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A Semi-Analytical Study of a Non-Linear Initial Value Problem for the Lassa Fever Model

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Abstract Mathematical modelling plays a crucial role in comprehending and exercising control over Lassa fever outbreaks. The Homotopy Analysis Method (HAM) is employed to address the system of nonlinear differential equations and generate a semi-analytical solution for the Lassa disease Susceptible-Exposed-Infected-Recovered (SEIR) model. Comparing with numerical simulations, the HAM technique yielded extremely precise approximate solutions, proving its accuracy and efficiency. The numerical simulation is carried out using MATLAB programming. There is a good agreement between the numerical simulation and the approximate analytical results. We also examine the effects of changing model parameters on the different compartments, obtaining insightful knowledge on how the model behaves in various scenarios. This analysis is crucial for understanding how variations in transmission rates, recovery rates, and other key factors affect the overall dynamics of Lassa fever, thereby guiding more effective public health strategies and disease management. Moreover, it underscores the potential of the Homotopy Analysis Method (HAM) as a powerful tool for exploring epidemic models and formulating control strategies.

Keywords Epidemic model, Lassa fever, non-linear initial value problem, homotopy analysis method (HAM), numerical simulation

MSC(2010) 34A05, 34A12, 34E05, 34E10.

1. Introduction

The Lassa virus causes Lassa fever, an acute viral disease. Humans are frequently infected with Lassa virus from other animals, particularly the Natal multimammate mouse. In West Africa, which includes Guinea, Ghana, Sierra Leone, Nigeria, and Liberia, Lassa fever is comparatively prevalent [12]. Lassa fever takes two to twenty-one days to incubate. Eighty percent of infected people have minimal or no symptoms. Many virus-infected people do not show any symptoms. Headaches,

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fever, weakness, vomiting, and muscle pains are some examples of these minor symptoms [7]. 20% of people may get more serious symptoms like vomiting, chest pain, breathing issues, and bleeding gums. Hearing loss is one of the long-term consequences. Approximately 1% of Lassa virus infections are fatal.

Humans contract the Lassa virus mostly through direct contact with infected rats or by consuming food or household goods tainted with the urine or faeces of an infected Mastomys rat. Direct contact between individuals can then result in spread. As of 2023, there is no human vaccination [24]. Isolating sick individuals and reducing interaction with the rats are necessary for prevention. Food and grain should be kept in sealed containers, good personal hygiene should be promoted, and trash should be disposed of far from the house to maintain clean homes as further measures to prevent the spread of illness. Goggles, lab coats, gloves, and masks are recommended while interacting with an infected individual in order to prevent coming into contact with bodily fluids like blood [38]. Improving symptoms and treating dehydration are the goals of treatment.

Abidemi and Owolabi [2] used the least squares approach to fit the model to the Nigeria Centre for Disease Control database, which contained the matching cumulative number of weekly reported cases. The next-generation matrix approach is used to determine the model's fundamental reproduction number, and sensitivity analysis is performed to further identify the most sensitive parameters influencing the dynamics of disease transmission.In order to account for five time-dependent control variables, Abidemi et al. [3] extended the seven-dimensional deterministic model of LF dynamics. Using Pontryagin's maximal principle and optimal control theory, Abidemi et al. [3] were able to determine the prerequisites for the existence of an optimal control quintuple. Additionally, he conducted cost-effectiveness and efficiency analyses to identify the least expensive control strategy that can be used to stop the spread of LF in the population and to identify the most effective interventions among the group of various control strategies under consideration. Abiola et al. [4] calculated the fundamental reproduction number using the next-generation matrix approach. Additionally, Pontryagin's Maximum Principle was used to derive the associated optimality system, and the Lassa fever pandemic scenario was articulated as an optimal control issue.

In order to determine the most sensitive epidemiological parameters to lessen or mitigate the Lassa virus spread in Nigeria and beyond, Abdulhamid et al. [1] employed qualitative analysis to demonstrate the existence of backward bifurcation and the partial rank correlation coefficient. For Lassa fever, Agbi et al. [5] developed and investigated a non-linear mathematical compartmental model that incorporated human and rodent populations together with an infected environment. To determine the reproduction number, the next-generation method was applied and also he depicted rodent predation utilizing the Holling type II functional response.

Ajala et al. [6] tested and validated the solution's uniqueness using an invariant condition, and they calculated the reproduction number using the next generation matrix. Ayoade et al. [8] used numerous mathematical theorems to investigate and prove the model's validity. A threshold for the eradication of the disease and its equilibrium were determined. In order to determine the required and sufficient criteria for the model's equilibria to be stable both locally and globally, the stability was examined. Additionally, sensitivity analysis was performed to examine the relative contributions of different parameters to the treatment and spread of Lassa disease.

For Lassa fever, Ayoade et al. [9] determined the endemic and disease-free equilibria by equilibrium analysis. The findings of Bakare et al. [10] provided a fundamental basis for organizing and creating economical plans for effective interventions aimed at eradicating Lassa disease. By determining the fundamental reproduction number, Bawa et al. [11] were able to manage the disease's dynamics of transmission and create the prerequisites for both local and global stability of the disease-free equilibrium. To determine pooled estimates of case-fatality ratios and the time between symptom onset and hospital admission, Doohan et al. [13] conducted a meta-analysis. Using a fractional Caputo derivative, Farman et al. [15] examined the global stability of the proposed system employing the Volterra-type Lyapunov function.

In the absence of Lassa fever, Hamam et al. [16] analyzed the model's stability at an equilibrium point. Sensitivity analysis was used to identify the critical factors that have a major impact on the infection's propagation. The Basic reproduction number and global stability were investigated by Ibrahim and Denes [17]. Isma'ila et al. [18] used the next-generation matrix to calculate the fundamental reproduction number, and MATLAB was used to test the model equations' stability. The Jacobian matrix approach has been used by Sarki et al. [35] to study the infection free stability. Using a vector model of a regularly driven seasonal non autonomous system of ordinary differential equations, the population dynamics of the rodent reservoir may be the reason for the rise in human Lassa virus cases as demonstrated by McKendrick et al. [25].

Model fitting and parameterization were carried out by Ojo et al. [27], [28] using the non-linear least squares approach. Each reproduction number parameter's sensitivity analysis is also covered, and the constructed model's solutions were obtained using an iterative numerical method called the six-stage, fifth-order Runge–Kutta method [27], [28]. For achieving stability, Olupitan et al. [29] used model-fitting parameters to determine the model. Oluyo and Adejumo [30] investigated the existence of equilibrium points and used the next generation matrix operator to determine the fundamental reproduction number. Centre manifold theory is used to do bifurcation analysis. Additionally, using the Jacobian matrix method, the model's local and global stability around the Lassa fever free equilibrium was examined.

By adding a number of control intervention strategies, including treatment, isolation, external protection, and rodent control, Onah et al. [31] extended the model. The extended model was examined and contrasted with the basic model using a numerical simulation approach and suitable qualitative analysis. The theory of differential equations is used to qualitatively examine the suggested model, and Peter et al. [32] used a next-generation matrix technique to determine the threshold quantity that indicates the dominating eigenvalue. Ramzan et al. [33] used empirical data to analyze the model's accuracy and assess the infection-free equilibrium point's stability. Also, sensitivity analysis is used to find important factors affecting the dynamics of transmissions [33].

Ogwuche et al. [26] converted the deterministic model into a system of stochastic differential equations (SDEs) and employed the Milstein method to obtain a numerical solution. It was found that none of them explicitly present the approximate analytical expression for the model.The Ogwuche et al. [26] research serves as our inspiration, thus we start by solving the model to produce approximate analytical solutions. When the results obtained from the Homotopy analysis method are compared to other numerical results, an explicit response is obtained that is very useful

for assessing the epidemic model based on disease transmission and comprehending the parameters. It is not guaranteed that the Homotopy analysis method is used to find approximate analytical expressions for all first order non-linear initial value problem. But we used this method to solve the first order non-linear problem of Lassa fever and provided semi analytical expressions explicitly. Our models' results demonstrate that the best possible combinations of early hospitalization, public health education, prompt detection and treatment, and pest control measures will significantly slow the spread of the disease. Furthermore, the outbreak might be eradicated within 100 days if these suggested control methods are diligently put into practice.

This research's primary goal is to use Homotopy analysis method (HAM) to approximate the analytical solution for Lassa fever epidemiology. The approximate analytical result and numerical simulation are then compared and graphically depicted. To illustrate the impact of multiple factors, including the Recovery rate from infected to recovered state γ, Transmission rate from exposed to infected state κ, and Natural death rate μ, graphical representations are shown.

2. Mathematical formulation of the problem

Let's examine the transmission dynamics of Lassa fever model as presented by Ogwuche et al. [26] in which the classes "$S(t)$" stands for susceptible, "$E(t)$" for exposed, "$I(t)$" for infected, and "$R(t)$" for recovered. We specify a number of parameters for this model: κ stands for Transmission rate from exposed to infected state, λ is the Natural birth rate, β represents Transmission rate from susceptible to exposed state, γ is the Recovery rate from infected to recovered state and μ is the Natural death rate.The overall population size, $N(t)$ is determined by:

$$N(t) = S(t) + E(t) + I(t) + R(t). \tag{2.1}$$

Assumption of the Model [26]

i. None of the recruits are infected or immune.

ii. Birth is used to recruit members of the susceptible class.

iii. The vector is not killed by the virus; instead, they may die naturally or unintentionally, and members of each class may pass away spontaneously.

iv. Since it is believed that rats do not have Lassa fever in their body, humans are unable to infect them.

v. Since it is expected that the majority of sick persons will not travel, infected immigrants are excluded.

vi. The recovered human population is not immigrating. Natural death is the way that people depart from the population.

vii. The vector's infectious period ends when it dies, hence it never fully recovers from its infectious state.

The Model Equations

The dynamics of transmission of Lassa fever can be depicted schematically in Figure 1.

The model flowchart and the following set of ordinary differential equations describe the dynamics of disease transmission by Ogwuche et al. [26] as follows:

$$\frac{dS}{dt} = \lambda - (\beta + \mu)S, \tag{2.2}$$

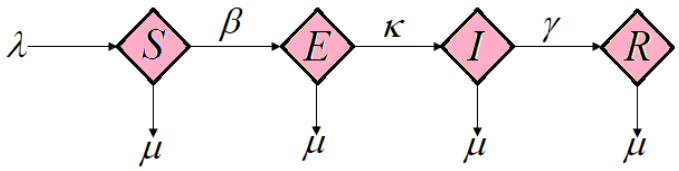

Figure 1. The Basic Lassa fever Transmission Diagram

$$\frac{dE}{dt} = \beta S - (\kappa + \mu)E, \tag{2.3}$$

$$\frac{dI}{dt} = \kappa E - (\gamma + \mu)I, \tag{2.4}$$

$$\frac{dR}{dt} = \gamma I - \mu R \tag{2.5}$$

with initial condition

$$At \;\; t = 0, S(0) = c_1 > 0, E(0) = c_2 > 0, I(0) = c_3 > 0, R(0) = c_4 > 0. \tag{2.6}$$

3. Approximate analytical solution of the equations (2.2)-(2.6) by utilizing the homotopy analysis technique

Laplace adomain decomposition procedure [14], Homotopy analysis approach [34, 37], Homotopy perturbation technique [19], and the new approach to Homotopy perturbation strategy [36] are asymptotic methods used to derive approximate analytic solutions to non-linear differential equations. The Homotopy analysis method(HAM) yields a simpler, more accurate solution than any of these other approaches. Liao [20]- [23] introduced the homotopy analysis approach, a potent approximate analytical technique for non-linear situations. This method's benefit is that it only requires one convergence control parameter within the system, making it suitable for a variety of solutions of non-linear differential equations.

The approximate analytical solutions of Eqns. (2.2)-(2.6) using Homotopy analysis method(HAM) are given below.

$$(1-p)\left[\frac{dS}{dt} + (\beta+\mu)S\right] = hp\left[\frac{dS}{dt} - \lambda + (\beta+\mu)S\right], \tag{3.1}$$

$$(1-p)\left[\frac{dE}{dt} + (\kappa+\mu)E\right] = hp\left[\frac{dE}{dt} - \beta S + (\kappa+\mu)E\right], \tag{3.2}$$

$$(1-p)\left[\frac{dI}{dt} + (\gamma+\mu)I\right] = hp\left[\frac{dI}{dt} - \kappa E + (\gamma+\mu)I\right], \tag{3.3}$$

$$(1-p)\left[\frac{dR}{dt} + \mu R\right] = hp\left[\frac{dR}{dt} - \gamma I + \mu R\right]. \tag{3.4}$$

The approximate analytical solution to Eqns. (3.1)-(3.4) is as follows:

$$S = S_0 + pS_1 + p^2S_2 + ..., \tag{3.5}$$

$$E = E_0 + pE_1 + p^2E_2 + ..., \tag{3.6}$$

$$I = I_0 + pI_1 + p^2 I_2 + ..., \tag{3.7}$$

$$R = R_0 + pR_1 + p^2 R_2 + ... \tag{3.8}$$

For Eqns. (3.1)-(3.4), the initial approximations are given by

$$S_0(0) = c_1, \;\; E_0(0) = c_2, \;\; I_0(0) = c_3, \;\; R_0(0) = c_4, \tag{3.9}$$

$$S_i(0) = 0, \;\; E_i(0) = 0, \;\; I_i(0) = 0, \;\; R_i(0) = 0, i = 1, 2, 3, ... \tag{3.10}$$

We have to put Eqns. (3.5)-(3.8) into Eqns. (3.1)-(3.4) and compare the coefficients of the powers of p^0 and p^1 so as to arrive at the following equations.

Zeroth iterations:

$$p^0 : \frac{dS_0}{dt} + (\beta + \mu)S_0 = 0, \tag{3.11}$$

$$p^0 : \frac{dE_0}{dt} + (\kappa + \mu)E_0 = 0, \tag{3.12}$$

$$p^0 : \frac{dI_0}{dt} + (\gamma + \mu)I_0 = 0, \tag{3.13}$$

$$p^0 : \frac{dR_0}{dt} + \mu R_0 = 0. \tag{3.14}$$

Initial iterations:

$$p^1 : \frac{dS_1}{dt} + (\beta + \mu)S_1 - \frac{dS_0}{dt} - (\beta + \mu)S_0 - h\left(\frac{dS_0}{dt} - \lambda + (\beta + \mu)S_0\right) = 0, \tag{3.15}$$

$$p^1 : \frac{dE_1}{dt} + (\kappa + \mu)E_1 - \frac{dE_0}{dt} - (\kappa + \mu)E_0 - h\left(\frac{dE_0}{dt} - \beta S_0 + (\kappa + \mu)E_0\right) = 0, \tag{3.16}$$

$$p^1 : \frac{dI_1}{dt} + (\gamma + \mu)I_1 - \frac{dI_0}{dt} - (\gamma + \mu)I_0 - h\left(\frac{dI_0}{dt} - \kappa E_0 + (\gamma + \mu)I_0\right) = 0, \tag{3.17}$$

$$p^1 : \frac{dR_1}{dt} + \mu R_1 - \frac{dR_0}{dt} - \mu R_0 - h\left(\frac{dR_0}{dt} - \gamma I_0 + \mu R_0\right) = 0. \tag{3.18}$$

We can obtain the following results by solving Eqns. (3.11)-(3.14) using the constraints in Eqn. (3.9):

$$S_0 = c_1 \; e^{-(\beta+\mu)\,t}, \tag{3.19}$$

$$E_0 = c_2 \; e^{-(\kappa+\mu)\,t}, \tag{3.20}$$

$$I_0 = c_3 \; e^{-(\gamma+\mu)\,t}, \tag{3.21}$$

$$R_0 = c_4 \; e^{-(\mu)\,t}. \tag{3.22}$$

We can obtain the following results by solving Eqns. (3.11)-(3.14) and (3.15)-(3.18) using the constraints (3.9) and (3.10)

$$S_1 = \frac{h\,\lambda \; e^{-(\beta+\mu)\,t}}{\beta + \mu} - \frac{h\,\lambda}{\beta + \mu}, \tag{3.23}$$

$$E_1 = \frac{h\,\beta\,c_1 \; e^{-(\kappa+\mu)\,t}}{\kappa - \beta} - \frac{h\,\beta\,c_1\,e^{-(\beta+\mu)\,t}}{\kappa - \beta}, \tag{3.24}$$

$$I_1 = \frac{h\,\kappa\,c_2\;e^{-(\gamma+\mu)\,t}}{\gamma-\kappa} - \frac{h\,\kappa\,c_2\;e^{-(\kappa+\mu)\,t}}{\gamma-\kappa}, \tag{3.25}$$

$$R_1 = -h\;c_3\;e^{-(\mu)\,t} + h\;c_3\;e^{-(\gamma+\mu)\,t}. \tag{3.26}$$

According to HAM technique, we have

$$S = \lim_{p\to 1} S(t) = S_0 + S_1, \tag{3.27}$$

$$E = \lim_{p\to 1} E(t) = E_0 + E_1, \tag{3.28}$$

$$I = \lim_{p\to 1} I(t) = I_0 + I_1, \tag{3.29}$$

$$R = \lim_{p\to 1} R(t) = R_0 + R_1. \tag{3.30}$$

As a result, by substituting the Eqns. (3.19)-(3.26) into the Eqns. (3.27)-(3.30), we have the following approximate analytical solutions.

$$S(t) = c_1\;e^{-(\beta+\mu)\,t} + \frac{h\,\lambda\;e^{-(\beta+\mu)\,t}}{\beta+\mu} - \frac{h\,\lambda}{\beta+\mu}, \tag{3.31}$$

$$E(t) = c_2\;e^{-(\kappa+\mu)\,t} + \frac{h\,\beta\,c_1\;e^{-(\kappa+\mu)\,t}}{\kappa-\beta} - \frac{h\,\beta\,c_1\,e^{-(\beta+\mu)\,t}}{\kappa-\beta}, \tag{3.32}$$

$$I(t) = c_3\;e^{-(\gamma+\mu)\,t} + \frac{h\,\kappa\,c_2\;e^{-(\gamma+\mu)\,t}}{\gamma-\kappa} - \frac{h\,\kappa\,c_2\;e^{-(\kappa+\mu)\,t}}{\gamma-\kappa}, \tag{3.33}$$

$$R(t) = c_4\;e^{-(\mu)\,t} - h\;c_3\;e^{-(\mu)\,t} + h\;c_3\;e^{-(\gamma+\mu)\,t}. \tag{3.34}$$

4. Numerical simulation

The effectiveness of our approximate-analytical solution is demonstrated by comparing the results with numerical simulation. In MATLAB, the function graphmain 3 is utilised for Eqns. (2.2)-(2.6). The MATLAB code is given in Appendix A. There is a satisfactory agreement between the numerical simulation and our approximate analytical results.

5. Results and discussion

In this part, we discuss the graphical illustration based on the derived approximate analytical results specified in Eqns. (3.31) to (3.34). Figs 2 to 13 compares the approximate analytical results with numerical simulation using MATLAB.

Fig. 2 illustrates the total population against time for the considered epidemic model. This figure shows the Susceptible, Exposed, Infected as well as Recovered classes of population against time for some fixed parameters involved in the model. From this figure, our approximate analytical results coincide with numerical simulation with an acceptable range.

For Susceptible class: The Susceptible class $S(t)$ is plotted against time (t) (days) Figs. 3-5 using Eqn. (3.31). As shown in Figure 3, the value of Natural birth rate λ rises, and the corresponding susceptible class $S(t)$ also increases. Figures 4 and 5 show that, as the amount of Transmission rate from susceptible to exposed state β and Natural death rate μ rise, the associated susceptible class $S(t)$ falls.

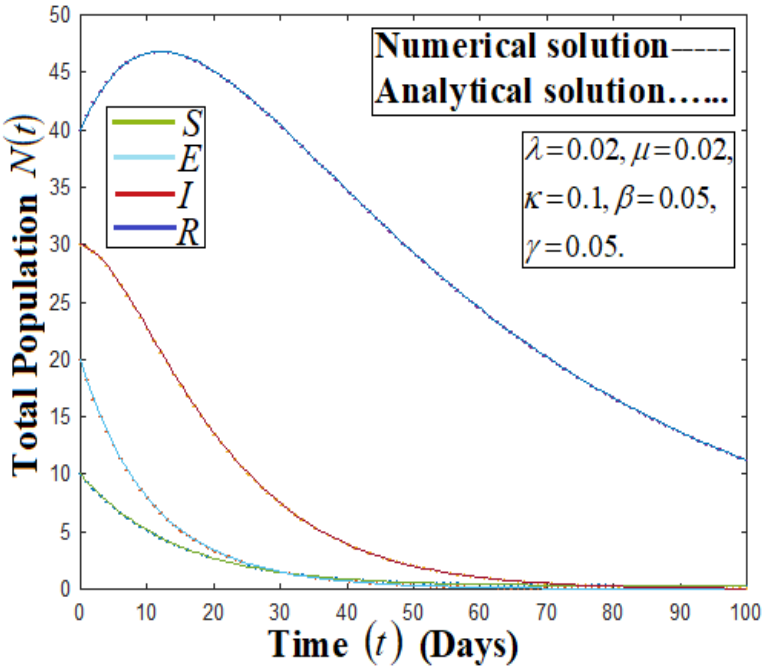

Figure 2. Total population against time for the epidemic model. The curves are plotted using the eqns. (3.31)-(3.34) for various values of dimensionless parameters.

For Exposed class: Using Eqn. (3.32), Figs. 6-8 display the exposed class $E(t)$ versus time (t) (days). Figure 6 shows that when Transmission rate from susceptible to exposed state β increases, the Exposed class $E(t)$ also rises. The rates of Transmission rate from exposed to infected state κ and the Natural death rate μ all show rising values; the matched Exposed class $E(t)$ experiences falling rates. These findings are illustrated in Figures 7 and 8.

For Infected class: Using Eqn. (3.33), Figs. 9-11 plot the infected class $I(t)$ against time (t) (days). According to Figure 9 when the Transmission rate from exposed to infected state κ rises, the infected class $I(t)$ also increases. Figs. 10 and 11 depict that when the Recovery rate from infected to recovered state γ, and Natural death rate μ all rise, the corresponding infected class $I(t)$ falls.

For Recovered class: Figs. 12 and 13 show the Recovered class $R(t)$ versus time (t) (days) by using Eqns. (3.34). The virtues of Natural death rate μ increase, the corresponding Recovered class $R(t)$drops as shown in Fig. 12. As illustrated in Figure 13, there is a positive correlation between the increases in the Recovery rate from infected to recovered state γ and the recovered class $R(t)$.

6. Conclusion

The approximate analytical results for the Susceptible $S(t)$, Exposed $E(t)$, Infected $I(t)$ and Recovered $R(t)$ of Lassa fever models were derived for all parameter values using the Homotopy analysis method. The graphical depictions for all parameters involved in the model are provided to show the effectiveness of the method. The result leads to the following: The agreement between the numerical simulation and

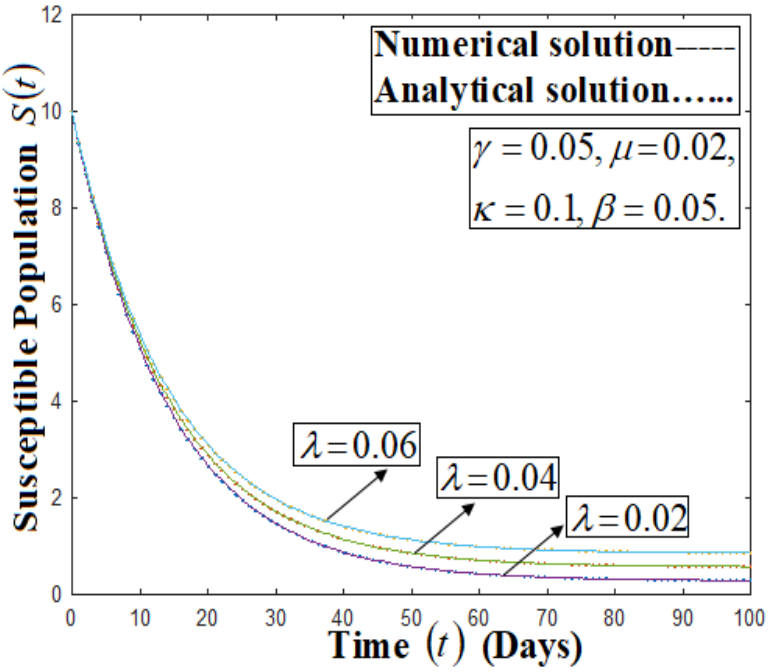

Figure 3. The impact of natural birth rate λ in Susceptible $S(t)$.

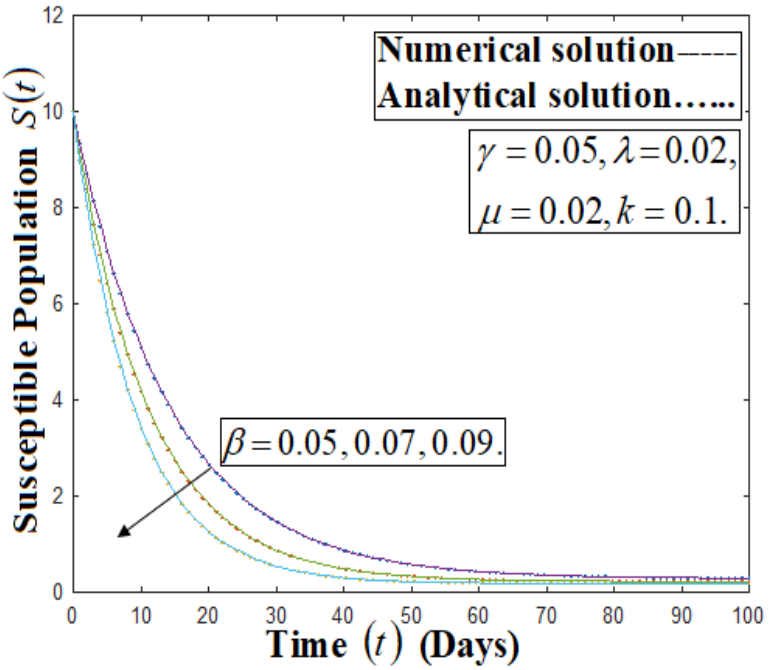

Figure 4. The influence of transmission rate from susceptible to exposed state β in susceptible $S(t)$.

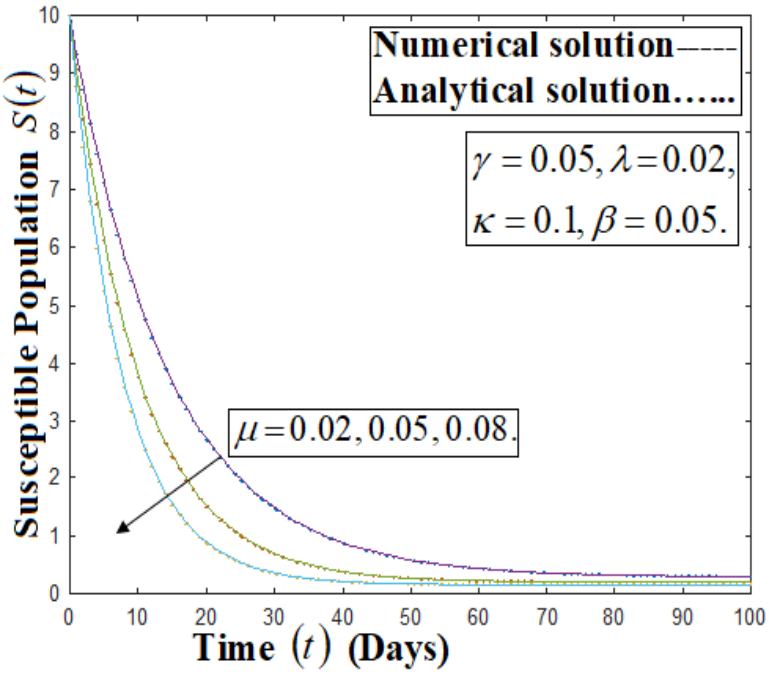

Figure 5. Effects of natural death rate μ in susceptible $S(t)$.

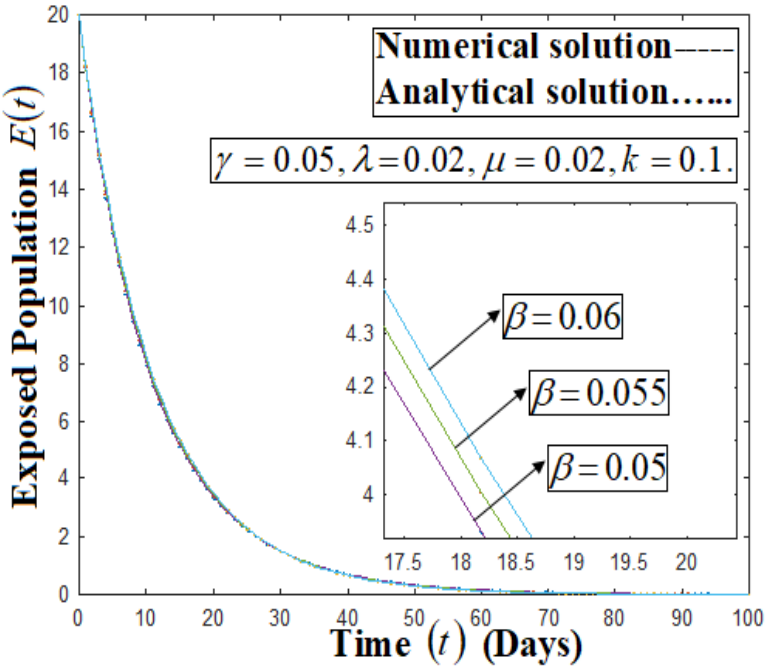

Figure 6. The influence of transmission rate from susceptible to exposed state β in exposed $E(t)$.

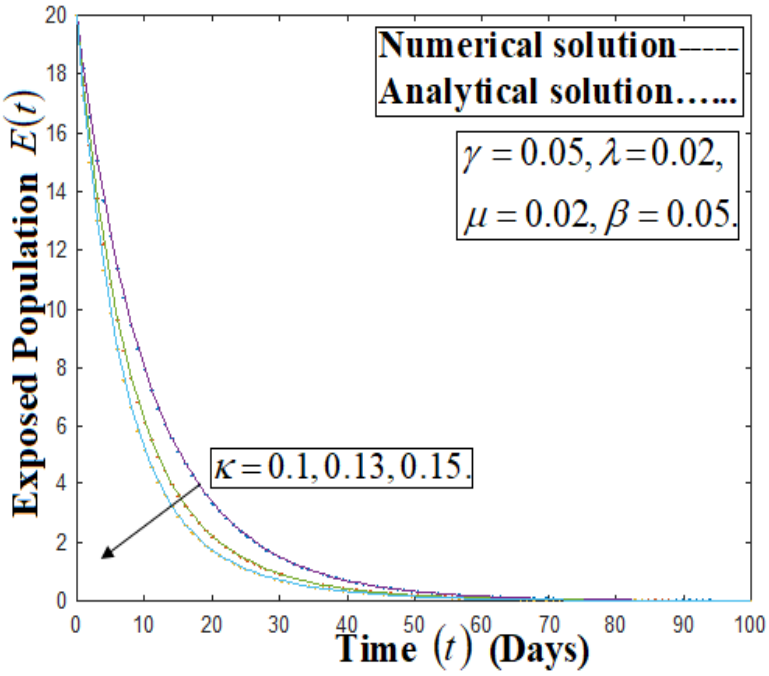

Figure 7. The effects of transmission rate from exposed to infected state κ in exposed $E(t)$.

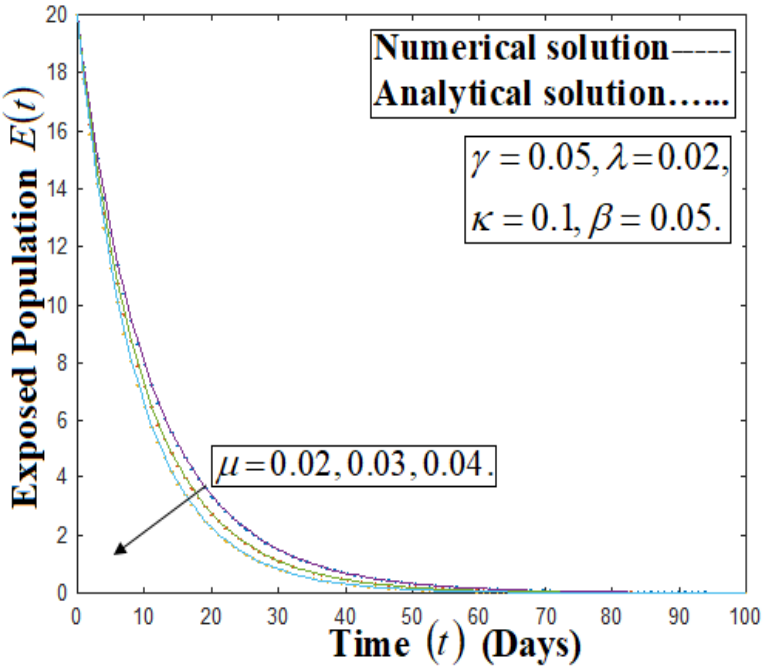

Figure 8. Effects of natural death rate μ in exposed $E(t)$.

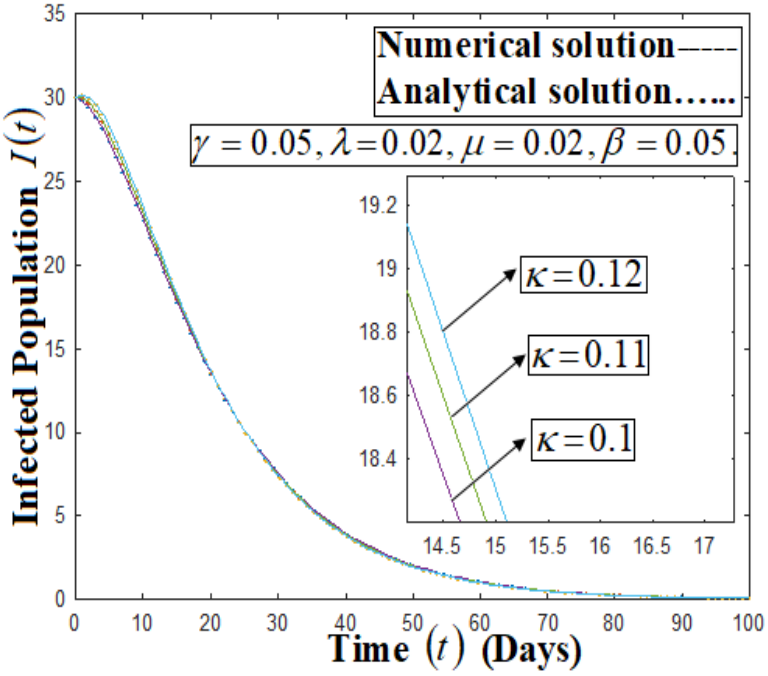

Figure 9. Impact of transmission rate from exposed to infected state κ in Infected $I(t)$.

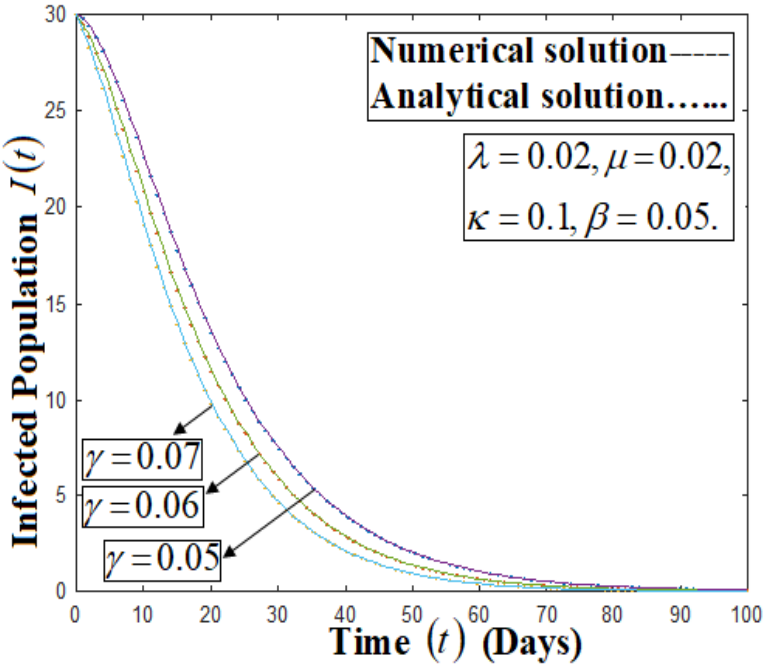

Figure 10. The influence of recovery rate from infected to recovered state γ in Infected $I(t)$.

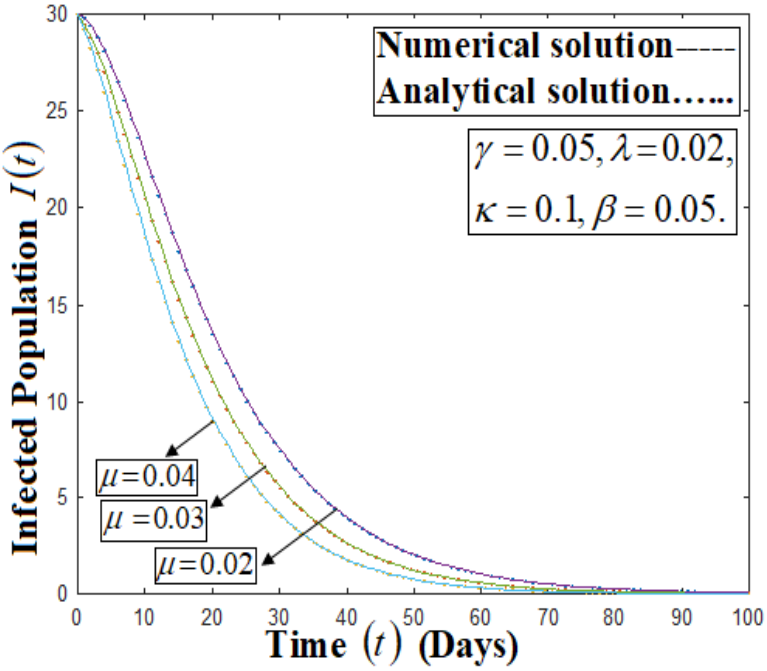

Figure 11. The effects of natural death rate μ in Infected $I(t)$.

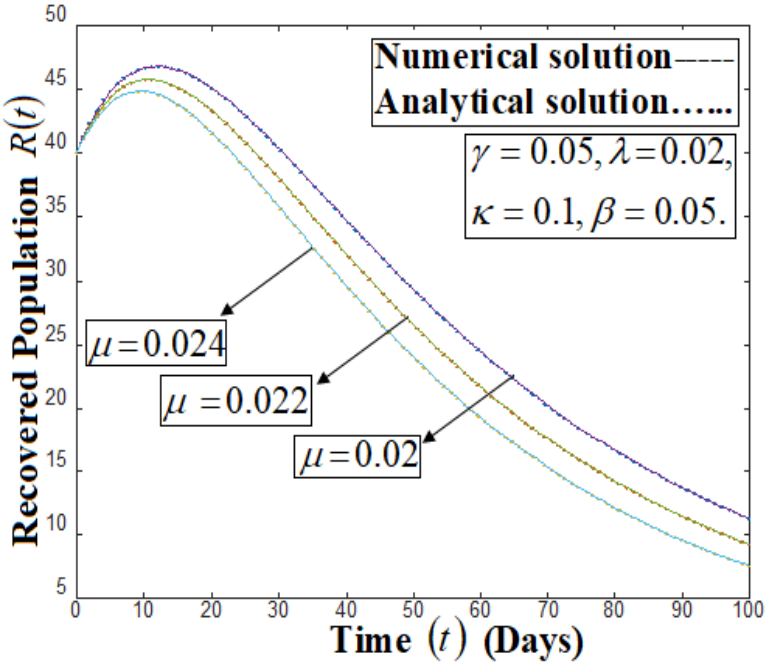

Figure 12. The influence of natural death rate μ in Recovered $R(t)$.

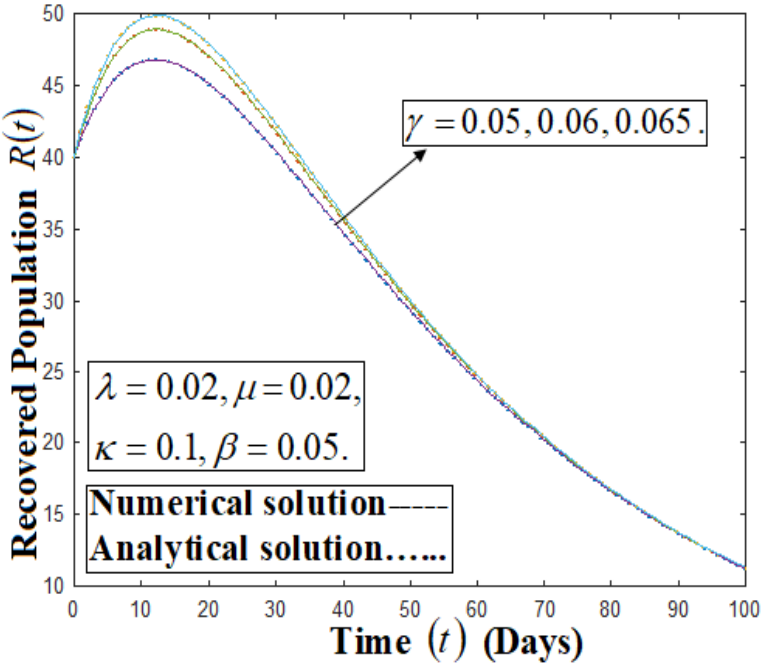

Figure 13. Variation of recovery rate from infected to recovered state γ in recovered $R(t)$.

our approximate analytical results was found to be satisfactory.

• According to our research, if we increase the transmission rate from exposed to infected state κ, we may quickly identify susceptible individuals $S(t)$ and reduce the number of exposed individuals $E(t)$ by providing guidance on viral control to assist our community in overcoming the infected state $I(t)$.

• Infected class $I(t)$ decreases by raising the recovery rate from infected to recovered state γ in the infected population.

• We propose that in order to raise the recovered population $R(t)$, the recovery rate from infected to recovered state γ should be increased.

•By increasing the transmission rate from exposed to infected state people κ, the exposed people $E(t)$ is identified completely as an infected people. This impact causes infected population $I(t)$ to increase.

•By raising the rate of recovery from infected to recovered individuals γ, it will be possible to detect infections early and help people recover quickly. Consequently, the population that has recovered $R(t)$ grows.

•In a critical scenario, the rate of transmission from a susceptible to an exposed state β is a significant factor in reducing the number of susceptible individuals $S(t)$. Through the identification of susceptible individuals $S(t)$, the transmission rate from susceptible to exposed β condition is increased, hence boosting the exposed population $E(t)$.

7. Appendices

Appendix A:
MATLAB program to find the numerical simulation of Eqns. (2.2)-(2.6)

```
functiongraphmain3
options = odeset('RelTol', 1e − 6,' Stats',' on');
Xo = [10, 20, 30, 40];
tspan = [0, 100]; ss
tic
[t, X] = ode45(@TestFunction, tspan, Xo, options);
toc
figure
holdon
plot(t, X(:, 1),' −')
plot(t, X(:, 2),' −')
plot(t, X(:, 3),' −')
plot(t, X(:, 4),' −')
legend('S',' E',' I',' R')
ylabel('Population')
xlabel('Time')
return
function[dx_dt] = TestFunction( , x)
lambda = 0.02; mu = 0.02; beta = 0.05; kappa = 0.1; gamma = 0.05;
dx_dt(1) = (lambda) − ((beta + mu) ∗ x(1));
dx_dt(2) = ((beta) ∗ x(1)) − ((kappa + mu) ∗ x(2));
dx_dt(3) = ((kappa) ∗ x(2)) − ((gamma + mu) ∗ x(3));
dx_dt(4) = ((gamma) ∗ x(3)) − ((mu) ∗ x(4));
dx_dt = dx_dt';
return
```

Appendix B: Nomenclature

Symbol	Meaning	Values(per day)
$N(t)$	Total population	
$S(t)$	Susceptible population	10(assumed)
$E(t)$	Exposed population	20(assumed)
$I(t)$	Infected population	30(assumed)
$R(t)$	Recovered population	40(assumed)
κ	Transmission rate from exposed to infected state	0.1
λ	Natural birth rate	0.02
β	Transmission rate from susceptible to exposed state	0.05
γ	Recovery rate from infected to recovered state	0.05
μ	Natural death rate	0.02

Conflict of interests

The authors declare that there is no conflict of interests.

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