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Controlling the Spread of Malaria-Cholera Co-infection with Effective Intervention Strategy

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Abstract This paper proposes an optimal control of intervention strategies for malaria-cholera co-infection with emphasis on five control strategies namely: treated bed nets, treatments of malaria, indoor residual spray, sanitation and treatment of cholera to prevent disease spread in a population. A non-linear system of differential equations is formulated to study the dynamics of the proposed model. The disease-free equilibrium (DFE) and endemic equilibrium (EE) states were obtained. The basic reproduction numbers, $\mathcal{R}_0^C$, $\mathcal{R}_0^V$ that determine the transmission of the diseases were derived. A sensitivity analysis on the reproduction numbers to determine the parameters that have impact on the reproduction number were carried out. Using the Routh-Hurwitz criterion and Castillo-Chavez techniques, the conditions for stability of the disease-free equilibrium were established. The result of the stability revealed that, if the reproduction number is kept below to unity, malaria-cholera co-infection can be entirely eradicated. The sensitivity analysis results paved way for the implementation of a controlled system, which was solved using Pontryagin's Maximum Principle (PMP) and an optimality system was obtained. The optimality system was then solved numerically using forward backward sweep approach and graphs were produced.

MSC: 26A33, 34A12, 34A43, 34D20
Keywords: Stability; Hamiltonian; Transmission; Equilibrium states; Epidemiology; Invariant region

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1. Introduction

When the vibrio cholera bacteria is ingested through food or drink, it can cause acute diarrhea. Cholera continues to be a global public health concern and a sign of inequality and underdevelopment in society. According to research estimates, cholera causes between 1.3 and 4.0 million infections and between 21 000 and 143 000 fatalities globally each year [23]. Though vibrio cholerae (V. cholerae) has several serogroups, only O1 and O139 cause outbreaks. Every recent outbreak has been caused by V. cholerae O1. V. cholerae O139, which was discovered for the first time in Bangladesh in 1992, has historically caused outbreaks but has only lately been linked to isolated cases. The illnesses brought on by the two serogroups are identical. Cholera originated in the Ganges delta of India and spread around the world throughout the 19th century, according to [23]. Millions of people died worldwide as a result of six more pandemics that followed. The current epidemic started in 1961 in South Asia, moved to Africa in 1971, and finally reached the Americas in 1991. Many nations have had a cholera outbreak [22].

Africa saw numerous cholera outbreaks, and the previously stated cholera serogroups were connected to fatalities in Egypt, South Africa, Uganda, Kenya, and other nations. Additionally, there has been an epidemic in Somalia, in Eastern Africa. The Ministry of Health in Somalia has identified 81 new suspected cases of cholera; no deaths have been reported. Cases have also been discovered in the subtropical region of west Africa in nations where the disease is associated with mortality, including Guinea in 2012, where the sickness was introduced by fisherman from Sierra Leone who crossed the border. Furthermore, Togo experienced a recent outbreak. Between November 11 and December 20, 2020. Two deaths (case fatality ratio: 3%), along with 67 probable cholera cases with vomiting and diarrhea, were reported from the communities of "Golfe 1" and "Golfe 6" in Lom, Togo. At least one incidence was confirmed by four health areas (Katanga, Adakpam, Gbtsogb in Golfe 1 and Kangnikop in Golfe 6) in the affected municipalities [23].

The disease cholera is curable quite quickly. Oral rehydration solution (ORS) is a successful treatment for most patients when administered promptly. Dissolve the WHO/UNICEF ORS regular sachet in one liter (L) of clean water. Adult patients may require up to 6 L of ORS on the first day in order to correct mild dehydration. Patients who are critically dehydrated require intravenous fluids right away because they run the danger of slipping into shock. Antibiotics are also administered to these patients in an effort to reduce the duration of diarrhea, the volume of rehydration fluids required, and the quantity and timing of V. cholerae excretion in the stool. Large doses of antibiotics are not advised as they may cause antibiotic resistance and have no proven effect on the spread of cholera. Quick access to medical attention is crucial during a cholera outbreak. Oral rehydration should be accessible in communities in addition to larger treatment centers that have the capacity to provide IV fluids and round-the-clock medical attention. If timely and appropriate care is provided, the case fatality rate can remain around 1%. For children under five, zinc is a useful supplementary treatment because it shortens the duration of diarrhea and may prevent further episodes of severe watery diarrhea from unrelated sources.

A feverish illness with a high death rate is malaria. In a non-immune individual, symptoms usually appear 1015 days following the infectious mosquito bite. The initial symptoms of malaria, which include chills, fever, and headaches, can be vague and challenging to recognize. If P. falciparum malaria is not treated within 24 hours, it might

cause fatality and severe sickness. Children with severe malaria often exhibit indications such as cerebral malaria, respiratory failure due to metabolic acidosis, and extreme anemia. In adults, multi-organ failure is also typical. People who live in malaria-endemic regions may become partially immune, allowing infections to occur without symptoms.

In 2019, malaria threatened over 50% of the world's population. Sub-Saharan Africa accounts for the majority of malaria infections and deaths worldwide. The WHO has declared that the Americas, Western Pacific, South-east Asia, and the Eastern Mediterranean are all at danger. There are some persons who are far more likely than others to get malaria and become really ill. Among them are newborns, kids under five, expectant women, HIV/AIDS patients, non-immune refugees, nomadic populations, and tourists. Given their unique circumstances, national malaria control systems need to take extra precautions to prevent malaria infection in these susceptible groups [22].

Vector control is the main strategy for stopping and minimizing malarial transmission. If there is sufficient coverage of vector control actions within a certain geographic region, then a certain level of security would be granted to the entire population. Human immunity is also important, especially for adults who reside in moderately or severely infected areas. Years of exposure gradually develop partially protective immunity, which lowers the chance of serious illness from malaria infection even if it never offers complete protection. Because of this, children under five account for the majority of malaria deaths in Africa; nevertheless, all age groups are at risk in places with low immunity and low infection rates. The WHO advises using effective malaria vector control measures to protect everyone who is at risk of contracting malaria. Insecticide-treated mosquito nets and indoor residual spraying are two efficient vector control methods under a variety of conditions [17].

Nigeria accounted for the greatest number of deaths (24 percent of all malaria deaths worldwide) and the greatest number of cases worldwide (25 percent of all malaria cases) in 2018, according to the 2019 World Malaria Report. Between 2015 and 2018, the number of cases reached a plateau, ranging from 292 to 296 per 1000 of the population at risk. In the same period, however, deaths decreased by 21%, from 0.62 to 0.49 per 1000 at-risk population. Nigeria is a country where malaria is spread across; 24% of the population lives in low transmission areas and 76% of the population lives in high transmission areas. In the southern portion of the country, the transmission season might run for the entire year, but in the northern part, it lasts for three months or less [22].

[4] examine a cholera model $SVR-B$ with insufficient immunization. The analysis of the associated characteristic equations establishes the local stability of both the endemic and disease-free equilibriums. They compute the control reproduction number RV, which is a certain threshold. The diseases will be eradicated from the society if $RV < 1$, which provides sufficient conditions for the global asymptotic stability of the disease-free equilibrium. Through a comparative analysis of the arguments, it is demonstrated that in cases where $RV > 1$, the disease continues to spread and the globally asymptotically stable unique endemic equilibrium is achieved through the application of autonomous convergence theorems and second compound matrix techniques. An imperfect vaccination is always helpful in slowing the spread of disease within the community, as demonstrated by their sensitivity analysis of RV on the parameters to evaluate their relative contribution to disease transmission.

[11] investigates the dynamics of cholera transmission in settings with few resources, which are typical in underdeveloped nations. The model helped shed light on how the

health care system's resources affected the disease's ability to spread and be controlled. We developed and examined a deterministic model with a nonlinear recovery rate. The backward bifurcation phenomenon in the model was demonstrated through the application of centre manifold theory. The model study demonstrates that at the threshold $\mathcal{R}_0 < 1$, the disease-free steady state is locally stable. Additionally, the model's many equilibria and backward bifurcation phenomenon were demonstrated; these findings have consequences for the infection of cholera.

According to [12], a mathematical model built on a system of ordinary differential equations is developed to investigate the dynamics of cholera transmission in Kenya, using treatment through quarantine and health education campaigns as preventative measures. The next generation matrix approach is used to calculate the effective basic reproduction number. The model's equilibrium points were identified, and their stability was examined. The disease-free equilibrium is both locally and globally asymptotically stable when $\mathcal{R}_0 < 1$, according to stability analysis results, but the endemic equilibrium is both locally and globally asymptotically stable when $\mathcal{R}_0 > 1$. A numerical simulation shown that the number of cholera cases falls more quickly in instances where health education campaigns are effective, suggesting that these campaigns are essential for halting the disease's spread.

An $SEIQR$ (Susceptible-Educated-Infected-Quarantined-Recovered) type model was presented by [18], which considers the bacterial concentration in the dynamics of cholera spread. The treatment of populations under quarantine and intervention efforts as a tactic to stop the disease from spreading through better sanitation and education were the three controls that were taken into consideration to decrease the spread of cholera. In addition, the Pontryagin Minimum Principle approach was used to tackle the dynamics optimization problem. The goal was to reduce the numbers of bacteria and diseased humans while lowering the expenses associated with improving cleanliness, education, and quarantine conditions. Numerical findings are provided to demonstrate how well the three controls can stop the cholera virus from spreading.

In [19], the dynamics of malaria transmission with age structure for the vector population are described using a mathematical model of nonautonomous ordinary differential equations. Mosquito biting rate was thought to be a positive periodic function dependent on meteorological conditions. After the model's fundamental reproduction ratio was determined, it became clear that this parameter marked the boundary between the disease's extinction and persistence. Thus, they demonstrated that if the basic reproduction ratio is less than unity, the disease-free equilibrium is globally asymptotically stable; if it is greater than unity, at least one positive periodic solution exists. This was accomplished by applying the comparison theorem and the theory of uniform persistence. Lastly, numerical simulations were run to show the analytical outcomes.

[17] examined the dynamics of malaria disease transmission in both mosquito and human populations, taking into account the impacts of vertical transmissionthe transfer of malaria from mother to child before or during birthas well as the effects of malaria vaccination on the human population. The model separates the population of mosquitoes into two classes and the human population into three classes. The resulting nonlinear system takes into account the effect of the vaccination as well as the afflicted infant. The Routh-Hurwitz criterion was used to demonstrate that the endemic equilibrium is locally asymptotically stable if $\mathcal{R}_0 > 1$ and that the disease-free equilibrium is globally asymptotically stable if $\mathcal{R}_0 < 1$. The analysis predictions are supported by the numerical simulations and graphical outcomes.

In this study, we propose an optimal control of cholera-malaria with five control strategies that do not require permanent immunity for both illnesses, motivated by the reviewed literature. The following is the sectional presentation of the paper: Section 2 presents the formulation of the malaria-cholera model. In Section 3, the malaria-cholera model is mathematically analyzed. In Section 4, the optimal control issue and analysis are given. Section 5 contains the discussion and numerical results. At last, Section 6 served as our conclusion.

2. Formulation of malaria-cholera co-infection model

The human population has six compartments, i.e. susceptible humans S_H, infected humans with malaria I_H^V, infected humans with cholera I_H^C, infected humans with both malaria and cholera I_H^{VC}, recovered humans from malaria R_H^V, recovered humans from cholera R_H^C. The mosquito population compartment are susceptible vector S_V and infected vector I_V and while the vibro concentration in the environment as B. So that, the total human population,$N(t)$ at time t becomes

$$N(t) = S_H(t) + I_H^V(t) + I_H^C(t) + I_H^{VC}(t) + R_H^V(t) + R_H^C(t), \tag{2.1}$$

and vector total population is:

$$S_V(t) + I_V(t). \tag{2.2}$$

Λ_H is the recruitment rate into human population, μ represent the natural death rate, δ is the induced disease death rate and γ the recovery rate. The susceptible humans increase due to new born and loss of immunity from cholera and malaria infection at rates Λ_H, ω and ψ respectively. The susceptible population decreases by natural death that occurs in all classes and a terms $\frac{\varepsilon\beta_V I_V S_H}{N_H}$ and $\frac{\beta_C I_H^C S_H + \beta_B B S_H}{N_H}$, where ε, β_V, β_C and β_H are biting rate of mosquitoes. contacted rate of infected mosquitoes with infected humans, transmission rate of cholera from human to human and contact rate of infected human with susceptible mosquitoes respectively. So that the equation becomes

$$\frac{dS_H}{dt} = \Lambda_H + \psi R_H^V + \omega R_H^C - \frac{\varepsilon\beta_V I_V S_H}{N_H} - \frac{\beta_C I_H^C S_H + \beta_B B S_H}{N_H} - \mu_H S_H.$$

The population of infected humans with malaria at time t, $I_H^V(t)$, increases with the term $\frac{\varepsilon\beta_V I_V S_H}{N_H}$ and decreases by disease induced death rate δ_H, natural death rate μ_H, recovery rate from malaria due to treatment γ_H and a term $\frac{\beta_C I_H^C I_H^V S_H}{N_H}$.

$$\frac{dI_H^V}{dt} = \frac{\varepsilon\beta_V I_V S_H}{N_H} - \frac{\beta_C I_H^C I_H^V}{N_H} - (\delta_H + \mu_H + \gamma_H) I_H^V.$$

The population of infected humans with cholera at time t $I_H^C(t)$, increases with the term $\frac{\beta_C I_H^C S_H + \beta_B B S_H}{N_H}$, where β_B is the transmission rate of vibro in the environment and the population decreases with disease induced death rate δ_C, natural death rate μ_H, and recovery rate due to treatment, γ_C and contamination of the environment with bacterium at the rate ξ. The additional term that further decrease the population is $\frac{\varepsilon\beta_V I_V I_H^C}{N_H}$.

$$\frac{dI_H^C}{dt} = \frac{\beta_C I_H^C S_H + \beta_B B S_H}{N_H} - \frac{\varepsilon\beta_V I_V I_H^C}{N_H} - (\xi + \gamma_C + \mu_H + \delta_C) I_H^C.$$

The population of infected humans with malaria and cholera at time t, $I_H^{VC}(t)$, increases with the terms $\frac{\beta_C I_H^C I_H^V S_H}{N_H}$ and $\frac{\varepsilon\beta_V I_V I_H^C}{N_H}$. The population decreases with malaria induced death rate δ_H, cholera induced death rate δ_C, natural death rate μ_H, and recovery rate of humans with both malaria and cholera due to treatment, γ.

$$\frac{dI_H^{VC}}{dt} = \frac{\beta_C I_H^C I_H^V}{N_H} + \frac{\varepsilon\beta_V I_V I_H^C}{N_H} - (\delta_H + \delta_C + \mu_H + \gamma) I_H^{VC}.$$

The recovered malaria class increases with the terms $\gamma_H I_H^C, (1-\upsilon)\gamma I_H^{VC}$ and decreases due to loss of immunity against malaria and natural death at the rates ψ and μ_H. Thus, the equation is given as

$$\frac{dR_H^V}{dt} = \gamma_H I_H^V + (1-\upsilon)\gamma I_H^{VC} - (\psi + \mu_H) R_H^V.$$

The recovered cholera class increases with the terms $\gamma_C I_H^C, \upsilon\gamma I_H^{VC}$ and decreases due to loss of immunity against cholera and natural death at the rates ω and μ_H. Thus, the equation is given as

$$\frac{dR_H^C}{dt} = \gamma_C I_H^C + \upsilon\gamma I_H^{VC} - (\omega + \mu_H) R_H^C.$$

The susceptible mosquitoes class increases due to recruitment of mosquitoes at the rate Λ_V. The class decreases with the term $\frac{\varepsilon\beta_H S_V (I_H^V + I_H^{VC})}{N_V}$, death due to use of indoor residual spray and natural death at the rates θ_V and μ_V. Thus, the equation is given as

$$\frac{dS_V}{dt} = \Lambda_V - \frac{\varepsilon\beta_H S_V (I_H^V + I_H^{VC})}{N_V} - (\mu_V + \theta_V) S_V,$$

where N_V is the total vector population.
The infected mosquitoes class increases with the term $\frac{\varepsilon\beta_H S_V (I_H^V + I_H^{VC})}{N_V}$. The class decreases with the term $\frac{\varepsilon\beta_H S_V (I_H^V + I_H^{VC})}{N_V}$, death due to use of indoor residual spray and natural death at the rates θ_V and μ_V. Thus, the equation is given as

$$\frac{dI_V}{dt} = \frac{\varepsilon\beta_H S_V (I_H^V + I_H^{VC})}{N_V} - (\mu_V + \theta_V) I_V.$$

Finally, the bacteria class increases due to the shedding of bacteria from humans infected with cholera at a rate ξ. The class decreases natural death of bacteria and sanitation at the rates μ and δ_B. Thus, the equation is given as

$$\frac{dB}{dt} = \xi I_H^C - (\mu + \delta_B) B.$$

Therefore, the maleria-cholera co-infection model equations is:

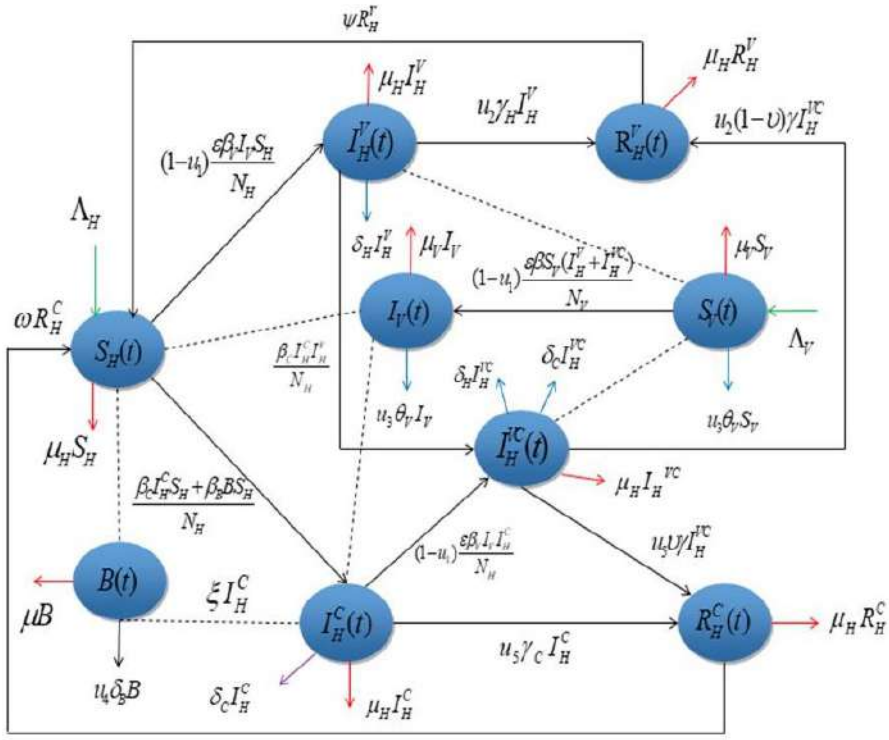


FIGURE 1. The schematic diagram for the co-infection model with constant controls.

$$
\begin{aligned}
\frac{dS_H}{dt} &= \Lambda_H + \psi R_H^V + \omega R_H^C - \frac{\varepsilon\beta_V I_V S_H}{N_H} - \frac{\beta_C I_H^C S_H + \beta_B B S_H}{N_H} - \mu_H S_H,\\
\frac{dI_H^V}{dt} &= \frac{\varepsilon\beta_V I_V S_H}{N_H} - \frac{\beta_C I_H^C I_H^V}{N_H} - (\delta_H + \mu_H + \gamma_H) I_H^V,\\
\frac{dI_H^C}{dt} &= \frac{\beta_C I_H^C S_H + \beta_B B S_H}{N_H} - \frac{\varepsilon\beta_V I_V I_H^C}{N_H} - (\xi + \gamma_C + \mu_H + \delta_C) I_H^C,\\
\frac{dI_H^{VC}}{dt} &= \frac{\beta_C I_H^C I_H^V}{N_H} + \frac{\varepsilon\beta_V I_V I_H^C}{N_H} - (\delta_H + \delta_C + \mu_H + \gamma) I_H^{VC},\\
\frac{dR_H^V}{dt} &= \gamma_H I_H^V + (1-\upsilon)\gamma I_H^{VC} - (\psi + \mu_H) R_H^V\\
\frac{dR_H^C}{dt} &= \gamma_C I_H^C + \upsilon\gamma I_H^{VC} - (\omega + \mu_H) R_H^C,\\
\frac{dS_V}{dt} &= \Lambda_V - \frac{\varepsilon\beta_H S_V (I_H^V + I_H^{VC})}{N_V} - (\mu_V + \theta_V) S_V,\\
\frac{dI_V}{dt} &= \frac{\varepsilon\beta_H S_V (I_H^V + I_H^{VC})}{N_V} - (\mu_V + \theta_V) I_V,\\
\frac{dB}{dt} &= \xi I_H^C - (\mu + \delta_B) B,
\end{aligned}
\tag{2.3}
$$

with the initial conditions:

$$\begin{cases} S_H(0) = S_H(0) \geq 0, I_H^V(0) = I_H^V \geq 0, \\ I_H^C(0) = I_H^C(0) \geq 0, I_H^{VC}(0) = I_H^{VC}(0) \geq 0, \\ R_H^V(0) = R_H^V(0) \geq 0 R_H^C(0) = R_H^C \geq 0, \\ S_V(0) = S_V(0) \geq 0, I_V(0) = I_V(0) \geq 0, \\ B(0) = B(0) \geq 0. \end{cases} \tag{2.4}$$

Table 1. Malaria-cholera co-infection model variables.

Variable	Description
$S_H(t)$	Number of susceptible humans at time t
$I_H^V(t)$	Number of infected humans with malaria at time t
$I_H^C(t)$	Number of infected humans with cholera at time t
$I_H^{VC}(t)$	Number of malaria cholera co-infection at time t
$R_H^V(t)$	Number of recovered humans from a malaria infection at time t
$R_H^C(t)$	Number of recovered humans from cholera infection at time t
$S_V(t)$	Number of susceptible mosquitoes at time t
$I_V(t)$	Number of infected mosquitoes at time t
$B(t)$	Total number of cholera bacteria in the environment at time t

TABLE 2. Maleria-cholera co-infection model parameters notation and values.

Parameters	Description	Values	Source
Λ_H	Recruitment rate human	100	[13]
Λ_V	Recruitment rate mosquitoes	1000	[13]
μ_H	Natural death rate of human	0.2	Asummed
μ_V	Natural death rate of mosquitoes	0.1429	[13]
ε	Biting rate of mosquitoes	0.2	[13]
β_V	The contact rate of infected mosquitoes with a susceptible humans	0.502	[13]
β_H	The contact rate of infected humans with a susceptible mosquitoes	0.833	[13]
δ_H	The disease induced death rate for humans infected with malaria	0.05	[14]
ω	Loss of immunity against cholera	0.62	Assumed
ψ	Loss of immunity against malaria	0.7902	[13]
γ_H	The rate of recovery for infected human with malaria	0.005	[13]
θ_V	The death rate of mosquitoes population with indoor residual spraying	0.25	[13]
δ_C	Disease induced death rate of humans infected with cholera in co-infected class	0.2407	[13]
δ_H	Disease induced death rate of humans infected with malaria in co-infected class	0.05	[14]
γ	Recovery rate of human with both infections in co-infected class	0.35	[14]
δ_B	The bacteria death rate due to sanitation	0.5	[15]
μ	Natural death rate of the bacteria	2	Assumed
ξ	Shedding rate of bacteria from humans infected with cholera to the environment	10	Assumed
β_C	Transmission rate from human to human infected with cholera	0.0011	Assumed
β_B	Transmission rate of vibro in the environment	0.5	Assumed
υ	Proportion of humans who recovered from cholera from co-infection class	0.5	[18]
A_1	Weight factor for infected malaria	100	Assumed
A_2	Weight factor for infected cholera	90	Assumed
A_3	Weight factor for both malaria and cholera co-infection	70	Assumed
A_4	Weight factor for mosquitoes population	50	Assumed
A_5	Weight factor for population of vibrio in the environment	20	Assumed
D_1	Weight factor for control u_1	20	[1]
D_2	Weight factor for control u_2	65	[1]
D_3	Weight factor for control u_3	10	[1]
D_4	Weight factor for control u_4	30	Assumed
D_5	Weight factor for control u_5	15	Assumed

3. Analysis of the co-infection model

3.1. Basic properties of the model

Theorem 3.1. *Let the initial solution set* $S_H(0), I_H^V(0), I_H^C(0), I_H^{VC}(0), R_H^V(0), R_H^C(0), S_V(0), I_V(0), B(0) > 0$ *be non-negative initial conditions, then system* (2.3) *has a non-negative solution* $S_H(t), I_H^V(t), I_H^C(t), I_H^{VC}(t), R_H^V(t), R_H^C(t), S_V(t), I_V(t), B(t) > 0$ *for all* $t > 0$. *Moreover,* $\limsup_{t\to\infty} N_H(t) \leq \dfrac{\Lambda_H}{\mu_H}$, $\limsup_{t\to\infty} N_V(t) \leq \dfrac{\Lambda_V}{\mu_V + \theta_V}$ *and* $\limsup_{t\to\infty} N_B(t) \leq \dfrac{\xi}{\mu + \delta_B}$. *In addition, if* $N_H(0) \leq \frac{\Lambda_H}{\mu_H}$, *then* $N_H(t) \leq \frac{\Lambda_H}{\mu_H}$, $N_V(0) \leq \frac{\Lambda_V}{\mu_V+\theta_V}$, *then* $N_V(t) \leq \frac{\Lambda_V}{\mu_V+\theta_V}$ *and* $N_B(0) \leq \frac{\xi}{\mu+\delta_B}$, *then* $N_B(t) \leq \frac{\xi}{\{\mu+\delta_B}$. *Therefore, the feasible region of system* (2.3) *given as:*

$$\Omega = \Omega_H \times \Omega_V \times \Omega_B \in \mathbb{R}_+^6 \times \mathbb{R}_+^2 \times \mathbb{R}_+, \tag{3.1}$$

where

$$\Omega_H = \left\{ (S_H, I_H^V, I_H^C, I_H^{VC}, R_H^V, R_H^C) \in \mathbb{R}_+^6 : N_H \leq \frac{\Lambda_H}{\mu_H} \right\}, \tag{3.2}$$

$$\Omega_V = \left\{ (S_V, I_V) \in \mathbb{R}_+^2 : N_V \leq \frac{\Lambda_V}{\mu_V + \theta_V} \right\}, \tag{3.3}$$

$$\Omega_B = \left\{ B \in \mathbb{R}_+ : N_B \leq \frac{\xi}{\mu + \delta_B} \right\}, \tag{3.4}$$

is positively invariant and attracting.

Proof. From the first equation of system (2.3), we have the following

$$\frac{dS_H}{dt} + \frac{\varepsilon\beta_V I_V S_H}{N_H} + \frac{\beta_C I_H^C S_H + \beta_B B S_H}{N_H} + \mu_H S_H \geq 0. \tag{3.5}$$

From time $t = 0$ to $t = t$, we integrated (3.5) to get

$$\frac{d}{dt}\left[S_H(t) \exp\left\{ \int_0^t \frac{\varepsilon\beta_V I_V}{N_H} + \frac{\beta_C I_H^C S_H + \beta_B B}{N_H} + \mu_H \right\} \right] \geq 0.$$

This means that

$$S_H(t) \geq S_H(0) \exp\left\{ -\left(\int_0^t \frac{\varepsilon\beta_V I_V}{N_H} + \frac{\beta_C I_H^C + \beta_B B}{N_H} + \mu_H \right) \right\} > 0, \forall t > 0.$$

We applied similar method to establish that $I_H^V(t), I_H^C(t), I_H^{VC}(t), R_H^V(t), R_H^C(t), S_V(t), I_V(t), B(t) > 0$ remain non-negative for all $t > 0$. In other to prove the second part of Theorem (3.1), the first six equations of system (2.3) were added to get

$$\frac{dN_H}{dt} = \Lambda_H - \mu_H N_H.$$

This implies that $N_H(t) = N_H(0)\exp^{-\mu_H t} + \frac{\Lambda_H}{\mu_H}(1 - \exp^{-\mu_H t})$.

Thus, $\limsup_{t\to\infty} N_H(t) \leq \dfrac{\Lambda_H}{\mu_H}$. Similarly, proving for the vector and bacteria populations, we have that if $N_V(t) = N_V(0)\exp^{-(\mu_V+\theta_V)t} + \frac{\Lambda_V}{\mu_V+\theta_V}(1 - \exp^{(-\mu_V+\theta_V)t}$, then $\limsup_{t\to\infty} N_V(t) \leq \dfrac{\Lambda_V}{(\mu_V+\theta_V}$ and $\limsup_{t\to\infty} N_B(t) \leq \dfrac{\xi}{(\mu+\delta_B)}$. Thus, proves the boundedness of the solutions inside Ω. Hence, the solutions to system (2.3) are positively invariant and attracting in a region Ω. We notice that system (2.3) is feasible biologically and mathematically well posed in Ω. ∎

3.2. The cholera model

In order to analyse the cholera model, we ignore the malaria and co-infection compartments in (2.3) to get

$$\begin{cases} \dfrac{dS_H}{dt} = \Lambda_H + \omega R_H^C - \left(\dfrac{\beta_C I_H^C S_H + \beta_B B S_H}{N_H}\right) - \mu_H S_H, \\ \dfrac{dI_H^C}{dt} = \dfrac{\beta_C I_H^C S_H + \beta_B B S_H}{N_H} - (\xi + \gamma_C + \mu_H + \delta_C) I_H^C, \\ \dfrac{dR_H^C}{dt} = \gamma_C I_H^C - (\omega + \mu_H) R_H^C, \\ \dfrac{dB}{dt} = \xi I_H^C - (\mu + \delta_B) B. \end{cases} \tag{3.6}$$

3.3. The cholera free equilibrium point

To obtain the the cholera free equilibrium point, we set the right-hand side of equation (3.6) to zero to get

$$\begin{cases} \Lambda_H + \omega R_H^C - \left(\frac{\beta_C I_H^C S_H + \beta_B B S_H}{N_H}\right) - \mu_H S_H = 0, \\ \frac{\beta_C I_H^C S_H + \beta_B B S_H}{N_H} - (\xi + \gamma_C + \mu_H + \delta_C) I_H^C = 0, \\ \gamma_C I_H^C - (\omega + \mu_H) R_H^C = 0, \\ \xi I_H^C - (\mu + \delta_B) B = 0. \end{cases} \tag{3.7}$$

In the absence of the cholera in the population, we set $I_H^C = R_H^C = B = 0$. Therefore, the cholera free equilibrium is given by $E_0^C = (S_H^0, I_H^{C0}, R_H^{C0}, B^0) = \left(\frac{\Lambda_H}{\mu_H}, 0, 0, 0\right)$.

3.4. The cholera basic reproduction number

The basic reproduction number is an important non-dimensional quantity in epidemiology as it set the threshold in the study of a disease both for predicting its outbreak and for evaluating its control strategies. When $\mathcal{R}_0 < 1$, each infected individual produces on average less than one infected individual, so it is expected that the disease dies out. On the other hand, if $\mathcal{R}_0 > 1$ then each individual produces more than one Cholera new infected individual so it is expected that the disease would spread in the population. To find the basic reproduction number of equation (3.6), we followed the method in [5] to get

$$F = \begin{pmatrix} \frac{\beta_C I_H^C S_H + \beta_B B S_H}{N_H} \\ 0 \end{pmatrix},$$

and

$$V = \begin{pmatrix} (\xi + \gamma_C + \mu_H + \delta_C) I_H^C & 0 \\ -\xi I_H^C & (\mu + \delta_B) B \end{pmatrix}.$$

Thus, the cholera model reproduction number is:

$$R_0^C = \rho(FV^{-1}) = R_H^1 + R_B, \tag{3.8}$$

where

$$R_H^1 = \frac{\beta_C}{(\xi + \gamma_C + \mu_H + \delta_C)},$$

and

$$R_B = \frac{\xi \beta_B}{(\xi + \gamma_C + \mu_H + \delta_C)(\mu + \delta_B)}.$$

R_H^1 is basic reproduction number generated by cholera infectious humans and R_B is the basic reproductive number generated by the cholera vibro in environment.

3.5. Local stability analysis of cholera free equilibrium

Theorem 3.2. *The cholera free equilibrium of system (3.6) is locally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if otherwise.*

Proof. The Jacobian matrix of the system (3.6) at cholera free equilibrium $J(E^0)$ is be given by

$$J(E^0) = \begin{pmatrix} -\mu_H & -\beta_C & \omega & -\beta_B \\ 0 & \beta_C - k_1 & 0 & \beta_B \\ 0 & \gamma_C & -k_2 & 0 \\ 0 & \xi & 0 & -k_3 \end{pmatrix},$$

where
$k_1 = (\xi + \gamma_C + \mu_H + \delta_C)$, $k_2 = (\omega + \mu_H)$, $k_3 = (\mu + \delta_B)$. From matrix $J(E^0)$, we see that the first two eigenvalues are $\lambda_1 = -\mu_H < 0$ and $\lambda_2 = -(\omega + \mu_H) < 0$. Thus, matrix $J(E^0)$ reduces to

$$\begin{pmatrix} \beta_C - k_1 - \lambda & \beta_B \\ \xi & -k_3 - \lambda \end{pmatrix}. \tag{3.9}$$

to obtain the remaining two eigenvalues. The characteristics polynomial of equation (3.9) is given as:

$$A_0 \lambda^2 + A_1 \lambda + A_2 = 0, \lambda = \lambda_i, \ i = 3, 4 \tag{3.10}$$

where

$A_0 = 1,$

$A_1 = (k_1 + k_3 - \beta_C),$

$A_2 = (1 - R_0^C).$

According to Routh-Hurwitz criterion, all roots of the polynomial equation (3.10) have negative real parts if and only if the coefficients A_i for $i = 0, 1, 2$ are positive and the determinant of the matrices $H_i > 0$ for $i = 0, 1, 2$. It is clear that $H_1 > 0$. Therefore, all the eigen values of the polynomial (3.10) have negative real parts, implying that $\lambda_3 < 0, \lambda_4 < 0$. Since all the values of $\lambda_1 < 0$ for $i = 0, 1, 2, 3, 4$, when $\mathcal{R}_0^C < 1$ and $\frac{\beta_C}{k_1+k_3} < 1$, we conclude that the cholera-free equilibrium point is locally asymptotically stable (LAS). ∎

3.6. The malaria model

Similarly, in order to analyse the malaria model, we ignore the cholera and co-infection compartments in (2.3) to get

$$\begin{cases} \dfrac{dS_H}{dt} = \Lambda_H - \varphi R_H^V - \dfrac{\varepsilon\beta_V I_V S_H}{N_H} - \mu_H S_H, \\ \dfrac{dI_H^V}{dt} = \dfrac{\varepsilon\beta_V I_V S_H}{N_H} - (\delta_H + \mu_H + \gamma_H) I_H^V, \\ \dfrac{dR_H^V}{dt} = \gamma_V I_H^V - (\varphi + \mu_H) R_H^V, \\ \dfrac{dS_V}{dt} = \Lambda_V - \dfrac{\varepsilon\beta_H S_V I_H^V}{N_V} - (\mu_V + \theta_V) S_V, \\ \dfrac{dI_V}{dt} = \dfrac{\varepsilon\beta_H S_V I_H^V}{N_V} - (\mu_V + \theta_V) I_V. \end{cases} \tag{3.11}$$

3.7. The malaria free equilibrium point

The right-hand side of equation (3.11) is set to zero to obtain the malaria free equilibrium point. Thus, we have

$$\begin{cases} \Lambda_H + \varphi R_H^V - \frac{\varepsilon\beta_V I_V S_H}{N_H} - \mu_H S_H = 0, \\ \frac{\varepsilon\beta_V I_V S_H}{N_H} - (\delta_H + \mu_H + \gamma_H) I_H^V = 0, \\ \gamma_V I_H^V - (\varphi + \mu_H) R_H^V = 0, \\ \Lambda_V - \frac{\varepsilon\beta_H S_V I_H^V}{N_V} - (\mu_V + \theta_V) S_V = 0, \\ \frac{\varepsilon\beta_H S_V I_H^V}{N_V} - (\mu_V + \theta_V) I_V = 0. \end{cases} \tag{3.12}$$

Considering that there is no malaria in the population, we set $I_H^V = R_H^V = I_V = 0$. Therefore, the malaria free equilibrium is given as: $E_0^V = (S_H^0, I_H^{V0}, R_H^{V0}, S_V^0, I_V^0) = \left(\frac{\Lambda_H}{\mu_H}, 0, 0, \frac{\Lambda_V}{(\mu_V+\theta_V)}, 0\right)$.

3.8. Malaria model basic reproduction number

The system equation (3.11) was used to derive the basic reproduction number. The method adopted is [5] where the new infection term is

$$F = \begin{pmatrix} \frac{\varepsilon\beta_V S_H}{N_H} \\ \frac{\varepsilon\beta_H I_H}{N_V} \end{pmatrix},$$

and transfer term is

$$V = \begin{pmatrix} (\delta_H + \mu_H + \gamma_H) I_H^V \\ (\mu_V + \theta_V) I_V \end{pmatrix}.$$

Thus, the reproduction number of the malaria model (3.11) denoted by $R_0^V = \rho(FV^{-1})$ is given as

$$R_0^V = \rho(FV^{-1}) = \sqrt{\frac{\mu_V \varepsilon^2 \beta_H \beta_V}{(\mu_V + \theta_V)^2(\delta_H + \mu_H + \gamma_H)}}. \tag{3.13}$$

3.9. Local stability analysis of malaria free equilibrium

Theorem 3.3. *The malaria free equilibrium of system (3.11) is locally asymptotically stable (LAS) if $\mathcal{R}_0^V < 1$ and unstable $\mathcal{R}_0^V > 1$.*

Proof. The Jacobian matrix of the system (3.11) at malaria free equilibrium $J(E_0^V)$ is

$$J(E_0^V) = \begin{pmatrix} -\mu_H & 0 & \varphi & 0 & -\varepsilon\beta_V \\ 0 & -(\delta_H + \mu_H + \gamma_H) & 0 & 0 & \varepsilon\beta_V \\ 0 & \gamma_V & -(\varphi + \mu_H) & 0 & 0 \\ 0 & \frac{-\varepsilon\beta_H\mu_V}{(\mu_V+\theta_V)} & 0 & -(\mu_V + \theta_V) & 0 \\ 0 & \frac{\varepsilon\beta_H\mu_V}{(\mu_V+\theta_V)} & 0 & 0 & -(\mu_V + \theta_V) \end{pmatrix}.$$

We see that the first three eigenvalues are
$\lambda_1 = -\mu_H,\ \lambda_2 = -(\varphi+\mu_H),\ \lambda_3 = -(\mu_V+\theta_V)$. To obtain the remaining two eigenvalues, from matrix $J(E_0^V)$, we have the reduced matrix as

$$\begin{pmatrix} -M_1 & \varepsilon\beta_V \\ \frac{\varepsilon\beta_V\mu_V}{(\mu_V+\theta_V)} & -M_2 \end{pmatrix}, \tag{3.14}$$

where $M_1 = (\delta_H + \mu_H + \gamma_H)$ and $M_2 = (\mu_V + \theta_V)$. The characteristics polynomial (3.14) is given as follows:

$$B_0\lambda^2 + B_1\lambda + B_2 = 0,\ \lambda = \lambda_i,\ i = 4, 5, \tag{3.15}$$

and the coefficients of (3.15) are given as
$B_0 = 1$,

$B_1 = (M_2 + M_5)$,

$B_2 = (1 - R_0^{V^2})$.

According to Routh-Hurwitz criterion again, all roots of the polynomial equation (3.15) have a negative real part if and only if the coefficients B_i are positive and the determinant of the matrices $H_i > 0$ for $i = 0, 1, 2$. Therefore, the remaining two eigenvalues have negative real parts since $B_i > 0,\ i = 0, 1, 2$ and $H_i > 0, i = 0, 1, 2$. implying that $\lambda_4 < 0, \lambda_5 < 0$. Since all the values of $\lambda_1 < 0$ for $i = 1, 2, 3, 4, 5$, when $\mathcal{R}_0^V < 1$, we conclude that the malaria-free equilibrium point is locally asymptotically stable (LAS). ∎

3.10. Analysis of the malaria-chlolera co-infection model

3.11. Equilibrium points

The system (2.3) equilibrium points are established by setting the system to zero. Thus, we have

$$\begin{cases} \dfrac{dS_H}{dt} = \Lambda_H + \psi R_H^V + \omega R_H^C - \dfrac{\varepsilon\beta_V I_V S_H}{N_H} - \dfrac{\beta_C I_H^C S_H + \beta_B B S_H}{N_H} - \mu_H S_H = 0, \\ \dfrac{dI_H^V}{dt} = \dfrac{\varepsilon\beta_V I_V S_H}{N_H} - \dfrac{\beta_C I_H^C I_H^V}{N_H} - (\delta_H + \mu_H + \gamma_H) I_H^V = 0, \\ \dfrac{dI_H^C}{dt} = \dfrac{\beta_C I_H^C S_H + \beta_B B S_H}{N_H} - \dfrac{\varepsilon\beta_V I_V I_H^C}{N_H} - (\xi + \gamma_C + \mu_H + \delta_C) I_H^C = 0, \\ \dfrac{dI_H^{VC}}{dt} = \dfrac{\beta_C I_H^C I_H^V}{N_H} + \dfrac{\varepsilon\beta_V I_V I_H^C}{N_H} - (\delta_H + \delta_C + \mu_H + \gamma) I_H^{VC} = 0, \\ \dfrac{dR_H^V}{dt} = \gamma_H I_H^C + (1-\upsilon)\gamma I_H^{VC} - (\psi + \mu_H) R_H^V = 0, \\ \dfrac{dR_H^C}{dt} = \gamma_C I_H^C + \upsilon\gamma I_H^{VC} - (\omega + \mu_H) R_H^C = 0, \\ \dfrac{dS_V}{dt} = \Lambda_V - \dfrac{\varepsilon\beta_H S_V (I_H^V + I_H^{VC})}{N_V} - (\mu_V + \theta_V) S_V = 0, \\ \dfrac{dI_V}{dt} = \dfrac{\varepsilon\beta_H S_V (I_H^V + I_H^{VC})}{N_V} - (\mu_V + \theta_V) I_V = 0, \\ \dfrac{dB}{dt} = \xi I_H^C - (\mu + \delta_B) B = 0. \end{cases} \tag{3.16}$$

Solving equation (3.16) give the disease free equilibrium as:

$$(S_H^0, I_H^{V0}, I_H^{C0}, I_H^{CV0}, R_H^{V0}, R_H^{C0}, S_V^0, I_V^0, B^0) = \left(\frac{\Lambda_H}{\mu_H}, 0, 0, 0, 0, 0, \frac{\Lambda_H}{(\mu_V + \theta_V)}, 0, 0\right), \tag{3.17}$$

and the disease presence equilibrium as:

$$\begin{cases} S_H^{**} = \dfrac{(\Lambda_H + \omega R_H^{C*} + \psi R_H^{V*})N_H^*}{B^*\beta_B + \mu_H N_H^* + \beta_C I_H^{C*} + \varepsilon\beta_V I_V^*}, \\ I_H^{V**} = \dfrac{\varepsilon\beta_V I_V^* S_V^*}{(\gamma_H + \mu_H + \delta_H) + N_H^* + \beta_C I_H^{C*}}, \\ I_H^{C**} = \dfrac{B^*\beta_C S_H^{C*}}{(\xi + \gamma_C + \mu_C + \delta_H)N_H^* - \beta_C S_H^* + \varepsilon\beta_V}, \\ I_H^{VC**} = \dfrac{I_H^{C*}(\beta_C I_H^{V*} + \varepsilon\beta_V I_V^*)}{(\gamma + \delta_C + \mu_H + \delta_H)N_H^*}, \\ R_H^{V**} = \dfrac{\gamma_H I_H^{V*} + (1-\upsilon)\gamma I_H^{VC*}}{(\psi + \mu_H}, \\ R_H^{C**} = \dfrac{\gamma_C I_H^{C*} + \upsilon\gamma I_H^{VC*}}{(\psi + \mu_H)}, \\ S_V^{**} = \dfrac{N_V^* A\Lambda_V - \varepsilon\beta_H S_V^*(I_H^{V*} + I_H^{VC*})}{N_V(\mu_V + \theta_V)}, \\ I_V^{**} = \dfrac{\varepsilon\beta_H S_V^*(I_H^{V*} + I_H^{VC*})}{(\mu_V + \theta_V)N_V^*}, \\ B^{**} = \dfrac{\xi I_H^{C*}}{(\mu_V + \delta_B)}. \end{cases} \tag{3.18}$$

3.12. Cholera-malaria model basic reproduction number

The techniques in [20] was adopted to obtain the basic reproduction number for the cholera-malaria model. Thus, the basic reproduction number for the co-infection model (3.16) is

$$\mathcal{R}_0^{CV} = \max(\mathcal{R}_0^C, \mathcal{R}_0^C). \tag{3.19}$$

where

$$\mathcal{R}_0^C = \frac{\beta_C}{(\xi + \gamma_C + \mu_H + \delta_C)} + \frac{\xi\beta_B}{(\xi + \gamma_C + \mu_H + \delta_C)(\mu + \delta_C)},$$

$$\mathcal{R}_0^V = \sqrt{\frac{\mu_V\varepsilon^2\beta_H\beta_V}{(\mu_V + \theta_V)^2(\delta_H + \mu_H + \gamma_H)}}.$$

$\mathcal{R}_0^C$ is basic reproduction number generated by cholera model and $\mathcal{R}_0^V$ is the basic reproductive number generated by the malaria model.

3.13. Global stability of the cholera and malaria co-infection free equilibrium

The approach in [5] is adopted to investigate the global asymptotic stability (GAS) of the cholera and malaria co-infection free equilibrium for the model (2.3).The model is written as follows

Lemma 3.4. *The cholera and malaria free equilibrium is globally asymptotically stable if $\mathcal{R}_0^{CV} < 1$ and unstable otherwise. Let system (2.3) be in the form*

$$\begin{cases} \dfrac{dX}{dt} = H(X,Z), \\ \dfrac{dZ}{dt} = G(X,Z,), (X,0) = 0, \end{cases} \tag{3.20}$$

where $X = (S_H, R_H^C, R_H^V, S_V)$ and $Z = (I_H^V, I_H^C, I_H^{VC}, I_V, B)$ and components of $X \in \mathbb{R}^4$ represent the population that are not infected and components of $Z \in \mathbb{R}^5$ represent the population that are infected. Considering the malaria cholera co-infection free equilibrium $E_0^{CV} = (X^*, 0)$, where

$$X^0 = \left(\frac{\Lambda_H}{\mu_H}, 0, 0, \frac{\Lambda_V}{(\mu_V + \theta_V}\right) \tag{3.21}$$

The conditions that must be met to guarantee a global asymptotic stability are:
$H_1 : \frac{dX}{dt} = H(X,Z)$, X^* is (GAS).
$H_2 : G(X,Z) = CZ - \hat{G}(X,Z), \hat{G}(X,Z) \geq 0$ for $(X,Z) \in \Omega_H$, where $C = D_z G(X^*, 0)$ is an M-matrix and Ω_H is the biological feasible region. Hence, E_0^{CV} is (GAS) if $\mathcal{R}_0^{CV} < 1$.

Theorem 3.5. *The malaria and cholera free equilibrium of system (2.3) is (GAS) if $\mathcal{R}_0^{CV} < 1$ and unstable if otherwise.*

Proof. We have to establish that the conditions (H_1) and (H_2) hold when $\mathcal{R}_0^{CV} < 1$. For the uninfected population we have

$$F(X,0) = \begin{pmatrix} \Lambda_H - \mu_H S_H \\ 0 \\ 0 \\ \Lambda_V - (\mu_V + \theta_V) S_V \end{pmatrix}, \tag{3.22}$$

and $Z \in \mathbb{R}^5$ denotes the infected compartments in the model (2.3), we have

$$G(X,Z) = CZ - \hat{G}(X,Z),$$

$$C = \begin{pmatrix} -(\delta_H + \mu_H + \gamma_H) & 0 & 0 & \varepsilon\beta_V & 0 \\ 0 & -(\xi + \gamma_C + \mu_H + \delta_C) + \beta_C & 0 & 0 & \beta_B \\ 0 & 0 & -(\delta_H + \delta_C + \mu_H + \gamma) & 0 & 0 \\ 0 & \frac{\varepsilon\beta_H^0 S_V^0}{N_V 0} & \frac{\varepsilon\beta_H^0 S_V^0}{N_V 0} & -(\mu_V + \theta_V) & 0 \\ 0 & \xi & 0 & 0 & -(\mu + \delta_B) \end{pmatrix}.$$

Thus,

$$\hat{G}(X,Z) = \begin{pmatrix} \varepsilon\beta_V I_V (1 - \frac{S_H}{N_H}) + \beta_C I_H^C I_H^V \\ \beta_C I_H^C (1 - \frac{S_H}{N_H}) + \beta_B B (1 - \frac{S_H}{N_H}) + \varepsilon\beta_V I_V I_H^C \\ -(\frac{\beta_C I_H^C I_H^V}{N_H} + \frac{\varepsilon\beta_V I_V I_H^C}{N_H}) \\ \varepsilon\beta_H I_H^V (\frac{S_V^0}{N_V^0} - \frac{S_V}{N_V}) + \varepsilon\beta_H I_H^{VC} (\frac{S_V^0}{N_V^0} - \frac{S_V}{N_V}) \\ 0 \end{pmatrix}. \tag{3.23}$$

Since $S_H < N_H$, $\frac{S_V^0}{N_V^0} \geq \frac{S_V}{N_V}$, it implies that $\hat{G}_1(X,Z) \geq 0, \hat{G}_2(X,Z) \geq 0, \hat{G}_4(X,Z) \geq 0, \hat{G}_5(X,Z) = 0$, but $\hat{G}_3(X,Z) < 0$ at the disease free equilibrium, $X^* = \left(\frac{\Lambda_H}{\mu_H}, 0, 0, \frac{\Lambda_V}{(\mu_V + \theta_V)}\right)$ the cholera and malaria co-infection model may not be globally asymptotically stable. This completes the proof. ∎

3.14. Sensitivity analysis of the malaria cholera co-infection model

In this sub-section, we present the sensitivity analysis for the cholera-malaria co-infection model. To carry out the analysis, we followed the technique outlined by [3] and [2]. This technique develops a formula to obtain the sensitivity index of all the basic parameters, defined as: $\Delta_x^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial x} \times \frac{\partial x}{\partial \mathcal{R}_0}$, where x represents all the basic parameters. Results of sensitivity analysis is presented in Table 3.

Table 3. Sensitivity indices for co-infection reproduction number $\mathcal{R}_0^{CV}$.

Parameter	Baselines Value	Sensitivity Index (R_0^{CV})
β_C	0.00011	0.00055
β_B	0.5	0.99995
μ	2	-0.79970
ξ	10	0.02377
μ_H	0.02	-0.00195
δ_C	0.01407	-0.00235
δ_B	0.5	-0.19999
ε	0.2	1.00000
β_H	0.8333	0.50000
β_V	0.502	0.50000
δ_H	0.05	-0.33330
γ_H	0.005	0.03333
μ_V	0.1429	0.13629
θ_V	0.25	-0.63629

The sensitivity indices carried out on the basic reproduction number with respect to main parameters are arranged orderly in Table 3. Those parameters that have positive indices shows that they have great impact in expanding the disease in the community if their values are increased. In contrast, those parameters with negative sensitivity indices have an influence of minimizing the burden of malaria-cholera in the community if their values are increased.

4. Optimal control of the malaria cholera co-infection model

Based on the sensitivity analysis results in sub-section (3.14), which shows the impacts of some parameters on the reproduction number, we developed the optimal control model. We incorporated control interventions into the system (2.3) to get

$$\begin{cases} \frac{dS_H}{dt} = \Lambda_H + \psi R_H^V + \omega R_H^C - (1-u_1)\frac{\varepsilon\beta_V I_V S_H}{N_H} - \frac{\beta_C I_H^C S_H + \beta_B B S_H}{N_H} - \mu_H S_H, \\ \frac{dI_H^V}{dt} = (1-u_1)\frac{\varepsilon\beta_V I_V S_H}{N_H} - \frac{\beta_C I_H^C I_H^V}{N_H} - (\delta_H + \mu_H + u_2\gamma_H) I_H^V, \\ \frac{dI_H^C}{dt} = \frac{\beta_C I_H^C S_H + \beta_B B S_H}{N_H} - (1-u_1)\frac{\varepsilon\beta_V I_V I_H^C}{N_H} - (\xi + u_5\gamma_C + \mu_H + \delta_H) I_H^C, \\ \frac{dI_H^{VC}}{dt} = \frac{\beta_C I_H^C I_H^V}{N_H} + (1-u_1)\frac{\varepsilon\beta_V I_V I_H^C}{N_H} - (\delta_H + \delta_C + \mu_H + u_5\upsilon\gamma + u_2(1-\upsilon)) I_H^{VC}, \\ \frac{dR_H^V}{dt} = u_2\gamma_H I_H^V + u_2(1-\upsilon)\gamma I_H^{VC} - (\psi + \mu_H) R_H^V, \\ \frac{dR_H^C}{dt} = u_5\gamma_C I_H^C + u_5\upsilon\gamma I_H^{VC} - (\omega + \mu_H) R_H^C, \\ \frac{dS_V}{dt} = \Lambda_V - (1-u_1)\frac{\varepsilon\beta_H S_V (I_H^V + I_H^{VC})}{N_V} - (\mu_V + u_3\theta) S_V, \\ \frac{dI_V}{dt} = (1-u_1)\frac{\varepsilon\beta_H S_V (I_H^V + I_H^{VC})}{N_V} - (\mu_V + u_3\theta) I_V, \\ \frac{dB}{dt} = \xi I_H^C - (\mu + u_4\delta_B) B. \end{cases} \tag{4.1}$$

We present the objective functional for the model with five controls with the aim of controlling the transmission of malaria-cholera co-infection namely: the use of treated mosquito net, treatment for both diseases, indoor residual spraying and sanitation. The optimal control problem is developed in such way to minimize the number of infected individuals, total number of bacteria and mosquitoes while minimizing cost of implementing the control measures. The objective functional is developed in line with the approach discussed in [[8],[9],[10]] and it is given as

$$\begin{aligned} J(u) &= \int_0^{t_f} [(A_1 I_H^V(t) + A_2 I_H^C(t) + A_3 I_H^V C(t) + A_4 N_V + A_5 B + \frac{1}{2}(B_1 u_1^2 + B_2 u_2^2 \\ &+ B_3 u_3^2 + B_4 u_4^2 + B_5 u_5^2)]dt, \end{aligned} \tag{4.2}$$

given the objective functional equation (4.2) where t_f is the final time and the coefficients $A_1, A_2, A_3, A_4, A_5, B_1, B_2, B_3, B_4$ and B_5 are positive weight to balance the factors. The aim is to reduce the number of infected humans I_H^V, I_H^C, I_H^{VC}, the total population of the mosquitoes N_V and total population of vibrio in the environment while minimizing the cost of implementing the controls. We seek to optimize the controls $u_1^*, u_2^*, u_3^*, u_4^*$ and u_5^* such that

$$J(u_1^*, u_2^*, u_3^*, u_4^*, u_5^*) = \min_{u_1,u_2,u_3,u_4,u_5} \{J(u_1, u_2, u_3, u_4, u_5) \ni u_1, u_2, u_3, u_4, u_5 \in U\},$$

where U is the set of measurable functions defined from $[0, t_f]$ onto $[0, 1]$. This principle converts system (4.1) into a problem of minimizing point-wise Hamiltonian H, with respect to $(u_1, u_2, u_3, u_4, u_5)$. To obtained the Hamiltonian (H), we follow the approach of [16] such that

$$\begin{aligned}
H = {} & A_1 I_H^V + A_2 I_H^C + A_3 I_H^{VC} + A_4 N_V + A_5 B + \frac{1}{2}(D_1 u_1^2 + D_2 u_2^2 + D_3 u_3^2 + D_4 u_4^2 + D_5 u_5^2) \\
& + \lambda_{S_H}\left(\Lambda_H + \psi R_H^V + \omega R_H^C - \frac{(1-u_1)\varepsilon\beta_V I_V S_H}{N_H} - \frac{\beta_C I_H^C S_H + \beta_B B S_H}{N_H} - \mu_H S_H\right) \\
& + \lambda_{I_H^V}\left(\frac{(1-u_1)\varepsilon\beta_V I_V S_H}{N_H} - \frac{\beta_C I_H^C I_H^V}{N_H} - (\delta_H + \mu_H + u_2\gamma_H) I_H^V\right) \\
& + \lambda_{I_H^C}\left(\frac{\beta_C I_H^C S_H + \beta_B B S_H}{N_H} - \frac{(1-u_1)\varepsilon\beta_V I_V I_H^C}{N_H} - (\xi + u_5\gamma_C + \mu_H + \delta_C) I_H^C\right) \\
& + \lambda_{I_H^{VC}}\left(\frac{\beta_C I_H^C I_H^V}{N_H} + \frac{(1-u_1)\varepsilon\beta_V I_V I_H^C}{N_H} - (\delta_H + \delta_C + u_5\upsilon\gamma + u_2(1-\upsilon)\gamma) I_H^{VC}\right) \\
& + \lambda_R^V\left(u_2\gamma_H I_H^V + u_2(1-\upsilon)\gamma I_H^{VC} - (\psi + \mu_H) R_H^V\right) \\
& + \lambda_R^C\left(u_5\gamma_C I_H^C + u_5\upsilon I_H^{VC} - (\omega + \mu_H) R_H^C\right) \\
& + \lambda_{S_v}\left(\Lambda_V - \frac{(1-u_1)\varepsilon\beta_H S_V (I_H^V + I_H^{VC})}{N_V} - (\mu_V + u_3\theta_V) S_V\right) \\
& + \lambda_{I_V}\left(\frac{(1-u_1)\varepsilon\beta_H S_V (I_H^V + I_H^{VC})}{N_V} - (\mu_V + u_3\theta_V) I_V\right) \\
& + \lambda_B\left(\xi I_H^C - (\mu + u_4\delta_B) B\right),
\end{aligned} \tag{4.3}$$

where $\lambda_{S_H}, \lambda_{I_H^V}, \lambda_{I_H^C}, \lambda_{I_H^{VC}}, \lambda_{R_H^V}, \lambda_{R_H^C}, \lambda_{S_V}, \lambda_{I_V}, \lambda_B$ are the adjoint variables.

Theorem 4.1. *Let u_1^*, u_2^*, u_3^*, u_4^*, u_5^* be optimal controls and S_H, I_H^V, I_H^C, I_H^{VC}, R_H^V, R_H^C, S_V, I_V, and B be the solutions of the optimal control problem (4.1) and (4.2) that minimize $J(u_1, u_2, u_3, u_4, u_5)$ over U, then there exist adjoint variables λ_{S_H}, $\lambda_{I_H^V}$, $\lambda_{I_H^C}$, $\lambda_{I_H^{VC}}$, $\lambda_{R_H^V}$, $\lambda_{R_H^C}$, λ_{S_V}, λ_{I_V}, λ_B satisfying $-\frac{d\lambda_i}{dt} = \frac{\partial H}{\partial i}$. Where S_H, I_H^V, I_H^C, I_H^{VC}, R_H^V, R_H^C, S_V, I_V, and B are the adjoint variables and the controls u_1^*, u_2^*, u_3^*, u_4^*, u_5^* obeys the optimality conditions.*

$$\begin{aligned}
u_1^* &= min\left\{0, max\left(1, \frac{a_1 + a_2}{B_1}\right)\right\}, \\
u_2^* &= min\left\{0, max\left(1, \frac{(\lambda_{I_H^V} - \lambda_{R_H^V})\gamma_H I_H^V + (\lambda_{I_H^{VC}} - \lambda_{R_H^V})(1-\upsilon)\gamma I_H^{VC}}{B_2}\right)\right\}, \\
u_3^* &= min\left\{0, max\left(1, \frac{\lambda_{S_V}\theta_V S_V + \lambda_{I_V}\theta_V}{B_3}\right)\right\} \\
u_4^* &= min\left\{0, max\left(1, \frac{\lambda_B\delta_B B}{B_4}\right)\right\}, \\
u_5^* &= min\left\{0, max\left(1, \frac{(\lambda_{I_H^C} - \lambda_{R_H^C})\gamma_C I_H^C + (\lambda_{I_H^{VC}} - \lambda_{R_H^C})\upsilon\gamma I_H^{VC}}{B_5}\right)\right\}.
\end{aligned} \tag{4.4}$$

$a_1 = (\lambda_{I_H^V} - \lambda_{S_H})\frac{\varepsilon\beta_V I_V S_H}{N_H} + (\lambda_{I_H^{VC}} - \lambda_{I_H^C})\frac{\varepsilon\beta_V I_V I_H^C}{N_H},\ a_2 = (\lambda_{I_V} - \lambda_{S_V})\frac{\varepsilon\beta_H S_V (I_H^V + I_H^{VC})}{N_V}$

Proof. Differentiating the Hamiltonian partially with respect to the state variables and multiplying the results by a negative, we have

$$\begin{aligned}
\frac{d\lambda_{S_H}}{dt} =&\lambda_{S_H}\left(\frac{(1-u_1)\varepsilon\beta_V I_V}{N_H} + \frac{\beta_C I_H{}_C + \beta_B B}{N_H} + \mu_H\right) - \lambda_{I_H^V}\left(\frac{(1-u_1)\varepsilon\beta_V I_V}{N_H}\right),\\
&-\lambda_{I_H^C}\left(\frac{\beta_C I_H^C + \beta_B B}{N_H}\right),\\
\frac{d\lambda_{I_H^V}}{dt} =&(\lambda_{I_H^V} - \lambda_{I_H^{VC}})\frac{\beta_C I_H^C}{N_H} + (\lambda_{I_{S_V}} - \lambda_{I_V})\left(\frac{(1-u_1)\varepsilon\beta_H S_V}{N_V}\right),\\
&+(\lambda_{I_H^V} - \lambda_{R_H^V})u_2\gamma_H + \lambda_{I_H^V}(\delta_H + \mu_H) - A_1,\\
\frac{d\lambda_{I_H^C}}{dt} =&(\lambda_{I_H^V} - \lambda_{R_H^{VC}})\frac{\beta_C I_H^V}{N_H} + (\lambda_{S_H} - \lambda_{I_H^C})\frac{\beta_C S_H}{N_H} + (\lambda_{I_H^C} - \lambda_{I_H^{VC}})\left(\frac{(1-u_1)\varepsilon\beta_V I_V}{N_H}\right),\\
&+\left(\lambda_{I_H^C} - \lambda_{R_H^C}\right)u_5\gamma_C + \left(\lambda_{I_H^C} - \lambda_B\right)\xi + \lambda_{I_H^C}(\mu_H + \delta_C) - A_2,\\
\frac{d\lambda_{I_H^{VC}}}{dt} =&(\lambda_{I_H^{VC}} - \lambda_{R_H^V})u_2(1-\upsilon)\gamma + (\lambda_{I_H^{VC}} - \lambda_{R_H^C})u_5\upsilon\gamma + (\lambda_{S_V} - \lambda_{I_V})\frac{(1-u_1)\varepsilon\beta_H S_V}{N_V},\\
&+\lambda_{I_H^{VC}}(\delta_H + \delta_C + \mu_H) - A_3,\\
\frac{d\lambda_{R_H^V}}{dt} =&(\lambda_{R_H^V} - \lambda_{S_H})\psi + \lambda_{R_H^V}\mu_H,\\
\frac{d\lambda_{R_H^C}}{dt} =&(\lambda_{R_H^C} - \lambda_{S_H})\omega + \lambda_{R_H^C}\mu_H,\\
\frac{d\lambda_{S_V}}{dt} =&(\lambda_{S_V} - \lambda_{I_V})\left(\frac{(1-u_1)\varepsilon\beta_H I_H^V}{N_V}\right) + (\lambda_{S_V} - \lambda_{I_V})\left(\frac{(1-u_1)\varepsilon\beta_H I_H^{VC}}{N_V}\right),\\
&+\lambda_{S_V}(\mu_V + u_3\theta_V) - A_4\\
\frac{d\lambda_{I_V}}{dt} =&(\lambda_{S_H} - \lambda_{I_V})\left(\frac{(1-u_1)\varepsilon\beta_V S_H}{N_H}\right) + (\lambda_{I_H^C} - \lambda_{I_H^{VC}})\left(\frac{(1-u_1)\varepsilon\beta_V I_H^C}{N_H}\right),\\
&+\lambda_{I_V}(\mu_V + u_3\theta_V) - A_4,\\
\frac{d\lambda_B}{dt} =&(\lambda_{S_H} - \lambda_{I_H^{VC}})\frac{\beta_B S_H}{N_H} + \lambda_B(\mu + u_4\delta_B) - A_5
\end{aligned} \tag{4.5}$$

with transversality conditions:

$$\begin{cases}\lambda_{S_H}(t_f) = \lambda_{I_H^V}(t_f) = \lambda_{I_H^C}(t_f) = \lambda_{I_H^{VC}}(t_f) = \lambda_{R_H^V}(t_f) = \lambda_{R_H^C}(t_f),\\ = \lambda_{S_V}(t_f) = \lambda_{I_V}(t_f) = \lambda_B(t_f) = 0.\end{cases} \tag{4.6}$$

Now, we obtain the characterized control set by differentiating the Hamiltonian with respect to the controls: u_1^*, u_2^*, u_3^*, u_4^* and u_5^* such that

$$\frac{\partial H}{\partial u_i^*} = 0, i = 1, 2 \ldots, 5.$$

is satisfied.
Therefore,

$$u_1^* = \left(\frac{(\lambda_{I_H^V} - \lambda_{S_H})\frac{\varepsilon\beta_V I_V S_H}{N_H} + (\lambda_{I_H^{VC}} - \lambda_{I_H^C})\frac{\varepsilon\beta_V I_V I_H^C}{N_H} + (\lambda_{I_V} - \lambda_{S_V})\frac{\varepsilon\beta_H S_V (I_H^V + I_H^{VC})}{N_V}}{B_1}\right),$$

$$u_2^* = \left(\frac{(\lambda_{I_H^V} - \lambda_{R_H^V})\gamma_H I_H^V + (\lambda_{I_H^{VC}} - \lambda_{R_H^V})(1-\upsilon)\gamma I_H^{VC}}{B_2} \right),$$

$$u_3^* = \left(\frac{\lambda_{S_V}\theta_V S_V + \lambda_{I_V}\theta_V}{B_3} \right),$$

$$u_4^* = \left(\frac{\lambda_B \delta_B B}{B_4} \right),$$

$$u_5^* = \left(\frac{(\lambda_{I_H^C} - \lambda_{R_H^C})\gamma_C I_H^C + (\lambda_{I_H^{VC}} - \lambda_{R_H^C})\upsilon\gamma I_H^{VC}}{B_5} \right).$$

In accordance with [7], based on typical control arguments involving the bound on the controls, we conclude that, as a result of the apriori boundedness of the state system, the adjoint system, we obtained the uniqueness of optimality system (4.5),(4.6). There is a restriction on the length of time interval $[0, t_f]$ so that we can guarantee the uniqueness of the optimality system [13]. ∎

5. Numerical simulations

We used forward-backward sweep method for our numerical simulations. The method is a numerical technique for solving optimal control problems. It is one of the indirect methods in which the differential equations from the maximum principle are numerically solved [7]. We investigate the effect of the optimal control strategies on the spread cholera-malaria co-infection using parameters and variables values in Tables 4 and 5. We obtained the graphs from Figures 2 to 8. The strategies are;

I. Strategy A: Treated bed nets, treatment of malaria and treatment of cholera.
II. Strategy B: Treatment of malaria, indoor residual spray and treatment of cholera.
III. Strategy C: Treatment of malaria, sanitation and treatment of cholera.
IV. Strategy D: Treated bed nets, treatment of malaria, indoor residual spray and treatment of cholera.
V. Strategy E: Treated bed nets, treatment of malaria, sanitation and treatment of cholera.
VI. Strategy F: Treatment of malaria, indoor residual spray, sanitation and treatment of cholera.
VII. Strategy G: Treated bed nets, treatments of malaria, indoor residual spray, sanitation and treatment of cholera.

TABLE 4. Initial values used in the model.

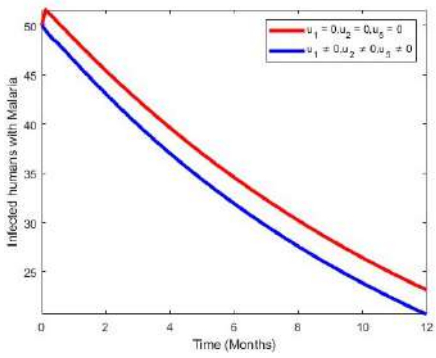
Variable	Value	Source
$S_H(0)$	250	[14]
$I_H^V(0)$	50	Assumed
$I_H^C(0)$	40	Assumed
$I_H^{VC}(0)$	200	Assumed
$R_H^V(0)$	15	Assumed
$R_H^C(0)$	10	[6]
$S_V(0)$	950	[14]
$I_V(0)$	30	[14]
$B(0)$	275000	[6]

TABLE 5. Parameters and values used in the model.

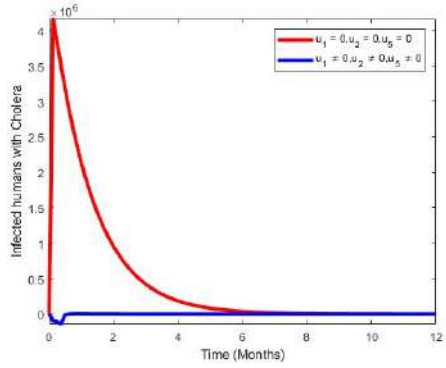
Parameter	Value	Source
Λ_H	100	[14]
Λ_V	1000	[14]
μ_H	0.02	Assumed
μ_V	1.1429	[14]
ε	0.2	[14]
β_V	0.502	[14]
ω	0.62	Assumed
ψ	0.7902	[14]
γ_H	0.005	[6]
θ_V	0.25	[14]
δ_C	0.2407	[14]
δ_H	0.05	[14]
γ	0.35	[14]
μ	2	Assumed
ξ	10	Assumed
δ_B	0.5	Assumed
γ_C	0.2	Assumed
β_H	0.8333	[14]
β_C	0.0011	Assumed
β_B	0.5	Assumed
υ	0.5	Assumed
A_1	100	Assumed
A_2	90	Assumed
A_3	70	Assumed
A_4	50	Assumed
A_5	20	Assumed
C_1	20	[1]
C_2	65	[1]
C_3	10	[1]
C_4	30	Assumed
C_5	15	Assumed

Strategy A: Treated bed nets, treatment of malaria and treatment of cholera

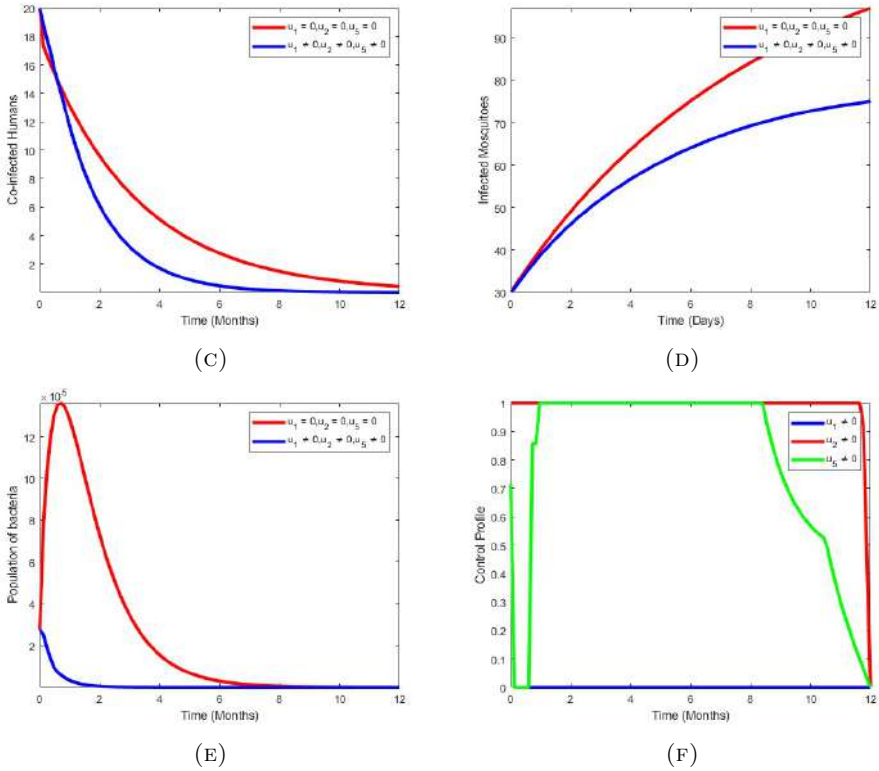
In strategy A, treated bed nets, treatment of malaria and treatment of cholera (u_1, u_2, u_5) are combined as control strategy against malaria-cholera diseases in a population to optimize the objective functional (J), while we set (u_3) and (u_4) to zero. Figures 2(A) - 2(E), shows a significant difference in the population at various states of infection, with optimal strategy $(u_1, u_2, u_5 \neq 0)$ when compared to the population without optimal control $(u_1, u_2, u_5 = 0)$. We observed in Figure 2(A) that due to the control strategy, number of infected human with malaria decreases, while the population increases when there is no control. Similarly, Figure 2(B) shows a decrease in the presence of control strategy for the infected human with cholera in the population, when compared to the case of no control. In Figure 2(C), we observed that, the population of co-infection human individuals with both diseases reduced due to the presence of control while the population increases in the absence of control. In Figure 2(D), the population of infected mosquitoes decreases due the treated bed nets, treatment of malaria and treatment of cholera as control strategy, while the population continued to grow in the absence of control. In Figure 2(E), it is observed that bacteria population decreases to the minimal level when the control strategy was implemented compared with the number of bacteria population when there is no control. The control profile in Figure 2(F), revealed that, using treated bed nets, treatment of malaria and treatment of cholera as a strategy for preventing co-infection, treatment of infected malaria and cholera are at upper bound for 11.9 and 8.6 months respectively before gradually dropping to the lower bound, while effort is at lower bound for treated bed nets through out the implementation.



(A)



(B)



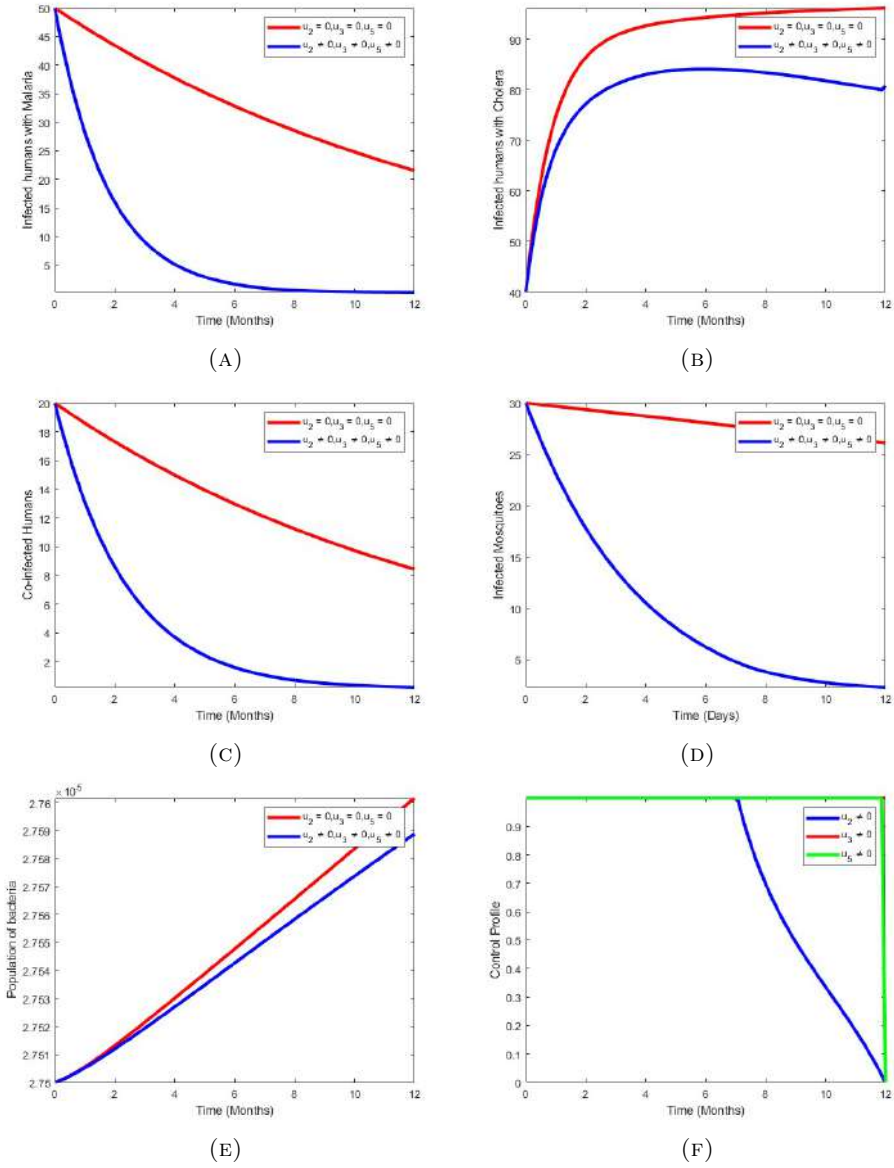
(C) (D)

(E) (F)

FIGURE 2. Treated bed nets, treatment of malaria and treatment of cholera.

STRATEGY B: TREATMENT OF MALARIA, TREATMENT OF CHOLERA, INDOOR RESIDUAL SPRAY AS CONTROL STRATEGY

This strategy shows the optimal treatment of malaria (u_2), indoor residual spray (u_3) and treatment of cholera (u_5) as the control strategy while other controls; (u_1) treatment bed nets and sanitation of the environment against bacteria (u_4) are set to zero. In Figures 3(A) - 3(E), the results show a significant decrease in infected humans with malaria, infected human with cholera, co-infected humans, infected mosquitoes and total population of bacteria when the strategy is implemented compared to the case without control. In Figure 3(F), we observed that the efforts put into implementation of the strategy is at the upper bound for 8 months, 12 months and 11.9 months for use of treated bed nets, treat before gradually dropping down to the lower bound.

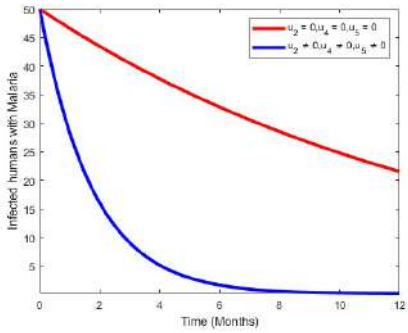


(A) (B) (C) (D) (E) (F)

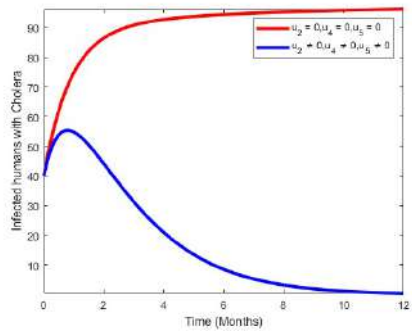
FIGURE 3. Treatment of malaria, indoor residual spray and treatment of cholera.

Strategy C: treatment of malaria, sanitation and treatment of cholera as control strategy

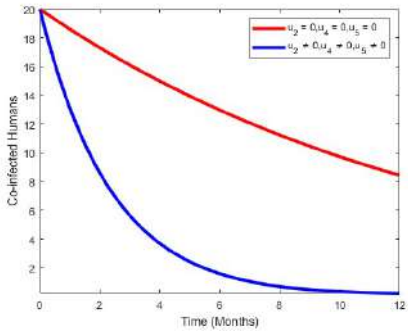
In strategy C, treatment of malaria, sanitation and treatment of cholera were considered as the control strategy setting treated bed nets (u_1) and indoor residual spray (u_3) to zero. Figures 4(A) - 4(E) show that in the presence of control, the number of infected individuals with malaria, number of infected individuals, number of infected individuals with co-infected, number of infected mosquitoes and total number of bacteria decrease to the minimal level compared to the case where there is no control. The control profile in Figure 4(F), reveals that efforts is at upper bound for $7,8$ months, 12 months and 11.9 months for treatment of malaria, sanitation spray and treatment of cholera respectively before gradually dropping down to the lower bound.



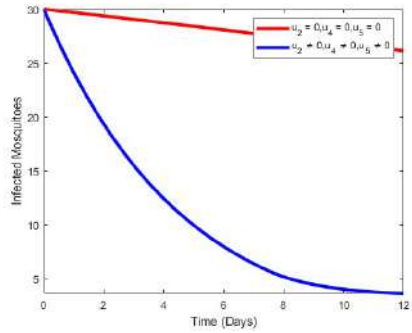
(A)



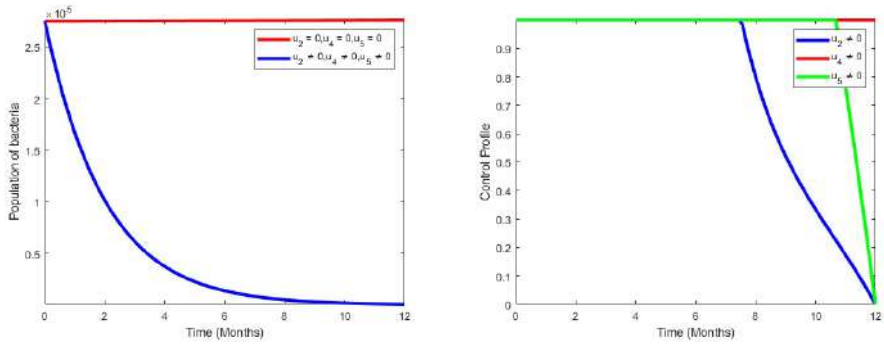
(B)



(C)



(D)

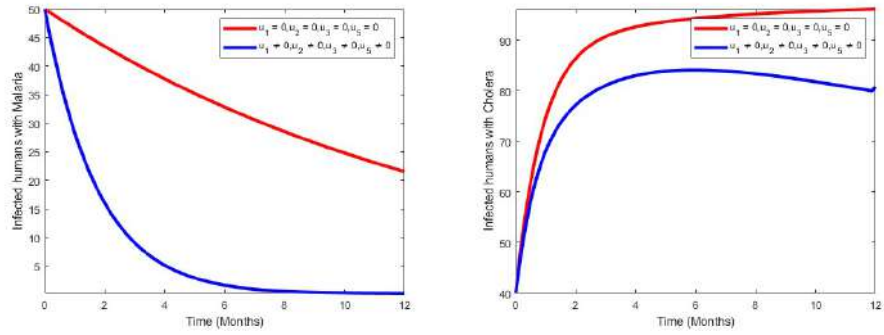


(E) (F)

FIGURE 4. Treatment of malaria, sanitation and treatment of cholera as control strategy.

STRATEGY D: TREATED BED NETS, TREATMENT OF MALARIA, INDOOR RESIDUAL SPRAY AND TREATMENT OF CHOLERA AS CONTROL STRATEGY

In strategy D, treated bed nets, treatment of malaria, indoor residual spray and treatment of cholera were considered as the control strategy setting sanitation (u_4) to zero. Figures 5(A) - 5(E) depict that in the presence of control, there is a greater decrease in the the number of infected individuals with malaria, number of infected individuals, number of infected individuals with co-infected, number of infected mosquitoes and total number of bacteria compared to the case with no control. The control profile in Figure 5(F), reveals that efforts put into implementation is at upper bound for $7, 8$ months, 12 months and 11.9 months for treatment of malaria, indoor residual spray and treatment of cholera respectively before gradually dropping down to the lower bound while effort is at lower bound for treated bed nets through out the study period.



(A) (B)

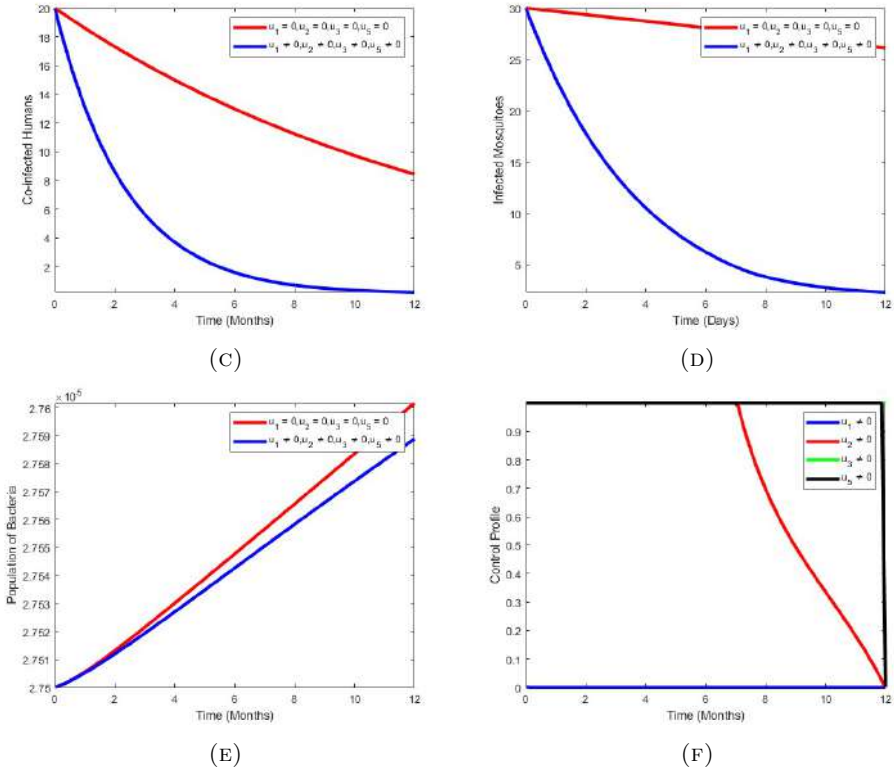


FIGURE 5. treated bed nets, treatment of malaria, indoor residual spray and treatment of cholera as control strategy.

STRATEGY E: TREATED BED NETS, TREATMENT OF MALARIA, SANITATION AND TREATMENT OF CHOLERA AS CONTROL STRATEGY

In strategy E, treated bed nets, treatment of malaria, sanitation and treatment of cholera were implemented as the control strategy setting indoor residual spray (u_3) to zero. Figures 6(A) - 6(E) show a significant decrease in the the number of infected individuals with malaria, number of infected individuals, number of infected individuals with co-infected, number of infected mosquitoes and total number of bacteria compared to the case with no control. The control profile in Figure 6(F), reveals that efforts put into implementation is at upper bound for 8 months, 12 months and 11 months for treatment of malaria, sanitation and treatment of cholera respectively before gradually dropping down to the lower bound while effort is at lower bound for treated bed nets through out the study period.

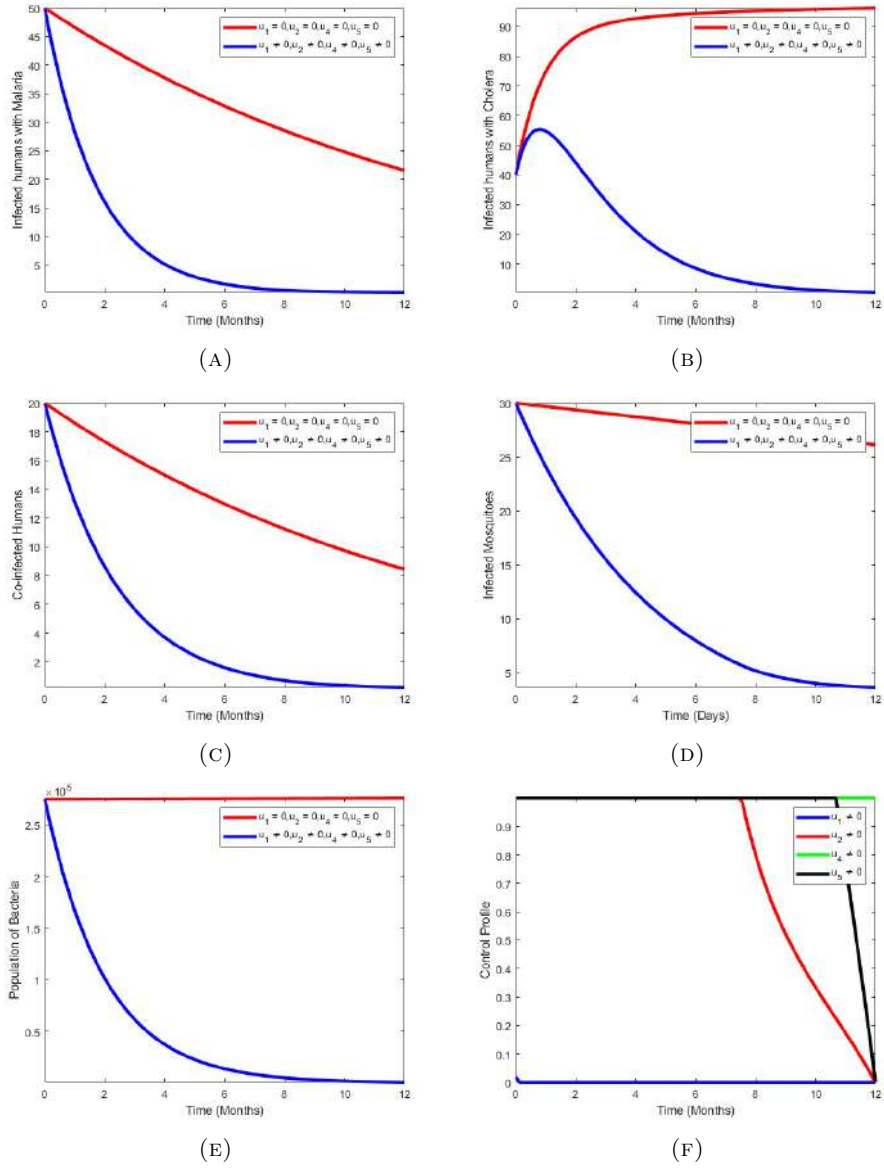


FIGURE 6. treated bed nets, treatment of malaria, sanitation and treatment of cholera as control strategy.

STRATEGY F: TREATMENT OF MALARIA, INDOOR RESIDUAL SPRAY, SANITATION AND TREATMENT OF CHOLERA AS CONTROL STRATEGY

In strategy F, treatment of malaria, indoor residual spray, sanitation and treatment of cholera were implemented as the control strategy setting treated bed nets (u_1) to zero.

Figures 7(A) - 7(E) show a better result compared to strategy A, B, C, D and E respectively. The control profile in Figure 7(F), reveals that efforts put into implementation of strategy F is at upper bound for 8 months, 12 months, 11 months and 12 months for treatment of malaria, indoor residual spray, sanitation and treatment of cholera respectively before gradually dropping down to the lower bound.

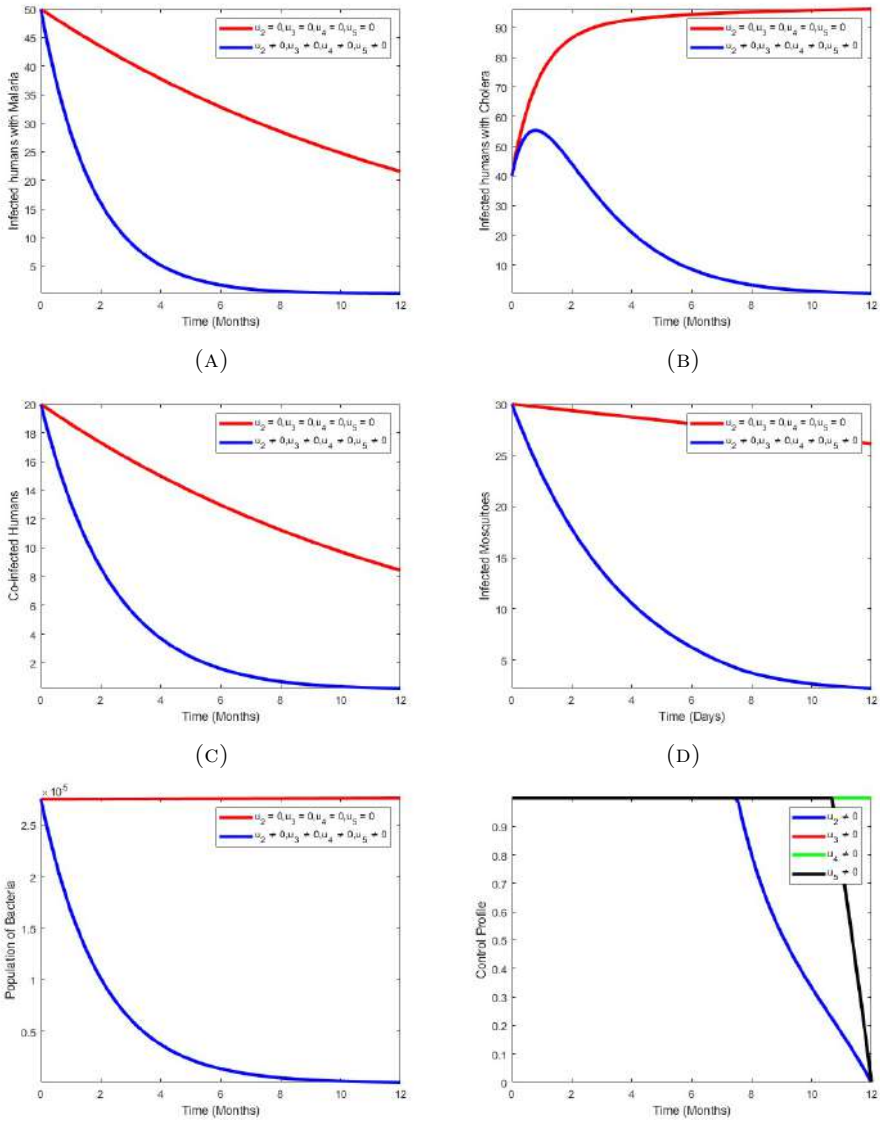


FIGURE 7. treatment of malaria, indoor residual spray, sanitation and treatment of cholera as control strategy.

Strategy G: treated bed nets, treatments of malaria, indoor residual spray, sanitation and treatment of cholera as control strategy

In strategy G, treated bed nets, treatment of malaria, indoor residual spray, sanitation and treatment of cholera were implemented as the control strategy. Figures 8(A) - 8(E) show the best result compared to the other implemented strategies. The control profile in Figure 8(F), reveals that efforts put into implementation of strategy G is at upper bound throughout the studied period for all strategies before gradually dropping down to the lower bound except, control profile for treated bed nets which drops to lower bound in less than a month.

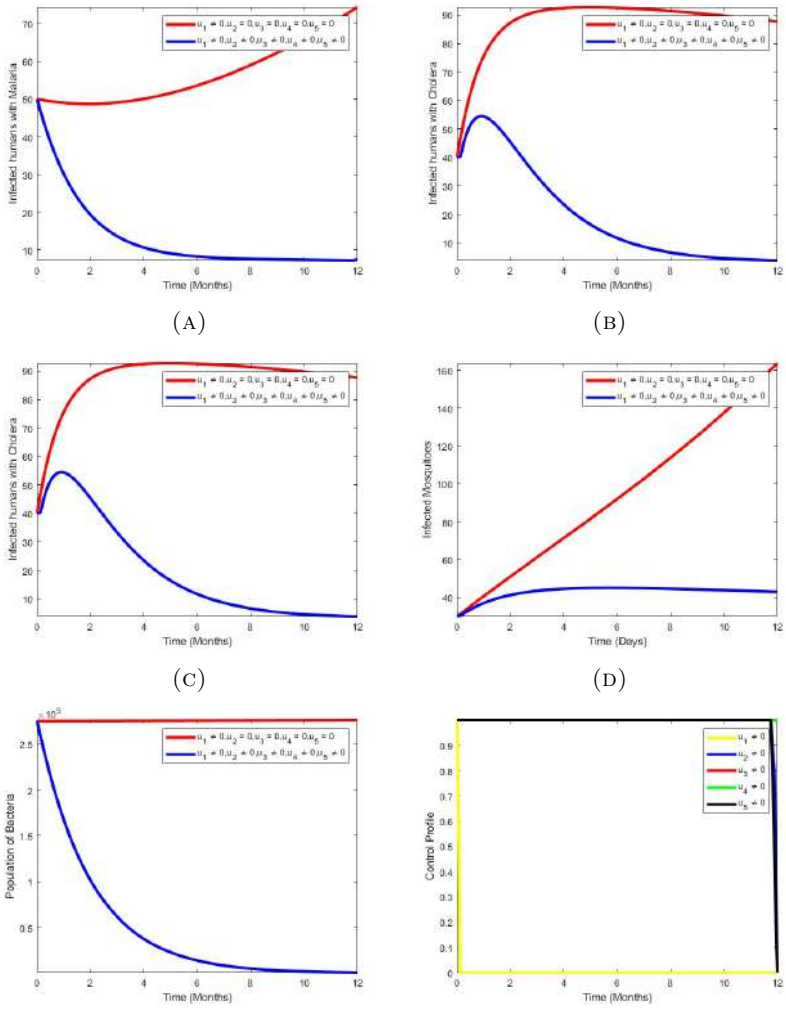


Figure 8. treated bed nets, treatments of malaria, indoor residual spray, sanitation and treatment of cholera as control strategy.

6. Conclusion

This study proposes a deterministic mathematical model with five countermeasures against co-infection of cholera and malaria. The system of nonlinear differential equations is mathematically and epidemiologically well-posed, and the positivism of the model equation solutions was established and determined to be positive. The next generation approach was used to calculate the fundamental reproduction number. The analysis that was done shows that the disease free is unstable if $\mathcal{R}_0 > 1$ and locally asymptotically stable when $\mathcal{R}_0 < 1$. We investigated the ideal quantity needed to stop the spread of the cholera-malaria illness in a community using Pontryagin's maximal principle. Combining treated bed nets, medication-assisted malaria therapy, indoor residual spray, sanitation, and cholera treatment is the best way to manage malaria and cholera.

Author contributions

The authors contributed equally in writing this article. All authors read and approved the final manuscripts.

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