On the Effect of poliomyelitis and immunity in poliovirus epidemiology and the Role of Vaccine.

H.A Gazali[1] and M.M. Altine[2]

[1]Department of Mathematics, Sokoto Cement Sec. Sch. Sokoto, Nigeria.

[2]Department of Mathematics, Federal University Birnin Kebbi, Nigeria.

[1]gazalianka@gmail.com, [2]altinemuhammad@gmail.com

[1]08060266665, [2]08064819725

Keywords: poliovirus, DFE, EE, Symptoms, model, stability

Abstract

In this paper, we studied the mathematical modeling of the effect of poliomyelitis and immunity in poliovirus epidemiology. It centers on the application of mathematics as a tool in explaining the dynamics of poliovirus transmission. The study is based on understanding the role of Vaccine. This work focuses on rate of Vaccine and the chronicle stage of the virus tested for the existence and uniqueness of solution for the model using the Lipchitz condition to ascertain the efficacy of the model and proceeded to determine both the Disease Free Equilibrium (DFE) and the Endemic Equilibrium (EE) for the system of equations. We have seen that the system equations has a Unique solution. The local stability of the (DFE) of the model was obtained using the Variational Matrix Criteria while the stability of (EE) was analyzed. The reproduction number was calculated and simulated. We demonstrated that the disease will die out, if the basic reproductive numbers for the disease free equilibrium $R_0 < 1$. This is the case of a disease free state, with no infection in the population. Otherwise the disease may become endemic if the basic reproductive number R_0 is bigger than unity (i.e $R_0 > 1$). The basic reproduction number at both the disease Free State and the endemic state were obtained and the result shows stability in the role of Vaccine as a means of reducing the spread of the disease in the society.

1. INTRODUCTION

Polio (also called poliomyelitis) is a contagious, historically devastating disease that was virtually eliminated from the western hemisphere in the second half of the 20th century. Although polio has plagued humans since ancient times, its most extensive outbreak occurred in the first half of the 1900s before the vaccine,created by Jonas Salk

[1]in 1952, became widely available in 1955. It can strike at any age, but affects mainly children under three (over 50% of all cases). The virus enters the body through the mouth and multiplies in the intestine.

The application of mathematical models to disease goes back to 1840. Ever since, scientists have been trying to analyze the spread of disease by means of mathematical models. A variety of mathematical models ranging from very simple ones to complicated ones have been developed and analyzed in order to capture different phenomena associated with the spread of diseases [2].Polio is a highly infectious disease caused by a virus. It invades the nervous system, and can cause total paralysis in a matter of hours. The virus is transmitted from person to person, spread mainly through the fecal oral route, less frequently by a common vehicle or via the surrounding environment without direct contact (e.g. contaminated water or food) and multiplies in the intestine.

Initial symptoms are fever, fatigue, headache, vomiting, and stiffness in the neck and pain in the limbs. One in 200 infections lead to irreversible paralysis (usually in the legs) among those paralyzed, less than 5% die when their breathing muscle become immobilized.

Polio is a viral illness that, in about 95% of cases, actually produces no symptoms at all (called asymptomatic polio). In the 5% of cases in which there are symptoms (called symptomatic polio), the illness appears in three forms:

i. A mild form called abortive polio (most people with this type may not even suspect they have it because their sickness is limited to mild flu-like symptoms such as mild upper respiratory infection, diarrhea, fever, sore throat and a general feeling of being ill).
ii. A more serious form associated with aseptic meningitis called non paralytic Polio (1% - 5% show neurological symptoms such as sensitivity to light and neck stiffness).
iii. A severe, debilitating form called paralytic polio (this occurs in 0.1% - 2% of cases).

People who have abortive polio or non paralytic polio usually make full recovery. However, paralytic polio, as its name implies, causes muscle paralysis – and can even result to death.

In paralytic Polio, the virus leaves the intestinal tract and enters the bloodstream, with the aim to attack the nerves while abortive or asymptomatic polio, the virus usually doesn't get past the intestinal tract. The virus may affect the nerves governing the muscles in the limbs and the muscles necessary for breathing, causing respiratory difficulty and paralysis of the arms and legs.

It can strike at any age, but affects mainly children under five through contaminated drinking water. In some cases, the poliovirus can even cause death. Poliovirus is a communicable disease which must be eradicated. Children that are not immunized are at high risk of being paralyzed by polio.

This Paper describes the mathematical modeling of the effect of poliomyelitis and immunity in poliovirus epidemiology. It centers on the application of mathematics as a tool in explaining the dynamics of poliovirus transmission. The study is based on understanding the role of Vaccine as a means of reducing the spread of the disease in the society.

Health groups are working towards wiping out polio throughout the world, and much progress has been made. But several countries still have polio circulating, which means that the virus could occur in others. If the polio reaches a country where not enough people have been immunized, it could spread from person to person, just as it happened in some countries in Africa and Asia. So until it has been eliminated worldwide, it is important to continue vaccinating kids against poliovirus. Two fresh infections of wild polio virus have emerged in Gwarzo and Jere local Government Areas of Borno State Nigeria, threatening Nigeria's progress toward a polio free status. It was due to be declared polio free by the World Health Organization [3] if it made it to July 24th 2017 without a new case. The last known infection was July, 2014. There had been concern about children on camps for displaced people in Borno missing immunization as Nigeria struggled to prevent a flare up.

Eguda and Yakubu [4], proposed a mathematical model to study the spread and control of polio virus introducing vaccination and treatment. The model equations were analyzed to obtain the characteristics equation and equilibrium states. Stability analysis of the zero and non-zero equilibrium states was carried out.

Momoh *et al* [5] consider the mathematical model for the control of polio virus epidemiology by presenting and analyzing an SEIR to ascertain the impact of exposed individuals at latent period on the transmission dynamic of polio virus.

This Paper extends the work of Manju and Bhadauria [6] by considering a class of recovered individual by reflection on the Oral polio vaccine induced immunity and we study its effect on the entire population using all the model analysis tools described by the aforementioned authors.

2. Model formulation

To formulate a non linear deterministic mathematical model with much details on vaccine strategy, such as waning rate, efficacy and permanent immunity induced by the vaccine. The total homogenous population N(t) at time t, is

stratified into subpopulation of unvaccinated individuals S(t), vaccinated individuals V(t); asymptomatic E(t) and symptomatic I(t) and Polio infected individuals and recovered individuals R(t).

The total population N(t) is given as

$$N(t) = S(t) + V(t) + E(t) + I(t) + R(t)$$

The model takes the form of the following deterministic system of non- linear equations.

$$\frac{dS}{dt} = m_1 + \theta_2 V - \Gamma S - k_1 S \qquad (1)$$

$$\frac{dV}{dt} = m_2 + \theta_1 S - (1 - \in)\Gamma V - k_2 V \qquad (2)$$

$$\frac{dE}{dt} = \Gamma S + (1 - \in)\Gamma V - k_3 E \qquad (3)$$

$$\frac{dI}{dt} = \gamma_1 E - k_4 I \qquad (4)$$

$$\frac{dR}{dt} = \gamma_2 I + \eta V - \mu R \qquad (5)$$

Where $k_1 = \mu + \theta_1, \quad k_2 = \theta_2 + \eta + \mu, \quad k_3 = \mu + \gamma_1, \quad k_4 = \mu + \delta + \gamma_2$

$m_1 = (1 - p)\Lambda + (1 - \phi)\pi, \qquad m_2 = p\lambda + \phi,$

But

$$\Gamma = \beta(I + rE) \qquad (6)$$

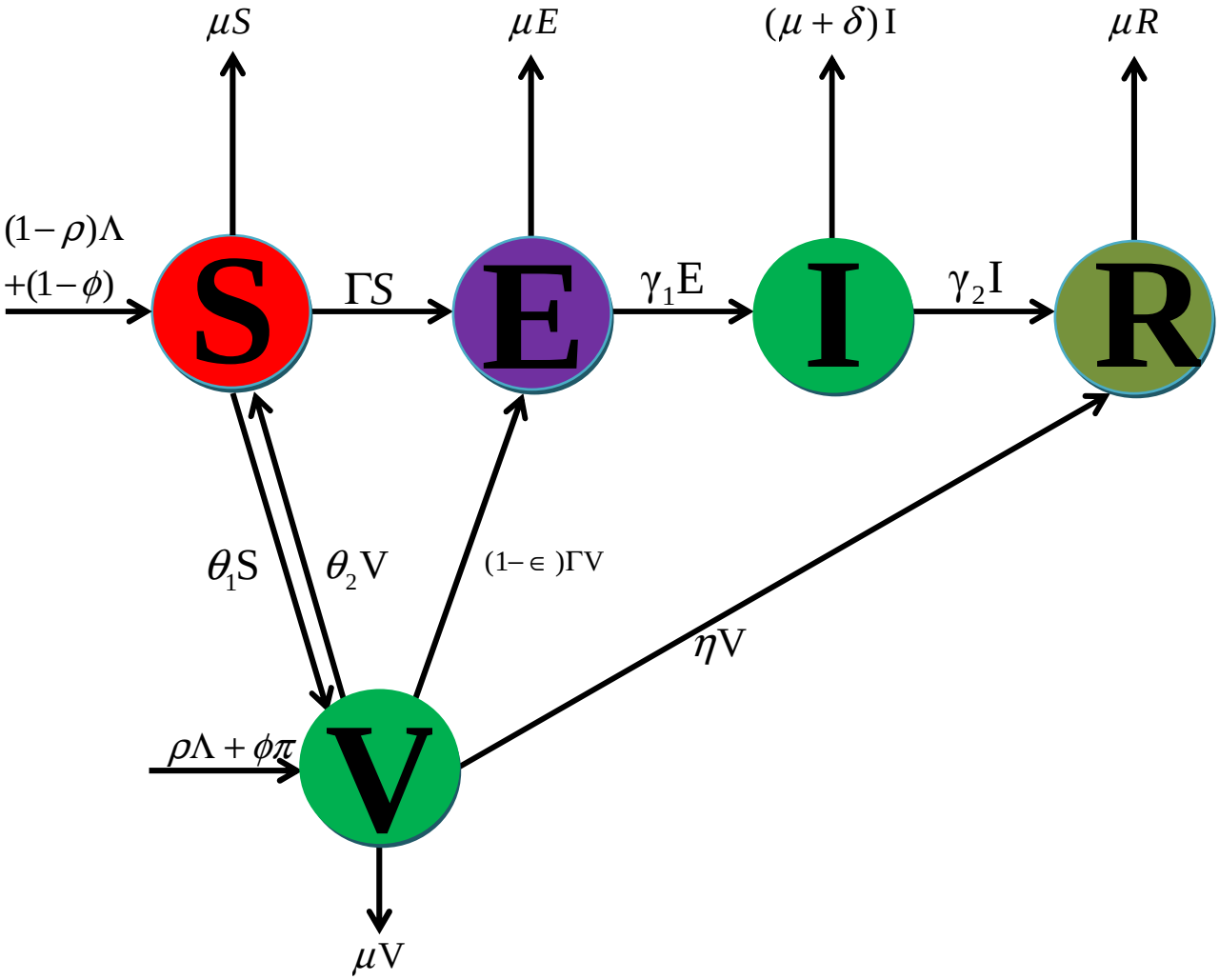

Figure 1: Schematic diagram of the model

NOMENCLATURE

Symbol Interpretation

Variable	Interpretation
S	Population of unvaccinated individuals
V	Population of vaccinated individuals
E	Population of asymptomatic infected individuals
I	Population of symptomatic infected individuals and polio infected individuals
R	Population of recovered individuals.

Parameter	Interpretation
π	Migration rate
ϕ	Rate of vaccinated migrant
Λ	Per capita birth rate

ρ	Vaccinated rate of new born
θ_1	Vaccinated rate of susceptible individuals
θ_2	Waning rate of Oral polio Vaccine
$\in$	Efficacy of Oral Polio Vaccine
r	Disease transmission coefficient
β	Birth rate
δ	Polio induced death rate
γ_1	Progression rate from E to I
γ_2	Recovery rate of I individuals due to treatment
μ	Natural death rate
η	Oral Polio vaccine induce immunity

3. Existence and uniqueness solution of the model

The validity and usability of any mathematical model depends on whether the given set of equations has a solution, if it has, is the solution Unique?

This section is concern with finding if the system of equations has a solution and if the solution to the system is unique. We shall use the lipchtiz condition to verify the existence and uniqueness of equations. Let

$$\begin{aligned} f_1 &= m_1 + \theta_2 V - \Gamma S - k_1 S \\ f_2 &= m_2 + \theta_1 S - (1 - \in \alpha)\Gamma V - k_2 V \\ f_3 &= \Gamma S + (1 - \in \alpha)\Gamma V - k_3 V \\ f_4 &= \gamma_1 E - k_4 I \\ f_5 &= \gamma_2 I + \eta V - \mu R \end{aligned} \qquad (7)$$

UsingDerrick and Grossman, let D denotes the region $|t - t_0| \le a, \|x - x_0\| \le b,$, $x = (x_1, x_2, ...x_n), x_0 = (x_{10}, x_{20}, ...x_n)$ and suppose that $f(t, x)$ satisfies the Lipschitz condition $\|f(t, x) - f(t, x_2)\| \le k\|x_1 - x_2\|$. Whenever the pairs (t, x) and (t, x_2) belong to D^1, where k is a positive constant, then there is a constant $\delta > 0$ such that there exist a unique continuous vectors solution $x(t)$ of the

system in the interval $t - t_0 \leq \delta$. It is important to note that the condition is satisfied by requirement that $\frac{\partial f_i}{\partial x_i}, i = 1, 2, ...,$ be continuous and bounded in D.

We now return to our model equations. We are interested in the region

$$0 \leq \alpha \leq m$$

We look for a bounded solution in this region and whose partial derivatives satisfy $0 \leq \alpha < \infty$, where α and δ are positive constants.

Let D denote the region $0 \leq \delta \leq m$, then equation have a unique solution. We show that $\frac{\partial f_i}{\partial x_i}, i = 1, 2, 3, 4, 5$ are continuous and bounded in D

Recall, from eqn (7)

Taking the partial derivative, i.e

$$\left|\frac{\partial f_1}{\partial S}\right| = \left|-(\Gamma + k_1)\right| < \infty; \quad \left|\frac{\partial f_1}{\partial V}\right| = \left|\theta_2\right| < \infty; \quad \left|\frac{\partial f_1}{\partial E}\right| = \left|-\beta r S\right| < \infty$$

$$\left|\frac{\partial f_1}{\partial I}\right| = \left|-\beta S\right| < \infty; \qquad \left|\frac{\partial f_1}{\partial R}\right| = 0 < \infty$$

Then, the partial derivatives of the whole system of equation exist, they are finite and bounded. Hence, the model system equation has a unique solution.

4. Equilibria State and Stability Analysis of the Model

4.1. Existence of equilibrium point

At equilibrium, the left hand side of (1) – (5) is equated to zero, i.e

$$\frac{dS}{dt} = \frac{dV}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0$$

Thus (1) – (5) becomes

$$m_1 + \theta_2 V - \Gamma S - k_1 S = 0 \quad (8)$$

$$m_2 + \theta_1 S - (1-\epsilon)\Gamma V - k_2 V = 0 \quad (9)$$

$$\Gamma S + (1-\epsilon)\Gamma V - k_3 E = 0 \quad (10)$$

$$\gamma_1 E - k_4 I = 0 \quad (11)$$

$$\gamma_2 I + \eta V - \mu R = 0 \quad (12)$$

From (8), we have

$$S = \frac{m_1 + \theta_2 V}{\Gamma + k_1} \quad (13)$$

Similarly from (9) we get

$$S = \frac{\left[(1-\epsilon)\Gamma + k_2\right]V - m_2}{\theta_1} \quad (14)$$

Substituting (13) into (14) to obtain

$$\frac{\left[(1-\epsilon)\Gamma + k_2\right]V - m_2}{\theta_1} = \frac{m_1 + \theta_2 V}{\Gamma + k_1}$$

$$\Rightarrow V = \frac{m_1\theta_1 + m_2\left[\Gamma + k_1\right]}{\left[(1-\epsilon)\Gamma + k_2\right]\left[\Gamma + k_1\right] - \theta_1\theta_2} \quad (15)$$

Use (15) in (13) to get

$$S = \frac{\left[(1-\epsilon)\Gamma + k_2\right]m_1 + \theta_2 m_2}{\left[(1-\epsilon)\Gamma + k_2\right]\left[\Gamma + k_1\right] - \theta_1\theta_2} \quad (16)$$

For convenience, (15) and (16) are re-written as

$$V = \frac{m_1\theta_1 + m_2\left[\Gamma + k_1\right]}{L_0} \tag{17}$$

$$S = \frac{\left[\left(1-\in\right)\Gamma + k_2\right]m_1 + \theta_2 m_2}{L_o} \tag{18}$$

Where $L_0 = \left[\left(1-\in\right)\Gamma + k_2\right]\left[\Gamma + k_1\right] - \theta_1\theta_2$, it is important to note that $L_0 > 0$ since $k_1k_2 - \theta_1\theta_2 = \left(\mu + \theta_1\right)\left(\eta + \mu\right) + \theta_2\mu > 0 \; and \;\; \in < 1$

From (11), we have

$$I = \frac{\gamma_1 E}{k_4} \tag{19}$$

Substituting (19) into Γ, we have

$$E = \frac{\Gamma k_4}{\beta\left(\gamma_1 + rk_4\right)} \tag{20}$$

From (12)

$$R = \frac{\gamma_2 I + \eta V}{\mu} \tag{21}$$

With (17) and (18), we have

$$S + \left(1-\in\right)V = \frac{\left[\left(1-\in\right)\Gamma + k_2\right]m_1 + \theta_2 m_2}{L_o} + \frac{\left(1-\in\right)\left\{m_1\theta_1 + m_2\left[\Gamma + k_2\right]\right\}}{L_0}$$

$$S + \left(1-\in\right)V = \frac{\left[\left(1-\in\right)\Gamma + k_2\right]m_1 + \theta_2 m_2}{L_o} + \frac{\left(1-\in\right)\left\{m_1\theta_1 + m_2\left[\Gamma + k_2\right]\right\}}{L_0}$$

$$S+(1-\epsilon)V=\frac{(1-\epsilon)\Gamma m_1+k_2m_1+\theta_2m_2+(1-\epsilon)m_1\theta_1+\Gamma m_2+(1-\epsilon)k_1m_2}{L_o}$$

$$S+(1-\epsilon)V=\frac{(1-\epsilon)(m_1+m_2)\Gamma+m_1\left[\theta_1(1-\epsilon)+k_2\right]+m_2\left[k_1(1-\epsilon)+\theta_2\right]}{L_o} \quad (22)$$

From (10) we obtain

$$k_3E=\Gamma\left[S+(1-\epsilon)V\right] \quad (23)$$

Substitute (20) into (23) to get

$$\frac{k_3k_4\Gamma}{\beta(\gamma_1+rk_4)}=\Gamma\left[S+(1-\epsilon)V\right]$$

$$\Gamma\left\{k_3k_4-\beta(\gamma_1+rk_4)\left[S+(1-\epsilon)V\right]\right\}=0$$

Thus, either

$$\Gamma=0 \ or \ \ S+(1-\epsilon)V=\frac{k_3k_4}{\beta(\gamma_1+rk_4)} \quad (24)$$

4.2. Existence of Disease Free Equilibrium

In the absence of infection, i.e $\Gamma^*=0$, from (24), hence from (10) and (11) we have $E^*=I^*=0$

From (15,16 and 21)

$$V^*=\frac{m_1\theta_1+m_2k_1}{k_1k_2-\theta_1\theta_2}, \qquad S^*=\frac{m_1k_2+m_2\theta_2}{k_1k_2-\theta_1\theta_2}, \qquad R^*=\frac{\eta\left[m_1\theta_1+m_2k_1\right]}{\mu\left[k_1k_2-\theta_1\theta_2\right]}$$

Let ε_0 denote the disease free equilibrium, such that

$$\varepsilon_0 = \left(S^* V^* E^* I^* R^*\right) = \left(\frac{m_1 k_2 + m_2 \theta_2}{k_1 k_2 - \theta_1 \theta_2}, \frac{m_1 \theta_1 + m_2 k_1}{k_1 k_2 - \theta_1 \theta_2}, 0, 0, \frac{\eta\left[m_1 \theta_1 + m_2 k_1\right]}{\mu\left[k_1 k_2 - \theta_1 \theta_2\right]}\right)$$

$$\therefore R_0 = \frac{\beta\left\{m_1\left[\theta_1 + (1-\in) + k_2\right] + m_2\left[k_1(1-\in) + \theta_2\right]\right\}(rk_4 + \gamma_1)}{\left[k_1 k_2 - \theta_1 \theta_2\right] k_3 k_4} \qquad (25)$$

5. Local stability of DFE

The Disease free equilibrium ε_0 is locally asymptotically stable whenever $R_c < 1$ and unstable whenever $R_c > 1$

The evaluated Jacobian matrix at ε_0 is given as

$$J(\varepsilon_0) = \begin{bmatrix} -k_1 & \theta_2 & -\beta r S^* & -\beta S^* & 0 \\ \theta_1 & -k_2 & -(1-\in)\beta V^* & -(1-\in)\beta V^* & 0 \\ 0 & 0 & \beta r\left[S^* + (1-\in)V^*\right] - k_3 & \beta\left[S^* + (1-\in)V^*\right] & 0 \\ 0 & 0 & \gamma_1 & -k_4 & 0 \\ 0 & \eta & 0 & \gamma_2 & -\mu \end{bmatrix}$$

Thus, the characteristics equation of $J(\varepsilon_0)$ is obtain as

$$(\lambda + \mu)\left[a_4 \lambda^4 + a_3 \lambda^3 + a_2 \lambda^2 + a_1 \lambda + a_0\right] = 0$$

Where the coefficient of the Eigen value (i.e λ) in the above equation are expressed in a simplified form as

$$a_4 = 1$$

$$a_3 = \frac{k_4\left[k_4 + k_2 + k_1\right] + Q\gamma_1 + k_3k_4\left(1 - R_0\right)}{k_4}$$

$$a_2 = \frac{k_3k_4\left[\gamma_1 + r\left(k_4 + k_2 + k_1\right)\right]\left(1 - R_0\right) + \left(k_1k_2 - \theta_1\theta_2\right)\left(\gamma_1 + rk_4\right) + \left(k_2 + k_1\right)\left[\gamma_1k_3 + k_4\left(\gamma_1 + rk_4\right)\right]}{\left(\gamma_1 + rk_4\right)}$$

$$a_1 = \frac{\left[k_4\left(\gamma_1 + rk_4\right) + k_3\gamma_1\right]\left[k_1k_2 - \theta_1\theta_2\right] + k_3k_4\left[\left(k_2 + k_1\right)\left(\gamma_1 + rk_4\right) + r\left(k_1k_2 - \theta_1\theta_2\right)\right]\left(1 - R_0\right)}{\left(\gamma_1 + rk_4\right)}$$

$$a_0 = k_3k_4\left(k_1k_2 - \theta_1\theta_2\right)\left(1 - R_0\right)$$

Hence, the Disease Free Equilibrium of the model is locally asymptotically stable.

5.1. Existence of Endemic Equilibrium Point

In the presence of infection, i.e $I^{**} \neq 0,\ E^{**} \neq 0,$ thus $\Gamma^{**} \neq 0$. Hence from (24) we have

$$S^{**} + (1-\in)V^{**} = \frac{k_3k_4}{\beta(\gamma_1 + rk_4)} \tag{26}$$

$$A\Gamma^{**2} + B\Gamma^{**} + C = 0$$

Where

$$A = k_3k_4\left(1-\in\right)$$

$$B = k_3k_4\left[k_1\left(1-\in\right) + k_2\right] - \beta\left[\gamma_1 + rk_4\right]\left(1-\in\right)\left[m_1 + m_2\right]$$

$$C = k_3k_4\left[k_1k_2 - \theta_1\theta_2\right]\left[1 - R_0\right]$$

Thus, $A > 0$ since $\in < 1$ and $C < 0$, if and only if $R_{c1} > 1$. Hence the theorem below is established.

The Model equations has a unique positive (endemic) equilibrium if and only if $R_{c1} > 1$

5.2. Local Stability of Endemic Equilibrium Point

The local stability of the unique endemic equilibrium ε_1 is investigated for the special case, where the imperfectness of the prophylactic vaccine (i.e OPV) is negligible or assumed to be absence i.e $\varepsilon = 1$ thus the associated reproductive number denoted by R_{c1} is given as

$$R_{c1} = \frac{\beta\{m_1 k_2 + m_2\theta_2\}(rk_4 + \gamma_1)}{(k_1 k_2 - \theta_1\theta_2)k_3 k_4}$$

The endemic equilibrium when $\varepsilon = 1,$ denoted by $\varepsilon_2 = \varepsilon_1 / \varepsilon = 1$ is locally asymptotically stable whenever $R_{c1} > 1$ and whenever $R_{c1} < 1$

The variational matrix of the model evaluated at $\varepsilon_2 = \varepsilon_1 / \varepsilon = 1$ is gotten as

$$J(\varepsilon_2) = \begin{bmatrix} \frac{-(\theta_2 V^{**} + m_1)}{S} & \theta_2 & -\beta r S^{**} & -\beta S^{**} & 0 \\ \theta_1 & -k_2 & 0 & 0 & 0 \\ \Gamma & 0 & -\frac{\beta S^{**} I^{**}}{E^{**}} & \beta^{**} S^{**} & 0 \\ 0 & 0 & \gamma_1 & -k_4 & 0 \\ 0 & \eta & 0 & \gamma_2 & -\mu \end{bmatrix}$$

Where

$$\frac{\theta_2 V^{**} + m_1}{S^{**}} = \Gamma^{**} + k_1 \qquad from \quad (1)$$

$$-\frac{\beta S^{**} I^{**}}{E^{**}} = \beta r S^{**} - k_3 \quad from \ (2) \quad when \in = 1$$

Thus, the characteristic equation is obtained as

$$(\lambda + \mu)\left[\lambda^4 + a_0\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3\right] = 0$$

Where

$$a_0 = \frac{\left(k_4 + k_2\right)E^{**}S^{**} + \beta S^{**2}I^{**} + E^{**}\left(\theta_2 V^{**} + m_1\right)}{E^{**}S^{**}}$$

$$a_1 = \frac{\left(k_4 E^{**} + \beta S^{**}I^{**}\right)\left(k_2 S^{**} + \theta_2 V^{**} + m_1\right) + E^{**}\left(m_2\theta_2 + m_1 k_2\right) + \Gamma^{**}\beta r S^{**2}E^{**}}{E^{***}S^{**}}$$

$$a_2 = \frac{\beta S^{**2}\Gamma^{**}E^{**}\left(\gamma_1 + rk_4\right) + k_4 E^{**}\left(m_2\theta_2 + k_2 m_1\right) + \beta S^{**2}\Gamma^{**}rE^{**}k_2}{E^{**}S^{**}}$$

$$a_3 = \beta\Gamma^{**}k_2 S^{**}\left[\gamma_1 + rk_4\right]$$

It is clearly seen that $a_i > 0 \forall i = 0,....3$ only if $R_0 > 1$, hence we concluded the proof since the Routh Hurtwiz Criterion is satisfied, that the system is locally asymptotically stable.

6. Numerical Simulation and Discussion of Result

The role played by some important epidemiological parameters, are investigated with the aid of maple software for the numerical simulation by comparing the model effective reproductive number, the parameters used, their estimated values and appropriate source are given in table 1.

Table 1: Numerical Simulation of the model

θ_1	θ_2	ε	η	R_0	$E^{**} + I^{**}$	Remark
0.9	0.2	0.9	1	0.94	0	Unstable
0.99	0	0.9	1	0.65	0	Unstable
0.99	0	0.8	1	0.75	0	Unstable
0.8	0	0.8	1	0.91	0	Unstable
0.9	1	0.1	0	68.87	896.15	Stable
0.8	0.8	0.4	0	60.83	893.75	Stable
0.8	0.6	0.4	0.2	9.19	789.94	Stable
0.6	0.4	0.6	0.4	4.45	629.30	Stable
0.6	0.6	0.6	0.6	4.04	577.94	Stable

7. Conclusion

In this paper, an endemic model on polio with effect of vaccination is considered. Two threshold parameters R_o and R_1 corresponding to interaction of susceptible with infective and exposed class respectively are found. The sum of these two threshold values, denoted by R, is proved to be a sharp threshold value which completely determines the stability dynamics and the outcome of the disease. It is found that if $R<1$, the disease free equilibrium is stable and the disease dies out, however if $R>1$, there exists a unique endemic equilibrium which is locally asymptotically stable. Persistence of the disease is shown for $R_o>1$ and $R_1> 1$. To substantiate the analytical findings, the model is numerically and for which the system of differential equation is integrated, which satisfy the stability conditions. Numerical simulation is performed for different initial starts and the results are displayed. It is concluded that endemic level of infective population increases with the increase in rate of transmission of infection from infective

to susceptible class, which can further be enhanced if transmission of infection occurs from latent hosts during incubation period. However, for a particular value of disease transmission coefficient r β, exposed and infective population die out. Variation of infective equilibrium size of the population with basic reproduction numbers determined and it is found that endemic infective level first rises with the increase in basic reproduction ratio and then becomes constant.

It is found that although vaccination helps in eradicating polio by decreasing endemic equilibrium level yet careful administration of vaccination is desired because if vaccine is administered in an individual during incubation period of polio, endemic equilibrium level increases and decrease spreads faster than usual pace. Like any other IM injection, it can precipitate paralysis in a patient who is already in incubation period of polio, as can occur during polio epidemics. Hence, IPV or any other IM injection should be avoided in an unimmunized sick child with fever especially during season of polio epidemics. It is found that the periodic outbreak of the disease occurs when infection due to exposed and infective class occurs at the same rate. It is pointed out from my study that population movement also contributes to the spread of the disease; constant migration in human population makes the disease endemic. The movement of infected people from areas where polio is still endemic to areas where the disease has been eradicated led to resurgence of the disease. It is further observed that transmission of infection due to population in exposed state of polio plays an important role in the spread of polio and hence some measures must be adopted to trace the population in exposed class as it is very difficult to trace them out because of absence of symptoms of disease in them.

The introduction of the virus into a population poses definite health threat, hence the need to adopt a preventive measure and intensify efforts to the fight against the pandemic.

REFERENCES

[1] Jonas S. (1952); population models for disease with no recovering; J Math Biol. Vol.28.

[2] Brauer A.W (1990); Models for the spread of universally fatal diseases, J Math, Biol, Springer-Verlag; PP 451-462.Bull. Math Biol. 94, pp. 1123-1148.

[3] World Health Organization Country Office Tajikistan, Who Regional Office for Europe (2010). European center for disease prevention and control outbreaks of Poliomyelitis in Tajikistan in 2010: risk for importation and impact on polio surveillance in Europe. Euro Surveill., vol.15 No. 17.

[4] Eguda R. and Yakubu E. (2010), *Mathematical model of study the spread and control of poliovirus introducing vaccination and treatment.*

[5] Momoh A., Ibrahim M.O., Uwanta I.J. and Manga A., (2013), *Chacteristerizing Measles transmission and evolution; Insight from modeling experiences with wild and vaccine related polioviruses.*

[6] Manju A. and Bhadauria A.S (2011), Modeling the Spread of polio with the role of Vaccination. An International Journal (AAM) Vol. 6, Issue 2, Pp. 552-571. Department of Mathematics and Astronomy, Lucknow University, Uttar Pradesh, India.