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Numerical Solution And Stability Analysis Of A Childhood-Disease Model With Vaccination And Relapse*

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Abstract

In this research, a childhood disease model that incorporates relapse and vaccination was developed and systematically analyzed using sets of non-linear Ordinary Differential Equations. The model exhibits disease-free equilibrium which is locally and globally asymptotically stable whenever the threshold parameter R_o is less than unity and unstable otherwise. It also exhibits endemic equilibrium which was proved to be locally asymptotically stable whenever R_o is greater than unity. The model was then modified to include vaccination programme capable of reducing disease burden. The global stability analysis of the endemic equilibrium points was carried out using geometric and compound matrix approach satisfying the Bendixon criterion when $R_v > 1$. The numerical solution of the model was performed using Adams method coded in Python Programming Language to explore the biological implication of force of infection, vaccination and relapse. The result shows that for the disease to be eradicated, the vaccination rate f must be robust, relapse rate ϵ reduced and the contact rate β among children should be avoided or minimized.

1 Introduction

Recently, childhood diseases have been a major public health hazard in the world. About three decades ago, more than 2 million children died of different forms of childhood diseases [24]. Typical examples of childhood diseases are measles, chicken pox, rubella, etc. Children within the age bracket 4-8 years are prone to these diseases due to their frequent contact with their peers at school, playing grounds and other places [7].

Honestly, the use of vaccines has typically reduced the incidence of infectious diseases among children, but recent studies show that childhood diseases still remain a public health problem. Poor immunization administration and unavailability of vaccine are some of the major reasons behind the resurgence of these deadly diseases [9]. At this age bracket, particularly for the uninfected children, the administration of vaccine may induce permanent immunity to the disease.

Some researchers like [8] studied the classical susceptible-exposed-infectious-removed (SEIR) model for the transmission dynamics of measles to better understand its complex dynamics. But some of the recent clinical researches have shown that the permanent immunity induced by the preventive vaccines for some of the aforementioned diseases wanes in no time. For example, [16] estimated the mean duration of vaccine-induced protection against measles in the absence of re-exposure to be 25 years. Similar clinical results have shown additional cases of waning immunity in vaccines that are expected to offer permanent immunity [20, 1, 3, 17, 21].

The immunity in preventive vaccines against childhood diseases also wanes. Therefore, the vaccine wanes immediately from the body of the children, those children become susceptible to the disease again and get relapsed. Hence, it becomes imperative to develop a model that incorporates vaccine-induced immunity with no or negligible waning rate.

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Biologically speaking, the return of a disease weeks or months after its apparent cessation is called relapse. That is, relapse is the return of a disease or the signs and symptoms of a disease after a period of improvement. This phenomenon has partly contributed to the wide spread of disease in the community [19]. As far as mathematical modeling is concerned, few researchers have incorporated this phenomenon in childhood disease model.

The unquantifiable dangers posed by the menace of childhood diseases which made public health workers, researchers, scientists and governments at both state and federal level to try their best to contain its spread has necessitated the attempt to embark on this research.

To this end, we develop, analyze and carry out the numerical solution of a childhood disease model incorporating vaccine-induced immunity to further understand its dynamics. It is worth noting here that, to the best of our knowledge, no childhood disease model has incorporated vaccination and relapse combined which necessitated the interest to embark on this work.

The work is arranged as follows; section two presents model 1 without vaccination, the meaning of some basic parameters, invariant region, positivity solution and its stability analyses. In section three, we present the vaccination model, its analyses and the contour plot. Section four contains the numerical solution of the model using the Adams method coded in Python Programming Language and MATLAB. Section five contains the conclusion and acknowledgment followed by references.

2 Model Formulation

The entire population of the model at time t, is divided in to four main classes namely: susceptible class $X(t)$, asymptomatic class $I_1(t)$, symptomatic class $I_2(t)$ and the recovered class $R(t)$ using set of nonlinear deterministic differential equations.
Taking $\frac{\beta X I_2(t)}{N}$ as the force of infection, ϵ as relapse rate, α as progression rate from asymptomatic to symptomatic class. Both birth and death rates are represented by μ, recovery rate by γ and the total population can be represented as

$$N(t) = X(t) + I_1(t) + I_2(t) + R(t). \tag{1}$$

The basic model assumptions are:

1. The asymptomatic class are only infected but not infectious;
2. Eruption of relapse due to the absence of vaccine or low vaccine efficacy;
3. The population is fixed;
4. The probability of been infected is not based on sex, race or tribe;
5. There is always homogeneous mixing and interaction between the four classes;
6. Individuals are recruited to the susceptible class only;
7. The vaccine is assumed to be perfect without waning.

The model is presented as follows:

$$\dot{X}(t) = \mu N - \frac{\beta X I_2(t)}{N} - \mu X, \tag{2}$$

$$\dot{I_1}(t) = \frac{\beta X I_2(t)}{N} - (\alpha + \mu) I_1, \tag{3}$$

$$\dot{I_2}(t) = \alpha I_1 + \epsilon R - (\mu + \gamma) I_2, \tag{4}$$

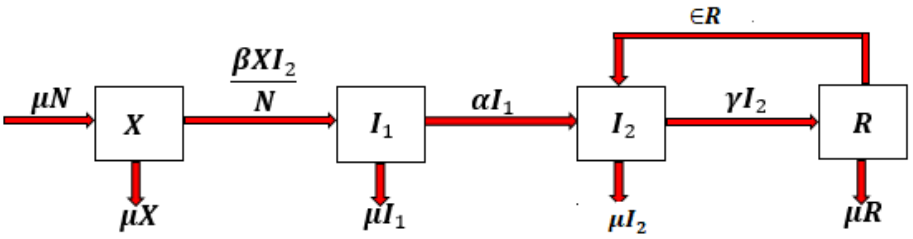

Figure 1: Vaccination-free Model Diagram.

$$\dot{R}(t) = \gamma I_2 - (\epsilon + \mu)R. \tag{5}$$

The above set of nonlinear differential equations can be normalized to give

$$\frac{dx(t)}{dt} = \mu - \beta x i_2 - \mu x, \tag{6}$$

$$\frac{di_1(t)}{dt} = \beta x i_2 - m_1 i_1, \tag{7}$$

$$\frac{di_2(t)}{dt} = \alpha i_1 + \epsilon r - m_2 i_2, \tag{8}$$

$$\frac{dr(t)}{dt} = \gamma i_2 - m_3 r, \tag{9}$$

where

$$x = \frac{X}{N}, i_1 = \frac{I_1}{N}, i_2 = \frac{I_2}{N}, r = \frac{R}{N}, m_1 = \alpha + \mu,$$
$$m_2 = \mu + \gamma, m_3 = \epsilon + \mu, n(t) = x(t) + i_1(t) + i_2(t) + r(t).$$

Parameter	Description	Value/year	Reference
μ	Recruitment & death rate	0.166	[7]
β	Contact rate	0.8	[22]
ϵ	Relapse rate	0.89	Assumed
α	Progression from I_1 to I_2	0.9	Assumed
γ	Recovery rate	0.5	[5]
f	Vaccination rate	(0,1)	[22]

Table 1: Description of Parameters and Their Hypothetical Value.

2.1 Positivity and Boundedness of Solution

Theorem 1 *The feasible region* Γ *defined by*

$$\Gamma = \left\{(x, i_1, i_2, r) \in \mathbb{R}^4_+ : x(t) + i_1(t) + i_2(t) + r(t) = 1\right\},$$

with initial condition

$$x(0) \geq 0,\ i_1(0) \geq 0,\ i_2(0) \geq 0,\ r(0) \geq 0, \tag{10}$$

is positively-invariant and attracting with respect to model equation (6)–(9).

Proof. Using equation (6), we have

$$\frac{dx(t)}{dt} = \mu - (\lambda + \mu)x(t) \quad \text{where} \quad \lambda(i_2) = \beta i_2(t).$$

With the integrating factor $\theta(t) = \exp\left\{\mu t + \int_o^\tau \lambda(\tau)d\tau\right\}$, the solution is given by

$$x(t)\exp\left\{\mu t + \int_o^\tau \lambda(\tau)d\tau\right\} = \mu\int_o^t \exp\left\{\mu t + \int_o^\tau \lambda(\tau)d\tau\right\}dt + x(0),$$

$$x(t) = \left[\mu\int_o^t \exp\left\{\mu t + \int_o^\tau \lambda(\tau)d\tau\right\}dt + x(0)\right]\exp\left\{-\mu t - \int_o^\tau \lambda(\tau)d\tau\right\},$$

where $x(0)$ is given by (10). This shows that the variable $x(t)$ is positive. Hence, the positiveness of the solution of $x(t)$ is guaranteed. The same approach can be extended to other variables $i_1(t)$, $i_2(t)$ and $r(t)$ to prove the positivity of their respective solution.
Moreover, adding all equations of the system (6)–(9) gives,

$$\frac{dn(t)}{dt} = \mu - \mu n(t).$$

Using the integrating factor $e^{\mu t}$, the solution is given by

$$n(t) = 1 + (n(0) - 1)\, e^{-\mu t},$$

where $n(t) = 1$ for any $t > 0$. This indicates that the solutions of system (6)–(9) are bounded above by 1 in a positive region $\mathbb{R}^4_+$. This implies that

$$\Gamma = \left\{(x(t), i_1(t), i_2(t), r(t)) \in {\mathbb{R}_+}^4 : x(t) + i_1(t) + i_2(t) + r(t) = 1\right\},$$

is positively invariant set of the system (6)–(9). It is then sufficient to study the childhood model since it is epidemiologically well-posed and biologically meaningful [10, 15, 11]. ■

2.2 Equilibrium Points

The disease-free equilibrium of the model (6)–(9) is given as

$$E^{'} = (1, 0, 0, 0), \text{ that is, } x^* = 1, i_1^* = 0, i_2^* = 0, r^* = 0. \tag{11}$$

2.3 The Basic Reproduction Number

According to [18], the linear stability of $E^{'}$ can be established using the next generation operator method on the model equation. The reproduction number is termed as the average number of secondary infection that can be obtained in the cause of a single primary infection introduced into a population of susceptible individuals [23]. Let $u = (i_1(t), i_2(t), r(t))^T \in \mathbb{R}^3$. Then the model equation can be written in the form $\frac{du}{dt} = \mathcal{F}(u) - \mathcal{V}(u)$, where

$$\mathcal{F}(u) = \begin{pmatrix} \beta x i_2 \\ 0 \\ 0 \end{pmatrix}, \quad \mathcal{V}(u) = \begin{pmatrix} m_1 i_1 \\ m_2 i_2 - \alpha i_1 - \epsilon r \\ m_3 r - \gamma i_2 \end{pmatrix}.$$

The derivative of the above expressions with respect to i_1, i_2, r evaluated at disease-free equilibrium gives

$$F = \begin{pmatrix} 0 & \beta & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} m_1 & 0 & 0 \\ -\alpha & m_2 & -\epsilon \\ 0 & -\gamma & m_3 \end{pmatrix}.$$

The reproduction number expressed as $\rho(FV^{-1})$ which is the spectra radius of FV^{-1} is given by

$$R_o = \frac{\beta\alpha m_3}{m_1(m_2m_3 - \epsilon\gamma)}.$$

The spread of the childhood disease among children is dependent on the value of the reproduction number.

2.4 Endemic Points

The endemic equilibrium point is known as the positive steady state solution where the disease is still prevalent in the population. The endemic equilibrium points E are given as

$$x^{**} = \frac{1}{R_o}, \quad i_1^{**} = \frac{\mu^2(\epsilon+\gamma+\mu)(R_o-1)}{\alpha m_3\beta}, \quad i_2^{**} = \frac{\mu(R_o-1)}{\beta}, \quad r^{**} = \frac{\gamma\mu(R_o-1)}{\beta m_3}, \tag{12}$$

where the endemic equilibrium exists only when $R_o > 1$.

2.5 Stability Analysis of the System

Using Theorem 2 in [18], the following result is established.

Theorem 2 *The disease-free equilibrium of the model (6)–(9) is locally asymptotically stable if $R_o < 1$ and unstable if $R_o > 1$.*

Theorem 3 *The endemic equilibrium of the model (6)–(9) is locally asymptotically stable if $R_o > 1$ and unstable if $R_o < 1$.*

Proof. We evaluate the Jacobian matrix of the model equation at endemic equilibrium points as follows:

$$J(E) = \begin{pmatrix} -\beta i_2^{**} - \mu & 0 & -\beta x^{**} & 0 \\ \beta i_2^{**} & -m_1 & \beta x^{**} & 0 \\ 0 & \alpha & -m_2 & \epsilon \\ 0 & 0 & \gamma & -m_3 \end{pmatrix}.$$

The characteristic equation of $J(E^1)$ is given as

$$f_2(\lambda) = \lambda^4 + h_3\lambda^3 + h_2\lambda^2 + h_1\lambda + h_o = 0. \tag{13}$$

where h_4, h_3, h_2, h_1, h_o are given as

$$h_4 = 1 > 0,$$

$$h_3 = m_1 + m_2 + m_3 + \beta i_2^{**} + \mu > 0,$$

$$h_2 = \beta i_2^{**}(m_1+m_2+m_3) + \mu(m_1+m_2+m_3) + m_2m_3 + m_1(m_2+m_3) - \beta x^{**}\alpha - \epsilon\gamma,$$

where

$$-\beta x^{**}\alpha - \epsilon\gamma = -\beta\alpha\left[\frac{\mu(\epsilon+\mu+\gamma)(\alpha+\mu)}{\alpha\beta(\epsilon+\mu)}\right] - \epsilon\gamma = \frac{\epsilon\gamma m_1}{m_3} - m_1m_2 - \epsilon\gamma$$

from the provision of the expressions in (12). Hence,

$$h_2 = \beta i_2^{**}(m_1+m_2+m_3) + \mu(m_1+m_2+m_3) + m_1m_3 + \frac{\epsilon\gamma m_1}{m_3} + \mu(\mu+\epsilon+\gamma) > 0,$$

$$\begin{aligned} h_1 \quad = \quad & \beta i_2^{**}(m_2m_3 - \epsilon\gamma) + \beta i_2^{**}m_1(m_2+m_3) - \beta x^{**}\alpha\mu - \epsilon\gamma\mu - \beta x^{**}\alpha m_3 \\ & -\epsilon\gamma m_1 + \mu(m_1m_2 + m_1m_3 + m_2m_3) + m_1m_2m_3, \end{aligned}$$

which can also be re-expressed as

$$h_1 = \beta i_2^{**} m_1(m_2 + m_3) + A_1 + A_2 + A_3,$$

where

$$A_1 = -\beta x^{**}\alpha m_3 - \epsilon\gamma m_1 = -m_1 m_2 m_3,$$
$$A_2 = -\beta x^{**}\alpha\mu - \epsilon\gamma\mu = \mu\left(\frac{\gamma\epsilon m_1}{m_3} - m_1 m_2 - \epsilon\gamma\right),$$
$$A_3 = \beta i_2^{**}(m_2 m_3 - \epsilon\gamma) = \mu^2(\mu + \gamma + \epsilon)(R_o - 1).$$

Substituting back A_1, A_2, A_3 gives

$$\begin{aligned} h_1 &= \beta i_2^{**} m_1(m_2 + m_3) \\ &\quad +\mu\left[\frac{\gamma\epsilon m_1}{m_3} + \mu(\mu + \epsilon + \gamma) + m_1 m_3 + \mu(\mu + \gamma + \epsilon)(R_o - 1)\right] > 0. \end{aligned}$$

Following the same approach we have, $h_o = \alpha\beta m_3\mu\left[\frac{R_o - 1}{R_o}\right] > 0$. Since all the coefficients are positive, we now finalize the proof by establishing the Routh-Hurwitz criterion given in Appendix 1. Hence, the endemic equilibrium of the model (6)–(9) is locally asymptotically stable if $R_o > 1$. ■

Theorem 4 *The disease-free equilibrium point of the model (6)–(9) is globally asymptotically stable if $R_o < 1$ and unstable if $R_o > 1$.*

Proof. We construct the Lyapunov function

$$V = \alpha m_3 i_1 + m_1 m_3 i_2 + m_1\epsilon r.$$

Obtaining the time derivative gives

$$\dot{V} = \alpha m_3 \dot{i_1} + m_1 m_3 \dot{i_2} + m_1\epsilon\dot{r}.$$

Substituting equations (7), (8) and (9), to have

$$\begin{aligned} \dot{V} &= \alpha m_3(\beta x i_2 - m_1 i_1) + m_1 m_3(\alpha i_1 + \epsilon r - m_2 i_2) + m_1\epsilon(\gamma i_2 - m_3 r) \\ &= i_2[m_1(\epsilon\gamma - m_2 m_3) + \alpha\beta m_3 x] = -i_2[m_1(m_2 m_3 - \epsilon\gamma) - \alpha\beta m_3 x] \\ &\leq -i_2\left[1 - \frac{\alpha\beta m_3}{m_1(m_2 m_3 - \epsilon\gamma)}\right] \text{ by the feasible region } \Gamma. \end{aligned}$$

Then $\dot{V} \leq i_2\mu(R_o - 1)(\mu + \epsilon + \gamma)$. Vividly, $\dot{V} \leq 0$ when $R_o \leq 1$ and $\dot{V} = 0$ if $i_2 = 0$. Then, by Lassalle's In-variance Principle [12], every solution of the system (6)–(9) having the stated initial conditions in Γ approaches the disease-free equilibrium as t tends to infinity. Hence, since the region Γ is positively invariant as established earlier, the disease-free equilibrium is globally asymptotically stable if $R_o < 1$ [6]. For the global stability of the endemic equilibrium points, we state the following theorem without proof. ■

Theorem 5 *The endemic equilibrium point of the model (6)–(9) is globally asymptotically stable if $R_o > 1$ and unstable if $R_o < 1$.*

For the proof, please see the proof of Theorem 9.

3 The Vaccination Model of Childhood Disease

Biologically speaking, it has been established that childhood disease can be prevented using vaccine with no or very negligible waning rate and high efficacy. To this end, we modified the model to include the vaccination group where the vaccine is being administered to the children from birth (susceptible children) so that we can determine the effect of such vaccine via mathematical modeling technique.

3.1 The Vaccination Model Formulation

The basic model (6)–(9) is extended to include the population of vaccinated individuals represented by $V(t)$ so that the total population becomes $N = X(t) + I_1(t) + I_2(t) + V(t) + R(t)$. This compartment is obtained by vaccination of susceptible group at birth at the rate f. The model is as follows

$$\frac{dx(t)}{dt} = (1-f)\mu - \beta x i_2 - \mu x, \tag{14}$$

$$\frac{dv(t)}{dt} = \mu f - \mu v, \tag{15}$$

$$\frac{di_1(t)}{dt} = \beta x i_2 - m_1 i_1, \tag{16}$$

$$\frac{di_2(t)}{dt} = \alpha i_1 + \epsilon r - m_2 i_2, \tag{17}$$

$$\frac{dr(t)}{dt} = \gamma i_2 - m_3 r. \tag{18}$$

The invariant region of the above model is given as:

$$\Gamma_1 = \left\{(x(t), v(t), i_1(t), i_2(t), r(t)) \in R_+^5 : x(t) + v(t) + i_1(t) + i_2(t) + r(t) = 1\right\}. \tag{19}$$

The disease-free equilibrium is given by

$$E^o = \{1-f, f, 0, 0, 0\} \text{ i.e. } x^* = 1-f,\ v^* = f,\ i_1^* = 0,\ i_2^* = 0,\ r^* = 0.$$

As usual, the basic reproduction number is calculated as follows:

$$\mathcal{F}(u) = \begin{pmatrix} \beta x i_2 \\ 0 \\ 0 \end{pmatrix}, \quad \mathcal{V}(u) = \begin{pmatrix} m_1 i_1 \\ m_2 i_2 - \alpha i_1 - \epsilon r \\ m_3 r - \gamma i_2 \end{pmatrix}.$$

The derivative of the above expressions with respect to i_1, i_2, r evaluated at disease-free equilibrium gives

$$F = \begin{pmatrix} 0 & \beta(1-f) & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} m_1 & 0 & 0 \\ -\alpha & m_2 & -\epsilon \\ 0 & -\gamma & m_3 \end{pmatrix}.$$

The reproduction number expressed as $\rho(FV^{-1})$ which is the spectra radius of FV^{-1} is given by

$$R_v = \frac{(1-f)\beta\alpha m_3}{m_1(m_2 m_3 - \epsilon\gamma)}.$$

Hence,

$$R_v = (1-f)R_o.$$

The endemic equilibrium points E^1 can also be presented as follows

$$x^{**} = \frac{1-f}{R_v}, \quad v^{**} = f, \quad i_1^{**} = \frac{\mu^2(\epsilon+\gamma+\mu)(R_v-1)}{\alpha m_3 \beta}, \quad i_2^{**} = \frac{\mu(R_v-1)}{\beta},$$

$$r^{**} = \frac{\gamma\mu(R_v-1)}{\beta m_3}. \tag{20}$$

3.2 Local Stability Analysis of the Vaccination Model

We establish the theorem below without proof, using Theorem 2 in [18].

Theorem 6 *The disease-free equilibrium points of the model (14)–(18) is locally asymptotically stable if* $R_v < 1$ *and unstable otherwise.*

For the endemic equilibrium, we evaluate the Jacobian matrix of the model equation at endemic equilibrium points as follows:

$$J(E^1) = \begin{pmatrix} -\beta i_2^{**} - \mu & 0 & 0 & -\beta x^{**} & 0 \\ 0 & -\mu & 0 & 0 & 0 \\ \beta i_2^{**} & 0 & -m_1 & \beta x^{**} & 0 \\ 0 & 0 & \alpha & -m_2 & \epsilon \\ 0 & 0 & 0 & \gamma & -m_3 \end{pmatrix}.$$

Obviously, $\lambda_1 = -\mu$ is the first eigenvalue, the sign of the remaining eigenvalues can be determined by the reduced matrix:

$$J(E^1) = \begin{pmatrix} -\beta i_2^{**} - \mu & 0 & -\beta x^{**} & 0 \\ \beta i_2^{**} & -m_1 & \beta x^{**} & 0 \\ 0 & \alpha & -m_2 & \epsilon \\ 0 & 0 & \gamma & -m_3 \end{pmatrix},$$

$$f_4(\lambda) = \lambda^4 + b_1\lambda^3 + b_2\lambda^2 + b_3\lambda + b_4,$$

where

$$b_1 = m_1 + m_2 + m_3 + \beta i_2^{**} + \mu > 0,$$

$$b_2 = \beta i_2^{**}(m_1 + m_2 + m_3) + \mu(m_1 + m_2 + m_3) + m_1m_3 + \frac{\epsilon\gamma m_1}{m_3} + \mu(\mu + \epsilon + \gamma) > 0,$$

$$b_3 = \beta i_2^{**} m_1(m_2 + m_3) + \mu\left[\frac{\gamma\epsilon m_1}{m_3} + (\mu + \epsilon + \gamma) + m_1m_2 + \mu(\mu + \gamma + \epsilon)(R_v - 1)\right] > 0,$$

$$b_4 = \alpha\beta m_3\mu\left[\frac{R_v - 1}{R_v}\right] > 0,$$

where i_2^{**} is as given in equation (20). Since all the coefficients are positive when $R_v > 1$, the proof can be finalized by establishing the Routh-Hurwitz criterion similar to the one presented in Appendix 1. Therefore, the endemic equilibrium point is locally asymptotically stable if $R_v > 1$. Hence, we establish the theorem below.

Theorem 7 *The endemic equilibrium points of the model (14)–(18) is locally asymptotically stable if* $R_v > 1$ *and unstable if* $R_v < 1$.

3.3 Global Stability Analysis of the Vaccination Model

The global stability analysis of the disease-free equilibrium can be obtained using the following Theorem.

Theorem 8 *The disease-free equilibrium point of the model (14)–(18) is globally asymptotically stable if* $R_v < 1$ *and unstable if* $R_v > 1$.

We construct the Lyapunov candidate function

$$L = \alpha m_3 i_1 + m_1 m_3 i_2 + m_1 \epsilon r.$$

Obtaining the time derivative gives

$$\dot{L} = \alpha m_3 \dot{i}_1 + m_1 m_3 \dot{i}_2 + m_1 \epsilon \dot{r}.$$

Substituting the respective values of $\dot{i_1}$, $\dot{i_2}$ and $\dot{r}$, we have

$$\begin{aligned}\dot{L} &= \alpha m_3(\beta x i_2 - m_1 i_1) + m_1 m_3(\alpha i_1 + \epsilon r - m_2 i_2) + m_1\epsilon(\gamma i_2 - m_3 r)\\ &= i_2[m_1(\epsilon\gamma - m_2 m_3) + \alpha\beta x m_3] = -i_2[m_1(m_2 m_3 - \epsilon\gamma) - \alpha\beta m_3 x]\\ &= -i_2\left[1 - \frac{\alpha\beta m_3 x}{m_1(m_2 m_3 - \epsilon\gamma)}\right] m_1(m_2 m_3 - \epsilon\gamma)\\ &\leq i_2\left[\frac{\alpha\beta m_3(1-f)}{m_1(m_2 m_3 - \epsilon\gamma)} - 1\right] m_1(m_2 m_3 - \epsilon\gamma) \text{ by the feasible region } \Gamma_1.\end{aligned}$$

Then

$$\dot{L} \leq i_2\mu(\alpha+\epsilon)(\mu+\epsilon+\gamma)(R_v - 1).$$

Vividly, $\dot{L} \leq 0$ if $R_v \leq 1$ and $\dot{L} = 0$ if and only if $i_2 = 0$. Then, by Lassalle's Invariance Principle [12], every solution of the system (14)–(18) having the stated initial conditions $s(0)$, $i_1(0)$, $i_2(0)$, $r(0)$ in Γ_1 approaches the disease-free equilibrium as t tends to infinity. Hence, since the region Γ_1 is positively invariant as established before, the disease-free equilibrium is globally asymptotically stable if $R_v < 1$ [6].

3.4 Global Stability Analysis of the Endemic Equilibrium by Geometric Method.

Here, we shall examine the global stability analysis of the endemic equilibrium E^1 of the system (14)–(18) when $R_v > 1$. A geometrical approach developed by [13] for proving global stability will be adopted. This kind of approach is specifically based on the use of higher-order generalization of Bendixions' criterion which precludes the existence of non-constant periodic solution [14]. The instability of E^o implies the uniform persistence, i.e. there exists a constant $a > 0$ such that any solution $x(t)$, $i_1(t)$, $i_2(t)$, $r(t)$ with $x(0)$, $i_1(0)$, $i_2(0)$, $r(0)$ in the orbit of the system (14)–(18) satisfies

$$\min\left\{\liminf_{t\longrightarrow\infty} x(t), \liminf_{t\longrightarrow\infty} i_1(t), \liminf_{t\longrightarrow\infty} i_2(t), \liminf_{t\longrightarrow\infty} r(t)\right\} > a.$$

The following Lemma will provide some insight in the analysis.

Lemma 1 (Li and Muldowney [13]) *If the system*

$$\frac{dx}{dt} = f(x),$$

where $x \longrightarrow f(x) \in \mathbb{R}^n$, be a C^1 function for x in an open set $\Gamma_1 \subset \mathbb{R}^n$ such that

(i) *it has a unique equilibrium x^* in Γ_1 and*

(ii) *[3], there exists a compact absorbing set $Z \subset \Gamma_1$, then the equilibrium x^* in Γ_1 is globally asymptotically stable provided that a $\binom{n}{2} \times \binom{n}{2}$ matrix-valued function $P(x)$ and Lozinskii measure θ of F with respect to a vector norm $|\cdot|$ in $\mathbb{R}^N$, $N = \binom{n}{2}$ (where N is the number of combinations of n by 2 and n is the number of compartments) exist such that the quantity B is given by*

$$B = \limsup_{t\to\infty}\sup_{x\in Z}\frac{1}{t}\int_0^t \theta[F(x, i_1, i_2, r)]ds < 0,$$

under the condition that

$$F = P_f P^{-1} + P J^{[2]} P^{-1}, \tag{21}$$

the matrix P_f is obtained by replacing each entry P_{ij} of P by its derivative in the direction of f and $J^{[2]}$ is the second additive compound matrix [2, 13] of the Jacobian matrix J i.e, $J(x) = Df(x)$ and

$$\theta(F) = \lim_{h\to 0^+}\frac{||I + hF|| - 1}{h}$$

where I is an identity matrix.

Theorem 9 *If $R_v > 1$, then the endemic equilibrium E^1 of system (14)–(18) is globally asymptotically stable provided that $z = max\{\gamma - m_2, \epsilon - m_3\}$, and*

$$\hat{q} = min\{\beta - (\mu + m_1), \alpha + z - (2\beta + \mu), 2\beta + z - m_3, \alpha - (m_2 + m_3)\}.$$

Proof. The provision of Theorem 1 is enough to show that the model equations is uniformly persistent whenever $R_v > 1$. In other words, the system (14)–(18) is uniformly persistent in the bounded set Γ_1 is the same as the existence of a compact absorbing set $Z \subset \Gamma_1$. Hence, conditions (i) and (ii) are satisfied since $R_v > 1$. Since v doesn't appear elsewhere in the model equations, equation (15) will be consequently ignored.

The Jacobian matrix of the model equation can be expressed as follows:

$$J(E^1) = \begin{bmatrix} -\beta i_2^{**} - \mu & 0 & -\beta x^{**} & 0 \\ \beta i_2^{**} & -m_1 & \beta x^{**} & 0 \\ 0 & \alpha & -m_2 & \epsilon \\ 0 & 0 & \gamma & -m_3 \end{bmatrix}.$$

The second additive compound matrix is given below

$$J^{[2]} = \begin{bmatrix} g_{11} & \beta x^{**} & 0 & \beta x^{**} & 0 & 0 \\ \alpha & g_{22} & \epsilon & 0 & 0 & 0 \\ 0 & \gamma & g_{33} & 0 & 0 & -\beta x^{**} \\ 0 & \beta i_2^{**} & 0 & g_{44} & \epsilon & 0 \\ 0 & 0 & \beta i_2^{**} & \gamma & g_{55} & \beta x^{**} \\ 0 & 0 & 0 & 0 & \alpha & g_{66} \end{bmatrix},$$

where

$$g_{11} = -(\beta i_2^{**} + \mu + m_1), g_{22} = -(\beta i_2^{**} + \mu + m_2), g_{33} = -(\beta i_2^{**} + \mu + m_3)$$
$$g_{44} = -(m_1 + m_2), g_{55} = -(m_1 + m_3), g_{66} = -(m_2 + m_3).$$

Let

$$P = \text{diag}\left(\frac{1}{I}, \frac{1}{I}, \frac{1}{I}, \frac{1}{I}, \frac{1}{I}, \frac{1}{I}, \frac{1}{I},\right), \; P^{-1} = \text{diag}(I, I, I, I, I, I),$$
$$P_f = -\text{diag}\left(\frac{I'}{I^2}, \frac{I'}{I^2}, \frac{I'}{I^2}, \frac{I'}{I^2}, \frac{I'}{I^2}, \frac{I'}{I^2}\right),$$
$$P_f P^{-1} = -\text{diag}\left(\frac{I'}{I}, \frac{I'}{I}, \frac{I'}{I}, \frac{I'}{I}, \frac{I'}{I}, \frac{I'}{I},\right). \tag{22}$$

We evaluate

$$F = P_f P^{-1} + P J^{[2]} P^{-1} = \begin{bmatrix} g_{11} - \frac{I'}{I} & \beta x^{**} & 0 & \beta x^{**} & 0 & 0 \\ \alpha & g_{22} - \frac{I'}{I} & \epsilon & 0 & 0 & 0 \\ 0 & \gamma & g_{33} - \frac{I'}{I} & 0 & 0 & -\beta x^{**} \\ 0 & \beta i_2^{**} & 0 & g_{44} - \frac{I'}{I} & \epsilon & 0 \\ 0 & 0 & \beta i_2^{**} & \gamma & g_{55} - \frac{I'}{I} & \beta x^{**} \\ 0 & 0 & 0 & 0 & \alpha & g_{66} - \frac{I'}{I} \end{bmatrix}.$$

From here, we have the following sets

$$F_{11} = g_{11} - \frac{I'}{I}, \; F_{12} = (\beta x^{**}, 0), \; F_{13} = (\beta x^{**}, 0), \; F_{14} = (0),$$

$$F_{21} = (\alpha, 0)^T,\ F_{22} = \begin{pmatrix} g_{22} - \frac{I^{'}}{I} & \epsilon \\ \gamma & g_{33} - \frac{I^{'}}{I} \end{pmatrix},\ F_{23} = 0,\ F_{24} = (0, -\beta x^{**})^T,$$

$$F_{31} = (0),\ F_{32} = \begin{pmatrix} \beta i_2^{**} & 0 \\ 0 & \beta i_2^{**} \end{pmatrix},\ F_{33} = \begin{pmatrix} g_{44} - \frac{I^{'}}{I} & \epsilon \\ \gamma & g_{55} - \frac{I^{'}}{I} \end{pmatrix},$$

$$F_{34} = (0, \beta x^{**})^T,\ F_{41} = (0),\ F_{42} = (0),\ F_{43} = (0, \alpha),\ F_{44} = \left(g_{66} - \frac{I^{'}}{I} \right).$$

Let $u = (u_1, u_2, u_3, u_4, u_5, u_6)$ denote a vector in $R^6 \cong R \begin{pmatrix} 4 \\ 2 \end{pmatrix}$, we select a norm in R^6 as

$$\|(u_1, u_2, u_3, u_4, u_5, u_6)\| = \max\left\{|u_1|, |u_2| + |u_3|, |u_4| + |u_5|, |u_6|\right\}.$$

Now we have

$$\theta[F(x, i_1, i_2, r)] \leq \sup\left\{u_1, u_2, u_3, u_4\right\}$$

where

$$u_1 = \theta_1(F_{11}) + |F_{12}| + |F_{13}| + |F_{14}|, u_2 = \theta_1(F_{22}) + |F_{21}| + |F_{23}| + |F_{24}|,$$
$$u_3 = \theta_1(F_{33}) + |F_{31}| + |F_{32}| + |F_{34}|, u_4 = \theta_1(F_{44}) + |F_{41}| + |F_{42}| + |F_{43}|,$$
$$\theta_1(F_{11}) = -(\beta i_2^{**} + \mu + m_1 + \frac{I^{'}}{I}), \theta_1(F_{22}) = -\frac{I^{'}}{I} - (\beta i_2^{**} + \mu) + z,$$

and

$$\theta_1(F_{33}) = -\frac{I^{'}}{I} - m_3 + z,$$

where $z = \max\left\{\gamma - m_2, \epsilon - m_3\right\}$. After some simplifications, we have the following

$$u_1 = \theta_1(F_{11}) + |F_{12}| + |F_{13}| + |F_{14}| \leq -\frac{I^{'}}{I} - (\beta + \mu + m_1) + 2\beta,$$

$$u_2 = \theta_1(F_{22}) + |F_{21}| + |F_{23}| + |F_{24}| \leq -\frac{I^{'}}{I} - (2\beta + \mu) + \alpha + z,$$

$$u_3 = \theta_1(F_{33}) + |F_{31}| + |F_{32}| + |F_{34}| \leq -\frac{I^{'}}{I} + 2\beta - m_3 + z,$$

$$u_4 = \theta_1(F_{44}) + |F_{41}| + |F_{42}| + |F_{43}| \leq -\frac{I^{'}}{I} - (m_2 + m_3) + \alpha.$$

And the non differential part is given as

$$\hat{q} = \min\left\{\beta - (\mu + m_1), \alpha + z - (2\beta + \mu), 2\beta + z - m_3, \alpha - (m_2 + m_3)\right\}$$

such that

$$\theta(F) \leq -\frac{I^{'}}{I} - \hat{q}. \tag{23}$$

Given $[x(0), i_1(0), i_2(0), r(0)] \in n$ as the initial conditions of the system (16)–(20) when $t \to \infty$, we have

$$\frac{1}{t}\int_0^t \theta(F)ds \leq \frac{1}{t}\int_0^t \left(-\frac{I^{'}}{I} - \hat{q}\right) ds = \frac{\ln I(0) - \ln I(t)}{t} - \hat{q} = \frac{1}{t}\ln\left(\frac{I(0)}{I(t)}\right) - \hat{q}.$$

Therefore,

$$B = \limsup_{t\to\infty} \sup_{x\in Z} \frac{1}{t}\int_0^t \theta[F(x, i_1, i_2, r)]ds \leq -\hat{q} < 0.$$

Provided that $\hat{q} > 0$. This completes the proof. ■

3.5 Threshold Analysis, Vaccine Impact and Effect of Relapse.

To further understand the effect of vaccine and contact rate, we carry out the threshold analysis by obtaining the partial derivative of the reproduction number with respect to f and β as follows:

$$\frac{\partial R_v}{\partial f} = \frac{-\alpha\beta m_3}{m_1(m_2m_3 - \epsilon\gamma)} = -R_o,$$

$$\frac{\partial R_v}{\partial \beta} = \frac{\alpha m_3(1-f)}{m_1(m_2m_3 - \epsilon\gamma)} = (1-f)\frac{R_o}{\beta},$$

$$\frac{\partial R_v}{\partial \epsilon} = \frac{\alpha\beta(1-f)\gamma}{\mu m_1(\mu+\epsilon+\gamma)^2}.$$

It follows that $\frac{\partial R_v}{\partial f} < 0$ for $0 \leq f < 1$. Hence, R_v is a decreasing function of f. Since the reproduction number is expected to signify reduction in disease persistence. This shows that the proposed vaccine would have a positive impact for any $f > 0$ since the vaccination of any fraction of the susceptible child would reduce the disease burden. Furthermore there is a unique $\bar{f}$ such that $R_v(\bar{f}) = 1$ given by

$$\bar{f} = 1 - \frac{1}{R_o}.$$

On the other-hand, $\frac{\partial R_v}{\partial \beta} > 0$ for $0 \leq f < 1$. Hence, R_v is an increasing function of β. This shows that increase in the contact rate β will increase R_v and results in the increase in disease burden. Furthermore there is a unique $\bar{\beta}$ such that $R_v(\bar{\beta}) = 1$ given by

$$\bar{\beta} = \frac{m_1(m_2m_3 - \epsilon\gamma)}{\alpha m_3(1-f)} = \frac{\beta}{R_v}.$$

Finally, $\frac{\partial R_v}{\partial \epsilon} > 0$ for $0 \leq f < 1$. Hence, R_v is an increasing function of ϵ. This shows that an increase in the relapse rate ϵ will increase R_v and results in the increase in disease burden. Furthermore there is a unique $\bar{\epsilon}$ such that $R_v(\bar{\epsilon}) = 1$ given by

$$\bar{\epsilon} = \frac{\mu[\alpha\beta(1-f) - \{\mu(\mu+\gamma+\alpha) + \alpha\gamma\}]}{\mu(\mu+\alpha) + \alpha\beta(f-1)}.$$

The effect of contact rate β and relapse rate ϵ on the reproduction number R_v is better understood using the contour plot presented in Figure 2. Parameter value used are $\mu = 0.016$, $\alpha = 0.9$, $\gamma = 0.5$, $f = 0.1$, $\beta = \epsilon = (0, 1)$. Figure 2 is the contour plot that shows that the reproduction number increases with increase in both contact rate and relapse rate. The reproduction number is minimal when both ϵ and β are minimal and maximal when both parameters are maximal. This consequently confirms the analytic result presented earlier that R_v is an increasing function of β and ϵ hence, to ensure disease eradication, effort must be made to reduce them to the barest minimum.

4 Numerical Solution of the System and Discussion of Results.

Numerical solution of the system of equation is always carried out to understand the behavior of the model and its parameters. In this section, we present the solution of the model equation which are numerically verified using Python Programming Language and Maple 18 software. Firstly, we consider the case when the reproduction number is more than unity ($R_v = 2.3471$) with parameter value $\beta = 0.8, \mu = 0.166, \epsilon = 0.89, \alpha = 0.9, \gamma = 0.5, f = 0.15$ which biologically signifies endemicity of the childhood disease in the community.

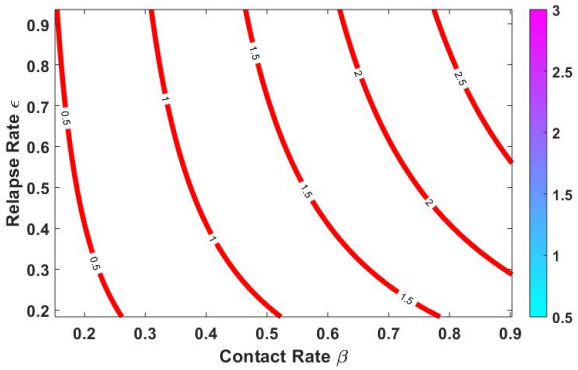

Figure 2: Effect of contact rate β and relapse rate ϵ on reproduction number R_v.

According to Figure 3, we carried out the solution in a small population size distributed over the five compartments with the following initial values

$$(x(0), v(0), i_1(0), i_2(0), r(0)) = (0.32, 0.31, 0.2, 0.11, 0.06),$$

$$(0.42, 0.22, 0.04, 0.23, 0.09),\ (0.57, 0.19, 0.11, 0.02, 0.11),\ (0.54, 0.1, 0.07, 0.11, 0.18),$$

where in each case, $x(0) + v(0) + i_1(0) + i_2(0) + r(0) = 1$. We discovered that the susceptible and vaccinated population declined steadily, while the other three infected population classes grow up steadily as time goes on due to high contact rate β, relapse rate ϵ and low vaccination rate f which consequently makes $R_v > 1$. This is in confirmation with theorem 7 and disease-free equilibrium points become unstable when $R_v > 1$.

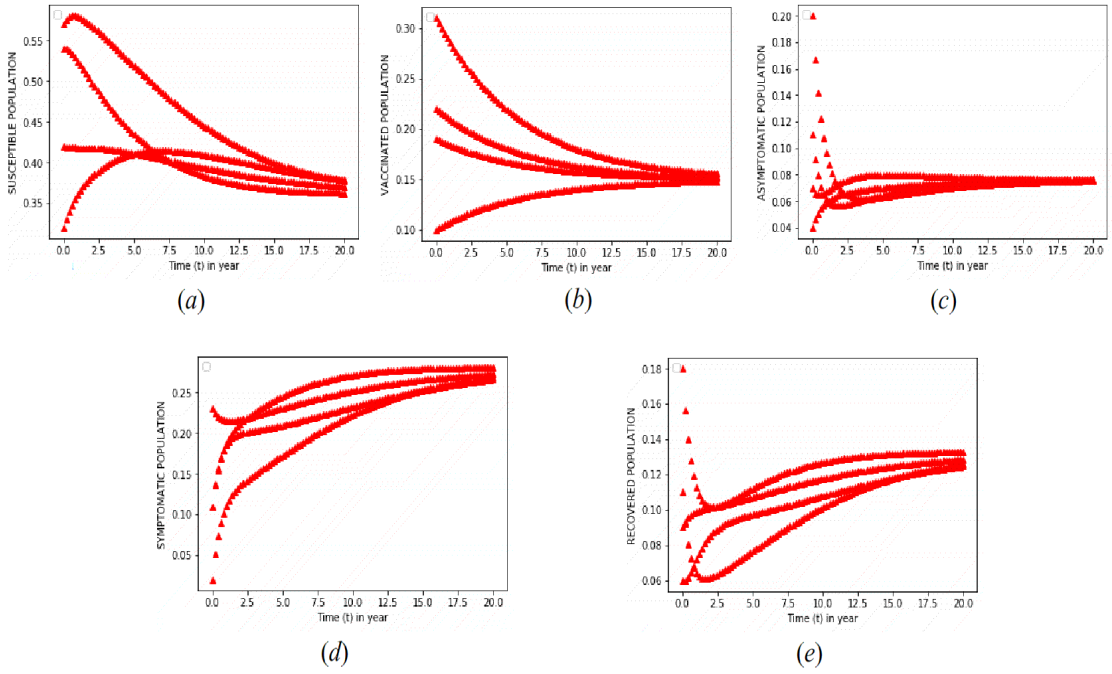

Figure 3: Graphical solution of x, v, i_1, i_2, r with $R_v > 1$.

With the same initial conditions, the graphical solution is presented in Figure 4 when ($R_v = 0.0588 < 1$) using parameter values $\beta = 0.1, \mu = 0.166, \epsilon = 0.2, \alpha = 0.12, \gamma = 0.5, f = 0.45$. It can be easily seen that the

susceptible and vaccinated population classes grow up steadily, while the other three infected populations decrease drastically (almost zero level) due to low contact rate β, relapse rate ϵ and high vaccination rate f which consequently makes $R_v < 1$. This is in confirmation with theorem 6 and endemic equilibrium points becomes unstable when $R_v < 1$.

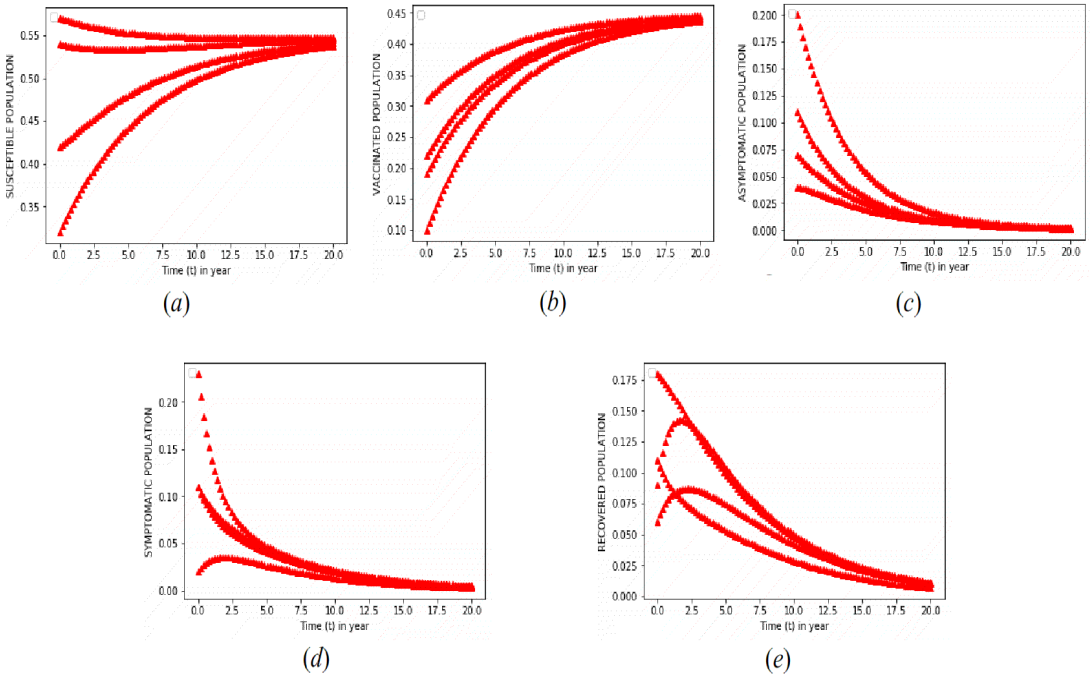

Figure 4: Graphical solution of x, v, i_1, i_2, r with $R_v < 1$.

In both figures, we can easily verify that when the relapse and the rate of contact between the children are high under low/no vaccination programme more people will get infected and the whole population will consequently wiped out in no time. Conversely, if the contact rate is low (with negligible relapse rate) under a very robust vaccination programme, few children will be infected and the disease spread will be kept at minimal level and under control. This underlines the effect of vaccination, relapse rate and contact rate in the spread of childhood disease. Finally, we present here the effect of relapse on the recovered population. It is worth noting that relapse contribute to the spread of childhood disease and this is graphically represented in Figure 5. It can be seen that the higher the rate at which children relapse, the fewer the recovered population and vice-versa. The recovered population is maximum when relapse rate $\epsilon = 0$ and the population is minimal when $\epsilon = 1$.

5 Conclusion and Acknowledgment

5.1 Conclusion

In this research, we developed a new childhood disease model that incorporates vaccine-induced immunity with relapse. The two models were rigorously analyzed to understand their dynamics. The global stability of the disease-free equilibrium points of model 2 was done using Lyapunov direct method while that of the endemic equilibrium was carried out using geometric method and compound matrix approach satisfying the Bendixon criterion. The threshold analysis, vaccine impact and effect of relapse rate were also investigated using the basic reproduction number of the vaccination model to understand the effect of vaccine and relapse in disease transmission. The results obtained show that, for the disease to be eradicated, the contact rate

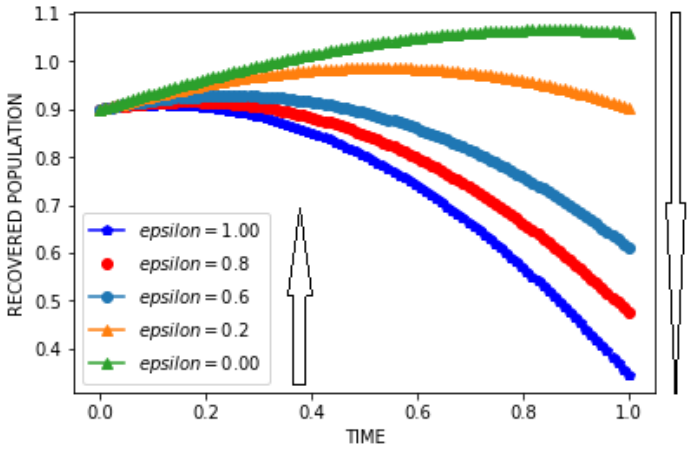

Figure 5: Effect of Relapse on Recovered Population.

β and relapse rate ϵ must be kept as minimal as possible while vaccine administration is at maximum level which in turn ensure that R_v is less than unity, hence disease is eradicated.

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6 Appendix 1: Proof of Routh-Hurwitz criterion of Equation (13)

Clearly, $h_i > 0$ for $i = 0, 1, 2, 3, 4$ and matrices $M_i > 0$ for $i = 1, 2, 3, 4$. The matrices are found positive as follows:

$$M_1 = h_3 > 0, \quad M_2 = \begin{vmatrix} h_3 & h_4 \\ h_1 & h_2 \end{vmatrix} > 0, \quad M_3 = \begin{vmatrix} h_3 & h_4 & 0 \\ h_1 & h_2 & h_3 \\ 0 & h_o & h_1 \end{vmatrix} > 0,$$

$$M_4 = \begin{vmatrix} h_3 & h_4 & 0 & 0 \\ h_1 & h_2 & h_3 & h_4 \\ 0 & h_o & h_1 & h_2 \\ 0 & 0 & 0 & h_o \end{vmatrix} > 0.$$

We will prove $M_2 > 0$ only as the proof of $M_3 > 0$ and $M_4 > 0$ directly follows.

$$\begin{aligned} M_2 &= h_2h_3 - h_1h_4 \\ &= \left[(\beta i_2^{**} + \mu)(m_1 + m_2 + m_3) + m_1m_3 + \frac{\epsilon\gamma m_1}{m_3} + \mu(\mu + \epsilon + \gamma)\right] A \\ &\quad - \left[\beta i_2^{**} m_1(m_2 + m_3) + \mu\left[\frac{\gamma\epsilon m_1}{m_3} + B + m_1m_2 + \mu(\mu + \gamma + \epsilon)(R_o - 1)\right]\right]. \end{aligned}$$

where $A = (m_1 + m_2 + m_3 + \beta i_2^{**} + \mu)$, $B = (\mu + \epsilon + \gamma)$. By putting back the original value of R_o and expressions in (12) coupled with some serious algebraic simplifications, we expand with Maple 18 and the

result is as follows:

$$
\begin{aligned}
&2\gamma m_1\epsilon + \beta i_2^{**}(m_1m_2 + m_2m_3 + \gamma\epsilon + 2m_1m_3) + \beta x^{**}(\alpha\mu + \alpha m_3) + \mu(\gamma\epsilon + \mu^2)\\
&+m_1(\mu m_1 + 2\mu^2 + m_1m_3) + m_2(\mu m_2 + 2\mu^2) + \mu^2(\gamma + \epsilon) + \mu(\gamma m_3 + \epsilon m_2)\\
&+m_3\mu(m_3 + 2\mu) + 2\beta i_2^{**}\mu(m_1 + m_2 + m_3) + \frac{\epsilon\gamma m_1^2}{m_3} + \beta i_2^{**}(\gamma\mu + \mu\epsilon + m_1^2 + \mu^2 + \beta i_2^{**}m_2)\\
&+\mu\gamma(m_1 + m_2) + \beta i_2^{**}\left[\beta i_2^{**}m_1 + m_2^2 + \beta i_2^{**}m_3 + \frac{\epsilon\gamma m_1}{m_3}\right] + \beta i_2^{**}m_3^2\\
&+\mu m_3\epsilon + \frac{\epsilon\gamma m_1}{m_3}(\mu + m_2) + m_1(\mu m_2 + m_3^2) + 2\mu m_3(m_1 + m_2) > 0
\end{aligned}
$$

which is strictly greater than zero.

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