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 (Ro) of the model is obtained, where it was shown that if Ro<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 Ro>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 \( \beta \) 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 \( \rho \) 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 \( \delta \) denotes the rate at which vaccination wanes in vaccinated individuals’ overtime. \( \mu \) represents the natural death rate applicable to all sub-population of individuals in the total host population. In the sub-population of infected individuals, \( \alpha \) denotes the disease induced death rate and \( \gamma \) 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

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

(1)
Subject to the initial conditions $S(0) = S_0$, $I(0) = I_0$, $V(0) = V_0$, $R(0) = R_0$.

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

$$\frac{dN}{dt} = \frac{ds}{dt} + \frac{dv}{dt} + \frac{dr}{dt}. \quad (2)$$

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

\[ A - (1 - \rho) - \mu N(t) \geq e^{-\mu t} e^{c_1} \]  \hspace{1cm} (8)

so that as \( t = 0 \), and \( N(0) = N_0 \) in (8) becomes

\[ A - (1 - \rho) - \mu N(t) \geq e^{c_1} \]  \hspace{1cm} (9)

Substituting (9) into (8) yields

\[ A - (1 - \rho) - \mu N(t) \geq (A - (1 - \rho) - \mu N_0)e^{-\mu t} \]  \hspace{1cm} (10)

And

\[ A - \mu N(t) \geq A - (1 - \rho) - [A - (1 - \rho) - \mu N_0]e^{-\mu t}. \]  \hspace{1cm} (11)

Since \( N(0) = N_0 \) and \( A \) is a constant, after simple re-arrangement and simplification, yields

\[ N(t) \leq \frac{A - (1 - \rho)}{\mu} - \left[ \frac{A - (1 - \rho) - \mu N_0}{\mu} \right] e^{-\mu t}. \]  \hspace{1cm} (12)

As \( t \to \infty \) in (12), the population \( N(t) \to \frac{A - (1 - \rho)}{\mu} \) 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

\[ \Omega = \left\{ (S, I, V, R) \in \mathbb{R}^4 : N \leq \frac{A - (1 - \rho)}{\mu} \right\}. \]  \hspace{1cm} (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)

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

Integrating both sides of (14) yields

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

where

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

and

\[ S(t) \geq S_0 e^{-(\beta I + [\rho + (1 - \rho)] + \mu)t} \geq 0. \]  \hspace{1cm} (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

$$I(t) \geq I_0 e^{-(bS+\mu+\gamma)dt} \geq 0,$$

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

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

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

3. Equilibrium Solutions and Reproduction Number ($R_o$)

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

$$E_0 = (S, I, V, R) = \left( \frac{A}{\rho+(1-\rho)-\mu}, 0, \frac{\rho A}{\mu+\delta}, 0 \right).$$

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

$$E^* = (S^*, I^*, V^*, R^*)$$

where

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

$$V^* = \left( \frac{\rho}{\mu+\delta} \right) \left( \frac{V^* A}{\beta I + \mu + 1} \right), R^* = \frac{\gamma}{\mu+\alpha+\gamma}.$$
\[ x_i = F_i(x) - V_i(x), i = 1, 2, 3 \ldots \text{ and } V_i(x) = V_i^- - V_i^+ \]. F is a non-negative matrix and V is a non-singular matrix. Therefore;

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

(24)

and

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

(25)

Also,

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

(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

\[
R_0 = \frac{\beta A}{(\rho + \mu)(\mu + \alpha + \gamma)}.
\]

(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
The characteristic polynomial of \((28)\) yields
\[
\frac{(-\mu+\rho+\lambda+(-\mu^2+(\rho-\alpha-\gamma-\lambda))\mu+(-\alpha-\gamma-\lambda)\rho+\beta A)(\mu+\lambda)^2(1-R_o)}{\rho+\mu}.
\]
\[(29)\]

The trace and determinant of \((28)\) are respectively given by
\[-\rho - 4\mu + \frac{\beta A}{\rho+\mu} - \alpha - \gamma < 0 \]
\[(30)\]

and
\[
\frac{(-\rho-\mu)\mu^2(\lambda-\rho-\alpha-\gamma-\lambda)}{\rho+\mu} > 0.
\]
\[(31)\]

In \((28)\), all the real parts are negative except
\[
\beta \left( \frac{A}{\rho+(1-\rho)-\mu} \right) - (\mu + \alpha + \gamma) > 0
\]
\[(32)\]

which implies that, for \(R_o\) to be less than unity,
\[
\beta \left( \frac{A}{\rho+(1-\rho)-\mu} \right) - (\mu + \alpha + \gamma) > \frac{(\mu+\alpha+\gamma)}{(\mu+\alpha+\gamma)}
\]
\[(33)\]

so that
\[
R_o - 1 > 0, -R_o > -1, \ R_o < 1.
\]
\[(34)\]

Hence, since \(R_o < 1\), 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 \(R_o < 1\).

**Proof:** Given that \(R_o < 1\), there exists only the infection free equilibrium \(E^0\). Consider a Lyapunov function candidate of the form \(V(S,I,V)\): \(R^3 \to R^+\) defined as
\[
V(S,I,V) = \psi I, \ \psi > 0
\]
\[(35)\]
Substituting the second equation in (1) into (35) yields

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

(36)

Since \( E_0 = S = \frac{A}{[\rho+(1-\rho)-\mu]} \) let \( \psi = \frac{1}{(\mu+\alpha+\gamma)} \) so that, in the absence of disease i.e., \( I = 0 \),

\[ \dot{V} = \psi(\beta A - (\rho+\mu)(\mu+\alpha+\gamma))I \]  

(37)

\[ \dot{V} = [R_0 - 1]I \leq 0 \]  

(38)

\( \dot{V} \leq 0 \) for \( R_0 < 1 \) 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

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

(39)

Suppose that the function \( f \) has \( (n + 1) \) derivatives on the interval \( (c - r, c + r) \), for some \( r > 0 \) and \( \lim_{n \to \infty} R_n(x) \) 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;

\[ f(x) = \sum_{k=0}^{n} \frac{1}{k!} (f^{(k)}(c)(x - c)^k) = 0. \]  

For all \( x \in (c - r, c + r) \).  

(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

\[ f(x) = \frac{1}{k!} \left[ \frac{d^k f(x)}{dx^k} \right]_{x=x_0} \]  

(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

\[ f(x) = \sum_{k=0}^{\infty} (x - x_0)^k F(k). \]  

(42)
Substituting (41) and (42), yields

\[ f(x) = \sum_{k=0}^{\infty} \left( x - x_0 \right)^k \frac{\partial^k f(x)}{\partial x^k} \bigg|_{x=x_0}. \]  

(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. If \( f(x) = g(x) \pm h(x) \), 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 \( f(x) = \frac{d^n g(x)}{dx^n} \), then \( Y(k) = (k + m)(k + 1) \ldots (k + m)G(k + m) \)
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 \( F(k) = \delta(k - m) = 1 \), if \( k = m \)
8. If \( f(x) = g(x) \, h(x) \), then \( F(x) = \sum_{m=0}^{k} H(m)G(k - m) \)
9. If \( f(x) = e^{mx} \), then \( F(k) = \frac{m^k}{k!} \)
10. If \( f(x) = (1 + x)^m \), then \( F(k) = \frac{m(m-1)(m-2) \ldots (m-k+1)}{k!} \)

(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)

\[ S(k + 1) = \frac{1}{k+1} \left[ A - [\rho + (1 - \rho) - \mu]S(k) - \beta \sum_{m=0}^{k} S(m)I(k - m) \right] + \delta_1V(k), \]  

(45)

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

(46)

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

(47)

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

(48)

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

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

(49)

\[ I(t) = \sum_{m=0}^{k} t^k I(k) = 10 + 17.76t - 197.16762t^2 - 1127.63213t^3 - 1461.870336t^4 + 14781.11872t^5 + 111163.5841t^6 + 309595.0066t^7, \]  

(50)
\[ V(t) = \sum_{k=0}^{\infty} t^k V(k) = 20 - 37.74t + 13.0051935t^2 + 53.38854263t^3 + 36.00885688t^4 + 25.36091675t^5 - 1899.04723t^6 - 10030.88445t^7, \]

\[ R(t) = \sum_{m=0}^{\infty} t^k R(k) = 30 - 0.24t + 2.784t^2 - 20.60936848t^3 - 87.37824368t^4 - 89.26343635t^5 + 770.2844243t^6 + 4942.395198t^7. \]

(51) 

(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:

| 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 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     | 41.40762143 | 13.66517011  | 15.8642119  | 30.66536487 |
| 0.4     | 32.81524285 | 17.33034021  | 11.72924238 | 30.1372974 |
| 0.6     | 24.2286428   | 20.99551032  | 7.59836537  | 30.19609462 |
| 0.8     | 15.63048571  | 24.66068043  | 2.45848476  | 30.26145949 |
| 1.0     | 11.03810713  | 29.03810713  | 1.57689405  | 30.32682435 |

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

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 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.
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.

References

Ahmad, M. Z., Alsarayreh, D., & Alsarayreh, A. (2017). Differential Transform Method (DTM) for solving SIS and SI epidemic models. *Sains Malaysiana*. 46(10), pp2007 – 2017.

Breaur, F. (2017). A new epidemic model with indirect transmission. *Journal of Biological Dynamics*. vol. 11, 285-293.

Cui, Q., Zhang Q. (2014). Global stability of a discrete SIR epidemic model with vaccination and treatment. *Journal of Difference Equation and Application*. vol 21, issue 2, pp.334-338.

Klouach, D. & Boulasair, L. (2018). Stationary distribution and dynamic behaviour of a stochastic SIVR epidemic model with imperfect vaccine. *Journal of Applied Mathematics*. Volume 2018 (2018), Article ID 1291402, 11 pages.

El-Koufi, A., Adnani, J., Abdelkrim, B., & Yousfi N. (2019). Analysis of a stochastic SIR model with vaccination and non – linear incidence rate. *International Journal of differential equations*. vol 2019, article id 9275051, 9pgs.

Fan, M., (2001). Global stability of an SEIS epidemic model with recruitment and varying total population size. *Mathematical Biosciences*. 170: 199-208.

Gani, S. R., & Halawar, S. (2017). Analysis of an SIVR epidemic model with different control strategies. *Res. J. Mathematical & Statistical Sci.*, Volume 5, Issue (2), 5-13.

Idowu, A. A., Ibrahim, O. M., James, P. O., Sylvanus, A., & Abiodun, O. F. (2018). Differential transform method for solving mathematical model of SEIR and SEI spread of malaria. *International Journal of Science: Basic and Applied Research*. vol 40, no 1, pp.197-219.

Kermack W., & McKendrick A. (1927). A contribution to the Mathematical theory of epidemics. https://doi.org/10.1098/rspa.1927.0118 01 August 1927.

Liu, L., Wang J., & Xianning L. (2015). Global stability with age dependent latency with relapse. *Nonlinear Analysis: Real world applications*, vol 24, 18-35.

Li, M. Y., (1999). Global dynamics of a SEIR model with varying total population size. *Mathematical Biosciences*. Vol. 160, No 2, 191-213.

Ogunmiloro, O. M., Fadugba S. E., & Ogunlade T. O., (2018). Stability analysis and optimal control of vaccination and treatment of a SIR epidemiological deterministic model with relapse. *International Journal of Mathematical Modelling and Computations*. vol 8., no 01, winter 2018, 39-51.

Ross, R., (1911). Some quantitative studies in epidemiology. *International Journal of Nature*, 87: 466-467.

Sun, C. (2010). Global dynamics for a special SEIR epidemic model with nonlinear incidence rates. *Chaos Solitons and Fractals*. 33, 290-297.

Sudhipa, S., Misra O. P., & Dhar J. (2014). Stability analysis of SIR model with vaccination. *American journal of computational and applied mathematics*, 4(1), 17-23, doi:10.5923/j.ajcam.20140401.03.

Wang, J., Lang J., & Chen Y. (2017). Global threshold dynamics of an SIVR model with age dependent infection and relapse. *Journal of Biological Dynamics*, vol 11, no 52, pp. 427-454.

WHO. (2018). Release of the 2018 assessment report of the global vaccine action plan https://www.who.int/immunization/newsroom/news_release_gvap_2018...report/en/

Yang, W., Sun, C., & Arino, J. (2010). Global analysis for a general epidemiological model with vaccination and varying population. *Journal of Mathematical Analysis and Applications*, 372(1), 208-223.

Zhou, J., (1986). Differential Transformation and its Application to Electrical Circuits. Huazhong University Press (in Chinese). 361