants strictly positive.

Consequently, if $R_0 > 1$ and all the Routh-Hurwitz conditions hold true, then all the eigenvalues of $J(E^*)$ will have negative real parts, which means that E^* will be a locally asymptotically stable equilibrium.

If $R_0 > 1$ then the EE, which is the solution to the model (2) is stable for any small perturbation of the EE, as stated in Theorem 5. Epidemiologically, it means that if a few individuals with the disease enter a susceptible population and new secondary infections are generated whenever $R_0 > 1$ then cholera would remain in the population.

IV. CONCLUSION

A deterministic compartmental model of cholera transmission dynamics which include immunisation, pathogen mutation, environmental transmission, sanitation and compliance with hygiene practices, has been formulated and analysed in this study. This model divides the human population into susceptible, vaccinated, wild-type infected, mutant infected and recovered compartments, as well as a reservoir of bacteria in the environment.

Mathematic analyses were used to determine Positivity and Boundedness of solutions. This establishes the epidemiological relevance of the model since all the human population compartments are non-negative as well as the concentration of bacteria in the environment if the initial conditions are non-negative. Boundedness also has implications for the space containing the human population as it remains within a space that has a biological meaning.

The disease-free equilibrium condition was made by assuming that the infected populations and bacterial concentration in the environment is zero. The next-generation matrix method was employed to calculate the basic

reproduction number R_0 . A baseline value that was calculated was 0.78398, which is R_0 . If the value is below 1, then a new cholera infection is not likely to sustain itself in the population, for the baseline values of the parameters within the model. This is consistent with the local stability analysis carried out in the previous step of the disease free equilibrium on the basis of which it is shown that the disease free equilibrium will be locally stable if $R_0 < 1$ and locally unstable if $R_0 > 1$.

This endemic equilibrium occurs when $R_0 > 1$ and that its local stability is determined by Routh-Hurwitz conditions. When all of the Routh Hurwitz conditions are fulfilled, the endemic equilibrium will be locally asymptotically stable. This means that a small amount of EE will tend to converge to EE if $R_0 > 1$ which means cholera will remain in the population.

RECOMMENDATIONS

- The model needs to be used as a basis for sensitivity analysis and determine the most influential parameters when it comes to cholera control.
- Disease dynamic should be modelled numerically to explore how intervention strategies impact disease dynamic.
- The model should be adapted to Kenya specific epidemiological and environmental information to enable better predictions.
- In planning cholera control programs, decisions and regulations must be orientated to the broader, combined impact of the interventions of vaccination, treatment, hygiene and sanitation.

CONCERNS FOR FUTURE RESEARCH.

- Future work might involve (if possible) developing suitable Lyapunov functions for the existence of the endemic equilibrium in the entire global dynamics.
- The model used in this work is based on the assumption of a homogeneous population. Spatial or multi-patch simulations may be used in a future study to represent the variations in transmission across the region.
- Environmental variability and randomness of transmission could be modeled using stochastic models.
- Control techniques may be used optimally to estimate intervention strategies to use that are economical.
- More further extensions are possible to the model by adding more strains of the pathogen or incorporating strain-specific difference, thereby adding more complexity to the pathogen evolution.

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