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Evaluating Vaccine Hesitancy and Its Effects on Polio Spread Using Mathematical Models

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ABSTRACT: Vaccine hesitancy poses a significant barrier to global polio eradication efforts, particularly in regions where misinformation, cultural beliefs, and mistrust hinder immunization programs. This study evaluates the impact of vaccine hesitancy on the spread of polio using mathematical modeling approaches, focusing on Nigeria, where the disease has persisted despite extensive vaccination campaigns. A compartmental SIRV (Susceptible-Infected-Recovered-Vaccinated) model is employed to simulate the dynamics of polio transmission, incorporating vaccine uptake rates and hesitancy factors. The analysis explores how suboptimal vaccination coverage influences herd immunity thresholds and increases the risk of outbreaks and virus resurgence. Furthermore, the model assesses the effectiveness of various intervention strategies, such as intensified immunization campaigns and targeted communication programs aimed at reducing vaccine hesitancy. Results highlight the critical role of adaptive public health policies and targeted vaccination efforts in mitigating the adverse effects of vaccine hesitancy on polio transmission. This study provides valuable insights to guide policymakers in optimizing control strategies to achieve polio eradication in Nigeria and similar settings.

KEYWORDS: Vaccine, Hesitancy, Polio, Mathematical Modeling, Compartmental Models, Epidemiology, Immunization, Behavioral Factors, Transmission Dynamics,

I. INTRODUCTION

Poliomyelitis, commonly known as polio, remains a significant global health challenge despite substantial progress in eradication efforts. It is a highly infectious viral disease predominantly affecting children under five, with the potential to cause permanent paralysis and even death. Although the widespread use of vaccines has dramatically reduced polio cases worldwide, the virus persists in specific regions, including Nigeria, due to several complex factors [1,2]. One major obstacle to eradication is vaccine hesitancy, defined as the delay in acceptance or refusal of vaccines despite the availability of vaccination services [3]. This reluctance arises from a mix of sociocultural beliefs, misinformation, safety concerns, and distrust in healthcare systems, particularly in areas with a history of political instability and health system challenges [3,4].

Nigeria's history of vaccine hesitancy is exacerbated by past controversies regarding the oral polio vaccine (OPV), including unfounded rumors linking it to infertility, which significantly undermined public trust [5]. Efforts to overcome these challenges have been met with varying levels of success, influenced by regional disparities, community engagement, and the effectiveness of public health messaging [6]. Understanding the impact of vaccine hesitancy on the transmission dynamics of polio is thus critical for optimizing vaccination campaigns and enhancing eradication strategies.

The drivers of vaccine hesitancy are multifaceted, often rooted in cultural, social, and economic factors. In Nigeria, misinformation about vaccine safety has been prevalent, partly due to low literacy levels and limited access to credible health information. Religious beliefs also play a role, with some communities resisting vaccination on theological grounds [7,8]. Moreover, logistical challenges such as inadequate vaccine storage facilities and transportation further complicate vaccination efforts in remote regions [9]. Addressing these issues requires an understanding of the underlying causes and the development of targeted interventions aimed at promoting vaccine acceptance and increasing coverage [10,11].

Mathematical modeling has become an invaluable tool in epidemiology, providing insights into disease transmission and the potential outcomes of various control measures. In the context of polio, models can simulate different scenarios by incorporating factors such as vaccination rates, waning immunity, birth rates, and seasonal variations in transmission [12]. Recent advances in modeling have enabled the integration of data on vaccine hesitancy to evaluate the effects of

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suboptimal immunization coverage on polio dynamics. These models offer a framework for assessing the potential consequences of hesitancy and guiding policy decisions aimed at enhancing eradication strategies [1,13]. Through simulations, policymakers can better understand the thresholds needed for herd immunity and design more effective interventions to reduce the risk of outbreaks [14].

II. MATHEMATICAL FORMULATION

A compartmental model is often used to represent polio transmission, dividing the population into different groups or "compartments" based on disease status: Susceptible (S), Infected (I), Recovered (R), and Vaccinated (V). For polio, a common model used is the SIRV model, which includes the compartments:

- Susceptible (S): Individuals who are at risk of contracting polio because they are not vaccinated or have lost immunity.
- Infected (I): Individuals who are currently infected and capable of transmitting the virus.
- Recovered (R): Individuals who have recovered from infection and gained temporary or permanent immunity.
- Vaccinated (V): Individuals who have been vaccinated and have developed immunity.

Incorporating vaccine hesitancy into the model can be achieved by introducing a parameter that represents the proportion of the population that refuses vaccination. This parameter impacts the rate at which individuals move from the susceptible to the vaccinated compartment, thus affecting the overall vaccination coverage.

The equations governing the dynamics of the SIRV model can be written as a system of ordinary differential equations (ODEs), incorporating a parameter for vaccine hesitancy that affects the rate of vaccination. The set of equations that describe the dynamics becomes:

$$\frac{dS(t)}{dt} = \Lambda N - \beta\frac{S(t)I(t)}{N} - (1-h)\nu S(t) - \mu S(t) \qquad (1)$$

$$\frac{dI(t)}{dt} = \beta\frac{S(t)I(t)}{N} - \gamma I(t) - \mu I(t) \qquad (2)$$

$$\frac{dR(t)}{dt} = \gamma I(t) - \omega R(t) - \mu R(t) \qquad (3)$$

$$\frac{dV(t)}{dt} = (1-h)\nu S(t) - \mu V(t) \qquad (4)$$

- The term $\beta\frac{S(t)I(t)}{N}$ represents the rate at which susceptible individuals become infected, with β being the transmission rate and N the total population.
- The vaccination term $(1-h)\nu S$ indicates the rate at which susceptible are vaccinated, where ν is the vaccination rate and h is the hesitancy parameter (with $0 \le h \le 1$). If $h = 0$, there is no hesitancy, while $h > 0$ indicates increasing levels of hesitancy.
- $\mu S(t)$ represents the natural death rate among the susceptible individuals.
- The term γI represents the recovery rate of infected individuals, where γ is the recovery rate.
- The term μI accounts for the natural death rate among infected individuals.
- ωR represents the rate of waning immunity, where ω is the rate at which recovered individuals lose immunity and become susceptible again.
- μR accounts for the natural death rate among recovered individuals.
- The term μV represents the natural death rate among vaccinated individuals.
- The total population is given by $N = S + I + R + V$, assuming no other sources of population change (e.g., migration).

Table 1: Numerical Value of the Parameter

Parameter	Description	Value	Unit
Λ	Birth rate	0.02(10-30)	individuals/day
β	Transmission rate	0.1-0.5	per day
ν	Vaccination rate	0.001-0.01	per day
h	Vaccine hesitancy parameter	0.1-0.5	None
γ	Recovery rate	0.1-0.2	per day
ω	Waning immunity rate	0.001 -0.05	per day
μ	Natural death rate	0.01 (0.0001)	per day

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The initial data adopted is $S(0) = 800.000, I(0) = 10, R(0) = 5.000, V(0) = 195.000$

III. ANALYSIS OF THE MODEL

3.1 Existence and Uniqueness of Solutions the Model of Disease Dynamics

Theorem: Consider the system of differential equations (1) – (4) where S, I, R, V represent the susceptible, infected, recovered, and vaccinatedpopulations, respectively, and the parameters $\Lambda, N, \beta, \nu, h, \mu, \gamma, \omega$ are positive constants.

Let $\boldsymbol{X} = [S, I, R, V]^T$. If the initial conditions $\mathbf{X}(0) = [S(0), I(0), R(0), V(0)]^T$ are such that $S(0), I(0), R(0), V(0) \geq 0$, then: there exists a unique solution $\boldsymbol{X}(t)$ to the system of ODEs (1) – (4) in some interval $t \in [0, T)$ for some $T > 0$.

Proof: The System is expressed in vector form as:

$$\frac{d\boldsymbol{X}}{dt} = \boldsymbol{F}(\boldsymbol{X}), (5)$$

where

$$\boldsymbol{F}(\boldsymbol{X}) = \begin{bmatrix} \Lambda N - \beta \frac{SI}{N} - (1-h)\nu S - \mu S \\ \beta \frac{SI}{N} - \gamma I - \mu I \\ \gamma I - \omega R - \mu R \\ (1-h)\nu S - \mu V \end{bmatrix} (6)$$

The right-hand side functions F_1, F_2, F_3, F_4 are composed of rational and polynomial functions of $S, I, R,$and V. Each function is continuous for all non-negative values of these variables.Since all functions are continuous, the continuity condition is satisfied.

The partial derivatives of the functions becomes,

$$\begin{aligned} \frac{\partial F_1}{\partial S} &= -\beta \frac{I}{N} - (1-h)\nu - \mu & \frac{\partial F_1}{\partial I} &= -\beta NS \\ \frac{\partial F_2}{\partial I} &= \beta \frac{S}{N} - \gamma - \mu & \frac{\partial F_2}{\partial S} &= \beta NI \\ \frac{\partial F_3}{\partial I} &= \gamma - \mu & \frac{\partial F_3}{\partial R} &= -\omega - \mu \\ \frac{\partial F_4}{\partial S} &= (1-h)\nu & \frac{\partial F_4}{\partial V} &= -\mu \end{aligned} \quad (7)$$

These derivatives are bounded for all $S, I, R, V \geq 0$ because $\beta, \gamma, \mu, \nu, \omega$are constants.

Thus,

$$\| \boldsymbol{F}(\boldsymbol{x}) - \boldsymbol{F}(\boldsymbol{y}) \| \leq L \| \boldsymbol{x} - \boldsymbol{y} \| \text{ for any } \boldsymbol{x}, \boldsymbol{y} \in R^4 (8)$$

$$L = \max_{i \in [1,4]} \frac{\partial F_i}{\partial \Omega}, i = 1..4, \Omega = \{S, I, R, V\} (9)$$

$\boldsymbol{X} = [S, I, R, V]^T$. If the initial conditions $\mathbf{X}(0) = [S(0), I(0), R(0), V(0)]^T$ are such that $S(0), I(0), R(0), V(0) \geq 0$, then:

Therefore, the functions F_1, F_2, F_3, F_4 are continuous and satisfy the Lipschitz condition, there exists a unique solution $\boldsymbol{X}(t) = [S(t), I(t), R(t), V(t)]^T$ to the system of equations (1) – (4) for $t$in the interval $[0, T)$ for some $T > 0$, provided the initial conditions are non-negative:

$$\boldsymbol{X}(0) = [S(0) I(0) R(0) V(0)]^T \geq 0. (10)$$

This completes the proof.

3.2 Equilibria

(i). **Disease-Free Equilibrium (DFE):** The Disease-Free Equilibrium occurs when there is no infection in the population, i.e., $I = 0$. At the DFE, Set$I = 0, dI/dt = 0$, and solve for $S, R,$ and V. Solving the reduced system of equation (1) –(4), we have

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$$(S^0, I^0, R^0, V^0) = \left(\frac{\Lambda N}{\mu + (1-h)\nu}, 0, 0, \frac{(1-h)\nu\Lambda N}{\mu(\mu + (1-h)\nu)}\right) (11)$$

(ii). **Endemic Equilibrium (EE):** The Endemic Equilibrium occurs when $I \neq 0$. Therefore, we solve for S, I, R, and Vat the point where all derivatives are zero. This gives

$$(S^*, I^*, R^*, V^*) = \left(\frac{N(\gamma + \mu)}{\beta}, \frac{\Lambda N}{(\gamma + \mu)} - \frac{N\big((1-h)\nu + \mu\big)}{\beta}, \frac{\gamma I^*}{\omega + \mu}, \frac{(1-h)\nu S^*}{\mu}\right) (12)$$

3.3 Reproduction Number:

(i). Basic Reproduction Number, R0

The basic reproduction number, R0, is defined as the average number of secondary infections produced by a single infected individual in a fully susceptible population. It is occurred when $S^0 \leq S^*$, where S^0 is the number of susceptible before the breakout of the disease and S^* is the number of susceptible after the breakout of the disease.

Thus $R_0 = S^*/S^0$

$$R_0 = \frac{S^*}{S^0} = \frac{\Lambda N}{\mu + (1-h)\nu} \Big/ \frac{N(\gamma + \mu)}{\beta} = \frac{\Lambda\beta}{(\mu + (1-h)\nu)(\gamma + \mu)}$$

$$\Rightarrow R_0 = \frac{\Lambda\beta}{(\mu + (1-h)\nu)(\gamma + \mu)} (13)$$

(ii). **Effective Reproduction Number, R_e**:The effective reproduction number, Re, accounts for the current level of immunity in the population and is given by:

$$R_e = R_0\left(\frac{S}{N}\right) (14)$$

Where S represents the current number of susceptible individuals.

3.4 Stability Analysis

To assess the local stability, we need to analyze the Jacobian matrix evaluated at the DFE and EE.

Jacobian Matrix: The Jacobian matrix $J$for the system under consideration is:

$$J = \begin{bmatrix} \frac{\partial f_1}{\partial S}\frac{\partial f_1}{\partial I} & \frac{\partial f_1}{\partial R} & \frac{\partial f_1}{\partial V} \\ \frac{\partial f_2}{\partial S}\frac{\partial f_2}{\partial I} & \frac{\partial f_2}{\partial R} & \frac{\partial f_2}{\partial V} \\ \frac{\partial f_3}{\partial S}\frac{\partial f_3}{\partial I} & \frac{\partial f_3}{\partial R} & \frac{\partial f_3}{\partial V} \\ \frac{\partial f_4}{\partial S}\frac{\partial f_4}{\partial I} & \frac{\partial f_4}{\partial R} & \frac{\partial f_4}{\partial V} \end{bmatrix} = \begin{bmatrix} -\beta\frac{I}{N} - (1-h)\nu - \mu & -\beta\frac{S}{N} & 0 & 0 \\ \beta\frac{I}{N} & \beta\frac{S}{N} - \gamma - \mu & 0 & 0 \\ 0 & \gamma & \omega - \mu & 0 \\ (1-h)\nu & 0 & 0 & -\mu \end{bmatrix} (15)$$

where f_1, f_2, f_3,and f_4 are the right-hand sides of the differential equations for S, I, R, and V, respectively.

Evaluating (15) at DFE (11), the Jacobian matrix becomes:

$$J = \begin{bmatrix} -(1-h)\nu - \mu & -\frac{\Lambda\beta}{\mu + (1-h)\nu} & 0 & 0 \\ 0 & \frac{\Lambda\beta}{\mu + (1-h)\nu} - \gamma - \mu & 0 & 0 \\ 0 & \gamma & \omega - \mu & 0 \\ (1-h)\nu & 0 & 0 & -\mu \end{bmatrix} \quad (16)$$

The characteristic equation OF (16) is given by:

$$det(J - \lambda I) = 0 \quad (17)$$

Solving (17**), the** eigenvalues areobtained as follows:

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$$\lambda_1 = -(1-h)\nu - \mu, \lambda_2 = \frac{\Lambda\beta}{\mu + (1-h)\nu} - \gamma - \mu, \lambda_3 = -\omega - \mu, \lambda_4 = -\mu \quad (18)$$

From

$$\lambda_2 = \frac{\Lambda\beta}{\mu + (1-h)\nu} - \gamma - \mu = (\gamma + \mu)\left(\frac{\Lambda\beta}{\mu + (1-h)\nu(\gamma + \mu)} - 1\right) = (\gamma + \mu)(R_0 - 1)$$

Since all the parameters are positive and $0 \leq h \leq 1$ and

$$\lambda_2 = (\gamma + \mu)(R_0 - 1) \quad (19)$$

Therefore, all eigenvalues will have negative real partsif $R_0 < 1$.

Hence, the DFE is locally stable if $R_0 < 1$. If $R_0 > 1$, the DFE is unstable since $\lambda_2 > 0$.

Thus, R_0 acts as the threshold parameter for the stability of the disease-free equilibrium.

3.5 Stability of the endemic equilibrium

To determine the stability of the endemic equilibrium, we again use the Jacobian matrix evaluated at the endemic equilibrium.Evaluating (15) at (12)

$$J = \begin{bmatrix} -\frac{\Lambda\beta}{(\gamma + \mu)} - (1-h)\nu - \mu - \lambda & -(\gamma + \mu) & 0 & 0 \\ \frac{\Lambda\beta}{(\gamma + \mu)} & 0 - \lambda & 0 & 0 \\ 0 & \gamma & -\omega - \mu - \lambda & 0 \\ (1-h)\nu & 0 & 0 & -\mu - \lambda \end{bmatrix} \quad (20)$$

which imply

$$\left(-\frac{\Lambda\beta}{(\gamma + \mu)} - (1-h)\nu - \mu - \lambda\right)\begin{vmatrix} -\lambda & 0 & 0 \\ \gamma & -\omega - \mu - \lambda & 0 \\ 0 & 0 & -\mu - \lambda \end{vmatrix} + (\gamma + \mu)\begin{vmatrix} \frac{\Lambda\beta}{(\gamma + \mu)} & 0 & 0 \\ 0 & -\omega - \mu - \lambda & 0 \\ (1-h)\nu & 0 & -\mu - \lambda \end{vmatrix}$$

That is

$$\left(-\lambda\left(-\frac{\Lambda\beta}{(\gamma + \mu)} - (1-h)\nu - \mu - \lambda\right) + (\gamma + \mu)\left(\frac{\Lambda\beta}{(\gamma + \mu)}\right)\right)(-\omega - \mu - \lambda)(-\mu - \lambda)$$

$$\left(-\lambda\left(-\frac{\Lambda\beta}{(\gamma + \mu)} - (1-h)\nu - \mu - \lambda\right) + (\gamma + \mu)\left(\frac{\Lambda\beta}{(\gamma + \mu)}\right)\right) = 0$$

Or

$$(-\omega - \mu - \lambda)(-\mu - \lambda) = 0$$

From

$$\lambda^2 + \lambda\left(\frac{\Lambda\beta}{(\gamma + \mu)} + (1-h)\nu + \mu\right) + \Lambda\beta = 0$$

$$\lambda = \frac{-\left(\frac{\Lambda\beta}{(\gamma + \mu)} + (1-h)\nu + \mu\right) \pm \sqrt{\left(\frac{\Lambda\beta}{(\gamma + \mu)} + (1-h)\nu + \mu\right)^2 - 4\Lambda\beta}}{2}$$

From where

$$\lambda_{1,2} = \frac{-\left(\frac{\Lambda\beta}{(\gamma + \mu)} + (1-h)\nu + \mu\right) \pm \sqrt{\left(\frac{\Lambda\beta}{(\gamma + \mu)} + (1-h)\nu + \mu\right)^2 - 4\Lambda\beta}}{2}$$

Therefore,

$$\lambda_1 = \frac{-\left(\frac{\Lambda\beta}{(\gamma + \mu)} + (1-h)\nu + \mu\right) + \sqrt{\left(\frac{\Lambda\beta}{(\gamma + \mu)} + (1-h)\nu + \mu\right)^2 - 4\Lambda\beta}}{2}$$

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$$\lambda_2 = \frac{-\left(\frac{\Lambda\beta}{(\gamma+\mu)} + (1-h)\nu + \mu\right) - \sqrt{\left(\frac{\Lambda\beta}{(\gamma+\mu)} + (1-h)\nu + \mu\right)^2 - 4\Lambda\beta}}{2}$$

$$\lambda_3 = -\omega - \mu, \lambda_4 = -\mu$$

This imply that all eigenvalues have negative real parts, the endemic equilibrium is locally stable.

3.6 Global Stability of the System

Theorem: Global Stability of the Endemic Equilibrium for the SIRV model described by the system of equations (1) – (4), for any initial conditions $(S(0), I(0), R(0), V(0))$ as $t \to \infty$, the endemic equilibrium (12) is globally asymptotically stable.

Proof: Let a Lyapunov function be:

$$F(S,I,R,V) = a_1\left(S - S^* - S^* ln\frac{S}{S^*}\right) + a_2\left(I - I^* - I^* ln\frac{I}{I^*}\right) + a_3\left(R - R^* - R^* ln\frac{R}{R^*}\right) + a_4\left(V - V^* - V^* ln\frac{V^*}{V}\right) \quad (21)$$

where a_1, a_2, a_3, and a_4 are positive constants.

This function is non-negative and vanishes only at the endemic equilibrium (S^*, I^*, R^*, V^*), since the expressions $x - x^* - x^* ln\frac{x}{x^*}$ are non-negative for all x and equal zero only when $x = x^*$.

The time derivative of F along the trajectories of the system is given by:

$$\frac{dF}{dt} = a_1\left(1 - \frac{S}{S^*}\right)\frac{ds}{dt} + a_2\left(1 - \frac{I}{I^*}\right)\frac{dI}{dt} + a_3\left(1 - \frac{R}{R^*}\right)\frac{dR}{dt} + a_4\left(1 - \frac{V}{V^*}\right)\frac{dV}{dt}. \quad (22)$$

Substitute the right-hand sides of the equations (1) – (4):

1. For the S −component:

$$\frac{dF}{dt} = a_1\left(1 - \frac{S}{S^*}\right)\left(\Lambda N - \beta\frac{S(t)I(t)}{N} - (1-h)\nu S(t) - \mu S(t)\right) + a_2\left(1 - \frac{I}{I^*}\right)\left(\beta\frac{S(t)I(t)}{N} - \gamma I(t) - \mu I(t)\right) \quad (23)$$

$$+a_3\left(1 - \frac{R}{R^*}\right)\left(\gamma I(t) - \omega R(t) - \mu R(t)\right) + a_4\left(1 - \frac{V}{V^*}\right)\left((1-h)\nu S(t) - \mu V(t)\right)$$

At the endemic equilibrium point, (23) becomes

$$\frac{dF}{dt} = 0$$

The function is designed to decrease along trajectories of the system, except at the endemic equilibrium, where it equals zero. This ensures that:

$$\frac{dF}{dt} < 0 \text{ for all} (S,I,R,V) \neq (S^*, I^*, R^*, V^*).$$

Hence the endemic equilibrium point is global asymptotic stable.

3.6 Sensitivity Analysis

To determine the sensitivity indices of each parameter on the quantities $R^0, S^0, I^0, R^0, V^0, S^*, I^*, R^*, V^*$, we use the concept of "normalized sensitivity indices". The sensitivity index of a parameter p with respect to a quantity Q is given by:

$$\text{Sensitivity Index} = \frac{\partial Q}{\partial p}\cdot\frac{p}{Q}$$

We will compute the sensitivity indices for each parameter $(\Lambda, N, \beta, h, \nu, \mu, \gamma, \omega)$ on the quantities $R^0, S^0, I^0, R^0, V^0, S^*, I^*, R^*, V^*$.

1. Sensitivity Indices for $\boldsymbol{R}_0$

$$R_{0_\Lambda} = 1 \qquad R_{0_\beta} = 1 \qquad R_{0_h} = -\frac{(1-h)\nu}{\mu + (1-h)\nu} \qquad R_{0_\gamma} = -\frac{\gamma}{\gamma + \mu}$$

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$$R_{0_\nu} = -\frac{(1-h)\nu}{\mu+(1-h)\nu} \quad R_{0_\mu} = -\left[\frac{\gamma+\mu+(1-h)\nu}{\gamma+\mu}\cdot\frac{\mu}{\mu+(1-h)\nu}\right]$$

2. Sensitivity Indices for S_0

$$S_\Lambda^0 = 1 \quad S_N^0 = 1 \quad S_h^0 = -\frac{(1-h)\nu}{\mu+(1-h)\nu} \quad S_\nu^0 = -\frac{(1-h)\nu}{\mu+(1-h)\nu}$$

$$S_\mu^0 = -\frac{\mu}{\mu+(1-h)\nu}$$

3. Sensitivity Indices for V^0

$$V_\Lambda^0 = 1 \quad V_N^0 = 1 \quad V_h^0 = -\frac{(1-h)\nu}{\mu+(1-h)\nu} \quad V_\nu^0 = \frac{\nu}{\mu+(1-h)\nu} \quad V_\mu^0 = -\frac{\mu}{\mu+(1-h)\nu}$$

4. Sensitivity Indices for S^*

$$S_N^* = 1 \quad S_\beta^* = -1 \quad S_\mu^* = -\frac{\mu}{\gamma+\mu} \quad S_\gamma^* = \frac{\mu}{\gamma+\mu}$$

5. Sensitivity Indices for I^*

$$I_N^* = \frac{N\cdot\left(\Lambda\beta-(\gamma+\mu)\left((1-h)\nu+\mu\right)\right)}{\Lambda N\beta - N(\gamma+\mu)\cdot\left((1-h)\nu+\mu\right)} \qquad I_\beta^* = -\frac{\beta}{\Lambda\beta-(\gamma+\mu)\left((1-h)\nu+\mu\right)}$$

$$S_\beta^{R^*} = \frac{N(\gamma+\mu)(\mu+(1-h)\nu)}{\beta\left(\Lambda N - \frac{N(\gamma+\mu)(\mu+(1-h)\nu)}{\beta}\right)} \qquad I_h^* = -\frac{(1-h)\nu}{\mu+(1-h)\nu} \qquad I_\Lambda^* = 1 \qquad S_\gamma^{R^*} = 1$$

6. Sensitivity Indices for R^*

$$S_\mu^{R^*} = \mu\frac{\left[-(\gamma+\mu)-\frac{\Lambda(\beta(\gamma+\mu)+N(\mu+\omega))}{(\mu+\omega)(\gamma+\mu)}+(\mu+(1-h)\nu)\left((\gamma-\omega)+\frac{N}{\beta}\right)\right]}{\Lambda\beta-(\gamma+\mu)(\mu+(1-h)\nu)}$$

$$S_\nu^{R^*} = -N\nu(1-h)(\gamma+\mu)/(\beta\left(\Lambda N - \frac{N(\gamma+\mu)(\mu+(1-h)\nu)}{\beta}\right)$$

$$S_\Lambda^{R^*} = \frac{\beta\Lambda}{\beta\Lambda-(\gamma+\mu)(\mu+(1-h)\nu)}, \quad S_\omega^{R^*} = -\frac{\omega}{\mu+\omega}$$

$$S_h^{R^*} = \frac{Nh\nu(\gamma+\mu)}{\beta\left(\Lambda N - \frac{N(\gamma+\mu)(\mu+(1-h)\nu)}{\beta}\right)}$$

Sensitivity Indices for V^*

$$S_\beta^{V^*} = -1, \quad S_\gamma^{V^*} = \frac{\gamma}{\gamma+\mu}, \quad S_\mu^{V^*} = \frac{\gamma}{(\gamma+\mu)}, \quad S_h^{V^*} = -\frac{h}{1-h}, \quad S_\nu^{V^*} = 1$$

Table 2: Sensitivity Index

Parameter	R_0	S^0	V^0	S^*	I^*	R^*	V^*
$\Lambda = 0.020$	1.000	1.000	1.000	1.000	1.000	2.497	0.000
$\beta = 0.100$	1.000	0.000	0.000	-1.000	-124.844	1.000	-1.000
$\nu = 0.001$	-0.083	-0.083	0.092	0.000	0.000	-0.124	1.000
$h = 0.100$	-0.083	-0.083	-0.083	0.000	-0.083	0.014	-0.111
$\gamma = 0.100$	-0.909	0.000	0.000	0.091	0.000	0.000	0.909
$\omega = 0.001$	0.000	0.000	0.000	0.000	0.000	-0.091	0.000
$\mu = 0.010$	-0.925	-0.925	-0.917	-0.091	0.000	-90912.721	0.909

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IV. NUMERICAL SOLUTION

In other to access the numerical simulation of model under consideration, a numerical method RKF45 method, short for Runge-Kutta-Fehlberg 4th and 5th order method is used. This adaptive method combines the advantages of both the 4th-order and 5th-order Runge-Kutta methods, allowing it to provide accurate solutions while dynamically adjusting the step size based on the estimated error at each step. By estimating the solution using both the 4th and 5th orders, RKF45 can effectively determine the most suitable step size for the next iteration, thus balancing computational efficiency and accuracy. This adaptability makes it particularly valuable for problems where the solution exhibits rapid changes or where precise results are crucial. Consequently, RKF45 is implemented using Maple inbuild ode solver, Ascher and Petzold (1998).

The `dsolve` command, utilizing the `numeric` option or `type=numeric`, facilitates the computation of numerical solutions for ordinary differential equations (ODEs) or systems of ODEs subject to initial value problems (IVPs)Shampine and Corless (2000). Furthermore, the integration of the `odeplot` function enables the visualization of the resulting graphs, providing a comprehensive representation of the dynamics inherent in the mathematical model of polio transmission. This approach ensures a robust and systematic exploration of the ODEs, contributing to a deeper understanding of the underlying epidemiological processes.

V. RESULT AND DISCUSSION

5.1 Numerical Results

In this model, the population dynamics are governed by the flow of individuals between four compartments: susceptible (S), infected (I), recovered (R), and vaccinated (V).

This model effectively captures the dynamics of polio transmission and control within a population. Population dynamics depicted in Fig. 1 shows the dynamics between the subclasses in the system. It could be observed that withing the first 2-years, fluctuations in subclasses persist, which suggest a shifts in disease prevalence, immunity levels, and the effectiveness of vaccination efforts over time. The peak in S, I, R profiles (Fig. 1) indicate periods of increased infection risk if the susceptible population grows as the vaccinated population declines due to waning immunity or vaccine hesitancy. An increase in recovered individuals signals an outbreak phase that has led to natural immunity.

Birth rate (Λ) plays a fundamental role in the dynamics of infectious disease models by continuously adding new individuals to the susceptible compartment, thus impacting the overall population structure. A steady influx of new, unexposed individuals increases the pool of susceptible individuals as shown in Fig. 2, providing a continuous source of potential hosts for the disease. This influx is particularly influential in the persistence of polio diseases (Fig. 3), the disease has an ongoing opportunity to spread, as the susceptible population remain high even in the presence of other control strategies. Which also imply increase in Vaccinated class as more individuals is required to be vaccinated, Fig. 4. An increase in birth rate, therefore, lead to higher infection rates and potentially an increased basic reproduction number (R0), as it sustains a larger proportion of individuals who are at risk. Balancing birth rates with effective vaccination coverage is essential to controlling long-term disease dynamics.In Fig. 5, we displayed the effect of transmission rate on infected class. In this model, a high transmission rate indicates that infected individuals are more easily transmit the disease to susceptible individuals, thereby accelerating the spread of infection. A peak in the profile indicates a higher transmission rate could result into epidemics during the disease outbreak. This heightened transmission raises the overall infection rate within the population, increasing the likelihood that a single infection will lead to secondary cases. Reducing the transmission rate, whether through interventions like vaccination, social distancing, or hygiene measures, can thus significantly impact the overall progression of the disease. A lower βeffectively reduces the basic reproduction number (R0), making it harder for the infection to persist in the population and aiding in control efforts.

The vaccination rate (**v**) determines the rate at which susceptible individuals are transferred into the vaccinated compartment Fig. 6, effectively reducing the number of individuals at risk of infection. High vaccination rates are pivotal in disease control as they reduce the susceptible population, thereby lowering the potential for disease spread.In polio models, increasing νdirectly helps to prevent new infections, as more individuals are rendered immune through vaccination rather than exposure to the infection. This process also contributes to achieving herd immunity, as it lowers the probability that an

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infected person will encounter a susceptible individual. High vaccination rates are essential to achieving eradication goals for polio, as they prevent new infections from sustaining transmission cycles. Ensuring consistent and high vaccination coverage can counterbalance high birth rates or other factors that increase susceptibility within a population. Vaccine hesitancy (h) represents the proportion of individuals who, despite being eligible, avoid or delay vaccination. This parameter has gained attention due to its influence on disease dynamics, especially in the context of achieving herd immunity. When his high, the susceptible population remains larger than expected under an ideal vaccination program Fig. 7, creating a wider window for potential outbreaks. For polio, high vaccine hesitancy can jeopardize eradication efforts by maintaining a susceptible population large enough to sustain transmission. Vaccine hesitancy also indirectly impacts other model parameters, as it interacts with vaccination rates, leading to less effective control even with high vaccination availability Fig. 8. Addressing vaccine hesitancy through public health education, trust-building, and addressing misinformation is essential to achieving herd immunity and ensuring effective disease control.

In polio dynamics, a high recovery rate reduces the duration of infectiousness, effectively decreasing the likelihood of transmission to others Fig. 9. This shorter infectious period helps to lower the infection's spread by reducing the number of secondary infections an infected person can generate. In this model, increasing γlower the basic reproduction number, potentially reducing the persistence of infection within a population. Furthermore, if immunity post recovery is long-lasting or permanent, a high recovery rate can substantially aid control efforts by maintaining a higher proportion of immune individuals in the population. Waning immunity can undermine control efforts by continuously replenishing the susceptible compartment, particularly in settings where booster vaccinations or ongoing exposure are not maintained. A higher ω rate implies that individuals lose immunity more quickly, potentially leading to reduction in recovery class and resulted in periodic outbreaks as immunity levels wane across the population. This phenomenon highlights the importance of understanding immunity duration and considering booster doses or natural immunity through exposure to maintain long-term population protection.

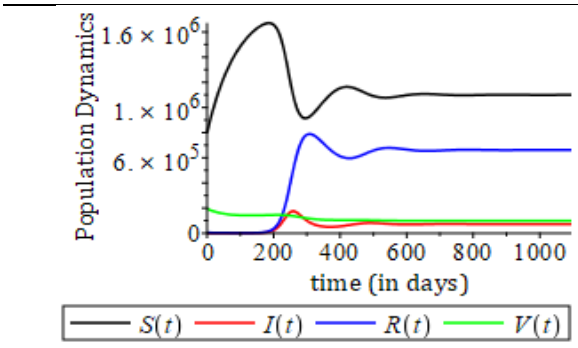

Fig. 1: Population Dynamics of Polio

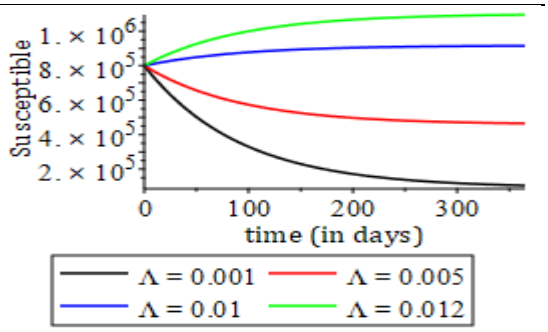

Fig. 2: Impact of Birth/recruitment rate on susceptible class

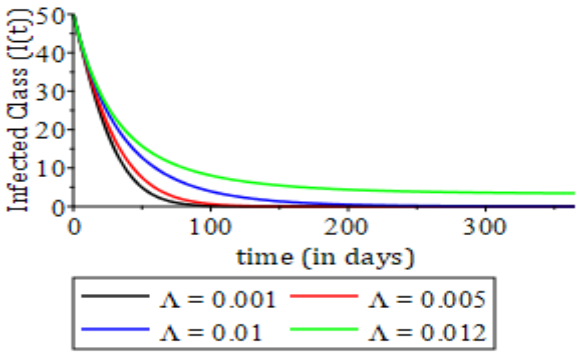

Fig. 3: Impact of Birth/recruitment rate on infected

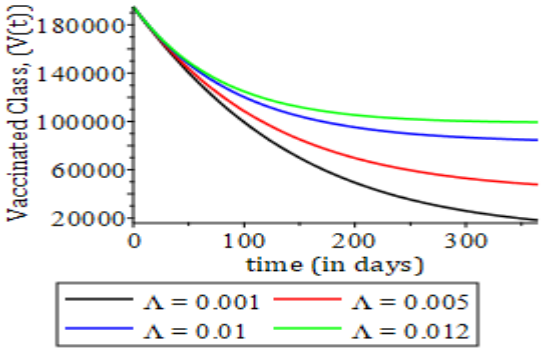

Fig. 4: Impact of Birth/recruitment rate on vaccinated class

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class

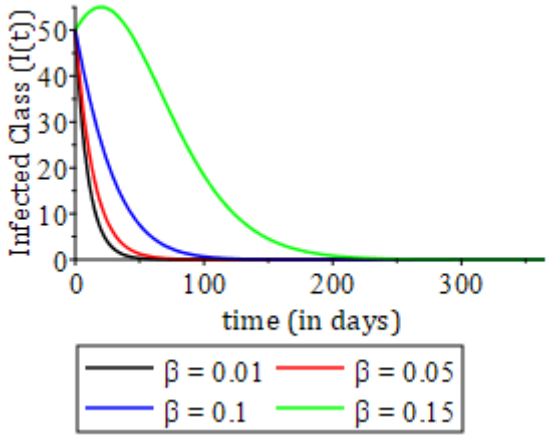

Fig. 5: Effect oftransmission rate on infected class

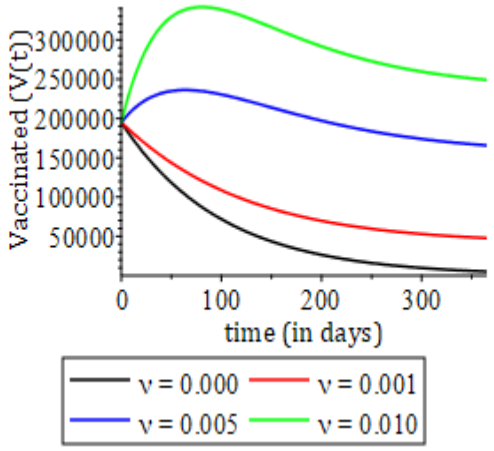

Fig. 6: Contribution of vaccination rate on vaccinated class

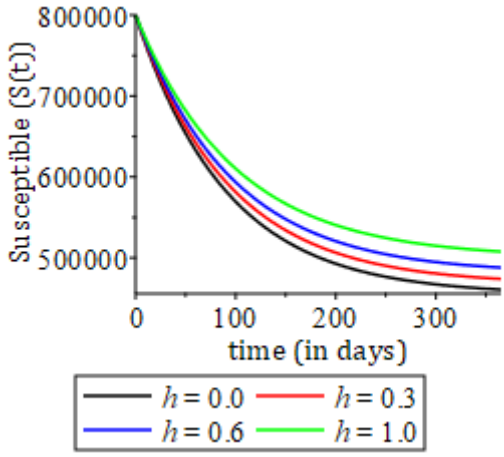

Fig. 7: Impact ofVaccine hesitancy parameter on susceptible class

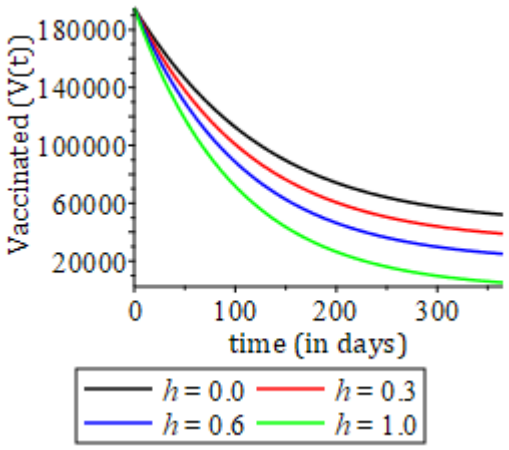

Fig. 8: Impact ofVaccine hesitancy parameter on vaccinated class

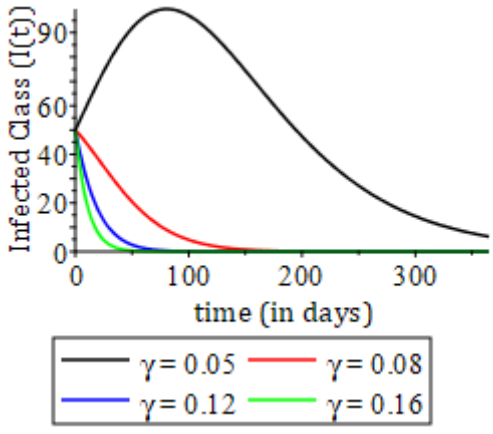

Fig. 9: Effect of Recovery rate on infected class

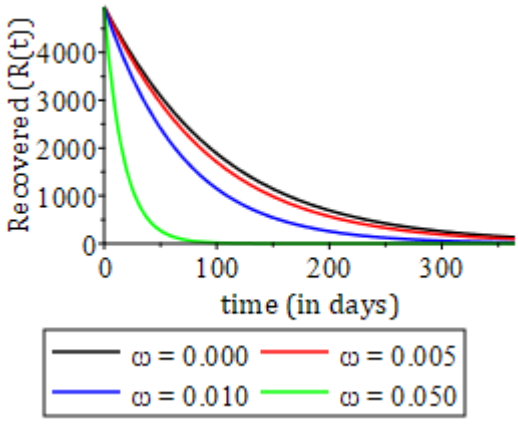

Fig. 10: Influence of wanning immunity rate on recovery class

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5.2 Sensitivity Analysis

The sensitivity of parameters on the basic reproduction number R0 provides valuable insights into how changes in each parameter affect the potential for disease transmission within a population.

5.2.1 Sensitivity index of R_0

The sensitivity index of R_0 with respect to the birth rate (Λ) is positive and equal to 1, indicating a direct, strong relationship where an increase in birth rate raises the susceptible population, thus enhancing transmission potential. This underscores the importance of birth rate management in disease control. Similarly, the sensitivity of R_0 to the transmission rate (β) is positive and also equal to 1, showing that a higher transmission rate allows infected individuals to infect more susceptible, increasing R_0 . This emphasizes the need for interventions to reduce transmission. The sensitivity index for vaccination rate (ν) is negative (-0.083), suggesting that increased vaccination reduces R_0 slightly by controlling the susceptible population, while vaccine hesitancy (h) also has a negative impact (-0.083), as it limits vaccination uptake, maintaining a larger susceptible population. Recovery rate (γ) shows a strong inverse effect on R0 with a sensitivity of -0.909, as quicker recovery reduces transmission time, highlighting the importance of effective healthcare. The waning immunity rate (ω) shows no sensitivity (0), suggesting minimal impact on R_0 at low rates. Lastly, the natural death rate (μ) has a strong negative sensitivity of -0.925, as increased mortality decreases the susceptible and infected compartments, lowering R_0, though this raises population health concerns. This reveals that the parameters most critical to controlling R_0 are the recovery rate and the natural death rate, which significantly reduce transmission potential when increased. Conversely, both the birth and transmission rates are positively correlated with R_0, emphasizing the need for effective public health interventions that target these areas to control disease spread effectively.

5.2.2 Sensitivity of the susceptible population at disease-free equilibrium (DFE)

The sensitivity of the susceptible population at disease-free equilibrium (DFE), S0, to the birth rate (Λ) is positive and equal to 1, showing a direct relationship where increased births elevate the susceptible population, creating a reservoir for potential disease spread. This underscores the role of managing birth rates in controlling transmission. The sensitivity to the transmission rate (β) is 0, meaning transmission dynamics affect spread but not the baseline count of susceptible at DFE, highlighting that lowering transmission primarily affects infection dynamics rather than susceptibility.

The sensitivity index to vaccination rate (ν) is slightly negative, showing that vaccination effectively reduces susceptibility, even if the impact is modest. As vaccination increases, fewer remain susceptible, supporting public health efforts. Vaccine hesitancy (h) similarly shows negative sensitivity, as increased hesitancy enlarges the susceptible group, indicating a need for strategies to improve vaccine acceptance. Lastly, the natural death rate (μ) has a strongly negative sensitivity, suggesting that higher mortality reduces susceptibility by decreasing the overall population, though raising ethical concerns around long-term public health stability. This analysis reveals that the birth rate has the most substantial positive impact on the initial susceptible population at DFE, while the natural death rate significantly reduces the number of susceptible individuals. Conversely, the vaccination rate and vaccine hesitancy slightly influence susceptibility, emphasizing the importance of vaccination programs and addressing vaccine hesitancy to maintain lower levels of susceptibility within the population. Understanding these sensitivities is crucial for devising effective public health strategies to control infectious diseases.

5.2.3Sensitivity of vaccination levels (V^0) at Disease-FreeEquilibrium (DFE)

The sensitivity of vaccination levels (V^0) at Disease-Free Equilibrium (DFE) to various parameters provides insights into how these factors influence vaccine uptake in a population. A positive sensitivity to birth rate implies that higher birth rates increase the pool eligible for vaccination, underscoring the need to consider population growth in maintaining effective coverage. While the transmission rate sensitivity is zero, suggesting it does not directly affect vaccination at DFE, reducing transmission remains key for controlling disease spread. A positive sensitivity to the vaccination rate itself suggests that even slight increases in vaccination efforts can significantly elevate immunity levels, supporting prevention. Conversely, vaccine hesitancy has a negative sensitivity, meaning higher hesitancy reduces vaccination, underscoring the importance of

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education and outreach to address it. The natural death rate's negative sensitivity indicates that higher mortality can reduce the pool for vaccination, impacting coverage. Overall, birth rate and vaccination rate positively impact vaccination levels, while vaccine hesitancy and natural death rate reduce it. These findings highlight the importance of targeted strategies to encourage vaccination, address hesitancy, and consider population dynamics to enhance vaccine coverage and control disease effectively.

5.2.4 Sensitivity of the susceptible population ($S*$) at EndemicEquilibrium

The sensitivity of the susceptible population ($S*$) at Endemic Equilibrium to various parameters reveals key influences on its size. The birth rate has a positive sensitivity, indicating that an increase in birth rate directly enlarges the susceptible pool, thereby sustaining potential transmission. Public health interventions should consider birth rates to manage new susceptibles, especially for vaccination strategies. In contrast, the transmission rate has a negative sensitivity, as a higher rate reduces the susceptible population by accelerating infections, underscoring the importance of controlling transmission to stabilize the susceptible pool. The recovery rate has a modest positive impact, meaning faster recovery returns individuals to susceptibility, slightly increasing the pool. On the other hand, the natural death rate reduces the susceptible population, as higher mortality removes individuals from this group, affecting disease spread. Overall, birth and transmission rates are the most significant factors influencing the susceptible population size, while recovery and death rates have smaller impacts, and vaccination rate and hesitancy show minimal effects on susceptibles at this stage. Understanding these sensitivities is essential for designing public health strategies to manage susceptibility and control disease in endemic settings.

5.2.5 Sensitivity of the infected population (I^*) at EndemicEquilibrium

The sensitivity of the infected population ($I*$) at Endemic Equilibrium to various parameters offers essential insights into infection dynamics. With a sensitivity index of 1.000, the birth rate shows a direct relationship with the infected population, indicating that higher birth rates increase infections by introducing more susceptible. This risks among new cohorts. Conversely, the transmission rate exhibits an extremely high negative sensitivity, meaning that even slight increases in transmission control measures (e.g., hygiene, distancing, vaccination) significantly reduce infections. This parameter is thus critical for epidemic control as reducing transmission rates can dramatically lower infection levels. The sensitivity of vaccine hesitancy, though negative, suggests that increased hesitancy marginally raises infections, as fewer individuals are vaccinated, leaving a larger susceptible pool. Addressing hesitancy through community engagement is crucial to boost vaccine uptake and lower infection rates. Overall, birth and transmission rates are the primary influences on endemic infection levels, with transmission control being pivotal for reducing infection burdens. Vaccine hesitancy remains a significant factor, emphasizing the need for public health strategies to enhance vaccination coverage.

5.2.6 Sensitivity analysis of the recovery population (R^*) atEndemic Equilibrium

The sensitivity analysis of the recovery population (R^*) at Endemic Equilibrium reveals how parameter changes impact the dynamics of recovered individuals. A strong positive sensitivity index (2.497) for the birth rate suggests that as birth rates increase, so does the recovery population, as newersusceptible enter, potentially get infected, and eventually recover. This underscores the importance of adequate health infrastructure and vaccination efforts for incoming cohorts to bolster recovery. Similarly, the transmission rate's sensitivity (1.000) shows that higher transmission results in more infected individuals who recover, stressing the value of transmission control to maintain manageable infection and recovery rates.

Conversely, the vaccination rate's negative sensitivity suggests that increasing vaccination reduces recovery, as fewer infections occur in the first place. This highlights vaccination's preventive focus over recovery-driven disease management. Vaccine hesitancy has a minimal positive sensitivity, indicating little immediate impact on recovery but suggesting long-term gains with improved vaccine uptake. The negative sensitivity of the waning immunity rate implies that higher rates of lost immunity reduce the recovered population, reinforcing the need for durable immunity strategies, such as boosters. Finally, the very high negative sensitivity of the natural death rate underscores how elevated mortality reduces the recovery pool, underscoring the importance of addressing mortality for

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sustained recovery rates. Overall, this analysis emphasizes that comprehensive strategies integrating infection control, vaccination, and mortality reduction are essential for supporting recovery and maintaining population health.

5.2.7 Sensitivity analysis of the vaccination population (V^*)

The sensitivity analysis of the vaccination population (V^*) at endemic equilibrium reveals how various parameters impact vaccination uptake, showing that transmission, vaccination efforts, recovery rates, and community perceptions shape immunization dynamics. A negative sensitivity index of -1.000 for the transmission rate indicates that higher transmission rates reduce vaccination uptake, as resources may shift toward outbreak control, underscoring the need for transmission management to support immunization. In contrast, a positive sensitivity of 1.000 for the vaccination rate shows that enhancing vaccination efforts directly increases coverage, affirming the critical role of well-resourced campaigns in public health. Vaccine hesitancy, with a modest negative

sensitivity, slightly decreases vaccination rates, indicating that targeted education and outreach can improve uptake. Additionally, a positive sensitivity (0.909) for the recovery rate suggests that as more people recover, vaccine uptake increases, possibly due to greater awareness of disease risks, highlighting an indirect benefit of recovery support on vaccination. Similarly, the same positive index for the natural death rate suggests that heightened mortality may prompt more aggressive vaccination campaigns, positioning immunization as a response to increased mortality threats. Altogether, controlling transmission, ensuring robust vaccination initiatives, supporting recovery, and engaging the community are essential to enhance vaccination rates and strengthen population health, demonstrating the intricate balance between disease dynamics and public health strategies.

5.2.8 Vaccine Hesitancy

Vaccine hesitancy profoundly affects polio spread by altering vaccination coverage levels, which, in turn, influence the dynamics of immunity and susceptibility within a population. In the absence of hesitancy ($h = 0$), vaccination rates are maximized, rapidly building immunity across the population and minimizing the number of susceptible individuals. This high coverage suppresses polio transmission efficiently, leading to a lower peak in infections and a swift decline in cases. Such conditions make it easier to approach or sustain herd immunity, thereby protecting those who cannot be vaccinated and reducing the risk of an outbreak.

Conversely, with moderate hesitancy ($h = 0.5$), fewer individuals receive the vaccine, leaving more people susceptible. This results in a higher infection peak, as the reduced immunity level cannot adequately curb polio spread. The decline in cases slows, extending the transmission period and increasing the likelihood of recurrent outbreaks. In scenarios of high hesitancy ($h = 0.9$), vaccination coverage drops significantly, leading to a sustained outbreak with a much higher peak in infections. The large pool of susceptible individuals perpetuates transmission, emphasizing the direct role of high vaccination coverage in controlling polio. These findings underscore that even moderate hesitancy can compromise herd immunity, with increased hesitancy posing a severe risk of persistent and intensified polio transmission.

VI. CONCLUSION

The research on evaluating vaccine hesitancy and its impact on polio transmission using mathematical models underscores the critical influence of vaccination coverage on disease dynamics and public health outcomes. Through a detailed sensitivity analysis of various vaccination hesitancy scenarios—from no hesitancy to high hesitancy—this study demonstrates how reluctance to vaccinate directly correlates with increased polio transmission rates, prolonged outbreaks, and challenges in achieving herd immunity. The model reveals that in the absence of hesitancy, robust vaccination campaigns significantly limit the number of infections, rapidly establishing a high level of immunity across the population. This outcome aligns with global public health efforts that emphasize the importance of maintaining high vaccination rates to keep diseases like polio at bay and protect vulnerable groups.

In moderate hesitancy scenarios, the model shows a less favorable outcome, with decreased vaccine uptake leaving more individuals susceptible to infection. This condition results in a higher peak of infections and a slower decline in case numbers, leading to extended transmission periods and heightening the likelihood of recurrent outbreaks. Moderate hesitancy, often stemming from concerns about vaccine safety, misinformation, or cultural beliefs, illustrates the need for targeted interventions that address specific

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causes of reluctance. Effective public health campaigns and education initiatives become essential in this context, as they can help reduce hesitancy by building public trust in vaccination. The model highlights that even a moderate drop in vaccine uptake can disrupt the progress toward eradicating polio, illustrating the narrow margin by which vaccination success can be maintained in susceptible communities.

High vaccine hesitancy (with values such as h=0.9) is shown in the model to be especially concerning, leading to substantial decreases in vaccination coverage and a dramatic increase in polio cases. Under these conditions, the number of susceptible individuals remains high, creating a fertile environment for sustained transmission and increasing the risk of severe outbreaks. Such findings underscore those high levels of hesitancy can nullify the benefits of vaccination, placing entire communities at risk and undermining years of public health progress. Additionally, the model emphasizes the compounding effects of vaccine hesitancy, as each hesitancy level creates a proportionate risk, with high hesitancy approaching near-endemic conditions. This scenario is particularly relevant in regions where trust in health institutions is fragile or where misinformation regarding vaccines proliferates.

The study's mathematical model serves as a valuable tool for understanding how even small shifts in vaccination behavior can dramatically influence disease transmission. It illustrates the intricate balance between vaccination uptake and infection rates, highlighting that maintaining high vaccine coverage is not merely a public health target but a necessity for sustaining polio control. Through sensitivity analyses of the birth rate, transmission rate, recovery rate, and vaccine hesitancy, the model further demonstrates that vaccine hesitancy can impede efforts to control transmission effectively, leaving populations vulnerable to resurgence and severe outbreaks. From the above discussions, the following deduction were made:

- Higher birth rates can increase the number of susceptible individuals, maintaining a population at risk for infection.
- Can sustain disease spread if not counterbalanced by high vaccination rates.
- High β accelerates infection spread, increasing disease prevalence.
- Reducing β is crucial for lowering the basic reproduction number (R0).
- High ν lowers the susceptible pool, reducing potential new infections.
- Higher h increases the susceptible population, sustaining potential transmission.
- Higher γ reduces the infectious period, leading to fewer secondary infections.
- High ω can lead to resurgence in infections by increasing the susceptible pool.
- Natural Death Rate (μ) - Applies to all compartments, affecting the overall population structure.
- Reduces susceptible and infected individuals, potentially slowing spread.
- Mathematical modeling highlights how vaccine hesitancy directly influences polio spread, with higher hesitancy leading to increased infection rates, prolonged outbreaks, and challenges in achieving herd immunity.
- Vaccine hesitancy levels have a profound impact on polio spread: no hesitancy enables high vaccination rates that limit infections and rapidly build immunity; moderate hesitancy results in higher infection peaks and prolonged transmission, highlighting the need for public health education; high hesitancy severely reduces coverage, leading to major outbreaks and prolonged transmission, potentially reversing public health gains.
- The model underscores that even small increases in vaccine hesitancy have disproportionately large effects on disease transmission, making high coverage essential to sustaining polio control.

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.

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REFERENCE

[1]. Agarwal, M., &Bhadauria, A. S. (2023). Modeling the spread of polio with vaccination dynamics. Applications and Applied Mathematics: An International Journal, 18(2), 714–736.

[2]. Eikenberry, S. E., Thunes, J., Del Valle, S. Y., & Munshi, S. P. (2021). Modeling the effect of hesitancy on polio eradication. Epidemics, 35, 100456.

[3]. Kasselman, S., Lindquist, K., & Wallace, H. (2023). Understanding vaccine reluctance and public health strategies. University of Michigan School of Public Health. Available online.
[4]. Lindquist, K. (2022). Addressing the social factors of vaccine hesitancy. Community and Global Public Health Journal, 6(2), 211–222.
[5]. Etsano, R., Gunnala, R., Nsubuga, P., Wannemuehler, K., Shuaib, F., Nguku, P., & Roy, S. (2021).
[6]. Ghinai, I., Crawford, T., Wiesen, E., & Mahoney, F. (2023). The influence of public health messaging on polio vaccination in Nigeria. BMC Public Health, 23, Article 145.
[7]. Larson, H., Jarrett, C., & Paterson, P. (2022). Vaccine hesitancy: Global impact and trends. Vaccine, 40(12), 1951–1962.
[8]. Bliss, C., & Morrison, R. (2022). The risks of misinformation and vaccine hesitancy during the COVID-19 crisis. Center for Strategic and International Studies. Available online.
[9]. Wallace, J. (2023). Community-centered approaches to vaccine uptake. Journal of Infectious Diseases, 228(5), 800–808.
[10]. Gleason, C. (2022). Factors contributing to vaccine hesitancy in low-resource settings. Journal of Public Health, 44(3), 580–588.
[11]. Shen, A., & Dubey, V. (2023). Vaccine hesitancy guidance for healthcare professionals. Canadian Family Physician, 69(4), 251–256.
[12]. Unraveling the transmission ecology of polio. (2024). Analysis of polio transmission dynamics using mechanistic models. PLOS Biology.
[13]. PLOS Biology. (2024). Unraveling the transmission ecology of polio. PLOS Biology, 22(4), e3002245.
[14]. Manju, A., & Archana, S. B. (2023). Numerical analysis of polio models with vaccination parameters. Applied Mathematics and Computation, 444, 127842.