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Mathematical Analysis of Vaccination and Immunity Dynamics in Polio Eradication Efforts in Nigeria

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Abstract

Polio eradication efforts in Nigeria have seen significant progress, marked by an ongoing commitment to vaccination programs and public health initiatives. This article explores the mathematical modeling of polio transmission dynamics, with a focus on the impact of vaccination and immunity on disease control. A system of differential equations is developed to represent the spread of polio and the role of vaccination strategies. The model accounts for susceptible, vaccinated, infectious, and recovered populations, as well as the waning of vaccine-induced immunity. The analysis provides insights into the threshold conditions required for polio eradication and the implications of vaccination coverage, immunity waning, and population dynamics. The results suggest that achieving and sustaining high vaccination coverage remains crucial for eliminating polio, alongside addressing vaccine hesitancy and improving immunization strategies.

Keywords: Mathematics, Vaccination, Nigeria

1. Introduction

Polio, an infectious disease caused by the poliovirus, has been a global public health challenge, particularly in countries with limited access to healthcare and vaccination services. In Nigeria, concerted efforts have been made to eradicate polio through mass immunization campaigns, routine vaccination programs, and surveillance for acute flaccid paralysis (Ghinai *et al*., 2013; Etsano *et al*., 2017) [5, 3]. The implementation of oral polio vaccines (OPV) and inactivated polio vaccines (IPV) has played a central role in reducing polio incidence (Wiysonge *et al*., 2016). However, achieving complete eradication requires addressing factors such as vaccine hesitancy, population immunity dynamics, and the risk of vaccine-derived poliovirus (Grassly *et al*., 2006; Ado *et al*., 2014) [6, 1].

Mathematical modeling serves as a valuable tool for understanding the transmission dynamics of polio and assessing the impact of vaccination strategies (Martcheva, 2015) [11]. Models that incorporate vaccination and immunity dynamics can help predict disease trends, identify critical thresholds for eradication, and guide public health interventions (Heesterbeek *et al*., 2015) [8]. This article aims to analyze the mathematical modeling of polio transmission in Nigeria, focusing on vaccination efforts and immunity dynamics, and to provide recommendations for optimizing eradication strategies.

The literature on polio transmission and vaccination dynamics highlights several key issues in the context of Nigeria's eradication efforts. Vaccine coverage has been identified as a significant factor in reducing polio cases (Hagan *et al*., 2018) [7], with higher coverage associated with lower transmission rates. However, waning immunity and the persistence of low-level transmission in some regions continue to pose challenges (Fine & Ritchie, 2006; Thompson & Duintjer Tebbens, 2006) [4, 12].

Vaccine-derived poliovirus (VDPV) remains a concern, as it can circulate in populations with insufficient immunity and cause outbreaks (Kew *et al*., 2005) [10]. Studies have shown that improving vaccination coverage and conducting supplementary immunization activities can mitigate the risk of VDPV emergence (Duintjer Tebbens *et al*., 2011) [2]. Moreover, the role of socio-cultural factors in influencing vaccination uptake cannot be overlooked, with evidence pointing to the need for community engagement to address vaccine hesitancy (Jegede, 2007; Ghinai *et al*., 2013) [9, 5].

Research on the "Mathematical Analysis of Vaccination and Immunity Dynamics in Polio Eradication Efforts in Nigeria" is vital due to ongoing challenges like vaccine hesitancy and fluctuating immunity levels that hinder eradication. There is need for Mathematical modeling to allows for simulating factors influencing polio spread, identifying vaccination thresholds needed

for herd immunity, and predicting outbreak scenarios. This research is aim at formulating a mathematical model representing the dynamics of polio transmission and vaccination in Nigeria and analyze the stability and threshold conditions for disease eradication as well as evaluating the impact of vaccination coverage, waning immunity, and population mobility on polio dynamics.

2. Mathematical Model Development

Poliomyelitis (polio) is a highly contagious viral disease that can lead to irreversible paralysis and sometimes death. Vaccination plays a crucial role in mitigating the spread and eventual eradication of polio. Mathematical modeling of polio transmission, incorporating vaccination strategies, provides insights into control and elimination efforts. The assumptions for this model are:

1. Homogeneous mixing of the population.
2. Constant total population (birth and natural death rates are balanced).
3. Vaccination is administered at a constant rate or periodically, with a certain efficacy.
4. Waning immunity is considered in previously vaccinated individuals.

2.1 Compartmental Model Framework

We develop a compartmental model to represent the dynamics of polio transmission, vaccination, and immunity in a population. The model divides the population into four compartments based on their disease status as follows:

$S(t)$: Susceptible individuals
$E(t)$: Exposed (infected but not yet infectious)
$I(t)$: Infectious individuals
$R(t)$: Recovered individuals (with immunity)
$V(t)$: Vaccinated individuals (immune)

The complex interplay between natural infection, recovery, vaccination, and loss of immunity, is represented by Fig 1 which represents the flow of individuals through the compartments in the dynamics of polio transmission and control.

Susceptible (S): Individuals enter this compartment via the birth rate Λ, non-adherence to vaccination guidelines and become susceptible after vaccination μ or immunity waning (ρ) to become susceptible.

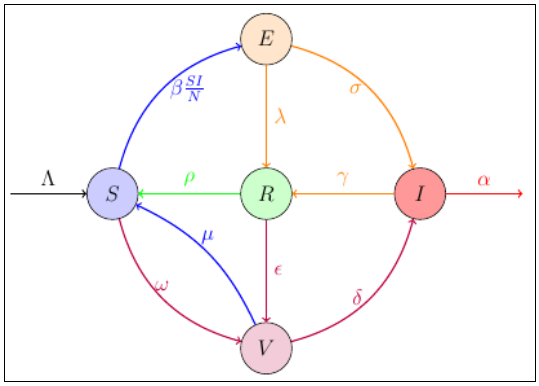

Fig 1: Compartmental Diagram for Polio Dynamics

They leave due to infection ($\beta SI/N$), vaccination (ω). Susceptible individuals who become infected enter this compartment at a rate $\beta SI/N$. They progress to the infectious state at rate σ or recover directly at rate λ. Exposed individuals progress here at rate σ. They recover at rate γ or die due to disease at rate α. Susceptible are vaccinated at rate ω, those who are already recovered lose vaccine-acquired immunity and goes for re-vaccination.

2.2 Model Equations

From the compartmental frame work describe in section 2.2 (above), we derive the following system of equation governing the dynamics of polio transmission and control.

$$\begin{cases} \frac{dS}{dt} = \Lambda - \beta\frac{SI}{N} - \omega S + \rho R + \mu V \\ \frac{dE}{dt} = \beta\frac{SI}{N} - \sigma E - \lambda E \\ \frac{dI}{dt} = \sigma E - \gamma I - \alpha I + \delta V \\ \frac{dR}{dt} = \gamma I - \rho R - \epsilon V + \lambda E \\ \frac{dV}{dt} = \omega S + \epsilon V - \delta V - \mu V \end{cases} \quad (1)$$

The equations incorporate rates such as infection, recovery, vaccination, waning immunity and disease induced death.

Table 1: Parameter Definitions for the Polio Model

Parameter	Description
Λ	Birth rate (rate at which new individuals enter the population)
β	Transmission rate (rate of disease transmission from infectious to susceptible)
N	Total population size
ω	Vaccination rate (rate at which susceptible individuals get vaccinated)
ρ	Waning immunity rate (rate at which recovered individuals lose immunity and become susceptible again)
ϵ	Loss of vaccine protection rate (rate at which vaccinated individuals lose vaccine-acquired immunity)
σ	Progression rate (rate at which exposed individuals become infectious)
λ	Recovery rate of exposed individuals (rate at which exposed individuals recover without becoming infectious)
γ	Recovery rate (rate at which infectious individuals recover and move to the recovered compartment)
α	Disease-induced death rate (rate at which infectious individuals die due to the disease)
δ	Waning vaccine immunity rate (rate at which vaccinated individuals become infectious)

3. Analysis of the Model

The analysis of a disease dynamics of polio transmission and control involves understanding its behavior, transmission and implications.

3.1 Existence and Uniqueness of Solutions for the System

To discuss the existence and uniqueness of solutions for the given system of ordinary differential equations (1), we rely on the Cauchy Lipschitz Theorem. This theorem states that if the system is well-posed (i.e., the functions defining the system are continuous and satisfy a Lipschitz condition), then a unique solution exists in a local neighborhood of the initial condition.

Theorem: Consider the system of ordinary differential equations (1) governing the dynamics of polio transmission

with initial conditions $S(0)=S_0, E(0)=E_0, I(0)=I_0, R(0)=R_0, V(0)=V_0$, satisfying $S_0, E_0, I_0, R_0, V_0 \geq 0$ and $N_0 = S_0 + E_0 + I_0 + R_0 + V_0 > 0$. Then, there exists a unique solution $S(t), E(t), I(t), R(t), V(t)$ to the system for all $t \geq 0$, and the solution remains non-negative and bounded for all t.

Proof: The proof is based on the *Cauchy Lipschitz Theorem*, which guarantees existence and uniqueness of solutions for a system of ODEs under the following conditions:
(i). The right-hand side of the system (1) is continuous with respect to the state variables.
(ii). The right-hand side satisfies a Lipschitz condition in the state variables.
1. *Continuity:* The right-hand side of the system consists of functions such as $\omega S, \gamma I$, etc and nonlinear terms ($\beta SI/N$). Each term is continuous for $S, E, I, R, V \geq 0$ and parameters like β, σ, ρ, etc.
Polynomial and rational functions are continuous for all values where $N \neq 0$ and $S, E, I, R, V \geq 0$.
Since $N = S + E + I + R + V$, assuming a population that is positive and finite, the right-hand sides are continuous.
Hence, the right-hand side of the system is continuous.
2. *Boundedness of the Domain:*
(i). In a physical context, $S, E, I, R, V \geq 0$ and $S + E + I + R + V \leq N$, so the domain is naturally bounded.
(ii). The continuity of the right-hand side functions over a bounded domain guarantees the **local existence** of solutions.

Uniqueness of Solutions
To guarantee uniqueness, we establish Lipschitz continuity.
Lipschitz Condition: To check the Lipschitz condition, consider any two points $(S_1, E_1, I_1, R_1, V_1)$ and $(S_2, E_2, I_2, R_2, V_2)$ in the state space. The Lipschitz condition requires that there exists a constant $L > 0$ such that:

$$\|f(S1, E1, I1, R1, V1) - f(S2, E2, I2, R2, V2)\| \leq L\|(S1, E1, I1, R1, V1) - (S2, E2, I2, R2, V2)\|, \quad (2)$$

where $f(S, E, I, R, V)$ is the vector of right-hand side functions.
In this case, for the term $-\beta\, SI/N$, we compute the partial derivatives:

$$\frac{\partial}{\partial S}\left(-\beta\frac{SI}{N}\right) = -\beta\frac{I}{N}, \frac{\partial}{\partial I}\left(-\beta\frac{SI}{N}\right) = -\beta\frac{S}{N}. \quad (3)$$

Since $S, I \geq 0$ and N is bounded, these derivatives are also bounded. Similarly, all other terms in the system involve linear or bounded coefficients, ensuring the Lipschitz condition is satisfied. By the *Cauchy Lipschitz Theorem*, these conditions guarantee the **local uniqueness** of solutions.

3.2 Global Existence and Uniqueness
To extend local solutions to global solutions **Non-Negativity and Boundedness is established**
Non-explosion Condition:
1. The system models a population, and the compartments S, E, I, R, V are constrained by $S + E + I + R + V = N$, where *N* is constant.
2. The boundedness of the population ensures that solutions cannot "blow up" in finite time, ensuring **global existence**.

Positivity of Solutions:
1. The initial conditions $S(0), E(0), I(0), R(0), V(0) \geq 0$ ensure that the state variables are initially non-negative.
2. The structure of the equations ensures that no compartment can become negative, as all flow terms are non-negative for $S, E, I, R, V \geq 0$.
3. The total population $N = S + E + I + R + V$ is conserved, so $N(t) \leq N(0)$ for all $t \geq 0$. This implies boundedness of the solution.

Thus, the system has a unique, globally defined solution for any non-negative initial condition that satisfies the population constraint.

3.3 Equilibrium Points
3.3.1 Disease-Free Equilibrium (DFE): The state where the disease does not exist in the population. Typically, this occurs when $\boldsymbol{I = I_0 = 0, E = E_0 = 0}$, and the population is distributed among the $\boldsymbol{S, R}$, and $\boldsymbol{V}$ compartments.

$$S = \frac{\Lambda\left((\delta+\mu)-\epsilon\right)}{\delta\omega}, E = 0, I = 0, R = \frac{\epsilon\Lambda}{\delta\rho}, V = \frac{\Lambda}{\delta} \quad (4)$$

3.3.2 Endemic Equilibrium: A state where the disease persists in the population at a constant level. This occurs when $I > 0$. Thus setting the derivatives to zero and solve for the variables, we have

$$\left.\begin{aligned} S^* &= \frac{-N\Lambda(\alpha+\gamma)(\lambda+\sigma)(\epsilon-\mu-\delta)}{(-\beta(\epsilon-\mu-\delta)\Lambda + N\alpha\delta\omega)\sigma + N\alpha\delta\lambda\omega}, \\ E^* &= -\frac{\Lambda^2\beta(\alpha+\gamma)(\epsilon-\mu-\delta)}{\alpha\left(-\beta\sigma(\epsilon-\mu-\delta)\Lambda + N\alpha\delta\omega(\lambda+\sigma)\right)}, \\ I^* &= \frac{\Lambda}{\alpha}, \\ R^* &= \frac{-\beta\left(\lambda\alpha+\gamma(\lambda+\sigma)\right)(\epsilon-\mu-\delta)\Lambda + (\lambda+\sigma)\omega\alpha\left(\epsilon\alpha+\gamma(\epsilon-\delta)\right)N\Lambda}{\alpha\left(-\beta\sigma(\epsilon-\mu-\delta)\Lambda + N\alpha\delta\omega(\lambda+\sigma)\right)\rho}, \\ V^* &= \frac{N\Lambda\omega(\alpha+\gamma)(\lambda+\sigma)}{(-\beta(\epsilon-\mu-\delta)\Lambda + N\alpha\delta\omega)\sigma + N\alpha\delta\lambda\omega}. \end{aligned}\right\} \quad (5)$$

The equilibrium points are given by $(S^*, E^*, I^*, R^*, V^*)$ where each value depends on the parameters $\Lambda, \beta, \omega, \rho, \epsilon, \sigma, \lambda, \gamma, \alpha$, and δ. These must typically be solved numerically, except in special cases where simplifications can be applied.
To ensure that E, I, R, S, and V are all non-negative (≥ 0), we need to analyze each expression and derive conditions based on the parameters. With the assumption that no parameter is less than zero i.e ($\Lambda, \alpha, \beta, \gamma, \delta, \mu, \epsilon, \sigma, \lambda, \omega, N > 0$).

Constraints:

$$-(\epsilon-\mu-\delta) \geq 0 \Rightarrow \epsilon-\mu-\delta \leq 0 \text{ i.e } \epsilon \leq \mu+\delta \text{ and } \epsilon-\delta \geq 0 \Rightarrow \epsilon \geq \delta \quad (6)$$

Equation (6) implies

$$0 \leq \epsilon - \delta \leq \mu \quad (7)$$

3.4 Basic and Effective Reproduction Numbers
The basic reproduction number (R_0) and the effective

reproduction number (R_{eff}) are key epidemiological quantities that measure the average number of secondary infections caused by a single infectious individual in a fully susceptible population (R_0) and in a partially susceptible population (R_{eff}).

3.4.1 Basic Reproduction Number (R_0)

The basic reproduction number is derived using the next-generation matrix (NGM) approach. To compute R_0, we consider the compartments directly involved in the transmission: Exposed (E) and Infectious (I). The equations governing these compartments are:

$$\frac{dE}{dt} = \beta\frac{SI}{N} - (\sigma + \lambda)E,$$
$$\frac{dI}{dt} = \sigma E - (\gamma + \alpha)I. \quad (8)$$

Infection terms (F): Equation (8) are the terms representing new infections:

$$\mathcal{F} = \begin{bmatrix} \beta\frac{S}{N}I \\ 0 \end{bmatrix}. \quad (9)$$

Transition terms ($\mathcal{V}$): Equation (9) are the terms representing the transition in and out of compartments:

$$\mathcal{V} = \begin{bmatrix} (\sigma + \lambda)E \\ -(\sigma E - (\gamma + \alpha)I) \end{bmatrix}. \quad (10)$$

The Jacobians of $\mathcal{F}$ and $\mathcal{V}$ are computed at the disease-free equilibrium (4),
Jacobian of $\mathcal{F}$:

$$F = \begin{bmatrix} 0 & \beta\frac{\Lambda((\delta+\mu)-\epsilon)}{N\delta\omega} \\ 0 & 0 \end{bmatrix}.$$

Jacobian of $\mathcal{V}$:

$$V = \begin{bmatrix} \sigma+\lambda & 0 \\ -\sigma & \gamma+\alpha \end{bmatrix}$$

The next-generation matrix is:

$$K = FV^{-1}.$$

But

$$V^{-1} = \begin{bmatrix} \frac{1}{\sigma+\lambda} & 0 \\ \frac{\sigma}{(\sigma+\lambda)(\gamma+\alpha)} & \frac{1}{\gamma+\alpha} \end{bmatrix}.$$

Therefore,

$$K = FV^{-1} = \begin{bmatrix} 0 & \beta\frac{\Lambda((\delta+\mu)-\epsilon)}{N\delta\omega} \\ 0 & 0 \end{bmatrix}\begin{bmatrix} \frac{1}{\sigma+\lambda} & 0 \\ \frac{\sigma}{(\sigma+\lambda)(\gamma+\alpha)} & \frac{1}{\gamma+\alpha} \end{bmatrix}.$$

$$K = \begin{bmatrix} \frac{\beta\sigma\Lambda((\delta+\mu)-\epsilon)}{N\delta\omega(\sigma+\lambda)(\gamma+\alpha)} & \frac{\beta\sigma\Lambda((\delta+\mu)-\epsilon)}{N\delta\omega(\gamma+\alpha)} \\ 0 & 0 \end{bmatrix}.$$

Now, let $\Im$ be the eigenvalue, the root of

$$\det(K - \Im I)$$

is the eigenvalue, where I is the identity matrix. Thus

$$\Im = 0 \text{ or } \frac{\beta\sigma\Lambda((\delta+\mu)-\epsilon)}{N\delta\omega(\sigma+\lambda)(\gamma+\alpha)} \quad (11)$$

Then largest eigenvalue is:

$$\frac{\beta\sigma\Lambda((\delta+\mu)-\epsilon)}{N\delta\omega(\sigma+\lambda)(\gamma+\alpha)}$$

Hence

$$R_0 = \frac{\beta\sigma\Lambda((\delta+\mu)-\epsilon)}{N\delta\omega(\sigma+\lambda)(\gamma+\alpha)} \quad (12)$$

- R_0 represents the average number of secondary infections caused by one infectious individual in a fully susceptible population.
- If $R_0 < 1$, the disease will die out over time.
- If $R_0 > 1$, the disease will spread and may become endemic.

3.4.2 Effective Reproduction Number (R_{eff})

R_{eff} represents the average number of secondary infections caused by a single infectious individual in a partially susceptible population. The effective reproduction number adjusts R_{eff} to account for the fraction of the population that is susceptible. The fraction of susceptible individuals in the population is S^*/N, where S^* is the disease-free equilibrium.

Then:

$$R_{eff} = R_0 \cdot \frac{S^*}{N} \quad (13)$$

Hence, substituting for the parameters and expression for R_0, we have

$$R_{eff} = \frac{\beta\sigma\Lambda((\delta+\mu)-\epsilon)}{N\delta\omega(\sigma+\lambda)(\gamma+\alpha)} \cdot \frac{\Lambda((\delta+\mu)-\epsilon)}{\delta\omega N} \quad (14)$$

These quantities depend on the parameters of the system

3.5 Stability Analysis

We examine the stability of equilibrium points of a system (1), a Lyapunov function $V(t)$ is chosen to be

$$V(t) = \frac{1}{2}S(t)^2 + \frac{1}{2}E(t)^2 + \frac{1}{2}I(t)^2 + \frac{1}{2}R(t)^2 + \frac{1}{2}V(t)^2$$

Then

$$\frac{d}{dt}V(t) = S(t)\frac{d}{dt}S(t) + E(t)\frac{d}{dt}E(t) + I(t)\frac{d}{dt}I(t) + R(t)\frac{d}{dt}R(t) + V(t)\frac{d}{dt}V(t) \quad (15)$$

Evaluating (15) at the derivatives, we have

$$\begin{aligned}\frac{d}{dt}V(t) = S\left(\Lambda - \beta\frac{SI}{N} - \omega S + \rho R + \mu V\right) + E(t)\left(\beta\frac{SI}{N} - \sigma E - \lambda E\right) \\ + I(\sigma E - \gamma I - \alpha I + \delta V) + R(\gamma I - \rho R - \epsilon V + \lambda E) \\ + V(\omega S + \epsilon V - \delta V - \mu V)\end{aligned} \quad (16)$$

3.5.1 Local Stability Analysis: For local stability, we evaluate equation (16) at DFE (4), we have

$$\begin{aligned}\frac{d}{dt}V(t) = \Lambda\frac{(-\epsilon + \mu + \delta)}{\delta\omega}\left(\Lambda - \Lambda\frac{(-\epsilon + \mu + \delta)}{\delta} - \frac{\epsilon\Lambda}{\delta} + \mu\frac{\Lambda}{\delta}\right) \\ + \frac{\Lambda}{\delta}\left(\Lambda\frac{(-\epsilon + \mu + \delta)}{\delta} + \frac{\epsilon\Lambda}{\delta} - \frac{(\delta + \mu)\Lambda}{\delta}\right) = 0\end{aligned} \quad (17)$$

That is, the time derivative of (17) is zero.

Since all terms in $\dot{V}$ vanish at the Disease-Free Equilibrium (DFE), this implies that $\dot{V} = 0$. This means that the Lyapunov function does not decrease at the DFE, which suggests that the system is not strictly globally asymptotically stable at this point. Thus, the system is *marginally stable* at the Disease-Free Equilibrium (DFE).

3.5.2 Global Stability: Now, evaluate equation (16) at DEE, we have

$$\begin{aligned}\frac{d}{dt}V(t) = \Big(2\big((\lambda + \sigma)((\rho + \omega)\epsilon - \rho(\delta + \mu))N\alpha^2 + \big((\lambda + \sigma)((\rho + \omega)\epsilon + (-\rho - \omega)\delta - \mu\rho)N\gamma - \lambda\beta(\epsilon - \mu - \delta)\big)\alpha \\ - \beta\gamma(\epsilon - \mu - \delta)(\lambda + \sigma)\big) \\ \times \Lambda^2\left(N\epsilon\omega(\lambda + \sigma)\alpha^2 + \left(N\omega(\epsilon - \delta)(\lambda + \sigma)\gamma + \frac{1}{2}\lambda\beta(\epsilon - \mu - \delta)(\Lambda - 1)\right)\alpha\right. \\ \left. + \frac{1}{2}\beta\gamma(\epsilon - \mu - \delta)(\lambda + \sigma)(\Lambda - 1)\right)\Big)\frac{1}{\left(-\beta\sigma(\epsilon - \mu - \delta)\Lambda + N\alpha\delta\omega(\lambda + \sigma)\right)^2\rho\alpha^2}\end{aligned}$$

Thus for

$$\left(\frac{\rho + \omega}{\delta + \mu}\right)\epsilon > \rho \text{ and } (\rho + \omega)(\epsilon - \delta) > \mu\rho$$

$$\frac{d}{dt}V(t) < 0$$

3.6 Sensitivity Analysis

Sensitivity analysis is a method used to determine how changes in the parameters of a mathematical model affect the output or behavior of the model. The sensitivity analysis aims to evaluate how variations in model parameters influence the basic reproduction number R_0.

Definition: Normalized Forward Sensitivity Index

The normalized forward sensitivity index (S_p) of θ with respect to a parameter p is defined as:

$$S_p = \frac{\partial\theta}{\partial p}\cdot\frac{p}{\theta}$$

This measures the relative change in θ due to a relative change in p. A positive index indicates that θ increases with p, while a negative index indicates that θ decreases with p.

Sensitivity Indices for Parameters in R_0

Calculation for β:

$$\frac{\partial R0}{\partial\beta} = \frac{\sigma\Lambda(\delta + \mu) - \epsilon}{N\delta\omega(\sigma + \lambda)(\gamma + \alpha)}, \quad S\beta = \frac{\partial R_0}{\partial\beta}\cdot\frac{\beta}{R_0} = 1$$

Calculation for σ:

$$\frac{\partial R0}{\partial\sigma} = \frac{(\beta\Lambda(\delta + \mu) - \epsilon)}{N\delta\omega(\sigma + \lambda)(\gamma + \alpha)}, \quad S\beta = \frac{\partial R_0}{\partial\sigma}\cdot\frac{\sigma}{R_0} = 1$$

The process is repeated for all parameters in R_0 and R_{eff}, the following table is obtained.

Table 2: Sensitivity Indices of Parameters for R_0

Parameter	Description	Sensitivity Index (R_0)	Sensitivity Index (R_{eff})
β	Transmission rate	1	1
σ	Progression rate	1	1
Λ	Birth rate	1	2
δ	Waning vaccine immunity rate	-1	-2
μ	Loss of vaccine-acquired immunity rate	$-\frac{\epsilon}{\delta+\mu-\epsilon}$	$-\frac{2\epsilon}{\delta+\mu-\epsilon}$
ϵ	Loss of vaccine-acquired immunity rate	$-\frac{\epsilon}{\delta+\mu-\epsilon}$	$-\frac{2\epsilon}{\delta+\mu-\epsilon}$
ω	Vaccination rate	-1	-2
λ	Recovery rate of exposed individuals	$-\frac{\lambda}{\sigma+\lambda}$	$-\frac{\lambda}{\sigma+\lambda}$
γ	Recovery rate	$-\frac{\gamma}{\gamma+\alpha}$	$-\frac{\gamma}{\gamma+\alpha}$
α	Disease-induced death rate	$-\frac{\alpha}{\gamma+\alpha}$	$-\frac{\alpha}{\gamma+\alpha}$
N	Total population size	-1	-2

4. Numerical Simulations

For numerical solutions of our model, we need to define the initial conditions for each compartment and the model parameters.

Table 3: Initial Values for SEIRV Model Simulation

Parameter	Value
Initial Susceptible Population ($S0$)	999,000
Initial Exposed Population ($E0$)	0
Initial Infectious Population ($I0$)	1,000
Initial Recovered Population ($R0$)	0
Initial Vaccinated Population ($V0$)	0
Simulation Time Frame	365 days
Time Step	1 day

Table 4: Suggested Values and Units for Parameters

Parameter	Value	Units	Source/Notes
Λ	10	Individuals/day	Estimated from population growth
β	0.5	Per individual per day	Literature on polio dynamics
N	10^6	Individuals	Population size
ω	0.01	Per day	Vaccination campaign data
ρ	0.002	Per day	Immunity waning rates
ϵ	0.005	Per day	Vaccine effectiveness studies
σ	0.1	Per day	Polio incubation period
λ	0.05	Per day	Rate of recovery in exposed
γ	0.05	Per day	Recovery rate from infectious stage
α	0.01	Per day	Disease-induced mortality
δ	0.005	Per day	Waning vaccine immunity

For solving the system of ODEs**,** a Runge-Kutta method of order 4 (RK4) is used for better accuracy and implemented in Maple 2023 release. The results are resented in the Figs. (2) -()

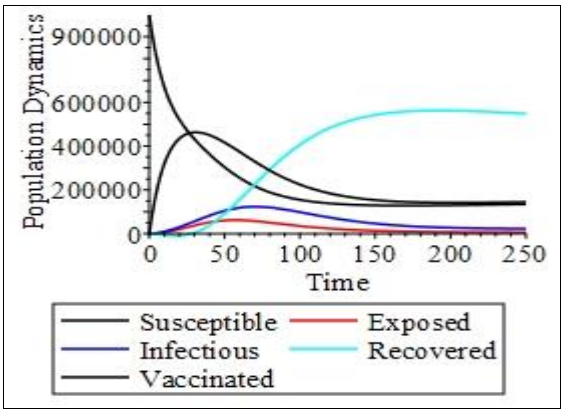

Fig 2: Population Dynamics

5. Discussion of Results

5.1 Sensitivity Indices on R_0 and R_{eff}

The numerical estimates for Ro and Reff is presented in Table 5 below.

5.1.1 Sensitivity Indices for R_0

The sensitivity index for the transmission rate (β) is $S_\beta = 1$, indicating that R_0 is directly proportional to β. A 10% increase in β results in a 10% increase in R_0. Similarly, the progression rate (σ) has a sensitivity index of $P_\sigma = 1$, showing the same proportional relationship, where a 10% increase in σ results in a 10% increase in R_0. The birth rate (Λ) also exhibits direct proportionality with R_0, having a sensitivity index of $S_\Lambda = 1$. Therefore, any percentage increase in Λ translates into the same percentage increase in R_0.

Table 5: Sensitivity of Parameters

Parameter	Description	R_0	R_{eff}
β	Transmission rate	1	1
σ	Progression rate	1	1
Λ	Birth rate	1	2
δ	Waning vaccine immunity rate	−1	−2
μ	Loss of vaccine-acquired immunity rate	−0.33	−0.67
ϵ	Vaccine effectiveness decay rate	−0.33	−0.67
ω	Vaccination rate	−1	−2
λ	Recovery rate of exposed individuals	−0.33	−0.33
γ	Recovery rate	−0.83	−0.83
α	Disease-induced death rate	−0.17	−0.17
N	Total population size	−1	−2

The waning vaccine immunity rate (δ) has a sensitivity index of $S_\delta = -1$, indicating an inverse relationship with *R*0. For instance, a 10% increase in δ leads to a 10% decrease in R_0. The loss of vaccine-acquired immunity rate (μ) and the vaccine effectiveness decay rate (ϵ) both have sensitivity indices of $S_\mu = -0.33$ and $S_\epsilon = -0.33$, respectively. This implies that a 10% increase in either parameter results in a 3.3% decrease in R_0.

The vaccination rate (ω) has a sensitivity index of $S_\omega = -1$, showing that R_0 decreases linearly with increases in ω. A 10% increase in ω results in a 10% reduction in R_0. The recovery rate of exposed individuals (λ) has a sensitivity index of $S_\lambda = -0.33$, meaning that a higher recovery rate moderately reduces *R*0. The recovery rate (γ) is more influential, with a sensitivity index of $S_\gamma = -0.83$, indicating that a 10% increase in γ results in an 8.3% decrease in R_0. The disease-induced death rate (α) has a relatively minor effect, with $S_\alpha = -0.17$, where a 10% increase in α leads to a 1.7% reduction in *R*0. Finally, the population size (N) has a sensitivity index of $S_N = -1$, implying that *R*0 decreases inversely with population size, with a 10% increase in *N* leading to a 10% decrease in R_0.

5.1.2 Sensitivity Indices for R_{eff}

The sensitivity index for the transmission rate (β) in *R*eff is $S_\beta = 1$, identical to that of R_0. This indicates a direct proportional relationship where a 10% increase in β results in a 10% increase in R_{eff}. Similarly, the progression rate (σ) has a sensitivity index of $S_\sigma = 1$, maintaining a proportional relationship. However, the birth rate (Λ) exhibits a higher sensitivity with $S_\Lambda = 2$, meaning a 10% increase in Λ results in a 20% increase in R_{eff}.

The waning vaccine immunity rate (δ) has a sensitivity index of $S_\delta = -2$, showing that R_{eff} is inversely proportional to δ, with a 10% increase in δ resulting in a 20% decrease in R_{eff}. Similarly, the vaccination rate (ω) has a sensitivity index of $S_\omega = -2$, indicating a significant inverse relationship, where a 10% increase in ω results in a 20% reduction in R_{eff}. Both the loss of vaccine-acquired immunity rate (μ) and the vaccine effectiveness decay rate (ϵ) have sensitivity indices of $S_\mu = -0.67$ and $S_\epsilon = -0.67$, respectively. This means a 10% increase in these parameters leads to a 6.7% reduction in R_{eff}.

The recovery rate of exposed individuals (λ) has a moderate sensitivity index of *S*λ = -0.33, indicating a similar trend to R_0. The recovery rate (γ) has a sensitivity index of *S* γ = -0.83, showing that a 10% increase in γ leads to an 8.3% decrease in R_{eff}. The disease-induced death rate (α) has minimal influence with $S_\alpha = -0.17$. Lastly, the population size (N) is highly significant, with $S_N = -2$, meaning a 10% increase in *N* results in a 20% decrease in R_{eff}.

5.2 Sensitivity of parameters on the dynamics

In this study, we analyzed the dynamics of polio transmission using a compartmental model that considers five distinct populations: Susceptible (S), Exposed (E), Infectious (I), Recovered (R), and Vaccinated (V). The system of differential equations governing the interactions among these compartments was solved numerically to understand the effect of different parameters on the disease dynamics. The results were analyzed to observe how variations in key parameters, such as the transmission rate (β), vaccination rate (ω), waning immunity rate (ρ), and recovery rate (γ), affect the behavior of the populations. The model dynamics is shown in Fig., which provide valuable

insights into the dynamics of polio transmission. The variations in the parameters and their impacts on the populations of Exposed, Vaccinated, and Recovered individuals are discussed below.

5.2.1 Effect of Transmission Rate (β)

The transmission rate (β) represents the likelihood of an interaction between a susceptible individual and an infectious individual that leads to the transmission of the disease. As shown in the results Fig. 3, an increase in β leads to a higher number of exposed individuals (E) and consequently a higher number of infectious individuals (I) over time. This is due to the increased rate of infection spread in the population, which leads to a more rapid transition from susceptible to exposed individuals. In contrast, a decrease in β reduces the number of new infections, leading to fewer exposed and infected individuals, and hence, slower disease spread.

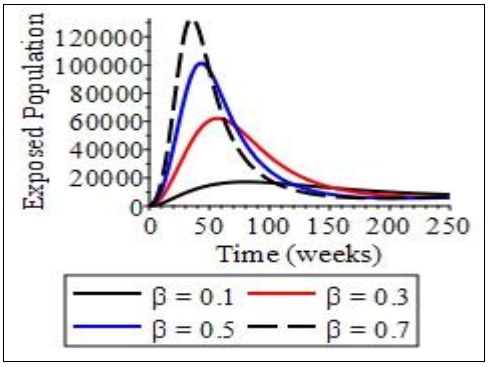

Fig 3: Effect of Transmission Rate on Exposed Population

5.2.2 Effect of Vaccination Rate (ω)

The vaccination rate (ω) determines the rate at which susceptible individuals are vaccinated Fig.4-6, which reduces the susceptible population and helps in preventing future infections. As expected, an increase in the vaccination rate results in a higher number of vaccinated individuals (V), which consequently reduces the number of susceptible individuals Fig 5. This leads to a decrease in the transmission of the disease (Fig 4), as there are fewer individuals available for infection. A higher vaccination rate thus helps to control the spread of polio more effectively (Fig 6). In contrast, a lower vaccination rate allows the disease to spread more rapidly through the population.

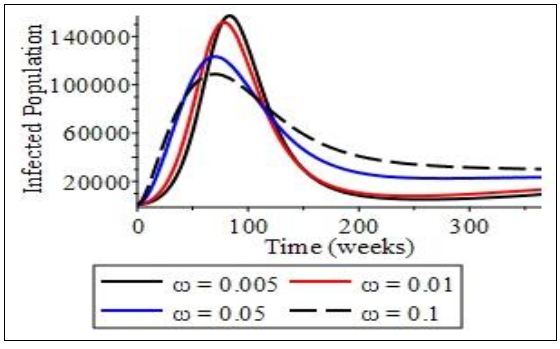

Fig 4. Rate of Loss of Natural Immunity on Infected Population

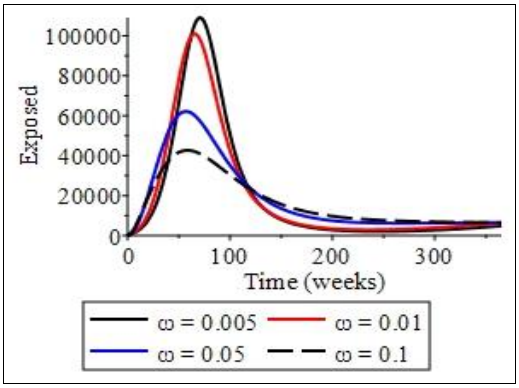

Fig 5: Rate of Loss of Natural Immunity on exposed Population

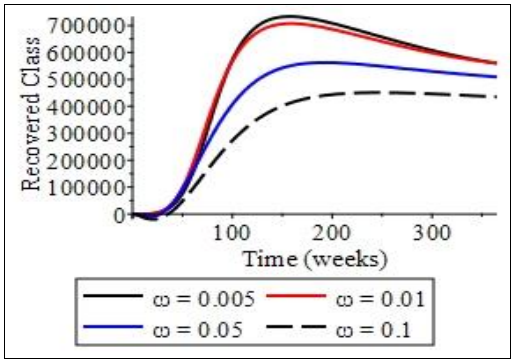

Fig 6: Rate of Loss of Natural Immunity on Recovered Class

5.2.3 Effect of Waning Immunity Rate (ρ)

The waning immunity rate (ρ) represents the rate at which recovered individuals lose their immunity over time and become susceptible again. An increase in ρ leads to more individuals moving from the recovered compartment (R) back to the susceptible compartment (S), which increases the potential for new infections, Fig 7. Consequently, a higher ρ results in a resurgence of the disease as previously immune individuals become vulnerable again. On the other hand, a lower ρ reduces the number of individuals returning to the susceptible class, thereby lowering the overall disease burden in the population, Fig 8.

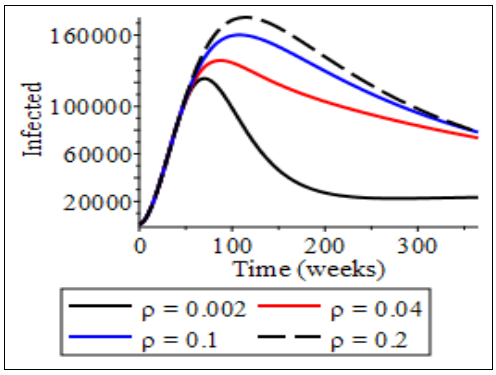

Fig 7: Rate of Waning Immunity on Infected Class

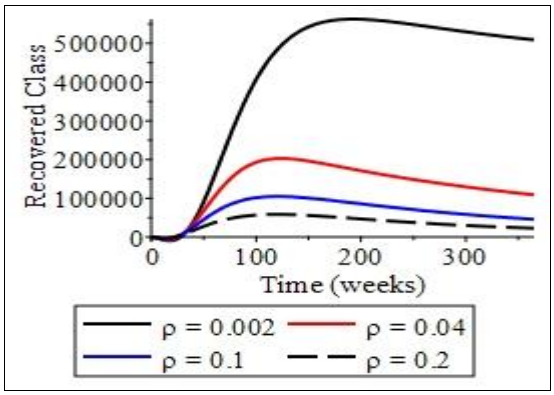

Fig 8: Rate of Waning Immunity on Recovered Class

5.2.4 Effect of Recovery Rate (γ)

The recovery rate (γ) determines how quickly infected individuals recover from the disease. An increase in γ leads to a faster transition from the infectious class (I) to the recovered class (R), reducing the number of infectious individuals in the population. This results in a reduction in the spread of the disease, as fewer individuals remain infectious for long periods. Conversely, a decrease in γ leads to a slower recovery process, maintaining a higher number of infectious individuals and thus allowing the disease to spread more effectively.

5.2.5 Effect of Progression Rate on Infected Population (σ)

The progression rate from of exposed individual to infected class is displayed in Figs. 9 and 10. From these figures, increase in progression rate leaves the exposed class lowers as many migrated out of the class to infected class while, it resulted in increase in the infected class.

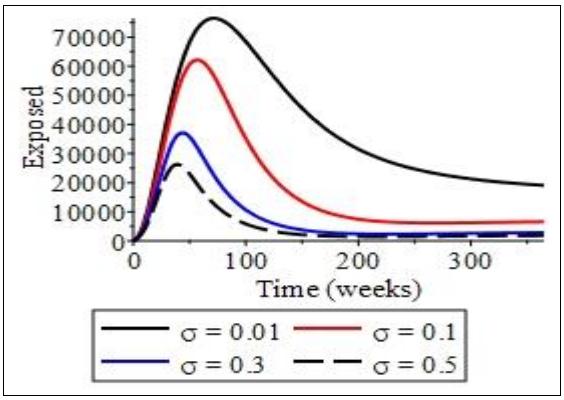

Fig 9: Progression Rate on Exposed Population

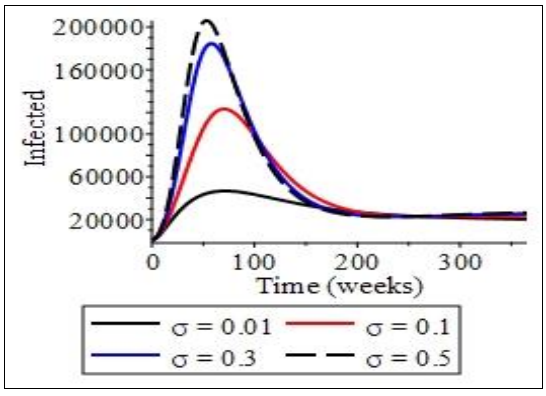

Fig 10: Progression Rate on Infected Population

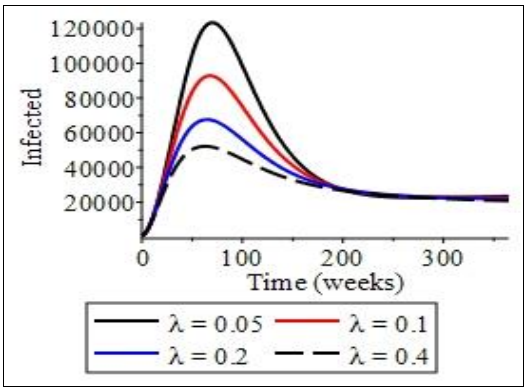

Fig 11: Recovery Rate on Infected Population

5.2.6 Waning Vaccine Immunity and Loss of Vaccine-Acquired Immunity

The combined effects of waning vaccine immunity and vaccine effectiveness decay on different compartments of a mathematical model analyzing vaccination and immunity dynamics in polio eradication efforts in Nigeria provide critical insights into the disease's control strategies.

Figures 12 to 15 specifically explore the influence of waning vaccine immunity and the loss of vaccine-acquired immunity rates on various compartments, including recovered, exposed, infected, and vaccinated populations.

1. **Recovered Population (Fig 12)**: As waning immunity progresses, a noticeable decline in the recovered population is observed. The gradual loss of vaccine-acquired immunity returns individuals to a susceptible or exposed state, which undermines long-term disease-free conditions. This underscores the importance of booster doses to maintain high immunity levels in the population.

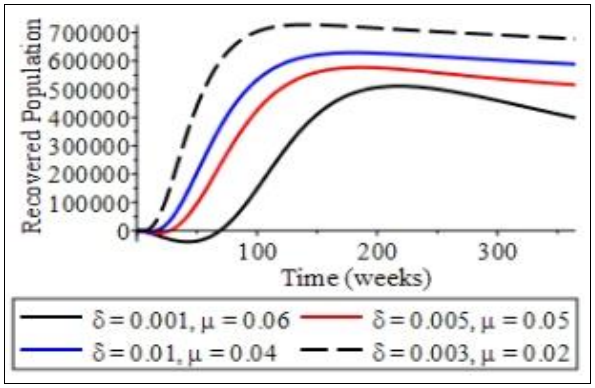

Fig 12: Waning Vaccine/Loss of Vaccine Acquired Immunity Rate on Recovered Population

2. **Exposed Population (Fig 13)**: The exposed compartment shows an upward trend when the waning of vaccine-acquired immunity increases. The loss of protection creates a vulnerable segment of the population that becomes more susceptible to viral exposure. This dynamic poses a threat to herd immunity and requires effective surveillance and timely vaccination interventions.
3. **Infected Population (Fig 14)**: The infected population responds inversely to changes in immunity levels. As vaccine protection wanes, the infection rate increases, leading to a larger infected cohort. This outcome highlights the critical role of sustained vaccine coverage

and the need to mitigate immunity loss through public health campaigns.

4. **Vaccinated Population (Fig 15)**: A declining trend in the vaccinated compartment is evident with increasing waning immunity. This shift reflects the erosion of protection among vaccinated individuals, emphasizing the necessity for policy strategies that promote re-vaccination or periodic booster doses.

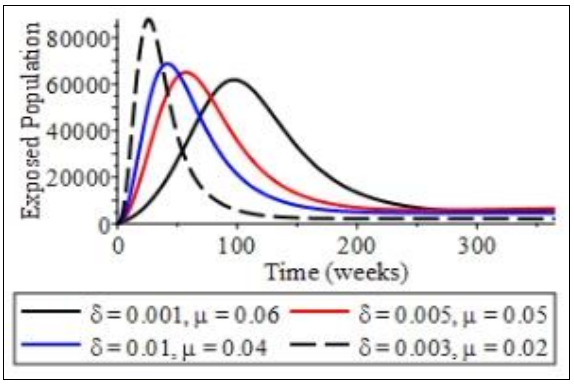

Fig 13: Waning Vaccine/Loss of Vaccine Acquired Immunity Rate on Exposed Population

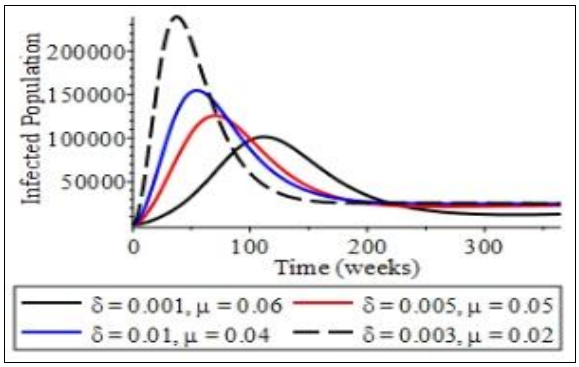

Fig 14: Waning Vaccine and Loss of Vaccine Acquired Immunity Rate on Infected Population

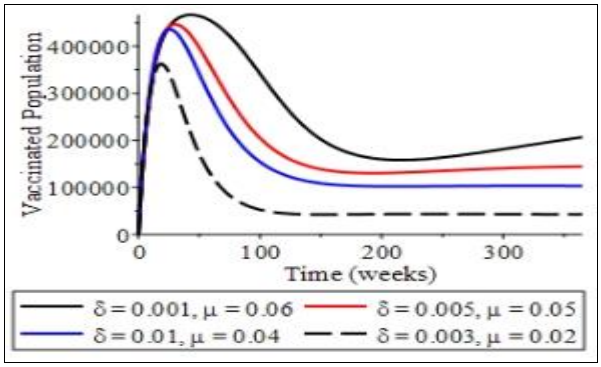

Fig 15: Waning Vaccine and Loss of Vaccine Acquired Immunity Rate on Vaccinated Population

5.2.7 Vaccine Effectiveness Decay Rate and Loss of Vaccine-Acquired Immunity

Figures 16 to 19 investigate the combined impact of vaccine effectiveness decay and the loss of vaccine-acquired immunity on various compartments, revealing compounding effects on disease dynamics.

1. **Exposed Population (Fig 16)**: As vaccine effectiveness decays, coupled with immunity loss, there is a significant increase in the exposed population. The dual effect exacerbates vulnerability to infection, particularly in regions where vaccination uptake is suboptimal. Strengthening vaccine effectiveness and enhancing coverage are essential to mitigating this risk.

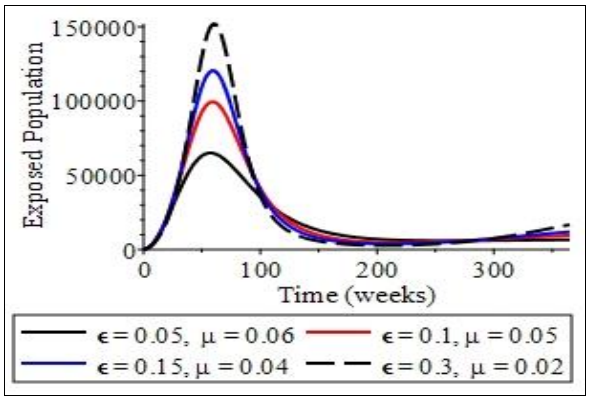

Fig 16: Vaccine Effectiveness Decay Rate and Loss of Vaccine Acquired Immunity Rate on Exposed Population

2. **Infected Population (Fig 17)**: A pronounced rise in the infected compartment occurs due to reduced vaccine efficacy and loss of acquired immunity. The synergy between these factors accelerates disease transmission and can undermine eradication efforts. Continuous monitoring of infection rates and adaptive vaccination strategies are crucial to counter this trend.

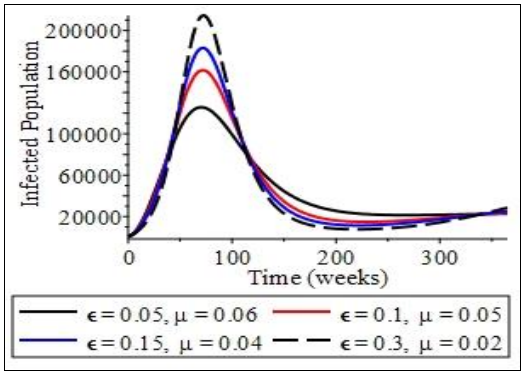

Fig 17: Vaccine Effectiveness Decay Rate and Loss of Vaccine Acquired Immunity Rate on Infected Population

3. **Vaccinated Population (Fig 18)**: The vaccinated compartment diminishes over time as vaccine effectiveness decays and immunity wanes. This depletion poses a risk to maintaining herd immunity and necessitates efforts to improve vaccine durability and promote booster administration.

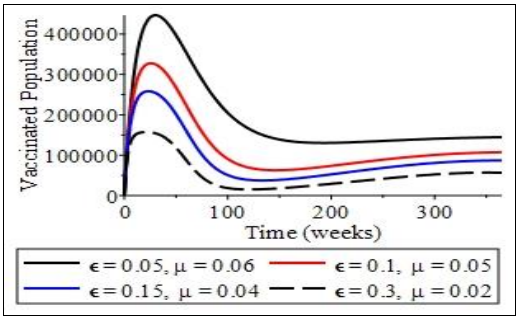

Fig 18: Vaccine Effectiveness Decay Rate and Loss of Vaccine Acquired Immunity Rate on Vaccinated Population

4. **Recovered Population (Fig 19)**: The recovered population also sees a downward shift under these conditions. The loss of immunity and diminishing vaccine effectiveness revert recovered individuals to susceptible or exposed states, thereby increasing the risk of future outbreaks.

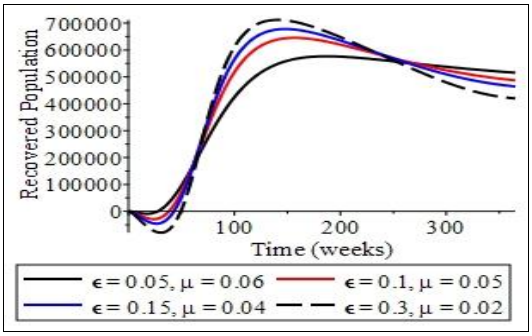

Fig 19: Vaccine Effectiveness Decay Rate and Loss of Vaccine Acquired Immunity Rate on Recovered Population

5.3 Summary

The analysis of the polio transmission model revealed the following key findings:

- The transmission rate (β) plays a critical role in determining the speed at which the disease spreads. A higher transmission rate leads to more rapid infection, while a lower rate slows the disease progression.
- Vaccination rate (ω) is crucial in controlling the disease. Increasing vaccination rates reduces the number of susceptible individuals, thereby preventing new infections.
- The waning immunity rate (ρ) and the loss of vaccine protection (ϵ) can significantly influence disease dynamics. Higher values of ρ and ϵ increase the susceptible population, contributing to disease resurgence.
- The recovery rate (γ) influences the duration for which individuals remain infectious. A higher recovery rate reduces the duration of infection and helps control the spread of the disease.

5.4 Conclusion and Observations

In conclusion, the polio transmission model demonstrates that disease dynamics are highly sensitive to variations in key parameters, such as the transmission rate, vaccination rate, waning immunity, and recovery rate. The model emphasizes the importance of vaccination programs and maintaining immunity in the population to prevent the spread of polio.

The following observations were made:

- Increasing the vaccination rate (ω) is one of the most effective ways to control the spread of polio.
- The loss of immunity, both from natural recovery (ρ) and from vaccination (ϵ), can significantly hinder disease eradication efforts.
- Recovery rates (γ) must be sufficiently high to ensure that infected individuals are removed from the transmission chain as quickly as possible.
- Efforts to reduce the transmission rate (β) through public health interventions, such as social distancing or improved hygiene, could also help in controlling the spread of polio.

In light of these findings, it is crucial for policymakers to focus on enhancing vaccination coverage and strengthening public health initiatives to minimize the impact of polio on the population. Conclusion

6. Conflict of interest:

The authors declare no conflicts of interest in the research on vaccine hesitancy and polio spread, ensuring independent study and unbiased findings without influence from external entities or relationships.

7. Acknowledgments:

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