



Research Article

Equilibrium and Stability Analysis of a Deterministic Diphtheria Transmission Model

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Abstract

This study presents a deterministic mathematical model for analysing the transmission dynamics and control of diphtheria using a Susceptible, Vaccinated, Latent, Infectious, and Removed (SVLIR) framework. The study was motivated by the persistent occurrence of diphtheria outbreaks in regions with inadequate vaccination coverage, weak healthcare systems, and poor public health awareness despite the availability of effective vaccines. The main aim of the study is to investigate the transmission dynamics of diphtheria and evaluate the effectiveness of vaccination and other control measures in achieving disease eradication. Specifically, the objectives were to formulate an SVLIR deterministic model, analyse the qualitative properties of the model, determine the disease-free and endemic equilibrium states, derive the basic reproduction number (R_0), investigate the local and global stability of the disease-free equilibrium, and perform numerical simulations to examine the effects of epidemiological parameters on disease transmission. The total population was divided into five epidemiological compartments namely Susceptible, Vaccinated, Latent, Infectious, and Removed individuals. The model incorporated recruitment, vaccination, disease transmission, progression from latent to infectious stage, recovery, waning immunity, natural death, and disease-induced mortality. The positivity and boundedness of the model were established to ensure epidemiological validity. The basic reproduction number was derived using the next-generation matrix method, while the local and global stability analyses were established using the Jacobian matrix, Routh–Hurwitz criterion, Lyapunov function, and LaSalle’s invariance principle. Numerical simulations using the fourth-order Runge–Kutta method revealed that increased vaccination and recovery rates significantly reduce infection prevalence, whereas high transmission and progression rates intensify disease spread. The study further showed that routine vaccination remains the most effective long-term strategy for diphtheria eradication. The findings established that when ($R_0 < 1$), the disease-free equilibrium is both locally and globally

asymptotically stable, implying that diphtheria will eventually die out from the population. The study therefore highlights the importance of sustained immunization programs, early treatment, and public health awareness in controlling and preventing diphtheria outbreaks.

Keywords: Diphtheria Transmission; Disease-Free Equilibrium; Endemic Equilibrium; Basic Reproduction Number; Stability Analysis; Local Stability and Global Stability.

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Introduction

Diphtheria is a severe infectious disease that can be prevented through vaccination and is caused by the toxin-producing bacterium *Corynebacterium diphtheriae*. It mainly targets the upper respiratory tract, where it produces a thick grey membrane that may block the airway and result in breathing difficulties. In more serious cases, the toxin can spread into the bloodstream, leading to complications such as myocarditis, nerve damage, kidney failure, and even death. The disease is primarily transmitted through respiratory droplets, close personal contact, or contact with contaminated surfaces. Although the widespread use of the diphtheria toxoid vaccine has significantly reduced its global prevalence, outbreaks still occur in areas with low immunization coverage, inadequate disease monitoring, population displacement, and a fragile healthcare system (Johnson et al., 2024)

Despite worldwide vaccination efforts, diphtheria continues to persist in many developing countries, with occasional outbreaks occurring in highly populated or conflict-affected areas. Contributing factors to its resurgence include reluctance to vaccinate, declining immunity over time, population movement, and insufficient access to booster vaccinations. Mathematical studies have further demonstrated that migration can reintroduce the infection into previously disease-free communities, thereby influencing the long-term pattern of disease transmission (Soleh et al., 2025). Furthermore, public health education efforts, enhanced disease surveillance, and the prompt use of diphtheria antitoxin have been shown to greatly lower the prevalence of infection. These results highlight the need to incorporate behavioural factors and intervention strategies into epidemiological models (Andrawusa et al., 2025).

Infectious diseases often have devastating consequences, especially for children's survival in developing countries. Addressing this challenge requires a comprehensive global perspective that accounts for biocomplexity-the many interconnected factors

influencing the emergence and persistence of infectious agents. Achieving this understanding calls for interdisciplinary collaboration among specialists such as biologists, ecologists, chemists, epidemiologists, mathematicians, statisticians, and atmospheric scientists to better inform strategies for controlling or minimizing the impact of these diseases (Katz and Fischer, 2021).

A deterministic diphtheria transmission model that includes public awareness as a key control factor is analysed through equilibrium and stability methods, illustrating how behaviour changes driven by awareness can markedly reduce disease spread by lowering the basic reproduction number (R_0). By applying Jacobian linearization and stability conditions, the study indicates that when ($R_0 < 1$), the disease-free equilibrium is stable, and diphtheria can be eliminated, emphasizing the need to combine vaccination efforts with strong public health education strategies (Abubakar et al., 2025).

Considering the findings of (Fauzi et al., 2024 and Kamadjeu et al., 2025), booster vaccination plays a critical role in reducing the basic reproduction number (R_0) and maintaining the stability of the disease-free equilibrium, while R_0 serves as a key threshold for determining whether diphtheria persists or dies out. These studies demonstrate that achieving high vaccination coverage and keeping ($R_0 < 1$) are essential for effective disease control. Building on these findings, the present study focuses on disease-free equilibrium and stability analysis to provide deeper mathematical insights into the effects of vaccination, migration, and treatment on diphtheria transmission, thereby supporting evidence-based strategies for disease elimination and long-term epidemiological stability.

Model Formulation

To analyse the transmission dynamics of diphtheria within a population, a deterministic model was developed. The total population, denoted by $N(t)$, was partitioned into five distinct epidemiological compartments based on disease status at time (t). These include the susceptible $S(t)$, comprising individuals who are uninfected but at risk; the vaccinated group $V(t)$, made up of individual who have received immunization; the latent group $L(t)$, made up of individuals who are infected but not yet infectious; the infectious group $I(t)$, which includes those capable of transmitting the disease; and the removed group $R(t)$, representing individuals who have acquired immunity following infection. Thus, the total population size is given by:

$$N(t) = S(t) + V(t) + L(t) + I(t) + R(t)$$

The model assumes that individuals enter the susceptible class through recruitment (birth or migration) at a constant rate Λ . Susceptible individuals can become infected after effective contact with infectious individuals at a transmission rate β , or they may be vaccinated at a rate ρ , moving into the vaccinated class. All individuals experience natural death at rate μ . Vaccinated individuals transition to the removed class at rate ψ , while also being subjected to natural death. Upon exposure to infectious individuals, susceptible individuals may contract the infection: a fraction τ of newly infected individuals enters the latent class, representing an incubation stage during which they are infected but not yet infectious, while the remaining fraction $(1-\tau)$ moves directly into the infectious class. Individuals in the latent class may progress to the infectious stage at rate γ , recover without becoming infectious at rate σ , or die naturally at rate μ . Infectious individuals recover at rate ε , die naturally at rate μ , or die due to diphtheria at rate α . Recovered individuals accumulate in the removed class and eventually exit the population through natural mortality. Based on the assumptions, the transmission dynamics of diphtheria are governed by the following system of nonlinear ordinary differential equations below:

Equations of the Model

$$\frac{dS}{dt} = \Lambda - \beta SI - \rho S - \mu S \quad (1)$$

$$\frac{dV}{dt} = \rho S - \psi V - \mu V \quad (2)$$

$$\frac{dL}{dt} = \tau\beta SI - \gamma L - \sigma L - \mu L \quad (3)$$

$$\frac{dI}{dt} = (1 - \tau)\beta SI + \gamma L - \varepsilon I - (\mu + \alpha)I \quad (4)$$

$$\frac{dR}{dt} = \varepsilon I - \sigma L - \psi V - \mu R \quad (5)$$

Characteristics of the Model

Positively Invariant and Boundedness

Let

$$\Omega = \left[(S, V, L, I, R) \in \mathbb{R}^5 : (S \geq 0, V \geq 0, L \geq 0, I \geq 0, R \geq 0), 0 < N \leq \Lambda / \mu \right]$$

It can be shown that the set Ω is positively-invariant and attracts all positive solutions of the system (1)-(5)

Theorem 1. The region Ω is positively invariant for the system (1)-(5)

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Proof. The rate of change of the total population is given as:

$$\frac{dN(t)}{dt} = \frac{dS}{dt} + \frac{dV}{dt} + \frac{dL}{dt} + \frac{dI}{dt} + \frac{dR}{dt} \quad (6)$$

substituting (1) into (2), and simplifying it gives

$$\frac{dN(t)}{dt} = \Lambda - \mu S - \mu V - \mu L - (\mu + \alpha)I - \mu R \quad (7)$$

When there is no diphtheria disease in the population, $I = 0$ and $L = 0$, then (7) becomes

$$\frac{dN(t)}{dt} = \Lambda - \mu N \quad (8)$$

so, we have

$$\frac{dN(t)}{dt} + \mu N \leq \Lambda \quad (9)$$

using the integrating factor method, we have

$$\frac{dN(t)}{dt} e^{\mu t} + \mu N e^{\mu t} \leq \Lambda e^{\mu t} \quad (10)$$

Simplifying,

$$\frac{d(Ne^{\mu t})}{dt} \leq \Lambda e^{\mu t} \quad (11)$$

taking the integral,

$$\int \frac{d(Ne^{\mu t})}{dt} \leq \int \Lambda e^{\mu t} \quad (12)$$

Simplifying,

$$N e^{\mu t} \leq \frac{\Lambda}{\mu} e^{\mu t} + C \quad (13)$$

When $t = 0$

$$C = N(0) - \frac{\Lambda}{\mu} \quad (14)$$

substituting (14) into (13), and simplify,

$$N \leq N(0)e^{\mu t} + \frac{\Lambda}{\mu}[1 - e^{\mu t}] \quad (15)$$

If $N \leq \frac{\Lambda}{\mu}$ then $N(0) = \frac{\Lambda}{\mu}$ so Ω is positively invariant set under model described in (1) - (5).

Positivity of the Model

Positivity of the model using the Invariant Region method showing that the population remains non-negative and bounded. Consider equations (1) - (5)

Theorem 2. Let the initial data for the model (1)- (5) be $(S, V, L, I, R) \in R^5 : (S > 0, V \geq 0, L \geq 0, I \geq 0, R \geq 0)$,

then the solution $S(t), V(t), L(t), I(t), R(t)$ with positive initial data will remain positive for all time $t \geq 0$

Proof. The $(t \geq 0 : S(0) > 0, V(0) \geq 0, L(0) \geq 0, I(0) \geq 0, R(0) \geq 0)$, therefore, can be connected with simplifying (1)

$$\frac{dS}{dt} = \Lambda - (\beta I + \rho + \mu)S \quad (17)$$

Solving for $S(t)$:

$$\text{Let } ks(t) = (\beta I + \rho + \mu)$$

$$\frac{dS}{dt} + ks(t)S = \Lambda \quad (18)$$

Using an integrating factor,

$$Zs(t) = e^{\int ks(u)du > 0} \text{ then } Zs(t)' = \Lambda Zs(t) \geq 0$$

Implies that, $Zs(t)S(t) \geq Zs(0)S(0)$

Therefore,

$$S(t) = S(0)e^{-(ks)} + \int \Lambda e^{-(ks)} dS > 0 \quad (19)$$

Using the same approach for V, L, I, R, gives

$$V(t) = V(0)e^{-(kv)} + \int \rho S e^{-(kv)} dV > 0 \quad (20)$$

$$L(t) = L(0)e^{-(kl)} + \int \tau \beta S I e^{-(kl)} dL > 0 \quad (21)$$

$$I(t) = I(0)e^{-(ki)} + \int \gamma L e^{-(ki)} dI > 0 \quad (22)$$

$$R(t) = R(0)e^{-(kr)} + \int (\varepsilon I + \sigma L + \psi \lambda) e^{-(kr)} dR > 0 \quad (23)$$

for all $t \geq 0$

Implies that the populations $S(t), V(t), L(t), I(t), R(t)$ remain positive at all times, meaning that the compartments representing the populations are biologically meaningful (non-negative).

Disease-Free Equilibrium (E^*)

At the disease-free equilibrium, there are no infected or latent individuals; that is: all the infected and latent classes are considered to be zero at this state, and the entire human population will consist of the susceptible class, vaccinated class, and removed class (i.e. $I = L = 0$).

Thus, we set

$$\frac{dS}{dt} = 0, \frac{dV}{dt} = 0, \frac{dL}{dt} = 0, \frac{dI}{dt} = 0, \frac{dR}{dt} = 0$$

and solve for the equilibrium values of S^*, V^*, R^*

Theorem 3. The Disease-Free Equilibrium (E^*) of the model (1) - (5) exists

Proof. At the Disease-Free Equilibrium, let

$$(S^*, V^*, L^*, I^*, R^*) = (S, V, L, I, R)$$

Substituting $I = 0, L = 0, S = S^0$

The disease-free equilibrium is calculated to be:

$$E^* = \left(\frac{\Lambda}{(\rho + \mu)}, \frac{\rho \Lambda}{(\rho + \mu)(\psi + \mu)}, 0, 0, \frac{\rho \psi \Lambda}{\mu (\rho + \mu)(\psi + \mu)} \right)$$

This equilibrium exists uniquely and biologically whenever all parameters are positive.

Basic Reproduction Number ($R_0 < 1$)

The basic reproduction number (R_0) is a threshold parameter in disease modelling. It is defined as the expected number of infections caused by an infectious person introduced into a completely susceptible population. R_0 is the threshold condition traditionally applied for successful disease invasion (Nash et al., 2022). We only need to model the latent compartment L and the infected compartment I. Considering the model dynamics using the equations:

$$\frac{dL}{dt} = \tau \beta SI - \gamma L - \sigma L - \mu L \quad (30)$$

$$\frac{dI}{dt} = (1 - \tau) \beta SI + \gamma L - \varepsilon I - (\mu + \alpha) I \quad (31)$$

Decomposing the system at the disease-free equilibrium point into:

F(x): The new infections in each infected compartment

V (x): The transition terms, including removals and movement between compartments.

$$V^{-1} = \begin{pmatrix} \frac{1}{(\gamma + \sigma + \mu)} & 0 \\ \frac{1}{(\gamma + \sigma + \mu)(\varepsilon + \mu + \alpha)} & \frac{1}{(\varepsilon + \mu + \alpha)} \end{pmatrix}$$

$$F = \begin{pmatrix} \frac{(\beta \Lambda \tau)}{(\rho + \mu)} & 0 \\ \frac{(1 - \tau) \beta \Lambda}{(\rho + \mu)} & 0 \end{pmatrix}$$

$$G = F V^{-1}$$

$$G = \begin{pmatrix} \frac{\Lambda \tau \beta}{(\gamma + \sigma + \mu)(\rho + \mu)} & 0 \\ \frac{(1 - \tau) \beta \Lambda}{(\gamma + \sigma + \mu)(\rho + \mu)} & 0 \end{pmatrix}$$

Therefore,

$$R_0 = \frac{\beta \tau \Lambda}{(\gamma + \sigma + \mu)(\rho + \mu)}$$

1. Local Stability of the Disease-Free-Equilibrium

To determine the local stability, the Jacobian matrix of the DFE is analysed and the eigenvalues are shown below:

$$\lambda_1 = -(\rho + \mu)$$

$$\lambda_2 = -(\psi + \mu)$$

$$\lambda_3 = -\mu$$

Which are all negative. Considering the infected subsystem which involves L and I:

The characteristic equation is given as $\lambda^2 + a_1 \lambda + a_2 = 0$

Where: $a_1 = (\gamma + \sigma + \mu) + (\varepsilon + \mu + \alpha) - (1 - \tau) \beta S$

And $a_2 = (\gamma + \sigma + \mu)(\varepsilon + \mu + \alpha) - (1 - \tau) \beta S$

Applying Routh-Hurwitz Stability Condition,

$$a_1 > 0 \text{ and } a_2 > 0$$

since $a_2 > 0$ and $R_0 < 1$

Hence, all eigenvalues have negative real parts.

Stability Condition indicates that since $R_0 < 1$, all the eigenvalues of the Jacobian are negative; hence, the Disease-Free-Equilibrium is locally asymptotically stable.

2. GLOBAL STABILITY OF THE DISEASE-FREE EQUILIBRIUM

The infected compartments are L and I. Therefore, we analyse the subsystem by constructing the Lyapunov Function of L and I, $V(L, I) = L + kI$ where $k > 0$ differentiating

$$\frac{dV}{dt} = L' + kI'$$

Substituting the model equation

$$\frac{dV}{dt} = \tau\beta SI - (\gamma + \sigma + \mu) + k[(1 - \tau)\beta SI + \gamma L - \varepsilon I - (\mu + \alpha)I]$$

Expand

$$\frac{dV}{dt} = \tau\beta SI - (\gamma + \sigma + \mu) + k(1 - \tau)\beta SI + k\gamma L - k\varepsilon I - k(\mu + \alpha)I$$

Select $k = \tau(\varepsilon + \mu + \alpha)\gamma$

This simplifies the expression to give:

$$\frac{dV}{dt} \leq (\varepsilon + \mu + \alpha)(R_0 - 1)I$$

Where:

$$R_0 = \frac{\beta\tau\Lambda}{(\gamma + \sigma + \mu)(\rho + \mu)}$$

determining the sign of the derivative:

If $R_0 < 1$, then $\frac{dV}{dt} \leq 0$ and equality holds only when $I = 0, L = 0$.

Applying LaSalle's Invariance Principle: Since $V(L, I)$ is positive definite, which corresponds to the Disease-Free-Equilibrium.

Therefore, $R_0 < 1$ implies that E^* is globally asymptotically stable.

3. Endemic Equilibrium of the Model ($R_0 > 1$)

The endemic equilibrium represents a steady state where the disease persists in the population, meaning that the infected classes remain positive. Thus, $L > 0, I > 0$.

The endemic equilibrium is denoted as:

$$(R_0 > 1) = S^*, V^*, L^*, I^*, R^*$$

Where:

$$\begin{aligned} S^* &= \frac{\varepsilon + \mu + \alpha}{\beta[(1-\tau) + \frac{\gamma\tau}{\gamma + \sigma + \mu}]} \\ V^* &= \frac{\rho k_2 k_1}{\beta(\psi + \mu)[k_3 + (1-\tau)k_1]} \\ L^* &= \frac{\tau[(k_3 k_4 \Lambda \beta + \Lambda \beta + k_5 \Lambda \beta + \Lambda \beta \mu(1-\tau)) - (k_2 k_1(\rho + \mu))]}{\beta k_1[k_3 + (1-\tau)k_1]} \\ I^* &= \frac{\Lambda \beta[k_3 + k_4 + k_5 + \mu(1-\tau)] - k_2 k_1(\rho + \mu)}{\beta[k_2 k_1]} \\ R^* &= \left[\varepsilon \frac{\Lambda \beta[k_3 + k_4 + k_5 \mu(1-\tau)] - k_2 k_1(\rho + \mu)}{\beta(k_2 k_1)} \right] + \left[\sigma \frac{\tau((k_3 \Lambda \beta + k_4 \Lambda \beta + k_5 \Lambda \beta + \Lambda \beta \mu(1-\tau)) - (k_2 k_1(\rho + \mu)))}{\beta k_1[k_3 + k_1(1-\tau)]} \right] \\ &+ \left[\psi \frac{\rho k_2 k_1}{\beta(\psi + \mu)[k_3 + k_1(1-\tau)]} \right] \end{aligned}$$

4. Numerical Simulations

Model Parameters

In this section, the system of equations (1) - (5) was solved numerically using the Fourth-Order Runge-Kutta method implemented by computational software Maple. The parameter values are given in Table 1 for predicting diphtheria cases. The simulations were executed by setting

$S(0) = 253, V(0) = 111, L(0) = 0, I(0) = 8, R(0) = 3$, as initial values of the state variables. The results obtained were used to interpret the stability state of the disease extinction, the infectious persistence steady state of the disease, and the basic reproduction number numerically. It has been shown that the basic reproduction number $R_0 = 0.8069837$, which implies that $R_0 < 1$. The values of the parameters were shown in

Table 1. Most of the

parameters are from literature, and only two were assumed.

Table 1: Values of Parameters of the Model

Parameters	Description	Values	References
Λ	Recruitment rate	0.003	(Assumed)
μ	Natural death rate	0.000054	(Atkinson et al., 2012)
α	Disease-induced death	0.005	(Atkinson et al., 2012)
ρ	Vaccinated rate	0.02	(Wu et al., 2020)
β	Transmission rate	0.5	(Makeri et al., 2023)
Ψ	Vaccinated individuals removed from the entire population	0.05	(Assumed)
τ	Population entering latent class	0.95	(Wu et al., 2020)
γ	Progression rate from latent to infectious class	0.04	(Makeri et al., 2023)
σ	Recovery rate from latent class	0.048	(Makeri et al., 2023)
ϵ	Recovery rate from infectious class	0.025	(Makeri et al., 2023)

Susceptible Population Dynamics

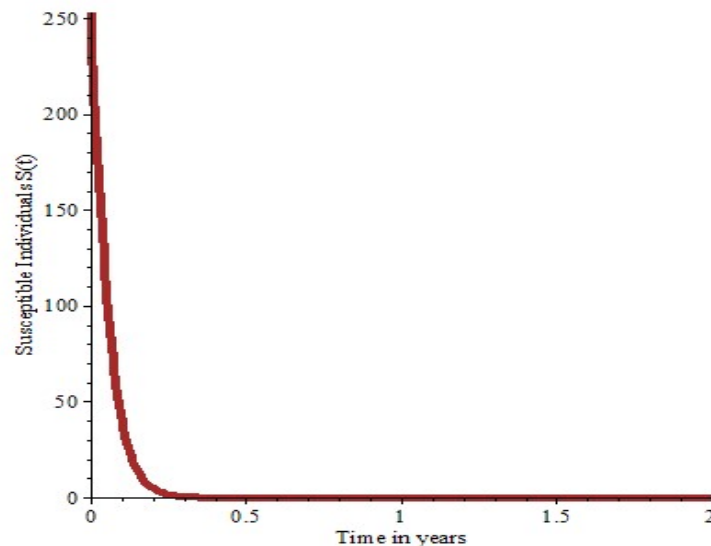


Figure 1: Time evolution of the susceptible population $S(t)$ under the SVLIR diphtheria transmission model when $R_0 = 0.8069837 < 1$.

Figure 1 displays the number of susceptible individuals against time. Initially, the susceptible population decreases due to vaccination and disease transmission. As time progresses, the rate of decline slows down and eventually approaches a stable equilibrium value. The susceptible population represents individuals who are at risk of contracting diphtheria. At the beginning of the simulation, a significant proportion of susceptible individuals leave the compartment through vaccination and infection. Consequently, the susceptible population declines steadily. Since the reproduction number is below unity $R_0 < 1$, the infection is unable to generate sufficient secondary cases to sustain transmission. Therefore, the susceptible population eventually stabilizes instead of continuing to decrease indefinitely. The stabilization indicates that the disease cannot invade the population successfully and that vaccination effectively reduces the pool of individuals available for infection.

Key Finding:

A reduction in susceptible individuals contributes directly to lowering disease transmission and maintaining R_0 below unity.

Vaccinated Population Dynamics

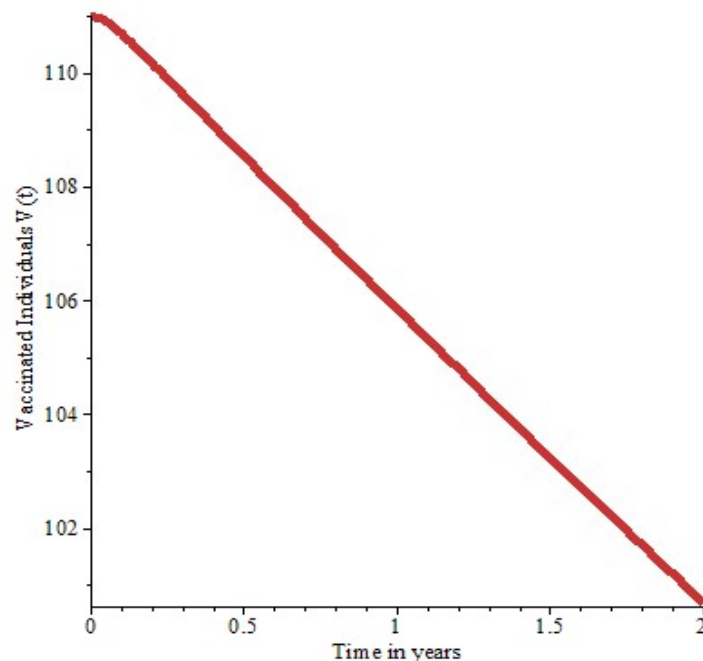


Figure 2: Time evolution of the vaccinated population $V(t)$ showing the effect of vaccination on diphtheria control when $R_0 = 0.8069837 < 1$.

Figure 2 illustrates the increase in vaccinated individuals over time. The curve rises rapidly during the early stages and later approaches a steady-state value. The vaccinated class consists of individuals who have received immunization against diphtheria. The simulation shows a continuous increase in vaccination coverage as susceptible individuals move into the vaccinated compartment. This increase plays a critical role in reducing the number of susceptible individuals available for infection. Because vaccination directly reduces the effective contact between susceptible and infectious individuals, it contributes significantly to lowering the basic reproduction number. The steady rise in vaccinated individuals demonstrates the effectiveness of routine immunization in controlling disease spread. The eventual stabilization of the curve indicates that the system reaches equilibrium where vaccination and natural mortality balance each other.

Key Finding:

Vaccination is the most effective long-term strategy for maintaining R_0 below one and preventing disease persistence.

The Latent Population Dynamics

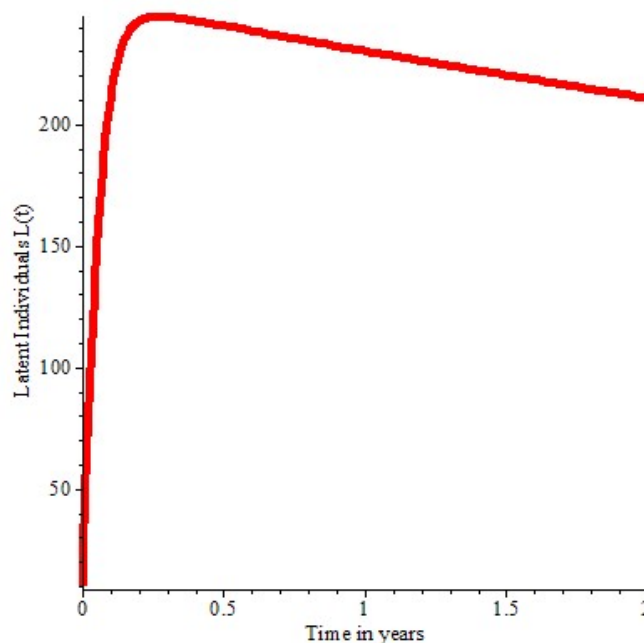


Figure 3: Dynamics of the latent population $L(t)$ under the SVLIR diphtheria model for $R_0 = 0.8069837 < 1$.

Figure 3 initially shows a slight increase in latent individuals followed by a gradual

decline toward zero as time increases. The latent compartment contains infected

individuals who have not yet become infectious. At the onset of the outbreak, exposure to infectious individuals causes the latent population to increase. However, because the reproduction number is less than one, the rate of new infections remains insufficient to sustain the epidemic. As a result, progression to the infectious stage, recovery, and natural mortality gradually reduce the latent population. The eventual convergence of the latent class to zero confirms the theoretical prediction that the disease-free equilibrium is globally asymptotically stable. This behaviour indicates that the disease transmission chain is interrupted before a large epidemic can develop.

Key Finding:

The decline of the latent class toward zero demonstrates that diphtheria transmission cannot be sustained when $R_0 < 1$.

Effect of Transmission Rate (β) on the Population Dynamics

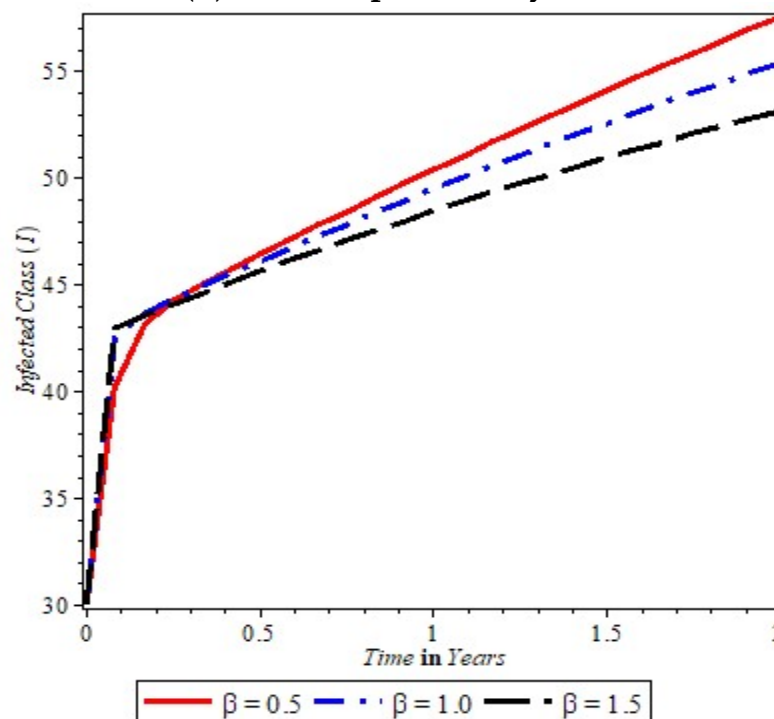


Figure 4: Influence of transmission rate on diphtheria infection dynamics model for $R_0 = 0.8069837 < 1$.

Increasing β causes a substantial increase in the infectious population (Figure 4). Higher transmission rates lead to more effective contacts between susceptible and infectious individuals, resulting in larger outbreaks. Since R_0 is directly proportional to β , increasing

transmission may push the system above the epidemic threshold.

Key Finding

Reducing contact rates through awareness and hygiene measures is essential for maintaining $R_0 < 1$.

Effect of Recovery Rate (ϵ) on The Population Dynamics

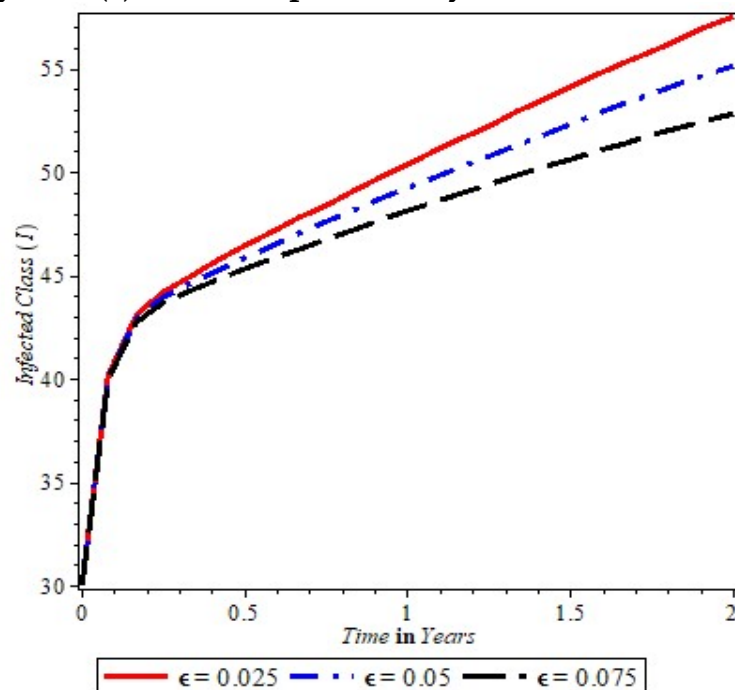


Figure 5: Effect of recovery rate on the infectious population model for $R_0 = 0.8069837 < 1$.

Figure 5 shows that an increase in the recovery rate reduces both the peak and duration of infection. Faster recovery shortens the infectious period, thereby reducing the number of secondary infections generated by infected individuals.

Key Finding

Early diagnosis and treatment contribute significantly to disease control.

Effect of Latent Progression Rate (Γ) on the Population Dynamics

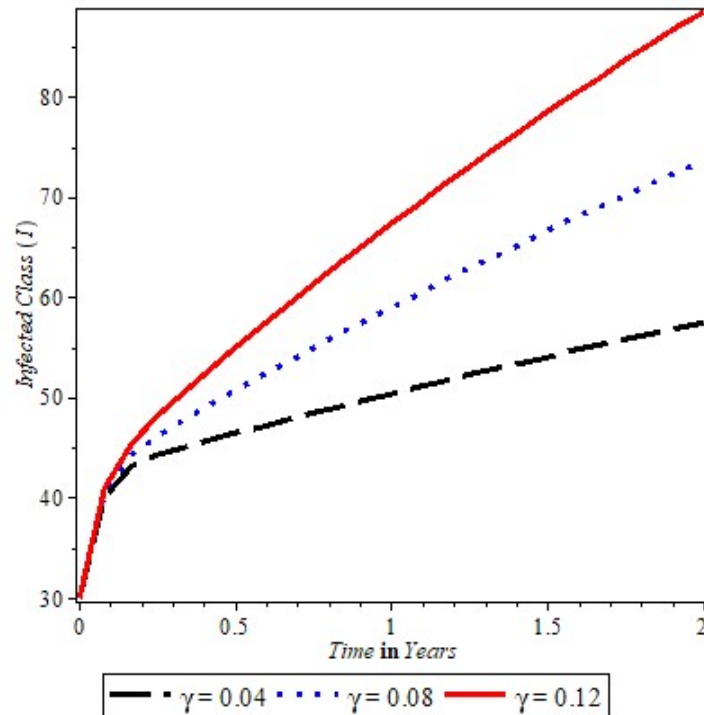


Figure 6: Impact of progression from latent to infectious stage on disease transmission model for $R_0 = 0.8069837 < 1$.

Figure 6 depicts that higher progression rates move individuals more rapidly into the infectious compartment, resulting in steeper epidemic peaks. Although infection peaks occur earlier, the overall epidemic duration becomes shorter.

Key Finding

Rapid progression intensifies outbreaks and increases short-term transmission risk.

Effect of Recruitment Rate and Progression into the Latent Class (Λ & T) on the Population Dynamics

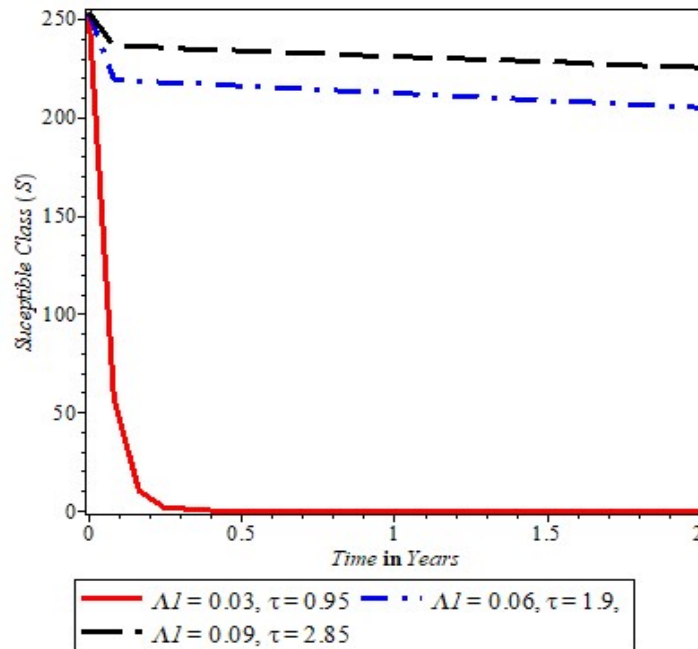


Figure 7: Effect of recruitment rate and progression into the latent class on diphtheria dynamics model for $R_0 = 0.8069837 < 1$.

As recruitment increases, more susceptible individuals enter the population (Figure 7). Without corresponding vaccination efforts, these new susceptible can sustain transmission and increase disease prevalence.

Key Finding

Population growth must be matched by vaccination programs to prevent resurgence.

Discussion and Results

This study developed and analysed a deterministic Susceptible, Vaccinated, Latent, Infectious, Removed (SVLIR) mathematical model to investigate the transmission dynamics and control of diphtheria. The model incorporated major epidemiological processes, including recruitment, vaccination, exposure, latent progression, recovery, natural death, and disease-induced mortality. Analytical and numerical methods were employed to examine the qualitative behaviour of the model and evaluate the effectiveness of vaccination in controlling disease transmission. The positivity and boundedness analyses confirmed that the model is biologically feasible and mathematically well posed, as all state variables remain non-negative and the total population remains within a biologically meaningful invariant region.

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The disease-free equilibrium was shown to exist uniquely under biologically realistic conditions, while the basic reproduction number was determined as $R_0 = 0.8069837$. Since $R_0 < 1$, the results indicate that an infected individual generates less than one secondary infection on average, implying that diphtheria transmission cannot be sustained and the disease will eventually die out. Local stability analysis using the Jacobian matrix and Routh–Hurwitz criterion, together with global stability analysis based on Lyapunov theory and LaSalle's Invariance Principle, further established that the disease-free equilibrium is both locally and globally asymptotically stable whenever $R_0 < 1$. Consequently, diphtheria elimination is guaranteed irrespective of the initial number of infected individuals.

The findings are consistent with previous studies. In particular, they corroborate the work of (Fauzi et al., 2024), which demonstrated that increased vaccination coverage significantly reduces disease transmission and the basic reproduction number. The results also agree with (Johnson et al., 2024), who reported that sustained vaccination campaigns effectively suppress outbreaks. Furthermore, the findings align with (Kamadjeu et al., 2025), who emphasized that maintaining $R_0 < 1$, is essential for disease elimination. Unlike many earlier studies that relied mainly on numerical simulations, the present study provides rigorous analytical proofs of local and global stability. Overall, the study demonstrates that sustained immunization, booster vaccination, prompt treatment, early diagnosis, and public awareness are indispensable for the long-term control and eventual eradication of diphtheria, while also providing a robust mathematical framework to support evidence-based public health policy and vaccination planning.

Conclusion and Recommendations

Conclusion

This study developed and analysed a deterministic SVLIR mathematical model for investigating the transmission dynamics and control of diphtheria. The model incorporated susceptible, vaccinated, latent, infectious, and removed populations and captured important epidemiological processes including recruitment, vaccination, infection, disease progression, recovery, and mortality. The positivity and boundedness analyses confirmed that the model is biologically feasible and epidemiologically meaningful. The disease-free equilibrium and endemic equilibrium were determined, while the basic reproduction number was derived using the next-generation matrix approach. The computed reproduction number, $R_0 = 0.8069837$, revealed that diphtheria

transmission cannot be sustained in the population under the specified parameter conditions. Local stability analysis using the Jacobian matrix and Routh–Hurwitz criterion established that the disease-free equilibrium is locally asymptotically stable whenever $R_0 < 1$, while global stability analysis based on Lyapunov theory and LaSalle's Invariance Principle demonstrated that disease elimination occurs irrespective of the initial infection level.

Numerical simulations validated the theoretical findings by showing a decline in the latent and infectious populations and a corresponding increase in vaccinated and removed individuals. Additional parameter investigations revealed that vaccination and recovery reduce disease burden, whereas increased transmission and recruitment may facilitate disease persistence if not properly controlled. Overall, the findings demonstrate that sustained immunization programs, timely treatment, and public health awareness are essential for maintaining $R_0 < 1$ and achieving long-term diphtheria eradication. The study therefore provides a useful mathematical framework for guiding evidence-based public health policies aimed at controlling and preventing future diphtheria outbreaks.

Recommendations

Based on the findings of this study, it is recommended that public health authorities continue to strengthen routine immunization programs to ensure that a large proportion of the population remains protected against diphtheria infection. Increasing vaccination coverage directly reduces the number of susceptible individuals and lowers the basic reproduction number, thereby preventing the disease from spreading within the community. It is also important to implement booster vaccination programs to maintain immunity over time. Also, future studies may extend the present model by incorporating additional epidemiological factors such as age structure, spatial distribution, and stochastic effects. Such extensions would provide a more detailed understanding of diphtheria transmission dynamics and help improve the accuracy of predictions for disease control strategies.

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APPENDIX

$$k_1 = (\gamma + \sigma + \mu)$$

$$k_2 = (\varepsilon + \mu + \alpha)$$

$$k_3 = (\gamma\tau)$$

$$k_4 = (\gamma(1-\tau))$$

$$k_5 = (\sigma(1-\tau))$$