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Mathematical analysis of a generalized epidemic model with nonlinear incidence function

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Abstract

Background: Though different forms of control measures have been deployed to curtail disease transmission, which are mostly through vaccination, treatment, isolation, etc., using mathematical models. Therefore, there is a need to consider the strict compliance or attendance of human individuals to medical awareness program through media outlets like radio, television, etc. In this work, a generalized mathematical model of two groups of infectious individuals who are compliant and non-compliant to medical awareness program is studied.

Results: A generalized Susceptible-Exposed-Infected-Recovered (SEIR) model with two groups of infectious individuals who attend or are compliant and those who do not attend or are non-compliant to medical awareness program is established. The analytical results of the model shows that the model is positive, well-posed, and epidemiologically reasonable. The two equilibria and the basic reproduction number R_0 of the model is computed and analyzed and it is shown that the disease-free equilibrium is locally and globally asymptotically stable when $R_0 < 1$ and the endemic equilibrium is globally stable when $R_0 > 1$. Simulations are carried out by varying some parameters when R_0 is less and above unity. The simulations suggest that control interventions are to be implemented and medical awareness program scaled up to mitigate the spread of diseases. Furthermore, two numerical methods of Runge-Kutta and Differential Transform Method (DTM) are employed to obtain the approximate solutions of the model system equations, and it is observed that the results of the two methods agreeably compare with each other in terms of efficiency and convergence.

Conclusion: This work should be taken into consideration by health policy makers and bio-mathematicians, because existing literature only take into consideration, how diseases spread and its management without considering the impact of strict compliance to consistent awareness program to mitigate the spread of diseases, which has been considered in this work. The limitation of this work is the unavailability of data on individuals in disease endemic regions who always and who do not comply with medical awareness programs.

Keywords: Susceptible, Reproduction number R_0 , Local stability, Global stability, Incidence function

1 Background

Mathematical models are important techniques that have been greatly explored to describe the transmission dynamics of evolving and re-evolving diseases in epidemiology. The essence of epidemic models is to understand, prepare for future occurrence of epidemic

breakout, and to implement necessary intervention strategies to curtail the spread of diseases in human and environment host population. One of the earliest publications on mathematical models of infectious disease are the works of [1–3]. The authors discussed on the division of the human host population into compartments and epidemic interactions between them. Also, several mathematical techniques have been employed by authors to quantitatively and qualitatively describe the dynamics

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of models of many diseases like onchocerciasis [4, 5], conjunctivitis [6], co-infection of malaria with filariasis and toxoplasmosis [7, 8], as well as using stability theorems and optimal control analysis to qualitatively and quantitatively analyse epidemic models [9–11]. Medical awareness programs through media outlets like radio, electronic prints, television, and social media are means of communicating to the human host community to be aware of how to mitigate disease spread and forestall healthy living [12, 13]. The basic reproduction number R_r is an epidemic threshold used to determine the number of secondary cases of infections arising as a result of an introduction of an infected individual into a susceptible host population during his or her period of infection. R_r have been used immensely to describe the reproductive rate of many diseases [14]. Also, [15] worked on the analysis of an SEIR epidemic model with saturated incidence and saturated treatment functions, while [16] investigated the impact of vaccination strategies for a SISV epidemic model guaranteeing the non-existence of endemic solutions. In addition, [17] worked on the SEIR model formulation and compartmental epidemic interactions in the human population, while [18] worked on assessing the inference of the basic reproduction number R_r in SIR model incorporating growth scaling parameter, see also [19, 20]. Other works on SEIR modeling includes [21–24], while the publications of [25, 26] proved useful to this study by employing the use of Lyapunov functions to analyze model stability domain. Numerical methods of Runge-Kutta and DTM have proved useful in obtaining the convergent approximate solutions of epidemic models, see [27, 28]. In view of the cited publications, we consider the SEIR epidemic model with two groups of infected individuals who attend or are compliant to medical awareness program and those who did not attend or are non-compliant to medical awareness program, with saturated incidence function. Section 2 discusses the positivity and invariant region analysis of the model, while the equilibria of the model and basic reproduction number R_r is obtained. Section 3 involves the local and global stability of model system at the disease-free and endemic equilibrium solutions. Furthermore, we employ the DTM and Runge-Kutta fourth-order method to obtain the approximate solutions of the model. The results obtained agreeably compare with each other. Also, we performed simulations involving some parameters of the model when $R_r < 1$ and $R_r > 1$.

2 Model establishment and analytical results

The total human host population denoted $N(t)$, at time $t > 0$ is classified into five compartments of susceptible denoted $S(t)$, which are the individuals who are at risk of acquiring the disease. Also, people who have been

latently infected but are not yet infectious are denoted by $E(t)$, symptomatic individuals who did not attend medical awareness program are denoted by $I_u(t)$, symptomatic individuals who did attend medical awareness program are denoted by $I_v(t)$, and the recovered individuals are denoted by $R(t)$, so that

$$N(t) = S(t) + E(t) + I_u(t) + I_v(t) + R(t). \quad (1)$$

The susceptible population is increased by the recruitment of individuals at the rate A , following an effective contact with infected individuals in the I_u and I_v compartment, the force of infection is denoted $\lambda(t)$, and described by the quantity

$$\lambda(t) = \beta \left(\frac{I_u}{1 + \phi_1 I_u} + \frac{\theta I_v}{1 + \phi_2 I_v} \right). \quad (2)$$

In (2), $\lambda(t)$ is the force of infection that takes into account the high saturation of infected individuals in the human host community, where β is the transmission rate of infection and θ is the modification parameter that takes into account the relative infectiousness of medical awareness non-compliant individuals to transmit infection at a higher rate than medical awareness compliant individuals. We adopt a nonlinear saturated incidence rate in the two groups of individuals to describe the behavioral change and crowding effect of infected humans where ϕ_1 and ϕ_2 measures the inhibitory effect. But if ϕ_1 and ϕ_2 are zeros, then the incidence function follows a bilinear incidence which is commonly adopted in many models. The population of the susceptible individuals is further decreased by natural death rate μ . Thus, the rate of change of the susceptible population is given by

$$\frac{dS}{dt} = A - (\mu + \lambda(t))S. \quad (3)$$

The population of exposed individuals is increased by the force of infection $\lambda(t)$. The compartment is further on decreased by the development of clinical symptoms, natural death and the disease-induced mortality at the rate ϵ , μ and d respectively, so that

$$\frac{dE}{dt} = \lambda(t)S - (\epsilon + \mu + d)E. \quad (4)$$

The population of the symptomatic individuals who did not attend medical awareness program is increased at the rate ϵ . It is decreased by natural recovery rate γ_o , the rate of emergence of new symptoms σ , natural death μ_1 , and disease-induced mortality rate α_1 . This is given by

$$\frac{dI_u}{dt} = \epsilon E - (\gamma_o + \mu_1 + \alpha_1 + \sigma)I_u. \quad (5)$$

The population of infected individuals who attended medical awareness program is increased progressively at the rate σ . The compartment is decreased by recovery rate γ_1 , natural death rate μ_2 , and disease-induced mortality α_2 . It is assumed that the disease-induced mortality rate of individuals who attended medical awareness program is low in comparison with infected individuals who did not attend medical awareness program, such that $\alpha_2 < \alpha_1$. Hence, the rate of change of this population is given by

$$\frac{dI_v}{dt} = \sigma I_u - (\mu_2 + \gamma_1 + \alpha_2)I_v. \quad (6)$$

Finally, the population of recovered individuals is generated by the recovery of individuals who attend and who did not attend medical awareness program at the rate γ_o and γ_1 , while it is decreased by natural death rate μ , so that

$$\frac{dR}{dt} = \gamma_o I_u + \gamma_1 I_v - \mu R. \quad (7)$$

Thus, the model for the transmission dynamics of a generalized infectious disease with non-linear incidence of two groups of infected individuals who attend and did not attend medical awareness follows a first order system of ordinary differential equations given by

$$\begin{aligned} \frac{dS}{dt} &= A - (\mu + \lambda(t))S, \\ \frac{dE}{dt} &= \lambda(t)S - (\epsilon + \mu + d)E, \\ \frac{dI_u}{dt} &= \epsilon E - (\gamma_o + \mu_1 + \alpha_1 + \sigma)I_u, \\ \frac{dI_v}{dt} &= \sigma I_u - (\mu_2 + \gamma_1 + \alpha_2)I_v, \\ \frac{dR}{dt} &= \gamma_o I_u + \gamma_1 I_v - \mu R. \end{aligned} \quad (8)$$

Subject to the initial conditions $S(0) = S_o$, $E(0) = E_o$, $I_u(0) = I_{uo}$, $I_v(0) = I_{vo}$, $R(0) = R_o$.

2.1 Positivity of the model

It is assumed that the initial conditions of the model are non-negative and it is necessary to show that the solution of the model is positive.

Theorem 1: Let $\Omega = \{(S, E, I_u, I_v, R) \in R_+^5 : S_o > 0, E_o > 0, I_{uo} > 0, I_{vo} > 0, R_o > 0\}$. Then the solutions of S, E, I_u, I_v, R are positive for $t \geq 0$.

Proof: From the model system of differential Eq. (8), considering the first state equation given by

$$\begin{aligned} \frac{dS}{dt} &= A - (\mu + \lambda)S, \\ \text{so that} \\ \frac{dS(t)}{dt} &\geq (\mu + \lambda)S, \\ \frac{dS(t)}{S} &\geq (\lambda + \mu)dt, \\ \text{and} \\ \int \frac{dS(t)}{S} &\geq \int (\lambda + \mu)dt. \end{aligned} \quad (9)$$

Solving (9) using separation of variable and applying the initial condition $S(0) = S_o$, yields

$$S(t) \geq S_o e^{-(\lambda + \mu)t} \geq 0. \quad (10)$$

Also, from the second state equation of (8),

$$\frac{dE}{dt} = \lambda S - (\epsilon + \mu + d)E. \quad (11)$$

Simplifying (11) further yields

$$\begin{aligned} \frac{dE}{dt} &\geq (\epsilon + \mu + d)E \\ \text{and} \\ \int \frac{dE}{E} &\geq \int (\epsilon + \mu + d)dt. \end{aligned} \quad (12)$$

On solving (12) using separation of variable and applying initial condition $E(0) = E_o$, yields

$$E(t) \geq E_o e^{-(\epsilon + \mu + d)t} \geq 0. \quad (13)$$

From the third state equation in (8),

$$\frac{dI_u}{dt} = \epsilon E(t) - (\gamma_o + \mu_1 + \alpha_1 + \sigma)I_u, \quad (14)$$

Simplifying (14) further become,

$$\begin{aligned} \frac{dI_u}{dt} &\geq (\gamma_o + \mu_1 + \alpha_1 + \sigma)I_u \\ \text{and} \end{aligned} \quad (15)$$

$$\int \frac{dI_u}{I_u} \geq \int (\gamma_o + \mu_1 + \alpha_1 + \sigma)dt.$$

Solving (15) using separation of variable and applying initial condition $I_u(0) = I_{uo}$, yields

$$I_u(t) \geq I_{uo} e^{-(\gamma_o + \mu_1 + \alpha_1 + \sigma)t} \geq 0. \quad (16)$$

In addition, taking the fourth state equation of (8),

$$\frac{dI_v}{dt} = \sigma I_u(t) - (\mu_2 + \gamma_1 + \alpha_2)I_v \quad (17)$$

where

$$\int \frac{dI_v}{I_v} \geq - \int (\mu_2 + \gamma_1 + \alpha_2)dt. \quad (18)$$

Solving (18) using separation of variable and applying initial condition $I_v(0) = I_{vo}$, yields

$$I_v(t) \geq I_{vo} e^{-(\mu_2 + \gamma_1 + \alpha_2)t} \geq 0. \quad (19)$$

Finally, taking the fifth state equation of (8),

$$\frac{dR}{dt} = \gamma_o I_u(t) + \gamma_1 I_v(t) - \mu R. \quad (20)$$

The simplification of (20) yields

$$\begin{aligned} \frac{dR}{dt} &\geq -\mu R, \\ \text{and} \\ \frac{dR}{R} &\geq -\mu dt, \\ \int \frac{dR}{R} &\geq - \int \mu dt. \end{aligned} \quad (21)$$

Solving (21) using separation of variable and applying initial condition $R(0) = R_o$, yields

$$R(t) \geq R_o e^{-\mu t} \geq 0. \quad (22)$$

From (10), (13), (16), (19), and (22), it is clear that at time $t > 0$, the model solutions are positive.

This completes the proof of the theorem.

2.2 Invariant region

In this section, the model system is analyzed in an invariant region and shown to be bounded. The addition of the whole model system Eq. (8) yields

$$N = S + E + I_u + I_v + R, \quad (23)$$

such that

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI_u}{dt} + \frac{dI_v}{dt} + \frac{dR}{dt} \quad (24)$$

and

$$\frac{dN}{dt} = A - \mu N - dE - \alpha_1 I_u - \alpha_2 I_v. \quad (25)$$

In the absence of natural and mortality due to disease, i.e., ($d = 0$, $\alpha_1 = 0$, $\alpha_2 = 0$), (25) becomes

$$\frac{dN}{dt} = A - \mu N. \quad (26)$$

Integrating both side of (26) yields

$$\int \frac{dN}{A - \mu N} \leq \int dt \quad (27)$$

and

$$\frac{1}{\mu} \ln(A - \mu N) \leq t. \quad (28)$$

Simplification of (28) become

$$A - \mu N \geq e^{-\mu t}. \quad (29)$$

Applying the initial condition, $N(0) = N_o$, (29) yields $A = A - \mu N_o$. Substituting $A = A - \mu N_o$ into (29) yields

$$A - \mu N_o \geq (A - \mu N_o) e^{-\mu t}. \quad (30)$$

Further simplification and re-arrangement of (30) yields

$$N \leq \left(\frac{A}{\mu} - \frac{A - \mu N_o}{\mu} \right) e^{-\mu t}. \quad (31)$$

As $t \rightarrow \infty$ in (31), the population size $N \rightarrow \frac{A}{\mu}$ implies that $0 \leq N \leq \frac{A}{\mu}$. Thus, the feasible solution set of the system equations of the model start and end in the region

$$\Omega = \left(\{S, E, I_u, I_v, R\} \in R^{+5} : N \leq \frac{A}{\mu} \right). \quad (32)$$

Therefore, the basic model (8) is well posed mathematically and epidemiologically reasonable. Hence, it is sufficient to study the dynamics of the model system (8) in Ω .

2.3 Equilibria

To find the disease-free equilibrium solutions, the right-hand side of the model system (8) is equated to zero, evaluating it at when there is no disease in the system, i.e., $E = I_u = I_v = 0$. Therefore, the disease-free equilibrium solutions are given by

$$E_o = (S, E, I_u, I_v, R) = \left(\frac{A}{\mu}, 0, 0, 0, 0 \right). \quad (33)$$

The endemic equilibrium is denoted $E^{**} = (S^{**}, E^{**}, I_u^{**}, I_v^{**}, R^{**})$ and it occurs when a disease persist in the human host population. Therefore

$$\begin{aligned} E^{**} = (S^{**}, E^{**}, I_u^{**}, I_v^{**}, R^{**}) &= \left(S^* = \frac{A}{m_1}, E^* = \frac{\lambda A}{m_2 m_3}, I_u^* = \frac{\lambda \epsilon A}{m_1 m_2 m_3}, \right. \\ &\left. I_v^* = \frac{\lambda \sigma \epsilon A}{m_1 m_2 m_3 m_4}, R^* = \frac{1}{\mu} \left[\frac{\lambda \epsilon \gamma_o A}{m_1 m_2 m_3} + \frac{\lambda \sigma \epsilon \gamma_1 A}{m_1 m_2 m_3 m_4} \right] \right). \end{aligned} \quad (34)$$

Where $m_1 = (\lambda + \mu)$, $m_2 = (\epsilon + \mu + d)$, $m_3 = (\gamma_o + \mu_1 + \alpha_1 + \sigma)$, $m_4 = (\mu_2 + \gamma_1 + \alpha_2)$.

2.4 Basic reproduction number (R_0)

We want to show how the threshold that governs the spread of a disease, called the basic reproduction number is obtained.

Theorem 2.

Define $X_s = \{X = 0 | X_i, i = 1, 2, 3, \dots\}$, in order to obtain R_0 , new infections are distinguished from other changes in the populations. Such that, $F_i(x)$ is the rate of new

manifestations of clinical symptoms in compartment i . Also, let V_i^+ be the rate at which individuals move out of compartment i . Then $x_i = F_i(x) - V_i(x)$, $i = 1, 2, 3, \dots$, and $V_i(x) = V_i^- - V_i^+$. F is a non negative matrix and V is a non-singular matrix.

Proof: We applied the next generation matrix method to the model equations starting with newly infective classes given by

$$\left. \begin{aligned} \frac{dE}{dt} &= \lambda(t)S - m_2E, \\ \frac{dI_u}{dt} &= \epsilon E - m_3I_u, \\ \frac{dI_v}{dt} &= \sigma I_u - m_4I_v. \end{aligned} \right\} \quad (35)$$

The rate of new clinical symptoms is given by

$$F = \begin{pmatrix} \beta S \left(\frac{I_u}{1 + \phi_1 I_u} + \frac{\theta I_v}{1 + \phi_2 I_v} \right) \\ 0 \\ 0 \end{pmatrix} \quad (36)$$

And the rate of transfer terms of individuals is given by

$$V = \begin{pmatrix} m_2E \\ \epsilon E - m_3I_u \\ \sigma I_u - m_4I_v \end{pmatrix}. \quad (37)$$

The Jacobian matrices of F and V evaluated at disease-free equilibrium solution (33) are given by

$$F = \begin{pmatrix} 0 & 0 & \frac{\beta A}{\mu} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \quad (38)$$

and

$$V = \begin{pmatrix} m_2 & 0 & 0 \\ \epsilon & m_3 & 0 \\ 0 & \sigma & m_4 \end{pmatrix}. \quad (39)$$

The inverse of V is given by

$$V^{-1} = \begin{pmatrix} \frac{1}{m_2} & 0 & 0 \\ \frac{\epsilon}{m_2 m_3} & \frac{1}{m_3} & 0 \\ \frac{\epsilon \sigma}{m_2 m_3 m_4} & \frac{\sigma}{m_4} & \frac{1}{m_4} \end{pmatrix} \quad (40)$$

and

$$FV^{-1} = \begin{pmatrix} \frac{\lambda A \epsilon \sigma}{\mu m_2 m_3 m_4} & \frac{\lambda A \sigma}{\mu m_3 m_4} & \frac{\lambda A}{\mu m_4} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}. \quad (41)$$

The eigenvalues of FV^{-1} in (41) are given by

$$\begin{aligned} \lambda_1 &= \lambda_2 = 0 \\ \text{and} \\ \lambda_3 &= \frac{\lambda A \epsilon \sigma}{\mu m_2 m_3 m_4}. \end{aligned} \quad (42)$$

The dominant eigenvalue in (42) is λ_3 . Therefore, the basic reproduction number R_r is given by

$$R_r = \frac{\lambda A \epsilon \sigma}{\mu m_2 m_3 m_4}. \quad (43)$$

The threshold in (43) measures the rate at which new cases of infection arises, when a typical infected individual is introduced into a susceptible population of humans during their course of infection.

3 Stability analysis of the model system equilibria

3.1 Local stability of the disease-free equilibrium

Theorem 2: The disease-free equilibrium solution E_o (33) is locally asymptotically stable if $R_r < 1$.

Proof: The Jacobian matrix of system (8) at the disease-free equilibrium solution E_o of (33) is given by

$$J(E_o) = \begin{pmatrix} -m_1 & 0 & 0 & 0 & 0 \\ \lambda & -m_2 & 0 & 0 & 0 \\ 0 & \epsilon_o & -m_3 & 0 & 0 \\ 0 & 0 & \sigma & -m_4 & 0 \\ 0 & 0 & \gamma_o & \gamma_1 & -\mu \end{pmatrix}. \quad (44)$$

The eigenvalues obtained in (44) yield

$$\left. \begin{aligned} &-\mu, \\ m_1 &= -(\lambda + \mu), \\ m_2 &= -(\epsilon + \mu + d). \end{aligned} \right\} \quad (45)$$

The remaining characteristics polynomial is given by

$$\lambda^2 + \lambda(m_3 + m_4) + m_3 m_4 = 0. \quad (46)$$

It is observed that (46) has strictly negative real root if and only if $m_3 > 0$ and $m_4 > 0$, and $m_3 > m_4$. Also, m_3 is positive and for m_4 to be positive, $1 - R_r$ must be positive which leads to $R_r < 1$. Therefore, the disease-free equilibrium E_o (33) of model (8) is locally asymptotically stable if $R_r < 1$.

3.2 Global stability of disease-free equilibrium

Theorem: The disease free equilibrium solution E_o (33) of model system (8) is globally asymptotically stable whenever $R_r < 1$.

Proof: A Lyapunov function is derived for the model system such that

$$F = \left(\frac{\epsilon m_4 + \sigma}{m_2 m_3} \right) E + \left(\frac{m_4 + \sigma}{m_3} \right) I_u + I_v. \quad (47)$$

Where

$$\dot{F} = \left(\frac{\epsilon m_4 + \sigma}{m_2 m_3} \right) \frac{dE}{dt} + \left(\frac{m_4 + \sigma}{m_3} \right) \frac{dI_u}{dt} + \frac{dI_v}{dt} \quad (48)$$

and

$$\begin{aligned} \dot{F} = & \left(\frac{\epsilon m_4 + \sigma}{m_2 m_3} \right) (\beta S - m_3 E) + \left(\frac{m_4 + \sigma}{m_3} \right) \\ & (\epsilon E + m_3 I_u) + (\sigma I_u - m_4 I_v). \end{aligned} \quad (49)$$

Since $\frac{d}{dt}$ in (33), further simplification becomes

$$\begin{aligned} \dot{F} \leq & \left(\frac{\epsilon m_4 + \sigma}{m_2 m_3} \right) \left(\frac{\beta A}{\mu} - m_3 E \right) + \left(\frac{m_4 + \sigma}{m_3} \right) \\ & (\epsilon E + m_3 I_u) + (\sigma I_u - m_4 I_v) \end{aligned} \quad (50)$$

and

$$\dot{F} = \frac{\lambda A \epsilon (m_4 + \sigma)}{\mu m_2 m_3} + (\sigma - (m_4 + \sigma)) I_u - m_4 I_v. \quad (51)$$

Therefore

$$\dot{F} = m_4 \left(R_{tr} - \frac{\lambda A \epsilon}{\mu m_2 m_3} I_u + I_v \right). \quad (52)$$

Since all the parameters and variables of the model system (8) are nonnegative, it follows that $\dot{F} \leq 0$ for $R_r < 1$ with $\dot{F} = 0$ if and only if $E = I_u = I_v = 0$. Hence, \dot{F} is a Lyapunov function in Ω . Therefore, the largest compact invariant subset of the set where $\dot{F} = 0$ is the singleton $\{(E, I_u, I_v) = (0, 0, 0)\}$. Thus, it follows, from the La-Salle's invariant principle [19], that as $t \rightarrow \infty$,

$$(E, I_u, I_v) \rightarrow (0, 0, 0). \quad (53)$$

Therefore the disease-free equilibrium (33), of model (8) is globally asymptotically stable if $R_{tr} < 1$.

3.3 Global stability of endemic equilibrium

Theorem: If $R_r > 1$, the endemic equilibrium solution (34) of the model system (8) is globally asymptotically stable.

Proof: A Lyapunov function candidate of the form

$$\begin{aligned} L(t) = & \left(S - S^* - S^* \text{Loge} \frac{S}{S^*} \right) \\ & + \left(E - E^* - E^* \text{Loge} \frac{E}{E^*} \right) \\ & + \left(I_u - I_u^* - I_u^* \text{Loge} \frac{I_u}{I_u^*} \right) \\ & + \left(I_v - I_v^* - I_v^* \text{Loge} \frac{I_v}{I_v^*} \right) \\ & + \left(R - R^* - R^* \text{Loge} \frac{R}{R^*} \right) \end{aligned} \quad (54)$$

is derived, such that the time derivative of (54) becomes

$$\begin{aligned} \frac{dL}{dt} = & \left(\frac{S - S^*}{S} \right) \frac{dS}{dt} + \left(\frac{E - E^*}{E} \right) \frac{dE}{dt} \\ & + \left(\frac{I_u - I_u^*}{I_u} \right) \frac{dI_u}{dt} + \left(\frac{I_v - I_v^*}{I_v} \right) \frac{dI_v}{dt} \\ & + \left(\frac{R - R^*}{R} \right) \frac{dR}{dt}, \end{aligned} \quad (55)$$

so that

$$\begin{aligned} \frac{dL}{dt} = & \left(\frac{S - S^*}{S} \right) (A - m_1 S) + \left(\frac{E - E^*}{E} \right) \\ & \times (\lambda S - m_2 E) + \left(\frac{I_u - I_u^*}{I_u} \right) (\epsilon E - m_3 I_u) \\ & + \left(\frac{I_v - I_v^*}{I_v} \right) (\sigma I_u - m_4 I_v) + \left(\frac{R - R^*}{R} \right) \\ & \times (\gamma_o I_u + \gamma_1 I_v - \mu R). \end{aligned} \quad (56)$$

and

$$\begin{aligned} \frac{dL}{dt} = & \left(\frac{S - S^*}{S} \right) (A - m_1 (S - S^*)) + \left(\frac{E - E^*}{E} \right) \\ & \times (\lambda (S - S^*) - m_2 (E - E^*)) + \left(\frac{I_u - I_u^*}{I_u} \right) \\ & \times (\epsilon (E - E^*) - m_3 (I_u - I_u^*)) \\ & \times \left(\frac{I_v - I_v^*}{I_v} \right) - m_4 + \left(\frac{R - R^*}{R} \right) \\ & \times (\gamma_o (I_u - I_u^*) + \gamma_1 (I_v - I_v^*) - \mu (R - R^*)). \end{aligned} \quad (57)$$

Further simplification of (57) yields

$$\begin{aligned}
\frac{dL}{dt} = & \left(\frac{(S-S^*)^2}{S} \right) \left(\frac{A}{(S-S^*)} - m_1 \right) \\
& + \left(\frac{(E-E^*)^2}{E} \right) \left(\frac{\lambda(S-S^*)}{(E-E^*)} - m_2 \right) \\
& + \left(\frac{(I_u-I_u^*)^2}{I_u} \right) \left(\frac{\epsilon(E-E^*)}{(I_u-I_u^*)} - m_3 \right) \\
& + \left(\frac{(I_v-I_v^*)^2}{I_v} \right) \left(\frac{\sigma(I_u-I_u^*)}{(I_v-I_v^*)} - m_4 \right) \\
& + \left(\frac{(R-R^*)^2}{R} \right) \\
& \times \left(\frac{\gamma_o(I_u-I_u^*)}{(R-R^*)} + \frac{\gamma_1(I_v-I_v^*)}{(R-R^*)} - \mu \right)
\end{aligned} \quad (58)$$

and

$$\begin{aligned}
\frac{dL}{dt} = & \frac{A(S-S^*)}{S} + \frac{\lambda(S-S^*)(E-E^*)}{E} \\
& + \frac{\epsilon(E-E^*)(I_u-I_u^*)}{I_u} \\
& + \frac{\sigma(I_u-I_u^*)(I_v-I_v^*)}{I_v} \\
& + \frac{\gamma_o(I_u-I_u^*)(R-R^*)}{R} \\
& + \frac{\gamma_1(I_v-I_v^*)(R-R^*)}{R} \\
& - \frac{m_1(S-S^*)^2}{S} - \frac{m_2(E-E^*)^2}{E} \\
& - \frac{m_3(I_u-I_u^*)^2}{I_u} - \frac{m_4(I_v-I_v^*)^2}{I_v} \\
& - \frac{\mu(R-R^*)^2}{R}.
\end{aligned} \quad (59)$$

Hence, by collecting positive terms together and negative terms together in (59) becomes

$$\frac{dL}{dt} = B_1 - B_2. \quad (60)$$

Where

$$\begin{aligned}
B_1 = & \frac{A(S-S^*)}{S} + \frac{\lambda(S-S^*)(E-E^*)}{E} \\
& + \frac{\epsilon(E-E^*)(I_u-I_u^*)}{I_u} \\
& + \frac{\sigma(I_u-I_u^*)(I_v-I_v^*)}{I_v} \\
& + \frac{\gamma_o(I_u-I_u^*)(R-R^*)}{R} \\
& + \frac{\gamma_1(I_v-I_v^*)(R-R^*)}{R}
\end{aligned} \quad (61)$$

And

$$\begin{aligned}
B_2 = & \frac{m_1(S-S^*)^2}{S} + \frac{m_2(E-E^*)^2}{E} \\
& + \frac{m_3(I_u-I_u^*)^2}{I_u} + \frac{m_4(I_v-I_v^*)^2}{I_v} \\
& + \frac{\mu(R-R^*)^2}{R}.
\end{aligned} \quad (62)$$

Thus if $B_1 < B_2$, then $\frac{dL}{dt} \leq 0$. Note that, $\frac{dL}{dt} = 0$ if and only if $(S = S^*, E = E^*, I_u = I_u^*, I_v = I_v^*, R = R^*)$. Therefore, the largest compact, invariant set in $[(S^*, E^*, I_u^*, I_v^*, R^*) \in \Omega: \frac{dL}{dt} = 0]$ is the singleton set E^* , where E^* is the endemic equilibrium solution (34) of the model system (8). By La-Salle's invariant principle [20], E^* is globally asymptotically stable in Ω if $B_1 < B_2$.

3.4 Approximate solution of the SEIR epidemic model

In this section, the approximate solution of the model system (8) is obtained, using the Runge-Kutta fourth order and DTM. The concept of DTM was first proposed by Zhou [28] for solving linear and nonlinear initial value problems in electrical circuit analysis. The method in many instances have been adopted to solve series of models based on differential equations. The concept of DTM is derived from the Taylor series expansion. This method requires that a system of differential equations together with its initial and boundary conditions are transformed into recurrent power series solutions.

Taylor series expansion of a function $f(x)$ about the point $x = 0$ is given by

$$f(x) = \sum_{n=1}^{\infty} \frac{1}{k!} (f^k(c)(x-c)^k) = 0. \quad (63)$$

For all $x \in (c-r, c+r)$ such that $x = c$ converges to $f(x)$.

Definition 1: The differential transformation $F(k)$ of a function $f(x)$ is defined as

$$F(k) = \frac{1}{k!} \left(\frac{d^k f(x)}{dt^k} \right)_{k=0}. \quad (64)$$

Definition 2: It follows from Eqs. (63) and (64) that the differential inverse transformation $f(x)$ of $F(k)$ is given by

$$f(x) = \sum_{k=0}^{\infty} \frac{t^k}{k!} \frac{d^k f(t)}{dt^k} \Big|_{x=x_0}. \quad (65)$$

Using (63)–(64), the following basic operations of DTM [26, 28] is tabulated below;

If $S(k)$, $E(k)$, $I_u(k)$, $I_v(k)$, and $R(k)$ denote the differential transformation of $S(t)$, $E(t)$, $I_u(t)$, $I_v(t)$, and $R(t)$ respectively, then the following recurrence relation of model system (8) are given by

$$\begin{aligned}
S(k+1) &= \frac{1}{k+1} \left(A\delta(k) - \mu S(k) - \beta \sum_{m=1}^{\infty} \left(\frac{S(m)I_u(k-m)}{\delta(k) + \phi_1 I_u(k)} + \frac{S(m)I_v(k-m)}{\delta(k) + \phi_2 I_v(k)} \right) \right), \\
E(k+1) &= \frac{1}{k+1} \left(-(\epsilon + \mu + d)E(k) + \beta \sum_{m=1}^{\infty} \left(\frac{S(m)I_u(k-m)}{\delta(k) + \phi_1 I_u(k)} + \frac{S(m)I_v(k-m)}{\delta(k) + \phi_2 I_v(k)} \right) \right), \\
I_u(k+1) &= \frac{1}{k+1} (\epsilon E(k) - (\gamma_o + \mu_1 + \alpha_1 + \sigma)I_u(k)), \\
I_v(k+1) &= \frac{1}{k+1} (\sigma I_u(k) - (\mu_2 + \gamma_1 + \alpha_2)I_v(k)), \\
R(k+1) &= \frac{1}{k+1} (\gamma_o I_u(k) + \gamma_1 I_v(k) - \mu R(k)).
\end{aligned} \quad (66)$$

Applying the values of variables and parameters in Table 1 together with the initial conditions of the model, yields the following results given by

$$\begin{aligned}
S(1) &= -17.768, E(1) = 16.089, I_u(1) \\
&= -5.322, I_v(1) = 27.500, R(1) \\
&= -2.162, S(2) = -10.330, E(2) \\
&= 9.559, I_u(2) = 1.496, I_v(2) \\
&= 5.710, R(2) = 1.513, S(3) = 0.878, E(3) \\
&= 0.778, I_u(3) = -0.252, I_v(3) \\
&= -1.591, R(3) = 0.946.
\end{aligned} \quad (67)$$

Substituting the values in (67) into (66) yields, the recurrence relation given by the following;

Table 1 The meanings of variables and parameters of the SEIR model (8)

Symbols	Meanings of Variables and Parameters	Values
A	Recruitment rate of Humans	0.781 day ⁻¹
β	Disease transmission rate	0.05 day ⁻¹
ϕ_1	Emergence of disease symptoms in I_u class	0.312 day ⁻¹
ϕ_2	Emergence of disease symptoms in I_v class	0.120 day ⁻¹
μ	Natural death rate	0.002 day ⁻¹
ϵ	Transition rate from E to I_u	0.006 day ⁻¹
d	Disease related death	0.11 day ⁻¹
μ_1	Natural death in I_u class	0.0132 day ⁻¹
μ_2	Natural death in I_v class	0.0012 day ⁻¹
α_1	Disease induced mortality in I_u class	0.12 day ⁻¹
α_2	Disease induced mortality in I_v class	0.21 day ⁻¹
γ_1	Recovery rate in I_u class	0.134 day ⁻¹
γ_2	Recovery rate in I_v class	0.123 day ⁻¹
σ	Infectious transition from I_u to I_v	0.3 day ⁻¹
θ	Modification parameter	0.0021 day ⁻¹
S	Susceptible Humans	50 day ⁻¹
E	Exposed Humans	20 day ⁻¹
I_u	Infected human who attend awareness program	10 day ⁻¹
I_v	Infected human who did not attend awareness program	7 day ⁻¹
R	Recovered humans	3 day ⁻¹

$$\begin{aligned}
S(t) &= \sum_{k=1}^{\infty} t^k S(k) = S(0) + tS(1) + t^2S(2) + t^3S(3) + \dots, \\
E(t) &= \sum_{k=1}^{\infty} t^k E(k) = E(0) + tE(1) + t^2E(2) + t^3E(3) + \dots, \\
I_u(t) &= \sum_{k=1}^{\infty} t^k I_u(k) = I_u(0) + tI_u(1) + t^2I_u(2) + t^3I_u(3) + \dots, \\
I_v(t) &= \sum_{k=1}^{\infty} t^k I_v(k) = I_v(0) + tI_v(1) + t^2I_v(2) + t^3I_v(3) + \dots, \\
R(t) &= \sum_{k=1}^{\infty} t^k R(k) = R(0) + tR(1) + t^2R(2) + t^3R(3) + \dots,
\end{aligned} \quad (68)$$

The closed form solutions when $k = 3$, in (68) is given by

$$\begin{aligned}
S(t) &= 50 - 17.768t - 10.330t^2 + 0.878t^3 + \dots, \\
E(t) &= 20 + 16.089t + 9.5585t^2 + 0.778t^3 + \dots, \\
I_u(t) &= 10 - 5.322t + 1.496t^2 - 0.252t^3 + \dots, \\
I_v(t) &= 7 + 27.500t + 5.710t^2 - 1.591t^3 + \dots, \\
R(t) &= 3 + 2.162t + 1.513t^2 - 0.946t^3 + \dots.
\end{aligned} \quad (69)$$

Also, the idea of Runge-Kutta method was conceived by Carl Runge and Wilhem Kutta. This work employs the Runge-Kutta fourth-order method because of its accuracy and faster convergence, which have been employed to solve several problems in differential equations applied to science, engineering, economics etc. The numerical scheme for this method is given by

$$y_{n+1} = y_n + \frac{1}{6}(z_1 + 2z_2 + 2z_3 + z_4). \quad (70)$$

Where

$$\left. \begin{aligned}
z_1 &= hf(x_n, y_n), \\
z_2 &= hf\left(x_n + \frac{1}{2}h, y_n + \frac{1}{2}z_1\right), \\
z_3 &= hf\left(x_n + \frac{1}{2}h, y_n + \frac{1}{2}z_2\right), \\
z_4 &= hf(x_n + h, y_n + z_3).
\end{aligned} \right\} \quad (71)$$

Applying the Runge-Kutta scheme to the model system (8) yields

$$\left. \begin{aligned}
S_{n+1} &= S_n + \frac{1}{6}(q_1 + 2q_2 + 2q_3 + q_4)h \\
E_{n+1} &= E_n + \frac{1}{6}(y_1 + 2y_2 + 2y_3 + y_4)h \\
I_{vn+1} &= I_{vn} + \frac{1}{6}(x_1 + 2x_2 + 2x_3 + x_4)h \\
I_{un+1} &= I_{vn} + \frac{1}{6}(u_1 + 2u_2 + 2u_3 + u_4)h \\
R_{n+1} &= R_n + \frac{1}{6}(v_1 + 2v_2 + 2v_3 + v_4)h
\end{aligned} \right\} \quad (72)$$

Where

Table 2 Numerical results of the model system equations using Runge-Kutta method

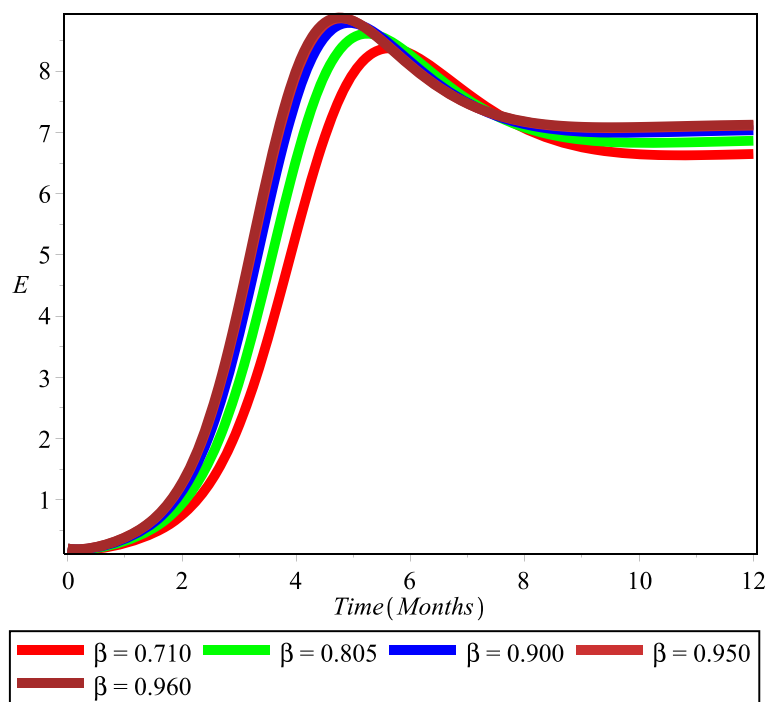
Time(t)	S(t)	E(t)	I _u (t)	I _v (t)	R(t)
0	50	20	10	7	3
0.2	46.3241401	23.38344467	8.99269207	7.023710128	3.47116912
0.4	42.6482802	26.76688931	7.98538415	7.047420250	3.94233820
0.6	38.9724204	30.15033400	6.97807623	7.071130380	4.41350730
0.8	35.2965055	33.53377866	5.97078631	7.094840511	4.88407660
1.0	31.6207006	36.91722233	4.96346038	7.118555066	5.35584500

$$\begin{aligned}
 q_1 &= A - \mu S_n - \beta \left(\frac{I_{un}}{1 + \phi_1 I_{un}} + \frac{\theta I_{vn}}{1 + \phi_2 I_{vn}} \right) S_n, \\
 y_1 &= \beta \left(\frac{I_{un}}{1 + \phi_1 I_{un}} + \frac{\theta I_{vn}}{1 + \phi_2 I_{vn}} \right) S_n - (\epsilon + \mu + d) E_n, \\
 x_1 &= \epsilon E_n - (\gamma_o + \mu_1 + \alpha_1 + \sigma_1) I_{un}, \\
 u_1 &= \sigma I_{un} - (\mu_1 + \gamma_1 + \alpha_2) I_{vn}, \\
 v_1 &= \gamma_o I_{un} + \gamma_1 I_{vn} - \mu R_n.
 \end{aligned}
 \tag{73}$$

Also,

$$\begin{aligned}
 q_2 &= A - \mu \left(S_n + q_1 \frac{h}{2} \right) - \beta \left(\frac{\left(I_{un} + q_1 \frac{h}{2} \right)}{1 + \phi_1 \left(I_{un} + q_1 \frac{h}{2} \right)} + \frac{\left(I_{vn} + q_1 \theta \frac{h}{2} \right)}{1 + \phi_1 \left(I_{vn} + q_1 \frac{h}{2} \right)} \right) \\
 &\quad \left(S_n + q_1 \frac{h}{2} \right), \\
 y_2 &= \beta \left(\frac{\left(I_{un} + y_1 \frac{h}{2} \right)}{1 + \phi_1 \left(I_{un} + y_1 \frac{h}{2} \right)} + \frac{\left(I_{vn} + y_1 \theta \frac{h}{2} \right)}{1 + \phi_1 \left(I_{vn} + y_1 \frac{h}{2} \right)} \right) \left(S_n + y_1 \frac{h}{2} \right) \\
 &\quad + (\epsilon + \mu + d) \left(E_n + y_1 \frac{h}{2} \right), \\
 x_2 &= \epsilon \left(E_n + y_1 \frac{h}{2} \right) + (\gamma_o + \mu_1 + \alpha_1 + \sigma) \left(I_{un} + x_1 \frac{h}{2} \right), \\
 u_2 &= \sigma \left(I_{un} + x_1 \frac{h}{2} \right) + (\gamma_1 + \mu_1 + \alpha_2) \left(I_{vn} + u_1 \frac{h}{2} \right), \\
 v_2 &= \gamma_o \left(I_{un} + x_1 \frac{h}{2} \right) + \gamma_1 \left(I_{vn} + u_1 \frac{h}{2} \right) - \mu \left(R_n + v_1 \frac{h}{2} \right).
 \end{aligned}
 \tag{74}$$

and

**Fig. 1** Varying $\beta(0.001 - 0.027)$ when $R_r < 1$

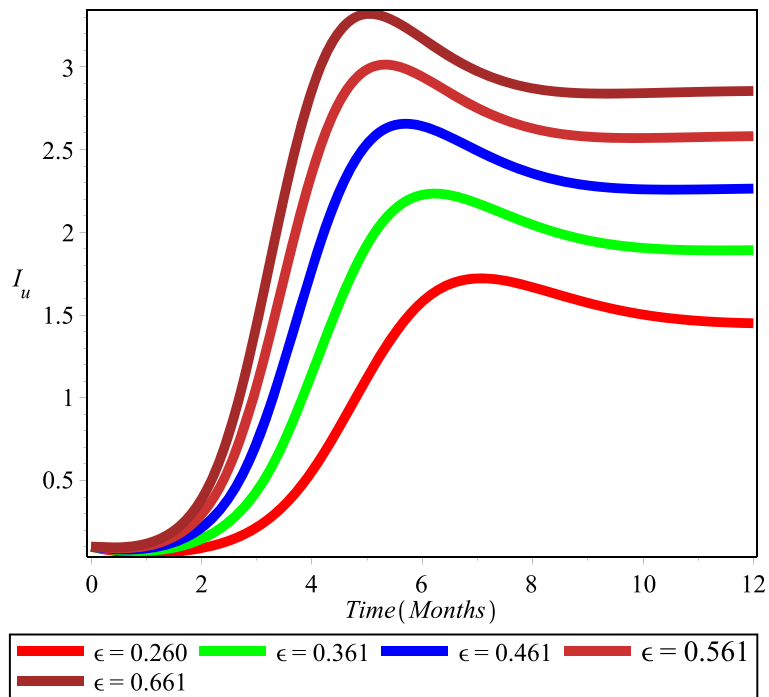


Fig. 2 Varying ϵ (0.001 – 0.0041) when $R_r < 1$

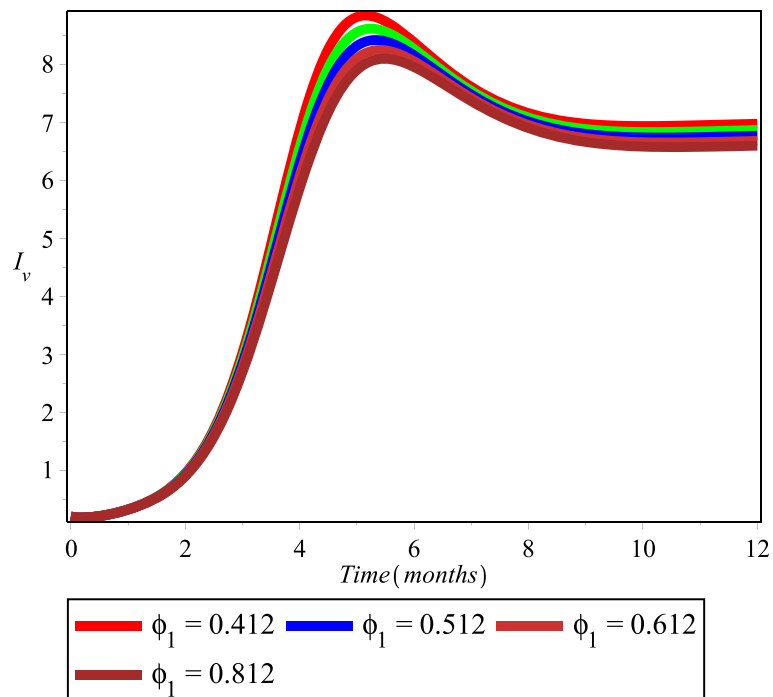


Fig. 3 Varying ϕ_1 (0.212 – 0.612) when $R_r < 1$

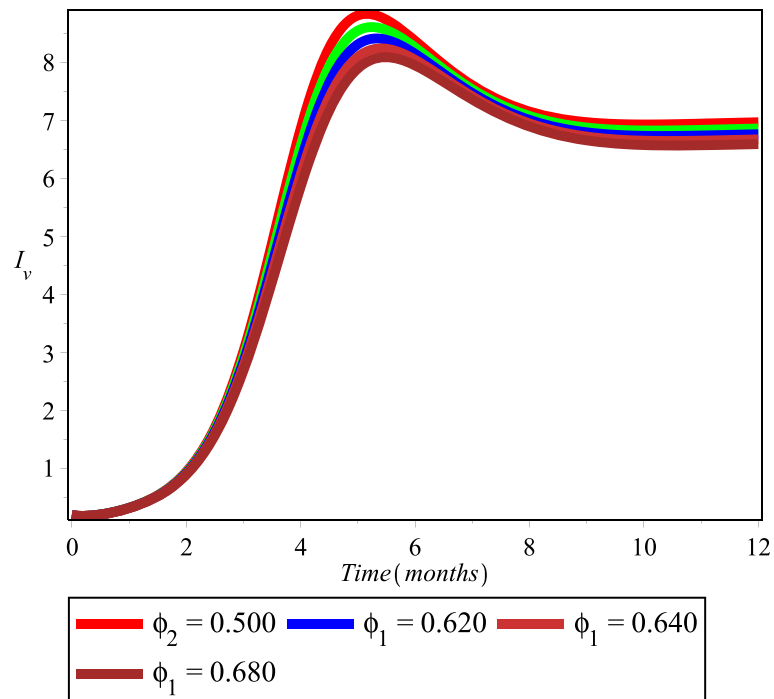


Fig. 4 Varying $\phi_2(0.40 - 0.220)$ when $R_r < 1$

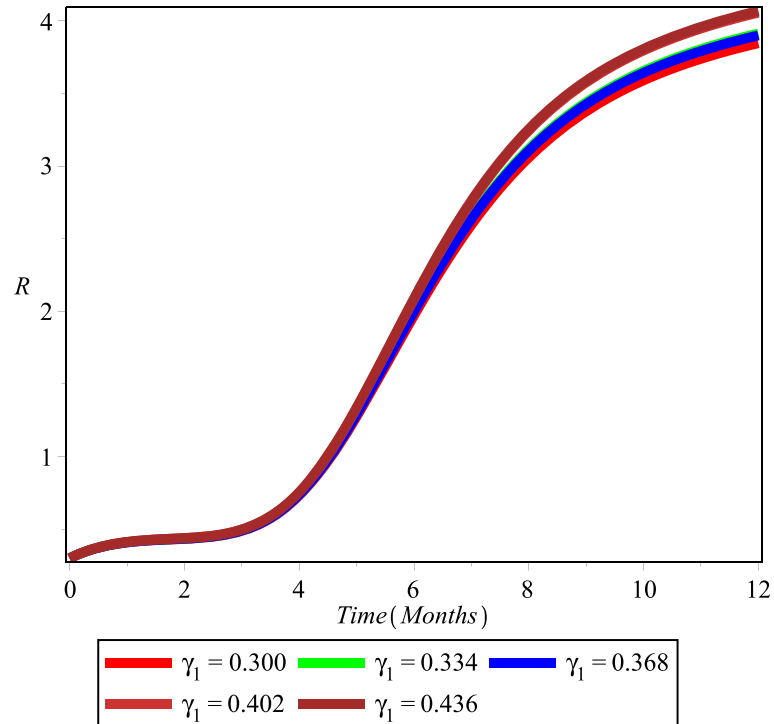


Fig. 5 Varying $\gamma_o(0.123 - 0.203)$ when $R_r < 1$

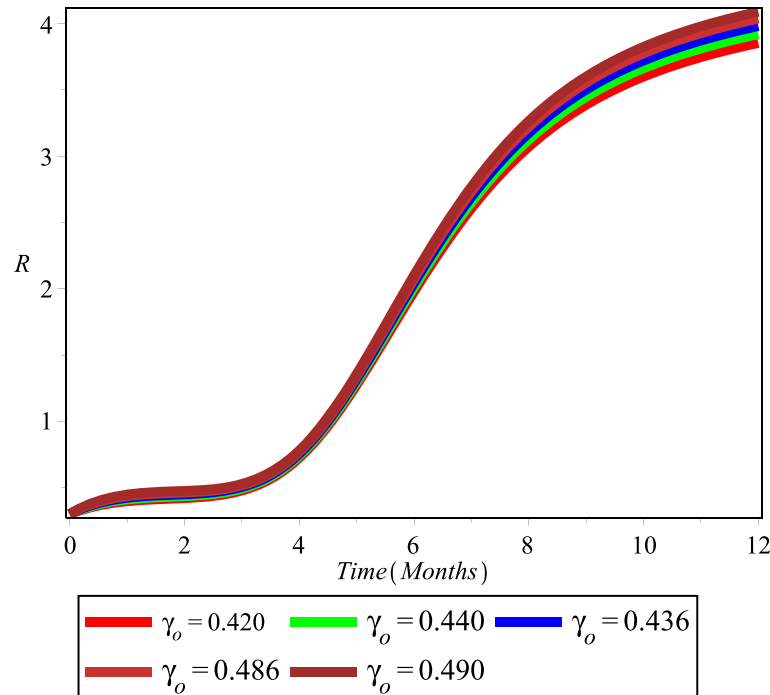


Fig. 6 Varying $\gamma_1(0.100 - 0.164)$ when $R_r < 1$

$$\begin{aligned}
 q_3 &= A - \mu \left(S_n + q_2 \frac{h}{2} \right) - \beta \left(\frac{\left(I_{un} + q_2 \frac{h}{2} \right)}{1 + \phi_1 \left(I_{un} + q_2 \frac{h}{2} \right)} + \frac{\left(I_{vn} + q_2 \theta \frac{h}{2} \right)}{1 + \phi_1 \left(I_{vn} + q_2 \frac{h}{2} \right)} \right) \\
 &\quad \left(S_n + q_2 \frac{h}{2} \right), \\
 y_3 &= \beta \left(\frac{\left(I_{un} + y_2 \frac{h}{2} \right)}{1 + \phi_1 \left(I_{un} + y_2 \frac{h}{2} \right)} + \frac{\left(I_{vn} + y_2 \theta \frac{h}{2} \right)}{1 + \phi_1 \left(I_{vn} + y_2 \frac{h}{2} \right)} \right) \left(S_n + y_2 \frac{h}{2} \right) \\
 &\quad + (\epsilon + \mu + d) \left(E_n + y_2 \frac{h}{2} \right), \\
 x_3 &= \epsilon \left(E_n + y_2 \frac{h}{2} \right) + (\gamma_o + \mu_1 + \alpha_1 + \sigma) \left(I_{un} + x_2 \frac{h}{2} \right), \\
 u_3 &= \sigma \left(I_{un} + x_2 \frac{h}{2} \right) + (\gamma_1 + \mu_1 + \alpha_2) \left(I_{vn} + u_2 \frac{h}{2} \right), \\
 v_3 &= \gamma_o \left(I_{un} + x_2 \frac{h}{2} \right) + \gamma_1 \left(I_{vn} + u_2 \frac{h}{2} \right) - \mu \left(R_n + v_2 \frac{h}{2} \right),
 \end{aligned} \quad (75)$$

And

$$\begin{aligned}
 q_4 &= A - \mu \left(S_n + z_3 h \right) - \beta \left(\frac{\left(I_{un} + z_3 h \right)}{1 + \phi_1 \left(I_{un} + z_3 h \right)} + \frac{\left(I_{un} + z_3 \theta h \right)}{1 + \phi_1 \left(I_{un} + z_3 h \right)} \right) \\
 &\quad \left(S_n + z_3 h \right), \\
 y_4 &= \beta \left(\frac{\left(I_{un} + z_3 h \right)}{1 + \phi_1 \left(I_{un} + z_3 h \right)} + \frac{\left(I_{un} + z_3 \theta h \right)}{1 + \phi_1 \left(I_{un} + z_3 h \right)} \right) \\
 &\quad \left(S_n + z_3 h \right) - (\epsilon + \mu + d) \left(E_n + y_3 h \right), \\
 x_4 &= \epsilon \left(E_n + y_3 h \right) - (\gamma_o + \mu_1 + \alpha_1 + \sigma_1) \left(I_{un} + x_3 h \right), \\
 u_4 &= \sigma \left(I_{un} + x_3 h \right) - (\mu_1 + \gamma_1 + \alpha_2) \left(I_{vn} + u_3 h \right), \\
 v_4 &= \gamma_o \left(I_{un} + x_3 h \right) + \gamma_1 \left(I_{vn} + u_3 h \right) - \mu \left(R_n + v_3 h \right).
 \end{aligned} \quad (76)$$

Substituting (73) to (76), together with the parameter values and initial conditions of the model system (8) into (72) yield the numerical results in Table 2.

3.5 Results

Simulations are carried out by varying some parameters of the models when the basic reproduction number $R_{tr} < 1$ as shown in Figs. 1, 2, 3, 4, 5, and 6, that is, the parameter solutions converges to the disease-free equilibrium. Tables 1 and 3 display the parameter and variable values used in the simulations of the model and the basic operations of DTM. Also, the numerical results of the model system is presented using Runge-Kutta fourth order and differential transform method (DTM) in Tables 2 and 4 which compare agreeably with each other, and the DTM performs better. Figure 1 is the description of the impact

Table 3 Basic Operations of DTM

Original function $f(x)$	Transformed function $F(k)$
$f(x) = g(x) \pm h(x)$	$F(k) = G(k) \pm H(k)$
$f(x) = cg(x)$	$F(k) = cG(k)$, where c is a constant
$f(x) = \frac{dg(x)}{dx}$	$F(k) = (k+1)G(k+2)$
$f(x) = \frac{d^m g(x)}{dx^m}$	$F(k) = (k+1)(k+2) \dots (k+m)G(k+m)$
$f(x) = 1$	$F(k) = \delta(k)$
$f(x) = x$	$F(k) = \delta(k-1)$
$f(x) = x^m$	$F(k) = \delta(k-m) = \begin{cases} 1, & \text{if } k=m \\ 0, & \text{if } k \neq m \end{cases}$
$f(x) = g(x)h(x)$	$F(k) = \sum_{m=0}^k H(m)G(k-m)$
$f(x) = e^{mx}$	$F(k) = \frac{m^k}{k!}$
$f(x) = (1+x)^m$	$F(k) = \frac{m(m-1)(m-2) \dots (m-k+1)}{k!}$

Table 4 Numerical results of the model system equations using DTM

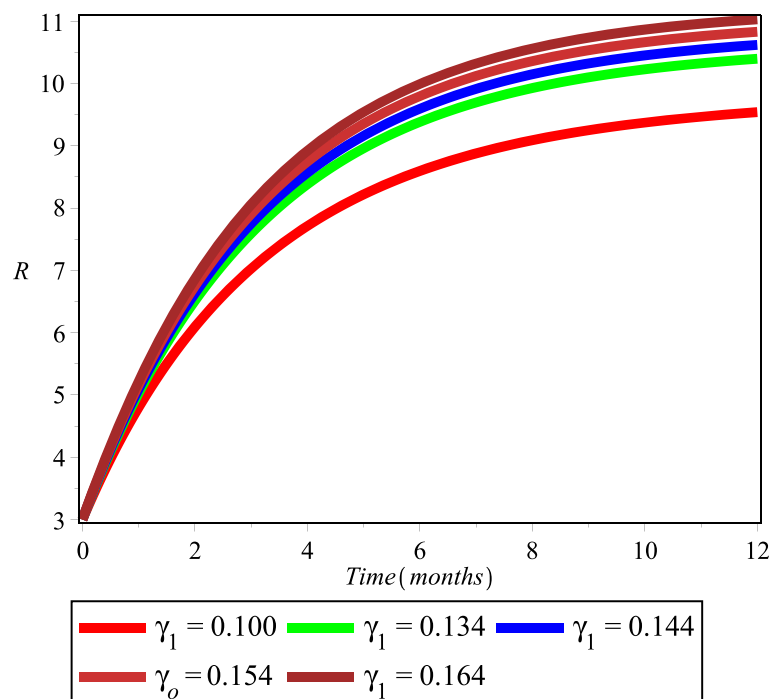
Time(t)	S(t)	E(t)	$I_u(t)$	$I_v(t)$	R(t)
0	50	20	10	7	3
0.2	46.0438184	23.59782685	8.99349350	7.741100	3.5004902
0.4	42.2352922	26.87814813	8.09538109	7.811010	4.1674360
0.6	38.7552644	30.57093498	7.29559566	7.211230	5.0462502
0.8	35.9730239	33.98343857	5.58543315	7.843968	6.1823670
1.0	31.5172160	36.08919010	4.95755349	7.619180	7.6211770

of disease transmission rate β . As time increases, transmission of disease increases, but the steady decline, indicate that more humans are being aware of their disease status leading to quick movement to recovery state or probably death in the case of fatality. It is observed in Fig. 2 that there is a decrease of infection from the 1st–6th month as time increases, but a gradual rise of infection from the 7th–12th month depict that emergence of infection will be on the rise in the absence of health intervention policies. Figures 3 and 4 is the depiction of the high saturation of diseases in human host population as time increases. Strict compliance, increase of medical awareness programs, and treatment are needed to mitigate the high saturation of the disease in human host community, while Figs. 5 and 6 is the depiction of the impact of the variation of treatment rates γ_o and γ_1 in infected humans. As time increases, a steady rise in the

curve indicate that treatment is essential in minimizing infection in human host community.

In addition, simulations of variations of some increased parameter values displayed in Figs. 7, 8, 9, 10, 11, and 12 reveal that the parameter solutions converges to the endemic state, that is, $R_r > 1$. This implies that, for the system not to remain in the endemic state, transmission and new clinical manifestations of disease must be minimized by scaling up treatment and medical awareness program rate.

Figure 13 is the description of the sub-population of susceptible individuals who are at the risk of acquiring the disease. As time increases in the absence of control, more susceptible individuals become exposed to the disease. Figure 14 is the description of the sub-population of exposed individuals who are latently infected. As time increases, there is a quick inflow of exposed individuals

**Fig. 7** Varying $\beta(0.710 - 0.960)$ when $R_r > 1$

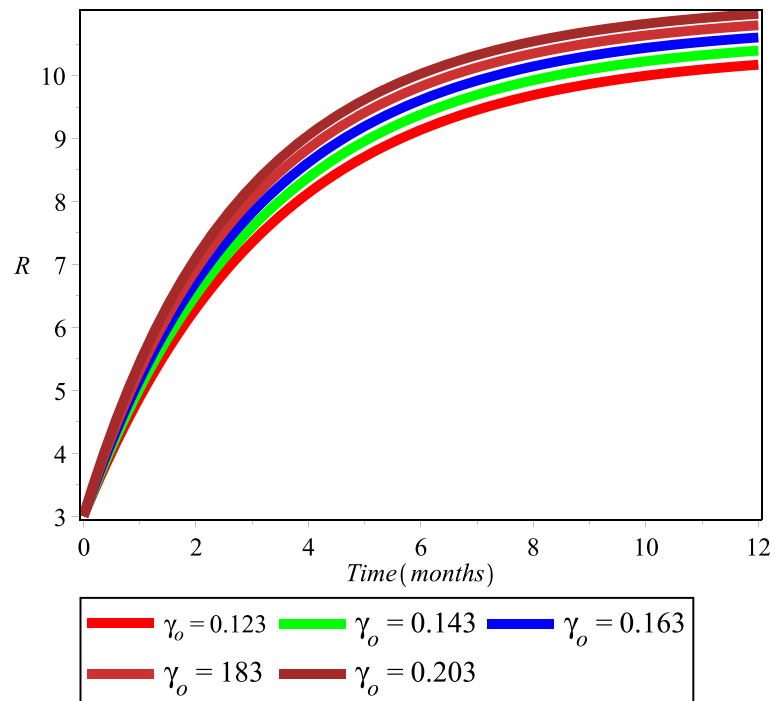


Fig. 8 Varying $\epsilon(0.260 - 0.661)$ when $R_r > 1$

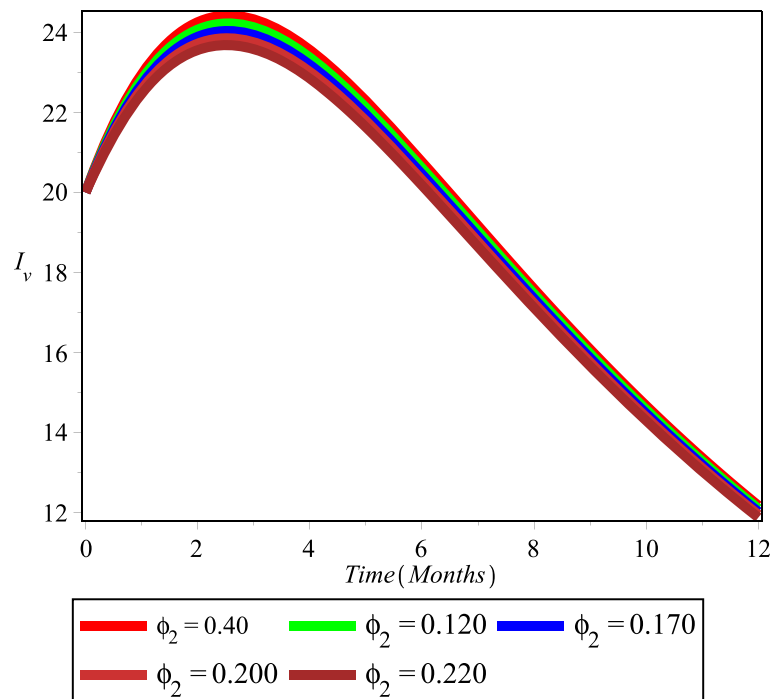


Fig. 9 Varying $\phi_1(0.412 - 0.812)$ when $R_r > 1$

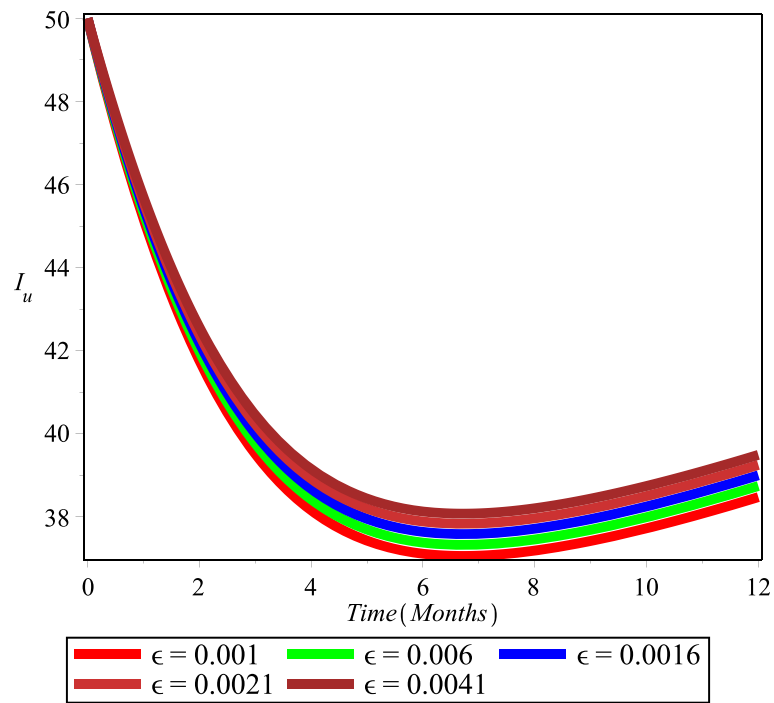


Fig. 10 Varying $\phi_2(0.500 - 0.680)$ when $R_r > 1$

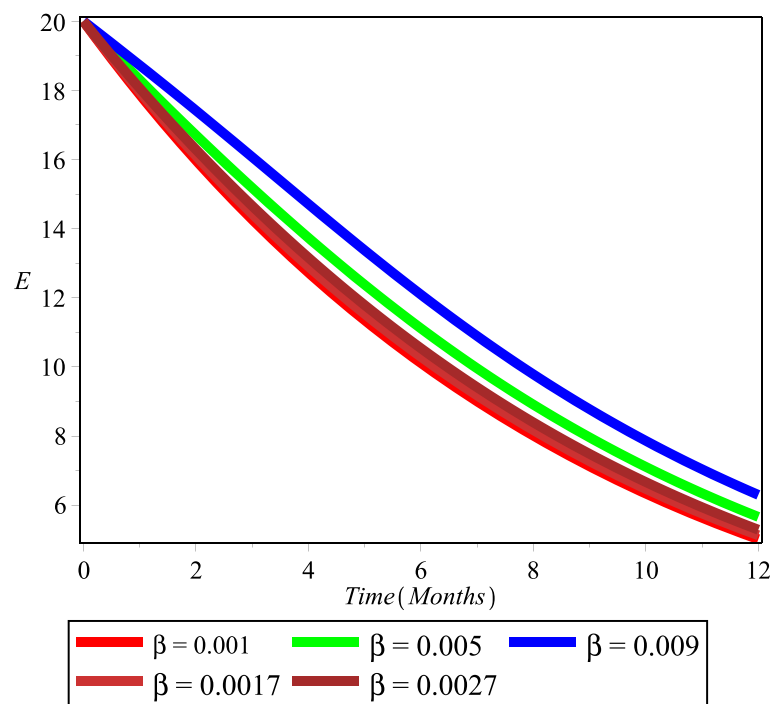


Fig. 11 Varying $\gamma_1(0.300 - 0.436)$ when $R_r > 1$

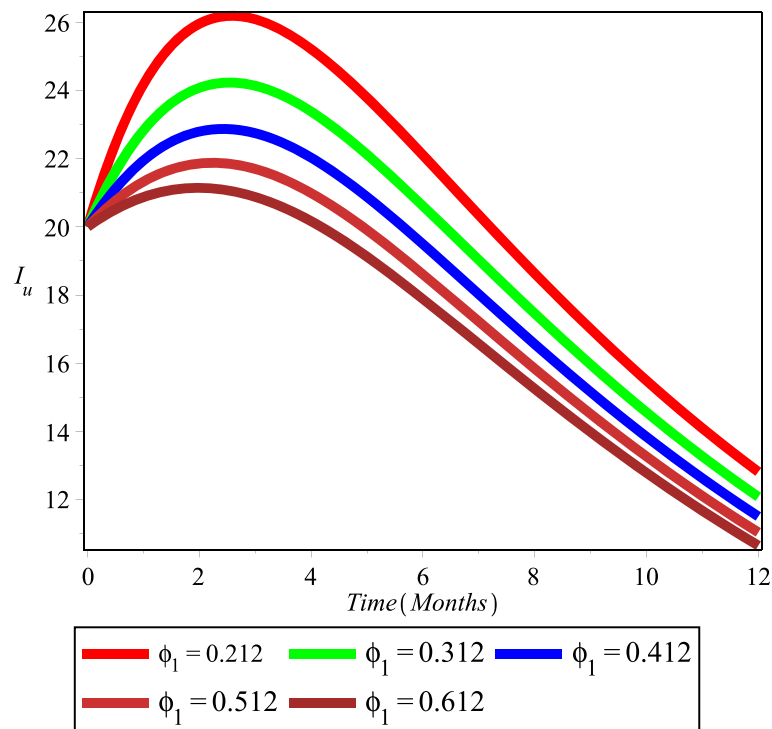


Fig. 12 Varying $\gamma_0(0.420 - 0.490)$ when $R_r > 1$

moving into the infectious class. Figure 15 is the depiction of the behavior of sub-population of infected individuals who attend or are compliant to medical awareness program. As time increases, their compliance to medical awareness program leads to decrease of infected individuals as they are available to treatment, care, etc. Figure 16 is description of the behavior of the sub-population of infected individuals who did not attend or are non-compliant to medical awareness program. As time increases, non compliance to medical awareness program results to fatal cases and probably death. Figure 17 is the description of the behavior of the recovered

sub-population. Recovered humans increases as healthy measures are adopted by infected individuals leading to the reduction and elimination of the disease prevalence in human host community as time increases.

4 Conclusion

A generalized SEIR model describing the transmission of disease in human host community of infected individuals who attend or compliant and who did not attend (non-compliant) medical awareness program is established. Qualitative and quantitative mathematical techniques were used to analyze the invariant region and

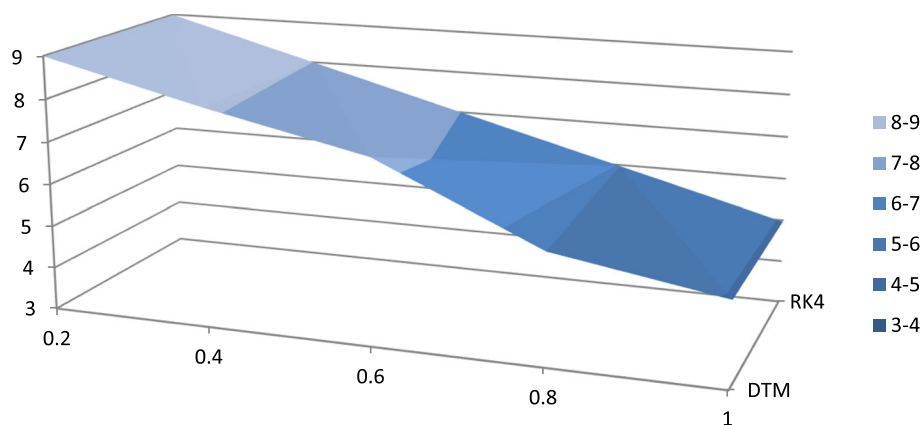


Fig. 13 Description of the sub-population of susceptible individuals who are at the risk of acquiring the disease

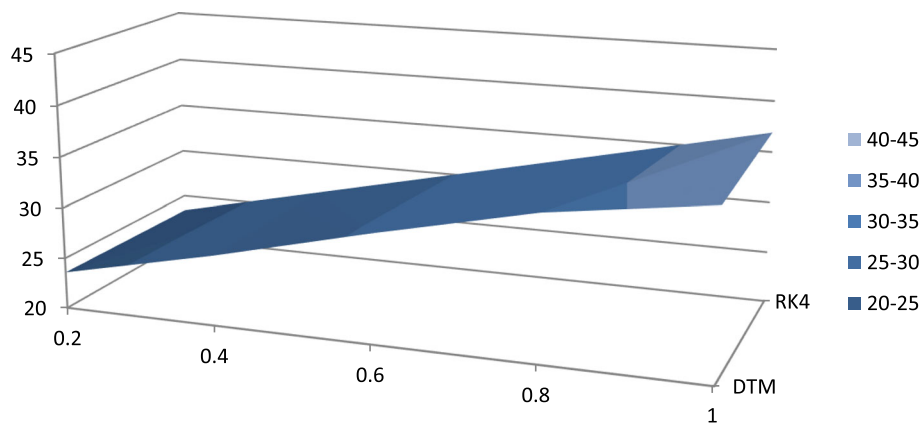


Fig. 14 Description of the sub-population of exposed individuals who are latently infected

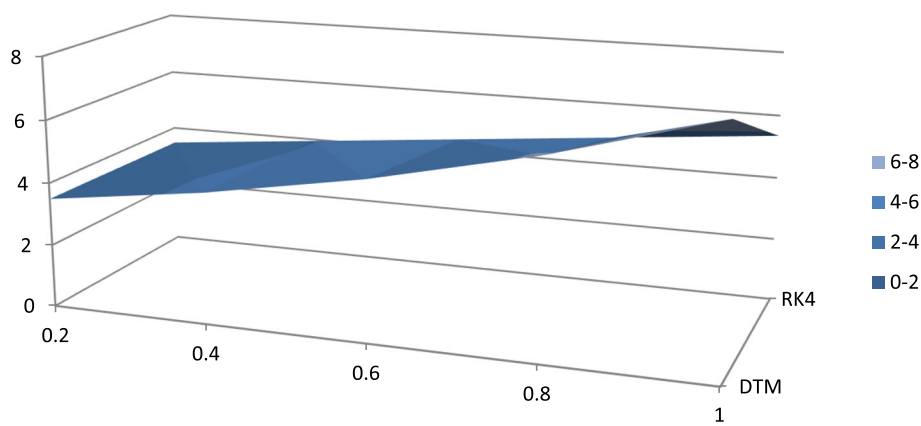


Fig. 15 Depiction of the behavior of sub-population of infected individuals who attend or are compliant to medical awareness program

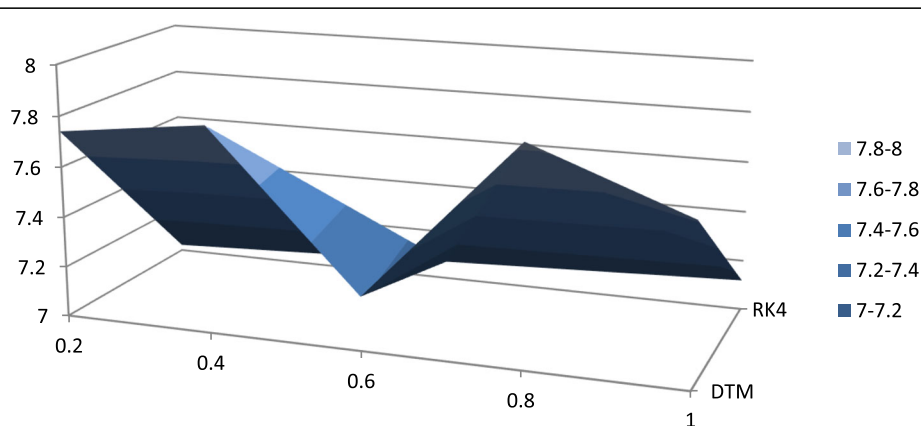


Fig. 16 Description of the behavior of the sub-population of infected individuals who did not attend or are non-compliant to medical awareness program

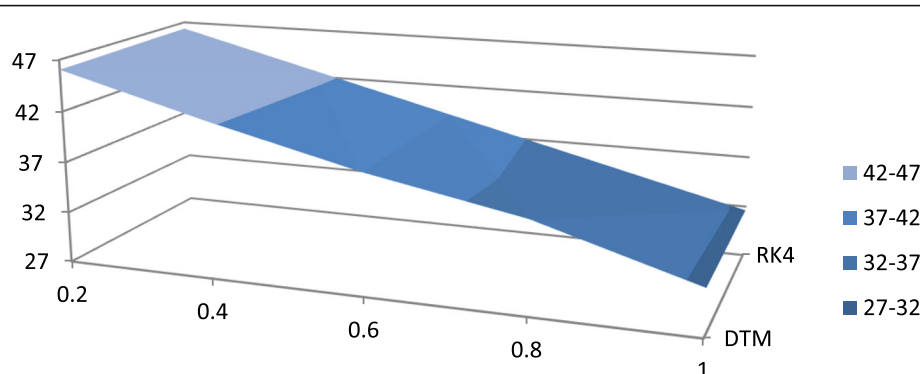


Fig. 17 Description of the behavior of the recovered sub-population

boundedness of the model. Also, the basic reproduction number (R_t) and the equilibria is obtained to show that if $R_t < 1$, the SEIR model disease-free equilibrium is locally and globally asymptotically stable and if $R_t > 1$, the endemic equilibrium solution is globally asymptotically stable. Numerical methods of DTM and Runge-Kutta fourth-order method are employed to obtain the approximate solutions of the model as shown in Tables 2 and 4, which reveal that the two methods agree favorably with each other. Simulations of the model parameters when $R_t < 1$ and $R_t > 1$ is performed in Figs. 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11 and 12 and the graphical behavior of the model in Figs. 13, 14, 15, 16 and 17 reveal that educational awareness about an epidemic breakout is essential in curbing the menace and endemicity of a disease. However, the limitation of this study is that the model cannot incorporate all the complexities involving human behavioral change toward compliance to medical awareness program and lack of real life data involving registration of human individuals that are medical awareness compliant or not in any endemic disease setting. To this end, this work is still recommended further for proper data fit to the model.

Abbreviations

SEIR: Susceptible - Exposed - Infected - Recovered; DTM: Differential Transform Method (DTM)

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Authors' contributions

OM reviewed and established the model as well as discussion of the numerical results, while KH performed the analysis and numerical computations of the work. All authors have read and approved the manuscript.

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References

- Kermack WO, McKendrick AG (1927) A contribution to the mathematical theory of epidemics. *Proc R Soc Lond* 115:700–721 <https://doi.org/10.1098/rspa.1927.0118>
- Anderson RM, May RM (1991) *Infectious disease of humans*. Oxford University Press, London
- Beretta E, Capasso V (1996) On the general structure of an epidemic system. *Comp Math Appl* 12:677–694
- Idowu AS, Ogunmiloro OM (2020) Transmission dynamics of onchocerciasis with two classes of infections and saturated treatment function. *Int J Model Simul Sci Comput* 11(05):2050045 (24 pages) <https://doi.org/10.1142/S1793962320500476>
- Ogunmiloro OM, Idowu AS (2020) On the existence of invariant domain and local asymptotic behaviour of a delayed onchocerciasis model. *Int J Mod Phys C* 2050047:24 <https://doi.org/10.1142/S0129183120501429>
- Ogunmiloro OM (2020) Stability analysis and optimal control of strategies of direct and indirect transmission dynamics of conjunctivitis. *Math Meth Appl Sci*:1–18 <https://doi.org/10.1002/mma.6756>
- Ogunmiloro OM (2019, 2019) Local and global asymptotic behavior of malaria–filariasis co-infections in complaint and non-complaint susceptible pregnant women to antenatal medical programs in the tropics. *e-J Anal Appl Math* (1):31–54 <https://doi.org/10.2478/ejaam-2019-0003>
- Ogunmiloro OM (2019) Mathematical modeling of the co-infection dynamics of malaria-toxoplasmosis in the tropics. *Biomed Lett* 56(2):139–163 <https://doi.org/10.2478/ejaam-2019-0003>
- Bahaa GM (2017) Fractional optimal control problem for variable-order differential system. *Fract Calc Appl Anal* 20(6):1447–1470 <https://doi.org/10.1515/fca-2017-0076>
- Bahaa GM (2017) Fractional optimal control problem for differential system with delay argument. *Adv Differ Equ* 69(2017):1447–1470 <https://doi.org/10.1186/s13662-017-1121-6>
- Bahaa GM (2017) Fractional optimal control problem for variational inequalities with control constraints. *IMA J Math Control Inf* 35(1):107–122 <https://doi.org/10.1093/imamci/dnw040>
- Fagbamigbe AF, Idemudia ES (2015) Barriers to antenatal care use in Nigeria: evidences from non-users and implications for maternal health programming. *BMC Pregnancy Child Birth* 15(95) <https://doi.org/10.1186/s12884-015-0527-y>

13. Makinde AO, Odimegwu CO (2020) Compliance with disease surveillance and notification by private health providers in south west Nigeria. *Pan Afr Med J* 35:114 <https://doi.org/10.11604/pamj.2020.35.114.21188>
14. Diekmann O, Heesterbeek JAP, Roberts MG (2010) Construction of next generation matrices for compartmental epidemic models. *J R Soc Biol Interface* 7:873–885 <https://doi.org/10.1098/rsif.2009.0386>
15. Zhang J, Jia J (2014) Song X (2010) Analysis of an SEIR model with saturated incidence and saturated treatment. *Sci World J* 910421:11 <https://doi.org/10.1155/2014/910421>
16. Alonso-Quesada S, De la Sen M, Nistal R (2018) On vaccination strategies for a SISV epidemic model guaranteeing the nonexistence of endemic solutions. *Discret Dyn Nat Soc* 20:9484121 <https://doi.org/10.1155/2018/9484121>
17. Chitnis N (2017) Introduction to SEIR models, workshop on mathematical models of climate variability, environmental change and infectious diseases, Trieste Italy May 8
18. Ganyani T (2018) Accessing inference of the basic reproduction number in an SIR model incorporating growth scaling parameter. *Stat Med* 37(29): 4490–45067 <https://doi.org/10.1002/sim.7935>
19. LaSalle J, Lifschetz S (1961) Stability of Lyapunov's direct method with applications. Academic, New York
20. Korobeinikov A (2004) Lyapunov functions and global properties for SEIR and SEIS epidemic models. *Math Med Biol* 21:75–83 <https://doi.org/10.1093/imammb21.2.75>
21. He S, Peng Y, Sun K (2020) SEIR modeling of the COVID-19 and its dynamics. *Nonlinear Dyn* <https://doi.org/10.1007/s11071-020-05743-y>
22. Adebimpe O, Abiodun OE, Olubodun O, Gbadamosi B (2020) Dynamics and stability analysis of SEIRS model with saturated incidence rate and treatment. In: 2020 international conference in mathematics, computer engineering and computer science, ICMCECS 2020. Institute of Electrical and Electronics Engineers Inc <https://doi.org/10.1109/ICMCECS47690.2020.246987>
23. Khan MA, Khan Y, Islam S (2018) Complex dynamics of an SEIR epidemic model with saturated incidence rate and treatment. *Physica A Stat Mech Appl* 493:210–227 <https://doi.org/10.1016/j.physa.2017.10.038>
24. Chowell G (2017) Fitting dynamic models to epidemic outbreak with quantified uncertainty, identifiability and forecasts. *Infect Dis Model* 2(3): 379–398 <https://doi.org/10.1016/j.idm.2017.08.001>
25. Side S, Utami AM, Surkana PMI (2018) Numerical solution of SIR model for transmission of tuberculosis by Runge – Kutta method. *J Phys Conf Ser* 1040:0122021 <https://doi.org/10.1088/1742-6596/1040/1/012021>
26. Ogunmiloro OM, Abedo FO, Kareem HA (2019) Numerical and stability analysis of the transmission dynamics of SVIR epidemic model with standard incidence rate. *Malays J Comput* 4(2):349–361 eISSN: 2600-8238 online
27. Tan D, Chen Z (2012) On a general formula for fourth order Runge–Kutta method. *J Math Sci Math Educ* 7(2):1–10
28. Zhou JK (1986) Differential transformation and its applications for Electrical Circuits (in Chinese). Huazhong University Press, Wuhan

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