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Modelling Childhood Disease Outbreak in a Community with Inflow of Susceptible and Vaccinated New-born

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Abstract

This paper investigates the transmission dynamics of a Childhood disease outbreak in a community with direct inflow of susceptible and vaccinated new-born. Qualitative analysis of the SEIR nonlinear model is performed for disease free and endemic equilibria using the stability theory of differential equations. The disease free state is found to be both locally and globally asymptotically stable when the vaccination reproductive number R_v is less than unity. In addition, the model exhibits transcritical forward bifurcation phenomenon and the sensitivity indices of the vaccination reproductive number with respect to various model parameters is determined. Using the Adomian decomposition method (ADM) and the fourth order Runge-Kutta integration scheme (RK4), the semi-analytical and numerical solutions of the nonlinear model are obtained. Pertinent results are displayed graphically and in tabular form. A vaccination coverage threshold is obtained above which the disease will be effectively eliminated from the community.

Keywords: Childhood diseases; Epidemiological model; Vaccination coverage; Forward bifurcation; Sensitivity indices; Adomian decomposition method; Runge-Kutta integration scheme.

INTRODUCTION

Childhood diseases are increasingly becoming the most common form of infectious diseases. These diseases include measles, mumps, Influenza, smallpox, chicken pox, Rubella, Polio etc. [14, 17, 19] which take special interest to children under-five years who are born highly susceptible. Childhood diseases have characteristics which make them well fit for mathematical modelling such as a relatively short incubation and infectious periods, confer permanent immunity when vaccinated etc. [11, 14, 17, 19].
As in most childhood diseases, there is a time delay between an individual becoming infected and developing disease symptoms. The exposed group only have the disease virus in the body due to exposure to the virus but have not developed the disease symptoms physically [15]. We have both the exposed and infected groups being infectious, but the exposed group are less infectious than the infected group since the infected individuals are already showing the symptoms of the disease. Childhood diseases such as Influenza, measles, poliomyelitis, hepatitis etc. in which substantial proportion of transmission is by exposed individuals makes Children under five years' effective spreaders since transmission is mainly through close contact, nasal excretion, faeces etc. regardless of the existence of visible symptoms.
Control measures include pharmaceutical interventions e.g. use of Drugs for treatment, vaccination of susceptible etc. Vaccination [1, 2, 20], if available is the most effective and safe preventive strategy against most childhood infectious diseases in the community. Since vaccination has been proved to be the most effective prevention strategy against childhood diseases, therefore, the need to obtain optimal vaccine coverage level needed to control the spread of childhood diseases is crucial in the twenty-first century.
Our main goal is to examine and analyse a SEIR model that monitors the transmission dynamics of Childhood diseases in the presence of preventive vaccine. This paper is organized as, in Section 2 we derive a SEIR model and state the underlying assumptions. In Section 3, the SEIR model is analysed qualitatively. In section 4 the sensitivity analysis is done. Section 5 discusses the bifurcation phenomenon of the model. In Sections 6,7 we apply Adomain decomposition method and Fourth order Runge-Kutta method respectively. The computational results to show the population dynamics and discussions are presented in Section 8. Section 9 was devoted to conclusions.

THE MATHEMATICAL MODEL

A SEIR model was formulated and analysed to investigate the transmission dynamics of Childhood diseases in a varying population size. The model has four epidemiological compartments: The susceptible *S*, an exposed *E*, an infective *I*, and a recovered group *R*, i.e. the vaccinated and treated group who poses permanent immunity to the disease. We assume the vaccines are 100% efficient and death rates μ due to causes other than the disease in the compartments are not equal to births, leading to varying population size N. Citizens are recruited into the community with the proportion of susceptible that are vaccinated as P (with $0 < P < 1$) and take the rest to be susceptible. We assume the vaccination rate as π. A susceptible person will progress into the exposed group by being in contact with an infective individual, estimated by a contact rate β_2 or through contact with an exposed individual, estimated by a contact rate β_1. An exposed individual Progresses from exposed to the infective compartment at a rate δ. An infective child progresses from infected to the recovered group due to treatment at a rate γ. We also assume α to be death rate due to disease infection.

The resulting differential equations for the model are,

$$\begin{cases} \dfrac{dS}{dt} = (1-P)\pi N - \left(\dfrac{\beta_1 E + \beta_2 I}{N}\right)S - \mu S, \\ \dfrac{dE}{dt} = \left(\dfrac{\beta_1 E + \beta_2 I}{N}\right)S - (\delta + \mu)E, \\ \dfrac{dI}{dt} = \delta E - (\alpha + \gamma + \mu)I, \\ \dfrac{dR}{dt} = P\pi N + \gamma I - \mu R, \end{cases} \tag{1}$$

Subject to the initial conditions $S(0) = S_0, E(0) = E_0, I(0) = I_0$ and $R(0) = R_0$. We also have that $N = S + E + I + R$ and assuming that $\mu, \beta_1, \beta_2, \pi, \alpha, \gamma, \delta$ are all non-negative parameters. Adding the equations of the governing model system (1) we have,

$$\frac{dN}{dt} = (\pi - \mu)N - \alpha I, \tag{2}$$

which exhibits a varying population size [1] with deaths due to fatal diseases.

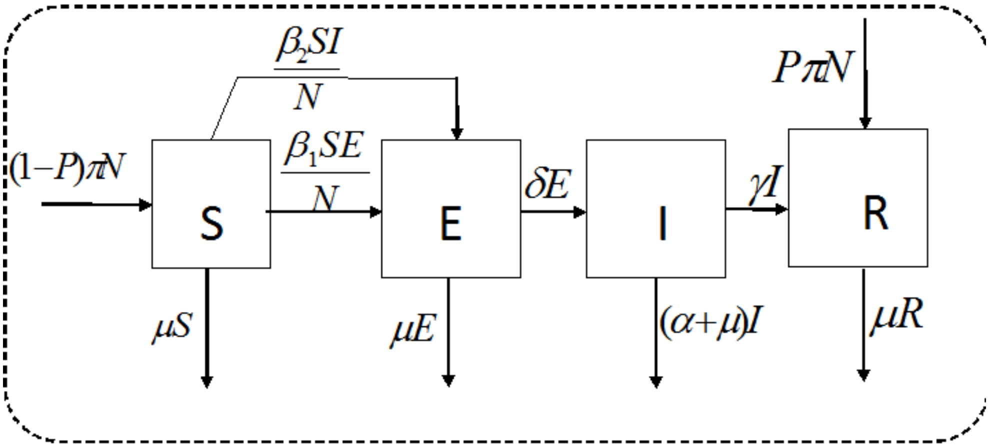

Figure 1: SEIR model flow chart

We introduce the following variables, $s = S/N, e = E/N, i = I/N$ and $r = R/N$, which normalizes the population, with $s+e+i+r=1$ and we obtain the following system,

$$\begin{cases} \frac{ds}{dt} = (1-P)\pi - (\beta_1 e + \beta_2 i)s - \pi s + \alpha is, \\ \frac{de}{dt} = (\beta_1 e + \beta_2 i)s - (\delta + \pi)e + \alpha ie, \\ \frac{di}{dt} = \delta e - (\alpha + \gamma + \pi)i + \alpha i^2, \\ \frac{dr}{dt} = P\pi + (\gamma + \alpha)I - \pi r. \end{cases} \quad (3)$$

We observe that the variable r appears in the fourth equation of system (3) only. Hence, we can study the system qualitatively by considering the sub-system,

$$\begin{cases} \frac{ds}{dt} = (1-P)\pi - (\beta_1 e + \beta_2 i)s - \pi s + \alpha is, \\ \frac{de}{dt} = (\beta_1 e + \beta_2 i)s - (\delta + \pi)e + \alpha ie, \\ \frac{di}{dt} = \delta e - (\alpha + \gamma + \pi)i + \alpha i^2, \end{cases} \quad (4)$$

where r can be determined from $r = 1 - s - e - i$ or $\frac{dr}{dt} = P\pi + (\gamma + \alpha)i - \pi r$.

The feasible solutions of the sub-system (4) in $\Re_+^3$ are confined in the region,

$$\Gamma = \{s, e, i \in \Re_+^3 : 0 \le s + e + i \le 1\}. \quad (5)$$

It is easy to verify that the region $\Gamma \subset \Re_+^3$ is positively invariant in relation to the sub-system (4) subject to initial conditions in $\Re_+^3$.

QUALITATIVE ANALYSIS OF THE MODEL.

The model was analysed qualitatively in the set Γ. A qualitative study reveals that the model has only two equilibria: Disease free equilibrium i.e. in the absence of infection and Endemic equilibrium i.e. in the presence of infection. The Jacobian Matrix [10, 13,18] of the governing model sub-system (4) yields,

$$J = \begin{bmatrix} a & -\beta_1 s & (\alpha - \beta_2)s \\ \beta_1 e + \beta_2 i & b & -\beta_2 s + \alpha e \\ 0 & \delta & c \end{bmatrix}, \quad (6)$$

where $a = -(\beta_1 e + \beta_2 i) - \pi + \alpha i, b = \beta_1 s - (\delta + \pi) + \alpha i$ and $c = -(\alpha + \gamma + \pi) + 2\alpha i$.

Stability of Disease-free equilibrium :

In the absence of an infection i.e. Disease die out, the solution asymptotically tends to a disease-free state which exists and is given by, $E^0 = (1-P,0,0)$. The threshold parameter value (vaccination reproduction number, R_v) that determines the stability of equilibria was computed using the Jacobian approach [10, 13] computed at the disease-free state (E^0). Thus, the vaccination reproduction number,

$$R_v = \frac{[\beta_1(\alpha+\gamma+\pi)+\delta\beta_2](1-P)}{(\delta+\pi)(\alpha+\gamma+\pi)}. \tag{7}$$

A local stability analysis of Disease-free state using the Jacobian method [10, 13,18] reveals that If $R_v < 1$ the disease-free state of the SEIR model is locally asymptotically stable(LAS) and unstable if $R_v > 1$. The Global stability analysis revealed that the maximum invariant set contained in the set $\{(s,e,i) \in \Gamma : \frac{dV}{dt} = 0\}$,with $V = (\alpha+\gamma+\pi)e + \beta_2 i$ as the Lyapunov function [10],is the disease-free equilibrium (E^0). Global asymptotic stability for disease-free equilibrium (E^0) is achieved by using Lasalle-Lyapunov theorem for $R_v < 1$[10, 13, 18, 19]. From equation (7), we compute the critical vaccination proportion (P_c) by setting $R_v = 1$ which gives,

$$P_c = 1 - \frac{1}{R_0}, \tag{8}$$

where, $R_0 = \frac{\beta_1(\alpha+\gamma+\pi)+\delta\beta_2}{(\delta+\pi)(\alpha+\gamma+\pi)}$., i.e. R_0 is the basic reproduction number [10, 13]. Hence, if $R_v < 1 \Leftrightarrow P > P_c$ i.e. the disease-free equilibrium is stable since the vaccination coverage is large enough and can lead to successful prevention of the disease.

The Existence of endemic equilibrium:

In the presence of an infection $(e \neq 0, i \neq 0)$, the model has an endemic equilibrium, $E^u = (s^*, e^*, i^*)$ calculated as follows:

$$\begin{cases} (1-P)\pi - \left(\beta_1 e^* + \beta_2 i^*\right)s^* - \pi s^* + \alpha i^* s^* = 0, \\ \left(\beta_1 e^* + \beta_2 i^*\right)s^* - (\delta+\pi)e^* + \alpha i^* e^* = 0, \\ \delta e^* - (\alpha+\gamma+\pi)i^* + \alpha i^{*2} = 0, \end{cases} \tag{9}$$

with $s^* > 0, e^* > 0$ *and* $i^* > 0$. We determine s^* and e^* uniquely as,

$$s^* = \frac{[(\delta+\pi)-\alpha i^*][(\alpha+\gamma+\pi)-\alpha i^*]}{\beta_1(\alpha+\gamma+\pi)+\delta\beta_2-\alpha\beta_1 i^*}, \qquad (10)$$

and

$$e^* = \frac{(\alpha+\gamma+\pi)i^* - \alpha i^{*2}}{\delta}. \qquad (11)$$

Adding equations of subsystem (9) yields,

$$(\pi-\alpha i^*)(1-s^*-e^*-i^*) = \gamma i^* + P\pi \qquad (12)$$

and we obtain the range of i^* as,

$$0 < i^* < \min\{1, \frac{\pi}{\alpha}\}. \qquad (13)$$

We direct our readers to [16] for a proof of equation (13).

Substituting s^* and e^* to subsystem (9) and evaluating simultaneously, we have that i^* satisfies $f(i^*) = R_v$ where,

$$f(i^*) = \frac{\alpha\beta_1(1-P)}{(\delta+\pi)(\alpha+\gamma+\pi)} i^* + (1-\frac{\alpha}{\pi}i^*)(1-\frac{\alpha}{\delta+\pi}i^*)(1-\frac{\alpha}{\alpha+\gamma+\pi}i^*) +$$
$$\frac{1}{\delta}(1-\frac{\alpha\beta_1(1-P)}{R_v(\delta+\pi)(\alpha+\gamma+\pi)}i^*)(1-\frac{\alpha}{\delta+\pi}i^*)(1-\frac{\alpha}{\alpha+\gamma+\pi}i^*)i^*,$$

and R_v is as defined in equation (7).

The roots of $f(i^*)$ are $i_1^* = \frac{\pi}{\alpha}, i_2^* = \frac{\delta+\pi}{\alpha}, i_3^* = \frac{\alpha+\gamma+\pi}{\alpha}$ and $i_4^* = \frac{R_v(\delta+\pi)(\alpha+\gamma+\pi)}{\alpha\beta_1(1-P)}$. We note that the roots i_2^*, i_3^* and i_4^* all lie outside $[0, \frac{\pi}{\alpha}]$ when $R_v > 1$.

We observe that

$$f(0) = 1 \qquad (14)$$

and

$$f(\frac{\pi}{\alpha}) = R_v[\frac{\pi^2}{\alpha\delta(1-P)} + \frac{\pi(\alpha+\delta+\gamma+2\pi)}{\alpha\delta(1-P)} + \frac{(\delta+\pi)(\alpha+\gamma+\pi)}{\delta(1-P)} - \frac{\pi^3}{\alpha\delta(\alpha+\gamma+\pi)}] + K > R_v, \qquad (15)$$

where

$$\text{K} = \frac{1}{\alpha\delta(\delta+\pi)(\alpha+\gamma+\pi)}\{\frac{\delta\pi^3\beta_2}{\alpha+\gamma+\pi} + \pi^2(\beta_1(\alpha+\delta+\gamma+2\pi)-\delta\pi) + \pi[\beta_1(\delta+\pi)(\alpha+\gamma+\pi)$$
$$-\alpha\delta(\alpha+\delta+\gamma+2\pi)] + \alpha\delta\beta_1(1-P)\pi - \alpha\delta[\pi(\delta+\pi)+\pi(\alpha+\gamma+\pi)+(\delta+\pi)(\alpha+\gamma+\pi)]\} + 1.$$

Also

$$f(1) = R_v[\frac{\alpha^2}{\pi\delta(1-P)} + \frac{\alpha(\alpha+\delta+\gamma+2\pi)}{\pi\delta(1-P)} + \frac{\alpha(\delta+\pi)(\alpha+\gamma+\pi)}{\delta\pi(1-P)} - \frac{\alpha^3}{\pi\delta(\alpha+\gamma+\pi)}] + L > R_v, \quad (16)$$

where

$$\mathrm{L} = \frac{1}{\pi\delta(\delta+\pi)(\alpha+\gamma+\pi)}\{\frac{\delta\alpha^3\beta_2}{\alpha+\gamma+\pi} + \alpha^2(\beta_1(\alpha+\delta+\gamma+2\pi)-\delta\pi) + \alpha[\beta_1(\delta+\pi)(\alpha+\gamma+\pi) - \alpha\delta(\alpha+\delta+\gamma+2\pi)] + \alpha\delta\beta_1(1-P)\pi - \alpha\delta[\pi(\delta+\pi)+\pi(\alpha+\gamma+\pi)+(\delta+\pi)(\alpha+\gamma+\pi)]\} + 1.$$

The above observations (see equations (14,15,16)) imply that, when $R_v > 1$, the line $f(i^*) = R_v$ has only one point $(i^*, f(i^*))$ that intersects with the graph of $f(i)$. Thus, we achieve the following result.

Lemma 1. Suppose that $R_v > 1$, then the model subsystem (4) has a unique endemic equilibrium $E^u = (s^*, e^*, i^*)$.

The endemic equilibrium results to a quartic polynomial given by,

$$P(i^*) = Ai^{*4} + Bi^{*3} + Ci^{*2} + Di^* + E = 0, \quad (17)$$

where,

$$A = \frac{\alpha^3}{\pi\delta(\delta+\pi)(\alpha+\gamma+\pi)}(\frac{\delta\beta_2}{\alpha+\gamma+\pi} - \frac{R_v(\delta+\pi)}{1-P}),$$

$$B = \frac{\alpha^2}{\delta\pi}(\frac{R_v}{1-P} + \frac{\beta_1(\alpha+\delta+\gamma+2\pi)-\delta\pi}{(\delta+\pi)(\alpha+\gamma+\pi)}),$$

$$C = \frac{\alpha}{\delta\pi}(\frac{R_v(\alpha+\gamma+2\pi)}{1-P} + \frac{\beta_1(\delta+\pi)(\alpha+\gamma+\pi)-\delta\alpha(\alpha+\delta+\gamma+3\pi)}{(\delta+\pi)(\alpha+\gamma+\pi)}),$$

$$D = \frac{\alpha}{\delta\pi}(\frac{R_v(\delta+\pi)(\alpha+\gamma+\pi)}{1-P} + \frac{\delta\beta_1(1-P)\pi - \delta[\pi(\delta+\pi)+\pi(\alpha+\gamma+\pi)+(\delta+\pi)(\alpha+\gamma+\pi)]}{(\delta+\pi)(\alpha+\gamma+\pi)}),$$

$$E = 1 - R_v.$$

Stability of Endemic equilibrium:

The Jacobian matrix [10, 13,18] of subsystem (4) computed at the endemic equilibrium, $E^u = (s^*, e^*, i^*)$ yields,

$$J_{E^u} = \begin{bmatrix} \frac{-(1-P)\pi}{s^*} & -\beta_1 s^* & (\alpha-\beta_2)s^* \\ \beta_1 e^* + \beta_2 i^* & \frac{-\beta_2 s^* i^*}{e^*} & -\beta_2 s^* + \alpha e^* \\ 0 & \delta & \frac{-(\delta e^* - \alpha i^{*2})}{i^*} \end{bmatrix}. \quad (18)$$

The resulting polynomial valuated at the endemic equilibrium, E^u gives,

$$P(\lambda) = \lambda^3 + A_1\lambda^2 + A_2\lambda + A_3 \tag{19}$$

where,

$$A_1 = \frac{\beta_2 s^* i^*}{e^*} + (\alpha + \gamma + \pi) + \frac{(1-P)\pi}{s^*},$$

$$A_2 = \frac{\beta_2 s^* i^*}{e^*}[(\alpha + \gamma + \pi) + \frac{(1-P)\pi}{s^*} + \frac{(\alpha+\gamma+\pi)(1-P)\pi}{s^*} - (\delta\beta_2 s^* + \alpha\delta e^* + \beta_1^2 s^* e^* + \beta_1\beta_2 s^* i^*)],$$

$$A_3 = \frac{\beta_2 s^* i^*}{e^*}(\alpha + \gamma + \pi) + (\alpha + \gamma + \pi)(\beta_1^2 s^* e^* + \beta_1\beta_2 s^* i^*) -$$
$$\delta(\alpha - \beta_2)(\beta_1 e^* + \beta_2 i^*)s^* - \delta\beta_2 s^* - \alpha\delta e^*,$$

and

$$A_1A_2 - A_3 = [\frac{\beta_2 s^* i^*}{e^*} + (\alpha + \gamma + \pi) + \frac{(1-P)\pi}{s^*}][\frac{\beta_2 s^* i^*}{e^*}((\alpha + \gamma + \pi) +$$
$$\frac{(1-P)\pi}{s^*} + \frac{(\alpha+\gamma+\pi)(1-P)\pi}{s^*} - (\delta\beta_2 s^* + \alpha\delta e^* + \beta_1^2 s^* e^* +$$
$$\beta_1\beta_2 s^* i^*))] - \frac{\beta_2 s^* i^*}{e^*}(\alpha + \gamma + \pi) - (\alpha + \gamma + \pi)(\beta_1^2 s^* e^* + \beta_1\beta_2 s^* i^*) +$$
$$\delta(\alpha - \beta_2)(\beta_1 e^* + \beta_2 i^*)s^* + \delta\beta_2 s^* + \alpha\delta e^*].$$

Clearly, $A_1 > 0$ and we observe that $R_v > 1$ implies that $\beta_2 > \alpha$ which leads to $A_3 > 0$. Hence, by Routh-Hurwitz criteria [10, 21] the endemic equilibrium E^u, which exists if $R_v > 1$, gives a locally asymptotically stable provided $A_2 > 0$ and $A_1A_2 - A_3 > 0$.

SENSITIVITY ANALYSIS

Here we determine the changes occurring in childhood disease temporal dynamics by computing the sensitivity indices of the vaccination reproduction Number, R_v with respect to the parameter values in the model. The sensitivity indices measure the change in model variables when a parameter changes [14]. Sensitivity analysis is essential in determining certain key parameter values that are highly sensitive and influence the outcome of a disease outbreak and must be targeted by prevention strategies [14].

The sensitivity indices of R_v were computed using the following estimated parameter values; $P = 0.3846, \beta_1 = 0.2, \beta_2 = 0.3, \gamma = 0.1, \alpha = 0.04, \pi = 0.1$ and $\delta = 0.1$.

We used the normalized forward sensitivity indexof a variable, P, which depends differentially on a parameter, q, defined as [14, 23]:

$$Z_q^P = \frac{\partial P}{\partial q} \times \frac{q}{P}. \tag{20}$$

From equation (20), we derived the sensitivity index of R_v given by $Z_q^{R_v} = \frac{\partial R_v}{\partial q} \times \frac{q}{R_v}$ with respect to the parameters contained in R_v. Some illustrative examples of sensitivity values of R_v in relation to β_1 and P are given by; $Z_{\beta_1}^{R_v} = \frac{\partial R_v}{\partial \beta_1} \times \frac{\beta_1}{R_v} = 0.615385$ and $Z_P^{R_v} = \frac{\partial R_v}{\partial P} \times \frac{P}{R_v} = -0.624959$.

The other indices are computed as: $Z_\alpha^{R_v}, Z_\gamma^{R_v}, Z_\delta^{R_v}, Z_\pi^{R_v}$ and $Z_{\beta_2}^{R_v}$ using the same approach.

Table 1: Sensitivity indices of R_v.

Parameter	**Parameter description**	**sensitivity indices**
$\boldsymbol{\pi}$	Proportion of susceptible that are vaccinated at birth	−0.660256
$\boldsymbol{P}$	Vaccination rate at birth	−0.624959
$\boldsymbol{\beta_1}$	Susceptible contact rate of disease from exposed group	0.615385
$\boldsymbol{\beta_2}$	Susceptible contact rate of disease from infected group	0.384615
$\boldsymbol{\gamma}$	Progression rate from infected to recovered group	−0.160256
$\boldsymbol{\delta}$	Progression rate from exposed to infected group	−0.115385
$\boldsymbol{\alpha}$	The death rate due to disease infection	−0.064103

Table1 above shows the numerical values of sensitivity indices of R_v to the parameter values for the SEIR model. The parameters are listed from the highly sensitive to the ones with low sensitivity. The highly sensitive parameters include, the Proportion of susceptible children that are vaccinated at birth π, followed by vaccination coverage, P and then Susceptible contact rate of disease from exposed group, β_1 while the least sensitive parameter is the death rate due to disease infection, α. From *Table 1*, we see that when the parameters β_1 and β_2 are increased while the other parameters are held constant, the numerical value of R_v increases. Hence,they increase the persistence of the childhood diseases in the community since they have positive indices. Also, the parameters $\alpha, \delta, \gamma, P$ and π decreases the computational value of R_v when increased while holding the other parameters constant. Thus, they decrease the persistence of the disease as they have negative indices.

BIFURCATION ANALYSIS

We illustrate the phenomenon of Bifurcation [10, 17] by considering the polynomial (17) resulting from the endemic equilibrium. The estimated parameter values above are used to plot the diagram. Multiplying equation (17) by $\frac{\pi\delta(\delta+\pi)(\alpha+\gamma+\pi)}{\alpha^3}$ yields,

$$a_1 i^{*4} + a_2 i^{*3} + a_3 i^{*2} + a_4 i^* + a_5 = 0, \tag{21}$$

where,

$$a_1 = \frac{\delta\beta_2}{\alpha+\gamma+\pi} - \frac{R_v(\delta+\pi)}{1-P},$$

$$a_2 = \frac{1}{\alpha}\left(\frac{R_v(\delta+\pi)(\alpha+\gamma+\pi)}{1-P} + \beta_1(\alpha+\delta+\gamma+2\pi) - \delta\pi\right),$$

$$a_3 = \frac{1}{\alpha^2}\left(\frac{R_v(\alpha+\delta+\gamma+2\pi)(\delta+\pi)(\alpha+\gamma+\pi)}{1-P} + \beta_1(\delta+\pi)(\alpha+\gamma+\pi) - \delta\alpha(\alpha+\delta+\gamma+3\pi)\right),$$

$$a_4 = \frac{1}{\alpha^2}\left(\frac{R_v}{1-P} + \delta\beta_1(1-P)\pi - \delta[\pi(\delta+\pi) + \pi(\alpha+\gamma+\pi) + (\delta+\pi)(\alpha+\gamma+\pi)]\right),$$

$$a_5 = \frac{\pi\delta(\delta+\pi)(\alpha+\gamma+\pi)}{\alpha^3}(1-R_v),$$

and R_v is defined as in equation (7).We observe that equation (21) has a unique positive solution when $R_v > 1,$ since the constant term is negative. We consider equation (21) to define a curve of i^* against R_v in the positive quadrant.

ADOMAIN DECOMPOSITION METHOD

Adomain Decomposition approach [4, 6, 7, 8,9] was applied to the normalized system (3)to approximate the solutions. The method is efficient since it can be applied directly to many differential equations, linear andnonlinear etc. [4, 8]. The approach has the ability of minimizing the size of computation activities while upholding high levels of accuracy of the semi-numerical solution.

The reduced form of the system (3) of non-linear differential equations yields,

$$s(t) = s(0) + (1-P)\pi t - \beta_1\int_0^t esdt - (\beta_2 - \alpha)\int_0^t isdt - \pi\int_0^t sdt, \tag{22}$$

$$e(t) = e(0) + \beta_1\int_0^t esdt + \beta_2\int_0^t isdt - (\delta+\pi)\int_0^t edt + \alpha\int_0^t iedt, \tag{23}$$

$$i(t) = i(0) + \delta\int_0^t edt - (\alpha+\gamma+\pi)\int_0^t idt + \alpha\int_0^t i^2 dt, \tag{24}$$

$$r(t) = r(0) + P\pi t + (\gamma + \pi)\int_0^t i dt - \pi \int_0^t r dt. \tag{25}$$

The solutions of equations (22) -(25) are found as the sum of the following series:
The linear terms are given by,

$$s = \sum_{n=0}^{\infty} s_n, e = \sum_{n=0}^{\infty} e_n, i = \sum_{n=0}^{\infty} i_n, r = \sum_{n=0}^{\infty} r_n. \tag{26}$$

The non-linear terms are approximated as,

$$si = \sum_{n=0}^{\infty} A_n(s_0,...,s_n,i_0,...,i_n), ei = \sum_{n=0}^{\infty} B_n(e_0,...,e_n,i_0,...,i_n), se = \sum_{n=0}^{\infty} C_n(s_0,...,s_n,e_0,...,e_n)$$

and $i^2 = \sum_{n=0}^{\infty} D_n(i_0,...,i_n)$,

where

$$A_n = \frac{1}{n!}\left[\frac{d^n(\sum_{k=0}^{\infty} s_k\lambda^k)(\sum_{k=0}^{\infty} i_k\lambda^k))}{d\lambda^k}\right]_{\lambda=0}, B_n = \frac{1}{n!}\left[\frac{d^n(\sum_{k=0}^{\infty} e_k\lambda^k)(\sum_{k=0}^{\infty} i_k\lambda^k))}{d\lambda^k}\right]_{\lambda=0},$$

$$C_n = \frac{1}{n!}\left[\frac{d^n(\sum_{k=0}^{\infty} s_k\lambda^k)(\sum_{k=0}^{\infty} e_k\lambda^k))}{d\lambda^k}\right]_{\lambda=0}, D_n = \frac{1}{n!}\left[\frac{d^n(\sum_{k=0}^{\infty} i_k\lambda^k)^2}{d\lambda^k}\right]_{\lambda=0}.$$

The Adomain polynomials are the non-linear functions A_n, B_n, C_n and D_n. Substituting the Adomain polynomials and the linear terms (26) into equations (22)-(25) yields,

$$\sum_{n=0}^{\infty} s_n = s(0) + (1-P)\pi t - \beta_1\int_0^t \sum_{n=0}^{\infty} C_n dt - (\beta_2 - \alpha)\int_0^t \sum_{n=0}^{\infty} A_n dt - \pi\int_0^t \sum_{n=0}^{\infty} s_n dt, \tag{27}$$

$$\sum_{n=0}^{\infty} e_n = e(0) + \beta_1\int_0^t \sum_{n=0}^{\infty} C_n dt + \beta_2\int_0^t \sum_{n=0}^{\infty} A_n dt - (\delta + \pi)\int_0^t \sum_{n=0}^{\infty} e_n dt + \alpha\int_0^t \sum_{n=0}^{\infty} B_n dt, \tag{28}$$

$$\sum_{n=0}^{\infty} i_n = i(0) + \delta\int_0^t \sum_{n=0}^{\infty} e_n dt - (\alpha + \gamma + \pi)\int_0^t \sum_{n=0}^{\infty} i_n dt + \alpha\int_0^t \sum_{n=0}^{\infty} D_n dt, \tag{29}$$

$$\sum_{n=0}^{\infty} r_n = r(0) + P\pi t + (\gamma + \pi)\int_0^t \sum_{n=0}^{\infty} i_n dt - \pi\int_0^t \sum_{n=0}^{\infty} r_n dt. \tag{30}$$

Using equations (27)-(30), we have the initial conditions and recursive relationship as:

$$s_0 = s(0) + (1-P)\pi t, e_0 = e(0), i_0 = i(0), r_0 = r(0) + P\pi t \tag{31}$$

$$s_{n+1} = -\beta_1 \int_0^t C_n dt - (\beta_2 - \alpha)\int_0^t A_n dt - \pi \int_0^t s_n dt, \tag{32}$$

$$e_{n+1} = \beta_1 \int_0^t C_n dt + \beta_2 \int_0^t A_n dt - (\delta + \pi)\int_0^t e_n dt + \alpha \int_0^t B_n dt, \tag{33}$$

$$i_{n+1} = \delta \int_0^t e\ dt - (\alpha + \gamma + \pi)\int_0^t i_n dt + \alpha \int_0^t D_n dt, \tag{34}$$

$$r_{n+1} = (\gamma + \pi)\int_0^t i_n dt - \pi \int_0^t r_n dt. \tag{35}$$

Some few Adomain polynomials are computed as follows:

$A_0 = s_0 i_0, A_1 = s_0 i_1 + s_1 i_0, A_2 = s_0 i_2 + s_1 i_1 + s_2 i_0, A_3 = s_0 i_3 + s_1 i_2 + s_2 i_1 + s_3 i_0,$

$A_4 = s_0 i_4 + s_1 i_3 + s_2 i_2 + s_3 i_1 + s_4 i_0, A_5 = s_0 i_5 + s_1 i_4 + s_2 i_3 + s_3 i_2 + s_4 i_1 + s_5 i_0, \ldots$

$B_0 = e_0 i_0, B_1 = e_0 i_1 + e_1 i_0, B_2 = e_0 i_2 + e_1 i_1 + e_2 i_0, B_3 = e_0 i_3 + e_1 i_2 + e_2 i_1 + e_3 i_0,$

$B_4 = e_0 i_4 + e_1 i_3 + e_2 i_2 + e_3 i_1 + e_4 i_0, B_5 = e_0 i_5 + e_1 i_4 + e_2 i_3 + e_3 i_2 + e_4 i_1 + e_5 i_0, \ldots$

$C_0 = s_0 e_0, C_1 = s_0 e_1 + s_1 e_0, C_2 = s_0 e_2 + s_1 e_1 + s_2 e_0, C_3 = s_0 e_3 + s_1 e_2 + s_2 e_1 + s_3 e_0,$

$C_4 = s_0 e_4 + s_1 e_3 + s_2 e_2 + s_3 e_1 + s_4 e_0, C_5 = s_0 e_5 + s_1 e_4 + s_2 e_3 + s_3 e_2 + s_4 e_1 + s_5 e_0, \ldots$

$D_0 = i_0^2, D_1 = 2 i_0 i_1, D_2 = 2 i_0 i_2 + i_1^2, D_3 = 2 i_0 i_3 + 2 i_1 i_2, D_4 = 2 i_0 i_4 + 2 i_1 i_3 + i_2^2,$

$D_5 = 2 i_0 i_5 + 2 i_1 i_4 + 2 i_2 i_3, \ldots$

Substituting equations (31)-(35) and A_n, B_n, C_n, D_n into equation (27)-(30),with the help of Maple we approximate the solution with a few terms as follows:

$$s_N = \sum_{n=0}^{N} s_n, e_N = \sum_{n=0}^{N} e_n, i_N = \sum_{n=0}^{N} i_n, r_N = \sum_{n=0}^{N} r_n, \tag{36}$$

where,

$$s(t) = \lim_{N\to\infty} (s_N), e(t) = \lim_{N\to\infty} (e_N), i(t) = \lim_{N\to\infty} (i_N), r(t) = \lim_{N\to\infty} (r_N). \tag{37}$$

FOURTH ORDER RUNGE-KUTTA INTEGRATION METHOD

The pure numerical classical fourth order Runge-Kutta method was used to compute the approximate solution of the model system (3). The method is reliable, robust and attains a higher level of accuracy and efficiency if a small step-size, *h* is used [5, 12]. The governing model system (3) of differential equations are of the form:

$$\frac{ds}{dt} = f(t,s,e,i), \frac{de}{dt} = f(t,s,e,i), \frac{di}{dt} = f(t,e,i) \text{ and } \frac{dr}{dt} = f(t,i,r),$$

subject to $s(0) = s_0, e(0) = e_0, i(0) = i_0$ and $r(0) = r_0$.

We let $h = t_{n+1} - t_n, n = 0,1,2,...$ so that the Taylor series [12] of $y(t_{n+1}) = y_{n+1}$ about y_n is given by,

$$y_{n+1} = y_n + hf(t_n, y_n) + \frac{1}{2!}h^2 f^{'}(t_n, y_n) + ... \tag{38}$$

Then, the RK4 integration method [5, 12] for the equation (38) yields,

$$y_{n+1} = y_n + \frac{h}{6}(k_1 + 2k_2 + 2k_3 + k_4), \tag{39}$$

where,

$$k_1 = f(t_n, y_n),$$

$$k_2 = f(t_n + \frac{h}{2}, y_n + \frac{h}{2} * k_1),$$

$$k_3 = f(t_n + \frac{h}{2}, y_n + \frac{h}{2} * k_2),$$

$$k_1 = f(t_n + h, y_n + h * k_3),$$

and y_{n+1} represents the model variables $s_{n+1}, e_{n+1}, i_{n+1}$ and r_{n+1} about s_n, e_n, i_n and r_n respectively.

COMPUTATIONAL RESULTS AND DISCUSSION

In this section, we monitor the effect of vaccination coverage on the dynamics of a childhood disease as described by the SEIR model Equations of system (3). The estimated parameter values and initial conditions of variables used for computations and simulations are shown in Table 2.

Table 2: Effects of vaccination coverage on disease transmission $(\mathrm{P_c} = 0.3846)$.

Case	s_0	e_0	i_0	r_0	β_1	β_2	γ	α	π	δ	P	R_v	**Comments**
1	0.4	0.2	0.1	0.3	0.2	0.3	0.1	0.04	0.1	0.1	0.7	0.4875	LAS (E^0)
2	0.4	0.2	0.1	0.3	0.2	0.3	0.1	0.04	0.1	0.1	0.5	0.8125	LAS (E^0)
3	0.4	0.2	0.1	0.3	0.2	0.3	0.1	0.04	0.1	0.1	0.3	1.1375	Unstable (E^0)
4	0.4	0.2	0.1	0.3	0.2	0.3	0.1	0.04	0.1	0.1	0.1	1.4625	Unstable (E^0)

Table 3 below depicts case-1 and shows the comparison between ADM solution and fourth order Runge-Kutta numerical solution. The computational results for the case $P > P_c$ demonstrates that the ADM series solution only agreed with the fourth order Runge-Kutta solution for very small values of time, i.e. $t = 0.01, 0.05, 0.1, 0.2$ *and* 0.3. We note that the ADM series only converge to the exact solution as $t \to 0$ i.e. for very small values of time. Furthermore, as the values of time increases the Adomain solution diverge and does not agree with the RK4 numerical solution. We note that the fourth order Runge-Kutta solution converges to the Disease free equilibrium (see *Table 3* and *Figure 4*). It is noteworthy that unlike the ADM solution which fails to converge as time increases, the fourth order Runge-Kutta method gave results that are in agreement with the qualitative analysis findings. In order to improve the ADM series radius of convergence, several series summation and enhancement methods such as Padé Approximation technique [3] may be applied. By this manipulation and improvement, we can obtain a convergent ADM series solution.

Table 3: Computations showing comparison between ADM solution and 4th Order Runge-Kutta numerical solution for Case-1, $(P = 0.7,\ \mathrm{P_c} = 0.3846)$.

ADM Solution					RK4 numerical solution			
t	*s*	*e*	*i*	*r*	*s*	*e*	*i*	*r*
0.1	0.396380	0.202877	0.099658	0.305370	0.396396	0.198871	0.099638	0.305370
0.3	0.389262	0.208615	0.099083	0.315939	0.389403	0.196567	0.098906	0.315936
0.5	0.382300	0.214333	0.098645	0.326284	0.382687	0.194204	0.098162	0.326273
0.7	0.375491	0.220034	0.098340	0.336414	0.376240	0.191791	0.097405	0.336384
1.0	0.365552	0.228559	0.098114	0.351223	0.367050	0.188088	0.096244	0.351137
1.5	0.349689	0.242709	0.098310	0.374935	0.352944	0.181745	0.094242	0.374654
2.0	0.334653	0.256809	0.099151	0.397527	0.340236	0.175257	0.092157	0.396885

Figure 2 shows the existence and uniqueness result for i^* in the interval $0 < i^* < \min\{1, \frac{\pi}{\alpha}\}$. *It confirms that the model has a unique endemic equilibrium.Figure 3* clearly shows the existence of a unique stable equilibrium when $R_v < 1$, confirming that the governing model undergoes the phenomenon of forward bifurcation. The diagram exhibits a globally stable disease-free equilibrium when $R_v < 1$ and an unstable state if $R_v > 1$, while it is evident that a unique stable endemic equilibrium emerges from the bifurcation point $R_v = 1$ and increases rapidly when $R_v > 1$. It is clear that the disease-free state exists for all R_v while an endemic equilibrium only exists for $R_v > 1$.

Figure 4 Describes case-1 and demonstrates the impact of high vaccination coverage $(P > P_c)$ on the temporal dynamics of the population fractions with increasing time. The Susceptible group gradually increases by a small amount as time increases and asymptotically attains a steady state. The increase is due to the recruitment of susceptible children/new-born babies. The population fractions of the exposed and infective groups display a sharp decrease as time increases. The rapid decrease of exposed is due to low recruitment caused by high vaccination while the decrease in infective populations is due to treatment of infected children. The Recovered group displays a sharp increase as time increases, it reaches a peak and decreases slightly to a steady state. The steep rise is due to recruitment of more children/new-born babies into the recovered group through vaccination, in addition to the treated children. The slight decrease is due to losses by natural deaths. It is noteworthy that the Disease free equilibrium state is attained on application of high vaccination coverage $(P > P_c)$ and the entire population attains disease-free state asymptotically.

Figure 5 Displays Case 1 − 4 and shows that an increase in vaccination proportion results to a decrease in the number of susceptible children, $s(t)$. *Figure 6* Displays Case 1 − 4 and shows that an increase in the vaccination coverage led to increased recovered population proportion, $r(t)$. *Figure7*Displays Case 1 − 4 and shows that an increase in the vaccination coverage leads to a decrease in the exposed population, $e(t)$, with a sharp decrease observed when the vaccination coverage was high i.e. $P > P_c$. *Figure 8*Displays Case 1 − 4 and shows that an increase in the vaccination coverage led to a decrease in the infective population, $i(t)$. The *Figures 7* and *8*confirms that, if P is large enough then the disease will die out in the population.

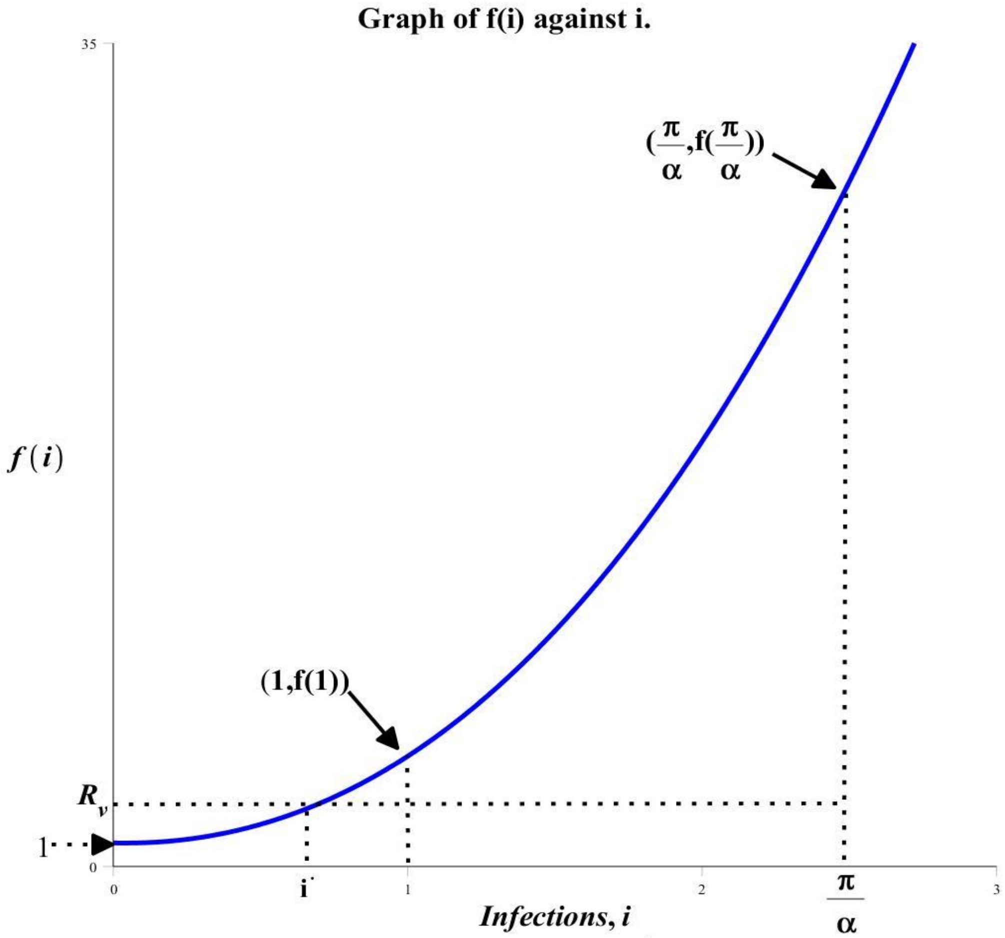

Figure 2: The existence and uniqueness of i^* in *the interval* $(0, 1)$.

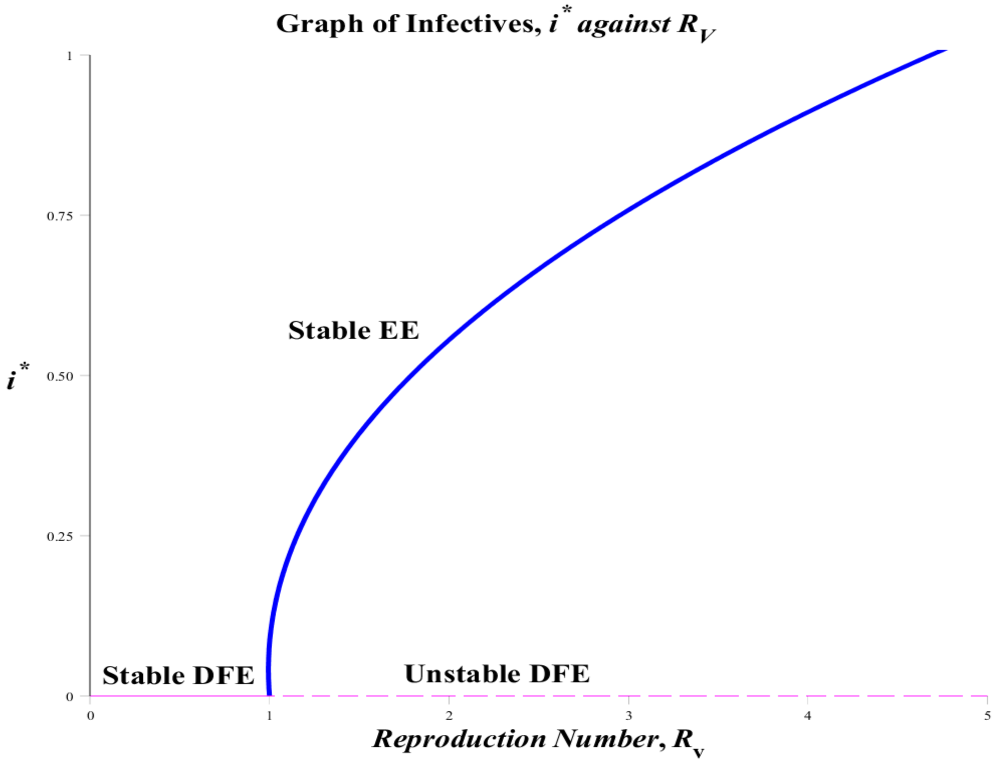

Figure 3: Forward bifurcation diagram for a SEIR childhood disease model.

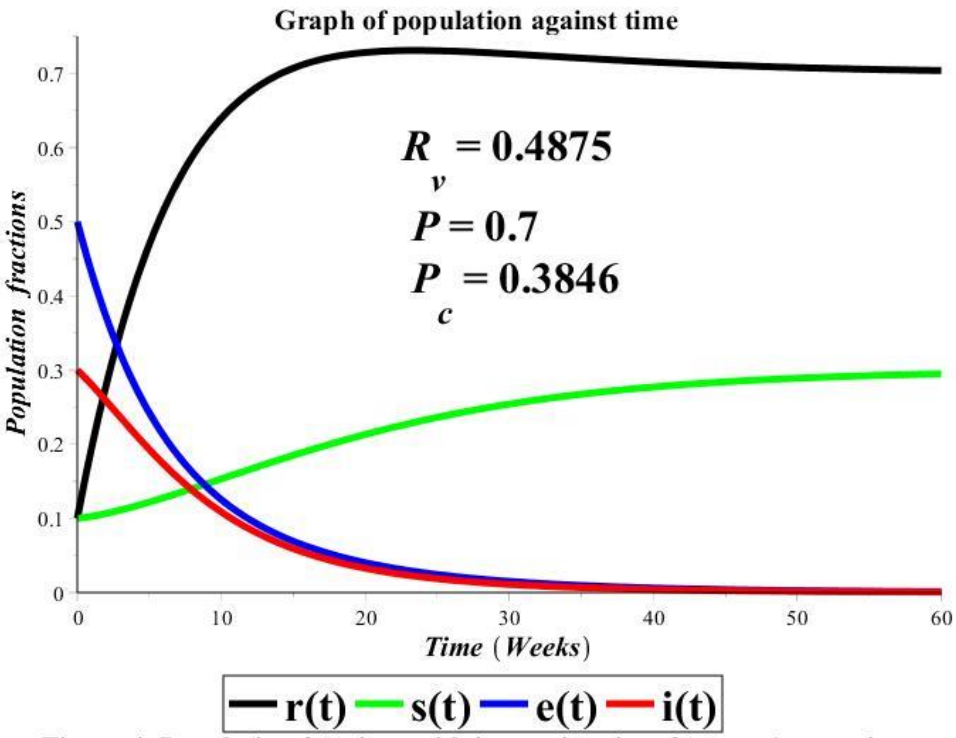

Figure 4: Population fractions with increasing time for case 1 scenario.

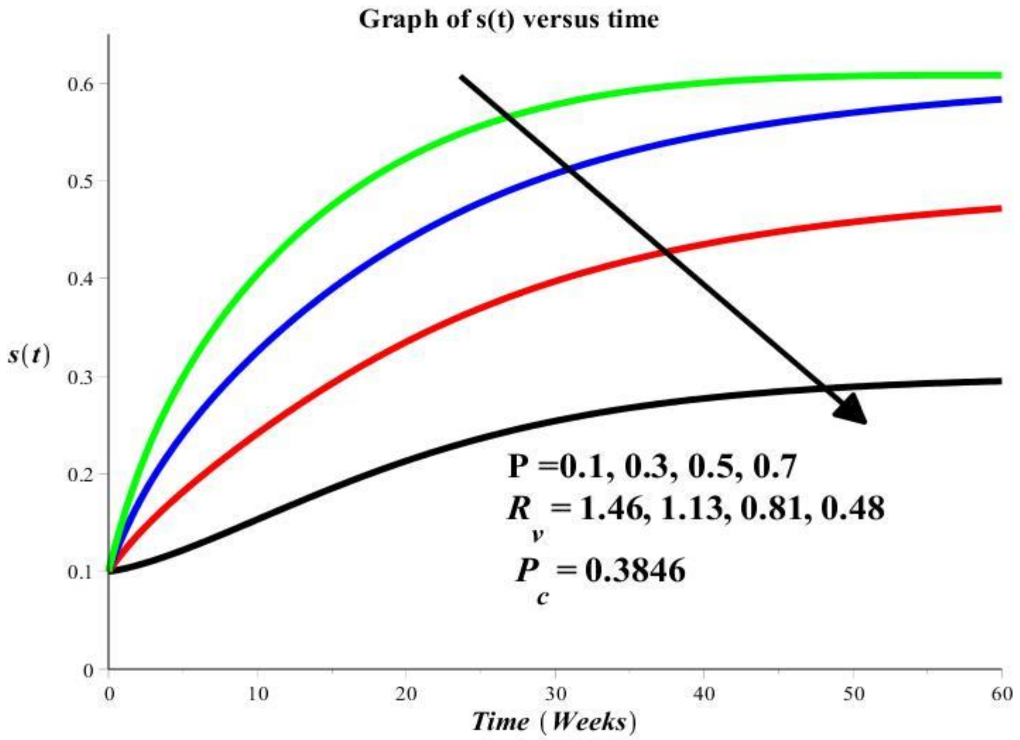

Figure 5: Effects of increasing population (P) on susceptible population.

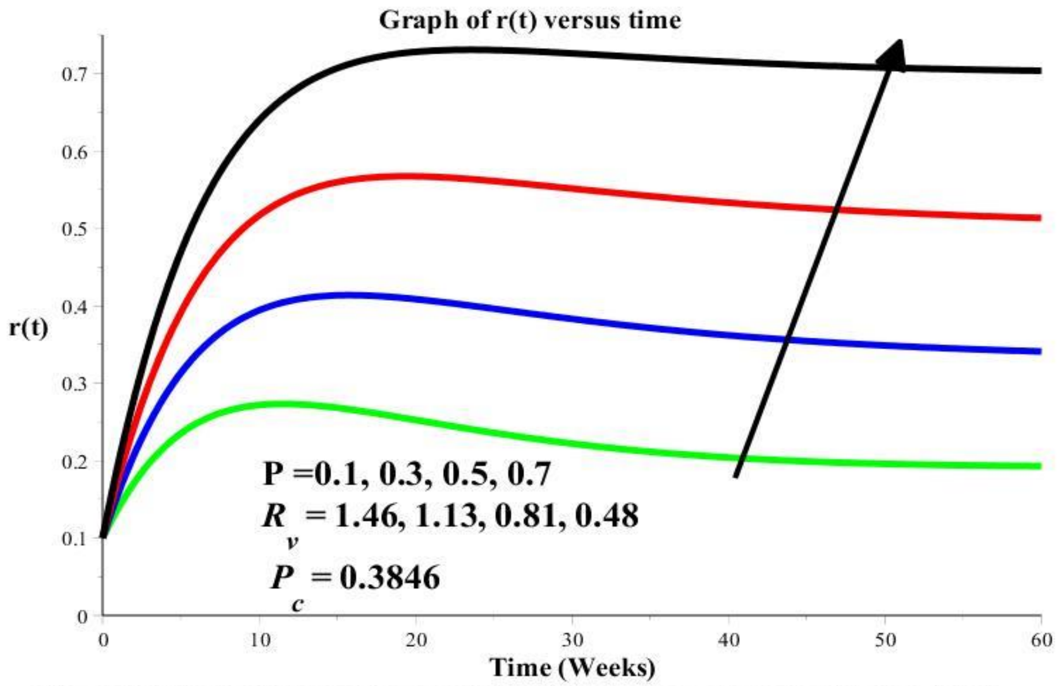

Figure 6: Effects of increasing population (P) on removed/vaccinated population.

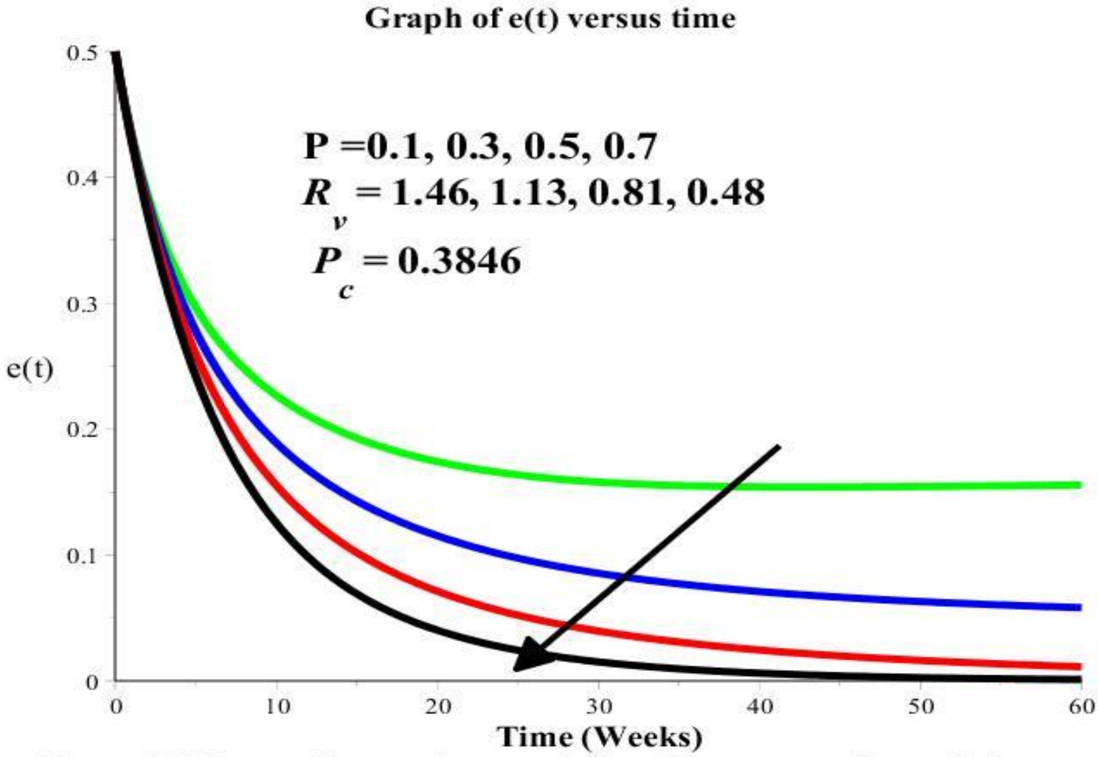

Figure 7: Effects of increasing population (P) on exposed population.

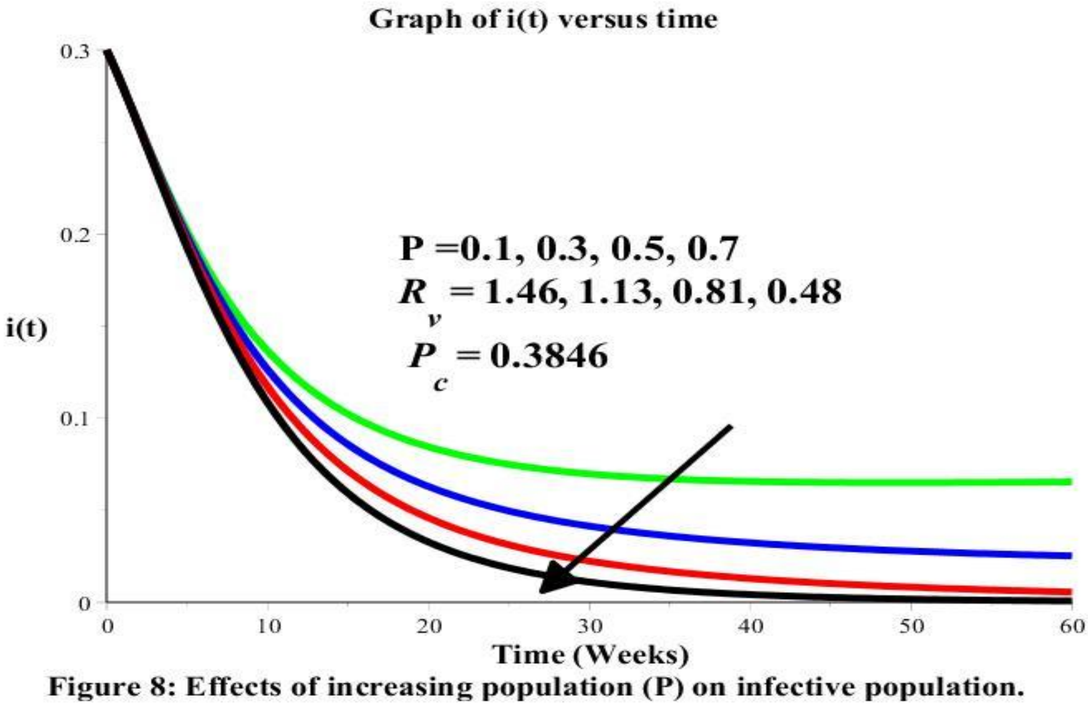

Figure 8: Effects of increasing population (P) on infective population.

CONCLUSION

In this paper, a SEIR deterministic model that monitors the temporal transmission dynamics of childhood diseases in the presence of preventive vaccine with varying population size was analysed. A qualitative and quantitative study of the SEIR model was done. The model incorporates the fact that exposed individuals are infectious to the community. The existence and uniqueness of the disease-free and endemic equilibrium were determined.

A qualitative analysis revealed that the disease-free equilibrium is both locally and globally asymptotically stable provided that the critical vaccination threshold value (P_c) is exceeded. A sensitivity analysis reveals that the proportion of susceptible that are vaccinated at birth and vaccination coverage have the highest impact on transmission dynamics of childhood diseases and death rate due to disease infection is the least sensitive parameter. Adomain decomposition method and fourth order Runge Kutta method are employed to compute the approximate solution of the model problem. The computations revealed that the ADM series only converge and Agree with RK4 for very small values of time and does not agree with RK4 as time increases. The fourth order Runge-Kutta scheme gave results that were in agreement with the qualitative analysis and converged for all time.

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APPENDIX A.

According to the Estimated parameter values in **Table 2**, the following ADM [4, 6, 7, 8] approximate series solutions were obtained:

Case 1:

$$s(t) = \frac{2}{5} - \frac{9}{2500}t + \frac{5141}{2500000}t^2 - \frac{419219}{3750000000}t^3 + \frac{65906323}{7500000000000}t^4 + O(t^5).$$

$$e(t) = \frac{1}{5} + \frac{18}{625}t - \frac{737}{2500000}t^2 + \frac{228073}{3750000000}t^3 - \frac{53761001}{7500000000000}t^4 + O(t^5).$$

$$i(t) = \frac{1}{10} - \frac{9}{2500}t + \frac{1161}{625000}t^2 - \frac{287453}{1875000000}t^3 + \frac{38544821}{3750000000000}t^4 + O(t^5).$$

$$r(t) = \frac{3}{10} + \frac{27}{500}t - \frac{369}{125000}t^2 + \frac{723}{3906250}t^3 - \frac{3747371}{375000000000}t^4 + O(t^5).$$

Case 2:

$$s(t) = \frac{2}{5} - \frac{41}{2500}t + \frac{991}{2500000}t^2 - \frac{265369}{3750000000}t^3 + \frac{50195873}{7500000000000}t^4 + O(t^5).$$

$$e(t) = \frac{1}{5} + \frac{18}{625}t + \frac{1013}{2500000}t^2 + \frac{273323}{3750000000}t^3 - \frac{41249251}{7500000000000}t^4 + O(t^5).$$

$$i(t) = \frac{1}{10} - \frac{9}{2500}t + \frac{1161}{625000}t^2 - \frac{243703}{1875000000}t^3 + \frac{34601071}{3750000000000}t^4 + O(t^5).$$

$$r(t) = \frac{3}{10} + \frac{17}{500}t - \frac{61}{31250}t^2 + \frac{14227}{9370000}t^3 - \frac{3128621}{375000000000}t^4 + O(t^5).$$

Case 3:

$$s(t) = \frac{2}{5} + \frac{9}{2500}t - \frac{3159}{2500000}t^2 - \frac{37173}{1250000000}t^3 + \frac{7995141}{2500000000000}t^4 + O(t^5).$$

$$e(t) = \frac{1}{5} + \frac{18}{625}t + \frac{2763}{2500000}t^2 + \frac{106191}{1250000000}t^3 - \frac{6079167}{2500000000000}t^4 + O(t^5).$$

$$i(t) = \frac{1}{10} - \frac{9}{2500}t + \frac{1161}{625000}t^2 - \frac{66651}{625000000}t^3 + \frac{10219107}{1250000000000}t^4 + O(t^5).$$

$$r(t) = \frac{3}{10} + \frac{7}{500}t - \frac{119}{125000}t^2 + \frac{5551}{46875000}t^3 - \frac{2509871}{375000000000}t^4 + O(t^5).$$

Case 4:

$$s(t) = \frac{2}{5} + \frac{59}{2500}t - \frac{7309}{2500000}t^2 + \frac{42331}{3750000000}t^3 - \frac{12725027}{7500000000000}t^4 + O(t^5).$$

$$e(t) = \frac{1}{5} + \frac{18}{625}t + \frac{4513}{2500000}t^2 + \frac{363823}{3750000000}t^3 + \frac{15274249}{7500000000000}t^4 + O(t^5).$$

$$i(t) = \frac{1}{10} - \frac{9}{2500}t + \frac{1161}{625000}t^2 - \frac{156203}{1875000000}t^3 + \frac{26713571}{3750000000000}t^4 + O(t^5).$$

$$r(t) = \frac{3}{10} - \frac{3}{500}t + \frac{3}{62500}t^2 + \frac{2659}{31250000}t^3 - \frac{1891121}{375000000000}t^4 + O(t^5).$$

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