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Abstract: The prevalence of measles as an infectious disease has been of major concern to the government health practitioners over the world. This paper intends to investigate the effect of vaccination as a control measure to control the incidence of it means. The basic reproduction number, local and global stabilities of the disease at equilibrium, sensitivity was obtained. The numerical simulation via variational iteration method was carried out. The result clearly shows that proper procurement of vaccine and its implementation is a good control strategy to reduce the rapid spread of the disease.

Keywords: Measles, Vaccine Implementation, Reproduction Number, Variational Iteration Method.

1. Introduction

In recent times, the prevalence of measles as an epidemic disease cannot be overemphasized by government of nations as its spread over the world is alarming. How and what has brought us to this point is a question for all. Mathematical model is a description of a system using mathematical concepts and language. The process of developing a mathematical model is termed mathematical modeling. These are used in the natural sciences and engineering disciplines, as well as in non-physical systems such as the social sciences. Measles is one of the common bacterial infectious diseases with rapid contact rate within 14 days by [1]. Humanity has the ability to control the environment within which it resides. In day-to-day life, humans interact with different beings co-habiting their world by [2], these interactions can sometimes be harmful or destructive to living beings and humans, and as a result, humanity has developed techniques, weapons and sophisticated technological instruments to help reduce the threat [3]. Despite technological advances, we are continuously exposed to new challenges, and constantly face biological threats within our environment in [4, 5]. Viruses are one such threat. Invisible to the human eye, they live in the air, soil, and water and on material surfaces and are responsible for a number of diseases that kill millions of people in [6]. Most recently, the rise of a new strain of corona virus SARS-COV-2 developed into a pandemic that claimed over 200,000 lives between its first documented case in December 2019 in Wuhan, China, and May 1, 2020.by [7–9]. More so, the basic reproduction number as the threshold which governs the stability i.e. $R_0 < 1$ and

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unstable if $R_0 > 1$ of the disease at equilibrium by [10]. $R_0$ is a prominent tool in the outbreak of a disease which projects the prevalence of this overtime as this will enable the predominant and trajectory with the rise or fall of the disease in the population by [11–13]. The local stability at disease free equilibrium is asymptotically stable or otherwise at an invariance of $(R_0 < 1$ or $R_0 > 1)$ in [14]. To combat these invisible enemies, we rely on the study of their behaviors in laboratories, analysis, and prediction, to perform the analysis and prediction, observed facts are converted into models using mathematical tools, including, differentiation, integration and statistical approaches. These models are analyzed and solved analytically or numerically for prediction using some obtained parameters and initial conditions. When dealing with large populations, as in the case of measles, compartmental mathematical models are used. In the deterministic model, individuals in the population are assigned to different subgroups, each representing a specific stage of the epidemic by [15]. The numerical simulation results obtained helps to facilitate that vaccination will help to suppress the rapid spread also project a good and vital role to more research and control policies to curb the spread of the disease.

2. Materials and method

2.1. Model description

Let $N(t)$ be the total population considered. The sets of subpopulation as susceptible class $S(t)$, vaccinated class $V(t)$, Exposed class $E(t)$, infected class $I(t)$ and also Recovered class in the population $R(t)$ representing an $SVEIR$ epidemic model. $\theta$ The number of an individual coming into the population, $\alpha$ vast spread of measles aided by its transmission factors through contact. The progression rate of an exposed individuals who have contracted the disease into the infected population $\gamma$ can be reduced through vaccinating such initial set of subpopulation $\beta_1$. Level of response to treatment is determined by the waning rate $\beta_2$, and if the immunity strength is such weak leading to death $\delta$ or natural phenomenon $\mu$. Parameter representation of each of the compartments state variables as illustrated in table 2.1, based on this consideration, the total population is $N(t) = S(t) + V(t) + (E) + (I) + (R)$.

![Schematic diagram of the model formulation](image.png)

Figure 1: Schematic diagram of the model formulation
Table 2.1: Descriptions, Parameter, Values and References.

<table>
<thead>
<tr>
<th>Description</th>
<th>Parameters</th>
<th>Values</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recruitment rate</td>
<td>$\theta$</td>
<td>0.7</td>
<td>Garba et al, [2]</td>
</tr>
<tr>
<td>Recovery rate</td>
<td>$\rho$</td>
<td>0.0375</td>
<td>Taiwo et al, [4]</td>
</tr>
<tr>
<td>Progression rate from exposed to infected class</td>
<td>$\sigma$</td>
<td>0.0001</td>
<td>Giffrin [3], Peter et al, [9]</td>
</tr>
<tr>
<td>Rate of vaccinating susceptible individuals</td>
<td>$\beta_1$</td>
<td>0.2</td>
<td>Mossong et al, [13]</td>
</tr>
<tr>
<td>Contact rate of infection</td>
<td>$\alpha$</td>
<td>0.885</td>
<td>Kolawole et al, [1]</td>
</tr>
<tr>
<td>Vaccine wane rate</td>
<td>$\beta_2$</td>
<td>0.013</td>
<td>Peter et al, [6]</td>
</tr>
<tr>
<td>Disease induced death rate</td>
<td>$\delta$</td>
<td>0.038</td>
<td>Ayoola et al, [10]</td>
</tr>
<tr>
<td>Natural death</td>
<td>$\mu$</td>
<td>0.02</td>
<td>Shinta et al, [7]</td>
</tr>
</tbody>
</table>

1.1 Proposed Modifications

The proposed compartmental based model for analyzing the dynamics of measles with vaccination as a control strategy, govern model is given by the system of linear ordinary differential equations below:

$$
\begin{align*}
\frac{dS}{dt} &= \theta - \alpha SI - \beta_1 S + \beta_2 V - \mu S \\
\frac{dV}{dt} &= \beta_1 S - (\beta_2 + \mu) V \\
\frac{dE}{dt} &= \alpha SI - (\mu + \sigma) E \\
\frac{dI}{dt} &= \sigma E - (\mu + \delta + \rho) I \\
\frac{dR}{dt} &= \rho I - \mu R
\end{align*}
$$

Subject to the following initial conditions

$S(0) = s_0, V(0) = v_0, E(0) = e_0, I(0) = i_0, R(0) = r_0 \geq 0$.

1.2 Existence of Solution

Consider the compartment of the system of equations on the population over the region

$$\psi = \{S(t), V(t), E(t), I(t), R(t), \in \mathbb{R}^5_+ \}$$

The derivatives obtained as;

$$
\begin{align*}
\frac{dN(t)}{dt} &= \frac{d}{dt}(S(t) + V(t) + E(t) + I(t) + R(t)) \\
\frac{dN(t)}{dt} &= \frac{dS}{dt} + \frac{dV}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dR}{dt} \\
\frac{dN(t)}{dt} &= \theta - \alpha SI - \beta_1 S + \beta_2 V - \mu S + \beta_1 S - (\beta_2 + \mu) V + \alpha SI - (\mu + \sigma) E + \sigma E - (\mu + \delta + \rho) I - \mu R
\end{align*}
$$

$$\frac{dN(t)}{dt} = \theta - \mu(S + V + E + I + R) - \delta I$$

$$\frac{dN(t)}{dt} \leq \theta - \mu N \quad C \text{ is a constant of integration}$$

$N(t) = \frac{\theta}{\mu} + C e^{\mu t}$, by the initial condition at $t = 0$

$C = N(0) - \frac{\theta}{\mu}$

$N(t)e^{\mu t} = \frac{\theta}{\mu} e^{\mu t} + C$

Taking the initial time $t$ and $N(t)$ such that;

$$\lim_{t \to \infty} N(t) \leq \lim_{t \to \infty} \left[ \frac{\theta}{\mu} + \left( N(0) - \frac{\theta}{\mu} e^{-\mu t} \right) \right] = \frac{\theta}{\mu}$$
If \( N(0) \leq \frac{\theta}{\mu} \), then \( N(t) \leq \frac{\theta}{\mu} \). This justification is considered enough for the dynamics of the model the domain \( \mathbb{R}_+^5 \). In this region the model can be considered to be mathematically and epidemiologically well posed. However, the nonnegative solution set of the model equations enters the feasible region, \( \psi \) which is a positively invariant set.

1.3 **Positivity of the model formation**

Consider all compartments of equation (1) at equilibrium in its initial conditions.

**Theorem 2.1.**

\[
\psi = \{ S(t), V(t), E(t), I(t), R(t), \in \mathbb{R}_+^5 \} \text{ as }
\]

\[
S(0) = s_0 > 0, V(0) = v_0 > 0, E(0) = e_0 > 0, I(0) = i_0 > 0, R(0) = r_0 > 0, t \geq 0
\]

Then:

**Proof.** from the system of differential equation (1),

\[
\frac{dS}{dt} = \theta - \alpha SI - \beta_1 S + \beta_2 V - \mu S(t)
\]

\[
\frac{dS}{dt} \geq - (\beta_1 + \mu) S(t)
\]

\[
\frac{dS}{dt} \geq - (\beta_1 + \mu) dt
\]

\[
\int \frac{dS}{dt} \geq - (\beta_1 + \mu) \int dt
\]

\[
S(t) \geq S_0 e^{- (\beta_1 + \mu) t} \geq 0
\]

Similarly for the second compartment,

\[
\frac{dV}{dt} = \beta_1 S - (\beta_2 V + \mu) V(t)
\]

\[
\frac{dV}{dt} \geq - (\beta_2 + \mu) V(t)
\]

\[
\frac{dV}{dt} \geq - (\beta_2 + \mu) dt
\]

\[
\int \frac{dV}{dt} \geq - (\beta_2 + \mu) \int dt
\]

\[
V(t) \geq V_0 e^{- (\beta_2 + \mu) t} \geq 0
\]

Also for the third compartment this yield;

\[
\frac{dE}{dt} = \alpha SI - (\mu + \sigma) E(t)
\]

\[
\frac{dE}{dt} \geq - (\mu + \sigma) E(t)
\]

\[
\frac{dE}{dt} \geq - (\mu + \sigma) dt
\]

\[
\int \frac{dE}{dt} \geq - (\mu + \sigma) \int dt
\]

\[
E(t) \geq E_0 e^{- (\mu + \sigma) t} \geq 0
\]
From the fourth, we obtain that;

\[ \frac{dI}{dt} = \sigma E - (\mu + \delta + \rho)I(t) \]

\[ \frac{dI}{dt} \geq -(\mu + \delta + \rho)I(t) \]

\[ \frac{dI}{dt} \geq -(\mu + \delta + \rho)I(t) \geq \int dt \]

\[ I(t) \geq I_0 e^{-\mu t} \geq 0 \]

Lastly we obtain,

\[ \frac{dR}{dt} = \rho I - \mu R(t) \]

\[ \frac{dR}{dt} \geq -\mu R(t) \]

\[ \frac{dR}{dt} \geq -\mu dt \]

\[ R(t) \geq R_0 e^{-\mu t} \geq 0 \]

Hence, this proofs the positivity of the theorem as a solution to the model.

1.4 Model Analysis

1.4.1 Disease free equilibrium state

At disease free equilibrium point, there is no outbreak of measles, \( S \neq 0, I = 0 \), as obtained;

\[
\begin{align*}
\theta - \alpha SI - \beta_1 S + \beta_2 V - \mu S &= 0 \\
\beta_1 S - (\beta_2 + \mu)V &= 0 \\
\alpha SI - (\mu + \sigma)E &= 0 \\
\sigma E - (\mu + \delta + \rho)I &= 0 \\
\rho I - \mu R &= 0
\end{align*}
\]

The disease free equilibrium

\[(E_1) = (S_0, V_0, E_0, I_0, R_0) = \left( \frac{\theta(\beta_2 + \mu)}{\mu(\beta_1 + \beta_2 + \mu)}, \frac{\theta \beta_1}{\mu(\beta_1 + \beta_2 + \mu)}, 0, 0, 0 \right) \]

1.4.2 Endemic equilibrium point

Let \( E^*_e = (S^*, V^*, E^*, I^*, R^*) \) as Endemic Equilibrium where \( I \neq 0 \) Consider the system of
Numerical Simulation of the Effect of Vaccine

equation (1) at equilibrium point whose results are;

\[ S^* = \frac{(\mu + \sigma)(\mu + \delta + \rho)}{\alpha \sigma} \]
\[ V^* = \frac{\beta_1[(\mu + \sigma)(\mu + \delta + \rho)]}{\alpha \sigma} \]
\[ E^* = \frac{\theta \sigma \alpha + (\mu + \sigma)(\mu + \delta + \rho)[\beta_1 \beta_2 - (\mu + \beta_1)]}{(\mu + \sigma)(\mu + \delta + \rho)\alpha \sigma} \]
\[ I^* = \frac{\theta \sigma \alpha + (\mu + \sigma)(\mu + \delta + \rho)[\beta_1 \beta_2 - (\mu + \beta_1)]}{(\mu + \sigma)(\mu + \delta + \rho)\alpha \sigma} \]
\[ R^* = \frac{\rho(\theta \sigma \alpha + (\mu + \sigma)(\mu + \delta + \rho)[\beta_1 \beta_2 - (\mu + \beta_1)])}{\mu(\mu + \sigma)(\mu + \delta + \rho)\alpha \sigma} \]

(2.13)

1.4.3 Basic Reproduction Number.
There are two disease states but only one to create new infection. Exposed and Infected compartments of the model are connected with from equation (1). This denote the number of secondary infection caused as a result of infected individuals in a population also known as the threshold parameter which governs the rapid spread of a disease in a population, where \( R_0 = G = \rho(F \times V^{-1}) \) via using next generation matrix as obtained below,

\[
\frac{dE}{dt} = \alpha SI - (\mu + \sigma)E \\
\frac{dI}{dt} = \sigma E - (\mu + \delta + \rho)I
\]

(2.14)

Considering the disease compartments where \( R_0 = F \times V^{-1} \). The transition and transmission matrix are obtained via partial derivatives of \( f \) and \( v \) evaluated at the disease free equilibrium.

\[ F = \left( \begin{array}{cc} \alpha \sigma I & \beta_1 \beta_2 - (\mu + \beta_1) \\ 0 & \beta_1 \beta_2 - (\mu + \beta_1) \end{array} \right) \]
\[ V = \left( \begin{array}{cc} (\mu + \sigma) & 0 \\ -\sigma & (\mu + \delta + \rho) \end{array} \right) \]
\[ V^{-1} = \left( \begin{array}{cc} \frac{1}{\mu + \sigma} & \frac{(\mu + \sigma)(\mu + \delta + \rho)}{(\mu + \delta + \rho)} \\ 0 & \frac{(\mu + \sigma)(\mu + \delta + \rho)}{(\mu + \delta + \rho)} \end{array} \right) \]

Since \( R_0 = F \times V^{-1} \)

\[ R_0 = \frac{\alpha \theta (\beta_2 + \mu)}{\mu[\beta_1 + \beta_2 + \mu](\mu + \delta + \rho)} \]

(2.16)

(2.17)

The invariance of the dominant Eigen-value is the basic reproduction number.

2.6 Model stability.

2.6.1 Local stability of disease free equilibrium

Proposition 1: The disease free equilibrium of the proposed epidemic model is locally
asymptotically stable if $R_0 < 1$ otherwise unstable if $R_0 > 1$ Local stability at disease free of the model is deduced for $R_0 < 1$ and unstable at $R_0 > 1$, invariantly as obtained by Jacobian matrix relatively as $|J_{E(0)} - \lambda I| = 0$.

$$J_{(E_1)} = \begin{pmatrix} - (\beta_1 + \mu) & \beta_2 & 0 & -\alpha & 0 \\ \beta_1 & - (\beta_2 + \mu) & 0 & 0 & 0 \\ 0 & 0 & -(\sigma + \mu) & 0 & 0 \\ 0 & 0 & \sigma & -(\mu + \delta + \rho) & 0 \\ 0 & 0 & 0 & \rho & -\mu \end{pmatrix}$$

(2.18)

$$\begin{vmatrix} - (\beta_1 + \mu) - \lambda & \beta_2 & 0 & -\alpha & 0 \\ \beta_1 & - (\beta_2 + \mu) - \lambda & 0 & 0 & 0 \\ 0 & 0 & -(\sigma + \mu) - \lambda & 0 & 0 \\ 0 & 0 & \sigma & -(\mu + \delta + \rho) - \lambda & 0 \\ 0 & 0 & 0 & \rho & -\mu - \lambda \end{vmatrix} = 0$$

Respectively the Eigen values are obtained as;

$$\lambda = -(\beta_1 + \mu), -(\beta_2 + \mu), -(\sigma + \mu), -(\mu + \delta + \rho), -\mu$$

(2.19)

Since all are negatively invariant, therefore they are locally asymptotically stable.

2.6.2 Local stability of endemic equilibrium

Proposition: The endemic equilibrium of the proposed model is locally asymptotically stable if $R_0 < 1$ unstable otherwise. Let $S = x + S^*, V = y + V^*, E = z + E^*, I = a + I^*, R = b + R^*$

$$\begin{align*}
\frac{dx}{dt} &= \theta - \alpha(x + S^*)(a + I^*) - \beta_1(x + S^*) + \beta_2(y + V^*) - \mu(x + S^*) \\
\frac{dy}{dt} &= \beta_1(x + S^*) - (\beta_2 + \mu)(y + V^*) \\
\frac{dz}{dt} &= \alpha(x + S^*)(a + I^*) - (\mu + \sigma)(z + E^*) \\
\frac{da}{dt} &= \sigma(z + E^*) - (\mu + \delta + \rho)(a + I^*) \\
\frac{db}{dt} &= \rho(a + I^*) - \mu(b + R^*)
\end{align*}$$

(2.20)

Via linearization of state variables, it is obtained that;

$$\begin{align*}
\frac{dx}{dt} &= -\alpha x a - \beta_1 x + \beta_2 y - \mu x + \text{higher order} + \text{non-linear} \\
\frac{dy}{dt} &= \beta_1 x - (\beta_2 + \mu)y + \text{higher order} + \text{non-linear} \\
\frac{dz}{dt} &= -\alpha x a - (\mu + \sigma)z + \text{higher order} + \text{non-linear} \\
\frac{da}{dt} &= \sigma z - (\mu + \delta + \rho)a + \text{higher order} + \text{non-linear} \\
\frac{db}{dt} &= \rho a - \mu b + \text{higher order} + \text{non-linear}
\end{align*}$$

(2.21)
Global stability of disease free equilibrium

The Jacobian of the resulting linearization have it that: \(|J_{E(0)} - \lambda I| = 0\).

\[
J_{E^*} = \begin{pmatrix}
-(\alpha a + \beta_1 + \mu) & \beta_2 & 0 & -\alpha x & 0 \\
\beta_1 & -(\beta_2 + \mu) & 0 & 0 & 0 \\
\alpha a & 0 & -(\mu + \sigma) & 0 & 0 \\
0 & 0 & \sigma & -(\mu + \delta + \rho) & 0 \\
0 & 0 & 0 & \rho & -\mu
\end{pmatrix}
\]

(2.22)

Consider the respective Eigen values via its characteristic equation as obtained below

\[
\begin{vmatrix}
-(\alpha a + \beta_1 + \mu) - \lambda & \beta_2 & 0 & -\alpha x & 0 \\
\beta_1 & -(\beta_2 + \mu) - \lambda & 0 & 0 & 0 \\
\alpha a & 0 & -(\mu + \sigma) - \lambda & 0 & 0 \\
0 & 0 & \sigma & -(\mu + \delta + \rho) - \lambda & 0 \\
0 & 0 & 0 & \rho & -\mu - \lambda
\end{vmatrix} = 0
\]

(2.23)

The resulting Eigen-value become,

\[-(\alpha a + \beta_1 + \mu) - \lambda - (\beta_2 + \mu) - \lambda - (\mu + \sigma) - \lambda - (\mu + \delta + \rho) - \lambda - \mu - \lambda\]

If \(p = -(\alpha a + \beta_1 + \mu), q = -(\beta_2 + \mu), r = -(\mu + \sigma), s = -(\mu + \delta + \rho), t = -\mu\)

\((p - \lambda)(q - \lambda)(r - \lambda)(s - \lambda)(t - \lambda) = 0\)

(2.24)

Algebraically, it is obtained that,

\[
\lambda^5 - (t + (r + s) + (p + q))\lambda^4 + ((p + q)(r + s) + pq + rs)(1 + t)\lambda^3 - (pq(r + s) + rs(p + q))(1 + s)\lambda^2 + s(pq(r + s) + rs(p + q))\lambda - pqrst = 0
\]

(2.25)

With the invariance of the Eigen values it is said to be locally asymptotically stable

### 2.6.3 Global stability of disease free equilibrium

The Lyapunov approach to obtain global asymptotic stability of the proposed model at disease free equilibrium whose derivatives are obtained with respect to time is illustrated below;

\[
V(t, S, V, E, I, R) = CI_1 + CI_2
\]

(2.26)

\[
\frac{dV}{dt} = C_1I_1^* + C_2I_2^*
\]

(2.27)

If \(C_1 = \frac{1}{(\mu + \sigma)}, C_2 = \frac{\alpha \theta(\beta_2 + \mu)}{\mu(\beta_1 + \beta_2 + \mu)(\mu + \sigma)(\mu + \delta + \rho)}\)

\[
\frac{dV}{dt} \leq \left[\frac{\alpha a \theta(\beta_1 + \mu)}{\mu(\beta_1 + \beta_2 + \mu)(\mu + \sigma)(\mu + \delta + \rho)} - \frac{(\mu + \sigma)}{\mu(\mu + \sigma)}\right] I + \left[\frac{\beta(1 - c)\lambda}{\mu(\mu + \epsilon + \tau_1)} - \frac{\lambda(1 - c)\beta(\mu + \gamma + \delta + \tau_2)}{\mu(\mu + \gamma + \delta + \tau_2)}\right] I
\]

(2.28)

\[
\frac{dV}{dt} \leq \eta \left[\frac{\alpha \theta(\beta_2 + \mu)}{\mu(\beta_1 + \beta_2 + \mu)(\mu + \delta + \rho) - 1}\right] I
\]

\[
\frac{dV}{dt} \leq \eta(R_0 - 1)
\]
Imperatively to note that $V^* = 0$ only when $I = 0$, the substitution of $I = 0$ into the model equation shows that at equilibrium $\eta > 0$ at $t \to \infty$. Based on LaSalle’s invariance principle it is globally asymptotically stable whenever $R_0 > 1$.

2.7 Sensitivity analysis of the reproductive ratio

The test for the sensitivity of $R_0$ is with respect to all the parameters in $R_0$. The normalized forward sensitivity index is defined as shown below:

$$\frac{\partial R_0}{\partial \rho} \times \frac{\rho}{\partial R_0} = \frac{\rho (\mu + \delta + \rho)}{(\mu + \delta)}$$
$$\frac{\partial R_0}{\partial \theta} \times \frac{\theta}{\partial R_0} = 1$$
$$\frac{\partial R_0}{\partial \alpha} \times \frac{\alpha}{\partial R_0} = 1$$

The test for the sensitivity of $R_0$ iteratively obtained as:

$$\frac{\partial R_0}{\partial \beta_1} \times \frac{\beta_1}{\partial R_0} = \frac{\beta_1 [\beta_1 + \beta_2 + \mu]}{(\beta_2 + \mu)}$$
$$\frac{\partial R_0}{\partial \beta_2} \times \frac{\beta_2}{\partial R_0} = \frac{\mu \beta_2 [\beta_1 + \beta_2 + \mu]}{(\beta_1 + \mu)(\beta_2 + \mu)}$$

$$\frac{\partial R_0}{\partial \mu} \times \frac{\mu}{\partial R_0} = \frac{[\beta_2 + \mu^2 (\beta_1 + \beta_2 + \mu)] (\mu + \delta + \rho)}{(\beta_1 + \beta_2)(\beta_2 + \mu)}$$
$$\frac{\partial R_0}{\partial \delta} \times \frac{\delta}{\partial R_0} = \frac{[\delta (\mu + \delta + \rho)]}{\mu + \rho}$$

Tabular representation of parameter and indices of sensitivity analysis deduced from their initial values are as follows:

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Sensitivity Indices $(R_0)$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\rho$</td>
<td>0.0157663</td>
</tr>
<tr>
<td>$\beta_1$</td>
<td>2.30704e-3</td>
</tr>
<tr>
<td>$\theta$</td>
<td>1.0000000</td>
</tr>
<tr>
<td>$\alpha$</td>
<td>1.0000000</td>
</tr>
<tr>
<td>$\beta_2$</td>
<td>1.290925e3</td>
</tr>
<tr>
<td>$\delta$</td>
<td>0.02077777</td>
</tr>
<tr>
<td>$\mu$</td>
<td>4.8814e-9</td>
</tr>
</tbody>
</table>

2.8 Numerical simulation

The numerical simulation for the epidemic model formulation via variational iteration method, constructing an iteration formula for each compartment adopted through maple software thus, iteratively obtained as:

$$S_{n+1} = S_n(t) - \int_0^t \left( \frac{d}{d\tau} S_n(\tau) - \theta - \alpha \cdot S_n(\tau) \cdot I_n(\tau) - \beta_1 \cdot S_n(\tau) + \beta_2 \cdot V_n(\tau) - \mu \cdot S_n(\tau) \right) d\tau$$

$$V_{n+1} = V_n(t) - \int_0^t \left( \frac{d}{d\tau} V_n(\tau) - \beta_1 \cdot S_n(\tau) - (\beta_2 + \mu) \cdot V_n(\tau) \right) d\tau$$

$$E_{n+1} = E_n(t) - \int_0^t \left( \frac{d}{d\tau} E_n(\tau) - \alpha \cdot S_n(\tau) \cdot I_n(\tau) - (\mu + \sigma) \cdot E_n(\tau) \right) d\tau$$

$$I_{n+1} = I_n(t) - \int_0^t \left( \frac{d}{d\tau} I_n(\tau) - \sigma \cdot E_n(\tau) - (\mu + \delta + \rho) \cdot I_n(\tau) \right) d\tau$$

$$R_{n+1} = R_n(t) - \int_0^t \left( \frac{d}{d\tau} R_n(\tau) - \rho \cdot I_n(\tau) - \mu \cdot R_n(\tau) \right) d\tau$$
At \( n = 0 \) the first iteration yield;
\[
S_1(t) = (\theta + \alpha_0 s_0 + \beta_1 s_0 - \beta_2 v_0 + \mu s_0) t + s_0 \\
V_1(t) = (\beta_1 s_0 + (\beta_2 + \mu) v_0) t + v_0 \\
E_1(t) = (\alpha_0 s_0 + (\mu + \sigma) e_0) t + e_0 \\
I_1(t) = (\alpha e_0 + (\mu + \delta + \rho) m_0) t + m_0 \\
R_1(t) = (\rho m_0 + \mu r_0) t + r_0
\]
\[
(2.31)
\]
At \( n = 1 \), with an initial condition;
\[
ic = \{ S_1(t) = (\theta + \alpha_0 s_0 + \beta_1 s_0 - \beta_2 v_0 + \mu s_0) t + s_0, S_1(\tau) = (\theta + \alpha_0 s_0 + \beta_1 s_0 - \beta_2 v_0 + \mu s_0) t + s_0, \\
V_1(t) = (\beta_1 s_0 + (\beta_2 + \mu) v_0) t + v_0, V_1(\tau) = (\beta_1 s_0 + (\beta_2 + \mu) v_0) t + v_0, \\
E_1(t) = (\alpha_0 s_0 + (\mu + \sigma) e_0) t + e_0, E_1(\tau) = (\alpha_0 s_0 + (\mu + \sigma) e_0) t + e_0 \\
I_1(t) = (\alpha e_0 + (\mu + \delta + \rho) m_0) t + m_0, I_1(\tau) = (\alpha e_0 + (\mu + \delta + \rho) m_0) t + m_0 \\
R_1(t) = (\rho m_0 + \mu r_0) t + r_0, R_1(t) = (\rho m_0 + \mu r_0) t + r_0 \}
\]
Its second iteration iteratively yields;
\[
S_2(t) = \text{collect (eval (} S_{n+1}, ic), t); \\
V_2(t) = \text{collect (eval (} V_{n+1}, ic), t); \\
E_2(t) = \text{collect (eval (} E_{n+1}, ic), t); \\
I_2(t) = \text{collect (eval (} I_{n+1}, ic), t); \\
R_2(t) = \text{collect (eval (} R_{n+1}, ic), t); \\
(2.33)
\]
\[
S_2(t) = \alpha(\theta + \alpha_0 s_0 + \beta_1 s_0 - \beta_2 v_0 + \mu s_0) (\sigma e_0 + (\mu + \delta + \rho) m_0) t^3 + (- \beta_2 (\beta_1 s_0 + (\beta_2 + \mu) v_0) + \beta_1 (\theta + \alpha_0 s_0 + \beta_1 s_0 - \beta_2 v_0 + \mu s_0) + \alpha_0 (\sigma e_0 + (\mu + \delta + \rho) m_0) \\
+ \alpha(\theta + \alpha_0 s_0 + \beta_1 s_0 - \beta_2 v_0 + \mu s_0) m_0 + \mu (\theta + \alpha_0 s_0 + \beta_1 s_0 - \beta_2 v_0 + \mu s_0) t^2 + (2(\beta_1 s_0 + 2(\beta_2 + \mu) v_0) + 2\beta_1 s_0 - 2\beta_2 v_0 + 2\mu s_0) t + s_0) \\
V_2(t) = (\beta_1 (\theta + \alpha_0 s_0 + \beta_1 s_0 - \beta_2 v_0 + \mu s_0) + (\beta_2 + \mu) (\beta_1 s_0 + (\beta_2 + v_0)) t^2 + (2\beta_1 s_0 + 2(\beta_2 + \mu) v_0) t + v_0 \\
E_2(t) = \alpha(\theta + \alpha_0 s_0 + \beta_1 s_0 - \beta_2 v_0 + \mu s_0) (\sigma e_0 + (\mu + \delta + \rho) m_0) t^3 + (\alpha s_0 (\alpha e_0 + (\mu + \delta + \rho) m_0) + \alpha(\theta + \alpha_0 s_0 + \beta_1 s_0 - \beta_2 v_0 + \mu s_0) m_0 + (\mu + \sigma)(\alpha_0 s_0 + (\mu + \sigma) e_0) t^2 + (2\alpha s_0 m_0 + 2(\mu + \sigma) e_0) t + e_0 \\
I_2(t) = \alpha(\alpha_0 s_0 + (\mu + \sigma) e_0) + (\mu + \delta + \rho) (\sigma e_0 + (\mu + \delta + \rho) m_0) t^2 + (2\alpha s_0 m_0 + 2(\mu + \sigma) e_0) t + e_0 \\
R_2(t) = (\rho (\sigma e_0 + (\mu + \sigma) m_0) + \mu (\rho m_0 + \mu r_0)) t^2 + (\mu + \delta + \rho) (\sigma e_0 + (\mu + \delta + \rho) m_0) t^2 + (2\rho m_0 + 2\mu r_0) t + r_0
\]
\[
(2.34)
\]
Evaluating at initial conditions;
\[
N = 722, s_0 = 500, v_0 = 120, e_0 = 65, i_0 = 23, r_0 = 14, \beta_1 = 0.0021, \beta_2 = 0.0013, \alpha = 0.0017, \sigma = 0.0011, \rho = 0.0115, \theta = 0.012, \mu = 0.02, \delta = 0.011
\]
\[
(2.35)
\]
Gives the following time dependent solutions,
\[
S(t) = 0.0543121848 \quad t^3 + 2.75086940t^2 + 60.9120t + 500 \\
V(t) = 0.14076540 \quad t^2 + 7.2120t + 120 \\
E(t) = 0.0543121848 \quad t^3 + 2.52392325t^2 + 41.8430t + 65 \\
I(t) = 0.06759615 \quad t^2 + 2.0980t + 23 \\
R(t) = 0.0229530 \quad t^2 + 1.0890t + 14
\]
\[
(2.36)
\]
Graphical illustration of the resulting iterations is thus shown below:
3.0 Discussion of results and recommendation.
Numerical simulation was carried out and presented graphically to investigate the effect of vaccine on the state variables as it is an efficient control measure to eradicate the rapid spread of the disease. Figure 1: It clearly shows that the rate of vaccination of susceptible individuals in the population increases as it reduces the infected population with time. Figure 2: Depicts that the exposed individual in the population reduces as vaccination is applied to the population of infected individuals. Figure 3: Depicts the effect of vaccine on the infected individual in the population as a strategic means to flatten the curve. It is therefore recommended that health practitioners implement vaccination as a control measure on infected individuals in the population in order to flatten the curves of this disease.

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References


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