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Research Article

A Semi-Analytical Study on Non-Linear Differential Equations in Typhoid Fever Disease

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Abstract. Typhoid infection dynamics is proposed in this work. The homotopy analysis method is used to solve the relevant equations, producing the approximate analytical solutions for the four compartments, such as Susceptible (S), Exposed (E), Infected (I) and Recovered (R). The numerical simulation is utilised using a MATLAB programme. In addition, the problem's numerical simulation is provided. A comparison between the numerical simulation and the analytical solution reveals excellent agreement. A number of other parameters are also discussed and graphically represented, such as the rate of innate dying ψ, the rate of human recruitment (birth) φ, the rate of disease interaction α, the rate of unprotected symptoms τ, the rate of infectious recovery θ, the rate at which humans who have recovered lose temporary immunity σ,and the total number of people who die from illness δin the compartment of Susceptible (S), Exposed (E), Infected (I) and Recovered (R). The homotopy analysis technique is employed to solve SVEIR, SEIR, SIR, and SVEIHR.

Keywords. Epidemic model, Typhoid infection, Homotopy Analysis Method (HAM), Numerical simulation, Non-linear initial value problem, Susceptible-Vaccinated-Exposed-Recovered (SVEIR), Susceptible-Exposed Infected-Recovered (SEIR), Susceptible-Infected-Recovered (SIR), Susceptible-Vaccinated-Exposed-Infected-Hospitalized-Recovered (SVEIHR)

Mathematics Subject Classification (2020). 34A05, 34A12, 34E05, 34E10

1. Introduction

Salmonella enterica serotype Typhi, commonly known as Salmonella typhi, is the pathogen responsible for typhoid fever, or simply typhoid. The symptoms can range in severity from mild to severe and often manifest six to thirty days following exposure. A rash with patches, the colour of roses appears on certain persons. Typhoid fever, commonly known as enteric fever, is brought on by the Salmonella bacteria. Wherever there is a low prevalence of the bacteria, typhoid fever is uncommon. Furthermore, the complete eradication of microorganisms is unusual in places where human waste disposal was regulated and water is treated (Ivanoff *et al.* [12]). In the United States, where typhoid disease is rare. The two regions with the highest incidence of cases or recurrent outbreaks are South Asia and Africa. It is extremely dangerous to the public's health in regions in which it is more widespread, especially for young people (Butler [6]).

The bacteria that cause typhoid fever are present in food and beverages. Additionally, intimate contact with a person who contains the salmonella bacteria might result in typhoid fever. One of the signs is a high fever, headache, constipation, diarrhoea, or abdominal pain (Nsutebu *et al.* [21]). Once antibiotic therapy is started, most people with typhoid fever recover within a week because the medication kills the bacterium. However, the risk of dying from complications related to typhoid fever is quite low in the absence of therapy (Schemmer [26]). Immunisations against typhoid disease may offer some defence (Bhan *et al.* [4]). However, they are unable to provide total defence against diseases caused by various strains of salmonella. Immunisations may lower the risk of typhoid illness.

To examine the typhoid disease model's worldwide stability, Mushayabasa *et al.* [19] compute the fundamental reproduction number. The impact of successful treatment and the probability of a newly infected individual being a carrier were also explored by numerical simulations. Rafiq *et al.* [24] using the notion of Lyapunov functions, global stabilisations has been conducted for the ebola epidemic model at both levels. They use both the Runge–Kutta technique of order 4 (RK4) and a non-standard finite difference (NSFD) scheme for the susceptible–exposed–infected–recovered (SEIR) model. Using above two method, Ahmad *et al.* [2] confirm theoretical conclusions with numerical simulations. Also, he concluded that the Ebola virus can be eradicated if people choose to voluntarily be vaccinated and if focused public education campaigns were launched at different coverage levels.

Ahmad *et al.* [3] proven the global stability of both equilibria by applying LaSalle's invariance assumption from the Lyapunov function theory. The (NSFD) and (RK4) method are two well-known numerical approaches that were used to solve the system of ODEs. These methods also serve to validate their theoretical results. Nazir *et al.* [20] using Khalil's conformable transform, memory effects were found for each example (States that are endemic and free of sickness) and designed to improve the accuracy of future forecasts. As you can see from the figures, the problem remained a constant in both the endemic and disease-free regimes.

In the sensitivity study of Tilahun *et al.* [27] for both endemic and disease-free equilibria, asymptotic stability criteria were found both locally and globally. A forward trans critical split that results from the model was viewed. An optimal control problem with three control techniques has been developed using the Pontryagin maximal concept: vaccination, good hygiene, and sanitation as a preventive measure. The stability study of the model was conducted in order to identify the parameters that promote the disease's spread within a particular community. In addition, an increase in protection results in a lower prevalence of disease in a community, according to numerical simulation of the model has been carried out by Nthiiri *et al.* [22].

Adeboye and Haruna [1] developed and studied a mathematical framework of co-infection between typhoid and malaria that addresses the management of both diseases' simultaneous spread. The goal of Brauer and Castillo-Chávez [5] was to encourage biological science students to approach science with a mathematical perspective. Chamuchi *et al.* [7] looked on the effectiveness of control measures to reduce the number of carriers of the typhoid virus in Kisii town. NSFD scheme is developed by Cui *et al.* [8] for a SIR epidemic model of paediatric disease that employs a constant technique. Using a NSFD method, the continuous SIR epidemic model is numerically discretised. The denominator function is selected so that the scheme upholds the population conservation law (Darti *et al.* [9]). Study was done on the dynamic behaviour of two discrete epidemiological models for diseases with nonlinear incidence rates (Hattaf *et al.* [11]). Euler methods are applied both forward and backward to derive both discrete models from the continuous case. The stability behaviour of the endemic equilibrium and the disease-free equilibrium were examined in relation to the two distinct discretization's.

Using the SVEIR epidemic model, Jia and Li [13] demonstrated the globally asymptotical stability of the disease-free equilibrium. The non-linear incidence rate was shown by the Liapunov-Lasalle invariant theorem. The endemic equilibrium's local asymptotic stability was established using the Hurwitz criterion. The analysis of the local stability of the disease-free equilibrium point took into account the Jacobian matrix, and the Brauer and Castillo-Chávez method was employed to ascertain the global stability of the disease-free equilibrium point (Karunditu *et al.* [14]). The global stability of the endemic equilibrium point was examined using the Lyapunov function. The typhoid fever epidemic's propagation patterns were examined by Musa *et al.* [18]. The model assesses the impact of public health education programs on the pathophysiology of typhoid fever, which can cause serious outbreaks, particularly in underdeveloped areas. In their evaluation of many school-based immunisation programs, Pitzer *et al.* [23] discovered that while vaccination by itself is unlikely to result in eradication, it is anticipated that vaccination will provide temporary indirect protection and lower typhoid incidence. sFor the biological models, NSFD are suggested. The presence of related discrete dynamical systems' equilibria was investigated by Rao. It was shown that equilibrium solutions will remain stable under sufficient parameter conditions.

Utilising HAM to approximate the analytical solution for the epidemiology of typhoid fever is the main objective of this study. We next compare and graphically illustrate the approximate analytical outcome and numerical simulation. To demonstrate the influence of several variables, such as therate of innate death ψ, the rate of human recruitment (birth) φ, the rate of disease interaction α, the rate of untreated symptoms τ, the rate of infectious recovery θ, the rate at which individuals who have recovered from an illness lose their temporary immunity σ, and the overall number of illness-related deaths δ, graphical illustrations are depicted.

2. Mathematical Formulation of the Problem

Let's examine the transmission dynamics of Typhoid disease model as presented by Khan *et al.* [15]. Presumably, there are four compartments within the total population $N(t)$: susceptible(S), exposed (E), infected (I), and recovered (R), i.e., $N(t) = S(t)+E(t)+I(t)+R(t)$. The following process is used by the model as follows: $S \to E \to I \to R$. Using the SEIR model, Figure 1 illustrates a fractional map of the typhoid disease transmission between exposed individual compartments:

$$\frac{dS}{dt} = \varphi + \sigma R - \alpha SI - \psi S, \tag{1}$$

$$\frac{dE}{dt} = \alpha SI - \tau E - \psi E, \tag{2}$$

$$\frac{dI}{dt} = \tau E - \tau I - \delta I - \psi I, \tag{3}$$

$$\frac{dR}{dt} = \theta I - \sigma R - \psi R, \tag{4}$$

with initial condition

$$\text{At } t = 0,\ S(0) = c_1 > 0,\ E(0) = c_2 > 0,\ I(0) = c_3 > 0,\ R(0) = c_4 > 0. \tag{5}$$

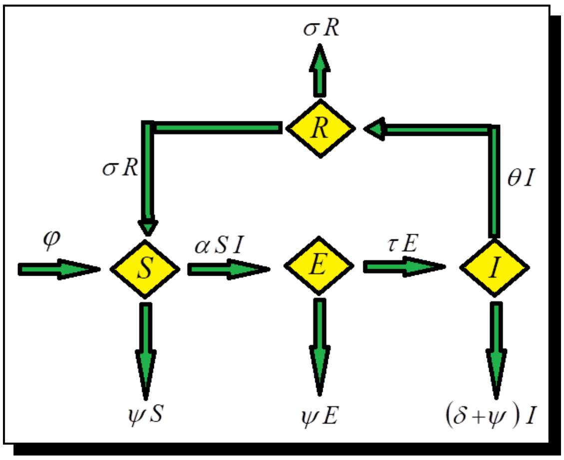


Figure 1. Diagrammatic representation of the epidemic model (Source: [15])

3. Approximate Analytical Solution of the Equations (1)-(5) by Utilising the Homotopy Analysis Technique

HAM is a non-perturbative analytical technique that has been successfully used to a broad range of scientific and engineering applications. It works by finding series solutions to non-linear differential equations. The convergence of a solution can be circumvented and modified by using the so-called convergence-control parameter, which is provided by HAM. As a result, HAM has demonstrated to be the most effective method for producing analytical solutions to non-linear differential equations. The majority of non-linear differential equations that HAM has been used to solve the unknown function and its derivatives to express the non-linearity as a polynomial. Liao introduced the homotopy analysis approach, a powerful analytical tool for non-linear problems (Liao [16, 17]). Regarding an endless power series, this approach offers an analytical solution. But evaluating this answer and deriving numerical numbers from the infinite power series are practically necessary. A finite number of terms in the *Homotopy Analysis Approach* (HAM) solution of the differential equations system were computed in order to verify its accuracy. Using the auxiliary parameter h, which is a component of the Homotopy analysis method, is a simple way to adjust and control the convergence zone of solution series. In comparison to other approaches, the HAM method is incredibly straightforward and shows great promise for solving additional non-linear equations. It is simple to expand this approach to solve all other non-linear equations.

Equations (1)-(5) can have their approximate analytical solutions using HAM

$$(1-p)\left[\frac{dS}{dt} + \psi S\right] = hp\left[\frac{dS}{dt} - \varphi - \sigma R + \alpha SI + \psi S\right], \tag{6}$$

$$(1-p)\left[\frac{dE}{dt}+(\tau+\psi)E\right]=hp\left[\frac{dE}{dt}-\alpha SI+\tau E+\psi E\right], \tag{7}$$

$$(1-p)\left[\frac{dI}{dt}+(\theta+\delta+\psi)I\right]=hp\left[\frac{dI}{dt}-\tau E+\theta I+\delta I+\psi I\right], \tag{8}$$

$$(1-p)\left[\frac{dR}{dt}+(\sigma+\psi)R\right]=hp\left[\frac{dR}{dt}-\theta I+(\sigma+\psi)R\right]. \tag{9}$$

The approximate analytical solution to equations (6)-(9) is as follows:

$$S=S_0+pS_1+p^2S_2+\dots, \tag{10}$$

$$E=E_0+pE_1+p^2E_2+\dots, \tag{11}$$

$$I=I_0+pI_1+p^2I_2+\dots, \tag{12}$$

$$R=R_0+pR_1+p^2R_2+\dots,. \tag{13}$$

For equations (6) to (9), the initial approximations are given by

$$S_0(0)=c_1,\ E_0(0)=c_2,\ I_0(0)=c_3,\ R_0(0)=c_4, \tag{14}$$

$$S_i(0)=0,\ E_i(0)=0,\ I_i(0)=0,\ R_i(0)=0. \tag{15}$$

We have to put equations (10)-(13) into equations (6)-(9) and compare the coefficients of the powers of p^0 and p^1 so as to arrive at the following equations.

Zeroth iterations:

$$p^0:\frac{dS_0}{dt}+\psi S_0=0, \tag{16}$$

$$p^0:\frac{dE_0}{dt}+(\tau+\psi)E_0=0, \tag{17}$$

$$p^0:\frac{dI_0}{dt}+(\theta+\delta+\psi)I_0=0, \tag{18}$$

$$p^0:\frac{dR_0}{dt}+(\sigma+\psi)R_0=0. \tag{19}$$

Initial iterations:

$$p^1:\frac{dS_1}{dt}+\psi S_1-\frac{dS_0}{dt}-\psi S_0-\left(h\frac{dS_0}{dt}-h\varphi-h\sigma R_0+h\alpha S_0I_0+h\psi S_0\right)=0, \tag{20}$$

$$p^1:\frac{dE_1}{dt}+(\tau+\psi)E_1-\frac{dE_0}{dt}-(\tau+\psi)E_0-\left(h\frac{dE_0}{dt}-h\alpha S_0I_0+h\tau E_0+h\psi E_0\right)=0, \tag{21}$$

$$p^1:\frac{dI_1}{dt}+(\theta+\delta+\psi)I_1-\frac{dI_0}{dt}-(\theta+\delta+\psi)I_0-\left(h\frac{dI_0}{dt}-h\tau E_0+h(\theta+\delta+\psi)I_0\right)=0, \tag{22}$$

$$p^1:\frac{dR_1}{dt}+(\sigma+\psi)R_1-\frac{dR_0}{dt}-(\sigma+\psi)R_0-\left(h\frac{dR_0}{dt}-h\theta I_0+h(\sigma+\psi)R_0\right)=0. \tag{23}$$

We can obtain the following results by solving equations (16) to (19) using the constraints in equations (14):

$$S_0=c_1e^{-\psi t}, \tag{24}$$

$$E_0=c_2e^{-(\tau+\psi)t}, \tag{25}$$

$$I_0=c_3e^{-(\theta+\delta+\psi)t}, \tag{26}$$

$$R_0=c_4e^{-(\sigma+\psi)t}, \tag{27}$$

$$S_1 = \frac{h\varphi}{\psi}e^{-\psi t} - hc_4 e^{-\psi t} - \frac{h\alpha c_1 c_3}{\theta+\delta-\psi}e^{-\psi t} - \frac{h\varphi}{\psi} + hc_4 e^{-(\sigma+\psi)t} + \frac{h\alpha c_1 c_3 e^{-(2\psi+\theta+\delta)t}}{\theta+\delta-\psi}, \tag{28}$$

$$E_1 = \frac{h\alpha c_1 c_3}{\tau-\psi-\theta-\delta}e^{-(\tau+\psi)t} - \frac{h\alpha c_1 c_3 e^{-(2\psi+\theta+\delta)t}}{\tau-\psi-\theta-\delta}, \tag{29}$$

$$I_1 = \frac{h\tau c_2 e^{-(\theta+\delta-\psi)t}}{\theta+\delta-\tau} - \frac{h\tau c_2 e^{-(\tau+\psi)t}}{\theta+\delta-\tau}, \tag{30}$$

$$R_1 = \frac{h\theta c_3 e^{-(\sigma+\psi)t}}{\sigma-\theta-\delta} - \frac{h\theta c_3 e^{-(\theta+\delta+\psi)t}}{\sigma-\theta-\delta}. \tag{31}$$

According to HAM technique, we have

$$S = \lim_{p\to 1} S(t) = S_0 + S_1, \tag{32}$$

$$E = \lim_{p\to 1} E(t) = E_0 + E_1, \tag{33}$$

$$I = \lim_{p\to 1} I(t) = I_0 + I_1, \tag{34}$$

$$R = \lim_{p\to 1} R(t) = R_0 + R_1. \tag{35}$$

As a result, by substitute the equations (24) to (31) into the equations (32) to (35), we have the following approximate analytical solutions:

$$S(t) = c_1 e^{-\psi t} + \frac{h\varphi}{\psi}e^{-\psi t} - hc_4 e^{-\psi t} - \frac{h\alpha c_1 c_3}{\theta+\delta-\psi}e^{-\psi t} - \frac{h\varphi}{\psi} + hc_4 e^{-(\sigma+\psi)t} + \frac{h\alpha c_1 c_3 e^{-(2\psi+\theta+\delta)t}}{\theta+\delta-\psi}, \tag{36}$$

$$E(t) = c_2 e^{-(\tau+\psi)t} + \frac{h\alpha c_1 c_3}{\tau-\psi-\theta-\delta}e^{-(\tau+\psi)t} - \frac{h\alpha c_1 c_3 e^{-(2\psi+\theta+\delta)t}}{\tau-\psi-\theta-\delta}, \tag{37}$$

$$I(t) = c_3 e^{-(\theta+\delta+\psi)t} + \frac{h\tau c_2 e^{-(\theta+\delta-\psi)t}}{\theta+\delta-\tau} - \frac{h\tau c_2 e^{-(\tau+\psi)t}}{\theta+\delta-\tau}, \tag{38}$$

$$R(t) = c_4 e^{-(\sigma+\psi)t} + \frac{h\theta c_3 e^{-(\sigma+\psi)t}}{\sigma-\theta-\delta} - \frac{h\theta c_3 e^{-(\theta+\delta+\psi)t}}{\sigma-\theta-\delta}. \tag{39}$$

4. Numerical Simulation

The effectiveness of our approximate-analytical solution is demonstrated by numerical simulation of non-linear differential equations. In MATLAB function graphmain3, is utilised for equations (1) to (5). We found that the numerical simulation and our approximate-analytical solution were agreed well from Figures 1-6.

5. Results and Discussion

In this section, we have discussed the graphical illustration based on the derived approximate analytical results specified in equations (36) to (39). Figure 2 to 6(d) compares the approximate analytical results with numerical simulation using MATLAB. As compared to numerical simulation, our approximate analytical findings reach a very good fit.

Figure 2 illustrates the total population against time for the considered epidemic model. This figure shows the Susceptible, Exposed, Infected as well as Recovered class 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.

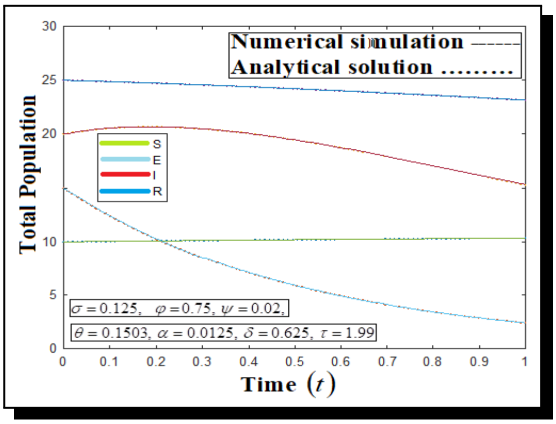
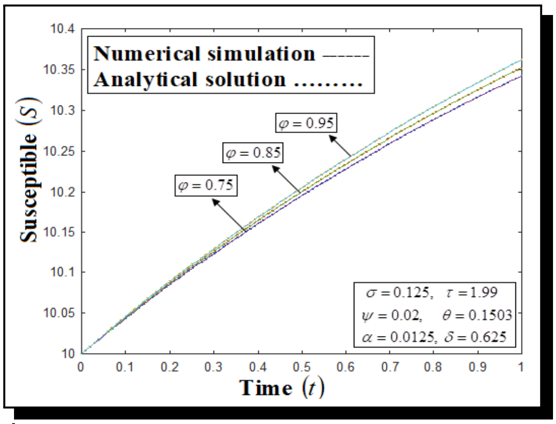
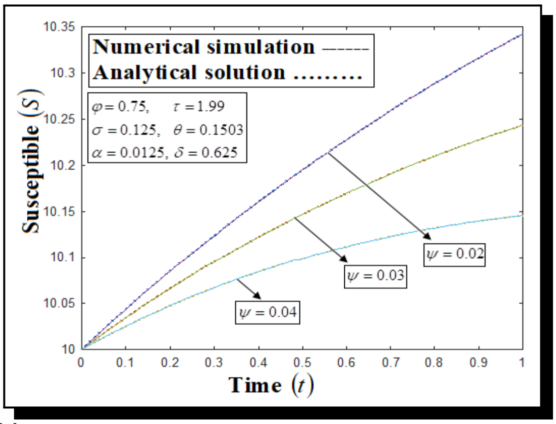


Figure 2. Total population against time for the epidemic model

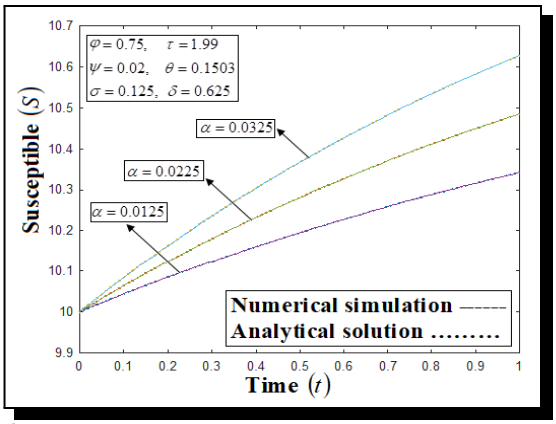
For Susceptible class. The Susceptible class $S(t)$ is plotted against time (t) (weeks) in Figures 3(a)-3(f) using equations (36). As shown in Figures 3(a), 3(c), and 3(f), the values of the rates of disease interaction α, the human recruitment (birth) φ, and the rate of transient immunity loss (lost immunity) in humans who have recovered σ are all rise, the corresponding susceptible class $S(t)$ also increases. Figures 3(b), 3(d), and 3(e) shows that, as the amount of the rates of infectious recovery θ, innate dying ψ and the number of illness-related deaths δ rise, the associated Susceptible class $S(t)$ falls.



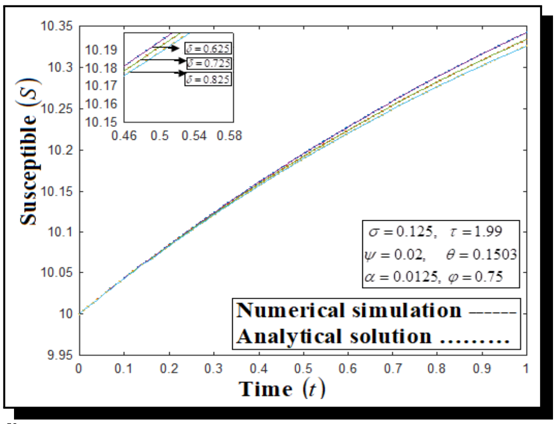
(a) The Impact of the pace of human recruiting (birth) φ in Susceptible $S(t)$



(b) The influence of the rate of innate dying ψ in Susceptible $S(t)$

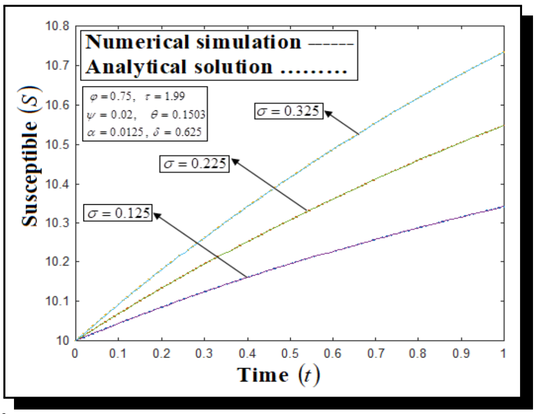


(c) The impacts of the disease interaction rate α in susceptible $S(t)$

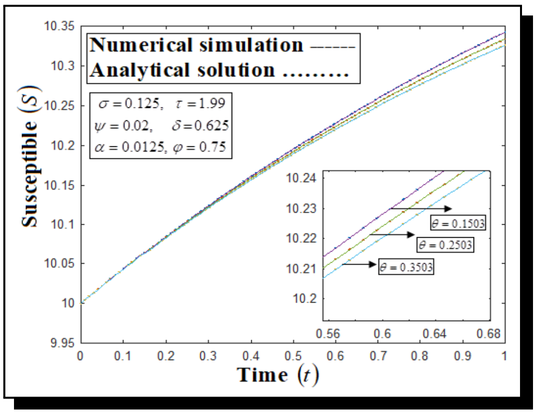


(d) Variation in the number of deaths due to sickness δ in Susceptible $S(t)$

Figure *continued*.



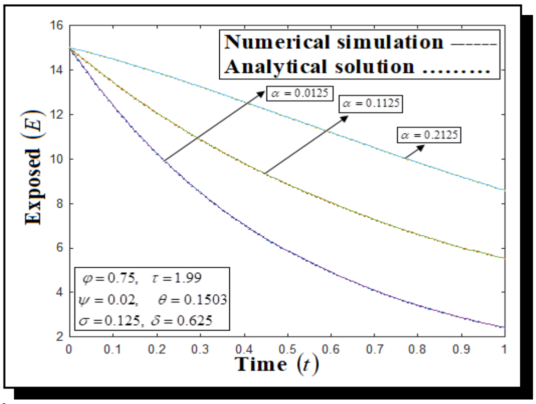
(e) Influence of the rate of infectious recovery θ in Susceptible $S(t)$



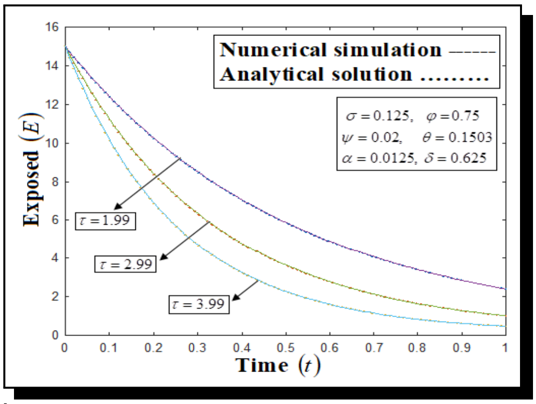
(f) Effects of the pace at which persons who have recovered lose their temporary immunity σin Susceptible $S(t)$

Figure 3

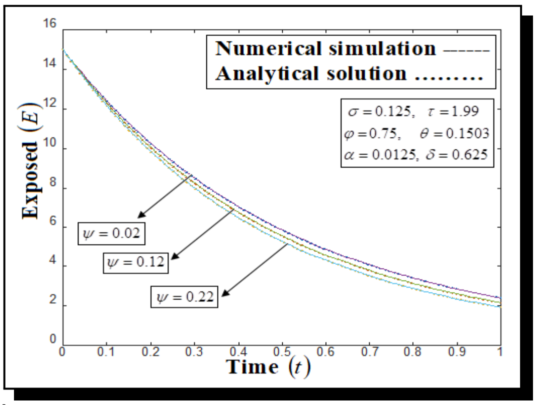
For Exposed class. Using equations (37), Figures 4(a)-4(e) display the exposed class $E(t)$ versus time (t) (weeks). According to Figure 4(a), when the rate of disease interaction increases, so does the corresponding Exposed class $E(t)$. The rates of unprotected symptoms τ, innate dying ψ, infectious recovery θ, and the number of illness-related deaths δ all show rising values; the matched Exposed class $E(t)$ experiences falling rates. These findings are illustrated in Figures 4(b)-4(e).



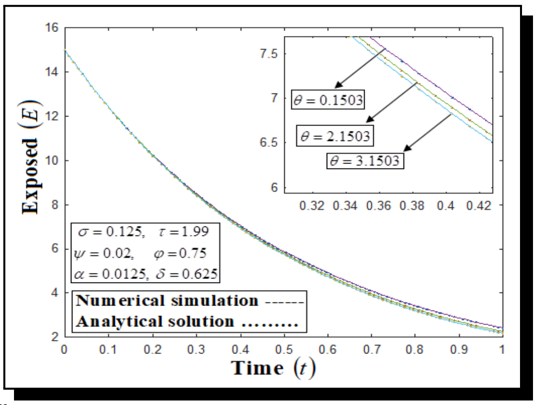
(a) The influence of the disease interaction rate α in Exposed $E(t)$



(b) The influence of the rate of innate dying ψ in Susceptible $S(t)$

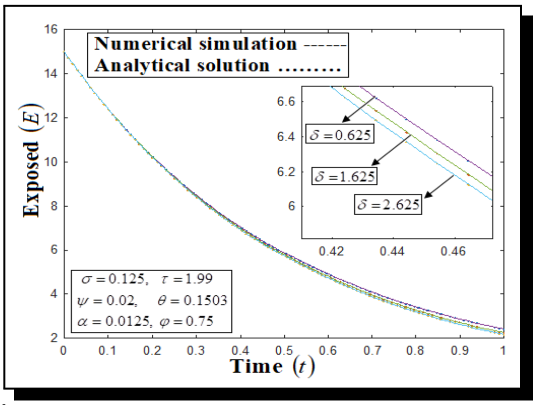


(c) Effects of the rate of innate dying ψ in Exposed $E(t)$



(d) Influence of the rate of infectious recovery θ in Exposed $E(t)$

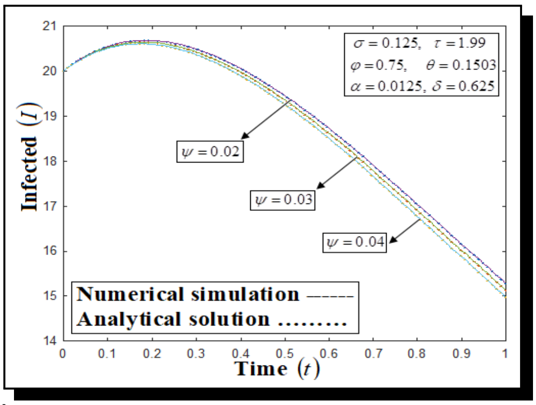
Figure *continued*.



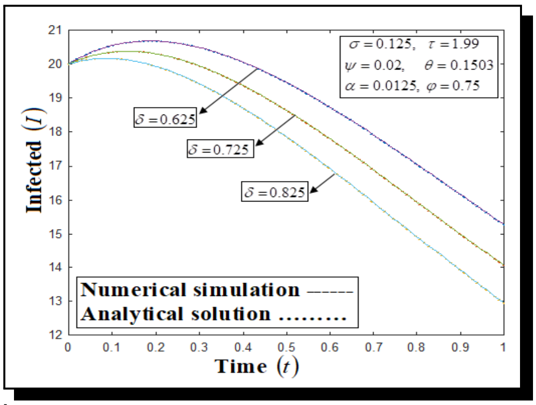
(e) The impact of the number of deaths due to sickness δ in Exposed $E(t)$

Figure 4

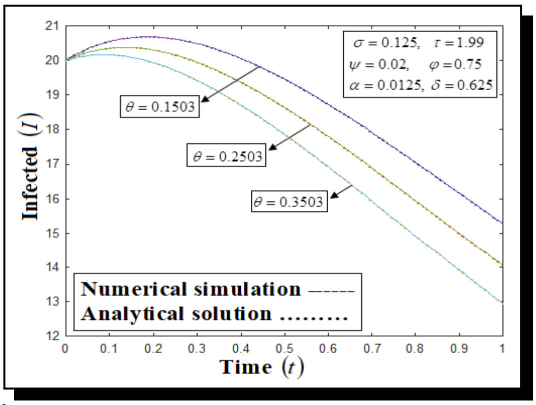
For Infected class. Using equations (38), Figures 5(a)-5(d) plots the infected class $I(t)$ against time (t) (weeks). In Figures 5(a)-5(c) depicts that when the rate of infectious recovery θ, the number of individuals who pass away from illness δ and the rate of innate dying ψ all rise, the corresponding infected class $I(t)$ get falls. Figures 5(d) illustrates how the infected class $I(t)$ rise for an increasing the rate of unprotected symptoms τ.



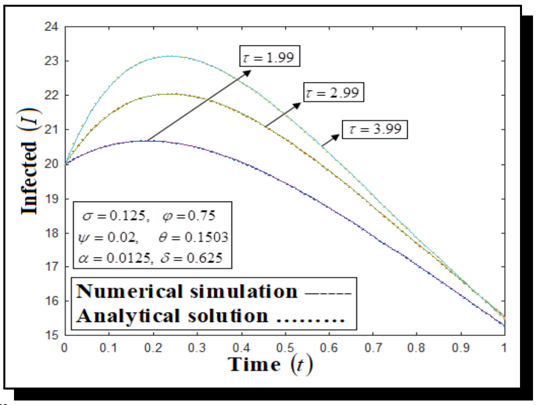
(a) Impact of the rate of innate dying ψ in Infected $I(t)$



(b) The influence of the number of individuals who pass away due to a disease δ in Infected $I(t)$



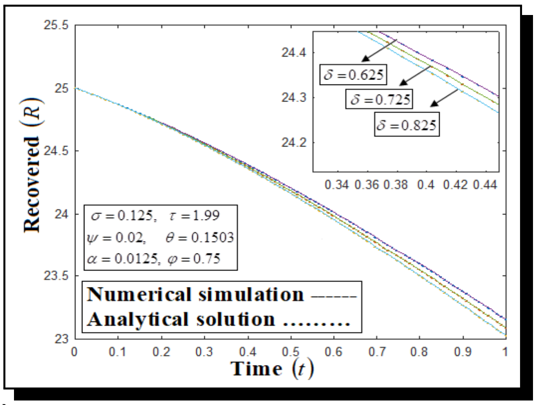
(c) The effects of the rate of infectious recovery θ in Infected $I(t)$



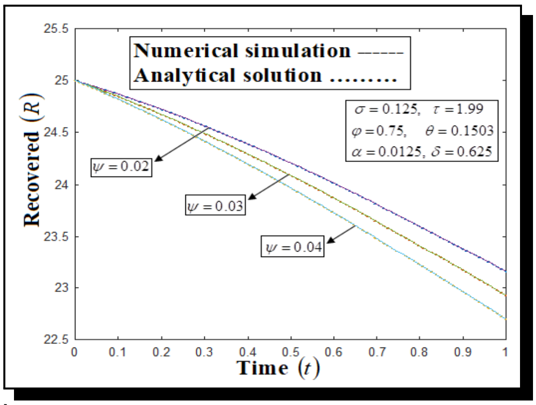
(d) Variation of the unprotected symptom rate τ in Infected $I(t)$

Figure 5

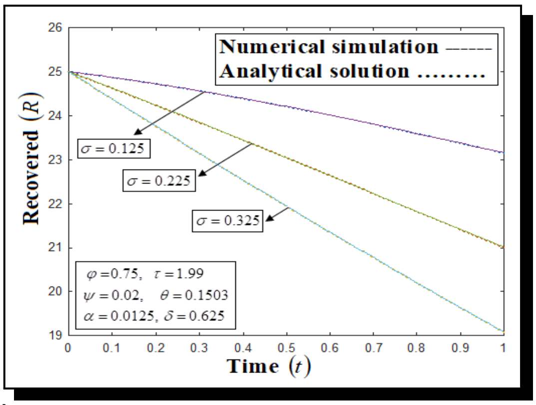
For Recovered class. Figures 6(a)-6(d) shows that the Recovered class $R(t)$ versus time (t) (weeks) by using equations (39). The virtues of natural death ψ, the amount of people who pass away due to illness δ, as well as how quickly individuals who have recovered lose their transient immunity σ all increase, the corresponding Recovered class $R(t)$ drops as shown in Figures 6(a)-6(c). As illustrated in Figure 6(d), there is a positive correlation between the increases in the infectious recovery rate θ and the recovered class $R(t)$.



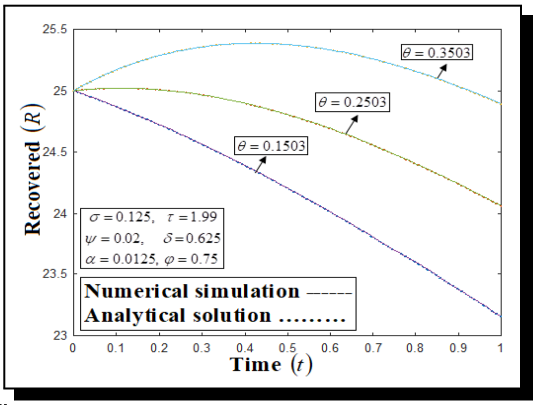
(a) The influence of the number of individuals who pass away from disease δ in recovered $R(t)$



(b) Variation of the rate of innate dying ψ in Recovered $R(t)$



(c) The impacts of the rate at which recovered humans lose temporary immunity σ in Recovered $R(t)$



(d) Impact of the rate of infectious recovery θ in Recovered $R(t)$

Figure 6

6. Conclusion

The approximate analytical result of the Susceptible (S), Exposed (E), Infected (I), and Recovered (R), of typhoid infection 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 effectiveness of the method. The result leads to the following: The agreement between the numerical simulation and our approximate analytical results was found to be satisfactory.

- Reducing the amount that sick people interact with one another is one strategy to reduce the number of susceptible people.
- We can lessen the number of persons who are susceptible to typhoid fever by boosting the immunity of the recovered individual.

- By avoiding the contact between the infected and uninfected individual, we can reduce the exposed population.
- The infected population was reduced due to an increase in the recovered people.

Competing Interests

The authors declare that they have no competing interests.

Authors' Contributions

All the authors contributed significantly in writing this article. The authors read and approved the final manuscript.

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