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Research Article

Mathematical Analysis of Malaria and Cholera Disease by Homotopy Perturbation Method

PN Vijaya Kumar[1], P Balaganesan[2], J Renuka[3]

[1]Research Scholar, [2]Professor, Department of Mathematics, Academy of Maritime Education and Training (AMET) Deemed to be University, Chennai, Tamil Nadu, India.
[3]Assistant Professor, Department of Mathematics, Women's Christian College, Chennai, Tamil Nadu, India.
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I N F O

Corresponding Author:
P Balaganesan, Department of Mathematics, AMET Deemed to be University, Chennai, Tamil Nadu, India.
E-mail Id:
balaganesanpp@gmail.com
Orcid Id:
https://orcid.org/0000-0003-4581-0608
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A B S T R A C T

The mathematical model for co-infection with malaria and cholera was developed in this problem, and it was researched to see if there was a synergistic link between the two diseases in the presence of medicines. The effects of malaria and its treatment on cholera dynamics were investigated in greater depth. Malaria infection raises the chances of cholera, while cholera infection doesn't really enhance the risk of malaria infection. The model was numerically investigated using the fourth-order Runge-Kutta method and analytically using the Homotopy Perturbation Method. The impact of each parameter on the governing equation was investigated and the effective control strategy was determined using the exact solutions (analytical).

Keywords: Malaria, Cholera, Co-infection, Homotopy Perturbation Method, Optimality Control of Disease

Introduction

Malaria is a disease transmitted by mosquitoes that may be prevented and treated. Mosquito biting is stopped by the distribution of low-cost bed nets and sprays, as well as by mosquito abatement tactics such as sprinkling insecticide indoors and draining filthy water where vectors thrive.[1] "Recent flooding in Africa and Asia has created a significant danger to environmental sanitation and clean water supply, allowing malaria and cholera to spread".[2]

John Snow was the first to examine cholera epidemiology, which paved the way for modern epidemiology study.[3] The relationshi p between cholera and contaminated water has long been known. Cholera is a life-threatening bacterium infection that can cause vomiting and diarrhoea. It is spread by drinking water infected with the bacteria *Vibrio cholerae* (typically via faeces or wastewater) and can lead to "severe thirst, electrolyte imbalance, and death", if not treated. Cholera colonisation by humans results in a super infectious condition that persists after transmission, contributing to epidemic illness. Good sanitation and water treatment can help prevent cholera.[4]

Mathematical modelling has been useful in gaining a better understanding of disease transmission dynamics as well as in making decisions about disease control intervention techniques. Ross, for example, was the first to establish "Mathematical models of malaria transmission".[5] His major objective was vector management, and he established that the mosquito population must be decreased to a

certain level in order to eradicate the infection. Koella's and Anita's inquiries are among the others,[6] Among them was a mosquito latent class. They evaluated various tactics for reducing "resistance spread and investigated the sensitivity of their findings to the parameters". Anderson and May developed "a malaria model based on the assumption that acquired immunity is unaffected by exposure length in malaria".[7] The effect of disease prevalence on transmission rate, as well as other control methods, were explored further. Stilianakis et al. performed a thorough examination of a dynamic model to characterise HIV infection aetiology.[8] With vaccination and various endemic states, "A basic two-dimensional SIS (susceptible-infected-susceptible) model" was created by Kribs-Zaleta and Vealsco-Hernandez.[9] There has been no research on the dynamics of malaria-cholera co-infection or the deployment of optimal methods of control to our understanding. A deterministic framework for HIV and malaria co-infection in a population was recently constructed and tested by Mukandavire et al.[10] Mtisi et al. also looked at a deterministic model for tuberculosis and malaria co-infection.[11] Mushayabasa and Bhunu developed a simple mathematical model and comprehensively analysed it to assess whether HIV infection is associated with an increased risk for cholera or not.[12] Nielan et al. developed a mathematical model for cholera that included key elements - "hyper-infectious, short-lived bacterial condition, a distinct class for mild human infections, and diminishing disease immunity".[13]

Okosun. et. al.14 found that malaria infection may be associated with an increased risk of cholera but however, cholera infection is not associated with an increased risk for malaria.[14] There have been significant mathematical modelling developments over the past eight years.[15] Osman and Adu established the SEIR model in 2017, which was used to predict the reproduction rate, and the disease-free and endemic equilibria. Additionally, they created an SEIR-SEI model of how malaria spreads between people and insects. Insecticides, mosquito-treated nets, and active anti-malarial medications can all assist in reducing mosquito populations and the transmission of malaria, according to the study's findings. Malaria can also be controlled by lowering the rate of human-mosquito interaction.

A historical review of the dynamics of malaria transmission and climate change was conducted in 2018 by Eikenberry and Gumel.[16] They discussed the main biological facets of malaria, techniques for formalising these into mathematical forms, and uncertainties and debates surrounding appropriate modelling approaches. Their goal was to present a timeline of some significant modelling initiatives, ranging from the classic works of Sir Ronald Ross and George Macdonald to more contemporary modelling studies that specifically focused on climate. Last but not least, they made an effort to set this mathematical study into a larger historical framework for the "million-murdering death" of malaria.

A mathematical model of malaria transmission dynamics with age structure for the vector population and a regular bite rate of female Anopheles mosquitoes was presented in 2018 by Bakary et al.[17] They examined the model's behaviour when the fundamental reproduction ratio R_0 was more than one or less than one, as well as the stability of the disease-free equilibrium. In 2018, Koutou et al. presented a model, constructed by considering two models: a model of vector population and a model of virus transmission.[18] Each model's threshold dynamics were identified, and a correlation between them was created. The Lyapunov principle was additionally used to investigate the stability of equilibrium points. The next-generation matrix was used to calculate the common basic reproduction number, and its implications for the management of malaria were examined.

In 2020, Hntsa and Kahsay took into account a mathematical model for the dynamics of cholera transmission and its countermeasure as one cohort of people.[19] They estimated the fundamental reproduction number R_0 and looked into the possibility of equilibria as well as their stability. According to the findings, the disease is eradicated and kills fewer people in places with appropriate preventive measures while becoming widespread and killing more people in areas with insufficient preventive measures. In 2020, Egeonu et al. conducted an analysis to determine how treatment controls affected a population's disease burden when there was drug resistance to malaria.[20] When the related reproduction number is smaller than unity, the dynamic feature of backward bifurcation is demonstrated to occur in the model. Pontryagin's Maximum Principle was used to establish the prerequisites for the existence of optimum control and the optimality system for the model. The study of a novel non-linear mathematical model for malaria disease was proposed by Ibrahim et al. in 2020.[21] Using the fundamental reproductive number R_0, local and global stability analysis of a disease-free equilibrium was investigated; if $R_0 < 1$, the system is stable; else, it is unstable. Under specific circumstances, the existence of a special endemic equilibrium was also established. The findings of this study demonstrate that in addition to a decline in the population of infected mosquitoes, a sizable increase in the population of susceptible humans has been

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observed. A non-linear mathematical model was proposed and used in 2020 by Baihaqi and Adi-Kusumo to analyse the dynamics of malaria transmission in a population.[22] Utilising differential equation stability theory, the deterministic compartmental model was investigated. The reproduction number was asymptotically stable for the disease-free conditions, and the endemic equilibria were identified. In addition, the qualitatively assessed model incorporated time-dependent variable controls that were intended to stop the spread of the malaria disease. Incorporating three control strategies—disease prevention with bed nets, treatment, and insecticides—the optimal control problem was formulated using Pontryagin's maximum principle. A mathematical model of malaria in the human population was considered in 2020 by Baihaqi and Adi-Kusumo,[22] who made the assumption that the disease's infection rate in the population is constant and only depends on the number of infected people. The existence of endemic and disease-free equilibrium points, as well as the local stability for both equilibrium points and the global stability for the disease-free equilibrium point, were also the focus of their analysis.

In 2021, Ndamuzi and Gahungu created the SEIR model and discovered the fundamental reproduction number R_0.[23] This model's findings demonstrated the need for effective control measures to decrease the number of mosquito bites on humans per unit of time (σ), the vector population of mosquitoes (N_v), the probability of infection for a person bitten by an infectious mosquito per unit of time (b), and the probability of infection for a mosquito per unit of time (c). The new fourteen-compartmental mathematical model was defined by Adeniran et al. in 2022.[24] In an effort to pinpoint the most sensitive factors, the model was examined for each factor influencing the spread of the illness. Full models of the co-infection of malaria and cholera were studied first, followed by sub-models of the two diseases separately. The relative sensitivity solution of the model was calculated for each parameter.

A fractional order model of the co-infection of malaria and cholera was developed in 2022 by Shah et al. using the definition of the Caputo fractional derivative with the order of derivative in the range (0,1).[25] The fractional order model has been shown to produce superior results. For malaria and cholera models, the basic reproduction number was calculated. Sinan et al.[26] started to look at the dynamics of malaria illness in humans in 2022, as well as the disease's vectors. It was also considered how the vector's (the mosquito) role in disease transmission might play a role. Ulam-Hyres stability analysis and optimal control strategies have been used in some theoretical analyses of existence, uniqueness, and stability. Different graphs show the results of fractional and classical order, and some numbers are shown to show the model's overall asymptotic stability. Finally, we have made an attempt to investigate the mathematical analysis of Malaria and cholera disease by HPM.[14]

Model Formulation

Humans who are susceptible (S_h), individuals only infected with malaria (I_m), individuals only infected with cholera (I_c), individuals only recovering from malaria (R_m), and individuals only recovering from cholera alone (R_c) are all sub-populations of the overall human population, denoted by N_h. As a result, $N_h = S_h + I_m + I_c + R_m + R_c$ vector population, N_v, is separated into susceptible mosquitoes and malaria-infected mosquitoes S_v. As a result, $= N_v = S_v + I_v$.[14]

Cholera Model

We will look at the cholera-only model here.[14]

$$\frac{dS_h}{dt} = \Lambda_h + \omega R_c - \lambda S_h - \mu_h S_h \quad (1)$$

$$\frac{dI_C}{dt} = \lambda S_h - (\delta + \mu_h + m) I_c \quad (2)$$

$$\frac{dR_c}{dt} = \delta I_c - (\omega + \mu_h) R_c \quad (3)$$

$$\frac{dB_c}{dt} = \rho I_c - \mu_b B_c \quad (4)$$

With the initial condition

$$S_h(0) \geq 0,\ I_c(0) \geq 0,\ R_c(0) \geq 0, B_c(0) \geq 0.$$

The analytical solution of the cholera model is obtained by the Homotopy perturbation method (HPM).

$$S_h(t) = 2498001.59e^{(-0.05004t)} + 1998.402 \quad (5)$$

$$I_c(t) = 1061.737 + 2834129.339e^{(-0.05004t)} - 2835191.075e^{(-0.09411t)} \quad (6)$$

$$R_c(t) = -4051995.2020e^{(-0.05004t)} + 2133316.4810e^{(-0.09411t)} + 1918678.7210e^{(-0.00104t)} \quad (7)$$

$$B_c(t) = 27213235.32e^{(-0.05004t)} - 68725429.18e^{(-0.09411t)} 41512193.86e^{(-0.0123t)} \quad (8)$$

Malaria Model

We will look at the malaria-only model here.[14]

$$\frac{dS_h}{dt} = \Lambda_h + kR_m - \beta_h S_h I_v - \mu_h S_h \quad (9)$$

$$\frac{dI_m}{dt} = \beta_h S_h I_v - (\alpha + \mu_h + \emptyset) I_m \quad (10)$$

$$\frac{dR_m}{dt} = \alpha I_m - (k + \mu_h) R_m \quad (11)$$

$$\frac{dS_v}{dt} = \Lambda_v - \beta_v S_v I_m - \mu_v S_v \quad (12)$$

$$\frac{dI_v}{dt} = \beta_v S_v I_m - \mu_v I_v \quad (13)$$

With the initial condition,

$$S_h(0) \geq 0, I_m(0) \geq 0, R_m(0) \geq 0, S_v(0) \geq 0, I_v(0) \geq 0$$

The analytical solution of the malaria model is obtained by HPM.

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$$S_h(t) = -928.4271e^{(-0.06671t)} - 94.9418e^{(-0.011409t)} + 181.78e^{(-0.011449t)} + 361.597e^{(-0.078079t)} - 708.826e^{(-0.078119t)} + 473.971e^{(-0.06667t)} - 0.013358e^{(-0.01004t)} + 0.000592e^{(-0.089528t)} + 454.468e^{(-0.0667t)} - 1.672663733e^{(-0.13338t)} + 0.8714282085e^{(-0.13334t)} - 0.000619e^{(-0.08948t)} + 0.942564e^{(-0.14479t)} - 0.49148e^{(-0.1448t)} + 0.8027e^{(-0.1334t)} - 24.0018e^{(-0.00004t)} + 0.00398e^{(-0.011409t)t} + 0.01896130957te^{(-0.0667t)} - 0.0198e^{(-0.0667t)t} - 0.01518265724te^{(-0.07808t)} + 0.000107e^{(-0.15616t)} - 0.002215e^{(-0.0767t)} - 0.00012e^{(-0.20005t)} - 0.4519e^{(-0.1449t)} + 0.002207e^{(-0.0768t)} + 0.01456788396te^{(-0.079*t)} + 347.218e^{(-0.079t)} - 86.827e^{(-0.01149t)} - 0.00010218e^{(-.15619t)} - 0.003628te^{(-0.0115t)} + 25 \quad (14)$$

$$I_m(t) = 12434.509e^{(-0.0115t)} - 0.01706te^{(-0.079t)} - 571.409e^{(-0.0667t)} + 0.5334e^{(-0.1448t)} - 1.0229e^{(-0.1448t)} + 1119.2269e^{(-0.0667t)} + 0.0026009e^{(-0.0767t)} + 0.00012e^{(-0.157t)} + 829.592e^{(-0.079t)} + 12426.0026e^{(-0.01149t)} - 24860.509e^{(-0.011449t)} + 1.829e^{(-0.13338t)} - 1.035073796e^{((-0.011409t)t)} - 0.00259e^{(-0.078t)} - 406.358e^{(-0.079t)} + 0.4905e^{(-0.1449t)} - 0.95269e^{(-0.1334t)} + 1.0348te^{(-0.011449t)} + 0.0239e^{(-0.06668t)t} - 0.8775e^{(-0.1335t)} - 547.85e^{(-0.0668t)} - 0.0229te^{(-0.0668t)} + 0.0178te^{(-0.079t)} \quad (15)$$

$$R_m(t) = 0.01336e^{(-0.01005t)} + 4.8295e^{(-0.011452t)} + 0.0028e^{(-0.0667t)} - 0.00266e^{(-0.0668t)} - 0.000208e^{(-0.0115t)t} - 4.8419e^{(-0.011449t)} - 0.00172009e^{(-0.079t)} + 0.0018e^{(-0.079t)} \quad (16)$$

$$S_v(t) = 0.1499 + 0.12613e^{(-0.13339t)} + 0.00155e^{(-0.06668t)t} + 1.2178e^{(-0.011409t)} - 0.08019e^{(-0.1448t)} + 0.08367e^{(-0.1448t)} - 0.131549e^{(-0.13334t)} - 36.3259e^{(-0.0667t)} + 37.1542e^{(-0.0668t)} + 33.2407e^{(-0.079t)} - 1.2183e^{(-0.011449t)} - 33.21625e^{(-0.078079t)} + 0.00139te^{(-0.078079t)} \quad (17)$$

$$I_v(t) = -0.12613e^{(-0.13338t)} - 0.001549e^{(-0.0667t)t} - 1.2178e^{(-0.011409t)} + 0.08019e^{(-0.1448t)} - 0.0836209e^{(-0.1448t)} + 0.131549e^{(-0.1334t)} + 37.1769e^{(-0.0667t)} - 37.1541e^{(-0.0667t)} - 33.2407e^{(-0.079t)} + 1.218236e^{(-0.01145t)} + 33.2163e^{(-0.07808t)} - 0.001394te^{(-0.079t)} \quad (18)$$

Table 1.Parameters and Their Values of Optimal Malaria - Cholera Model[14]

Parameter	Description	Value
Λ_h	Human birth rate	0.001
k	Malaria immunity waning rate	0.01
β_h	Probability of humans getting infected	0.034
μ_h	Natural death rate in humans	0.00004
α	Recovery rate of malaria-infected individual	0.001369
$\emptyset$	Malaria related death	0.01
Λ_v	Mosquitoes birth rate	0.01
β_v	Probability of mosquitoes getting infected	0.09

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μ_v	Natural death rate in mosquitoes	0.06667
ω	Cholera immunity waning rate	0.001
λ	Bacteria contact rate with humans	0.05
δ	Recovery rate of cholera-infected individual	0.07
ρ	Cholera-infected contribution to the aquatic	0.7
μ_b	Bacteria mortality rate	0.123
m	Cholera-related death	0.02407

Numerical Simulation and Discussion

Cholera Model

To observe the model system's dynamic behaviour, using the fourth-order Runge–Kutta method and the following parameters, the systematic Equations 1–4 were numerically computed:[14]

$\mu_b = 0.123, \omega = 0.001, \lambda = 0.05, \delta = 0.07, \mu_h = 0.00004, \rho = 0.7, \Lambda_h = 0.001, m = 0.02407$ with the Innitial Condition $S_h(0) \geq 0,\ I_c(0) \geq 0,\ R_c(0) \geq 0, B_c(0) \geq 0.$

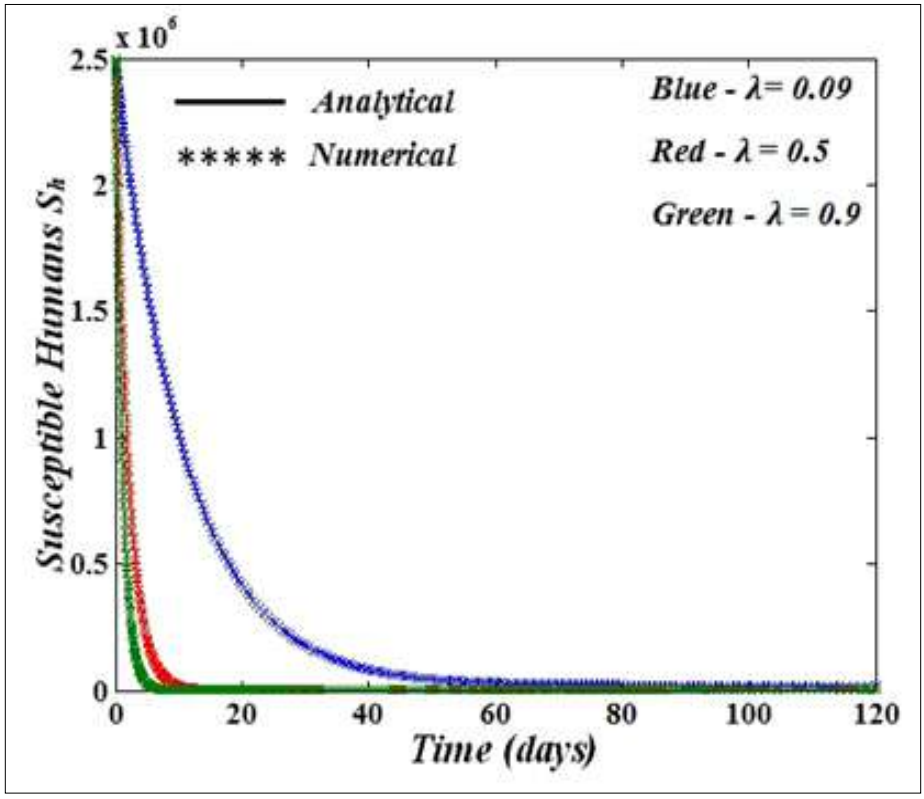

Figure 1.Variation in Number of Susceptible Humans with Change in Bacteria Contact with Humans (λ)

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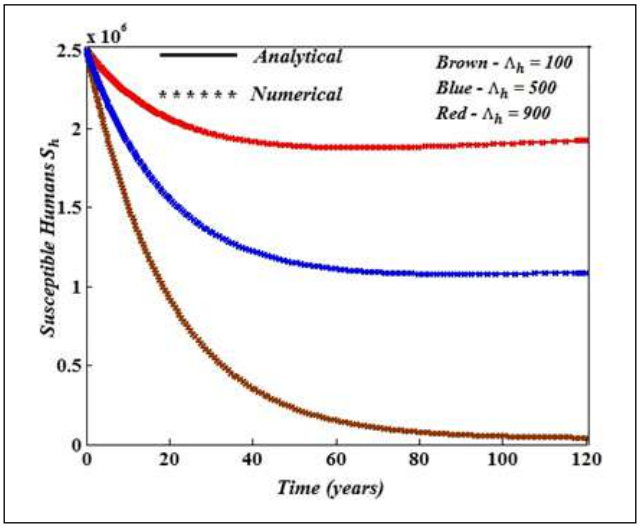

Figure 2.Variation in Number of Susceptible Humans with Change in Human Birth Rate (Λ_h)

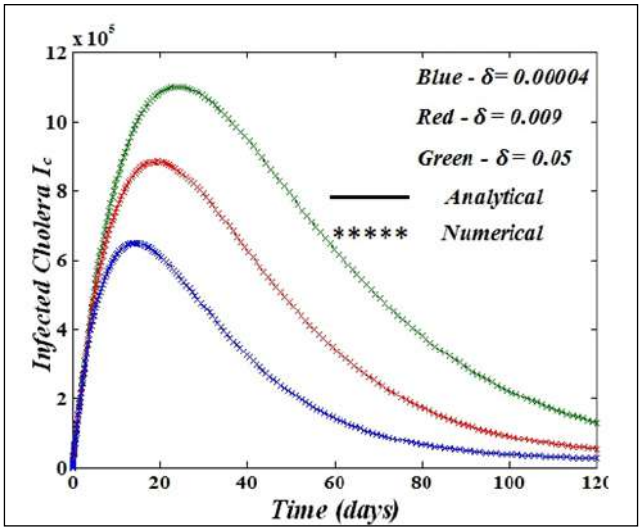

Figure 4.Variation in Number of Cholera-infected Individuals with Change in Recovery Rate of Cholera-infected Individuals (δ)

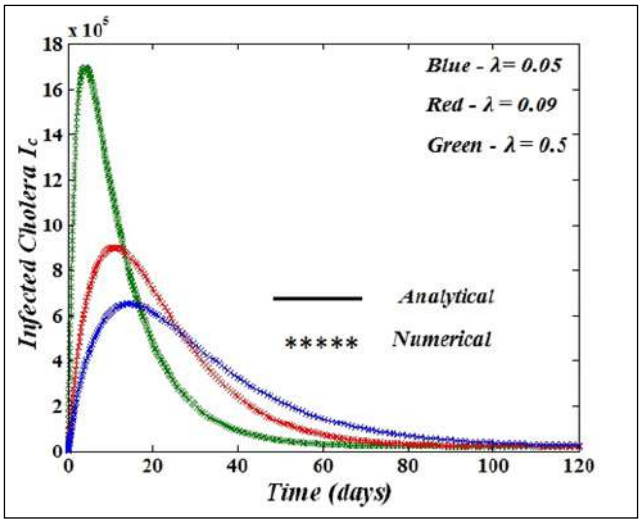

Figure 5.Variation in Number of Cholera-infected Individuals with Change in Bacteria Contact Rate with Humans (λ)

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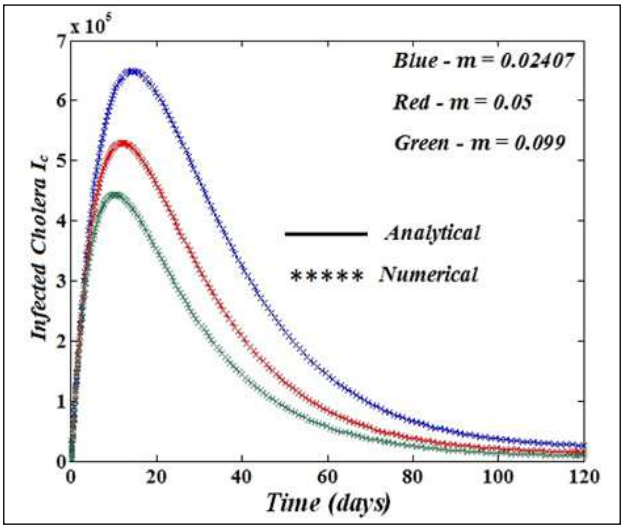
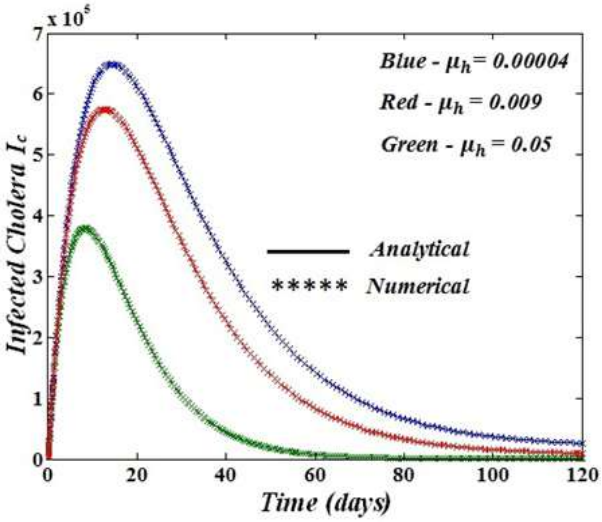


Figures 6.Variation in Number of Cholera-infected Individuals with Change in Cholera-related Deaths (m)



Figures 7.Variation in Number of Cholera-infected Individuals with Change in Values of Natural Death Rate in Humans (μh)

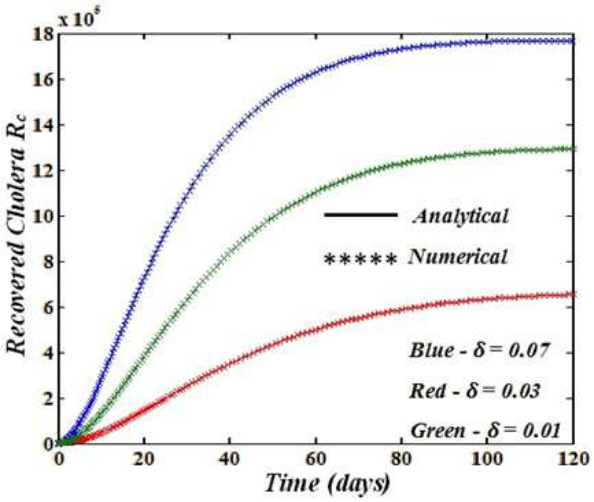


Figure 8.Variation in Number of Recovered Cholera Cases with Change in Recovery Rate of Cholera-infected Individuals (δ)

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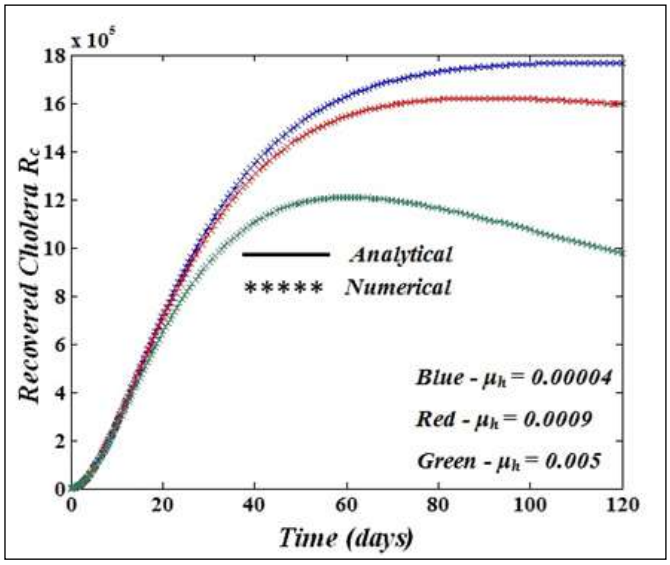

Figure 9.Variation in Number of Recovered Cholera Cases with Change in Natural Death Rate in Humans (*μ*h)

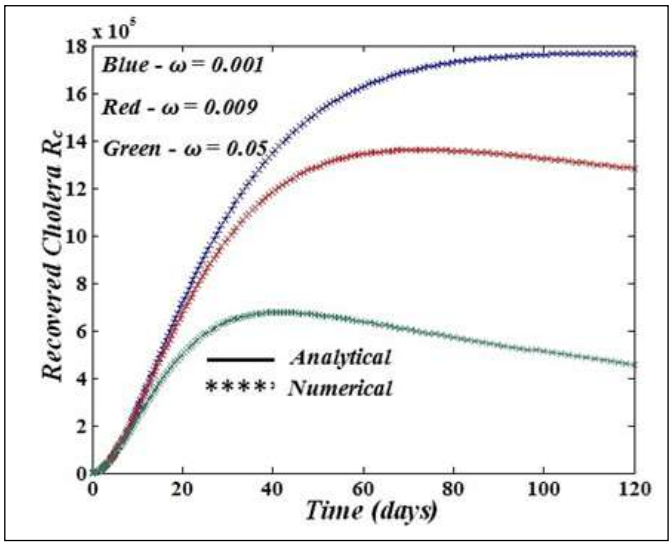

Figure 10.Variation in Number of Recovered Cholera Cases with Change in Cholera Immunity Waning Rate (ω)

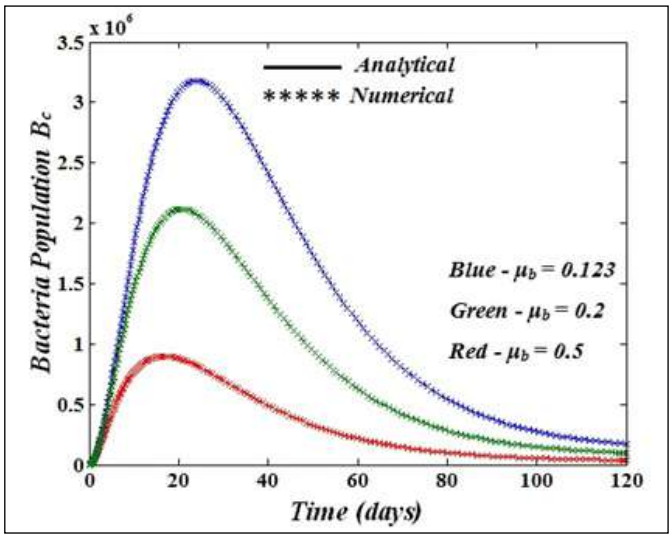

Figure 11.Variation in Bacteria Population with Change in Bacteria Mortality Rate (*μ*b)

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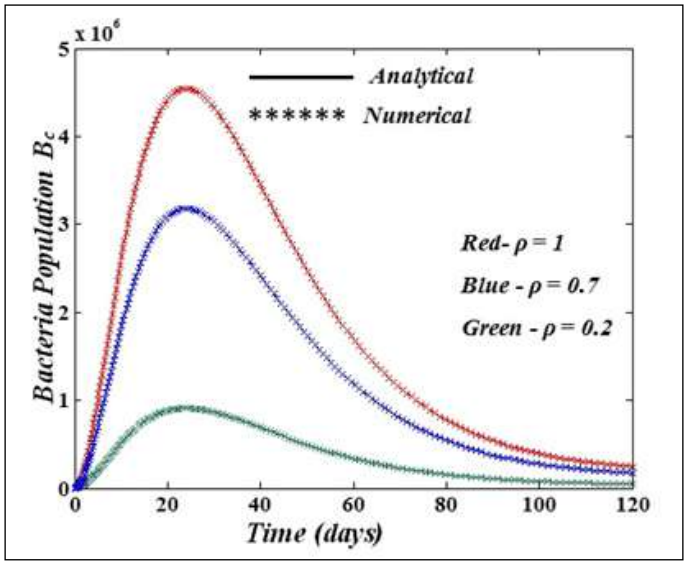

Figure 12.Variation in Bacteria Population with Change in Cholera-infected Contribution to the Aquatic (ρ)

Conclusion Derived from the Cholera Model

Figures 1 and 2 show the analytical and numerical simulation comparison of the parameters λ and Λ_h. By increasing the value of λ (0.09, 0.5, and 0.9), the number of susceptible humans was found to decrease. As shown in Figure 2, increasing the values of Λ_h led to an increase in the number of susceptible humans. Similarly, susceptible humans increased in number with an increase in cholera immunity waning rate (0.001, 0.009, and 0.05) (Figure 3). In Figure 4, it is shown that an increase in δ (0.00004, 0.009, and 0.05) led to an increase in the number of cholera-infected individuals. As shown in Figure 5, the number of cholera-infected individuals reached a high level when the value of λ was changed to 0.5.

As shown in Figures 6 and 7, the number of cholera-infected individuals decreased when the cholera-related death rate increased from 0.02407 to 0.099 and when the natural death rate in humans (μ_h) increased from 0.00004 to 0.05. Figures 8–10 show the variation of the number of recovered cholera cases with changes in the recovery rate of cholera-infected individuals (δ), natural death rate in humans (μ_h), and change in cholera immunity waning rate (ω), respectively. Figures 11 and 12 reveal the changes in the bacteria population with changes in bacteria mortality rate (μ_b) and cholera-infected contribution to the aquatic (ρ), respectively.

Malaria Model

The numerical computation of the systematic Equations 9–13 using the fourth-order Runge–Kutta method with the following parameters was used to see the dynamic behaviour of the model system:[14]

$\Lambda_h = 0.001, k = 0.01, \alpha = 0.001369, \beta_h = 0.034, \beta_v = 0.09,$
$\mu_h = 0.00004, \emptyset = 0.01, \Lambda_v = 0.01, \mu_v = 0.06667$

with the initial condition.

$S_h(0) \geq 0, I_m(0) \geq 0, R_m(0) \geq 0, S_v(0) \geq 0, I_v(0) \geq 0.$

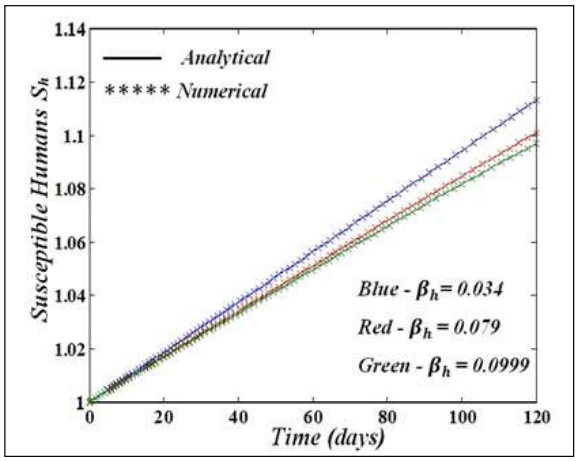

Figure 13.Variation in Number of Susceptible Humans with Change in Probability of Humans getting Infected (β_h)

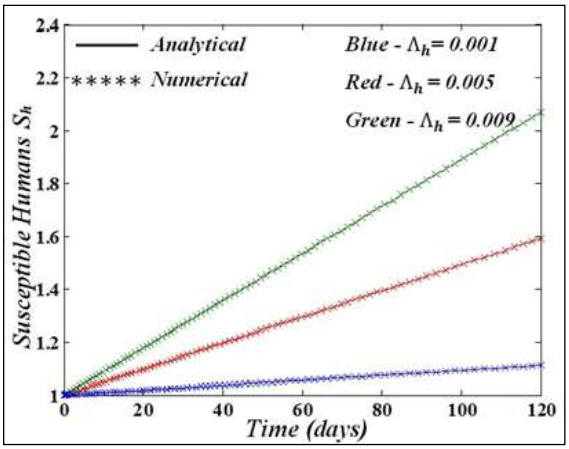

Figure 14.Variation in Number of Susceptible Humans with Change in Human Birth Rate (Λ_h)

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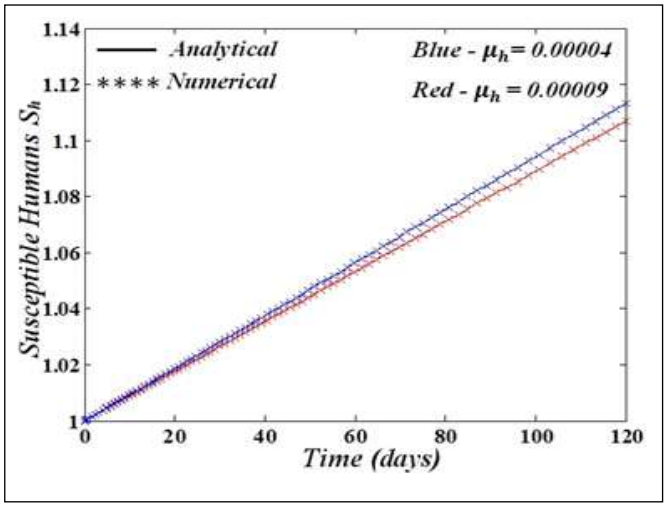

Figure 15.Variation in Number of Susceptible Humans with Change in Natural Death Rate in Humans (μ_h)

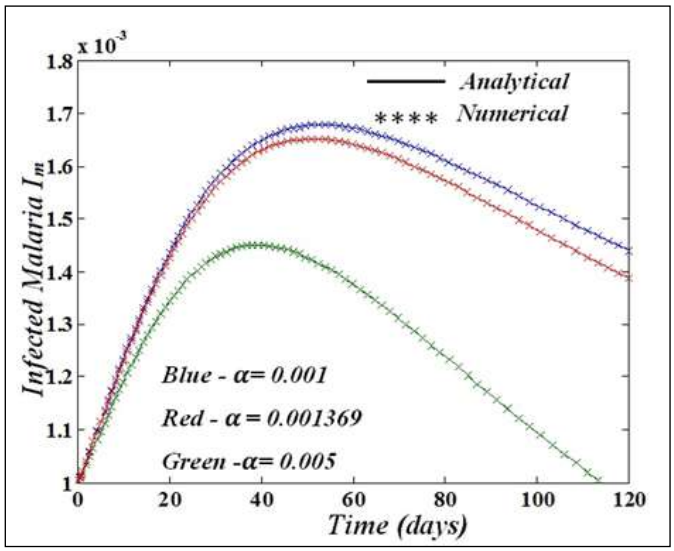

Figure 16.Variation in Number of Malaria-infected Individuals with Change in Recovery Rate of Malaria-infected Individuals ((α))

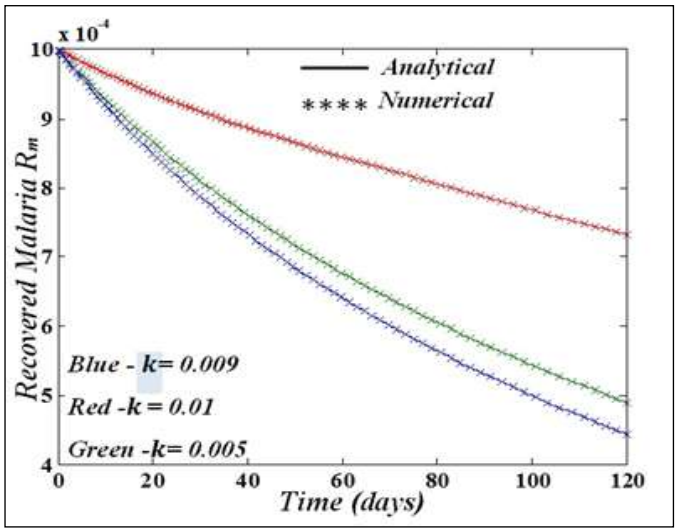

Figure 17.Variation in Number of Malaria-infected Individuals with Change in Natural Death Rate in Humans (μh)

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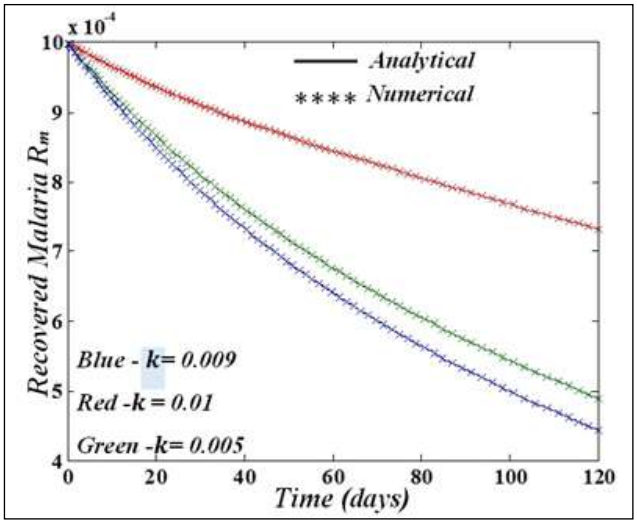

Figure 18.Variation in Number of Recovered Malaria Cases with Change in Malaria Immunity Waning Rate (k)

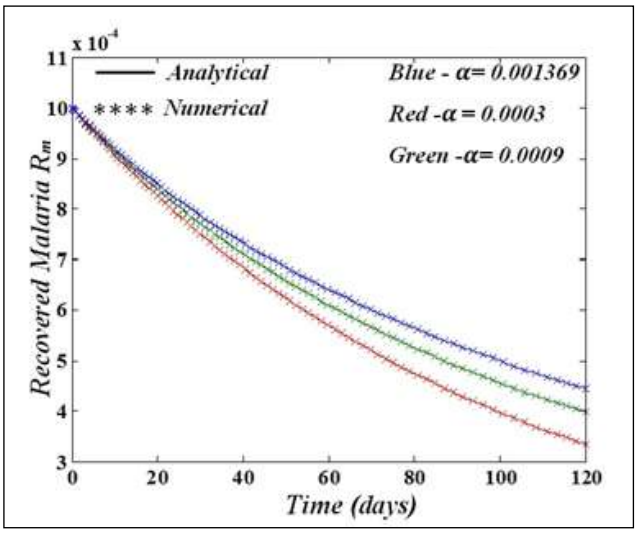

Figure 19.Variation in Number of Recovered Malaria Cases with Change in Recovery Rate of Malaria-infected Individuals (α)

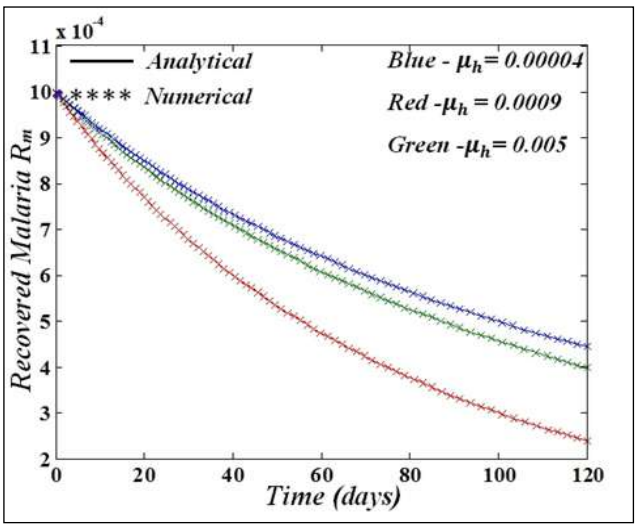

Figure 20.Variation in Number of Recovered Malaria Cases with Change in Natural Death Rate in Humans (μh)

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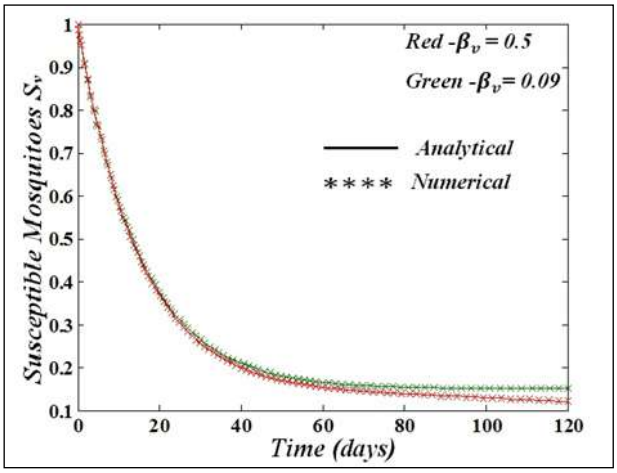

Figure 21.Variation in Number of Susceptible Mosquitoes with Change in Probability of Mosquitoes Getting Infected (β_v)

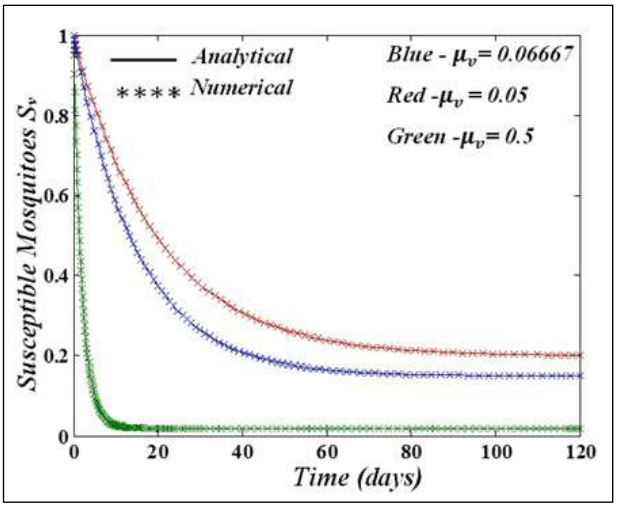

Figure 22.Variation in Number of Susceptible Mosquitoes with Change in Natural Death Rate in Mosquitoes (μv)

Conclusion Derived from the Malaria Model

Figures 13–15 show the change in the number of susceptible humans with changes in the value of β_h (0.034, 0.079, and 0.0999), Λh (0.001, 0.005, and 0.009), and μ_h (0.00004 and 0.00009), respectively.

Figures 16 and 17 indicate that the numbers of malaria-infected patients changed with respect to the recovery rate of malaria-infected individuals (α) and the natural death rate in humans (μ_h), respectively. Figures 18–20 show the recovery rate of malaria with respect to the malaria immunity waning rate (k), recovery rate of malaria-infected individuals (α) and natural death rate in humans (μ_h), respectively. Figures 21 and 22 explain the variation of susceptible mosquitoes with respect to the probability of mosquitoes getting infected (β_v) and the natural death rate in mosquitoes (μ_v).

Conclusion

A comprehensive mathematical analysis of the Malaria Cholera model using HPM has been presented in this research. The study is extended to the discussion of optimal control strategies for the Malaria Cholera model by presenting various parameter variation graphs. Both the linear and non-linear differential equations inherent in the Malaria Cholera model were systematically solved utilising both numerical methods and analytical approaches. Employing the MATLAB ode45 function and the generated numerical results demonstrates a notable consistency with the analytical solutions obtained through HPM. This convergence reinforces the reliability and validity of our mathematical analysis in understanding the Malaria Cholera model.

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