

NUMERICAL AND STABILITY ANALYSIS OF THE TRANSMISSION DYNAMICS OF SVIR EPIDEMIC MODEL WITH STANDARD INCIDENCE RATE

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ABSTRACT

In this article, a Susceptible – Vaccinated – Infected – Recovered (SVIR) model is formulated and analysed using comprehensive mathematical techniques. The vaccination class is primarily considered as means of controlling the disease spread. The basic reproduction number (R_0) of the model is obtained, where it was shown that if $R_0 < 1$, at the model equilibrium solutions when infection is present and absent, the infection-free equilibrium is both locally and globally asymptotically stable. Also, if $R_0 > 1$, the endemic equilibrium solution is locally asymptotically stable. Furthermore, the analytical solution of the model was carried out using the Differential Transform Method (DTM) and Runge - Kutta fourth-order method. Numerical simulations were carried out to validate the theoretical results.

Keywords: SVIR epidemic model, Reproduction number, Local stability, Global stability, DTM, Runge-Kutta

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1. Introduction

The classical susceptible – infectious – recovered (SIR) model originated from the seminar papers of Ross (1911) and one of the earliest fundamental contributions which provide basic framework for almost all later epidemic model was carried out by Kermack and Mckendrick, (1927). One of the modifications to SIR models is the introduction of vaccination compartment to study more complex disease and infection mechanism. The World Health Organization WHO (2018), reported that licensed vaccines are currently available to prevent or contribute to the prevention and control of twenty-five infections. Several mathematical models describing the transmission of disease and controls have been formulated. Wang *et al.*, (2017), considered a global threshold dynamic of an SVIR epidemic model with age dependent infection and relapse. Li (1999); Fan (2001) and Sun (2010), studied the global dynamics of SEIR models with varying population size and vaccination respectively. Klouach and Boulasair (2018), worked on the stochastic SVIR epidemic model with imperfect vaccine, also, a new epidemic model with indirect transmission was analytically discussed by Brauer (2017).

Moreover, Yang *et al.*, (2010) discussed the global analysis for a general epidemiological model with vaccination and varying populations, while Liu *et al.*, (2015) worked on the global stability with age dependent latency with relapse. Gani and Shreedevi (2017), applied optimal control strategies to a SIVR epidemic model. (El-Koufi *et al.*, 2019), considered the analysis of a stochastic SIR model with vaccination and nonlinear incidence rate. Also, (Ogunmiloro *et al.*, 2018), worked on the stability analysis and optimal control of vaccination and treatment of a SIR epidemiological deterministic model with relapse, while the

work of (Sudhipa *et al.*, 2014), on stability analysis of SIR model with vaccination and Cui and Zhang (2014) on global discrete SIR epidemic model with vaccination proved effective to this study. (Zhou, 1986) used DTM to solve linear and nonlinear initial value problems in electric circuit analysis and it has since be applied as a powerful to numerical tool to solve epidemic models, see (Idowu *et al.*, 2018) and (Ahmad *et al.*, 2017). All the articles cited have proved very useful to this study. Having gone through the articles cited, we formulated a SIVR epidemic model with standard incidence rate with some important parameters incorporated into the model. The model is analyzed and solved using qualitative and quantitative mathematical theorems and methods. Section 2 deals with the mathematical model formulation, analysis of the invariant region and positivity of the solutions. Section 3 involves, obtaining the equilibrium solutions at infection-free and infection-present states. Also, the reproduction number of model is obtained. Section 4 involves the local and global stability analysis of the model. Section 5 deals with the analytical solution of the model using the DTM and Runge – Kutta fourth order method.

2. Mathematical Model Formulation

The total human host population is subdivided into the population of susceptible individuals $S(t)$; infected individuals $I(t)$; vaccinated individuals $V(t)$; recovered individuals $R(t)$. The following parameters were incorporated into the model formulation as follows:

The susceptible host sub-population is increased by recruitment rate of susceptible individuals denoted A , while β is the transmission rate of infections between the susceptible and the infected individuals which leads to a reduction in the susceptible sub-population. Also, the susceptible sub-population is further reduced by the notation ρ which denotes the fraction of susceptible individual who are vaccinated and the term $(1 - \rho)$ which refers to the fraction of susceptible individuals that are not vaccinated and δ_1 denotes the rate at which vaccination wanes in vaccinated individuals' overtime. μ represents the natural death rate applicable to all sub-population of individuals in the total host population. In the sub-population of infected individuals, α denotes the disease induced death rate and γ represents the recovery rate of infected individuals. The assumptions guiding the model build up are that, there is permanent recovery, there is birth rate and the natural death rate is applicable to all sub-populations of individuals in the total human host population and vaccinations received by fractions of susceptible individuals wane overtime. The mathematical model derived after the incorporation of the assumptions, variables and parameters is given by

$$\begin{aligned} \frac{dS}{dt} &= A - \beta SI - [\rho + (1 - \rho)]S - \mu S + \delta_1 V, \\ \frac{dI}{dt} &= \beta SI - (\mu + \alpha + \gamma)I, \\ \frac{dV}{dt} &= \rho S - \mu V - \delta_1 V, \\ \frac{dR}{dt} &= \gamma I - \mu R. \end{aligned} \tag{1}$$

Subject to the initial conditions $S(0) = S_o, I(0) = I_o, V(0) = V_o, R(0) = R_o$.

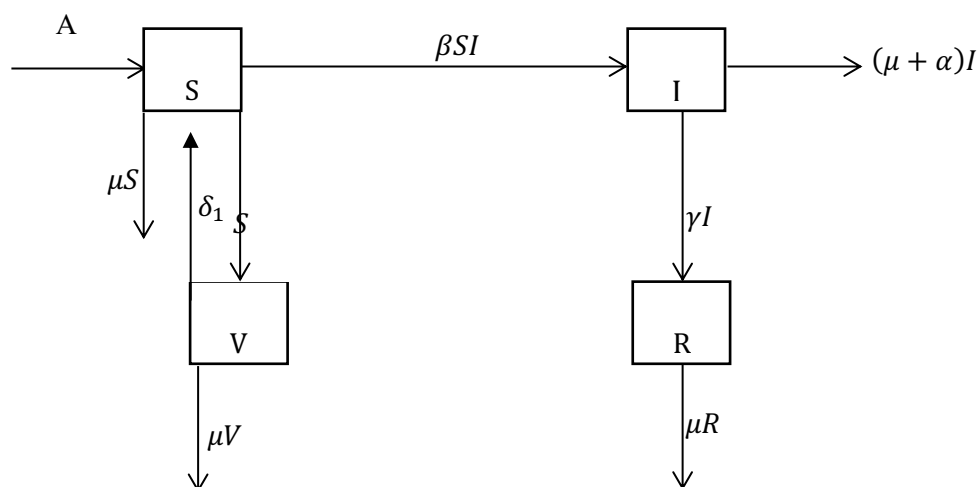


Figure. 1. Flow diagram of the model

2.1 Invariant Region

The positively invariant region is shown to be bounded by adding up the total human host population $N(t)$ such that

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dV}{dt} + \frac{dR}{dt}. \quad (2)$$

Addition of the model system equations in (2) yields

$$\frac{dN}{dt} = A - (1 - \rho)S - \alpha I - \mu N(t) \quad (3)$$

In the absence of disease induced death rate i.e., $\alpha = 0$, since the total human host population is constant, for convenience we assume that $N = S + I + V + R = 1$ and $S = N - I - V - R = 1$. Therefore (3) yields

$$\frac{dN}{dt} = A - (1 - \rho) - \mu N(t) \quad (4)$$

so that

$$\frac{dN}{dt} \leq A - (1 - \rho) - \mu N(t). \quad (5)$$

Moreover,

$$\int \frac{dN}{A - (1 - \rho) - \mu N(t)} \leq \int dt. \quad (6)$$

so that

$$\ln(A - (1 - \rho) - \mu N(t)) \geq t + C_1. \quad (7)$$

and

$$A - (1 - \rho) - \mu N(t) \geq e^{-\mu t} e^{c_1} \tag{8}$$

so that as $t = 0$, and $N(0) = N_o$ in (8) becomes

$$A - (1 - \rho) - \mu N(t) \geq e^{c_1} \tag{9}$$

Substituting (9) into (8) yields

$$A - (1 - \rho) - \mu N(t) \geq (A - (1 - \rho) - \mu N_o) e^{-\mu t} \tag{10}$$

And

$$A - \mu N(t) \geq A - (1 - \rho) - [A - (1 - \rho) - \mu N_o] e^{-\mu t}. \tag{11}$$

Since $N(0) = N_o$ and A is a constant, after simple re-arrangement and simplification, yields

$$N(t) \leq \frac{A-(1-\rho)}{\mu} - \left[\frac{A-(1-\rho)-\mu N_o}{\mu} \right] e^{-\mu t}. \tag{12}$$

As $t \rightarrow \infty$ in (12), the population $N(t) \rightarrow \frac{A-(1-\rho)}{\mu}$ implies that, $0 \leq N(t) \leq \frac{A-(1-\rho)}{\mu}$. Thus, the feasible solution set of the model system equations enters and remain in the region

$$\Omega = \left\{ (S, I, V, R) \in \mathbb{R}_+^4 : N \leq \frac{A-(1-\rho)}{\mu} \right\}. \tag{13}$$

The basic model is reasonable in an epidemic sense and mathematically well posed.

2.2 Positivity of the Model Solutions

Let $\Omega = \{(S, I, V, R) \in \mathbb{R}_+^4 : S_o > 0, I_o > 0, V_o > 0, R_o > 0\}$, then the solutions of $\{S, I, V, R\}$ are positive for time $t \geq 0$.

Taking the first equation in the model system equations (1)

$$\frac{dS}{dt} = A - (\beta I + [\rho + (1 - \rho)] + \mu)S \tag{14}$$

Integrating both sides of (14) yields

$$\ln S(t) \geq -(\beta I + [\rho + (1 - \rho)] + \mu)t + c, \tag{15}$$

where

$$S(t) \geq e^c e^{-(\beta I + [\rho + (1 - \rho)] + \mu)t} \geq 0 \tag{16}$$

and

$$S(t) \geq S_o e^{-(\beta I + [\rho + (1 - \rho)] + \mu)t} \geq 0. \tag{17}$$

From the initial condition that $S(0) = S_0$, (17) is positive. Applying the same procedure to the remaining state equations, the following are obtained as

$$I(t) \geq I_0 e^{-(\beta S + \mu + \alpha + \gamma)t} \geq 0, \tag{18}$$

$$V(t) \geq V_0 e^{-\mu t} \geq 0, \tag{19}$$

$$R(t) \geq R_0 e^{-\mu t} \geq 0. \tag{20}$$

Hence, the model solutions of (17), (18), (19), (20) are positive at time $t \geq 0$.

3. Equilibrium Solutions and Reproduction Number (R_0)

3.1 Equilibrium Solutions

The equilibrium solutions of the model system is obtained at the time-independent solutions, when infection is free and absent in the human host population. The infection-free equilibrium solutions are given by

$$E_0 = (S, I, V, R) = \left(\frac{A}{[\rho + (1-\rho) - \mu]}, 0, \frac{\rho A}{(\mu + \delta_1 [\rho + (1-\rho) - \mu])}, 0 \right) \tag{21}$$

Also, the endemic equilibrium solutions which occurs when infection persist in the human host population are given by

$$E^* = (S^*, I^*, V^*, R^*) \tag{22}$$

where

$$S^* = \frac{V^* \delta_1 A}{\beta I + \mu + 1}, I^* = \frac{\beta S^*}{\mu + \alpha + \gamma},$$

$$V^* = \left(\frac{\rho}{\mu + \delta_1} \right) \left(\frac{V^* \delta_1 A}{\beta I + \mu + 1} \right), R^* = \frac{\gamma}{\mu} \frac{\beta S^*}{\mu + \alpha + \gamma}. \tag{23}$$

3.2 Reproduction Number (R_0)

The reproduction number is the average number of secondary infections produced per unit time when a single infectious individual is introduced into the population of susceptible during his or her lifetime of infections. If $R_0 < 1$, infection dies out of the human host population. If $R_0 > 1$, then infection becomes endemic which necessitates some forms of control measures to curtail the disease spread. To obtain the R_0 of this model system, we define $X_S = \{X = 0 | X_i, i = 1, 2, 3 \dots n\}$, such that new infections are distinguished from other changes in the populations. Then, $F_i(x)$ is the rate of new manifestations of clinical symptoms in compartment i . Also, let V_i^+ be the rate at which individuals transfer out of compartment. 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. Therefore;

$$V^+ = \begin{bmatrix} A + \delta_1 V \\ 0 \\ \rho S \\ \gamma I \end{bmatrix}, \quad V^- = \begin{bmatrix} [\rho + (1 - \rho)]S - \mu S \\ -(\mu + \alpha + \gamma) I \\ -(\mu + \delta_1)V \\ -\mu R \end{bmatrix} \tag{24}$$

and

$$V = V^- - V^+ = \begin{bmatrix} A + \delta_1 V - [\rho + (1 - \rho)]S - \mu S \\ (\mu + \alpha + \gamma) I \\ \rho S + (\mu + \delta_1)V \\ \gamma I + \mu R \end{bmatrix}. \tag{25}$$

Also,

$$F = \begin{bmatrix} 0 & 0 & 0 & 0 \\ 0 & \beta \left(\frac{A}{[\rho + (1 - \rho) + \mu]} \right) & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & \gamma & 0 & 0 \end{bmatrix} \tag{26}$$

And

$$V = \begin{bmatrix} m_1 & 0 & 0 & 0 \\ 0 & m_2 & 0 & 0 \\ \rho & 0 & m_3 & 0 \\ 0 & \gamma & 0 & \mu \end{bmatrix}$$

where $m_1 = [\rho + (1 - \rho) - \mu], m_2 = (\mu + \alpha + \gamma), m_3 = (\mu + \delta_1)$. Therefore, the largest eigenvalue of $F.V^{-1} = R_0$, where

$$R_0 = \left[\frac{\beta A}{(\rho + \mu)(\mu + \alpha + \gamma)} \right]. \tag{27}$$

Is the basic reproduction number of the model system equations (1).

3.3 Local Stability of The Infection - Free Equilibrium

Theorem: The Infection-free equilibrium is locally asymptotically stable if $R_0 < 1$.

Proof: The Jacobian matrix J of the model system (1) at the disease free equilibrium yields

$$J = \begin{bmatrix} -(\rho + (1 - \rho) - \mu) - \lambda & -\beta \frac{A}{[\rho+(1-\rho)-\mu]} & \delta_1 & 0 \\ 0 & \left(\beta \left(\frac{A}{[\rho+(1-\rho)-\mu]} \right) - (\mu + \alpha + \gamma) \right) - \lambda & 0 & 0 \\ \rho & 0 & -(\mu + \delta_1) - \lambda & 0 \\ 0 & \gamma & 0 & -\mu - \lambda \end{bmatrix} \tag{28}$$

The characteristics polynomial of (28) yields

$$\frac{(-(\mu+\rho+\lambda)+(-\mu^2+(\rho-\alpha-\gamma-\lambda)\mu+(-\alpha-\gamma-\lambda)\rho+\beta A)(\mu+\lambda)^2(1-R_0))}{\rho+\mu} \tag{29}$$

The trace and determinant of (28) are respectively given by

$$-\rho - 4\mu + \frac{\beta A}{\rho+\mu} - \alpha - \gamma < 0 \tag{30}$$

and

$$\frac{(-\rho-\mu)\mu^2(A\beta-\alpha\mu-\alpha\rho-\gamma\mu-\gamma\rho-\mu^2-\mu\rho)}{\rho+\mu} > 0. \tag{31}$$

In (28), all the real parts are negative except

$$\beta \left(\frac{A}{[\rho+(1-\rho)-\mu]} \right) - (\mu + \alpha + \gamma) \tag{32}$$

which implies that, for R_0 to be less than unity,

$$\beta \left(\frac{A}{[\rho+(1-\rho)-\mu]} \right) - (\mu + \alpha + \gamma) > \frac{(\mu+\alpha+\gamma)}{(\mu+\alpha+\gamma)}. \tag{33}$$

so that

$$R_0 - 1 > 0, -R_0 > -1, R_0 < 1. \tag{34}$$

Hence, since $R_0 < 1$, the infection-free equilibrium is locally asymptotically stable.

3.4 Global Stability of The Infection-Free Equilibrium

Theorem: The infection-free equilibrium is locally asymptotically stable if and only if $R_0 < 1$.

Proof: Given that $R_0 < 1$, there exists only the infection free equilibrium E^0 . Consider a Lyapunov function candidate of the form $V(S, I, V): R^3 \rightarrow R^+$ defined as

$$V(S, I, V) = \psi I, \quad \psi > 0 \tag{35}$$

Substituting the second equation in (1) into (35) yields

$$\dot{V} = \psi[(\beta S - \mu + \alpha + \gamma)I] \tag{36}$$

Since $E_o = S = \frac{A}{[\rho+(1-\rho)-\mu]}$, let $\psi = \frac{1}{(\mu+\alpha+\gamma)}$ so that, In the absence of disease i.e., $I = 0$,

$$\dot{V} = \psi\left(\frac{\beta A - (\rho+\mu)(\mu+\alpha+\gamma)}{\rho+\mu}\right)I \tag{37}$$

$$\dot{V} = [R_o - 1]I \leq 0 \tag{38}$$

$\dot{V} \leq 0$ for $R_o < 1$ and $\dot{V} = 0$ if and only if $I = 0$. Hence, the infection-free equilibrium is globally asymptotically stable.

4. Numerical Solution of The SIVR Model (1)

In this section, the numerical solution of the model system (1) is obtained by solving it by using the Runge - Kutta 4th order and Differential Transform Method (DTM). This idea was first introduced by Zhou (1986) for solving linear and nonlinear initial value problems in electrical circuit analysis. The method had since been applied to solve a variety of problems that are modelled with differential equations. The concept of differential transformation is derived from the Taylor series expansion. In this method, given system of differential equations and related initial conditions are transformed into a system of recurrence equations that finally leads to a system of algebraic equations whose solutions are the coefficients of a power series solution. Taylor series polynomial of a degree n is defined as

$$P_n(x) = \sum_{k=0}^n \frac{1}{k!} (f^k(c)(x - c)^k) = 0. \tag{39}$$

Suppose that the function f has $(n + 1)$ derivatives on the interval $(c - r, c + r)$, for some $r > 0$ and $\lim_{n \rightarrow \infty} R_n(x)$ is the error between $p_n(x)$ and the approximated function $f(x)$ then the Taylor series expanded about $x = c$ converges to $f(x)$ that is;

$$f(x) = \sum_{k=0}^n \frac{1}{k!} (f^k(c)(x - c)^k) = 0. \text{ For all } x \in (c - r, c + r). \tag{40}$$

Definition 1: Ahmed et al., (2017), Idowu et al., (2018). The differential transformation of the function $f(x)$ for the k^{th} derivative is defined as

$$f(x) = \frac{1}{k!} \left[\frac{d^k f(x)}{dx^k} \right]_{x=x_o} \tag{41}$$

where $f(x)$ is the original function and $F(k)$ is the transformed function.

Definition 2: (Ahmad et al., 2017), (Idowu et al., 2018). The inverse differential transformation $F(k)$ is given by

$$f(x) = \sum_{k=0}^{\infty} (x - x_o)^k F(k). \tag{42}$$

Substituting (41) and (42), yields

$$f(x) = \sum_{k=0}^{\infty} (x - x_0)^k \frac{1}{k!} \left[\frac{d^k f(x)}{dx^k} \right]_{x=x_0}. \tag{43}$$

Equation (43) is the Taylor's series of $f(x)$ at $x = x_0$. The fundamental operations can be deduced from (41), (42), (43) as listed below. See (Ahmed et al., 2017).

1. $f(x) = g(x) \pm h(x)$, then $F(k) = G(k) \pm H(k)$
2. If $f(x) = c g(x)$, then $F(k) = c G(k)$, where c is a constant
3. If $f(x) = \frac{dg(x)}{dx}$, then $F(k) = (k + 1)G(k + 1)$
4. If $f(x) = \frac{d^m g(x)}{dx^m}$, then $Y(k) = (k + 1)(k + 2)\dots(k + m)G(k + m)$.
5. If $f(x) = 1$, then $F(k) = \delta(k)$.
6. If $f(x) = x$, then $F(k) = \delta(k - 1)$.
7. If $f(x) = x^m$, then $F(k) = \delta(k - m) = \begin{cases} 1, & \text{if } k = m \\ 0, & \text{if } k \neq m. \end{cases}$
8. If $f(x) = g(x) h(x)$, then $F(k) = \sum_{m=0}^k H(m)G(k - m)$
9. If $f(x) = e^{mx}$, then $F(k) = \frac{m^k}{k!}$.
10. If $f(x) = (1 + x)^m$, then $F(k) = \frac{m(m-1)(m-2)\dots(m-k+1)}{k!}$ (44)

Now, we consider the SIVR Model given by equation (1) with the initial conditions and parameter values as: $S(0) = 50, I(0) = 10, V(0) = 20, R(0) = 30$ and parameter value $\beta = 0.05, A = 0.0123, \rho = 0.24, \mu = 0.112, \alpha = 0.3, \gamma = 0.312, \delta_1 = 0.011$.

Let $S(k), I(k), V(k)$ and $R(k)$ denote the differential transformation of $S(t), I(t), V(t)$ and $R(t)$ respectively, then by using the fundamental operations of differential transformation method. We obtained the following recurrence relation to each equation of the model system (1)

$$S(k + 1) = \frac{1}{k+1} [A - [\rho + (1 - \rho) - \mu]S(k) - \beta \sum_{m=0}^k S(m)I(k - m)] + \delta_1 V(k), \tag{45}$$

$$I(k + 1) = \frac{1}{k+1} [-(\mu + \alpha + \gamma)I(k) + \beta \sum_{m=0}^k S(m)I(k - m)], \tag{46}$$

$$V(k + 1) = \frac{1}{k+1} [\gamma I(k) - (\mu + \delta_1)V(k)], \tag{47}$$

$$R(k + 1) = \frac{1}{k+1} [\gamma I(k) - \mu R(k)]. \tag{48}$$

Applying the values of parameters and initial conditions, the closed form solution, when $k=7$ are given by the following as;

$$S(t) = \sum_{m=0}^k t^m S(k) = 50 - 42.5877t - 227.6369148t^2 - 819.889052t^3 - 184.2728757t^4 + 16044.28588t^5 + 99195.61185t^6 + 232603.6549t^7, \tag{49}$$

$$I(t) = \sum_{m=0}^k t^m I(k) = 10 + 17.76t - 197.16762t^2 - 1127.632131t^3 - 1461.870336t^4 + 14781.11872t^5 + 111163.5841t^6 + 309595.0066t^7, \tag{50}$$

$$V(t) = \sum_{m=0}^k t^k V(k) = 20 - 37.74t + 13.0051935t^2 + 53.38854263t^3 + 36.00885688t^4 + 25.36091675t^5 - 1899.04723t^6 - 10030.88445t^7, \tag{51}$$

$$R(t) = \sum_{m=0}^k t^k R(k) = 30 - 0.24t + 2.784t^2 - 20.60936848t^3 - 87.37824368t^4 - 89.2634363t^5 + 770.2844243t^6 + 4942.395198t^7. \tag{52}$$

4.1 Numerical Results and Graphical Illustrations

In this section, the numerical results of the model system are presented in Tables 1 and 2, obtained from the numerical solutions of the model using Runge - Kutta 4th order and Differential Transform Method (DTM). The results compared favourably with each other and the plots are shown below:

Table 1. Numerical results of the model system equations using DTM

Time(t)	S(t)	I(t)	V(t)	R(t)
0	50	10	20	30
0.2	41.28304317	10.11248907	13.2151140	30.01609462
0.4	32.78142716	17.24039028	11.6298719	30.11609462
0.6	24.14789101	20.81490719	7.40975069	30.29230922
0.8	15.06439541	28.01207890	2.55292702	30.74353763
1.0	11.01690514	29.29564134	1.47181908	30.07705655

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

Time(t)	S(t)	I(t)	V(t)	R(t)
0	50	10	20	30
0.2	41.40762143	13.66517011	15.8642119	30.66536487
0.4	32.81524285	17.33034021	11.72924238	30.13072974
0.6	24.2286428	20.99551032	7.59386357	30.19609462
0.8	15.63048571	24.66068043	2.45848476	30.26145949
1.0	11.03810713	29.03810713	1.57689405	30.32682435

The results are further described in Figures 1,2,3 and 4. Figure 1 describes the behaviour of susceptible subpopulation with time. As time increases, the gradual decline depicts that there is a quick inflow of susceptible individuals becoming infected as they come in contact with the infected except the fraction of those that are vaccinated. At the same time, Figure 2 describes the behaviour of infected subpopulation with time. As time increases, the gradual rise depicts that in the absence of interventions, more human individuals get infected.

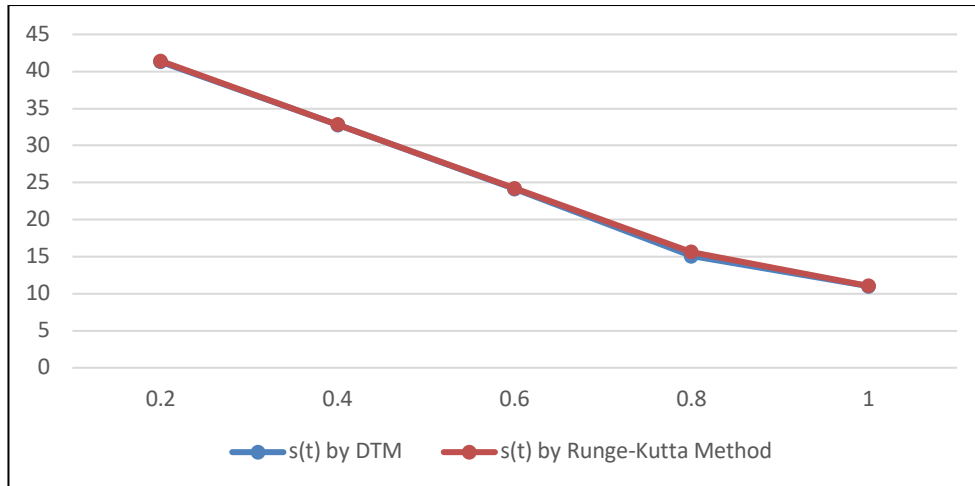


Figure. 1. S(t) against Time(t)

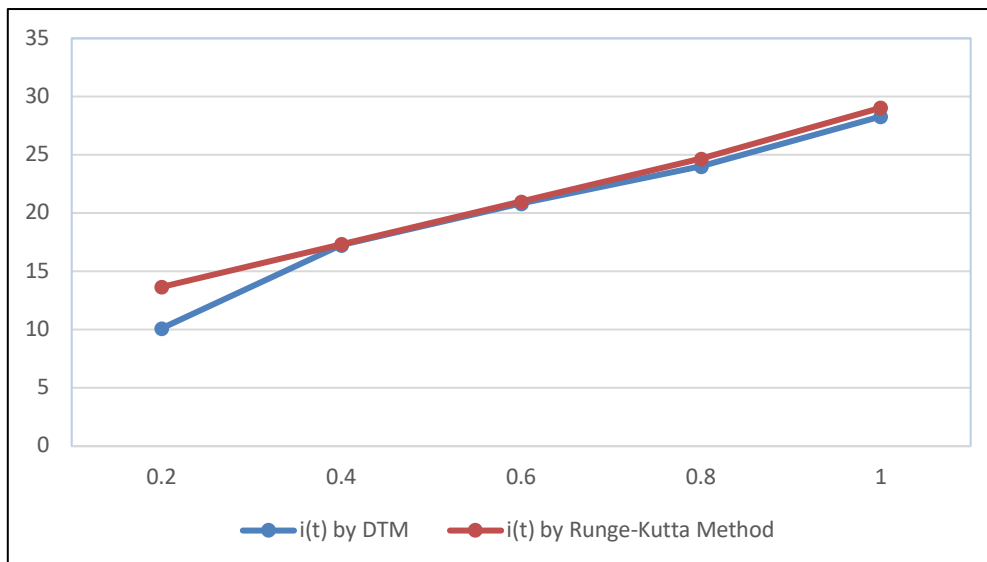


Figure. 2. I(t) against Time(t)

Figure 3 describes the behaviour of vaccinated sub-population with time. As time increases, the gradual decline depicts that as more human individuals get vaccinated, infection becomes low in the human host community. Similarly, Figure 4 describes the behaviour of the recovered sub-population with time. As time increases, the behaviour shows that more human individuals recover with compliance to vaccination and medical intervention strategies.

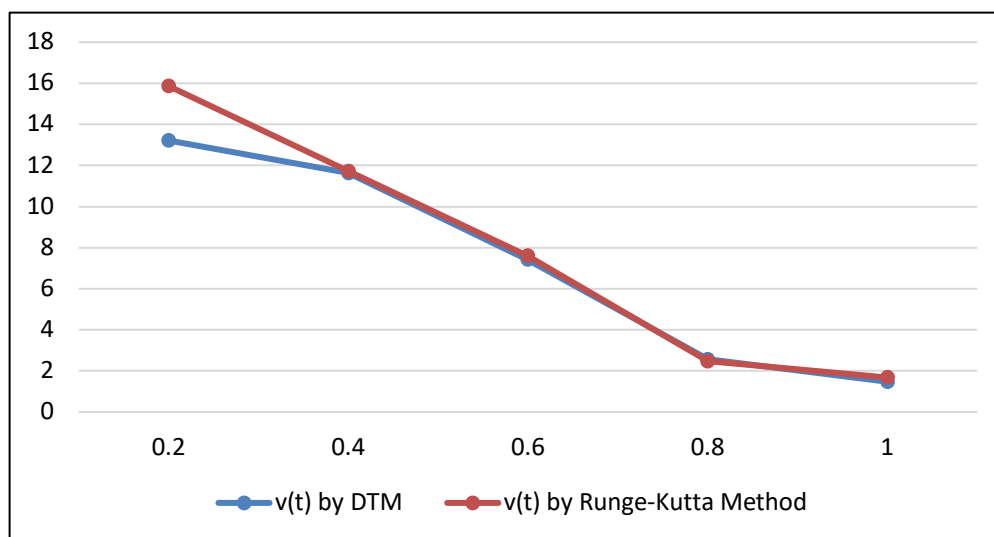


Figure. 3. V(t) againstTime (t)

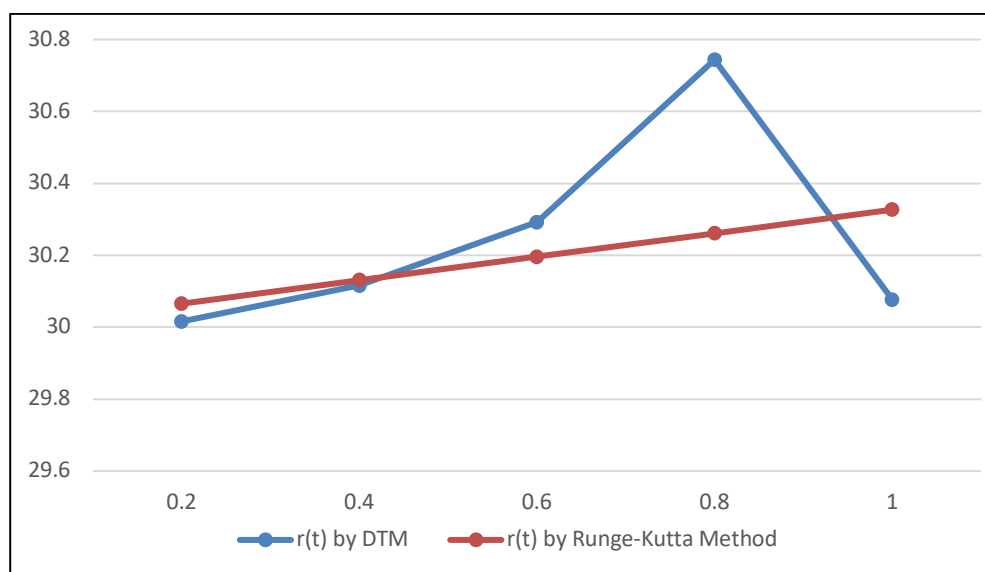


Figure. 4. R(t) againstTime (t)

4.2 Conclusion and Recommendations

The differential transformation method is an efficient way to solve SIVR epidemic model when either computation or iteration is costly. That is, it is capable of reducing the size of computational work and still accurately provides the series solution with faster convergence rate. In this paper, a mathematical epidemic model of SIVR is formulated based on a system of first order differential equation. The model is analyzed in a positively invariant region. The reproduction number (R_0) of the model is obtained via the next generation matrix method, it was shown that if $R_0 < 1$, the model is locally stable at the infection-free equilibrium solutions. The numerical solution of the model is obtained by using Differential Transform Method (DTM) and Runge - Kutta fourth order method. The numerical results obtained show that it compares favourably with each other and that DTM method perform better. It is recommended

that this work can be further extended into an optimal control problem, age structure, fitting a real life data on some epidemic to the model considered.

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