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Mathematical Modeling of Polio Virus Infection Incorporating Immigration and Vaccination

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Abstract

A deterministic mathematical model for polio infection dynamics with emphasis on immigration and vaccination was formulated and analyzed. We derived the basic reproduction number, R_0 of the model formulated. The effective reproduction number was computed using the next generation matrix to enable a qualitative analysis to be carried out on the model. Also, the disease-free equilibrium and endemic equilibrium points were computed. On analyzing the equilibrium points, we found that the disease-free equilibrium point is locally asymptotically stable if $R_0 < 1$ and the condition for existence on an Endemic Equilibrium point was also established. More so, numerical simulations showed that vaccination coverage of about 75% would be enough to eradicate polio from the population.

Keyword: Modeling, Polio, Vaccination, Equilibrium, Stability, Immigration.

1. Introduction

Polio is an infectious disease caused by poliovirus. There is always muscle weakness which could lead to inability to move in about 0.5% of cases. This can happen over a couple of hours to a couple of days. The weakness could include the legs as well as the muscles of the head, neck and diaphragm. Many but not all individuals infected can recover completely from this infection. In those with muscle weakness, about 2% to 5% of children and 15% to 30% of adults die. 25% of people have minor symptoms such as fever and a sore throat and up to 5% have headache, neck stiffness and pains in the arms and legs. These individuals get back to normal after a couple of weeks (Hamborsky *et al*., 2015). There may be no symptoms in up to 70% of infections. Post-polio syndrome may occur years after recovery with a moderate advancement of muscle weakness like what the individual had at the initial age of the illness

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(Post-Polio Syndrome Fact Sheet, 2014). It can cause lifelong paralysis (can't move parts of the body), and it can be deadly (CDC, 2014). Up to 95% of individuals leaving with polio virus have no symptoms. However, polio can be spread by individuals leaving with polio virus without symptoms and then cause others to develop polio infection (CDC, 2014).

Over the years, different authors have used different approaches to model the disease. For instance; a simple SIR epidemic model was developed by (Shulgin *et al.,* 1998) with pulse vaccination. The result shows that pulse vaccination could lead to total eradication of the epidemic if certain conditions like the period of pulses and the magnitude of vaccination are fulfilled. A simple two-dimensional SIS model with vaccination was developed and analyzed by (Kribs-Zaleta & Velasco- Hernandez, 2000). The result of their model resulted into backward bifurcation. (Farrington, 2003) developed and analyzed a mathematical model of polio virus. Their model shows a positive result of vaccination on the transmission dynamics of polio. (Gumel & Moghadas, 2003) also proposed a mathematical model of polio virus. They were able to obtain the optimal vaccine coverage needed for the total eradication of the infection. (Manju & Archana 2010) analyzed an epidemic model on Polio with vaccination to determine the impact of vaccination when it is administered in susceptible and exposed population.

(Okuonghae *et al.*, 2015) built a deterministic model for the transmission dynamics of two strains of polio, the vaccine-derived polio virus (VDPV) and the wild polio virus (WPV) in a population. It was observed that Oral Polio Vaccine (OPV) reversion (leading to increased incidences of WPV and VDPV strains), together with the joint impact of giving vaccine to certain unvaccinated susceptible individuals and children who are susceptible and missed up, could lead to backward bifurcation if the effective reproduction number is less than one. In the absence of OPV reversions (leading to the co-existence of both strains in the population), it was noticed that the disease-free equilibrium of the model is globally-asymptotically stable if the effective reproduction number is less than one. The result of the numerical simulations shows that the model developed experiences the phenomenon of competitive exclusion, where the strain with the largest effective reproduction number gives room for the other one to go into extinction.

In this study, we formulated a deterministic mathematical model for transmission dynamics of polio virus. The model developed considered the total population to be non-constant and non-aged structured. It should be clearly known that we are interested in investigating the effect(s)

of vaccination and immigration on the transmission dynamics of polio virus in a non-constant population.

2. Model Formulation

In this section, we formulate a deterministic mathematical model for polio virus which incorporates immigration and vaccination strategy. The total population N (t) is divided into four compartments namely: Susceptible S(t), Vaccinated V(t), Exposed E(t), Infectious and paralyzed individuals I(t). In this model, individuals are recruited into the population either by immigration at the rate Λ or per capital birth rate π. We assume that proportions ρ of newborns in the population and θ of the immigrants were vaccinated at birth or at one point in their life to protect them against infection. A proportion θ of the recruits are vaccinated, the remaining 1-θ are not vaccinated so the join the susceptible compartment. A proportion ρ of the newborns are vaccinated, the remaining 1-ρ are not vaccinated so they join the susceptible compartment.

We assume that the population of the susceptible will receive a vaccine at the rate γ to have a permanent immunity. Furthermore, we assume that the natural death rate μ is constant, the disease induced death rate is μ_I, the members of the population mix homogeneously, β probability that a susceptible individual becomes infected by one infectious individual. Susceptible individuals enter the exposed class at a rate λ which is the force of infection. The exposed individuals are those ones who just got the infection (asymptomatic) but they can still infect others. After some time, the exposed now move from the exposed class into the infectious class at the rate α. According to the nature of the disease, most infected individual cannot recover from the infection (WHO, 2018) hence, we would not consider recovered class.

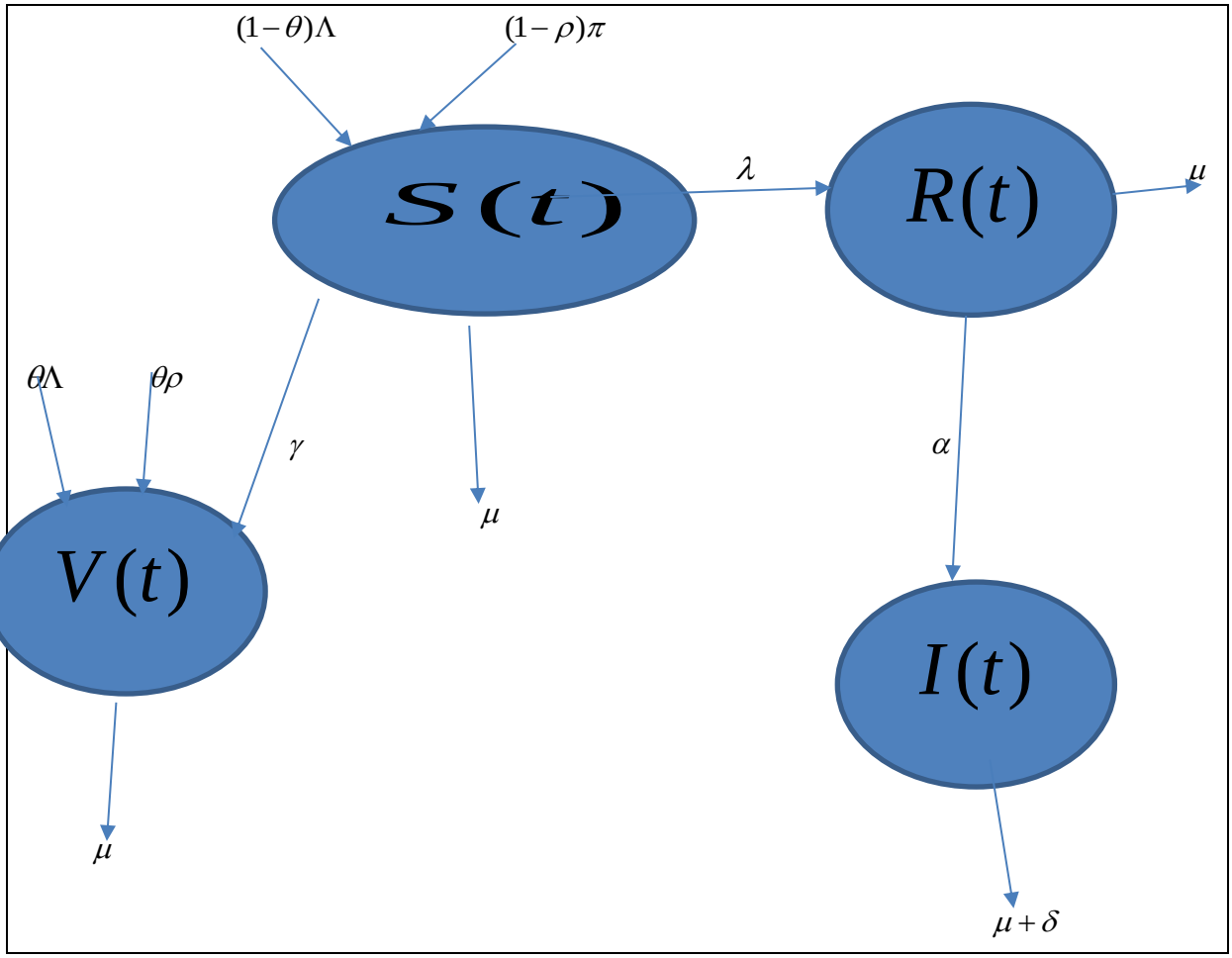

Figure 1: The flow diagram.

Table 1: State variables and model parameters.

Parameters and State Variables	Description
S	Susceptible individuals
E	Exposed individuals
I	Infectious individuals and Paralyzed individuals
V	Vaccinated individuals
N	Total population
β	Probability of an infected individuals to infect others
c	Per capital contact rate
μ	Natural death rate

Λ	Immigration
π	Per capital birth rate
γ	Rate at which susceptible individuals move to vaccinated class
λ	Force of infection
ρ	proportion of the newborn that are vaccinated
θ	proportion of the recruits that are vaccinated
α	Rate at which exposed individuals move to Infectious class
$(1-\rho)$	proportion of the newborn that are not vaccinated
$(1-\theta)$	proportion of the recruits that are not vaccinated
δ	Disease induced death rate

2.1 The Model Equations

From the assumptions and the dynamics between the compartments shown in the model compartments in figure 1, the effect of immunization on the epidemiology of polio virus is modeled by the following system of ordinary differential equations;

$$\left.\begin{aligned}
\frac{dS}{dt} &= (1-\rho)\pi + (1-\theta)\Lambda - (\lambda+\gamma+\mu)S \\
\frac{dE}{dt} &= \lambda S - (\alpha+\mu)E \\
\frac{dI}{dt} &= \alpha E - (\mu+\delta)I \\
\frac{dV}{dt} &= \rho\pi + \theta\Lambda + \gamma S - \mu V
\end{aligned}\right\}, \tag{1}$$

where λ is the force of infection; $\lambda = \dfrac{\beta c(E+I)}{N}$

3. Model Analysis

We provide comprehensive qualitative analysis of the model equation in this section.

3.1 The Positive Invariant Region

N=S+E+I+V, (2)

$$\frac{dN}{dt}=\frac{dS}{dt}+\frac{dE}{dt}+\frac{dI}{dt}+\frac{dV}{dt}, \tag{3}$$

$$\frac{dN}{dt}=(1-\rho)\pi+(1-\theta)\Lambda+\theta\Lambda+\rho\pi-\mu S-\mu E-\mu I-\mu V-\delta I, \tag{4}$$

The positive invariant region can be obtained by using the following theorem as applied by (Bolarin & Omatola, 2016).

Theorem 1:

The solutions of the system (1) are feasible for $t>0$ if they enter the invariant region D.

Proof:

Let $D=\left(S,E,I,V\right)\in \mathrm{R}^{4}_{+}$ be any solution of the system (1) with non-zero initial conditions.

Assuming there are no disease-induced deaths, equation (4) now becomes;

$$\frac{dN}{dt}\le(1-\rho)\pi+(1-\theta)\Lambda+\theta\Lambda+\rho\pi-\mu N, \tag{5}$$

$$\frac{dN}{dt}+\mu N\le(1-\rho)\pi+(1-\theta)\Lambda+\theta\Lambda+\rho\pi, \tag{6}$$

$$N(t)=\frac{(1-\rho)\pi+(1-\theta)\Lambda+\theta\Lambda+\rho\pi}{\mu}+Ce^{-\mu t}. \tag{7}$$

Applying the initial condition $t=0;\ N(0)=N_0$:

$$N_0\le\frac{(1-\rho)\pi+(1-\theta)\Lambda+\theta\Lambda+\rho\pi}{\mu}+C, \tag{8}$$

$$N_0-\frac{(1-\rho)\pi+(1-\theta)\Lambda+\theta\Lambda+\rho\pi}{\mu}\le C, \tag{9}$$

$$\Rightarrow \quad N(t) \le \frac{(1-\rho)\pi + (1-\theta)\Lambda + \theta\Lambda + \rho\pi}{\mu} + \left(N_0 - \frac{(1-\rho)\pi + (1-\theta)\Lambda + \theta\Lambda + \rho\pi}{\mu} \right) e^{-\mu t}. \tag{10}$$

Therefore, as $t \to \infty$ in equation (10) the human population N approaches $K = \frac{(1-\rho)\pi + (1-\theta)\Lambda + \theta\Lambda + \rho\pi}{\mu}$, that is, $N \to K = \frac{(1-\rho)\pi + (1-\theta)\Lambda + \theta\Lambda + \rho\pi}{\mu}$ the parameter $K = \frac{(1-\rho)\pi + (1-\theta)\Lambda + \theta\Lambda + \rho\pi}{\mu}$ is called the carrying capacity. Hence all feasible solution set of the model (1) enter the region

$$D = \left\{ (S, E, I, V) \in R^4 : S > 0, E \ge 0, I \ge 0, V \ge 0, N \le \frac{(1-\rho)\pi + (1-\theta)\Lambda + \theta\Lambda + \rho\pi}{\mu} \right\}.$$

Therefore, the region D is positively-invariant and system (1) is epidemiologically meaningful and mathematically well-posed in the domain D.

3.2 Positivity of the Solutions

Theorem 2:

Let the initial data be $\{S(0) > 0, (E(0), I(0), V \ge 0)\} \in D$.

Then the solution set $\{S, E, I, V\}(t)$ of the system of equations (1) to (4) is positive for all $t > 0$

Proof:

From the first equation of (1), we have:

$$\frac{dS}{dt} = (1-\rho)\pi + (1-\theta)\Lambda - (\lambda + \gamma + \mu)S \ge -(\lambda + \gamma + \mu)S, \tag{11}$$

$$\frac{dS}{dt} \ge -(\lambda + \gamma + \mu)S, \tag{12}$$

$$S(t) \ge Ke^{-(\lambda+\gamma+\mu)t}, \tag{13}$$

where K= e^C , using the initial condition $t = 0 \Rightarrow S(0) \ge K$.

Therefore

$$S(t) \ge S(0)e^{-(\lambda + \gamma + \mu)t} \ge 0, \tag{14}$$

from the equation (2), we have:

$$\frac{dE}{dS} = \lambda S - (\alpha + \mu)E \geq -(\alpha + \mu)E\,, \tag{15}$$

$$\frac{dE}{dS} \geq -(\alpha + \mu)E\ , \tag{16}$$

$$E(t) \geq Ke^{-(\alpha+\mu)t}\,. \tag{17}$$

Applying the initial condition $t = 0 \Rightarrow E(0) \geq K$ to have:

$$E(t) \geq E(0)e^{-(\alpha+\mu)t}\,. \tag{18}$$

Similarly, it can be verified that the rest of the equations are positive for all $t > 0$, since $e^{\omega} > 0$ $\forall \omega \in \Re$.

3.3 Disease Free Equilibrium State

The disease-free equilibrium of the model (1) is obtained by setting

$$\frac{dS}{dt} = \frac{dE}{dt} = \frac{dE}{dt} = \frac{dV}{dt} = 0\,. \tag{19}$$

In this case there is no disease: E = I = 0. Hence, the DFE of our equation is given by:

$$\begin{pmatrix} S^* \\ E^* \\ I^* \\ V^* \end{pmatrix} = \begin{pmatrix} \dfrac{(1-\rho)\pi + (1-\theta)\Lambda}{(\lambda + \gamma + \mu)} \\ 0 \\ 0 \\ \dfrac{\rho\pi + \theta\Lambda}{\mu} + \gamma\left(\dfrac{(1-\rho)\pi + (1-\theta)\Lambda}{\mu(\gamma + \mu)}\right) \end{pmatrix} \tag{20}$$

3.4 Basic Reproduction Number, R_0

Poletti *et al.* (2013) defined R_0 as the average number of secondary infections produced by individuals that are infectious during his or her entire period of infectiousness. R_0 determines if a disease will persist or will die out in a community. If $R_0 < 1$ it indicates that infectious individual will cause less than one secondary infection and hence the disease will not remain, then when $R_0 > 0$ the disease will take over the population. In a more complicated epidemic, the

R_0 can be calculated by using the next generation operator approach by (van den Driessche & Watmough, 2002).

From the system (1) we define f_i *and* v_i as:

$$f_i = \begin{bmatrix} \frac{\beta c(E+I)S}{N} \\ 0 \end{bmatrix} \quad and \quad v_i = \begin{bmatrix} (\alpha+\mu)E \\ \alpha E - (\mu+\delta)I \end{bmatrix}, \tag{21}$$

$$FV^{-1} = \begin{bmatrix} \frac{\partial f_i(E_0)}{\partial x_j} \end{bmatrix} \begin{bmatrix} \frac{\partial v_i(E_0)}{\partial x_j} \end{bmatrix}^{-1}$$
$$= \begin{bmatrix} \frac{\beta c S^*}{N} & \frac{\beta c S^*}{N} \\ 0 & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{(\alpha+\mu)} & 0 \\ \frac{\alpha}{(\mu+\delta)(\alpha+\mu)} & \frac{1}{(\mu+\delta)} \end{bmatrix} = \begin{bmatrix} \frac{\beta c S^*}{N(\alpha+\mu)} + \frac{\alpha\beta c S^*}{N(\mu+\delta)(\alpha+\mu)} & \frac{\beta c S^*}{N(\mu+\delta)} \\ 0 & 0 \end{bmatrix} \tag{22}$$

Therefore, the basic reproduction number $R_0 = \rho\left(FV^{-1}\right) =$ spectra radius of FV^{-1} and hence

$$R_0 = \frac{\beta c\left[(1-\rho)\pi + (1-\theta)\Lambda\right]}{N(\alpha+\mu)(\gamma+\mu+\lambda)} + \frac{\alpha\beta c\left[(1-\rho)\pi + (1-\theta)\Lambda\right]}{N(\mu+\delta)(\alpha+\mu)(\gamma+\mu)} \tag{23}$$

3.5 Local Stability Analysis of Disease Free Equilibrium State.

Theorem 3: The disease-free equilibrium, E^* of (23) is locally asymptotically stable (LAS) in D if $R_0 < 1$.

Proof: We shall use Jacobean stability technique to carry out the local stability analysis of the disease disease-free equilibrium.

Jacobean matrix of the system of equations at disease-free equilibrium is:

$$J(\frac{(1-\rho)\pi+(1-\theta)\Lambda}{(\lambda+\gamma+\mu)}\ ,\ 0\ 0\ \frac{\rho\pi+\theta\Lambda}{\mu}+\gamma\left(\frac{(1-\rho)\pi+(1-\theta)\Lambda}{\mu(\gamma+\mu)}\right))=$$

$$\begin{bmatrix} -(\gamma+\mu) & -\frac{\beta c(1-\rho)\pi+(1-\theta)\Lambda}{N(\lambda+\gamma+\mu)} & -\frac{\beta c(1-\rho)\pi+(1-\theta)\Lambda}{N(\lambda+\gamma+\mu)} & 0 \\ 0 & \frac{\beta c(1-\rho)\pi+(1-\theta)\Lambda}{N(\lambda+\gamma+\mu)}-(\alpha+\mu) & \frac{\beta c(1-\rho)\pi+(1-\theta)\Lambda}{N(\lambda+\gamma+\mu)} & 0 \\ 0 & \alpha & -\mu-\delta & 0 \\ \gamma & 0 & 0 & -\mu \end{bmatrix} \tag{24}$$

Determinant gives

$$\mu^2(\gamma+\mu)(\alpha+\mu)\left(1-\left(\frac{\beta c\left[(1-\rho)\pi+(1-\theta)\Lambda\right]}{N(\alpha+\mu)(\gamma+\mu)}+\frac{\alpha\beta c\left[(1-\rho)\pi+(1-\theta)\Lambda\right]}{N(\mu+\delta)(\alpha+\mu)(\gamma+\mu)}\right)\right), \tag{25}$$

but $R_0=\frac{\beta c\left[(1-\rho)\pi+(1-\theta)\Lambda\right]}{N(\alpha+\mu)(\gamma+\mu)}+\frac{\alpha\beta c\left[(1-\rho)\pi+(1-\theta)\Lambda\right]}{N(\mu+\delta)(\alpha+\mu)(\gamma+\mu)}$. (26)

Hence, DFE is Locally Asymptotically Stable (LAS) if $R_0<1$. The epidemiology implication of the theorem is that polio can be eliminated (control) from the population when $R_0<1$, if the initial size of the sub-populations are in the basin of attraction of the DFE.

3.6 Global Stability of Disease Free Equilibrium (E^*)

Lemma 1: (Castillo-Chavez *et al.*, 2002)**:** Let $x\to f(x)\in\Re^n$ be a C^1 function for x in an open set $D\subset\Re^n$. Consider the differential equation

$$x^{'}=f(x). \tag{27}$$

Denote by $x(t,x_0)$ the solution to (27) such that $x(0,x_0)=x_0$. A set K is said to be *absorbing* in D for (27) if $x(t,K_1)\subset K$ for each compact $K_1\subset D$ and t sufficiently large. We make the following two basic assumptions:

(H_1) There exists a compact absorbing set $K\subset D$

(H_2) Equation (27) has a unique equilibrium $\bar{x}$ in D

The equilibrium $\bar{x}$ is said to be globally stable in D if it is locally stable and all trajectories in D converges to $\bar{x}$.

Note: (H_1) is equivalent to uniform persistence of equation (27) (see Butler & Waltman, 1986; Waltman, 1991).

Theorem 4: The disease-free equilibrium E^* is Globally Asymptotically Stable (GAS) in D if $R_0 < 1$.

Proof:

To establish this, we re-write the model equation as follows:

$$\left.\begin{aligned} \frac{dS}{dt} &= (1-\rho)\pi + (1-\theta)\Lambda - (\lambda+\gamma+\mu)S \\ \frac{dE}{dt} &= \lambda S - (\alpha+\mu)E \\ \frac{dI}{dt} &= \alpha E - (\mu+\delta)I \end{aligned}\right\}, \tag{28}$$

with

$$\frac{dN}{dt} = \Omega - \mu N - \gamma S - \delta I \tag{29}$$

where

$$\Omega = (1-\rho)\pi + (1-\theta)\Lambda . \tag{30}$$

The reproduction number of the reduced system is given as:

$$R_0 = \frac{\lambda\alpha\Omega}{(\alpha+\mu)(\mu+\delta)} . \tag{31}$$

Now, consider a Lyapunov function $L = \alpha E + (\alpha+\mu)I$, the time derivative is given by

$$L' = \alpha\{\lambda S - (\alpha+\mu)E\} + (\alpha+\mu)\{\alpha E - (\mu+\delta)I\} . \tag{32}$$

After carrying out algebraic manipulations, we have

$$L' = \frac{\alpha\lambda\Omega}{\mu}\left(\frac{\mu S}{\Omega} - \frac{I}{R_0}\right), \tag{33}$$

recall that $\lambda = \frac{\beta c(E+I)}{N}$, then equation (33) becomes:

$$L^{'} = \frac{\alpha\beta cE\mu S}{N\Omega} + \frac{\alpha\beta cI\mu S}{N\Omega} - \frac{I}{R_0}$$
$$\leq \left(\frac{\alpha\beta c\mu S}{N\Omega} - \frac{1}{R_0} \right) I \leq 0$$

Noting that $S \leq N$ for all time t, $L^{'} < 0$ if and only if $R_0 < 1$, and $L^{'} = 0$ if and only if $I = 0$. Therefore, L is a Lyapunov function for the system equation (28). Thus, it follows by the LaSalle's Invariant Principle [cite], that the DFE of the model (1) is GAS whenever $R_0 < 1$ otherwise by Lemma 1, model (1) has an Endemic Equilibrium whenever $R_0 \geq 1$ because $L^{'} > 0$ for S sufficiently close to the invariant region except when $E = I = 0$.

3.7 Existence of Endemic Equilibrium Point in Terms of force of Infection

$E^{'} = (S, E, I, V) = (S^{**}, E^{**}, I^{**}, V^{**})$ is the endemic equilibrium point.

$$(1-\rho)\pi + (1-\theta)\Lambda^{**} - \frac{\beta cE^{**}S^{**}}{N^{**}} - \frac{\beta cI^{**}S^{**}}{N^{**}} - (\gamma + \mu)S^{**} = 0, \tag{34}$$

$$\frac{\beta cE^{**}S^{**}}{N^{**}} + \frac{\beta cI^{**}S^{**}}{N^{**}} - (\alpha + \mu)E^{**} = 0, \tag{35}$$

$$\alpha E^{**} - (\mu + \delta)I^{**} = 0, \tag{36}$$

$$\rho\pi + \theta\Lambda + \gamma S^{**} - \mu V^{**} = 0, \tag{37}$$

$\Rightarrow$

$$(1-\rho)\pi + (1-\theta)\Lambda - \lambda S^{**} - (\gamma + \mu)S^{**} = 0, \tag{38}$$

$$\lambda S^{**} - (\alpha + \mu)E^{**} = 0, \tag{39}$$

$$\alpha E^{**} - \mu I^{**} = 0, \tag{40}$$

$$\rho\pi + \theta\Lambda + \gamma S^{**} - \mu V^{**} = 0, \tag{41}$$

where $\lambda^{**} = \frac{\beta c(E^{**} + I^{**})}{N^{**}}$ (42)

$$S^{**} = \frac{(1-\rho)\pi + (1-\theta)\Lambda}{(\lambda^{**} + \gamma + \mu)}. \tag{43}$$

Putting equation (43) in equation (41) we have

$$V^{**} = \frac{\rho\pi + \theta\Lambda}{\mu} + \frac{\gamma(1-\rho)\pi + (1-\theta)\gamma\Lambda}{\mu(\lambda^{**} + \gamma + \mu)}, \tag{44}$$

putting equation (43) into equation (39) we have

$$E^{**} = \frac{\lambda^{**}[(1-\rho)\pi + (1-\theta)\Lambda]}{(\lambda^{**} + \gamma + \mu)(\alpha + \mu)}, \tag{45}$$

and putting equation (45) into equation (40) we have

$$I^{**} = \frac{\alpha\lambda^{**}[(1-\rho)\pi + (1-\theta)\Lambda]}{\mu(\lambda^{**} + \gamma + \mu)(\alpha + \mu)}. \tag{46}$$

Hence, the endemic equilibrium points of our model equation in terms of forces of infection are given as;

$$\begin{bmatrix} S^{**} \\ E^{**} \\ I^{**} \\ V^{**} \end{bmatrix} = \begin{bmatrix} \frac{(1-\rho)\pi + (1-\theta)\Lambda}{(\lambda^{**} + \gamma + \mu)} \\ \frac{\lambda^{**}[(1-\rho)\pi + (1-\theta)\Lambda]}{(\lambda^{**} + \gamma + \mu)(\alpha + \mu)} \\ \frac{\alpha\lambda^{**}[(1-\rho)\pi + (1-\theta)\Lambda]}{(\mu + \delta)(\lambda^{**} + \gamma + \mu)(\alpha + \mu)} \\ \frac{\rho\pi + \theta\Lambda}{\mu} + \frac{\gamma(1-\rho)\pi + (1-\theta)\gamma\Lambda}{\mu(\lambda^{**} + \gamma + \mu)} \end{bmatrix}. \tag{47}$$

Putting equations (9) and (10) into equation (6) we have:

$$\lambda^{**} = \frac{\beta c}{N^{**}}\left[\frac{\lambda^{**}[(1-\rho)\pi + (1-\theta)\Lambda]}{(\lambda^{**} + \gamma + \mu)(\alpha + \mu)} + \frac{\alpha\lambda^{**}[(1-\rho)\pi + (1-\theta)\Lambda]}{\mu(\lambda^{**} + \gamma + \mu)(\alpha + \mu)}\right], \tag{48}$$

$$\lambda^{**}\left[N^{**}(\alpha + \mu)\mu\right] + N^{**}(\alpha + \mu)(\gamma + \mu) = \mu\beta c[(1-\rho)\pi + (1-\theta)\Lambda] + \beta c\alpha[(1-\rho)\pi + (1-\theta)\Lambda] \tag{49}$$

$$A\lambda^{**} + B\ , \tag{50}$$

where

$$\mathrm{A} = N^{**}(\alpha + \mu)\mu\ ,$$

$$\mathrm{B} = N^{**}(\alpha + \mu)(\gamma + \mu)\mu - \mu\beta c[(1-\rho)\pi + (1-\theta)\Lambda] - \alpha\beta c[(1-\rho)\pi + (1-\theta)\Lambda],$$

$$\mathrm{B} = N^{**}(\alpha + \mu)(\gamma + \mu)\mu\left[1 - \left(\frac{\beta c[(1-\rho)\pi + (1-\theta)\Lambda]}{N^{**}(\alpha + \mu)(\gamma + \mu)} + \frac{\alpha\beta c[(1-\rho)\pi + (1-\theta)\Lambda]}{N^{**}(\alpha + \mu)(\gamma + \mu)}\right)\right],$$

$$B = N^{**}(\alpha+\mu)(\gamma+\mu)\mu\left[1-R_0\right].$$

4. Numerical Simulation

We used maple software to plot the graph of our model equations. Since, most of the parameters were not readily available; we assumed some and obtain the rest from the papers we reviewed just for illustration. The total number of the population of sample considered is 1,460. For us to be able to investigate the effects of vaccine on recruits or newborn which are vaccinated or susceptible, graphical representations showing the time graphs of different state variables are provided.

Table 2: Initial conditions for the state variables and parameters values.

Parameters and State Variables	Value	Source
S	500	Manju & Archana (2011)
E	200	Manju & Archana (2011)
I	160	Manju & Archana (2011)
V	600	Manju & Archana (2011)
N	1,460	Calculated
Λ	1000	Assumed
π	1000	Assumed
θ	0.25	Assumed
μ	0.5	Manju & Archana (2011)
γ	0.25	Assumed
μ_I	0.6	Manju & Archana (2011)
β	0.5	Assumed
ρ	0.25	Assumed
α	0.25	Assumed
C	0.5	Assumed

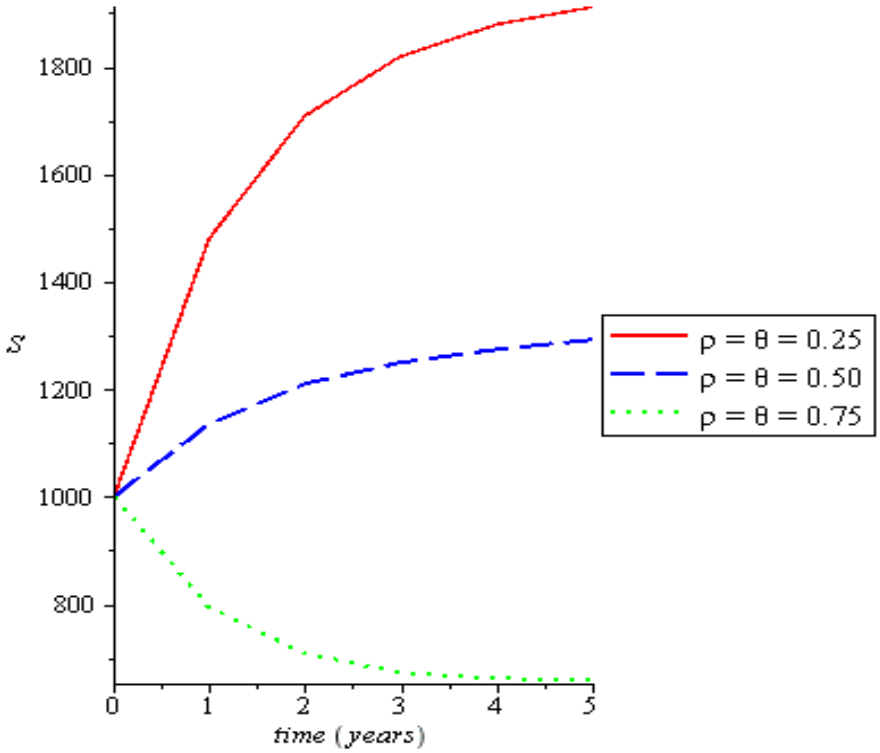

Figure 1: The graph of susceptible individuals versus time.

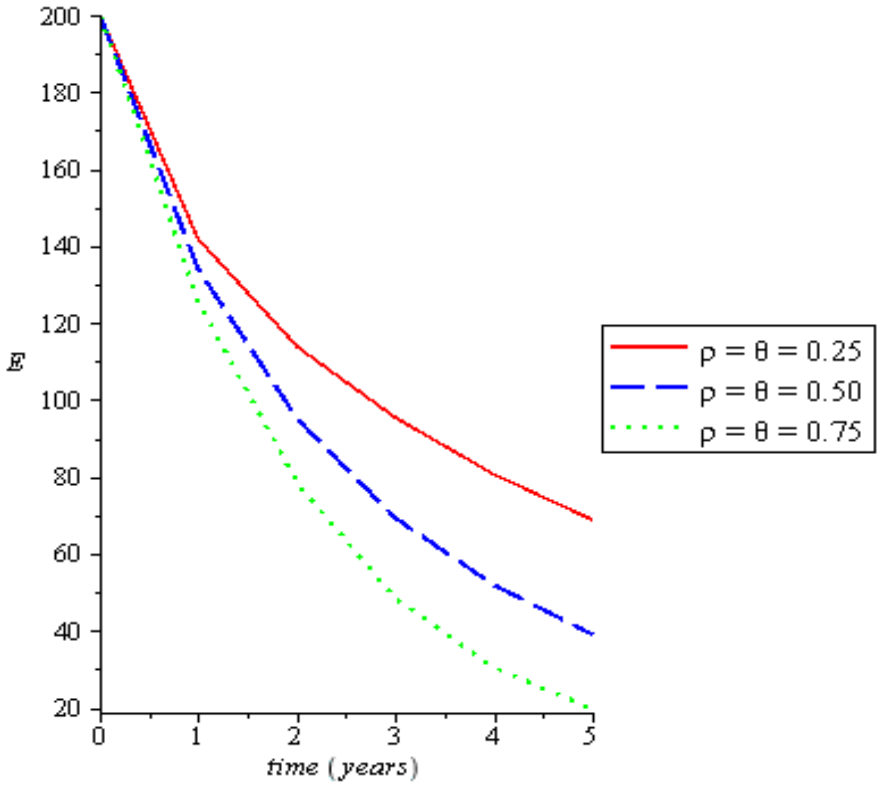

Figure 2: The graph of Exposed individuals versus time.

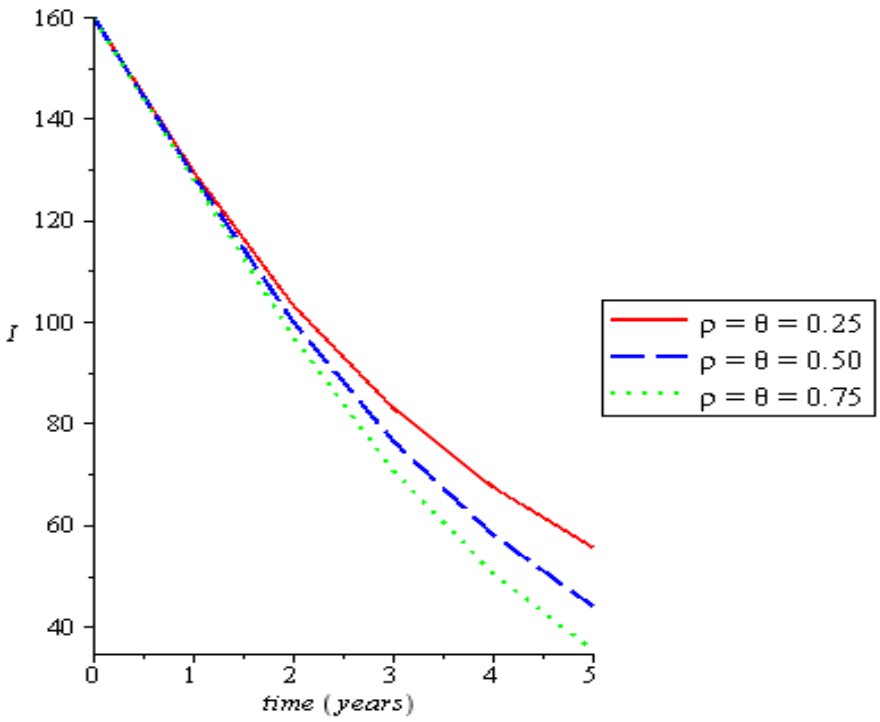

Figure 3: The graph of Infectious individuals versus time.

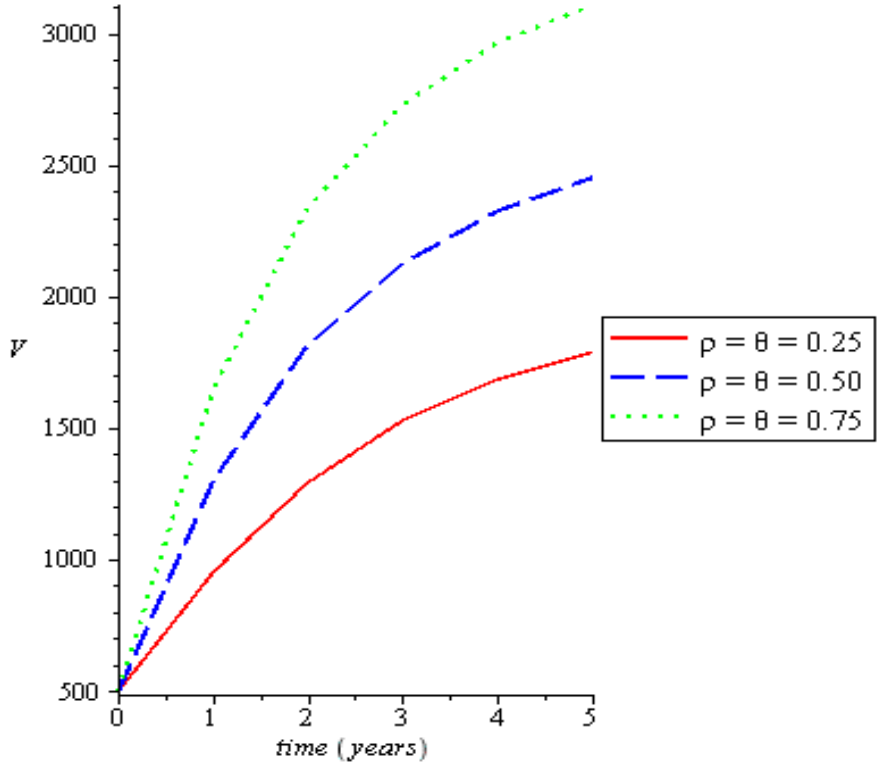

Figure 4: The graph of Vaccinated individuals versus time.

4.1 Discussion

Figure 1 is the graph of susceptible individuals versus time. The graph shows that the population of the susceptible individuals increases at low and moderate vaccination rates but goes to zero at high vaccination coverage. Figure 2 is the graph of Exposed individuals versus time. The graph shows that the population of the Exposed individuals decreases as the vaccination coverage increases and brought down the population Exposed individuals to zero at high vaccination rate. This means the polio can be eradicated completely in the population at time (t) = 5 years as per our graph.

Figure 3 is the graph of Infectious and paralyzed individuals versus time. The graph shows that the population of the Infectious and paralyzed individuals decreases as the vaccination coverage increases and brought down the population Infectious and paralyzed individuals to zero at high vaccination rate. This means that, at high vaccination coverage, a disease-free equilibrium can be reached. Figure 4 is the graph of vaccinated individuals versus time. The graph validates the effects of vaccination on the dynamics of the disease in the population.

5. Concluding Remarks

In this work, we formulated a deterministic mathematical model of Polio infection dynamics, with the aim of performing a theoretical analysis of epidemiological meaningfulness. We derived the basic reproduction number, R_0 of the model, and it was used to perform a qualitative analysis on the model. We obtained both the Disease-Free Equilibrium (DFE) and the Endemic Equilibrium points of the model. We further proved that that the DFE is locally and globally asymptotically when $R_0 < 1$ which means the disease will die out, in addition to that, using Lemma 1 we show the existence of Endemic Equilibrium when $R_0 \geq 1$. The result of the numerical simulation reveals that with 75% vaccination coverage for the immigrants and new-born, polio would be eradicated completely. Therefore, we have been able to prove the assertion that prevention is better than cure even at just about 75% vaccination coverage.

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