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Mathematical Modeling and Analysis of Cholera Control Strategies in Resource-Limited Regions

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Abstract Cholera, a major public health concern caused by the Vibrio cholerae bacterium, remains a pressing issue in remote regions of Nigeria, particularly during the dry season when access to clean and treated water is scarce. We seeks to develop a robust model to better understand the rapid transmission of cholera in these areas and assess the effectiveness of various intervention strategies, including public health education, antibiotic treatments, improved water treatment practices, and enhanced environmental sanitation. This addresses the existence and uniqueness of the model, its positivity and boundedness properties, and calculates the basic reproduction number, (R_0), which serves as a critical threshold for disease dynamics. Specifically, when $(R_0 < 1)$, the disease spread will diminish over time, while $(R_0 > 1)$ indicates sustained transmission. Both local and global stability analyses of the model were conducted, alongside a sensitivity analysis to identify key parameters influencing disease control. Furthermore, numerical simulations utilizing the homotopy perturbation method were employed to evaluate the specific contributions of control measures in reducing cholera transmission. The findings offer critical insights into designing effective interventions, particularly by enhancing access to safe water and implementing targeted public health strategies, to curb cholera outbreaks in resource-limited settings.

Keywords: Mathematical Modeling, Cholera Control, Epidemiology, Resource-Limited Regions, Disease Dynamics

I. Introduction

Cholera is a life-threatening disease primarily spread through the fecal-oral route, often resulting from contaminated water and poor sanitation practices [1]. The disease is caused by the bacterium vibrio cholerae, which thrives in environments with inadequate hygiene, leading to acute diarrhea and severe dehydration that can be fatal without timely treatment. Cholera outbreaks impose significant burdens on healthcare systems, increase mortality rates, and deplete medical resources, particularly affecting vulnerable groups such as children and the elderly [2, 3]. In Northern Nigeria, cholera remains a recurring public health issue, exacerbated by an arid climate, limited access to safe drinking water, and insufficient sanitation infrastructure in [4]. The interplay of geographic and socio-economic factors, combined with environmental conditions during the dry season, creates an ideal environment for the proliferation of vibrio cholerae, making disease control efforts particularly complex [5, 6]. To better understand and mitigate cholera outbreaks in such high-risk settings, it is essential to develop models that capture the dynamics of cholera transmission. The Susceptib

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le (S), Vaccinated (S), Exposed (E), Infected (I), Recovered (R) and Bacteria (B) as (SEIRB) model provides a comprehensive mathematical framework for analyzing cholera spread in regions like Northern Nigeria [7-9]. This model integrates critical epidemiological factors, including bacterial transmission, population immunity, and environmental influences on bacterial survival, such as the environmental bacterial persistence coefficient (c) [10]. Cholera spreads through human-environment interactions, especially via contaminated water in areas with poor sanitation [11]. In Northern Nigeria, dry-season water scarcity intensifies human-to-human and environmental transmission by [12, 13]. The environmental bacteria coefficient, influenced by factors like water quality and temperature, drives bacterial growth. Population immunity, shaped by prior exposure, vaccination, and health status, affects vulnerability. The (SEIRB) model captures these dynamics for targeted intervention in [14]. This compartment represents individuals with no prior exposure to cholera and no immunity, making them highly susceptible to infection during outbreaks. Vaccinated individuals, having received the cholera vaccine, possess some degree of protection, underscoring the importance of vaccination in controlling cholera, especially in outbreak-prone areas by [15-19]. Exposed individuals have encountered vibrio cholerae but remain asymptomatic. They may either develop symptoms after the incubation period or recover silently, depending on their immune system [20, 21]. Actively infected individuals can transmit cholera to others, either directly or by contaminating water sources. Recovered individuals, having survived the disease, gain temporary or permanent immunity, influenced by the cholera strain and their health condition [22]. The bacteria compartment includes environmental reservoirs, such as contaminated water, where the bacterium persists and facilitates transmission [23]. The (SEIRB) model effectively captures these dynamics, offering insights into cholera's spread and persistence in Northern Nigeria. Varied immunity levels, shaped by factors like age, nutrition, and healthcare access, impact vulnerability, with children, the elderly, and the immune compromised being at greater risk [24, 25]. A key factor, the environmental bacteria coefficient (c), determines how vibrio cholerae multiplies in water. This parameter, influenced by temperature, nutrient levels, and contaminants, escalates during the dry season in Northern Nigeria [26]. Water scarcity drives reliance on contaminated sources, worsening bacterial growth and cholera risk [27]. The model uses differential equations to describe transitions among compartments (S, V, E, I, R, B), incorporating transmission, recovery, and environmental impacts. Accurate parameter estimation, including that of (c), is challenging due to variations in climate, sanitation, and population density in Northern Nigeria, where these factors significantly affect cholera transmission [28, 29]. A multifaceted approach is necessary, incorporating improved sanitation, better water access, robust healthcare systems, public awareness campaigns and international collaboration [30]. Leveraging numerical simulations to fine-tune control strategies can help develop solutions tailored to Northern Nigeria's unique challenges [31]. By addressing immediate and long-term needs, these measures aim to reduce cholera incidence, improve health outcomes, and build resilient communities in affected regions.

II. Materials and Methods

A. Model formulation

A total population N(t) is considered which is divided into sub-populations of S(t) susceptible, E(t) exposed, I(t) infected, R(t) recovered and B(t) bacteria causing population. The level of individuals migrating into the population at Λ, effective contact rate of an individual τ and the level of the spread induced rate at δ. Transmission rate in cholera disease between the two or more population of individuals being exposed at β . The modification of the disease capacity multiplicative effect is at a rate c and enlightenment through educational program initiatives on the rapid spread on how deadly cholera is at a rate of ω. Prevention on the spread with a waning rate η and regular treatment of cholera disease with antibiotics is at rate of ε. An infected individual are subjected to recover at a rate of r and individuals that are hospitalized having been infected is $(1-\varepsilon)$ while that of infected are said to recover at a rate of $(r+\varepsilon)$. More so, set of bacteria individual form back into the susceptible population through water treatment occurs at rate of T when immunity level is high. Respective individuals across the sub-population are subjected to death naturally by μ. Pictorial illustration of this can be displayed from the fig. 1 and model equation is given by equation 1 below.

$$\frac{dS}{dt} = \Lambda - \beta S(t)I(t) - (\tau+\omega)S(t) + TB(t) - \mu S(t)$$

$$\frac{dE}{dt} = \beta S(t)I(t) - (c+\eta+\mu)E(t)$$

$$\frac{dI}{dt} = \tau S(t) + (c+\eta)E(t) - (\varepsilon+\delta+r+\mu)I(t) \quad (1)$$

$$\frac{dR}{dt} = \omega S(t) + (r+\varepsilon)I(t) - \mu R(t)$$

$$\frac{dB}{dt} = -(T+\mu)B$$

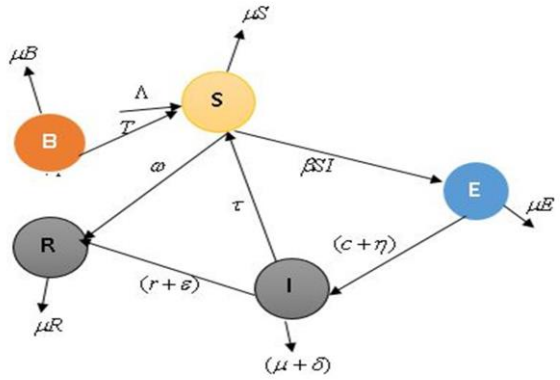

Fig. 1: Schematic Diagram of the Model.

By initial condition that $0 < T \leq 1$. When $T = 0$, vulnerable individuals are not immunized or immunization does not affect the vulnerable compartment. The description of the model parameters can be illustrated in Table1 as shown below.

B. Existence and uniqueness of model solution

The model in equation (1), which represents the spread of an epidemic disease within a human population, requires that its parameters be nonnegative for its existence and uniqueness of the model solution. To ensure that the system of differential equations in equation (1) is both mathematically valid and epidemiologically sound, it is important to establish that the model's state variables remain nonnegative. Equation (1) is considered well-defined at the initial point if the initial conditions are nonnegative.

$S(0) = s_0,, E(0) = e_0, I(0) = i_0, R(0) = r_0, B(0) = b_0$; In that case, the solutions of system (1) will persist in being nonnegative throughout their.

Table 1.Description of model parameters and values

Variable	Description	
$S(t)$	Susceptible population	
$E(t)$	Exposed population	
$I(t)$	Infected population	
$R(t)$	Recovered population	
$B(t)$	Bacteria population	
Parameter	Description	Values
N	Total population	0.1625
Λ	Recruitment rate into the susceptible population	0.001
β	Transmission Coefficient	0.5
τ	Vibro cholera multiplicative effect	0.2
T	Water treatment rate on rapid cholera growth	0.03
μ	Natural death	1.0
ω	Rate of educational program	0.0016
η	Immunity waning rate	0.113
ε	Environmental bacteria capacity	1.0126
c	Treatment rate with antibiotics	1.1927
δ	Induced death	1.0126
r	Recovery rate	1.8273

evolution, $t > 0$ and that these positive solutions are bounded. We thus apply the following theorems

i. Theorem 1

Let (x, y) be distinct points of normed linear space $(X, \|\cdots\|)$ over $\Re$. Then the map of $p : [0,1] \subseteq \Re \to (X, \|\cdots\|)$ such that $p(\lambda) = \lambda x + (1-\lambda)y$ is continuous on [0, 1].

Proof:

Let $\lambda_0 \in [0,1]$ then $p(\lambda_0) = \lambda_0 x + (1-\lambda_0)y$ for any $\lambda_0 \in [0,1]$,

$$\|p(\lambda) - p(\lambda_0)\| = \|(\lambda - \lambda_0)x + (\lambda - \lambda_0)y\| \leq |\lambda - \lambda_0|(\|x\| + \|y\|) \quad (2)$$

If $\varepsilon > 0$ is given, let $\delta = \dfrac{\varepsilon}{\|x\| + \|y\|}$ .If $|\lambda - \lambda_0| < \delta$, then the $\|p(\lambda) - p(\lambda_0)\| < \varepsilon$. Therefore, p is continuous at λ_0. Since λ_0 is an arbitrary point in [0, 1]. Then p is continuous on [0, 1]. Let X be a linear space over $\Re$. If (x, y) are distinct points of X, the set $\lambda x + (1-\lambda)y$ lies in $0 \leq \lambda \leq 1$

Hence, the solutions of system (1) are bounded if we consider the total population. The variation in the total population concerning time is given by:

$$N(t) = \mathrm{S(t) + E(t) + I(t) + R(t) + B(t)} \quad (3)$$

The variation in the total population concerning time is given by:

$$\frac{dN(t)}{dt} = \frac{d}{dt}\left(\mathrm{S(t) + E(t) + I(t) + R(t) + B(t)}\right) \quad (4)$$

Such that

$$\frac{dN(t)}{dt} = \Lambda - \mu(S + E + I + R + B) \quad \Rightarrow \frac{dN(t)}{dt} \leq \Lambda - \mu N \quad (5)$$

Hence, it is obtained that

$\frac{dN(t)}{dt} + \mu N \le \Lambda$, using the integrating factor concept on the total population N(t) and this leads to $N(t)e^{\mu t} = \frac{\Lambda}{\mu}e^{\mu t} + c$ (6)

Firstly,

$N(0) = \frac{\Lambda}{\mu} + ce^{-\mu(0)}$, resulting to

$$c = N(0) - \frac{\Lambda}{\mu} \quad (7)$$

Thus, substituting (6) into (5) as time progressively increases yields:

$$\lim_{t\to\infty} N(t) \le \lim_{t\to\infty}\left[\frac{\Lambda}{\mu} + \left(N(0) - \frac{\Lambda}{\mu}\right)e^{-\mu t}\right] = \frac{\Lambda}{\mu} \quad (8)$$

Then $N(0) \le \frac{\Lambda}{\mu}$, then $N(t) \le \frac{\Lambda}{\mu}$.

This is a positive invariant set under the flow described by (8) so that no solution path leaves through any boundary $\Re_+^5$. Hence, it is sufficient to consider the dynamics of the model in the domain $\Re_+^5$. In this region, the model can be considered has be mathematically and epidemiologically well-posed. This shows that the total population and the subpopulation $S(t), E(t), I(t), R(t), B(t)$ of the model are bounded and is a unique solution. Hence, its applicability to studying physical systems is feasible.

C. Positivity and Boundedness of Model

This shows that the total population $N(t)$, and the subpopulation $S(t), E(t), I(t), T(t), R(t)$ of the model are bounded and is a unique solution. Hence, its applicability to study physical systems is feasible.

ii. Theorem 2

Suppose $X = x_0$ is a space of consecutive real number and which are defined as

$$L(x,y) = \left(\sum_{i=1}^{n}|x_i|^{\Omega}\right)^{\frac{1}{\Omega}} \qquad \Omega \ge 1 \quad (9)$$

X with the metric is called ξ_n^{Ω} space. If $\sum^{\infty}|x|^{\Omega} < \infty$ or absolutely convergent and

$L(x,y) = \left(\sum_{i=1}^{\infty}|x_i - y_i|^{\Omega}\right)^{\frac{1}{\Omega}}$, then X with this metric is called an ξ^{Ω} space.

Proof:

It can be checked that for each n:

$$0 \le x_1^2 + x_2^2 + x_3^2 + \ldots + x_n^2 \le \left(|x_1| + |x_2| + |x_3| + \ldots + |x_n|\right)^2 \quad (10)$$

This will result to;

$$x_1^2 + x_2^2 \le \left(|x_1| + |x_2|\right)^2 \quad (11)$$

Therefore,

$0 \le \left(x_1^2 + x_2^2 + x_3^2 + \ldots + x_n^2\right)^{\frac{1}{2}} \le |x_1| + |x_2| + |x_3| + \ldots + |x_n|$,

If $\sum_{n=1}^{\infty}|x_n|$ converges , that is $\sum_{n=1}^{\infty}|x_n|$ is absolutely convergent, then

$$0 \le \left(x_1^2 + x_2^2 + x_3^2 + \ldots + x_n^2\right)^{\frac{1}{2}} \le |X_1| + |X_2| + |X_3| + \ldots + |X_n| = \sum_{n=1}^{\infty}|X_n| < \infty \quad (12)$$

Therefore,

$$0 \le x_n = x_1^2 + x_2^2 + x_3^2 + \ldots + x_n^2 \le \left[\sum_{n=1}^{\infty}|x_n|\right] < \infty \quad (13)$$

The sequence x_n is monotone increasing and bounded above, it therefore converges. Thus $\sum_{n=1}^{\infty} x_n$ converges absolutely, i. E if $x_n \in \xi^1$, then $x_n \in \xi^2$ where $\xi^1 \leq \xi^2$. In case of ξ^1 denote the set of all sequences of x_n of real numbers such that $\sum_{n=1}^{\infty} x_n$ is convergent absolutely. i.e $\sum_{n=1}^{\infty} |x_n| < \infty$ whereas ξ^2 denote the set of all sequence x_n of real numbers such that $\sum_{n=1}^{\infty} x_n^2 < \infty$ converges. From the proceeding $x_n \in \xi^1 \Leftrightarrow x_n \in \xi^2$ i.e $\xi^1 \subseteq \xi^2$. Further, if $x_n = \frac{1}{n^{\frac{3}{4}}}$, then $\sum_{n=1}^{\infty} |x_n|$ diverges and thus $x_n \notin \xi^1$. But $\sum_{n=1}^{\infty} x_n^2 = \sum_{n=1}^{\infty} \frac{1}{n^{\frac{3}{4}}}$ converges, implying that $x_n \in \xi^2$. We conclude that $\xi^1 \subseteq \xi^2$ and thus $\xi^1 \neq \xi^2$. If (x_n, y_n) are sequences of real numbers, then;

$$\sum_{n=1}^{\infty} (x_i - y_i)^2 \leq \sum_{n=1}^{\infty} x_i^2 + \sum_{n=1}^{\infty} y_i^2 + 2\left[\sum_{n=1}^{\infty} x_i^2\right]^{\frac{1}{2}}\left[\sum_{n=1}^{\infty} y_i^2\right]^{\frac{1}{2}} \quad (14)$$

Therefore if $\sum_{n=1}^{\infty} x_i^2 < \infty$ and $\sum_{n=1}^{\infty} y_i^2 < \infty$ then $\sum_{n=1}^{\infty} (x_i - y_i)^2 < \infty$ for all n. The monotone increasing sequence $\left[\sum_{n=1}^{\infty} (x_i - y_i)^2\right]$ is then bounded above and hence converges i.e. $\sum_{n=1}^{\infty} (x_i - y_i)^2 < \infty$. Thus $(x_i - y_i)^2 \in \xi^2$ if $(x_n, y_n) \in \xi^2$.

Given that the $S(0) = s_0 > 0, V(0) = v_0 > 0, E(0) = e_0 > 0, I(0) = i_0 > 0, T(0) = t_0 > 0, R(0) = r_0 > 0$, and $t > 0$, then the solutions S of the system (1) will always be nonnegative. Let:

$$\Psi = \left\{ \left(S(t), E(t), I(t), R(t), B(t)\right) \in \Re_+^5 : N(t) \leq \frac{\Lambda}{\mu} \right\} \quad (15)$$

If $f_i, i = 1, 2....5$ where f is a constant.

Then,

$$\left|\frac{df_1}{dS}\right| = |(\beta + \mu + \tau + \omega)| < \infty, \left|\frac{df_1}{dE}\right| = |0| < \infty, \left|\frac{df_1}{dI}\right| = |\beta| < \infty, \left|\frac{df_1}{dR}\right| = |0| < \infty, \left|\frac{df_1}{dB}\right| = |0| < \infty,$$

$$\left|\frac{df_2}{dS}\right| = |\beta| < \infty, \left|\frac{df_2}{dE}\right| = |c + \varepsilon + \mu| < \infty, \left|\frac{df_2}{dI}\right| = |\beta| < \infty, \left|\frac{df_2}{dR}\right| = |0| < \infty, \left|\frac{df_2}{dB}\right| = |0| < \infty$$

$$\left|\frac{df_3}{dS}\right| = |0| < \infty, \left|\frac{df_3}{dE}\right| = |c + \eta)| < \infty, \left|\frac{df_3}{dI}\right| = |(\varepsilon + \delta + r + \mu)| < \infty, \left|\frac{df_3}{dR}\right| = |0| < \infty, \left|\frac{df_3}{dB}\right| = |0| < \infty$$

$$\left|\frac{df_4}{dS}\right| = |\omega| < \infty, \left|\frac{df_4}{dE}\right| = |0| < \infty, \left|\frac{df_4}{dI}\right| = |(r + \varepsilon)| < \infty, \left|\frac{df_4}{dR}\right| = |\mu| < \infty, \left|\frac{df_4}{dB}\right| = |0| < \infty \quad (16)$$

$$\left|\frac{df_5}{dS}\right| = |0| < \infty, \left|\frac{df_5}{dE}\right| = |0| < \infty, \left|\frac{df_5}{dI}\right| = |0| < \infty, \left|\frac{df_5}{dR}\right| = |0| < \infty, \left|\frac{df_5}{dB}\right| = |(T + \mu)| < \infty$$

Equation (16) confirms that system (1) is bounded, invariantly and attractively influence the bounded region of $\Re_+^5$

D. Cholera-Non-Infected Equilibrium State

The equilibrium state of non-infected individuals with cholera signifies a system devoid of vibro-cholerae, encompassing individuals categorized as infected (I), exposed (E), and recovered (R) $I = E = R = 0$.

$$0 = \Lambda - \beta S(t)I(t) - (\tau + \omega)S(t) + TB(t) - \mu S(t)$$

$$0 = \beta S(t)I(t) - (c + \eta + \mu)E(t)$$

$$0 = \tau S(t) + (c + \eta)E(t) - (\varepsilon + \delta + r + \mu)I(t) \quad (17)$$

$$0 = \omega S(t) + (r + \varepsilon)I(t) - \mu R(t)$$

$$0 = -(T + \mu)B$$

At no outbreak of tuberculosis, the class of the disease is subjected as $t = 0$, from (21),

$0 = \Lambda - \beta S(t)I(t) - (\tau + \omega)S(t) + TB(t) - \mu S(t)$, $R = 0$.where, $S = \frac{\Lambda}{\mu}$

Thus, the disease-free equilibrium yields:

$$(S, E, I, R) = \left(S_0 = \frac{\Lambda}{\mu}, E_0 = 0, I_0 = 0, R_0 = 0 \right) \quad (18)$$

E. Steady-State Prevalence

It is crucial to highlight the dynamic nature of cholera prevalence, especially its central role in sustaining outbreaks within a population. To analyse the system at equilibrium, consider the set of equations in (1), where the equilibrium points represent the endemic states of cholera prevalence. $\Phi = (S^\bullet, E^\bullet, I^\bullet, R^\bullet, B^\bullet)$ and $t > 0$

$$S^* = \frac{1 + c\Lambda(\mu + \delta)(\varepsilon E^* + \delta R^*)(T + \gamma + \omega)}{\beta - \mu(1 + r)(T + \gamma + \omega)(\mu + \delta + \varepsilon)(\mu + \gamma + T)},$$

$$E^* = \frac{(\varepsilon E + \delta + \omega)(T + \gamma + \omega) - \beta(\mu + \varepsilon + \delta)(\omega + \gamma + \tau)}{\beta - \mu(\omega + \delta + r)(T + \gamma + \omega)(\mu + \delta + c)(\mu + \gamma + T)},$$

$$I^\bullet = \frac{(\mu + \delta + c)(\varepsilon E^* + \delta R^* + \omega)(T + \gamma + \delta)}{(T + \gamma + \omega)(\mu + \delta + \tau)(\mu + \gamma + T)} \quad (19)$$

$$R^\bullet = \frac{\sqrt{(\mu + \delta + \omega)(\varepsilon E^* + \delta R^*)} + (T + \gamma + \omega)}{(T + \gamma + r)(\mu + \delta + \omega)(\mu + \gamma + T)}$$

$$B^\bullet = \frac{(r + \mu + \delta)(\varepsilon E^* + \delta R^*)(T + \gamma)}{(T + c + r)(\mu + \delta + \omega)(\mu + r + T)}$$

F. **The Disease Threshold R_***

The basic reproduction number, denoted as R_*. To quantify the likelihood of new cholera infections arising from a single infectious individual in a previously unexposed population, we apply the next-generation matrix approach to construct the system outlined in (1), with a focus on infectious compartments. In this method, the F and V matrices are computed, representing the rate of new infections and the rate of transitions into and out of the infected compartment, respectively. This approach captures the dynamics of cholera transmission and reinfection, emphasizing the importance of treatment as a critical control measure. These matrices are obtained using a complex derivation from the equations in System (1), $R_* = \rho(G - \lambda I)$ taking $G = F \times V^{-1}$ and ρ is the spectral radius of the matrix $|G - \lambda I|$.From the system of equation (1) it is obtained for matrix

$$F \text{ and } V : F_i = \left(\frac{\partial f_i(x_i)}{\partial x_j} \right) V_i = \left(\frac{\partial v_i(x_i)}{\partial x_j} \right) \quad (20)$$

And such that

$$f = \begin{pmatrix} \beta S_0 \\ 0 \end{pmatrix} \quad \text{and}$$

$$v = \begin{pmatrix} (c + \eta + \mu)E(t) \\ \tau S(t) + (c + \eta)E(t) - (\varepsilon + \delta + r + \mu)I(t) \end{pmatrix} \quad (21)$$

Then,

$$F=\begin{pmatrix}\frac{\beta\Lambda}{\mu}\\0\end{pmatrix}V=\begin{pmatrix}(c+\eta+\mu) & 0\\ (c+\eta) & (\varepsilon+\delta+r+\mu)\end{pmatrix}$$

$$FV^{-1}=\frac{1}{(c+\mu+\varepsilon)(\mu+r+T)}\begin{pmatrix}\frac{\beta\Lambda}{\mu} & 0\\ 0 & 0\end{pmatrix}\begin{pmatrix}(c+\mu+\eta) & 0\\ (c+\eta) & (r+\delta+\mu+\varepsilon)\end{pmatrix}$$

$$R_*=\frac{\beta\omega\Lambda}{\mu(r+\mu+\varepsilon)(\mu+\gamma+T)(\delta+r+c)} \quad (22)$$

It results that the basic reproductive ratio determines the number of infected individual migrating to the subpopulation of exposed and infected, as this affect the level of recovery form the spread of tuberculosis. The leading eigenvalue of the non-invariant is the basic reproduction number of the disease model

G. Local stability of the disease-free state

We examined the local stability of the disease-free state for cholera by analysing the minimal recurrence rate impact. When the recurrence rate $R_*<1$, the disease declines, to determine stability using a Jacobian matrix and a characteristic equation.

Lemma 1

The disease-free state of the model is locally asymptotically stable $R_*<1$, otherwise $R_*>1$ if and only if the disease state prevails

Proof:

The disease-free equilibrium obtained as the Jacobian matrix of the system of (1) is evaluated at the disease free State using the linearization thus;

$$J_{\ell_0}=\begin{pmatrix}(-\beta+\tau+\omega+\mu) & 0 & -\beta & 0 & 0\\ \beta & -(c+\eta+\mu) & 0 & 0 & 0\\ \tau & (c+\eta) & -(\varepsilon+\delta+r+\mu) & 0 & 0\\ \omega & 0 & (r+\varepsilon) & 0 & -\mu\\ 0 & 0 & 0 & 0 & -(T+\mu)\end{pmatrix} \quad (23)$$

$$\begin{vmatrix}(-\beta+\tau+\omega+\mu)-\lambda & 0 & -\beta & 0 & 0\\ \beta & -(c+\eta+\mu)-\lambda & 0 & 0 & 0\\ \tau & (c+\eta) & -(\varepsilon+\delta+r+\mu)-\lambda & 0 & 0\\ \omega & 0 & (r+\varepsilon) & -\mu-\lambda & 0\\ 0 & 0 & 0 & 0 & -(T+\mu)-\lambda\end{vmatrix}=0$$

Computing for the eigenvalues, $\left|J_{E_1}-\lambda_i I\right|=0$, from the Jacobian matrix the respective eigen values of the matrix can be obtained as;

$$\begin{vmatrix}-(\mu+\gamma+T)-\lambda & \delta\\ T+\gamma & -(\mu+\delta)-\lambda\end{vmatrix}=0 \quad (24)$$

$$(-(\mu+\gamma+T)-\lambda_3)(-(\mu+\delta+\omega)-\lambda_4)=0,\lambda_4=-(\mu+c+\delta),\lambda_3=-(\mu+\gamma+T)\lambda_3=-(\mu+r+T)-(\mu+\varepsilon)-\lambda_2|A|=0$$

$$\lambda_2=-(\mu+\varepsilon+\delta)\lambda_1=-\mu,\lambda_1=-\mu,\lambda_2=-(\mu+\varepsilon+\tau),\lambda_3=-(\mu+\gamma+T),\lambda_4=-(\mu+\delta+\omega)$$

The negativity of the invariants region with respective eigen values obtained for the model equation is asymptotically stable

H. Local Stability of Endemic Equilibrium Point

Theorem: The regional resilience of the persistent equilibrium of the proposed model is locally asymptotically Stable if and unstable otherwise if $R_*>1$

Proof:

Suppose,

$$S=x+S^*,E=y+E^*,I=z+I^*,R=a+R^*,B=b+B^* \quad (25)$$

Linearizing equation (1), is then obtained as

$$\left.\begin{aligned}
\frac{dx}{dt} &= -2\beta\kappa z(1+z)^{-1} - \mu\kappa + \text{higher order} + \text{nonlinear terms...}\\
\frac{dy}{dt} &= 2\beta\kappa z(\tau+\omega+\delta)^{-1} - (\mu+\varepsilon+r)y + \text{higher order} + \text{nonlinear terms...}\\
\frac{dz}{dt} &= \varepsilon y + (\mu+\gamma+\omega)z - Tz + \delta a + \text{higher order} + \text{nonlinear terms...}\\
\frac{da}{dt} &= (T+\gamma+\delta)z - (\mu+\delta+c)a + \text{higher order} + \text{nonlinear terms...}\\
\frac{db}{dt} &= zb - \mu b + \text{higher order} + \text{nonlinear terms...}
\end{aligned}\right\} \quad (26)$$

Jacobian matrix of the system of,

$$\begin{vmatrix}
-(\beta(\omega+r+c)^{-1}+\mu) & 0 & (2\beta(\mu+\gamma+c)^{-1}+\mu) & 0\\
(\beta(\omega+r+c)^{-1}+\mu) & -(\mu+\varepsilon) & (2\beta(\tau+c+\gamma)^{-1}+\mu) & 0\\
0 & (\varepsilon+\gamma+\eta) & -(\mu+\gamma+T) & 0\\
0 & 0 & (\gamma+\omega+r+c) & -(\mu+\delta+c)
\end{vmatrix} = 0 \quad (27)$$

The resulting eigenvalue of the above matrix is obtained as;

$$\left(-(2\beta(\omega+r+c)+\mu)-\lambda_1\right)\left(-(\varepsilon+\eta+\mu)-\lambda_2\right)\left(-(T+\gamma+\mu)-\lambda_3\right)\left(-(\delta+\mu+c)-\lambda_4\right)=0$$

If

$$a=-(2\beta(1+\alpha)^{-1}, b=-(\varepsilon+\mu), c=-(T+\gamma+\mu), d=-(\delta+\mu), e=-\mu \quad (28)$$

It is therefore obtained that

$$(a-\lambda)(b-\lambda)(c-\lambda)(d-\lambda)(e-\lambda)$$

$$\lambda^5-[(a+b)(c+d)+ab+cde]\lambda^4-[abe(c+d)+(b+e)+d(a+c)]\lambda^3$$

$$[b(a+b)+(c+d)+a(d+e)]\lambda^2+[ae+ad+bd+ac]\lambda+abcde=0 \quad (29)$$

Therefore, the persistent resilience of the respective Eigen values in the model invariance region of $\Re_5^+$ is asymptotically stable.

I. Numerical simulation

We conducted numerical simulation on the mathematical model, we create the following iterative scheme of Laplace adomian decomposition method for the model equation. The Laplace adomian decomposition method was employed to computationally analyse the epidemic model. MAPLE 18 software facilitated the generation of iteration formulas for each compartment. These formulas were then iteratively solved, enabling the numerical evaluation of the model's dynamics and providing insights into the epidemic's behaviour and progression. Taking the Laplace transform of both sides of the above equation.

$$L\left[\frac{dS}{dt}\right] = L[\Lambda] - L[\beta S(t)I(t) - (\tau+\omega)S(t) + TB(t) - \mu S(t)]$$

$$L\left[\frac{dE}{dt}\right] = L[\beta S(t)I(t)] - L[(c+\eta+\mu)E(t)]$$

$$L\left[\frac{dI}{dt}\right] = L[\tau S(t) + (c+\eta)E(t)] - L[-(\varepsilon+\delta+r+\mu)I(t)] \quad (30)$$

$$L\left[\frac{dR}{dt}\right] = L[\omega S(t) + (r+\varepsilon)I(t)] - L[\mu R(t)]$$

$$L\left[\frac{dB}{dt}\right] = L[-(T+\mu)B]$$

Substituting into (30) to yield

$$mL[S(t)] = S(0) + \Lambda - L[\beta S(t)I(t) - (\tau+\omega)S(t) + TB(t) - \mu S(t)]$$

$$mL[E(t)] = E(0) + L[\beta S(t)I(t)] - L[(c+\eta+\mu)E(t)]$$

$$mL[I(t)] = I(0) + L[\tau S(t) + (c+\eta)E(t)] - L[-(\varepsilon+\delta+r+\mu)I(t)]$$

$$mL[R(t)] = R(0) + L[\omega S(t) + (r+\varepsilon)I(t)] - L[\mu R(t)]$$

$$mL[B(t)] = B(0) + L[-(T+\mu)B] \quad (31)$$

Subsequently, iteration result obtained form the above equation is deduced as;

$$\sum_{k=0}^{\infty} S_n(t) = s_0 + \pi t + L^{-1}\left(\frac{1}{m}L\left[-\alpha\sum_{k=0}^{\infty}\pi_n - \phi_1\sum_{k=0}^{\infty}S_n + \phi_2\sum_{k=0}^{\infty}V_n - \mu\sum_{k=0}^{\infty}S_n\right]\right)$$

$$\sum_{k=0}^{\infty} E_n(t) = v_0 + \pi t + L^{-1}\left(\frac{1}{m} L\left[-\sum_{k=0}^{\infty}((1-\varepsilon)\beta) - \beta_1 \sum_{k=0}^{\infty} V_n + \beta_2 \sum_{k=0}^{\infty} V_n - \mu \sum_{k=0}^{\infty} E_n\right]\right)$$

$$\sum_{k=0}^{\infty} I_n(t) = e_0 + L^{-1}\left(\frac{1}{m} + L\varphi \sum_{k=0}^{\infty} \varepsilon_n - L[\beta - (k+\mu)]\sum_{k=0}^{\infty} I_n\right)$$

(32)

$$\sum_{k=0}^{\infty} R_n(t) = i_0 + L^{-1}\left(\frac{1}{m} + L\delta \sum_{k=0}^{\infty} E_n - L[(\alpha+\mu+\gamma+\phi_2)]\sum_{k=0}^{\infty} R_n\right)$$

$$\sum_{k=0}^{\infty} B_n(t) = i_0 + L^{-1}\left(\frac{1}{m} + L\alpha \sum_{k=0}^{\infty} E_n - L[(\varphi+\mu)]\sum_{k=0}^{\infty} B_n\right)$$

The initial approximations of each class are given by;

$$S_0(t) = s_0 + \Lambda t, E_0(t) = e_0, I_0(t) = i_0, R_0(t) = r_0, B_0(t) = b_0$$

Now, comparing the coefficients $n=1$. Using the recurrence relations obtained from the iterations. Compartmentally it is obtained that

$$S_1(t) = (\Lambda i_0 s_0 - \mu s_0 - \beta s_0 + r_1 e_0)t + \left(-\frac{1}{2}\alpha i_0 \varepsilon - \frac{1}{2}\mu\Lambda - \frac{1}{2}\delta\tau_1\right)t^2$$

$$E_1(t) = (\gamma i_0 s_0 - \mu e_0 - \delta e_0)t + \frac{1}{2}\tau\eta_1 t^2$$

$$I_1(t) = kE - (r+\mu+\gamma+c_2)I(-\delta i_0 - r + \mu + \gamma + c_2 e_0 i_0 + \delta e_0)t$$

$$R_1(t) = (-\mu r_0 + \tau\eta_2 s_0 v_0 + (\tau+\mu)i_0)t$$

$$B_1(t) = \frac{1}{3}\varepsilon\delta_2\left(-\varepsilon + \mu r_0 + \frac{1}{2}(\delta+\mu)i_0\right)t$$

(33)

$$R_1(t) = (-\mu r_0 + \tau\eta_2 s_0 v_0 + (\tau+\mu)i_0)t$$

$$B_1(t) = \frac{1}{3}\varepsilon\delta_2\left(-\varepsilon + \mu r_0 + \frac{1}{2}(\delta+\mu)i_0\right)t$$

Further iterations are done to obtain successive iterative terms at n=2

$$S_2(t) = \begin{pmatrix} \frac{1}{2}\alpha^2 i^2 s_0 + \frac{1}{2}\alpha i s_0 + \frac{1}{3}\alpha i s_0\mu_0 + \frac{1}{2}\alpha i s_0\rho_0 - \frac{1}{2}\alpha i s_0 e_0 + \frac{1}{2}\alpha i s_0\beta_1 - \frac{1}{2}\alpha i s_0\beta_2 \\ + \frac{1}{2}\mu^2 s_0 + \beta_1\mu s_0 + \beta_1\mu v_0 + \frac{1}{2}\beta^2 s_0 + \frac{1}{2}\beta_1\beta_2 s_0 - \frac{1}{2}\beta_1\beta_2 v_0 - \frac{1}{2}\beta_2^2 v_0 \end{pmatrix} t^2$$
$$+\left(\frac{1}{6}\alpha^2 i^2\theta + \frac{1}{3}\alpha i_0\pi\delta + \frac{2}{3}\alpha i_0\pi\mu + \frac{1}{3}\alpha i_0\pi\rho - \frac{1}{3}\alpha e_0\theta\sigma + \frac{1}{3}\alpha i_0\pi\beta_1 + \frac{1}{6}\mu^2\theta + \frac{1}{3}\beta_0\pi\mu + \frac{1}{6}\beta_1^2\pi + \frac{1}{6}\beta_2\pi\beta_1\right)t^3$$

$$E_2(t) = \left(-\frac{1}{2}\alpha i s_0\beta_1 + \frac{1}{2}\mu^2 v_0 - \beta_1\mu s_0 + \beta_2\mu s_0 - \frac{1}{2}\beta^2 s_0 + \frac{1}{2}\beta_1\beta_2 s_0 - \frac{1}{2}\beta_1\beta_2 v_0 + \frac{1}{2}\beta_2^2 v_0\right)t^2$$
$$+\left(-\frac{1}{6}\alpha i_0\pi\beta_1 - \frac{1}{3}\beta_1\mu\pi - \frac{1}{6}\beta_1^2\pi + \frac{1}{6}\beta_2\pi\beta_1\right)t^3$$

$$I_2(t) = \left(-\frac{1}{6}\alpha^2 i^2\pi - \frac{1}{3}\alpha i_0\pi\delta - \frac{2}{3}\alpha i_0\pi\mu - \frac{1}{3}\alpha i_0\pi\rho + \frac{1}{3}\alpha e_0\pi\sigma_1 - \frac{1}{6}\mu^2\pi - \frac{1}{6}\alpha i_0\pi\beta_1\right)t^3$$
$$+\left(-\frac{1}{2}\alpha^2 i^2 s_0 - \frac{1}{2}\alpha i s_0 - \frac{2}{3}\alpha i s_0\mu_0 - \frac{1}{2}\alpha i s_0\rho_0 + \frac{1}{2}\alpha i s_0 v_0 - \mu^2 i e_0\beta_1 + \frac{1}{2}\alpha i e_0\sigma^2\right)t^2$$

(34)

$$R_2(t) = -\frac{1}{6}\alpha^2 i^2\theta + \begin{pmatrix} \frac{1}{2}\sigma\alpha i s_0 + \frac{1}{2}\delta^2 i_0 + \delta\mu i_0 - \frac{1}{2}\delta\sigma e_0 + \frac{1}{2}\mu^2 i_0 - \mu\rho i_0 \\ -\mu\sigma i_0 + \frac{1}{2}\rho^2 i_0 - \frac{1}{2}\rho\sigma e_0 - \frac{1}{2}\sigma^2 e_0 \end{pmatrix} t^2$$

$$B_2(t) = -\frac{1}{6}\alpha^2 i^2\theta + \begin{pmatrix} \frac{1}{2}\sigma\alpha i s_0 + \frac{1}{2}\delta^2 i_0 + \delta\mu i_0 - \frac{1}{2}\delta\sigma e_0 + \frac{1}{2}\mu^2 i_0 - \mu\rho i_0 \\ -\mu\sigma i_0 + \frac{1}{2}\rho^2 i_0 - \frac{1}{2}\rho\sigma e_0 - \frac{1}{2}\sigma^2 e_0 \end{pmatrix} t^2$$

and so on. This can be further till desired number of iterations are obtained. Thus, the obtained raw solution to each model compartment is obtained as:

$$S(t) = \sum_{k=0}^{3} s_k(t), V(t) = \sum_{k=0}^{3} v_k(t), E(t) = \sum_{k=0}^{3} e_k(t), I(t) = \sum_{k=0}^{3} i_k(t), R(t) = \sum_{k=0}^{3} r_k(t)$$

(35)

Evaluating these series results using the corresponding variables and parameter values,

$$S(t) = 500.012 - 30.4440t + 1.1315290300t^2 - -0.05075029853t^3 - 3.509616000\times10^{-13}t^5 - 5.179149070\times10^{-7}t^4$$

$$E(t) = 65 + 18.1785t - 1.171778775\,t^2 + 0.0492956076\,5t^3 + 5.087939775\times10^{-7}t^4 + 3.509616000\times10^{13}t^5$$
$$I(t) = 23.0.9 - 60t + 0.0292567500t^2 - 0.0008440367798t^3 - 4.378044000\times10^{-9}t^4$$
$$R(t) = 23.0.9 - 60t + 0.0292567500t^2 - 0.0008440367798t^3 - 4.378044000\times10^{-9}t^4 \quad (36)$$
$$B(t) = 14 - 0.0155t - 0.005054500000t^2 + 0.0001458242541t^3 + 2.473075000\times10^{-10}t^4$$

Hence from the results of successive iterations, comparison of control intervention effects on sub-populations in its graphical illustration depicts as;

III. Results and Discussion

From the simulation of iterative values of the model formulation using Laplace adomian decomposition method these are computed graphically with the help Maple-18 software as for respective compartments. The results of Fig. 2 to Fig.5 represents the effects of the target parameters on the compartments of the model solution, these are graphically illustrated as:

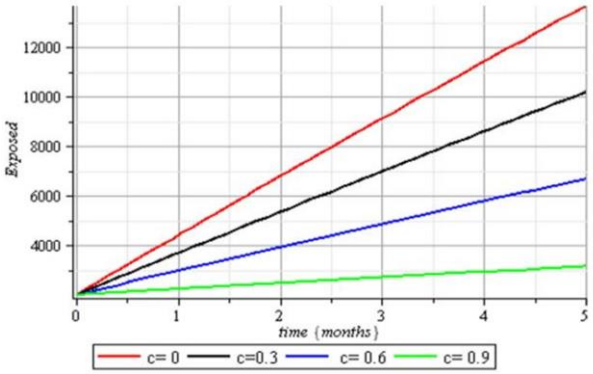

Fig 2. The Effect of Antibiotics (c) on the Exposed Population

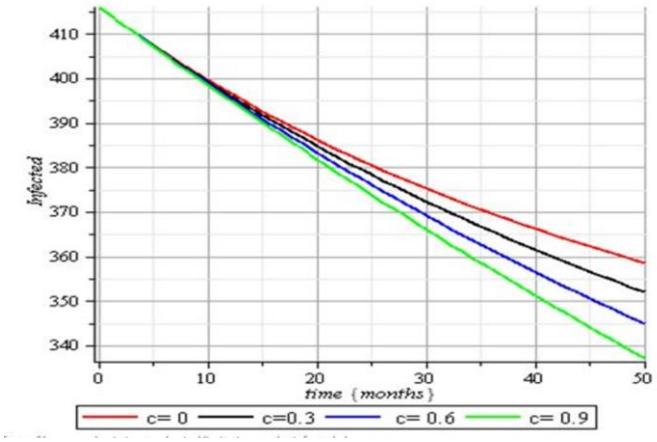

Fig 3. The Effect of Antibiotics (c) on the Infected Population

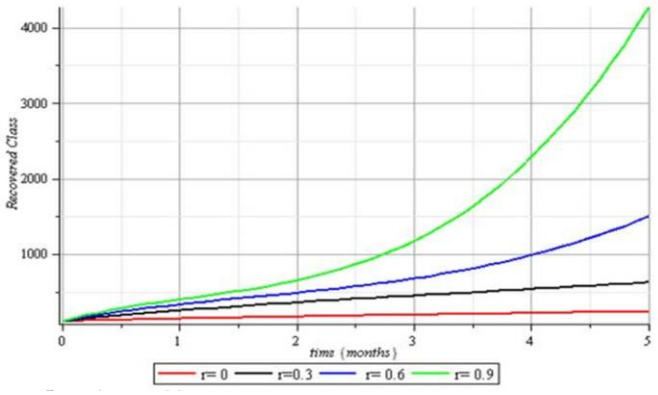

Fig 4. Water Treatment on Recovered Population brings about a Decline in the Disease Population

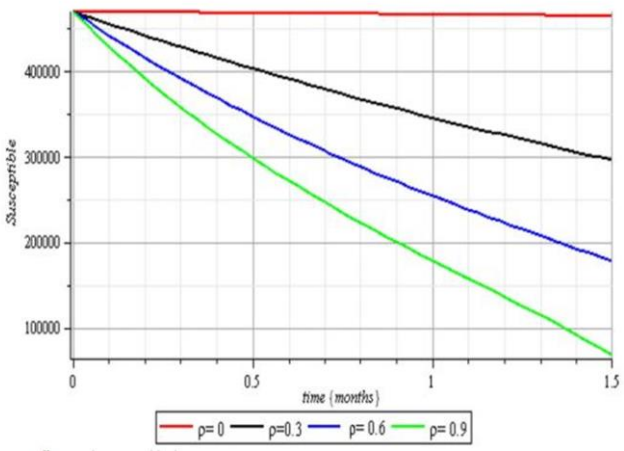

Fig 5. Reduction in Environmental Capacity Effect on the Susceptible Population.

A. Discussion

It is obtained that Fig 2: The effect of antibiotics is a prominent tool in reducing the spread of cholera to the vulnerable population. Fig 3: Depicts the effect of antibiotics on the infected population as these bring about a decline in the outbreak of cholera. Fig 4: Shows that treatment of water in this region will bring about a drastic measure to the control of cholera as the level of the spread reduces in the population while fig 5:

The level of environmental capacity and sensitization on the set of vulnerable populations reduces as it brings about a rapid decline in the spread cholera.

IV. Conclusion

The integration of mathematical modeling with comprehensive control strategies has been instrumental in mitigating cholera spread in remote Nigerian areas during the dry season. Incorporating educational programs, antibiotics, water treatment, and environmental cleanliness into the model has led to significant progress. These initiatives have raised awareness, enabled prompt treatment, ensured access to clean water, and improved sanitation, collectively reducing the impact of cholera outbreaks. Continued collaboration and targeted interventions are crucial for sustaining these efforts and enhancing resilience against cholera in vulnerable populations.

This research underscores the critical need for targeted interventions to address cholera outbreaks in remote areas of Nigeria during dry seasons. Based on the findings, it is recommended to implement enhanced water treatment infrastructure, promote sustainable farming practices and conduct community-specific educational programs. Additionally, policymakers, health practitioners should collaborate with local communities to develop and implement contextually relevant preventive measures. This comprehensive approach will contribute significantly to mitigating cholera spread and building resilience in vulnerable regions.

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