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Impact of Age Structure and Vaccine Efficacy on the Control and Prevention of Diphtheria

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ABSTRACT
This study presents a comprehensive mathematical model that explores the influence of age structure and vaccine efficacy on diphtheria control and prevention. Extending the basic epidemiological framework, our model incorporates multiple compartments representing various states of individuals across different age groups. By integrating key parameters such as age-specific transmission rates, vaccination coverage, vaccine efficacy, and natural death rates, our analysis delves into realistic scenarios. Through examination of Disease-Free Equilibrium (DFE) and Endemic Equilibrium (EE), we uncover the conditions necessary for disease elimination and persistence. Our sensitivity analysis reveals critical parameters positively influencing disease endemicity, such as the birth rate (ρ), age-specific transmission rate (β_i), and rate of progression from exposed to infectious (σ). Conversely, parameters like the natural death rate (δ), vaccination coverage rate (υ_i), recovery rate (γ), and disease-induced death rate (α) exhibit negative sensitivity, suggesting their potential to reduce disease spread. We find that higher vaccination rates and increased vaccine efficacy significantly bolster disease control, with age-specific strategies targeting high-risk groups proving particularly impactful in lowering infection rates. These insights underscore the importance of tailored public health interventions in managing diphtheria spread effectively.

Keywords*: Age structure, Vaccine efficacy, Disease control, public health interventions, Sensitivity analysis*

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I. INTRODUCTION

Diphtheria is a highly contagious vaccine-preventable disease caused mainly by *Corynebacterium diphtheria* but also *by Corynebacterium ulcerans.* It spreads between people mainly by direct contact or through the air via respiratory droplets. The disease can affect all age groups; however, unimmunized children are most at risk (WHO, 2023). People who are at high risk of catching diphtheria includes children and adults who don't have up-to-date vaccinations, people living in crowded or unsanitary conditions and anyone who travels to an area where diphtheria infections are more common. Diphtheria is rare in the United States and Western Europe due to decades of widespread childhood vaccination. However, it remains prevalent in developing countries where vaccination rates are low (Mayo, 2023). Diphtheria infection is treated with the administration of a diphtheria antitoxin (DAT), which can be given intravenously or through an intramuscular injection. Antibiotics, such as penicillin or erythromycin, are also administered to eliminate the bacteria and prevent further transmission. Close contacts of the patient should be monitored for signs and symptoms for 10 days from the last contact, and healthcare workers exposed to the patient's secretions or wounds should also be monitored and may receive prophylactic antibiotics for seven days (NCDC, 2023). To prevent diphtheria, parents should ensure their children are fully vaccinated with three doses of the pentavalent vaccine as recommended in the childhood immunization schedule. The Nigeria childhood immunization schedule advises these doses at the 6th, 10th, and 14th weeks of life. Additionally, the WHO recommends a 3-dose series of diphtheria toxoid-containing vaccines beginning at 6 weeks of age, followed by three booster doses during childhood and adolescence to ensure long-term protection (NCDC, 2023).

The global burden of diphtheria has long been markedly reduced in developed countries across the world, with significant control over the past decades in low- and middle- income countries such as Nigeria. The introduction of the diphtheria vaccine into immunization programs has helped improve global efforts toward its eradication. However, Nigeria is currently experiencing an alarming surge in the number of recorded cases across the nation, a situation that calls for concern and indeed necessary intervention (Abdulrasheed et al., 2023).The diphtheria outbreak in Kimba village, northeastern Nigeria, from February to November 2011, was primarily driven by extremely low vaccination rates. Among the 98 cases identified, 98% had never been immunized against diphtheria, underscoring the critical gap in vaccination coverage. The outbreak investigation

revealed that the absence of booster vaccinations for older children and adults, along with the routine DTP coverage being less than 1% in 2011, contributed significantly to the epidemic's severity. The delayed recognition of the disease by clinicians unfamiliar with diphtheria, compounded by the lack of access to healthcare and the absence of effective treatments, further exacerbated the situation. The high case-fatality ratio, particularly among children aged 0–4 years, highlights the urgent need for improved immunization programs and healthcare access in remote areas to prevent such outbreaks in the future (Besa et al., 2014). Abubakar et al., (2019) carried out a study on diphtheria outbreak that underscores the critical role of vaccination in preventing this potentially fatal disease. They made reports of nine cases of diphtheria which occurred within a period of six months. Out of the nine reported cases, eight had never been immunized against diphtheria, reflecting the extremely low immunization rates in northern Nigeria, where coverage is as low as 6%. This lack of vaccination contributed significantly to the outbreak's severity, as all cases developed severe complications and eight resulted in death. The absence of diphtheria antitoxin (DAT) further exacerbated the situation, highlighting the need for quick access to and administration of DAT for effective treatment. The study emphasized the necessity of improving immunization rates, addressing misconceptions and logistical challenges related to vaccine delivery, and establishing regional stockpiles of DAT to prevent and manage future outbreaks effectively.

Truelove et al., (2020) carried out a study on Diphtheria outbreaks which underscored the importance of vaccination, which is highly effective at preventing symptomatic disease (>87% with 3 doses) but does not prevent infection. Also, asymptomatic individuals can still transmit the disease, albeit at a lower rate (24%) than symptomatic cases, making diphtheria less transmissible but challenging to control through vaccination alone. Effective outbreak response requires combining vaccination with interventions such as rapid antibiotic treatment, contact tracing, and isolation. Full vaccination coverage alone interrupts transmission in only 27% of outbreaks, improving to 70% with prompt antibiotic treatment. The Rohingya outbreak highlighted the need for high vaccination coverage and integrated response strategies. Mass antibiotic administration, particularly with azithromycin, coupled with vaccination, proves effective in controlling diphtheria and reducing mortality, emphasizing the need for comprehensive immunization and outbreak preparedness efforts. The World Health Organization (WHO) recommends at least 90% coverage for the three-dose diphtheria vaccination (DPT) scheme, but Peru achieved only 72.4% between 2010 and 2019. As a result, Gonzales et al., (2022) carried out a study to identify key factors influencing DPT completion which included maternal age, language, education, and employment. They found out that older mothers (18-49 years) were more likely to complete the vaccination scheme compared to the youngest group (15-17 years). Spanish and Quechua-speaking mothers had higher completion rates, possibly due to better communication with healthcare providers. Surprisingly, rural mothers showed higher completion rates, likely due to recent government efforts to improve rural healthcare access. Higher maternal education levels were associated with lower completion rates, possibly due to vaccine hesitancy. Single mothers had higher completion rates than married or cohabiting women, reflecting increased economic independence and focus on child health. Employment also positively influenced completion rates by providing financial stability to cover additional healthcare costs. Access to healthcare facilities was crucial; mothers who knew the location of the nearest health center, had geographic access, and available transportation were more likely to complete the vaccination scheme. They highlighted the need for targeted strategies to improve vaccination rates in Peru, addressing economic, geographic, and sociodemographic barriers.

II. MODEL FORMULATION

Here we propose a mathematical model for diphtheria transmission to show the dynamics of disease, vaccination and immunity in a population over time, considering age-specific parameters and vaccine efficacy. In this model, the total population size N_i of age group i is divided into five compartments namely: $S_i(t)$ which is the number of susceptible individuals of age group i at time t**,** $E_i(t)$ **which is** the number of exposed individuals of age group i at time t**,** $I_i(t)$ which is the number of infectious individuals of age group i at time t**,** $V_i(t)$ **which is** the number of vaccinated individuals of age group i at time t **and** $R_i(t)$ **which is** the number of recovered individuals of age group i at time t**. Some a**ssumptions made in the model includes:

1. Age Structure: The model can be extended to include age-specific parameters such as susceptibility, transmission rate, and mortality rate to account for variations in these parameters across different age groups.
2. Heterogeneity: The model can be modified to include heterogeneity in susceptibility, transmission rate, and recovery rate, which can help to capture variations in these parameters within the population.
3. Vaccine Efficacy: The model can be adjusted to account for variations in vaccine efficacy.

The schematic diagram illustrating the interactions between the various compartments is shown in the figure below. This model serves as a valuable tool for studying diphtheria transmission dynamics and evaluating the effectiveness of vaccination strategies, considering age-specific factors.

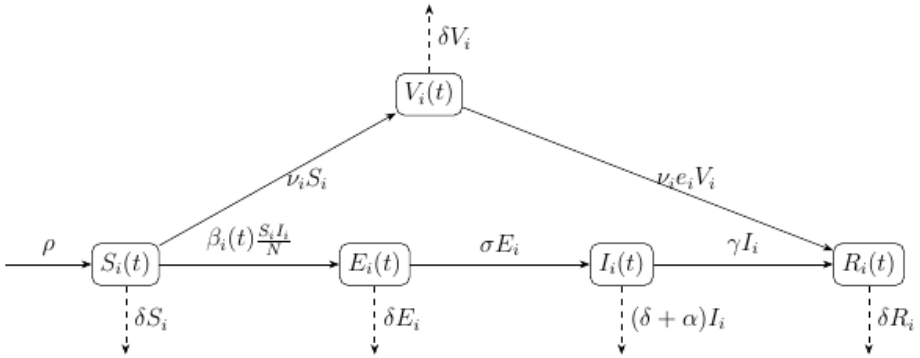

Figure 1: Schematic Diagram

The dynamics of each compartment are described by a set of differential equations which are given below

$$\frac{dS_i}{dt} = \rho - \frac{\beta_i(t)S_iI_i}{N_i} - \upsilon_i S_i - \delta S_i \quad (1)$$

$$\frac{dE_i}{dt} = \frac{\beta_i(t)S_iI_i}{N_i} - \sigma E_i - \delta E_i \quad (2)$$

$$\frac{dI_i}{dt} = \sigma E_i - \gamma I_i - (\delta+\alpha)I_i \quad (3)$$

$$\frac{dV_i}{dt} = \upsilon_i S_i - \upsilon_i e_i V_i - \delta V_i \quad (4)$$

$$\frac{dR_i}{dt} = \gamma I_i + \upsilon_i e_i V_i - \delta R_i \quad (5)$$

with $S_i(0) = 9000, E_i(0) = 100, I_i(0) = 50, V_i(0) = 800, R_i(0) = 50$
The total population is given by $N_i(t) = S_i(t) + E_i(t) + I_i(t) + V_i(t) + R_i(t)$ at any time t.

Table 1. Biological description of model parameters

Parameters	Biological significance	Values
ρ	birth rate	0.2
β_i	is the age-specific transmission rate influenced by seasonality and other factors	0.2
δ	natural mortality rate	0.001
α	disease induced death rate	0.008
γ	recovery rate	0.1
σ	rate of progression from exposed to infectious	0.09
e_i	age-specific vaccine efficacy rate	0.8
υ_i	age-specific vaccination coverage rate	0.05

III. MODEL ANALYSIS

This section presents a comprehensive analysis of the proposed mathematical model for diphtheria transmission, focusing on the impact of age structure and vaccine efficacy. We determine the Disease-Free Equilibrium (DFE) and Endemic Equilibrium (EE) to understand the conditions under which the disease can be eliminated or maintained. The local stability of these equilibrium points will be examined by analyzing the Jacobian matrix at the DFE and EE. This analysis helps us understand system behavior near these points and identify conditions for stability. Additionally, we calculate the basic reproduction number (R_0), which indicates the average number of secondary infections produced by a single infectious individual in a completely susceptible population. $(R_0 < 1)$ suggests the disease will die out, while $(R_0 > 1)$ indicates potential for spread. Sensitivity analysis of various parameters, such as transmission rate, vaccination rate, and natural death rate, is conducted to assess the effectiveness of public health interventions. This analysis aims to enhance understanding of diphtheria transmission dynamics and the factors influencing disease control and prevention.

3.1. Equilibrium Points

Identifying equilibrium points is vital for shaping effective control strategies and evaluating the potential for disease eradication. The two primary equilibrium points of interest are the disease-free equilibrium (DFE) and the disease-endemic equilibrium (DEE). Studying these points provides insights into the dynamics of

disease transmission, aiding researchers and public health professionals in making informed decisions about disease management and control.

3.1.1. The Disease-Free Equilibrium Point (DFEP)

The disease-free equilibrium represents a state in the population where there are no infections. This equilibrium denotes a stable condition with no new infections, providing a theoretical benchmark for evaluating and designing disease control strategies. Its analysis is crucial to help comprehend the circumstances essential to stop disease spread. For diphtheria, the effects of age structure, vaccine efficacy, and environmental factors are crucial considerations. When there are no infected individuals ($I_i = 0$), the system of equations (1) – (5) can be analyzed as follows:

$$S_i = \frac{\rho}{\delta + \upsilon_i}, E_i = I_i = 0, V_i = \frac{\rho\upsilon_i}{\delta\upsilon_i e_i + {\upsilon_i}^2 e_i + \delta^2 + \delta\upsilon_i}, R_i = \frac{\rho{\upsilon_i}^2 e_i}{(\delta\upsilon_i e_i + {\upsilon_i}^2 e_i + \delta^2 + \delta\upsilon_i)\delta}$$

Therefore, the disease-free equilibrium is given by,

$$(S_i, E_i, I_i, V_i, R_i) = \left(\frac{\rho}{\delta + \upsilon_i}, 0, 0, \frac{\rho\upsilon_i}{\delta\upsilon_i e_i + {\upsilon_i}^2 e_i + \delta^2 + \delta\upsilon_i}, \frac{\rho{\upsilon_i}^2 e_i}{(\delta\upsilon_i e_i + {\upsilon_i}^2 e_i + \delta^2 + \delta\upsilon_i)\delta}\right)$$

3.1.2. The Endemic Equilibrium (EE)

The Endemic Equilibrium (EE) is a state where the disease persists at a stable level within the population over time, meaning it remains present at a controllable level. At this point, the rates of new infections and recoveries are balanced, maintaining a relatively constant number of infected individuals without the disease completely disappearing. Solving the entire system of equations (1) – (5), we obtain the EE as follows:

$$S_{i_e} = \frac{N_i(\delta + \sigma)(\alpha + \delta + \gamma)}{\sigma\beta_i}$$

$$E_{i_e} = \frac{-(\delta + \upsilon_i)(\delta + \sigma)(\alpha + \delta + \gamma)N_i + \rho\sigma\beta_i}{\sigma(\delta + \sigma)\beta_i}$$

$$I_{i_e} = \frac{-(\delta + \upsilon_i)(\delta + \sigma)(\alpha + \delta + \gamma)N_i + \rho\sigma\beta_i}{\beta_i(\delta + \sigma)(\alpha + \delta + \gamma)}$$

$$V_{i_e} = \frac{\upsilon_i N_i(\delta + \sigma)(\alpha + \delta + \gamma)}{\sigma\beta_i(\upsilon_i e_i + \delta)}$$

$$R_{i_e} = \frac{((\delta + \sigma)\left((\upsilon_i{}^2 e_i - \gamma\sigma)\delta^2 + \left((\upsilon_i e_i - \gamma(e_i + 1)\right)\sigma + \upsilon_i e_i(\alpha + \gamma)\right)\upsilon_i\delta + \alpha\sigma\upsilon_i{}^2 e_i)}{(\alpha + \delta + \gamma)N_i + \rho\sigma^2\beta_i(\upsilon_i e_i + \delta)}{(\delta(\alpha + \delta + \gamma)(-(\delta + \upsilon_i)(\delta + \sigma)(\alpha + \delta + \gamma)N_i + \rho\sigma\beta_i)(\upsilon_i e_i + \delta))} E_{i_e}$$

3.2 Stability Analysis

In this section, we investigate the local stability of the Disease-Free Equilibrium (DFE) in our diphtheria transmission model. Stability analysis helps determine the conditions under which the disease will either die out or potentially spread within the population. By computing and evaluating the Jacobian matrix at the DFE, we can assess the behavior of the system near this equilibrium point and identify the critical factors that influence the stability of the disease-free state.

$$J|_{(S_i, E_i, I_i, V_i, R_i)} = \begin{bmatrix} -\delta - \upsilon_i & 0 & -\frac{\beta_i\rho}{(\delta + \upsilon_i)N_i} & \varsigma & 0 \\ 0 & -\sigma - \delta & \frac{\beta_i\rho}{(\delta + \upsilon_i)N_i} & 0 & 0 \\ 0 & \sigma & -\gamma - \delta - \alpha & 0 & 0 \\ \upsilon_i & 0 & 0 & -\upsilon_i e_i - \delta & 0 \\ 0 & 0 & \gamma & \upsilon_i e_i & -\delta \end{bmatrix}$$

The eigenvalues gotten are

$$\lambda = \begin{pmatrix} -\delta, -\delta - \upsilon_i, -\upsilon_i e_i - \delta, \\ \pm\sqrt{4}\sqrt{\left(\frac{1}{4}(\alpha + \gamma - \sigma)^2(\delta + \upsilon_i)N_i + \sigma\beta_i\rho\right)N_i(\delta + \upsilon_i)} + \\ \frac{1}{2}\frac{(-2\delta^2 + (-\alpha - \gamma - \sigma - 2\upsilon_i)\delta + (-\alpha - \gamma - \sigma)\upsilon_i)N_i}{(\delta + \upsilon_i)N_i} \end{pmatrix}$$

Since all eigenvalues have negative real parts, this shows that the disease-free equilibrium is stable.

3.3. The Basic Reproduction Number

In this section, we derive the basic reproduction number (R_0), a key epidemiological metric that indicates the average number of secondary infections produced by a single infectious individual in an entirely susceptible population. Calculating R_0 is essential for understanding the potential spread of the disease and the threshold conditions necessary for disease control and eradication. We employ the next generation matrix approach to obtain R_0, considering the state variables $X(t) = (E_i, I_i, V_i)$ to accurately represent the transmission dynamics within the population.

$$X^{'}(t) = \mathcal{F}(t) - \mathcal{V}(t)$$

where:

$$\mathcal{F}(t) = \begin{pmatrix} 0 & \frac{\beta_i S_i}{N_i} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \text{and} \quad \mathcal{V}(t) = \begin{pmatrix} -\sigma - \delta & 0 & 0 \\ \sigma & -\gamma - \delta - \alpha & 0 \\ 0 & 0 & -\upsilon_i e_i - \delta \end{pmatrix}$$

Evaluating the derivatives of F and V at the disease-free equilibrium point obtained above, yields $F\mathcal{V}^{-1}$ as seen below:

$$F\mathcal{V}^{-1} = \begin{pmatrix} -\frac{\beta_i S_i \sigma}{N_i(\sigma+\delta)(\gamma+\delta+\alpha)} & -\frac{\beta_i S_i}{N_i(\gamma+\delta+\alpha)} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

By solving the dominant eigenvalue of the next generation matrix $F\mathcal{V}^{-1}$, we get the basic reproduction number to be

$$R_0 = -\frac{\beta_i S_i \sigma}{N_i(\sigma+\delta)(\gamma+\delta+\alpha)}$$

Recall that

$$S_i = \frac{\rho}{\delta + \upsilon_i}$$

Therefore, the basic reproduction number of the given system becomes:

$$R_0 = -\frac{\beta_i \rho \sigma}{(\delta+\upsilon_i)N_i(\sigma+\delta)(\gamma+\delta+\alpha)}$$

3.4. Sensitivity Analysis

In this section, we conduct a sensitivity analysis to assess the impact of model parameters on the basic reproduction number (R_0), following the approach outlined by Chitnis et al. (2008). This analysis helps us understand how variations in parameters such as transmission rates, vaccination coverage, and vaccine efficacy affect the potential for disease spread. By computing sensitivity indices, we gain insights into the relative importance of each parameter in influencing R_0, thereby guiding the prioritization of interventions for effective disease control and prevention strategies. The sensitivity index for a parameter, say ρ is defined as

$$\xi_{\beta}^{R_0} = \frac{\partial R_0}{\partial \rho} \times \frac{\rho}{R_0}$$

Computing the sensitivity indices of each parameter we get the results below:

$$\xi_{\beta_i}^{R_0} = 1 > 0 \qquad \xi_{\rho}^{R_0} = 1 > 0 \qquad \xi_{\sigma}^{R_0} = \frac{\delta}{\sigma + \delta} > 0$$

$$\xi_{\delta}^{R_0} = -\frac{2\delta\left(\frac{3}{2}\delta^2 + (\alpha+\gamma+\sigma+\upsilon_i)\delta + \left(\frac{1}{2}\alpha + \frac{1}{2}\gamma + \frac{1}{2}\sigma\right)\upsilon_i + \frac{1}{2}\sigma(\alpha+\gamma)\right)}{(\delta+\upsilon_i)(\sigma+\delta)(\gamma+\delta+\alpha)} < 0$$

$$\xi_{\upsilon_i}^{R_0} = -\frac{\upsilon_i}{\delta + \upsilon_i} < 0 \qquad \xi_{\gamma}^{R_0} = -\frac{\gamma}{\gamma + \delta + \alpha} < 0 \qquad \xi_{\alpha}^{R_0} = -\frac{\alpha}{\gamma + \delta + \alpha} < 0$$

The sensitivity analysis revealed that the parameters positively influencing the basic reproduction number (R_0) are the birth rate (ρ) into the susceptible class, the age-specific transmission rate influenced by seasonality and other factors (β_i), and the rate of transition from exposed to infectious class (σ). Reducing the number of susceptible individuals, lowering the age-specific transmission rate, and effectively restricting the progression of exposed individuals to the infectious class can significantly decrease R_0, thereby enhancing the stability of the disease-free equilibrium. Conversely, increasing these positively sensitive parameters raises R_0, indicating higher disease endemicity. Parameters that negatively influence R_0include the natural death rate (δ), the age-specific vaccination coverage rate (υ_i), the recovery rate (γ), and the rate of disease-induced death (α). When the values of these negatively sensitive parameters are increased while keeping the other parameters

fixed, R_0decreases, suggesting a reduction in disease endemicity. This implies that enhancing vaccination coverage, improving recovery rates, and increasing the natural death rate can contribute to controlling the spread of diphtheria and reducing its endemicity.

Table 2: Numerical values of sensitivity indices of R_0

Parameter	Sensitivity Index
β_i	1.0000
ρ	1.0000
σ	0.0110
δ	-0.0398
υ_i	-0.9804
γ	-0.9174
α	-0.0734

IV. DISCUSSION OF RESULTS

The computed risk impact score helped in determining the birds risk impact. The birds and habitats were divided into three risk categories: High Risk (affected by 2 or more threats), Intermediary Risk (affected by one type of threat), and Low Risk (birds under pressure).

The age-specific vaccination coverage rate (υ_i) as shown in Figures 2, 3 and 4 plays a crucial role in shaping the dynamics of the susceptible, vaccinated, and recovered classes within a population. This parameter influences how effectively vaccination programs can reduce disease spread and provide long-term immunity in different age groups. The age-specific vaccination coverage rate (υ_i) directly decreases the number of susceptible individuals in the population. As υ_i increases, a higher proportion of the susceptible individuals (S_i) in the age group i receive vaccinations, transitioning them into the vaccinated class (V_i). This reduces the pool of individuals who are vulnerable to infection. Consequently, higher υ_i leads to a more significant reduction in the susceptible population, thereby lowering the overall risk of disease transmission within that age group. This effect is particularly vital in preventing outbreaks and achieving herd immunity, especially in highly susceptible age cohorts. An increase in the vaccination coverage rate (υ_i) directly boosts the number of individuals in the vaccinated class (V_i). As more susceptibles are vaccinated, the vaccinated class grows, reflecting the effectiveness of the vaccination program in the targeted age group. This transition is crucial because it not only reduces the immediate susceptibility of individuals but also, depending on the vaccine's efficacy (e_i), can provide long-term immunity. The vaccinated class represents a buffer against disease spread, as vaccinated individuals are less likely to become infected and, if the vaccine is effective, less likely to transmit the disease. The size and growth of the vaccinated class are therefore key indicators of the success of vaccination efforts. The interaction between vaccination and the recovered class (R_i) is somewhat indirect but still significant. As $\upsilon_i$increases and more individuals are vaccinated, the incidence of new infections decreases. This reduction in new infections subsequently decreases the number of individuals who would move from the infectious class (I_i) to the recovered class (R_i) through natural infection and recovery. However, if the vaccine provides immunity equivalent to natural infection, vaccinated individuals (represented by $\upsilon_i e_i V_i$) can transition into the recovered class. Thus, a high vaccination rate (υ_i) can lead to an increase in the recovered class without individuals having to suffer through the disease, effectively simulating a population-wide recovery scenario.

The age-specific vaccine efficacy rate (e_i) as seen in Figure 5 and 6 is a crucial parameter that determines how effectively a vaccine can prevent disease in vaccinated individuals within different age groups. This rate has significant implications for both the vaccinated and recovered classes. The vaccine efficacy rate (e_i) directly affects the quality of immunity provided to individuals in the vaccinated class (V_i). When $e_i$is high, it means that a large proportion of vaccinated individuals gain effective protection against the disease. In this scenario, the vaccinated class remains stable or grows as fewer vaccinated individuals succumb to the infection. High efficacy ensures that vaccinated individuals do not transition back to being susceptible or become part of the infected class, thereby maintaining or increasing the vaccinated population's overall health status. Conversely, if $e_i$is low, the vaccine is less effective in providing immunity, and a larger proportion of vaccinated individuals may still be susceptible to the disease. This situation can lead to breakthrough infections where vaccinated individuals contract the disease, reducing the vaccinated class's overall effectiveness in preventing disease spread. Thus, the higher the vaccine efficacy, the stronger the protective barrier created by the vaccinated class against the spread of the infection. The vaccine efficacy rate (e_i) also influences the dynamics of the recovered class (R_i), albeit indirectly. When $e_i$is high, effectively vaccinated individuals are less likely to contract the disease, reducing the number of individuals who would need to recover from an actual infection. In contrast, with low e_i, more vaccinated individuals may still get infected, increasing the flow of individuals from the vaccinated class to the infected class, and eventually to the recovered class through natural

infection and recovery. This scenario implies that the recovered class could grow not just from natural recovery but also due to the failure of the vaccine to provide adequate protection.

The age-specific transmission rate (β_i) seen in Figures 7 and 8 is a critical parameter that influences how rapidly a disease spreads within different age groups. This rate determines the likelihood of disease transmission from infectious individuals to susceptible ones, impacting both the susceptible and exposed classes. The age-specific transmission rate (β_i) directly affects the rate at which susceptible individuals (S_i) become exposed (E_i). A higher β_i indicates a greater probability that susceptible individuals will come into contact with and contract the disease from infectious individuals. Consequently, an increase in β_i leads to a more rapid decline in the susceptible class as more individuals transition from being susceptible to exposed. This rapid decrease in the number of susceptibles can significantly influence the overall dynamics of disease spread, leading to faster and potentially larger outbreaks within the age group. Conversely, a lower β_i reduces the likelihood of transmission, allowing the susceptible population to remain larger for a longer period. This slower transition helps to control the spread of the disease, providing more time for intervention measures such as vaccination or quarantine to be implemented effectively. Thus, the transmission rate β_i is a key factor in determining the vulnerability of the susceptible population to the disease. The exposed class (E_i) consists of individuals who have been infected but are not yet infectious themselves. The age-specific transmission rate (β_i) plays a pivotal role in the inflow of individuals into this class. As β_i increases, more susceptible individuals contract the disease and enter the exposed class. This leads to a rapid increase in the number of exposed individuals, potentially overwhelming the healthcare system if the exposed individuals quickly progress to become infectious. An elevated β_i can result in a substantial rise in the exposed class, which subsequently fuels the infectious class (I_i) as exposed individuals progress to the next stage of the disease. This escalation can drive the epidemic forward, making it more challenging to control. On the other hand, a lower β_i slows the rate at which susceptibles become exposed, thereby stabilizing the growth of the exposed class. This slower progression allows for more manageable disease dynamics and provides a buffer for public health responses.

The recovery rate (γ) shown in Figure 9 directly reduces the number of individuals in the infected class by accelerating their transition to recovery, leading to a lower prevalence of active cases. A higher γ shortens the infectious period, flattening the epidemic curve and easing the burden on healthcare systems. This also reduces transmission potential, indirectly lowering the basic reproduction number (R_0). Enhancing recovery rates through medical interventions can improve disease control and positively impact public health strategies. Also, the natural mortality rate (δ) as seen in Figure 10 and 11 influences both the susceptible and infected classes by reducing their respective populations over time. The natural mortality rate (δ) decreases the number of susceptible individuals (S_i) at a constant rate. This is represented by the term $-\delta S_i$ in the differential equation for the susceptible class. As δ increases, the susceptible population declines more rapidly due to natural causes, which can reduce the pool of individuals at risk of becoming infected. However, it does not directly impact the transmission dynamics of the disease, but it does lower the overall number of susceptibles available to contract the disease. In the infected class (I_i), the natural mortality rate (δ) contributes to the overall outflow of individuals, represented by the term $(\delta + \alpha)I_i$in the differential equation, where α is the disease-induced death rate. As δ increases, more infected individuals die from natural causes, leading to a faster decline in the infected population. This reduces the number of individuals who can potentially spread the disease, thereby indirectly affecting the transmission dynamics and possibly lowering the basic reproduction number (R_0).

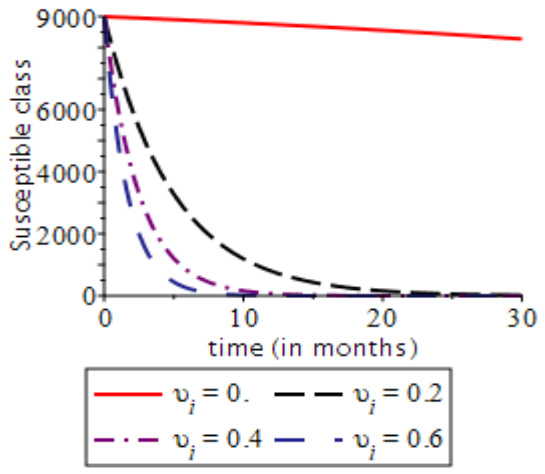

Figure 2: Effect of age-specific vaccination coverage on the susceptible class

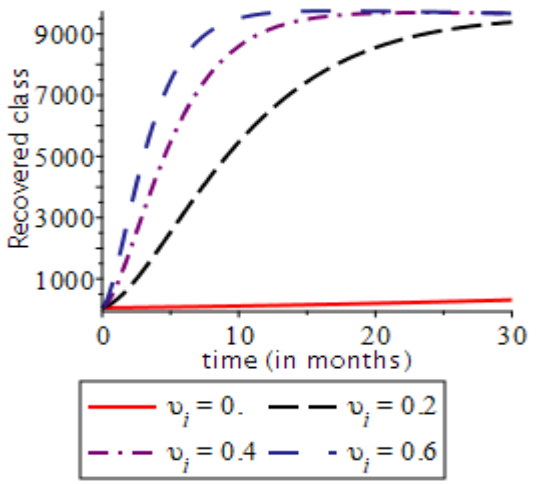

Figure 3: Effect of age-specific vaccination coverage on the recovered class

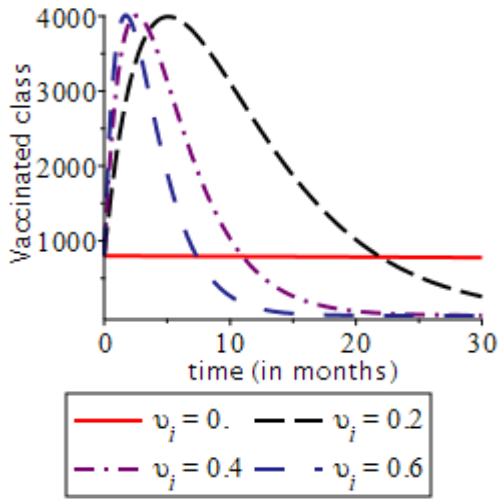

Figure 4: Effect of age-specific vaccination coverage on the vaccinated class

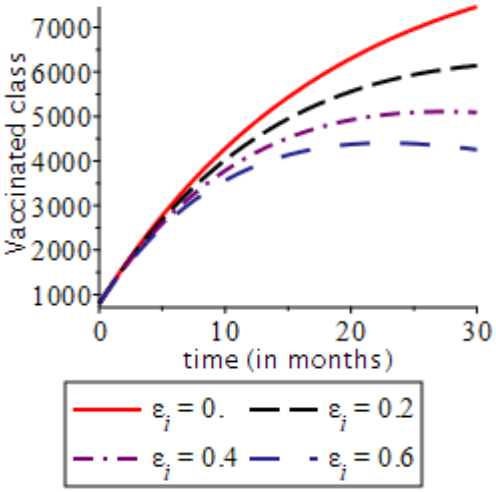

Figure 5: Effect of age-specific vaccine efficacy on the vaccinated class.

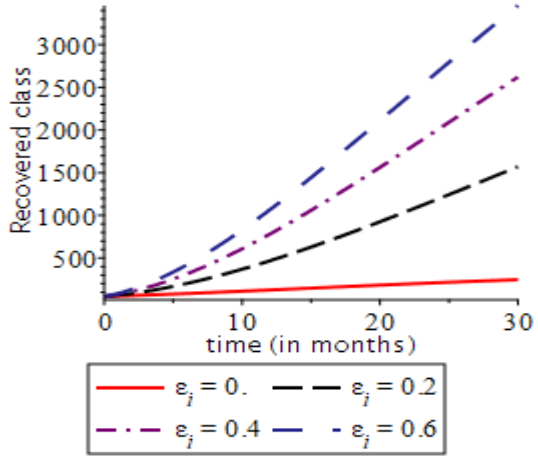

Figure 6: Effect of age-specific vaccine efficacy on recovered class.

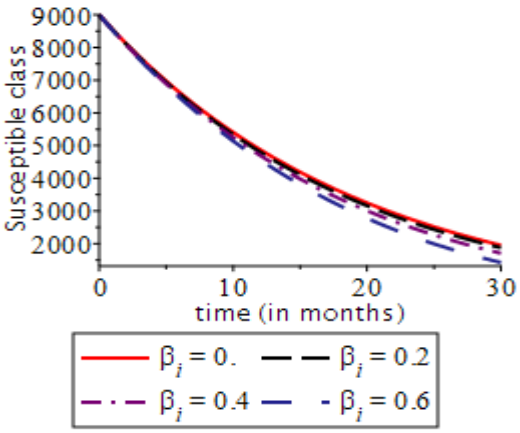

Figure 7: Effect of age-specific transition on the susceptible class.

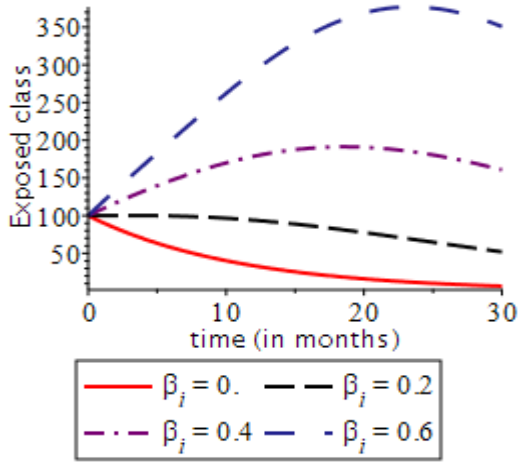

Figure 8: Effect of age-specific transition on the exposed class.

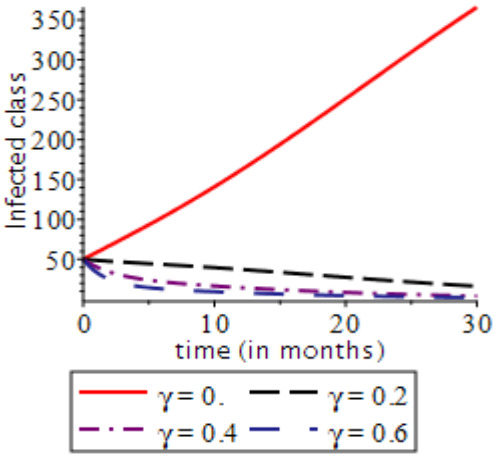

Figure 9: Effect of the recovery rate on the infected class.

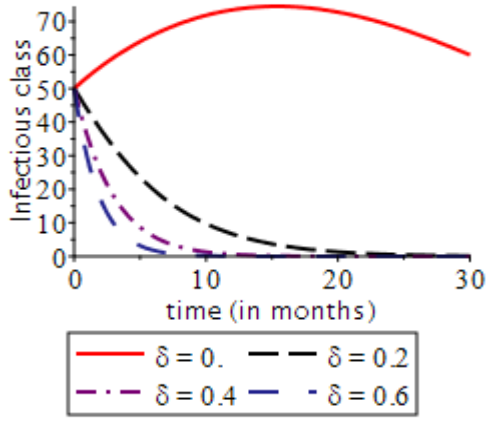

Figure 10: Effect of the natural mortality rate on the infected class.

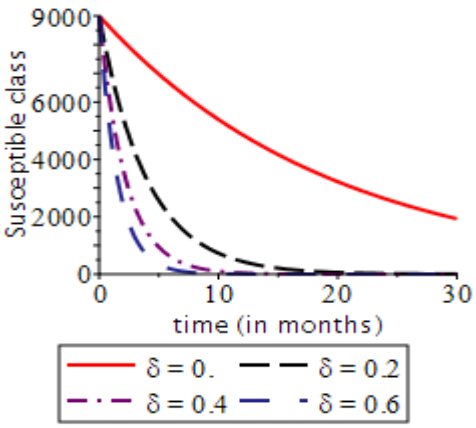

Figure 11: Effect of the natural mortality rate on the susceptible class.

V. DISCUSSION AND CONCLUSION

The various parameters in infectious disease modeling, such as age-specific vaccination coverage rate (υ_i), vaccine efficacy rate (e_i), transmission rate (β_i), recovery rate (γ), and natural mortality rate (δ), play critical roles in shaping the dynamics of disease spread and control within a population. Higher vaccination coverage and efficacy rates significantly reduce the susceptible and infected populations by providing effective immunity and minimizing breakthrough infections. The transmission rate directly impacts the transition of susceptible individuals to the exposed class, influencing the overall spread of the disease. An increased recovery rate leads to a quicker reduction in the infected class, easing healthcare burdens and lowering transmission potential. Lastly, the natural mortality rate reduces the number of susceptible and infected individuals, indirectly affecting disease dynamics by decreasing the overall population at risk. Together, these parameters highlight the importance of targeted public health interventions, effective vaccination programs, and medical treatments in managing and controlling infectious diseases.

As implementing effective strategies to control infectious diseases requires a multifaceted approach, priority should be given to vaccination campaigns across all age groups to achieve herd immunity and prevent outbreaks. Investment in research and development is essential to develop vaccines with high efficacy rates and improve formulations continually as well as public education campaigns to help increase vaccine uptake and combat misinformation. Also, targeted interventions based on age-specific transmission rates can effectively mitigate disease spread, while strengthening healthcare infrastructure is necessary to manage surges in cases and improve treatment outcomes.

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CONFLICT OF INTERESTS

The authors declare that there are no conflicts of interest regarding the publication of this article.

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