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Mathematical Modeling of Poliomyelitis with Control Measure

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Abstract

Poliomyelitis, also known as polio, is a contagious viral illness that predominantly impacts young children, leading to paralysis and, in severe instances, death. Despite worldwide initiatives aimed at elimination, the spread of the poliovirus persists in various areas, highlighting the need for robust vaccination strategies. This research employed a mathematical model to explore the dynamics of poliovirus transmission, integrating vaccination as a crucial method for disease control. The model was analyzed to determine the basic reproduction number (R_0) *and the stability properties of the disease-free and endemic equilibrium. Our findings demonstrated that the system achieves local asymptotic stability when the basic reproduction number is less than one, global asymptotic stability when it is exactly one, and maintains a stable endemic equilibrium when it is greater than one. Sensitivity analysis revealed critical parameters influencing the basic reproduction number, emphasizing the impact of vaccination coverage and disease transmission rates on polio dynamics. Numerical simulations further*

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elucidated the effectiveness of interventions such as reducing contact rates between susceptible and infected individuals and increasing the vaccination rate. Based on our results, we proposed recommendations to mitigate the polio burden, including enhanced vaccine availability, improved sanitation practices, and targeted healthcare interventions for vulnerable populations. This study contributes to the understanding of polio transmission dynamics and provides insights for optimizing control strategies towards global eradication efforts.

Keywords: Poliomyelitis, Mathematical modeling, Vaccination, Disease dynamics, Eradication strategies

INTRODUCTION

Poliomyelitis, commonly known as polio, is a highly infectious viral disease caused by the poliovirus, primarily affecting young children. Transmission primarily occurs through person-to-person contact via the fecal-oral route, although contaminated water and food can also spread the virus (CDC, 2023., GPEI, 2O21). Polio can result in a spectrum of outcomes, ranging from asymptomatic cases to severe paralysis, affecting approximately 1% of those infected (CDC, 2023., Alfaro-Murillo et al, 2020). Belonging to the Enterovirus genus within the Picornaviridae family, the poliovirus consists of three serotypes: P1, P2, and P3. It is a small, non-enveloped virus with a single-stranded RNA genome. Following initial infection in the gastrointestinal tract, the virus can disseminate to lymphatic tissues and the bloodstream. In some instances, it crosses the blood-brain barrier to infect motor neurons in the spinal cord and brainstem, causing acute flaccid paralysis, characteristic of paralytic poliomyelitis (WHO, 2023). Historically, polio has triggered widespread concern and significant morbidity, particularly during the mid-20th century in developed nations. The introduction of polio vaccines, notably the inactivated poliovirus vaccine (IPV) and the oral poliovirus vaccine (OPV), has resulted in a substantial decline in global polio cases (WHO, 2022., WHO, 2019) . Despite progress, polio persists in some regions due to challenges in vaccination coverage, surveillance, and obstacles related to conflict and accessibility.

The clinical presentation of polio varies, depending on the viral strain and individual immune response. Non-paralytic polio, also known as abortive poliomyelitis, typically causes symptoms such as fever, sore throat, headache, nausea, vomiting, and abdominal pain, which usually resolve within a week (Alfaro-Murillo et al, 20200). Paralytic poliomyelitis presents with sudden onset muscle weakness or paralysis, often asymmetrically affecting the legs predominantly. Diagnosis relies on clinical symptoms, history of exposure, and laboratory confirmation through viral isolation or detection of viral RNA in stool or cerebrospinal fluid. Serological testing for poliovirus antibodies aids in determining vaccination status and previous exposure (WHO, 20230).The global initiative to eradicate polio, led by the Global Polio Eradication Initiative (GPEI), involves collaboration among national governments, the World Health Organization (WHO), Rotary International, the U.S. Centers for Disease Control and Prevention (CDC), and UNICEF. Strategies include routine immunization with OPV or IPV, supplemental vaccination campaigns in high-risk areas, and robust surveillance systems to promptly detect and respond to outbreaks (WHO, 2022). While significant strides have been made, achieving a polio-free world requires sustained efforts in vaccination, research, and healthcare infrastructure development. The control of poliomyelitis centers largely around vaccination efforts aimed at achieving global eradication of the disease. Two main types of vaccines are pivotal in these efforts: the inactivated poliovirus vaccine (IPV) and the oral poliovirus vaccine (OPV). IPV, administered via injection, contains killed poliovirus strains of all three serotypes (P1, P2, and P3) and is highly effective in inducing immunity (CDC, 2022). OPV, administered orally, contains live attenuated poliovirus strains and offers the advantage of promoting intestinal immunity, aiding in interrupting transmission in communities with

poor sanitation. Both vaccines have been integral to reducing polio cases globally, with OPV particularly critical in mass immunization campaigns due to its ease of administration and ability to confer immunity through herd immunity (Efforts to control polio also involve surveillance to detect outbreaks early and immunization campaigns targeting high-risk populations. The Global Polio Eradication Initiative (GPEI) coordinates these efforts, focusing on achieving high vaccination coverage rates worldwide and ensuring that no child is left unvaccinated. Continued research into new vaccine formulations, improvements in delivery strategies, and addressing vaccine hesitancy are ongoing priorities in the quest to eliminate polio as a public health threat globally (Alfaro-Murillo et al, 2020).

There is currently no cure for poliomyelitis because the virus attacks the nervous system, causing irreversible damage, and there are limitations in effectively targeting and eliminating all traces of the virus from the body (Aylward, 2006), but various treatments aim to alleviate symptoms, hasten recovery, and prevent complications. Contemporary approaches focus on providing relief through measures such as antibiotics to thwart infections in weakened muscles, analgesics for pain management, moderate exercise, and a balanced diet. Long-term rehabilitation, encompassing occupational therapy, physical therapy, braces, corrective footwear, and occasionally orthopedic surgery, is often necessary for effective polio treatment (Daniel and Robbins, 1997). To aid breathing, portable ventilators may be essential. Historically, during acute polio infections, a noninvasive, negative-pressure ventilator known as an iron lung was employed to artificially sustain respiration until independence was regained (typically within one to two weeks). In the present day, many individuals enduring permanent respiratory paralysis utilize modern jacket-type negative-pressure ventilators worn over the chest and abdomen (Goldberg, 2002). Several researchers have studied mathematical modeling of infectious diseases.

Hsu and Yang (2023) studied the mathematical modeling of poliomyelitis transmission dynamics and control. They provided a comprehensive review of mathematical models used to analyze poliovirus transmission and control strategies, including compartmental and stochastic models. Their review highlighted the application of these models in assessing vaccination strategies, surveillance systems, and outbreak responses, synthesizing recent advancements and discussing their implications for global polio eradication efforts. Duintjer et al. (2016) focused on an economic analysis of poliovirus risk management policies for 2013–2052, using mathematical modeling to evaluate various policy options such as vaccination strategies and surveillance systems. Their research emphasized the cost-effectiveness of different interventions and provided insights into optimal resource allocation and policy decisions for sustaining polio eradication efforts. Kalkowska et al. (2015) developed and applied a mathematical model to manage imported type 1 wild poliovirus in Israel, evaluating the effectiveness of vaccination and surveillance strategies in preventing outbreaks and controlling transmission. Their study underscored the importance of rapid response and targeted interventions. Brouwe et al. (2018) investigated the epidemiology of a silent polio outbreak in Rahat, Israel, using environmental surveillance data. Their modeling study identified factors contributing to the outbreak and assessed the effectiveness of environmental surveillance in detecting and responding to poliovirus circulation. Brouwer et al. (2017) optimized silent environmental surveillance strategies for detecting poliovirus in southern Israel, evaluating the sensitivity and cost-effectiveness of various surveillance approaches. Their research highlighted the need for continuous monitoring and adaptive surveillance strategies to achieve and maintain polio eradication. Hsu and Yang (2023) studied the mathematical modeling of poliomyelitis transmission dynamics and control. They provided a comprehensive review of mathematical models used to analyze poliovirus transmission and control strategies, including compartmental and stochastic models. Their review highlighted

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MATERIALS AND METHODS

Model Formulation.

The human population at time t, given by $N(t)$ is sub-divided into five(5) mutually exclusive compartments of Susceptible humans $S(t)$, Vaccinated humans $V(t)$, Exposed humans $E(t)$, Infected humans $I(t)$ and Recovered humans $R(t)$. $N_h(t) = S(t) + V(t) + E(t) + I(t) + R(t)$ The recruitment rate of humans into the susceptible population is at the rate Λ. We denote β as the effective contact rate of humans with the probability of been infected per contact with the polio virus in the feces of the infected humans. The rate at which the susceptible individuals are vaccinated from polio is denoted as ϕ and the vaccinated individuals progress into the recovered class at the rate of ρ. The exposed individuals to polio becomes infected at the rate of θ. The rate at which the infected individuals recover is denoted as τ. The natural death rate of the individuals in the population is μ while the disease induced death rate is denoted as δ.

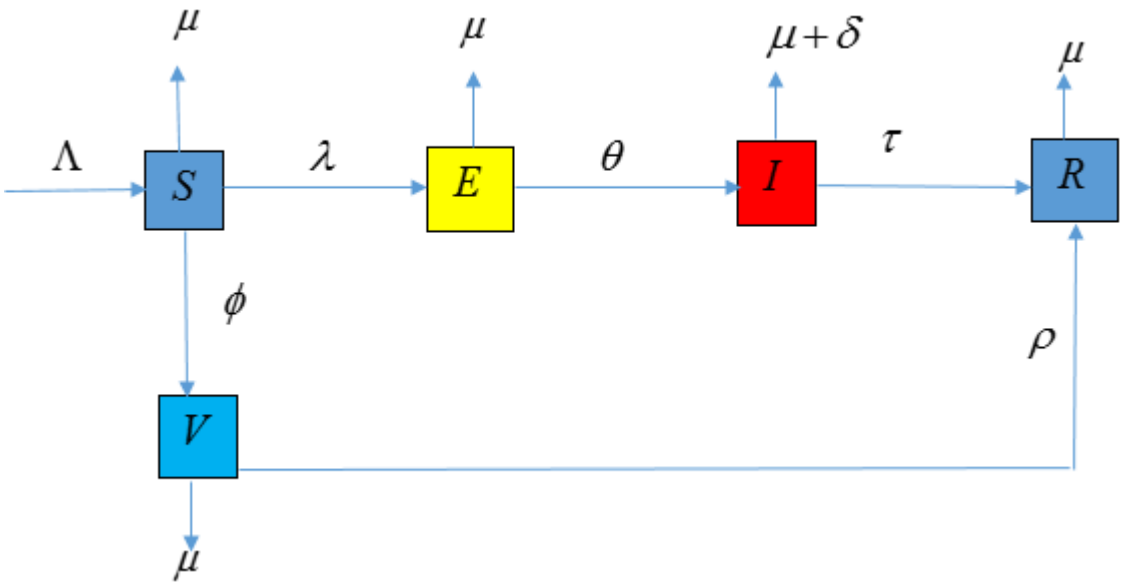


Fig. 1: Schematic diagram of the Poliomyelitis model

Assumptions used in the formulation of the model

- Recovery of infected individuals is possible even though it is minimal.
- Infected individuals can be vaccinated and their disease conditions are managed in the vaccination center.
- The vaccinated individuals recovered due to effective vaccination.
- Every individual is recruited into the population through birth.

Table 1: Variable and Parameters description

Variable	**Description**
S	Susceptible individuals
V	Vaccinated population
E	Exposed population
I	Infected Humans
R	Recovered Humans
Parameter Description	
Λ	Recruitment level of humans
μ	Natural death rate of humans
β	Contact rate of susceptible and infected humans
ϕ	Vaccination rate of susceptible humans
θ	Progression rate from exposed population to infected class.
δ	Disease induced death rate of infected humans
ρ	Recovery rate of vaccinated humans.
τ	Recovery rate of infected individuals.

Model Equations

Based on the model description provided earlier, the differential equations that represent the transmission dynamics of poliomyelitis in the population are formulated as follows:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - (\lambda + \phi + \mu)S, \\
\frac{dV}{dt} &= \phi S - (\rho + \mu)V, \\
\frac{dE}{dt} &= \lambda S - (\theta + \mu)E, \\
\frac{dI}{dt} &= \theta E - (\tau + \delta + \mu)I, \\
\frac{dR}{dt} &= \rho V + \tau I - \mu R.
\end{aligned} \tag{1}$$

The force of infection of the Poliomyelitis model in (1) is given as:

$$\lambda = \frac{\beta I}{N},$$

Let $K_1 = (\rho + \mu)$, $K_2 = (\theta + \mu)$, $K_3 = (\tau + \delta + \mu)$

Invariant region of the Poliomyelitis model

The solution set of the proposed Poliomyelitis model are feasible for all t > 0, if they enter the invariant region D, which is given by:

$$D = \left\{ (S, V, E, I, R) : S > 0, V > 0, E > 0, I > 0, R > 0, N < \frac{\Lambda}{\mu} \right\} \tag{2}$$

Proof

The total population of the humans in the Poliomyelitis model is given as

$$N(t) = S(t) + V(t) + E(t) + I(t) + R(t) \tag{3}$$

The sum of the differential equations is

$$N(t) = S'(t) + V'(t) + E'(t) + I'(t) + R'(t)$$

On evaluating the algebraic terms, we obtain

$$N'(t) = \Lambda - (S + V + E + I + R)\mu - \delta I$$

$$N'(t) = \Lambda - N\mu - \delta I$$

$$\frac{dN}{dt} \leq \Lambda - \mu N \tag{4}$$

Solving the above (4) equation, we obtained

$$N(t) \leq \frac{\Lambda}{\mu} + \left(N(0) - \frac{\Lambda}{\mu} \right) e^{-\mu t} \tag{5}$$

Using Birkhoff and Rota's theorem on the inequality, we obtain

$$0 \leq N \leq \frac{\Lambda}{\mu} \text{ as } t \to \infty \tag{6}$$

Therefore, D is a positively invariant set under the flow governed by model (2), ensuring that no solution trajectory exits through the boundary of D. Consequently, within this region, the poliomyelitis-only model can be deemed both epidemiologically and mathematically well-defined.

Positivity of solution of the Poliomyelitis model

To establish the epidemiological and mathematical integrity of the poliomyelitis model within the defined feasible region D, it is essential to demonstrate that all state variables remain nonnegative throughout the entire duration. The feasible region D is characterized by:

$$D=\left\{\left(S,V,E,I,R\right)\in R_{+}^{5}:\left(S+V+E+I+R\right)\le N\right\} \quad (7)$$

This is done by considering,

$$\left\{\left(S,V,E,I,R\right)\ge 0\in R_{+}^{5}\right\}$$

Lemma 1:

Suppose the initial data for the model (1) be $\left(S,V,E,I,R\right)>0$. Then the solution set, $\left(S,V,E,I,R\right)$ of the model (1) are positive for all time $t>0$

Proof

Let $t=\sup\left\{t>0:S>0,V>0,E>0,I>0,R>0\in[0,t]\right\}$. Thus $t>0.$

We have from the first equation that

$$\frac{dS}{dt}=\Lambda-\left(\lambda+\phi+\mu\right)S$$

$$\frac{dS}{dt}\ge-\left(\lambda+\phi+\mu\right)S$$

This can also be written as

$$\int\frac{dS}{S}\ge-\int\left(\lambda+\phi+\mu\right)dt$$

We obtained:

$$\ln S\ge-\left(\lambda+\phi+\mu\right)t+C$$

$$S(t)\ge Ce^{-\left(\lambda+\phi+\mu\right)t} \quad (8)$$

Applying the initial condition; when $t=0,\ S(0)=C$

Therefore, $S(t)\ge S(0)e^{-(\lambda+\phi+\mu)t}\ge 0$ since $\left(\lambda+\mu\right)>0$

Similarly, it can be demonstrated that $V,E,I,R>0$

Asymptotic stability analysis of the disease-free equilibrium of the Poliomyelitis model

The point at which there is no infection present, representing the absence of the disease, is referred to as the disease-free equilibrium (DFE) point, denoted as

$$\eta_0=\left\{S^*,V^*,E^*,I^*,R^*\right\}=\left\{\frac{\Lambda}{\mu},0,0,0,0\right\} \quad (9)$$

Basic Reproduction Number of the Poliomyelitis Model

The basic reproduction number (R_0) for poliomyelitis-infected individuals represents the average number of secondary infections generated by a single infectious individual over their entire period of infectiousness in a fully susceptible population (Agbata et al, 2024). This value is determined using the next generation operator method applied to the dynamic system (1). Therefore, we obtain our R_0 as follows

$R_0=\rho\left(FV^{-1}\right)$ where ρ is the dominant eigenvalue of FV^{-1}

$$F=\begin{bmatrix}0 & \dfrac{\beta K_1}{K_1+\phi}\\ 0 & 0\end{bmatrix},\ V=\begin{bmatrix}K_2 & 0\\ -\theta & K_3\end{bmatrix},$$

$$V^{-1}=\begin{bmatrix}\dfrac{K_3}{K_2K_3} & 0\\ \dfrac{\theta}{K_2K_3} & \dfrac{K_2}{K_2K_3}\end{bmatrix}$$

Simplifying the elements of the matrix: we obtained,

$$V^{-1}=\begin{bmatrix}\dfrac{1}{K_2} & 0\\ \dfrac{\theta}{K_2K_3} & \dfrac{1}{K_3}\end{bmatrix},\ FV^{-1}=\begin{bmatrix}\dfrac{\beta K_1\theta}{(K_1+\phi)K_2K_3} & \dfrac{\beta K_1}{(K_1+\phi)K_3}\\ 0 & 0\end{bmatrix}$$

Therefore, the greatest eigenvalue of FV^{-1} the basic reproduction number of the Poliomyelitis only model is

$$R_0=\frac{\beta K_1\theta}{(K_1+\phi)K_2K_3}$$

$$R_0=\frac{\beta(\rho+\mu)\theta}{((\rho+\mu)+\phi)(\theta+\mu)(\tau+\delta+\mu)} \tag{10}$$

Local Asymptotic Stability of the DFE of the Poliomyelitis Model

Theorem 1

The disease-free equilibrium point of the Poliomyelitis only is locally asymptotically stable (LAS) if $R_0<1$*, and unstable if* $R_0>1$.

Proof

Using Jacobian matrix to obtain the local stability of the disease free equilibrium point

$$J(\eta_0)=\begin{bmatrix}-(\phi+\mu) & 0 & 0 & -\dfrac{\beta K_1}{K_1+\phi} & 0\\ \phi & -K_1 & 0 & 0 & 0\\ 0 & 0 & -K_2 & \dfrac{\beta K_1}{K_1+\phi} & 0\\ 0 & 0 & \theta & -K_3 & 0\\ 0 & \rho & 0 & \tau & -\mu\end{bmatrix}$$

$$J_1(\eta_0)=\begin{bmatrix}-(\phi+\mu) & 0 & 0 & -\dfrac{\beta K_1}{K_1+\phi} & 0\\ \phi & -K_1 & 0 & 0 & 0\\ 0 & 0 & -K_2 & \dfrac{\beta K_1}{K_1+\phi} & 0\\ 0 & 0 & \theta & -K_3 & 0\\ 0 & \rho & 0 & \tau & -\mu\end{bmatrix}$$

Applying rows and Column reduction to the matrix, we obtain

$$J_2(\eta_0)=\begin{bmatrix} -K_2 & \dfrac{\beta K_1}{K_1+\phi} \\ \theta & -K_3 \end{bmatrix}$$

The characteristics polynomial of $J_2(\eta_0)$ is

$$\lambda^2+(K_3+K_2)\lambda+\frac{K_1\beta\theta(1-R_0)}{K_1+\phi}$$

Applying Routh-Hurwitz criterion to the Characteristics polynomial, we have that

$$(1-R_0)>0 \tag{10}$$

$$\Rightarrow R_0<1$$

Thus the DFE point of the Poliomyelitis only model is locally asymptotically stable.

Global Asymptotic Stability of the Disease-free equilibrium point of the Poliomyelitis Model.

To examine the global stability of the disease-free equilibrium, we employ the approach developed by Castillo-Chavez and Song. To accomplish this, we express the equation pertaining to the uninfected class as follows:

$$\frac{dX}{dt}=F(X,Z) \tag{11}$$

And we re-write the equation in the infected class as

$$\frac{dZ}{dt}=G(X,Z) \tag{12}$$

Where $X=(S,V,R)\in R^3_+$ represents the uninfected population and

$Z=(E,I,R)\in R^3_+$ represents the infected population

$\varepsilon_0=(X^*,0)$ represent the disease free equilibrium of the system, and it globally asymptotically stable if it satisfies the following conditions:

$H_1:\dfrac{dX}{dt}=F(X^*,0)$, X^* is globally asymptotically stable

$H_2:\dfrac{dZ}{dt}=D_Z G(X^*,0)Z-\hat{G}(X,Z)$

$\hat{G}(X,Z)\geq 0$ for all $(X,Z)\in D$ and where $D_Z G(X^*,0)$ is an M- matrix (i.e the diagonal elements are no-negative and it is also the Jacobian of $\hat{G}(X,Z)\geq 0$ evaluated at $(X^*,0)$.

If the system fulfills the condition stated above, then the following theorem applies.

Theorem 2

The equilibrium point $\eta_0=(X^*,0)$. *is globally asymptotically stable if* $R_0\leq 1$ and H_1,H_2 are satisfied.

$$F(X,Z)=\begin{bmatrix} \Lambda-(\lambda+\phi+\mu)S \\ \phi S-(\rho+\mu)V \\ \rho V+\tau I-\mu R \end{bmatrix},\ G(X,Z)=\begin{bmatrix} \dfrac{\beta I}{N}S-(\theta+\mu)E \\ \theta E-(\tau+\delta+\mu)I \end{bmatrix}$$

At disease free equilibrium,

H_1:

$$\left.\begin{aligned}\frac{dS}{dt} &= \Lambda-(\phi+\mu)S\\ \frac{dV}{dt} &= \phi S-\mu V\\ \frac{dR}{dt} &= 0\end{aligned}\right\}$$

H_2:

$$D_z G(X^*,0)Z=\begin{bmatrix}\beta & -(\theta+\mu)\\ \theta & -(\tau+\delta+\mu)\end{bmatrix}\begin{bmatrix}E\\ I\end{bmatrix}=\begin{bmatrix}\beta I-(\theta+\mu)E\\ \theta E-(\tau+\delta+\mu)I\end{bmatrix}$$

$$\hat{G}(X,Z)=D_Z G(X^*,0)Z-G(X,Z)$$

$$\hat{G}(X,Z)=\begin{bmatrix}\beta\left(1-\frac{S}{N}\right)\\ 0\end{bmatrix}$$

Clearly, $1\geq\frac{S}{N}$ this implies that $\hat{G}(X,Z)\geq 0$.

Therefore, it follows that the disease free equilibrium of the Poliomyelitis only model is globally asymptotically stable.

Endemic Equilibrium Point of the Poliomyelitis Model

The endemic equilibrium point is the steady state where there is persistence or prevalence of a disease in the population.

Theorem 3

The endemic equilibrium point of the Poliomyelitis model in (1) is stable if $R_0>1$ *and unstable if* $R_0<1$

Proof

To obtain the endemic equilibrium we set the RHS of the differential equations in (1) to zero and solve for the state variables.

Thus, at the endemic equilibrium point,

$$\frac{dS}{dt}=\frac{dV}{dt}=\frac{dE}{dt}=\frac{dI}{dt}=\frac{dR}{dt}=0. \qquad (13)$$

Let $\eta^{**}=\left(S^{**},V^{**},E^{**},I^{**},R^{**}\right)$ be the endemic equilibrium point.

We have that,

$$S^{**}=\frac{\Lambda}{\lambda^{**}+\phi+\mu}$$

$$V^{**}=\frac{\Lambda\phi}{\left(\lambda^{**}+\phi+\mu\right)K_1}$$

$$E^{**}=\frac{\Lambda\lambda^{**}}{\left(\lambda^{**}+\phi+\mu\right)K_2}$$

$$I^{**}=\frac{\Lambda\lambda^{**}\theta}{\left(\lambda^{**}+\phi+\mu\right)K_2K_3}$$

$$R^{**} = \frac{\Lambda\left(K_1\lambda^{**}\tau\theta + K_2K_3\phi\rho\right)}{\left(\lambda^{**}+\phi+\mu\right)K_2K_3K_1\mu}$$

Substituting them into the force of infection, $\lambda^{**} = \frac{\beta I^{**}}{N^{**}}$, we obtained the following:

$\lambda^{**}\left(\left(K_1K_3\mu + K_1\mu\theta + K_1\tau\theta\right)\lambda^{**} + \left(K_2K_3\phi\rho + \left(1-R_0\right)\right)\right) = 0$ and $\lambda^{**} = 0$ denotes the disease free equilibrium point of the Poliomyelitis model, thus at the endemic equilibrium point $\lambda^{**} \neq 0$.

$$\Rightarrow \lambda^{**} = \frac{\left(K_2K_3\phi\rho + \left(R_0 - 1\right)\right)}{\left(K_1K_3\mu + K_1\mu\theta + K_1\tau\theta\right)} \tag{14}$$

Thus for λ^{**} to be positive at the endemic equilibrium point, $R_0 - 1 > 0$.

$\Rightarrow R_0 > 1$ and the endemic equilibrium point is stable.

2.10 Sensitivity Analysis of the Poliomyelitis Model

Sensitivity analysis is conducted to identify parameters that influence both the propagation and containment of an infection within a population [16]. The sensitivity index of the reproduction number for the poliomyelitis model concerning a parameter (p) is expressed as follows:

$$\Im_p^{R_0} = \frac{\partial R_0}{\partial p} \times \frac{p}{R_0} \tag{15}$$

Given that

$$R_0 = \frac{\beta K_1\theta}{\left(K_1+\phi\right)K_2K_3}$$

$$R_0 = \frac{\beta\left(\rho+\mu\right)\theta}{\left(\rho+\mu+\phi\right)\left(\theta+\mu\right)\left(\tau+\delta+\mu\right)}$$

$$\Im_\beta^{R_0} = 1.0000$$

$$\Im_\theta^{R_0} = \frac{\mu}{\theta+\mu} = \frac{0.5}{0.9+0.5} = 0.3571$$

$$\Im_\mu^{R_0} = -2\frac{\left(\begin{array}{l}\mu^3+(\phi/2+2\rho+\tau/2+\delta/2+\theta/2)\mu^2\\ +\rho(\phi+\rho+\tau+\delta+\theta)\mu+(\tau/2+\delta/2+\theta/2)\rho^2\\ +1/2\phi(\tau+\delta+\theta)\rho-1/2\phi\theta(\tau+\delta)\end{array}\right)\mu}{(\rho+\mu+\phi)(\theta+\mu)(\tau+\delta+\mu)(\rho+\mu)}$$

$$\Im_\mu^{R_0} = -2\frac{\begin{array}{l}(0.5^3+(0.3+2(0.34)+0.115+0.3+0.45)0.5^2\\ +0.34(0.6+0.34+0.23+0.6+0.9)0.5+(0.115+0.3+0.45)0.34^2\\ +0.3(1.73)0.34-0.3(1.1)0.6)0.5\end{array}}{(1.44)(1.4)(1.33)(0.84)} = -0.4850$$

$$\Im_\tau^{R_0} = -\frac{\tau}{\tau+\delta+\mu} = -\frac{0.23}{0.23+0.6+0.5} = -0.1729$$

$$\Im_\delta^{R_0} = -\frac{\delta}{\tau+\delta+\mu} = -\frac{0.6}{0.23+0.6+0.5} = -0.4511$$

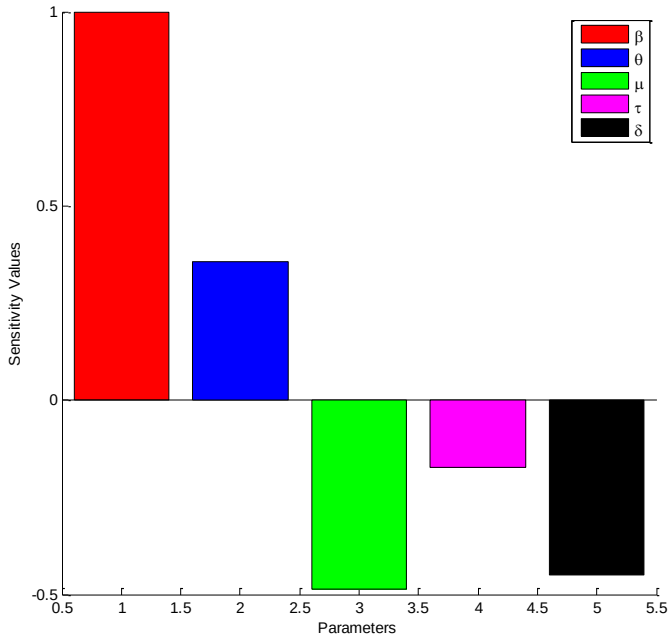


Figure 2 Bar chat of Poliomyelitis sensitivity Indices

Interpretation of the Poliomyelitis Sensitivity Analysis

From the sensitivity analysis above, it is observed that the parameters like β, θ which are the contact rates of susceptible and infected humans and the progression rate from exposed to infected classes with positive sensitivity indices enhances the spread of Poliomyelitis within the human population.

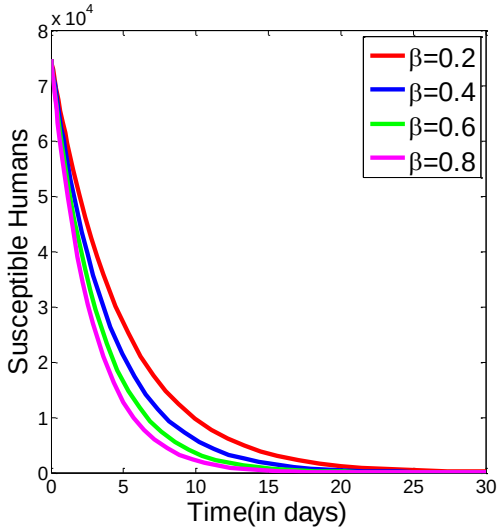
Conversely, the parameters τ, δ, μ which are the rate of recovery of infected humans, the natural death rate and the disease induced death rate of infected humans with negative sensitivity indices reduces the prevalence of Poliomyelitis within the human population.

Table 2. Parameter Values and Sources

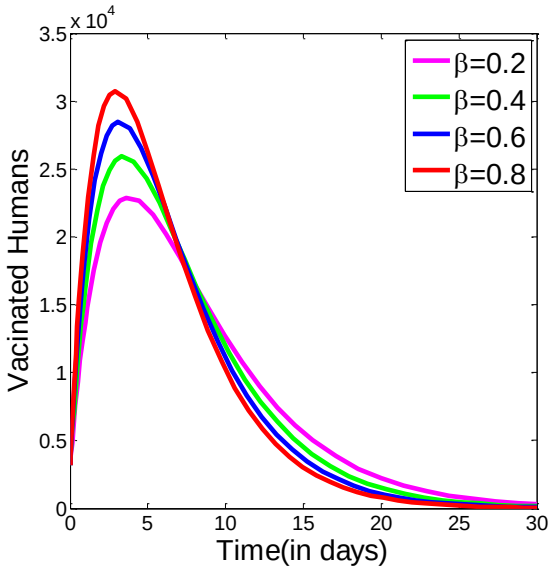
Parameter	Value	Source
Λ	1000	Xuan et al, 2022
β	0.002	Xuan et al, 2022
ρ	0.34	Estimated
ϕ	0.6	Xuan et al, 2022
μ	0.5	Xuan et al, 2022
τ	0.23	Estimated
θ	0.9	Bunimovich-Mendrazitsk & Stone, (2005)
δ	0.6	Assumed

Discussions and Numerical Simulations

In this segment, we present the graphs derived from our numerical simulation. The initial values of the state variables used for our numerical simulations are



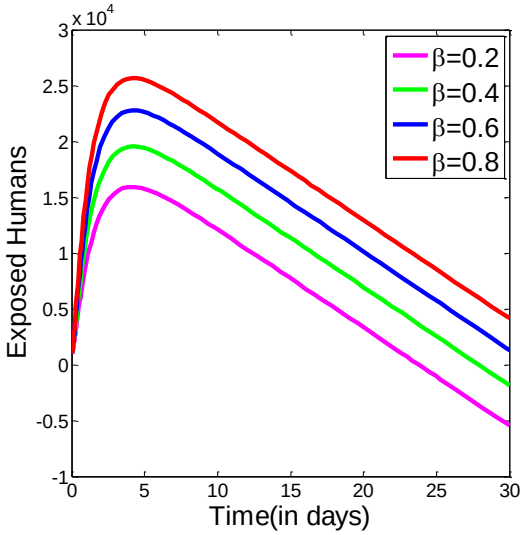
a. Effect of varying β on the susceptible class



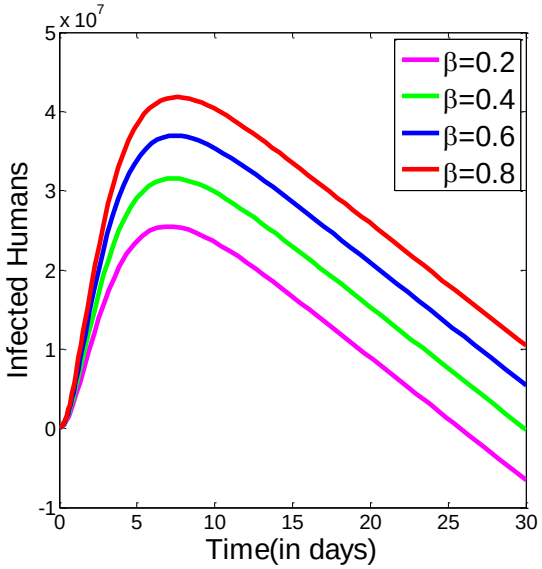
b. Effect of varying β on the Exposed class

Figure 3

From the graph in figure **3a**, we observed an increase in the population of the susceptible humans due to the effect of the vaccination strategy implemented into the population. This implies that with time, less individuals becomes susceptible to Polio if vaccination of susceptible humans can be fully implemented and adhered to. We also observed from the graph in figure **3b** an initial increase in the population of the vaccinated humans due to the influx of individuals from the susceptible humans. The later decrease observed in the vaccinated population is due to the fact that the effective vaccines administered to the susceptible humans ultimately leads to their recovery from polio. Hence the government should improve its commitment the provision of viable and potent vaccines so as to reduce the burden of polio within the human population.



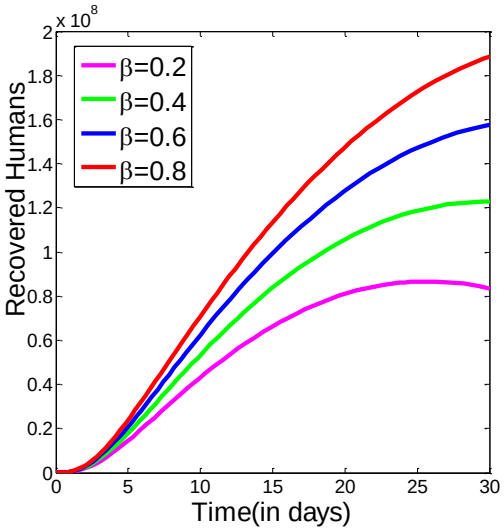
a. Effect of varying β on the Exposed Humans



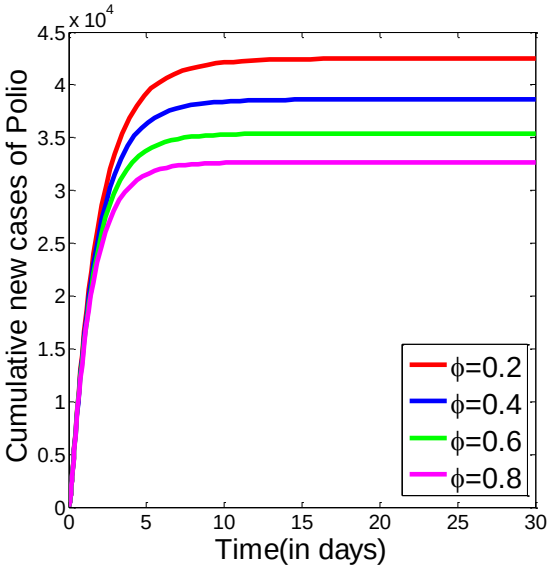
b. Effect of varying β on the Infected Humans

Figure 4

We observed from figure **4a** an initial increase in the population of the exposed humans to polio due the influx of humans who might at some certain times refuse to be vaccinated against the deadly disease. A later decrease is observed due to the high infectiousness of Polio which leads to their progression into the infected class.



a. Effect of varying β on the Cumulative new cases of polio



b. Effect if varying ϕ on the Recovered class

Figure 5

From figure **5a**, we observed that an increase in the rate of vaccination leads to the rapid recovery of individuals from polio within the human population. This can also be seen in figure **5b**, that an increase in the rate of vaccination of the susceptible humans leads to a decrease in the cumulative new cases of Polio.

CONCLUSION

In this study, we developed a mathematical model to explore the dynamics of Poliomyelitis transmission, incorporating vaccination as a means of control. We determined the basic reproduction number R_0 of the Poliomyelitis model, demonstrating that the system achieves local asymptotic stability when $R_0 < 1$, global asymptotic stability when $R_0 \leq 1$, and maintains a stable endemic equilibrium when $R_0 > 1$. Through sensitivity analysis, we assessed the impact of various parameters on the basic reproduction number. We found that parameters such as β, θ, which are the parameters that enhance the spread and burden of Poliomyelitis, exhibited positive sensitivity indices, thereby increasing the burden of the disease. Conversely, parameters such as τ, δ, μ, with negative sensitivity indices, mitigated Poliomyelitis transmission. Additionally, we conducted numerical simulations, varying parameters such as β, and ϕ, denoting the contact rates of susceptible humans and infected humans, and the rate of vaccination of susceptible humans. Our simulations revealed that reducing the contact between susceptible and infected individuals, and augmenting the treatment rate of infected individuals, substantially alleviate the Poliomyelitis burden within the human population. Based on our research findings, we recommend several strategies to mitigate the burden of Poliomyelitis. First, the government should enhance its commitment to providing effective vaccines to lower Polio incidence. Second, improved sewage disposal practices, particularly for feces, should be prioritized to minimize transmission through the

fecal-oral route. Lastly, there should be a strong emphasis on postnatal care for nursing mothers and newborns to reduce the risk of Polio infection in infants.

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Declaration of competing interests
The authors declare that they have no competing interests

Ethics Approval
Not Applicable

Availability of data.
The data applied in this study are referenced and given in table 2 above.

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