GSJ: Volume 11, Issue 5, May 2023, Online: ISSN 2320-9186
www.globalscientificjournal.com

MATHEMATICAL MODEL ON TRANSMISSION OF THE POLIO HOSPITALIZATION WITH VACCINATION

Adedayo, O.A, Sunday L. A., Akande S. A., Ugwu, U.C, Muhammed, I., Musa O.I. and [7]Tiamiyu. A.T.

[1,3,4] Department Mathematics,
Federal University of Technology, Minna Nigeria
[2]Department of Mathematics and Computer Science
Indiana University of Pennsylvania

[5]Department of Mathematics
Khalifa University, Abu Dhabi, UAE.

[6]Department of Microbiology
Federal University of Technology, Minna Nigeria

[7]Department of Mathematics
The Chinese University of Hong Kong,
Shatin, New Territories, Hong Kong

Corresponding author: adedayoolufemi@gmail.com

Abstract

The present study involved the development and thorough analysis of a deterministic mathematical model that accounts for the transmission dynamics of Polio Infection, taking into consideration the effects of Vaccination and hospitalization. The model incorporated a population of human beings affected by polio, which were subsequently categorized into six distinct compartments. The model exhibits two distinct equilibria, namely the diseases-free equilibrium (D.F.E) and the endemics equilibrium states (E.E). The stability of equilibrium states was analyzed in relation to the effective reproduction number. The findings indicate that the equilibrium state free from disease was stable, and the established criteria for stability of the endemic equilibrium state were observed. The study demonstrated that the equilibrium state of polio infection being free from contagion is both locally and globally asymptotically stable $R_0 < 1$. The Homotopy Perturbation Method (HPM) was utilized to derive the

analytical solution, and the effective reproduction number was subsequently calculated to evaluate the relative impact of individual or combined interventions for the purpose of achieving effective disease control. The model's numerical simulations indicate that the most effective strategy for combating the epidemiology of polio virus is through a combination of vaccination.

Keyword: Mathematical Model, Transmission, Polio, Hospitalisation, and Vaccination

1. Introduction

Poliovirus is the etiological agent responsible for the onset of polio, an infectious ailment. In approximately 0.5% of cases, there exists the potential for muscle weakness resulting in immobility. Poliomyelitis is an infectious ailment that can impact the central nervous system and, in some cases, lead to flaccid paralysis of varying degrees of severity. This medical condition has also been referred to as infantile paralysis or Heine-Medin disease, as documented by Avendano et al. (2011). The poliovirus exhibits a marked tropism for intestinal cells and is typically transmitted through the fecal-oral route, although respiratory transmission is also possible. It is noteworthy that humans are the sole natural host for this virus. According to Avendano, Ferres, and Spencer (2011), the typical progression of the virus involves an asymptomatic natural cycle that involves multiplication in the intestine. Following replication in the oropharynx and small intestine, with a particular emphasis on lymphatic tissue, the virus disseminates via the circulatory system to the Central Nervous System (Shors T 2009). According to Levinson (2006), poliomyelitis is characterized by the targeted degeneration of motor neurons, resulting in paralysis and potentially fatal respiratory failure in extreme cases. According to Avendano et al. (2011), the duration of the incubation period for polio ranges from 7 to 21 days, with a minimum of 4 days and a maximum of 40 days. The poliovirus is classified within the enterovirus group (Prats G 2013), which is a constituent of the picornavirus family, encompassing rhinoviruses as well (Shors T 2009). The picornaviruses are characterized by their small size, lack of an envelope, and unique structure consisting of an icosahedral nucleocapsid and a single-stranded RNA genome. Their diameter ranges from 20 to 30 nanometers. Picornaviruses undergo replication within the cellular cytoplasm. Lipid solvents, such as ether, do not render them inactive due to the absence of a protective envelope. Shors (2009) is the cited source. It is noteworthy that enteroviruses exhibit a high degree of resistance to both physical and chemical agents, thereby enabling their persistence in the environment. Their elimination is primarily achieved through fecal matter, a process that can extend for up to six weeks (Levinson, 2006).

Throughout the years, various authors have employed diverse methodologies to construct models of the illness. An SIR epidemic model incorporating pulse vaccination was developed by Shulgin et al. (1998). The findings indicate that the implementation of pulse vaccination has the potential to result in complete eradication of the epidemic, provided that specific conditions, such as the duration of the pulses and the extent of vaccination, are met. (Kribs-Zaleta & Velasco-Hernandez, 2000) developed and analyzed a basic two-dimensional SIS model that incorporates vaccination. The model they proposed exhibited a phenomenon known as backward bifurcation. Farrington (2003) formulated and evaluated a mathematical framework for the poliovirus. The model presented demonstrates a favorable impact of vaccination on the transmission dynamics of polio. A mathematical model of the polio virus was also proposed by Gumel and Moghadas in 2003. The optimal vaccine coverage required

for complete eradication of the infection was successfully attained. The study conducted by Manju and Archana in 2010 involved an analysis of an epidemic model pertaining to Polio, with a focus on the impact of vaccination when it is administered to populations that are susceptible and exposed. The present investigation involved the development of a deterministic mathematical model to examine the transmission dynamics of the polio virus. The developed model takes into account a non-constant and structured total population. Our research aims to examine the impact of vaccination and immigration on the transmission dynamics of polio virus within a population that is not constant.

2. Model Formulation

In this chapter, was developed and analyzed a Mathematical Model for Polio Virus Transmission with virus Migration. In this model, the mathematical model for the human transmission dynamics were incorporated to come up with a mathematical model of polio disease.

Following Bolarin *et al.,* 2018, in this model, was formulate a deterministic mathematical model for polio virus which incorporates immigration and vaccination strategy. The total population $N(t)$ is divided into six compartments namely: Susceptible $S(t)$, Vaccinated $V(t)$, Infectious $I(t)$, Infectious, Hospitalized Individuals $H(t)$, Paralyzed individuals $P(t)$ and Death individuals $D(t)$. In this model, individuals are recruited into the population either by immigration at the rate Λ. We assume that proportions 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 $(1-\theta)$ are not vaccinated so the join the susceptible compartment.

Was assume that the population of the susceptible will receive a vaccine at the rate α to have a permanent immunity. Furthermore, we assume that the natural death rate μ is constant, the disease induced death rate is $\delta_I, \delta_H, \delta_P$, the members of the population mix homogeneously, β is Contact rate at susceptible individual becomes infected by one infectious individual. Susceptible individuals enter the infected class at a rate βSI which is the force of infection. The infected individuals are those infected but they can still infect others. After some time, the infected now move from the infected class into the Paralyzed

individuals class at the rate η, also into the Hospitalized Individuals class at the rate ρ and the hospitalizes individuals are those hospitalized but they can still paralyzed others. After some time, people in hospital now move from hospitalized class into the paralyzed class. After some time, the paralyzed people can lead to death class at the rate γ.According to the nature of the disease, most infected individual cannot recover from the infection (WHO, 2018) hence, we would not consider recovered class.

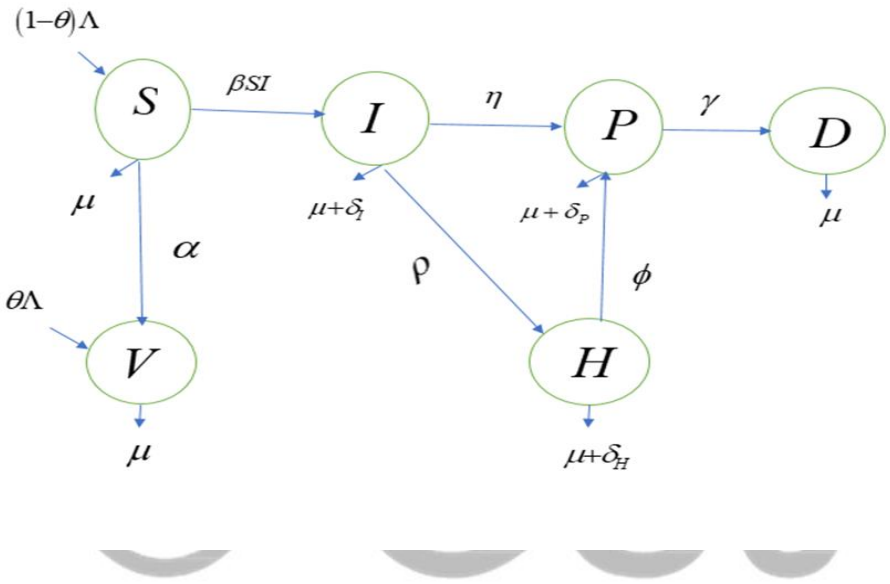

Figure 1: The Flow Diagram

Table 1: State variables and model parameters.

Symbol	Description
$N(t)$	Total number of Population
$S(t)$	Number of Susceptible Individuals
$V(t)$	Number of Vaccinated individuals
$I(t)$	Number of Infected individuals
$H(t)$	Number of Hospitalized individuals
$P(t)$	Number of Paralyzed individuals
$D(t)$	Number of Death individuals
Λ	Immigration
$(1-\theta)$	Proportion of the recruits that are not vaccinated
α	Rate at which susceptible individuals move to vaccinated class

μ	Natural death rate
$\delta_I, \delta_H, \delta_P$	Disease induced death rate
ρ	Rate at which infected individuals move to hospitalized class
η	Rate at which infected individuals move to Paralyzed class
ϕ	Rate at which hospitalized individuals move to Paralyzed class
γ	Rate at which Paralyzed individuals move to death class
ω	Death induced
β	Contact 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;

$$\left.\begin{aligned}
\frac{dS}{dt} &= (1-\theta)\Lambda - \beta SI - (\mu+\alpha)S \\
\frac{dV}{dt} &= \theta\Lambda + \alpha S - \mu V \\
\frac{dI}{dt} &= \beta SI - (\rho+\eta+\mu+\delta_I)I \\
\frac{dH}{dt} &= \rho I - (\phi+\mu+\delta_H)H \\
\frac{dP}{dt} &= \eta I + \phi H - (\mu+\delta_P+\gamma)P \\
\frac{dD}{dt} &= \gamma P - \omega D
\end{aligned}\right\} \quad (1)$$

2.2 Model Analysis

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

2.2.1 The Positive Invariant Region

$$N = S, V, I, H, P, D$$

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dV}{dt} + \frac{dI}{dt} + \frac{dH}{dt} + \frac{dP}{dt} + \frac{dD}{dt} \quad (2)$$

Adding equation (1) we have;

$$\frac{dN}{dt} = \Lambda - \mu\left(S + V + I + H + P + D\right) - \left(\delta_I I + \delta_H H + \delta_p P\right) - \omega D \tag{3}$$

The positive invariant region can be obtained by using the following theorem as applied by (Adedayo *et al.*, 2022).

Theorem 1: The solutions of the system of equations (1) are feasible if they are contained in the invariant region Ω. $\forall \ \ t \geq 0$.

Proof:

Let $\Omega = \left(S, V, I, H, P, D\right) \in R^6$ (4)

Be any solution of the system of equations (1) with non-negative initial conditions. Then from equation (4) we have

$$\frac{dN}{dt} \leq \Lambda - \mu N(t) \tag{5}$$

Since equation (7) is of the form

$$\frac{dy}{dx} + P(x)\,\mathrm{y} = Q(x) \tag{6}$$

with the Integrating Factor $e^{\int P(x)dx}$ as the solution.

We therefore seek the solution of the form

$$e^{\int \mu(\mathrm{t})\,\mathrm{dt}} = e^{\mu t} \tag{7}$$

as the integrating factor of equation (5).

by multiplying through with the integrating factor on equation (5) we obtain;

$$e^{\mu t}\left(\frac{dN}{dt} + \mu N\right) \leq \Lambda e^{\mu t} \tag{8}$$

By virtue of product rule in reverse we have;

$$\Rightarrow \frac{d}{dt}\left(Ne^{\mu t}\right) \leq \Lambda e^{\mu t} \tag{9}$$

Integrating both sides;

$$\int dNe^{\mu t} \leq \int \Lambda e^{\mu t} dt \tag{10}$$

We have;

$$Ne^{\mu t} \leq \frac{\Lambda}{\mu} e^{\mu t} + c \tag{11}$$

Applying the initial conditions $t = 0, N(0) = N_0$

$$N(t)e^{\mu t} \leq \frac{\Lambda}{\mu} e^{\mu t} + c \tag{12}$$

$$= N_0 e^{\mu(0)} \leq \frac{\Lambda}{\mu} e^{\mu(0)} + c \tag{13}$$

$$N_0 \leq \frac{\Lambda}{\mu} + c \Rightarrow N_0 - \frac{\Lambda}{\mu} \leq c \tag{14}$$

From equation (11)

$$Ne^{\mu t} - \frac{\Lambda}{\mu} e^{\mu t} \leq c \tag{15}$$

Divide through by $e^{\mu t}$

$$N - \frac{\Lambda}{\mu} \leq ce^{-\mu t} \tag{16}$$

Comparing (14) and (15)

$$N - \frac{\Lambda}{\mu} \leq (N_0 - \frac{\Lambda}{\mu})e^{-\mu t} \tag{17}$$

$$N(t) \leq \frac{\Lambda}{\mu} + (N_0 - \frac{\Lambda}{\mu})e^{-\mu t} \tag{18}$$

At $t \to \infty$ in equation (18) the human population $N(t)$ approaches $\kappa = \frac{\Lambda}{\mu}$ (that is $N \to k = \frac{\Lambda}{\mu}$) the parameter $\kappa = \frac{\Lambda}{\mu}$ is called the carrying capacity.

Therefore, all feasible solution of the human population of the Model is in the region

$$\Omega = \{(S,V,I,H,P,D) \in \Re^6 : S > 0, V > 0, I > 0, H > 0, P > 0, D > 0, N \le \frac{\Lambda}{\mu}\} \quad (19)$$

2.2.2 Positivity of the Solutions

Theorem 3 Let the initial solutions be $\{(S(0),V(0),I(0),H(0),P(0),D(0) \ge 0\} \in \Omega$ then the solutions $\{S(t),V(t),I(t),H(t),P(t),D(t)\}$ of the system (3.1 to 3.6) is positive $\forall t \ge 0$

Proof

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

$$\frac{dS}{dt} = (1-\theta)\Lambda - \beta SI - (\mu+\alpha)S \ge -(\mu+\alpha)S \quad (20)$$

$$\Rightarrow \frac{dS}{dt} \ge -(\mu+\alpha)S \quad (21)$$

By separating the variable and integrating equation we have;

$$\int \frac{dS}{S} \ge -\int (\mu+\alpha)\,dt \quad (22)$$

We have,

$$\ln S \ge -(\mu+\alpha)t + c \quad (23)$$

Take the e of both sides

$$e^{\ln S} \geq e^{-(\mu+\alpha)t+c} \tag{24}$$

we have

$$S(\mathrm{t}) \geq e^{-(\mu+\alpha)t+c} \tag{25}$$

$$= S(t) \geq e^{-(\mu+\alpha)t}.e^{c} \text{ let } e^{c} = A \tag{26}$$

$$S(t) \geq Ae^{-(\mu+\alpha)t} \tag{27}$$

From equation let $t = 0$ be initial population in the Susceptible compartment

$$S(\mathrm{t}) = \mathrm{S}(0) = \mathrm{S}_0 \; S(0) = \mathrm{A}\, e^{-(\mu+\alpha)(0)} \tag{28}$$

$$S(0) = A \tag{29}$$

$$\mathrm{S(t)} \geq \mathrm{S}(0)e^{-(\mu+\alpha)t} > 0 \tag{30}$$

From equation (1) we have;

$$\frac{dV}{dt} = \theta\Lambda + \alpha S - \mu V \geq -\mu V \tag{31}$$

$$\Rightarrow \frac{dV}{dt} \geq -\mu V \tag{32}$$

By separating the variable and integrating equation we have;

$$\int \frac{dV}{V} \geq -\int \mu dt \tag{33}$$

$$= \ln V \geq -\mu t + c \tag{34}$$

Take the e of both sides

$$e^{\ln V} \geq e^{-\mu t + c} \text{ let } e^{c} = A \tag{35}$$

Then;

$$V(t) = Ae^{-\mu t} \tag{36}$$

From equation let $t = 0$ be initial population in the Vaccinated compartment

$$V(\mathrm{t}) = V(0) = V_0 \; V(0) = Ae^{-\mu(0)} \tag{37}$$

$$V(0) = A \tag{38}$$

$$V(\mathrm{t}) \geq V(0)e^{-\mu t} > 0 \tag{39}$$

Similarly, it can be verified that the rest of the equations are positive for all $t > 0$,

Since $e^{\omega} > 0.,\ \forall \omega \in \Re$

2.2.3 The Existence and Uniqueness of Solution\

THEOREM 4. Let D denotes the region $0 \leq \Lambda \leq \Re$. Then the model system (1) has a unique solution if it is established that $\frac{\partial f_i}{\partial f_j}, i = 1, 2, 3, 4, 5, 6$ are continuous and bounded in D.

Proof Let equations (1) be represented by f_1, f_2, f_3, f_4, f_5 and f_6 respectively. From equation (1), the following partial derivatives are obtain

$$\left.\begin{aligned}
&\left|\frac{\partial f_1}{\partial S}\right| = \left|-\beta I - (\mu+\alpha)\right| < \infty \\
&\left|\frac{\partial f_1}{\partial V}\right| = 0 < \infty \\
&\left|\frac{\partial f_1}{\partial I}\right| = \left|-\beta S\right| < \infty \\
&\left|\frac{\partial f_1}{\partial H}\right| = 0 < \infty \\
&\left|\frac{\partial f_1}{\partial P}\right| = 0 < \infty \\
&\left|\frac{\partial f_1}{\partial D}\right| = 0 < \infty
\end{aligned}\right\} \tag{40}$$

The above partial derivatives exist, continuous and are bounded.

Similarly, (2)-(5) it can be verified that the rest of the equations are positive for all $t>0$,

Since $e^{\omega} > 0.,\ \forall \omega \in \Re$

2.2.4 Disease Free Equilibrium State

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

$$\frac{dS}{dt} = \frac{dV}{dt} = \frac{dI}{dt} = \frac{dH}{dt} = \frac{dP}{dt} = \frac{dD}{dt} = 0 \tag{41}$$

In this case there is no disease: $I = H = P = V = C = D = 0$. Hence, the DFE of our equation is given by

$$A_0 = \left(S^*, V^*, I^*, H^*, P^*, D^*\right) = \left(\frac{(1-\theta)\Lambda}{(\mu+\alpha)}, \frac{\Lambda(\theta\mu+\alpha)}{\mu(\mu+\alpha)}, 0, 0, 0, 0\right) \tag{42}$$

2.2.5 Basic Reproduction Number R_0,

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 $R_0 > 1$ the disease will take over the population. In a more complicated epidemic, the 0R 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:

$$F_i = \begin{bmatrix} \beta SI \\ 0 \\ 0 \end{bmatrix} \text{ and } V_i = \begin{bmatrix} (\rho+\eta+\mu+\delta_I)I \\ (\phi+\mu+\delta_H)H - \rho I \\ (\mu+\delta_P+\gamma)P - \eta I - \phi H \end{bmatrix} \tag{43}$$

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

$$R_0 = \frac{\beta(1-\theta)\Lambda}{(\mu+\alpha)K_2} \tag{44}$$

Where R_0 is the basic reproduction number of the Infected Humans respectively

2.2.6 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:

Let $K_1 = (\mu+\alpha), K_2 = (\rho+\eta+\mu+\delta_I), K_3 = (\phi+\mu+\delta_H), K_4 = (\mu+\delta_P+\gamma)$

$$J(E) = \begin{bmatrix} -K_1 & 0 & -\beta S^* & 0 & 0 & 0 \\ \alpha & -\mu & 0 & 0 & 0 & 0 \\ 0 & \alpha & \beta S^* - K_2 & 0 & 0 & 0 \\ 0 & 0 & \rho & -K_3 & 0 & 0 \\ 0 & 0 & \eta & \phi & -K_4 & 0 \\ 0 & 0 & 0 & 0 & \gamma & -\mu \end{bmatrix} \tag{45}$$

Using the Row echelon form to reduce the above Jacobian Matrix as used by Adedayo *et al.*, (2020).

From the leading diagonals we have

$$\left.\begin{aligned}\psi_1 &= -(\mu+\alpha)\\ \psi_2 &= -\mu\\ \psi_3 &= \frac{\beta\Lambda(1-\theta)-(\mu+\alpha)(\rho+\eta+\mu+\delta_I)}{(\mu+\alpha)}\\ \psi_4 &= -(\phi+\mu+\delta_H)\\ \psi_5 &= -(\mu+\delta_P+\gamma)\\ \psi_6 &= -\omega\end{aligned}\right\} \quad (46)$$

$$R_0 < 1 iff\ \psi_3 < 0 \quad (47)$$

Suppose $\psi_3 < 0$

$$\frac{\beta\Lambda(1-\theta)-(\mu+\alpha)(\rho+\eta+\mu+\delta_I)}{(\mu+\alpha)} < 0 \quad (48)$$

$$\beta\Lambda(1-\theta)-(\mu+\alpha)(\rho+\eta+\mu+\delta_I) < 0 \quad (49)$$

$$\frac{\beta\Lambda(1-\theta)}{(\mu+\alpha)(\rho+\eta+\mu+\delta_I)} - 1 < 0 \quad (50)$$

$$R_0 - 1 < 0 \quad (51)$$

$$R_0 < 1 \quad (52)$$

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.

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

Let $E^1 = (S, V, I, H, P, D) = (S^1, V^1, I^1, H^1, P^1, D^1)$ is the endemic equilibrium point.

$$(1-\theta)\Lambda - \beta S^{**} I^{**} - (\mu+\alpha) S^{**} = 0 \quad (53)$$

$$\theta\Lambda + \alpha S^{**} - \mu V^{**} = 0 \quad (54)$$

$$\beta S^{**} I^{**} - (\rho+\eta+\mu+\delta_I) I^{**} = 0 \quad (55)$$

$$\rho I^{**} - \left(\phi + \mu + \delta_H\right) H^{**} = 0 \tag{56}$$

$$\eta I^{**} + \phi H^{**} - \left(\mu + \delta_P + \gamma\right) P^{**} = 0 \tag{57}$$

$$\gamma P^{**} - \omega D^{**} = 0 \tag{58}$$

$$S^{**} = \frac{\left(\rho + \eta + \mu + \delta_I\right)}{\beta} \tag{59}$$

Putting equation (59) in equation (54) we have

$$V^{**} = \frac{\beta\theta\Lambda + \alpha\left(\rho + \eta + \mu + \delta_I\right)}{\mu\beta} \tag{60}$$

Adding equation (53) and (55)

$$\left(1-\theta\right)\Lambda - \left(\mu + \alpha\right) S^{**} - \left(\rho + \eta + \mu + \delta_I\right) I^{**} = 0 \tag{61}$$

putting equation (59) in equation (61)

$$I^{**} = \frac{\beta\left(1-\theta\right)\Lambda - \left(\mu + \alpha\right)\left(\rho + \eta + \mu + \delta_I\right)}{\beta\left(\rho + \eta + \mu + \delta_I\right)} \tag{62}$$

putting equation (62) into equation (56) we have

$$H^{**} = \frac{\rho\left(\beta\left(1-\theta\right)\Lambda - \left(\mu + \alpha\right)\left(\rho + \eta + \mu + \delta_I\right)\right)}{\beta\left(\rho + \eta + \mu + \delta_I\right)\left(\phi + \mu + \delta_H\right)} \tag{63}$$

putting equation (62) and (63) into equation (57) we have

$$P^{**} = \frac{\eta\left(\phi + \mu + \delta_H\right)\left(\beta\left(1-\theta\right)\Lambda - \left(\mu + \alpha\right)\left(\rho + \eta + \mu + \delta_I\right)\right) + \phi\rho\left(\beta\left(1-\theta\right)\Lambda - \left(\mu + \alpha\right)\left(\rho + \eta + \mu + \delta_I\right)\right)}{\beta\left(\rho + \eta + \mu + \delta_I\right)\left(\phi + \mu + \delta_H\right)\left(\mu + \delta_P + \gamma\right)} \tag{64}$$

And putting equation (64) into equation (59) we have

$$D^{**} = \frac{\gamma\left(\eta\left(\phi + \mu + \delta_H\right)\left(\beta\left(1-\theta\right)\Lambda - \left(\mu + \alpha\right)\left(\rho + \eta + \mu + \delta_I\right)\right) + \phi\rho\left(\beta\left(1-\theta\right)\Lambda - \left(\mu + \alpha\right)\left(\rho + \eta + \mu + \delta_I\right)\right)\right)}{\beta\omega\left(\rho + \eta + \mu + \delta_I\right)\left(\phi + \mu + \delta_H\right)\left(\mu + \delta_P + \gamma\right)}$$

(65)

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

$$\begin{pmatrix} S^{**} \\ V^{**} \\ I^{**} \\ H^{**} \\ P^{**} \\ D^{**} \end{pmatrix} = \begin{pmatrix} \frac{\left(\rho+\eta+\mu+\delta_I\right)}{\beta} \\ \frac{\beta\theta\Lambda+\alpha\left(\rho+\eta+\mu+\delta_I\right)}{\mu\beta} \\ \frac{\beta\left(1-\theta\right)\Lambda-\left(\mu+\alpha\right)\left(\rho+\eta+\mu+\delta_I\right)}{\beta\left(\rho+\eta+\mu+\delta_I\right)} \\ \frac{\rho\left(\beta\left(1-\theta\right)\Lambda-\left(\mu+\alpha\right)\left(\rho+\eta+\mu+\delta_I\right)\right)}{\beta\left(\rho+\eta+\mu+\delta_I\right)\left(\phi+\mu+\delta_H\right)} \\ \frac{\eta\left(\phi+\mu+\delta_H\right)\left(\beta\left(1-\theta\right)\Lambda-\left(\mu+\alpha\right)\left(\rho+\eta+\mu+\delta_I\right)\right)+\phi\rho\left(\beta\left(1-\theta\right)\Lambda-\left(\mu+\alpha\right)\left(\rho+\eta+\mu+\delta_I\right)\right)}{\beta\left(\rho+\eta+\mu+\delta_I\right)\left(\phi+\mu+\delta_H\right)\left(\mu+\delta_P+\gamma\right)} \\ \frac{\gamma\left(\eta\left(\phi+\mu+\delta_H\right)\left(\beta\left(1-\theta\right)\Lambda-\left(\mu+\alpha\right)\left(\rho+\eta+\mu+\delta_I\right)\right)+\phi\rho\left(\beta\left(1-\theta\right)\Lambda-\left(\mu+\alpha\right)\left(\rho+\eta+\mu+\delta_I\right)\right)\right)}{\beta\omega\left(\rho+\eta+\mu+\delta_I\right)\left(\phi+\mu+\delta_H\right)\left(\mu+\delta_P+\gamma\right)} \end{pmatrix} \quad (66)$$

3. Numerical Simulation

It is difficult to get a reliable data; we estimated the parameter value based on the available data from the Nigeria Centre for Disease Control (NCDC) and reliable literature. The estimates are clearly explained in the following sub-sections as shown in Table 4.1.

Table 1 Shows Initial Conditions for Each Plot and Parameters Value (NCDC, 2021).

Parameters and State Variables	Value	Source
$S(t)$	500	Manju & Archana (2011)
$V(t)$	600	Manju & Archana (2011)
$I(t)$	160	Manju & Archana (2011)
$H(t)$	200	Assumed
$P(t)$	300	Assumed
$D(t)$	105	Assumed
Λ	1000	Manju & Archana (2011)
μ	0.05	Assumed
δ_I	0.06	Assumed
δ_H	0.02	Assumed
δ_P	0.03	Assumed
β	0.0050	Assumed
θ	0.025	Assumed
γ	0.020	Assumed
ω	0.002	Assumed
α	0.025	Assumed
η	0.050	Assumed
ρ	0.025	Assumed
ϕ	0.030	Assumed

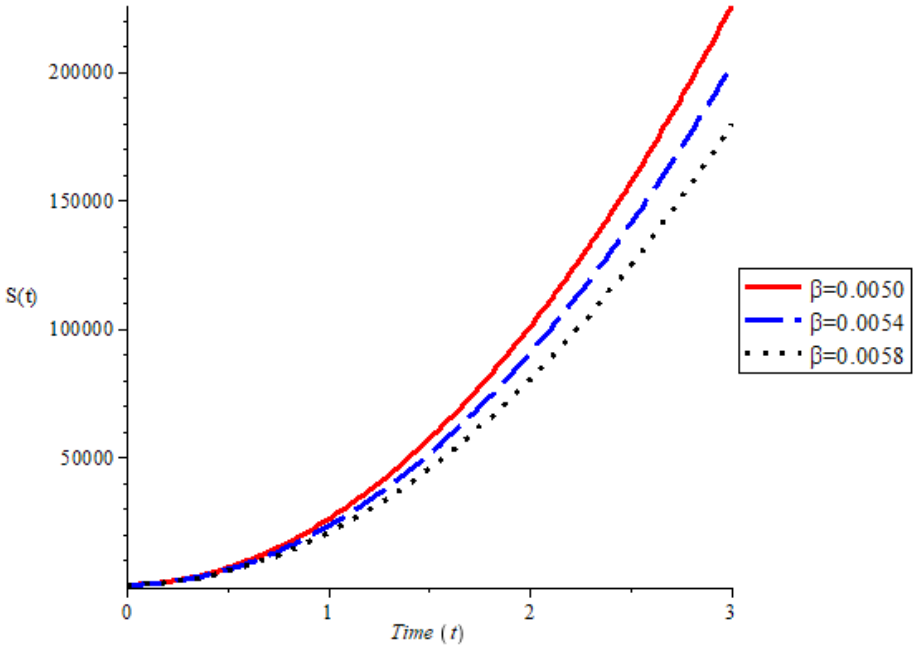

Figure 1: Graph of Susceptible Against Time for Different at the Contact rate.

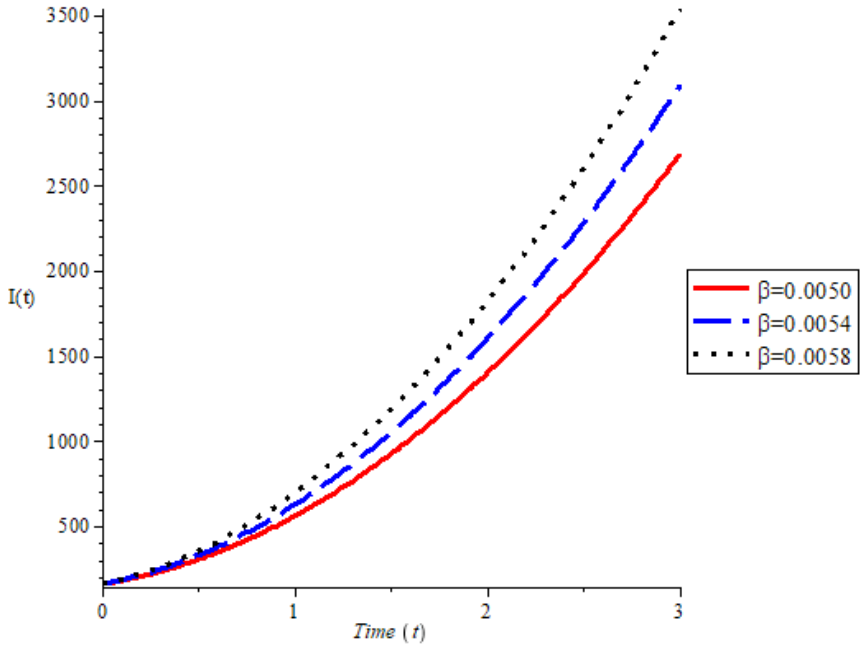

Figure 2: Graph of Infected Against Time for Different at the Contact rate

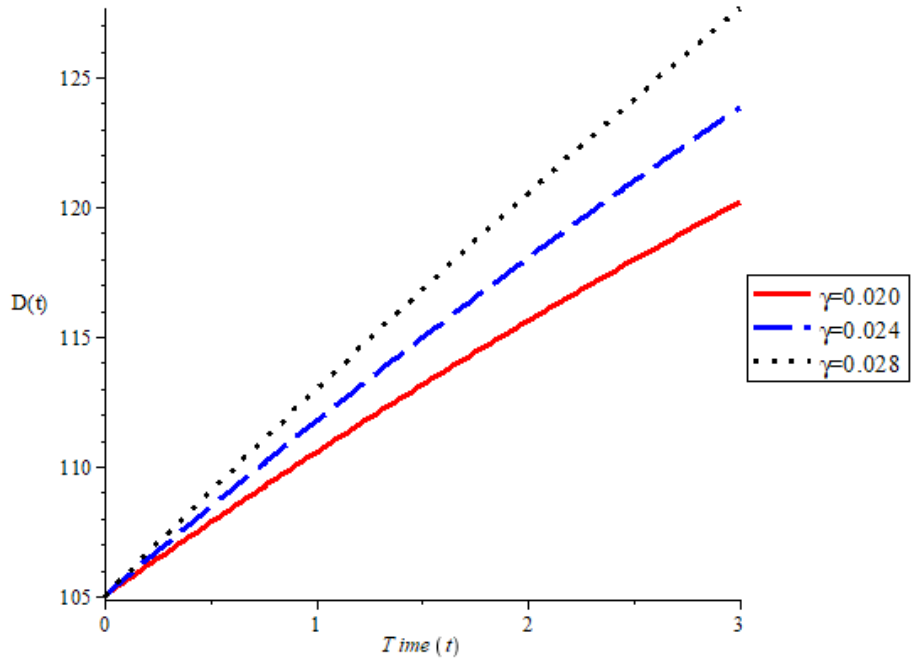

Figure 3: Graph of Death Individuals Against Time for Different at Rate at which Paralyzed individuals move to death class

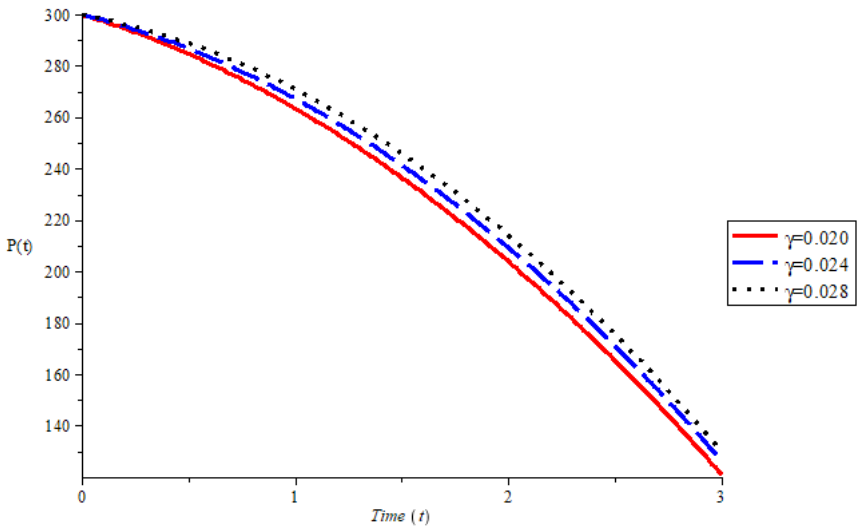

Figure 4: Graph of Paralyzed Individuals Against Time for Different at the Rate at which Paralyzed individuals move to Death

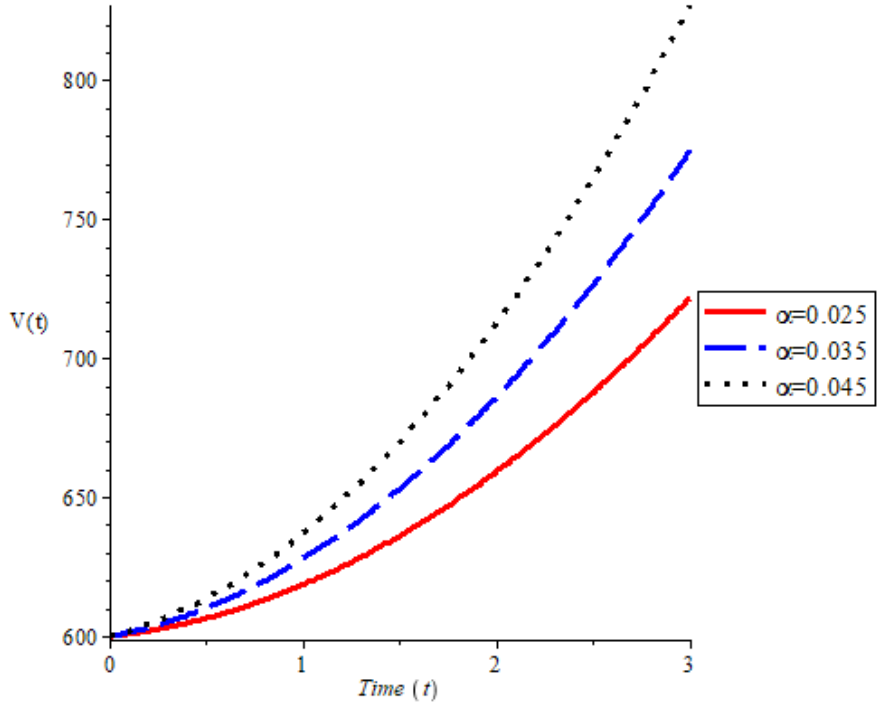

Figure 5: Graph of Vaccinated Individuals Against Time for Different at the Rate at which susceptible individuals move to vaccinated class

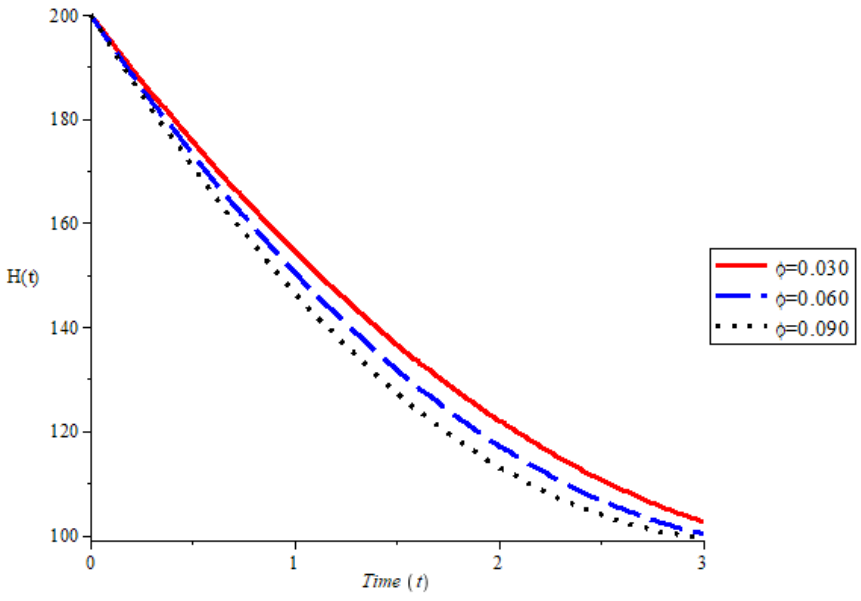

Figure 6: Graph of Hospitalized Individuals Against Time for Different at Rate at which hospitalized individuals move to Paralyzed class.

3.1 Discussion

Figure 1 is the graph of susceptible against time for different at the contact rate. The graph that the population susceptible increase as the at the contact decreases. Figure 2 is the graph of infected against time for different at the contact rate. The that the population infected increase as the at the contact rate also increases. Figure 3 is the graph of death individuals against time for different at Rate at which Paralyzed individuals move to death class. The graph shows that the population death individuals increase as the at the rate at which paralyzed individuals move to death also increases. Figure 4 is the graph of paralyzed Individuals against time for different at the rate at which paralyzed individuals move to death. The graph that the population paralyzed decrease as the at the rate at which paralyzed individuals move to death also decreases. Figure 5 is the graph of vaccinated Individuals against time for different at the rate at which susceptible individuals move to vaccinated class. The graph show that the population vaccinated individuals increase as the at the rate at which susceptible individuals move to vaccinated also increases. Figure 6 is the graph of hospitalized individuals against time for different at rate at which hospitalized individuals move to paralyzed class. The graph show that the population hospitalized individuals decrease as the at rate at which hospitalized individuals move to paralyzed also decreases.

3.2 Concluding Remarks

In this research work, we deterministic a mathematical model for transmission dynamics of Polio Infection incorporating Vaccination and hospitalization, with the aim of performing a perfect theoretical analysis of epidemiological meaningfulness. We derived the basic reproduction number R_0, of the model, and it was used to perform a 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 $R_0 > 1$. The result of the numerical simulation reveals that with 80% 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 80% vaccination coverage.

References

Adedayo, O.A**.** Akande, S.A. and Ugwu, U.C (2021), "A Mathematical Model of Tuberculosi (TB) with Respect to Drug Resistance to the First and Second Line of Treatment ". *Asian Journal of Mathematical Sciences (AJMS) Vol. 5 No. 4 doi*:10.22377/ajams.v6i1.400

Adedayo, O.A., Oyekunle, A.A., Ugwu, U.C., Akande, S.A., Oyedeji A.A. and Adebayo H.O. (2022), "A Mathematical Model for the Control on the Transmission Dynamics Classes COVID-19 Pandemic Containing Asymptomatic and Symptomatic Classes ". *Asian Journal of Mathematical Sciences (AJMS) Vol. 6 No. 1,* Available online here doi:10.22377/ajams.v6i1.414

Applications and Applied Mathematics: An International Journal. 6(2), 552 – 571.

Avendano L, Ferres M y Spencer E 2011 Virologıa Clınica (Santiago de Chile: Mediterraneo)

Biosciences.185, 89-109.

Bolarin, G., Omatola, I. U., Yusuf, A., Odo, C. E., Oguntolu, F. A. and Philip, M. A. (2019) Mathematical Modeling of Polio Virus Infection Incorporating Immigration and Vaccination. Applied Mathematics. 6(4), 65-72.

Farrington, C. P. (2003): On vaccine efficacy and reproduction numbers. Mathematical Gumel, A. B. and Moghadas, S. M. (2003): A qualitative study of vaccination model with Kribs-Zaleta, C. M. and Velasco-Hernandez, J. X. (2000): A simple vaccination model with

Levinson W 2006 Microbiologıa e Inmunologıa Medicas (Madrid: McGraw-Hill Interamericana de EspaÃśa S.L.)

Manju, A. and Archana, S. B. (2011): Modeling Spread of Polio with the Role of Vaccination.

Model. Bulletin of Mathematical Biology. 94, 1123-1148.

multiple endemic states. Mathematical Biosciences. 164(2), 183-200.

nonlinear incidence. Applied Mathematics and Computation. 143, 409-419.

Prats G 2013 Microbiolog´ıa y Parasitolog´ıa M´edica (Madrid: Editorial M´edica Panamericana)

Shors T 2009 Virus. Estudio molecular con orientaci´on Cl´ınica (Madrid: Editorial M´edica Panamericana)

Shulgin, B., Stone, L. and Agur, Z. (1998): Pulse vaccination strategy in the SIR epidemic

World Health Organization (2018). http://www.who.int/features/qa/07/en/