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Stability Analysis and Optimal Control of the Dynamics Diphtheria Disease Spread with Education Campaign and Treatment

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ABSTRACT. Diphtheria is an infectious disease caused by the bacterium Corynebacterium diphtheriae and remains a public health problem, particularly in areas with low immunization coverage. This study develops and analyzes a mathematical model to describe the dynamics of diphtheria transmission in a population through equilibrium-point, stability analysis, and the determination of the basic reproduction number. This analysis identifies the conditions distinguishing between disease persistence and elimination. Given the disease's complex transmission pathways, optimal control is required in the early stages, including educational campaign, treatment, and a combination of both interventions to reduce case numbers and lower patient care costs. Simulation results indicate that the simultaneous implementation of control measures is more effective in curbing disease spread than single-measure control. This model provides a structured quantitative basis for evaluating the impact of interventions and supports evidence-based decision-making in early intervention planning and resource allocation, thereby helping to prevent the resurgence of diphtheria.

1. Introduction

Diphtheria is a contagious disease caused by Corynebacterium diphtheriae with a high mortality rate [1]. This disease has the potential to cause serious complications and death, particularly in children, with a mortality rate ranging from 5–10% and potentially rising to 50% in settings with limited access to adequate diagnosis and treatment [2]. Transmission occurs through droplets from an infected individual or carrier and contact with contaminated objects. Globally, more than 25,107 cases were reported in 2024, with the highest concentrations in India and Nigeria [3]. In Indonesia, the trend in diphtheria cases has shown significant fluctuations over the past decade, with an increase from 259 cases in 2020 to 943 cases in 2024. Additionally,

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the number of deaths increased from 46 cases in 2022 to 68 cases in 2023 with the highest number of deaths occurring in several provinces such as West Java, North Sumatra, and East Java [4]. This situation indicates that diphtheria remains a public health issue requiring a more effective and evidence-based control approach.

A deep understanding of the mechanisms of diphtheria transmission is crucial for designing effective control strategies. Mathematical modeling has been widely used as an analytical tool to study the dynamics of infectious diseases, predict patterns of spread, and inform public health policy decisions ([5], [6]). Classic epidemiological models such as the SIR model introduced by Kermack and McKendrick form the basis for the development of various more complex models that account for population heterogeneity and control interventions [7]. In the context of diphtheria, several studies have developed models incorporating aspects of immunity, isolation compartments, and control strategies such as educational campaign and treatment ([8], [9], [10]). However, the global resurgence of diphtheria cases underscores the need for a more comprehensive and realistic modeling approach to represent the transmission dynamics of this disease [11].

To the best of the authors' knowledge, no previous study has simultaneously classified the infected population into asymptomatic and symptomatic exposed individuals, who were then placed in an isolation compartment to be separated and develop immunity, whether partial or full. Therefore, the model in this study was developed by adding an isolation compartment to prevent further transmission. The complexity of diphtheria transmission pathways allows the disease to persist in the population. Thus, this study also applies optimal control measures in the form of education campaign and treatment ([9], [10]). This study aims to analyze the equilibrium point, the stability of the equilibrium point, determine the basic reproduction number (R_0), design an optimal control strategy, and perform mathematical simulations of diphtheria spread with and without control measures.

2. Model Formulation

This model is deterministic, with balanced population entry and exit rates, and considers only one infectious disease namely diphtheria. The population is divided into seven compartments, including the susceptible population (S), the exposed population (E), the asymptomatic exposed population (A), the symptomatic exposed population (I), the isolated population (H), the partially immune population (T_p), and the fully immune population (T_f). The assumptions used in constructing the mathematical model for diphtheria transmission dynamics are:

- The population undergoes recruitment through natural births and reduction through natural deaths across all compartments, as well as deaths due to disease in infected individuals [10].

- The susceptible population has not yet developed immunity to diphtheria because they have not been vaccinated, putting them at high risk of infection. Transmission occurs through close contact with infectious individuals, whether asymptomatic or symptomatic, via respiratory droplets ([3], [12]).
- The exposed population remains in the latent phase (incubation period) for approximately 2–5 days, during which they do not exhibits symptoms and are not yet capable of transmitting the disease. Following the incubation period, the exposed population develops into either an asymptomatic exposed population or a symptomatic exposed population ([3], [13]).
- The asymptomatic exposed population does not exhibits symptoms but can transmit the bacteria to others, whereas the symptomatic exposed population has been infected with the bacteria, exhibits clinical signs or symptoms, and can transmit the disease to others ([3], [13]).
- The infected population can be identified through contact tracing or clinical screening and then transferred to isolation facilities to minimize interaction with vulnerable populations ([9], [13], [14]).
- The isolated population can recover and transition to either full immunity or partial immunity. Full immunity is achieved through full vaccination, whereas partial immunity reflects a suboptimal immune response or one that does not result in sterilizing immunity ([13], [15]).
- The susceptible population can achieve full immunity through diphtheria toxoid vaccination, which significantly reduces the risk of infection ([3], [8]).
- Immunity to diphtheria is not permanent. Populations with full immunity may experience a decline to partial immunity, and populations with partial immunity may revert to a susceptible state as a result of antibody levels falling below the protective threshold ([15], [16], [17]).

Based on the above assumptions, the compartments of the mathematical model for diphtheria are as follows.

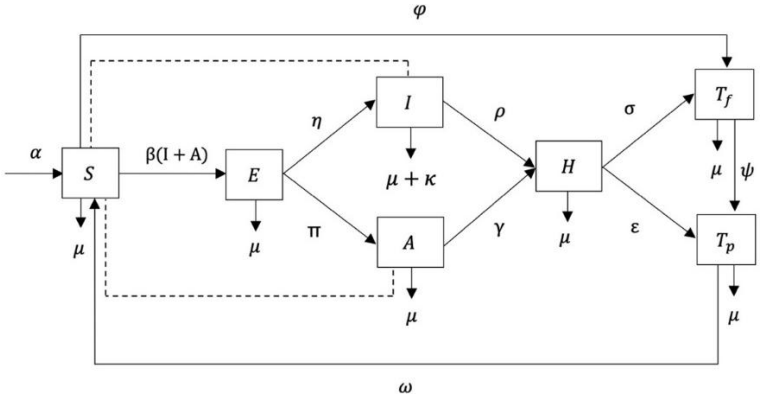


Figure 1. The Dynamics of the Diphtheria Model

Based on the compartment diagram shown in Figure 1, the dynamics of the Diphtheria model are described by the following nonlinear ordinary differential equation system.

$$\begin{cases} \frac{dS}{dt} = \alpha + \omega T_p - (\beta(I+A) + \varphi + \mu)S, \\ \frac{dE}{dt} = \beta(I+A)S - (\eta + \pi + \mu)E, \\ \frac{dA}{dt} = \pi E - (\gamma + \mu)A, \\ \frac{dI}{dt} = \eta E - (\rho + \mu + \kappa)I, \\ \frac{dH}{dt} = \rho I + \gamma A - (\sigma + \varepsilon + \mu)H, \\ \frac{dT_p}{dt} = \varepsilon H + \psi T_f - (\omega + \mu)T_p, \\ \frac{dT_f}{dt} = \sigma H + \varphi S - (\psi + \mu)T_f. \end{cases} \tag{2.1}$$

Based on the preceding conditions:

$$S(0) > 0, E(0) \geq 0, A(0) \geq 0, I(0) \geq 0, H(0) \geq 0, T_p(0) \geq 0, T_f(0) \geq 0, \tag{2.2}$$

where

$$N = S + E + A + I + H + T_p + T_f.$$

3. Qualitative Analysis of the Model

Lemma 3.1. *Assume that the initial conditions satisfy equation* (2.2)*, then all solutions of the system of equations* (2.1) *remain nonnegative for* $t \geq 0$.

Proof. Equations of system (2.1):

$$\frac{dS}{dt} = \alpha + \omega T_p - (\beta(I+A) + \varphi + \mu)S.$$

Since $T_p(t) \geq 0$, for every $t \geq 0$, we obtain:

$$\frac{dS}{dt} + (\beta(I+A) + \varphi + \mu)S \geq \alpha.$$

Let:

$$k_1(t) = \beta(I+A) + \varphi + \mu.$$

Thus, the above equation can be written as:

$$\frac{dS}{dt} + k_1(t)S \geq \alpha.$$

Using the method of integration by parts, we obtain

$$exp\left(\int_0^t k_1(\xi)d\xi\right).$$

Therefore:

$$S(t) \geq \left[S(0) + \int_0^t \alpha\, exp\left(-\int_0^s k_1(\xi)\, d\xi\right) ds\right] exp\left(-\int_0^t k_1(\xi)\, d\xi\right).$$

Since $S(0) \geq 0, \alpha > 0$, and the exponential function is always positive, it follows that $S(t) > 0$ for all $t \geq 0$. By applying a similar argument to the other equations (2.1), it can be concluded that all state variables S, E, A, I, H, T_p, T_f remain nonnegative for all $t \geq 0$. Therefore, all solutions of system (2.1) remain in the nonnegative region for all $t \geq 0$. ■

Lemma 3.2. *Let* $N = S + E + A + I + H + T_p + T_f$ *denote the total population. Then the region*

$$\Omega = \left\{\left(S, E, A, I, H, T_p, T_f\right) \in \mathbb{R}_+^7 : 0 < N \leq \frac{\alpha}{\mu}\right\},$$

remains positively invariant under system (2.1).

Proof. Summing all the differential equations corresponding to the population in system (2.1), we obtain:

$$\frac{dN}{dt} = \alpha - \mu N - \kappa I.$$

Since $I \geq 0$, it follows that:

$$\frac{dN}{dt} \leq \alpha - \mu N.$$

By solving the differential equation using the method of integrating factors, we obtain:

$$N(t) \leq \frac{\alpha}{\mu} + \left(N(0) - \frac{\alpha}{\mu}\right) e^{-\mu t},$$

so that

$$0 < N(t) \leq \frac{\alpha}{\mu}, \text{ for all } t \geq 0.$$

Therefore, all solutions of system (2.1) remain in the region Ω, and hence Ω is a positively invariant region. This ensures that the model is epidemiologically and biologically well-posed.■

Theorem 3.1. *The functions defined on the right hand side of system* (2.1) *consists of polynomial functions of the state variables* S, E, A, I, H, T_p, T_f. *Polynomial functions are continuous and have continuous partial derivatives on the domain* Ω, *and therefore satisfy the local Lipschitz condition. Consequently, by the*

standard existence and uniqueness theorem for ordinary differential equations, system (2.1) *has one unique solution for each initial value given by an initial condition*. ■

4. Model Analysis

4.1. **Equilibrium Point.** The equilibrium point of the system equation (2.1) is obtained when

$$\frac{dS}{dt} = \frac{dE}{dt} = \frac{dA}{dt} = \frac{dI}{dt} = \frac{dH}{dt} = \frac{dT_p}{dt} = \frac{dT_f}{dt} = 0, \tag{4.1}$$

so that it is obtained

$$\begin{cases} \alpha + \omega T_p - (\beta(I + A) + \varphi + \mu)S = 0, \\ \beta(I + A)S - (\eta + \pi + \mu)E = 0, \\ \pi E - (\gamma + \mu)A = 0, \\ \eta E - (\rho + \mu + \kappa)I = 0, \\ \rho I + \gamma A - (\sigma + \varepsilon + \mu)H = 0, \\ \varepsilon H + \psi T_f - (\omega + \mu)T_p = 0, \\ \sigma H + \varphi S - (\psi + \mu)T_f = 0. \end{cases} \tag{4.2}$$

From equation (4.2), the disease-free and endemic equilibrium point will then be sought.

4.1.1. *Disease-Free Equilibrium Point*. The disease-free equilibrium point exists when no one within the population has an infection present. The disease-free equilibrium point can be defined by assuming

$$E = A = I = H = 0.$$

From equation (4.2), the disease-free equilibrium point is obtained as follows

$$E^0 = \left(S^0, 0, 0, 0, 0, {T_p}^0, {T_f}^0\right),$$

where

$$\begin{cases} S^0 = \dfrac{\alpha(\psi + \mu)(\omega + \mu)}{(\varphi + \mu)(\psi + \mu)(\omega + \mu) - \omega\varphi\psi}, \\ {T_p}^0 = \dfrac{\alpha\varphi\psi}{(\varphi + \mu)(\psi + \mu)(\omega + \mu) - \omega\varphi\psi}, \\ {T_f}^0 = \dfrac{\alpha\varphi(\omega + \mu)}{(\varphi + \mu)(\psi + \mu)(\omega + \mu) - \omega\varphi\psi}. \end{cases} \tag{4.3}$$

4.1.2. *Endemic Equilibrium Point*. The endemic equilibrium point is a condition in which disease transmission occurs within a population and occurs when

$$E > 0, A > 0, I > 0, H > 0.$$

From equation (4.2), the endemic equilibrium point is obtained as follows

$$E^1 = \left(S^*, E^*, A^*, I^*, H^*, {T_p}^*, {T_f}^*\right),$$

where

$$\begin{cases} S^* = \dfrac{(\eta+\pi+\mu)(\rho+\mu+\kappa)(\gamma+\mu)}{\beta[\eta(\gamma+\mu)+\pi(\rho+\mu+\kappa)]}, \\ E^* = \dfrac{\rho+\mu+\kappa}{\eta} I^*, \\ A^* = \dfrac{\pi(\rho+\mu+\kappa)}{\eta(\gamma+\mu)} I^*, \\ I^* = \dfrac{a_2\left[a_1\left((\varphi+\mu)S^*-\alpha\right)\right]-\omega\psi\varphi S^*}{a_1[\omega(\rho\eta(\gamma+\mu)+\gamma\pi(\rho+\mu+\kappa))(\varepsilon(\psi+\mu)+\psi\sigma)-a_2a_3S^*]}, \\ H^* = \dfrac{\rho\eta(\gamma+\mu)+\gamma\pi(\rho+\mu+\kappa)}{\eta(\gamma+\mu)(\sigma+\varepsilon+\mu)} I^*, \\ {T_p}^* = \dfrac{(\rho\eta(\gamma+\mu)+\gamma\pi(\rho+\mu+\kappa))(\varepsilon(\psi+\mu)+\psi\sigma)}{\eta(\gamma+\mu)(\sigma+\varepsilon+\mu)(\psi+\mu)(\omega+\mu)} I^* + \dfrac{\psi\varphi S^*}{(\psi+\mu)(\omega+\mu)}, \\ {T_f}^* = \dfrac{\sigma(a_4I^*)+\varphi S^*}{\psi+\mu}. \end{cases} \tag{4.4}$$

Description:

$a_1 = (\psi+\mu)(\omega+\mu),$

$a_2 = \eta(\gamma+\mu)(\sigma+\varepsilon+\mu)(\psi+\mu)(\omega+\mu),$

$a_3 = \beta\left(1+\dfrac{\pi(\rho+\mu+\kappa)}{\eta(\gamma+\mu)}\right),$

$a_4 = \dfrac{\rho\eta(\gamma+\mu)+\gamma\pi(\rho+\mu+\kappa)}{\eta(\gamma+\mu)(\sigma+\varepsilon+\mu)}.$

4.2. The Equilibrium Point Local Stability Analysis

Lemma 4.1. *Let E be an equilibrium point of system* (2.1). *The equilibrium point E is said to be locally asymptotically stable if and only if all of the eigenvalues of its Jacobian matrix $J(E)$ have negative real parts. On the other hand, the equilibrium point is deemed unstable if there is at least one eigenvalue with a positive real component.*

Proof. Consider the nonlinear dynamical system described in equation (2.1). By linearizing the system around an equilibrium point E, the resulting linearized system can be written as

$$\frac{dX}{dt} = J(E)X,$$

where $J(E)$ is the Jacobian matrix evaluated at the equilibrium point. ■

Theorem 4.1. *The right–hand side of system* (2.1) *consists of polynomial functions of the state variables S, E, A, I, H, T_p, T_f. Polynomial functions are continuous and have continuous partial derivatives on the*

domain Ω, and therefore satisfy the local Lipschitz condition. Consequently, by the standard existence and uniqueness theorem for ordinary differential equations, system (2.1) *admits a unique solution for every given initial condition. The stability of the linearized system depends on the eigenvalues of $J(E)$ with all eigenvalues having negative real values guaranteeing that E is locally asymptotically stable. Otherwise, if at least one eigenvalue has a positive real part then E will become unstable.* ■

The stability analysis of the equilibrium point of system (2.1) is performed using the Jacobian matrix method as described in [18]. The Jacobian matrix of system (2.1) is expressed as follows

$$J(E) = \begin{bmatrix} x_1 & 0 & -x_2 & -x_2 & 0 & \omega & 0 \\ x_3 & x_4 & x_2 & x_2 & 0 & 0 & 0 \\ 0 & \pi & x_5 & 0 & 0 & 0 & 0 \\ 0 & \eta & 0 & x_6 & 0 & 0 & 0 \\ 0 & 0 & \gamma & \rho & x_7 & 0 & 0 \\ 0 & 0 & 0 & 0 & \varepsilon & x_8 & \psi \\ \varphi & 0 & 0 & 0 & \sigma & 0 & x_9 \end{bmatrix}, \tag{4.5}$$

where

$x_1 = -(\beta(I + A) + \varphi + \mu),$

$x_2 = \beta S,$

$x_3 = \beta(I + A),$

$x_4 = -(\eta + \pi + \mu),$

$x_5 = -(\gamma + \mu),$

$x_6 = -(\rho + \mu + \kappa),$

$x_7 = -(\sigma + \varepsilon + \mu),$

$x_8 = -(\omega + \mu),$

$x_9 = -(\psi + \mu).$

4.2.1. *The Stability Analysis of the Disease-Free Equilibrium Point* (E^0). The disease-free equilibrium point E^0 will be substituted into the matrix J in equation (4.5), yielding $J(E^0)$ as

$$J(E^0) = \begin{bmatrix} x_1 & 0 & -x_2 & -x_2 & 0 & \omega & 0 \\ 0 & x_3 & x_2 & x_2 & 0 & 0 & 0 \\ 0 & \pi & x_4 & 0 & 0 & 0 & 0 \\ 0 & \eta & 0 & x_5 & 0 & 0 & 0 \\ 0 & 0 & \gamma & \rho & x_6 & 0 & 0 \\ 0 & 0 & 0 & 0 & \varepsilon & x_7 & \psi \\ \varphi & 0 & 0 & 0 & \sigma & 0 & x_8 \end{bmatrix}, \tag{4.6}$$

where

$x_1 = -(\varphi + \mu),$

$x_2 = \beta S^0,$

$x_3 = -(\eta + \pi + \mu),$

$x_4 = -(\gamma + \mu),$

$x_5 = -(\rho + \mu + \kappa)$,
$x_6 = -(\sigma + \varepsilon + \mu)$,
$x_7 = -(\omega + \mu)$,
$x_8 = -(\psi + \mu)$.

The stability of E^0 is determined from the characteristic equation

$$\det(J(E^0) - \lambda I) = 0.$$

Based on the structure of $J(E^0)$, the characteristic polynomial can be factorized as

$$(\lambda - x_1)(\lambda - x_6)(\lambda - x_7)(\lambda - x_8)P(\lambda) = 0, \tag{4.7}$$

where

$$P(\lambda) = a_3\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0,$$

with

$a_3 = 1$,
$a_2 = (\eta + \pi + \mu) + (\gamma + \mu) + (\rho + \mu + \kappa)$,
$a_1 = (\eta + \pi + \mu)(\gamma + \mu) + (\eta + \pi + \mu)(\rho + \mu + \kappa) + (\gamma + \mu)(\rho + \mu + \kappa) - \beta S^0(\pi + \eta)$,
$a_0 = (\eta + \pi + \mu)(\gamma + \mu)(\rho + \mu + \kappa) - \beta S^0\pi(\rho + \mu + \kappa) - \beta S^0\eta(\gamma + \mu)$.

The first four eigenvalues are:

$$\begin{cases} \lambda_1 = -(\varphi + \mu) < 0, \\ \lambda_2 = -(\sigma + \varepsilon + \mu) < 0, \\ \lambda_3 = -(\omega + \mu) < 0, \\ \lambda_4 = -(\psi + \mu) < 0. \end{cases}$$

By the Routh–Hurwitz criterion for a cubic polynomial $P(\lambda)$, all roots have negative real parts if

$$a_3 > 0, a_2 > 0, a_1 > 0, a_0 > 0, a_2a_1 > a_3a_0.$$

Since $a_3 = 1$, the conditions reduce to

$$a_2 > 0, a_1 > 0, a_0 > 0, a_2a_1 > a_0.$$

Hence, $\lambda_5 < 0, \lambda_6 < 0$, and $\lambda_7 < 0$. Therefore, all eigenvalues of $J(E^0)$ have negative real parts whenever $R_0 < 1$, implying that E^0 is locally asymptotically stable. Conversely, E^0 is unstable when $R_0 > 1$.

Theorem 4.2. *The disease-free equilibrium point E^0 of system* (2.1) *is locally asymptotically stable if the basic reproduction number satisfies $R_0 < 1$ and unstable if $R_0 > 1$.*

Proof. From the characteristic equation of $J(E^0)$, four eigenvalues are explicitly negative, namely

$$\lambda_1 = -(\varphi + \mu), \lambda_2 = -(\sigma + \varepsilon + \mu), \lambda_3 = -(\omega + \mu), \lambda_4 = -(\psi + \mu).$$

The remaining three eigenvalues are determined from the cubic polynomial

$$P(\lambda) = a_3\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0, \quad a_3 = 1.$$

By the Routh–Hurwitz criterion, all roots of $P(\lambda)$ have negative real parts if

$$a_3 > 0, a_2 > 0, a_1 > 0, a_0 > 0, a_2a_1 > a_3a_0.$$

Hence, $\lambda_5 < 0, \lambda_6 < 0$, and $\lambda_7 < 0$. Therefore, all eigenvalues of $J(E^0)$ have negative real parts whenever $R_0 < 1$, implying that E^0 is locally asymptotically stable. Conversely, E^0 is unstable when $R_0 > 1$. ■

4.2.2. *The Stability Analysis of Endemic Equilibrium Point* (E^1). The endemic equilibrium point E^1 will be substituted into the matrix J in equation (4.5), yielding $J(E^1)$ as

$$J(E^1) = \begin{bmatrix} x_1 & 0 & -x_2 & -x_2 & 0 & \omega & 0 \\ x_3 & x_4 & x_2 & x_2 & 0 & 0 & 0 \\ 0 & \pi & x_5 & 0 & 0 & 0 & 0 \\ 0 & \eta & 0 & x_6 & 0 & 0 & 0 \\ 0 & 0 & \gamma & \rho & x_7 & 0 & 0 \\ 0 & 0 & 0 & 0 & \varepsilon & x_8 & \psi \\ \varphi & 0 & 0 & 0 & \sigma & 0 & x_9 \end{bmatrix}, \tag{4.8}$$

where

$x_1 = -(\beta(I^* + A^*) + \varphi + \mu),$
$x_2 = \beta S^*,$
$x_3 = \beta(I^* + A^*),$
$x_4 = -(\eta + \pi + \mu),$
$x_5 = -(\gamma + \mu),$
$x_6 = -(\rho + \mu + \kappa),$
$x_7 = -(\sigma + \varepsilon + \mu),$
$x_8 = -(\omega + \mu),$
$x_9 = -(\psi + \mu).$

The stability of E^1 is determined from the characteristic equation

$$\det(J(E^1) - \lambda I) = 0.$$

Based on the structure of $J(E^1)$, the characteristic polynomial can be factorized as

$$(\lambda - x_5)(\lambda - x_6)(\lambda - x_7)(\lambda - x_8)(\lambda - x_9)P(\lambda) = 0, \tag{4.9}$$

where

$$P(\lambda) = a_2\lambda^2 + a_1\lambda + a_0,$$

with

$a_2 = 1,$

$a_1 = (\beta(I^* + A^*) + \varphi + \mu) + (\eta + \pi + \mu),$

$a_0 = (\beta(I^* + A^*) + \varphi + \mu)(\eta + \pi + \mu) - \beta^2 S^*(I^* + A^*).$

The first five eigenvalues are:

$$\begin{cases} \lambda_1 = -(\gamma + \mu) < 0, \\ \lambda_2 = -(\rho + \mu + \kappa) < 0, \\ \lambda_3 = -(\sigma + \varepsilon + \mu) < 0, \\ \lambda_4 = -(\omega + \mu) < 0, \\ \lambda_5 = -(\psi + \mu) < 0. \end{cases}$$

By the Routh–Hurwitz criterion for a cubic polynomial $P(\lambda)$, all roots have negative real parts if

$$a_2 > 0, a_1 > 0, a_0 > 0.$$

Since $a_2 = 1$, the conditions reduce to

$$a_1 > 0 \text{ and } a_0 > 0.$$

Hence, $\lambda_6 < 0$ and $\lambda_7 < 0$. Therefore, all eigenvalues of $J(E^1)$ have negative real parts whenever $R_0 > 1$, implying that E^1 is locally asymptotically stable.

Theorem 4.3. *The endemic equilibrium point E^1 of system* (2.1) *is locally asymptotically stable whenever* $R_0 > 1$.

Proof. From the characteristic equation of $J(E^1)$, four eigenvalues are explicitly negative, namely

$$\lambda_1 = -(\gamma + \mu), \lambda_2 = -(\rho + \mu + \kappa), \lambda_3 = -(\sigma + \varepsilon + \mu), \lambda_4 = -(\omega + \mu), \lambda_5 = -(\psi + \mu).$$

The remaining two eigenvalues are determined from the quadratic polynomial

$$P(\lambda) = a_2\lambda^2 + a_1\lambda + a_0, \quad a_2 = 1.$$

By the Routh–Hurwitz criterion, all roots of $P(\lambda)$ have negative real parts if

$$a_2 > 0, a_1 > 0, a_0 > 0.$$

Hence, $\lambda_6 < 0$ and $\lambda_7 < 0$. Therefore, all eigenvalues of $J(E^1)$ have negative real parts whenever $R_0 > 1$, implying that E^1 is locally asymptotically stable. ■

4.3. **Equilibrium Point Global Stability Analysis**

4.3.1. The Global Stability Analysis of Disease-Free Equilibrium Point

Theorem 4.4. *If $R_0 < 1$, the disease-free equilibrium E^0 of system (2.1) is globally asymptotically stable in the feasible region Ω.*

Proof. To establish the global stability of the disease-free equilibrium, we employ the approach of Castillo–Chavez and Feng. First, the system (2.1) is rewritten by separating the uninfected and infected compartments.

Let the uninfected compartment vector be

$$X = \left(S^0, {T_p}^0, {T_f}^0\right),$$

and the infected compartment vector will be written as

$$I = (E, A, I, H).$$

Thus, we can write system (2.1) in the form:

$$\frac{dX}{dt} = F(X, I), \frac{dI}{dt} = G(X, I), G(X, 0) = 0.$$

Condition H_1

Consider the subsystem obtained when $I = 0, \frac{dX}{dt} = F(X, 0)$, namely:

$$\begin{cases} \dfrac{dS^0}{dt} = \alpha + \omega {T_p}^0 - (\varphi + \mu)S^0, \\ \dfrac{d{T_p}^0}{dt} = \psi {T_f}^0 - (\omega + \mu){T_p}^0, \\ \dfrac{d{T_f}^0}{dt} = \varphi S^0 - (\psi + \mu){T_f}^0. \end{cases}$$

The equilibrium point of this subsystem is

$$X^* = \left(\frac{\alpha(\psi + \mu)(\omega + \mu)}{(\varphi + \mu)(\psi + \mu)(\omega + \mu) - \omega\varphi\psi}, \frac{\psi}{(\omega + \mu)}S^0, \frac{\varphi}{(\psi + \mu)}S^0\right).$$

It is straightforward to verify that X^* is globally asymptotically stable. Thus, condition H_1 is satisfied.

Condition H_2

The infected subsystem is given by

$$G(X, I) = BI - \hat{G}(X, I),$$

where B is the Jacobian matrix of $G(X,I)$ evaluated at the disease-free equilibrium E^0. The matrix B is given by

$$B = \begin{bmatrix} \beta S^0 - (\eta + \pi + \mu) & \beta S^0 & \beta S^0 & 0 \\ \pi & -(\gamma + \mu) & 0 & 0 \\ \eta & 0 & -(\rho + \mu + \kappa) & 0 \\ 0 & \gamma & \rho & -(\sigma + \varepsilon + \mu) \end{bmatrix}.$$

It can be verified that B is an **M-matrix**, since all off-diagonal elements are nonnegative and all eigenvalues have negative real parts. Furthermore, $\hat{G}(X,I) \geq 0$ for all $(X,I) \in \Omega$. Thus condition H_2 is satisfied. Since conditions H_1 and H_2 are satisfied, it follows from the theorem of Castillo–Chavez that the disease-free equilibrium E^0 of system (2.1) is globally asymptotically stable whenever $R_0 < 1$. This completes the proof. ■

4.3.2. The Global Stability Analysis of Endemic Equilibrium Point

Theorem 4.5. *If $R_0 > 1$, then the endemic equilibrium point E^1 of system* (2.1) *is globally asymptotically stable in the feasible region M.*

Proof. Let the endemic equilibrium of system (2.1) be denoted by

$$E^1 = (S^*, E^*, A^*, I^*, H^*, {T_p}^*, {T_f}^*).$$

which exists whenever $R_0 > 1$.

To show E^1 is globally stable, we define an appropriate Lyapunov function:

$$L = S^*\left(\frac{S}{S^*} - ln\frac{S}{S^*} - 1\right) + E^*\left(\frac{E}{E^*} - ln\frac{E}{E^*} - 1\right) + A^*\left(\frac{A}{A^*} - ln\frac{A}{A^*} - 1\right) + I^*\left(\frac{I}{I^*} - ln\frac{I}{I^*} - 1\right) + H^*\left(\frac{H}{H^*} - ln\frac{H}{H^*} - 1\right) + {T_p}^*\left(\frac{T_p}{{T_p}^*} - ln\frac{T_p}{{T_p}^*} - 1\right) + {T_f}^*\left(\frac{T_f}{{T_f}^*} - ln\frac{T_f}{{T_f}^*} - 1\right).$$

Clearly, $L \geq 0$, and $L = 0$ if and only if $\mathrm{X} = E^1$, derivative of the Lyapunov function taking the time derivative of L along the solutions of system (2.1), we find

$$\frac{dL}{dt} = \sum_i \left(1 - \frac{X_i^*}{X_i}\right)\frac{dX_i^*}{dt}.$$

Substituting the model equations into the above expression, and using the equilibrium relations satisfied at E^1, the derivative simplifies to substituting the differential equations of the model into the above expression and simplifying yields

$$\frac{dL}{dt} \leq 0, \text{for all } X \in \Omega.$$

Invariant Set
Morever,

$$\frac{dL}{dt} = 0,$$

if and only if $X \in E^1$, Thus, the largest invariant set contained in:

$$\left\{X \in \Omega : \frac{dL}{dt} = 0\right\},$$

is the singleton set $\{E^1\}$.
By LaSalle's Invariance Principle, every solution of system (2.1) with initial conditions in Ω only go to the endemic equilibrium E^1 as $t \to \infty$. Hence, the endemic equilibrium E^1 is globally asymptotically stable whenever $R_0 > 1$.

4.4. **Basic Reproduction Number.** The basic reproduction number (R_0) is the average number of susceptible individuals who can be directly infected by one infected individual when the entire population is still completely susceptible to infection. When $R_0 < 1$, the disease will disappear, whereas when $R_0 > 1$, the disease will persist and spread within the population [19]. The calculation of R_0 uses the Next Generation Matrix approach, defined as $K = FV^{-1}$, where F is the new infection matrix and V is the transition rate matrix. Furthermore, R_0 can be expressed as $R_0 = \rho(FV^{-1})$, where $\rho(FV^{-1})$ is the spectral radius of the K matrix [20]. The basic reproduction number found from solving the system shown in equation (2.1), can be described as follows

$$R_0 = \beta S^0 \left(\frac{\pi}{(\eta + \pi + \mu)(\gamma + \mu)} + \frac{\eta}{(\eta + \pi + \mu)(\rho + \mu + \kappa)}\right). \tag{4.10}$$

5. Optimal Control Problems

This study applies an optimal control approach to a diphtheria disease spread model for testing multiple intervention strategies on diphtheria transmission dynamics. Two forms of control are used in the model, namely education campaign (u_1) and treatment (u_2). The objective is to determine the most effective intervention strategy in minimizing the number of infected individuals while reducing the intervention costs incurred. Three control scenarios were analyzed: (i) implementation of u_1, (ii) implementation of u_2, and (iii) a combination of u_1 and u_2.

5.1. **Education Campaign (u_1).** An educational campaign is a series of organized communication activities aimed at increasing public awareness of an issue. In diphtheria control, such campaign help reduce risky contacts and promote preventive behaviors, supporting overall control strategies [9]. The objective functional is defined as:

$$min\mathcal{J} = \min_{(u_1)} \int_{t_0}^{t_f} \left[AI + \frac{1}{2}B_1 u_1^2\right] dt \tag{5.1}$$

subject to the state system:

$$\begin{cases} \frac{dS}{dt} = \alpha + \omega T_p - \big((1-u_1)\beta(I+A) + \varphi + \mu\big)S, \\ \frac{dE}{dt} = (1-u_1)\beta(I+A)S - (\eta + \pi + \mu)E, \\ \frac{dA}{dt} = \pi E - (\gamma + \mu)A, \\ \frac{dI}{dt} = \eta E - (\rho + \mu + \kappa)I, \\ \frac{dH}{dt} = \gamma A + \rho I - (\sigma + \varepsilon + \mu)H, \\ \frac{dT_p}{dt} = \varepsilon H + \psi T_f - (\omega + \mu)T_p, \\ \frac{dT_f}{dt} = \sigma H + \varphi S - (\psi + \mu)T_f. \end{cases} \tag{5.2}$$

The symbol of t_f is the duration of the intervention effort, coefficient A is the weight of the population exposed to symptoms (I), and coefficient B_1 is the weight of the education campaign. Using Pontryagin Minimum Principle, the Hamiltonian function for this problem can be written as follows by (5.3).

$$H = A_1 I + \frac{1}{2}B_1 u_1^2 + \lambda_1 \dot{S} + \lambda_2 \dot{E} + \lambda_3 \dot{A} + \lambda_4 \dot{I} + \lambda_5 \dot{H} + \lambda_6 \dot{T_p} + \lambda_7 \dot{T_f}. \tag{5.4}$$

The equations for the state are defined as

$$\dot{x} = \frac{\partial H}{\partial \lambda}. \tag{5.5}$$

The equations for the costate are defined as

$$\dot{\lambda} = -\frac{\partial H}{\partial x}. \tag{5.6}$$

With the transversality conditions

$$\lambda_i(t_f) = 0, i = 1, \cdots, 7.$$

The stationary point can be derived by differentiating the Hamiltonian with respect to the control variable u_1 expressed as follows

$$\frac{\partial H}{\partial u_1} = 0,$$

$$\frac{\partial H}{\partial u_1} = B_1 u_1 - (\lambda_1 - \lambda_2)S,$$

$$u_1 = \frac{(\lambda_1 - \lambda_2)S}{B_1}.$$

The boundary condition for control u_1 is defined as $0 \leq u_1 \leq 1$, the optimal control is expressed as follows

$$u_1^* = min\left\{ max\left\{0, \frac{(\lambda_1 - \lambda_2)S}{B_1}\right\}, 1\right\}.$$

5.2. **Treatment** $(\boldsymbol{u_2})$. Treatment for diphtheria involves two main approaches: the use of antitoxins and antibiotics. Suspected cases of diphtheria must be immediately isolated and treated without waiting for laboratory confirmation, and kept isolated until cultures show negative results after treatment. The combination of isolation and treatment is effective in reducing morbidity and breaking the chain of transmission ([3], [21]). The objective functional is defined as:

$$min\mathcal{J} = \min_{(u_2)} \int_{t_0}^{t_f} \left[AI + \frac{1}{2}B_2 u_2^2\right] dt \tag{5.7}$$

subject to the state system:

$$\begin{cases} \frac{dS}{dt} = \alpha + \omega T_p - (\beta(I + A) + \varphi + \mu)S, \\ \frac{dE}{dt} = \beta(I + A)S - (\eta + \pi + \mu)E, \\ \frac{dA}{dt} = \pi E - (\gamma + \mu)A, \\ \frac{dI}{dt} = \eta E - (\rho + \mu + \kappa)I, \\ \frac{dH}{dt} = \rho I + \gamma A - (\sigma + u_2 + \varepsilon + \mu)H, \\ \frac{dT_p}{dt} = \varepsilon H + \psi T_f - (\omega + \mu)T_p, \\ \frac{dT_f}{dt} = \varphi S + (\sigma + u_2)H - (\psi + \mu)T_f. \end{cases} \tag{5.8}$$

The Hamiltonian function is given in equation (5.9), with the corresponding state in equation (5.10), the costate in equation (5.11), and the stationary condition given by

$$u_2 = \frac{(\lambda_5 - \lambda_7)H}{B_2}.$$

Thus, the optimal treatment control is

$$u_2^*(t) = \min\left\{ max\left\{0, \frac{(\lambda_5 - \lambda_7)H}{B_2}\right\}, 1\right\}.$$

5.3. **Combined Control of** $(\boldsymbol{u_1})$ **and** $(\boldsymbol{u_2})$. In this scenario, optimal control aims to minimize the objective function by combining education campaign (u_1) and treatment (u_2). The total cost function to be minimized is given by the following expression:

$$min\mathcal{J} = \min_{(u_1,u_2)} \int_{t_0}^{t_f} \left[AI + \frac{1}{2}B_1 u_1^2 + \frac{1}{2}B_2 u_2^2\right] dt \tag{5.12}$$

with constraints on the system of equation (5.13). The Hamiltonian function is defined in equation (5.14), from which the state equation (5.15), the costate equation (5.16), and the stationary is obtained using Pontryagin Minimum Principle.

Theorem 5.1. *Suppose u_1^* and u_2^* are optimal controls minimizing the objective functional subject to the state system. Then there exist adjoint variables $\lambda_i(t), i = 1, \cdots, 7$, such that the following conditions hold*:

1. State system.
   The state variables satisfy the controlled system given in Equation (5.13).
2. Costate system
   The adjoint variables satisfy $\dot{\lambda} = -\frac{\partial H}{\partial x}, i = 1, \cdots, 7$.
3. Optimal conditions
   $\lambda_i(t_f) = 0, i = 1, \cdots, 7$. The optimal controls are given by

$$u_1^* = min\left\{ max\left\{0, \frac{(\lambda_1 - \lambda_2)S}{B_1}\right\}, 1\right\}, u_2^* = min\left\{max\left\{0, \frac{(\lambda_5 - \lambda_7)H}{B_2}\right\}, 1\right\}.$$

which satisfy the Pontryagin Minimum Principle.

6. Numerical Simulation

Numerical simulations were performed to describe the dynamics of diphtheria transmission using a compartment model that represents the clinical conditions and immunity status of the population, namely susceptible (S), exposed (E), asymptomatic exposed (A), symptomatic exposed (I), isolated (H), partially immune (T_p), and fully immune (T_f). The simulation was performed by combining the Forward-Backward Sweep method with the fourth-order Runge–Kutta method to solve the nonlinear differential equation system. All simulations were performed using Python over a simulation period of 100 days.

The total population is $N(0) = 5{,}695{,}475$. Then $S(0) = 1{,}936{,}138$, $E(0) = 11$, $A(0) = 201$, $I(0) = 67$, $H(0) = 45$, $T_p(0) = 341{,}728$, $T_f(0) = 3{,}417{,}285$. For the optimal control simulations, the weighting coefficients were set to $A = 1.0, B_1 = 0.1$, and $B_2 = 0.1$. The total population was obtained from [22]. The number of exposed individuals with symptoms is used as the basis for determining the initial conditions of the diphtheria spread dynamics model obtained from [23]. The isolated population is assumed to be 60% of the exposed population with symptoms and 2.5% of the exposed population without symptoms ([9], [13]). The asymptomatic exposed population is assumed to be three times the symptomatic exposed population. The exposed population is determined based on a comparison of the incubation period, which ranges from 2 to 5 days, and the duration of diphtheria infection in untreated individuals, which can last an average of approximately 18.5 days. Based on the number of exposed cases with symptoms of 67

people and an average incubation period of about 3 days, the exposed population is estimated to be around 11 people ([3], [13]).

The fully immune population is assumed to be 60% of the total population. The partially immune population is assumed to be 10% of the fully immune population [13]. The susceptible population is determined as the difference between the total population and all other compartments. This approach was chosen due to the limited availability of detailed quantitative data on population distribution at each stage of diphtheria infection at the population level. Therefore, a combination of epidemiological surveillance data and conservative biological assumptions was used to maintain the biological realism and numerical stability of the model. The parameter values are presented in Table 1.

Table 1. Parameters Values for the Diphtheria Model

Parameter	Description	Values	Unit	Reference
α	Natural birth rate	30	Day^{-1}	[9]
β	Rate of movement from the vulnerable population to the exposed population	0.00003	Day^{-1}	[10]
η	Rate of movement from the exposed population to the exposed population with symptoms	0.2	Day^{-1}	[24]
π	Rate of movement from the exposed population to the exposed population without symptoms	0.143	Day^{-1}	[25]
ρ	Rate of movement from the exposed population with symptoms to the isolated population	0.1	Day^{-1}	[26]
γ	Rate of movement from the asymptomatic exposed population to the isolated population	0.214	Day^{-1}	[9]
σ	Rate of movement from the isolated population to the fully immune population	0.005	Day^{-1}	[9]
ε	Rate of movement from the isolated population to the partially immune population	0.0001	Day^{-1}	Assumed
φ	Rate of movement from the susceptible population to the fully immune population	0.10	Day^{-1}	[9]
ψ	Rate of migration from a fully immune population to a partially immune population	0.067	Day^{-1}	[10]
ω	Rate of migration from a partially immune population to a susceptible population	0.0667	Day^{-1}	[8]
μ	Natural death rate	0.0019	Day^{-1}	[5]
κ	Death rate due to infection	0.0005	Day^{-1}	[10]

Model parameters are determined based on real data, references from previous studies, and epidemiological assumptions. The natural birth rate (α), the rate of transition from exposed without symptoms to isolation (γ), the rate of transition from isolation to full immunity (σ), and the vaccination rate (φ) are obtained from [9]. The rate of transition from exposed to exposed with symptoms (η) are obtained from [24]. The transmission rate (β), the rate of decline from full immunity to partial immunity (ψ), and the mortality rate due to infection (κ) are obtained from [10]. The transition rate from exposed to asymptomatic exposed (π) are obtained from [25]. The transition rate from the symptomatic exposed population to isolation (ρ) are obtained from [26]. The transition rate from isolation to partial immunity (ε) is assumed based on the ratio of the initial value H to T_p. The transition rate from partial immunity to susceptibility (ω) are obtained from [8]. The natural death rate (μ) are obtained from [5].

6.1. **Stability Analysis for $R_0 < 1$.** Based on the results of numerical simulations using the initial values and parameter values, the condition $R_0 = 0.9 < 1$ was obtained. The disease-free equilibrium point is (4,081, 0, 0, 0, 0, 5,785, 5,923). All the resulting eigenvalues are negative, namely (-0.47722, -0.17960, -0.11875, -0.11875, -0.00700, -0.00636, -0.00190). This indicates that the disease-free equilibrium point is locally asymptotically stable.

6.2. **Model Parameter Sensitivity Index.** One widely used method of quantifying how sensitivity R_0 is to parameter changes in epidemiological models is the *Normalized Forward Sensitivity Index*. This method is used to assess the extent to which a change in a parameter affects the value of R_0 which is the main indicator in determining whether an infectious disease will spread or disappear [20]. This method is defined as a measure of the relative change in R_0 with respect to a change in parameter (p) [18], which is defined mathematically in the following manner

$$\Upsilon_p^{R_0} = \frac{\partial R_0}{\partial p} \cdot \frac{p}{R_0},$$

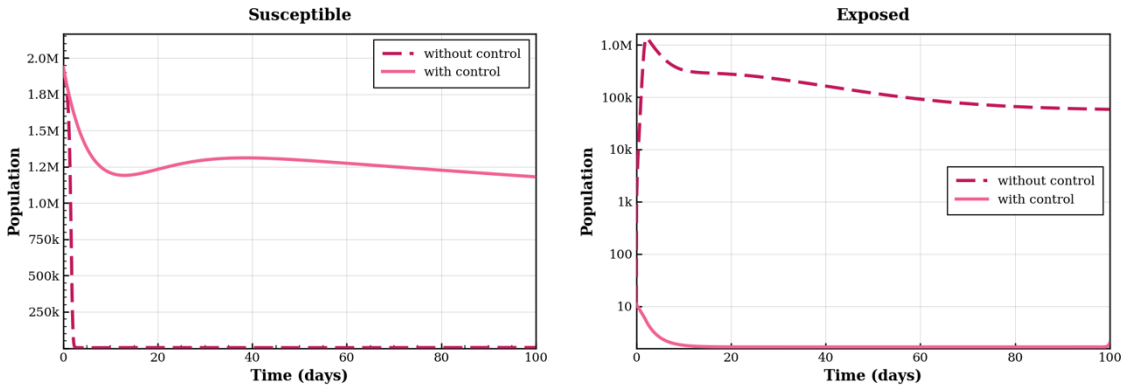
where p represents the model parameters.

A sensitivity analysis was conducted to identify the key parameters that influence the basic reproduction number (R_0). A positive sensitivity index indicates that an increase in the parameter value will increase R_0, while a negative index indicates the opposite effect. The parameters with the highest positive sensitivity indices are the susceptible population entry rate (α) and the transmission rate (β), both with a value of 1, followed by $\eta = 0.1668$, $\omega = 0.3562$, and $\psi = 0.3546$, all of which contribute to increasing R_0. Conversely, the parameters with the largest negative sensitivity indices are the natural mortality rate $\mu = -0.9909$, the vaccination rate $\varphi = -0.7415$, $\pi = -0.1613$, $\rho = -0.7292$, $\gamma = -0.2510$, and $\kappa = -0.0036$, which act to suppress the

value of R_0. These results confirm that increasing vaccination coverage and accelerating case isolation are the most effective strategies for controlling the spread of diphtheria.

6.3. **Stability Analysis for $R_0 > 1$.** Based on the results of the sensitivity analysis, increasing the parameter α to 35 and β to 0.000036, respectively, yields $R_0 = 1.1 > 1$, indicating that an increase in the birth rate of the susceptible population or in the intensity of transmission contact can sustain the spread of diphtheria within the population. Under conditions of increased α, the disease-free equilibrium point (4,761, 0, 0, 0, 0, 6,749, 6,910) has a single positive eigenvalue (0.00722) and is therefore unstable, while the endemic equilibrium point (4,395, 23, 15, 46, 1,149, 6,314, 6,463) has all negative eigenvalues and is therefore asymptotically stable. Similarly, as β increases, the disease-free equilibrium point (4,081, 0, 0, 0, 0, 5,785, 5,923) is unstable with one positive eigenvalue (0.00987), while the endemic equilibrium point (3,663, 27, 18, 53, 1,314, 5,287, 5,411) is asymptotically stable with all negative eigenvalues. These results indicate that when $R_0 > 1$, the disease-free equilibrium becomes unstable and the system converges to the stable endemic equilibrium, so that diphtheria tends to become established in the population.

6.4. **Numerical Simulation with Control**. Although the basic reproduction number is less than one, optimal control measures for diphtheria are still necessary to maintain long-term stability of the system because population dynamics, mobility, density, and potential decline in immunity can increase transmission again. Parameters with high sensitivity such as effective contact rate and infection progression have the potential to increase the basic reproduction number again if not controlled consistently. Therefore, numerical simulations were conducted in three scenarios: a model with an education campaign control variable (u_1), a model with a treatment control variable (u_2), and a model with two control variables (u_1) and (u_2).



(a) (b)

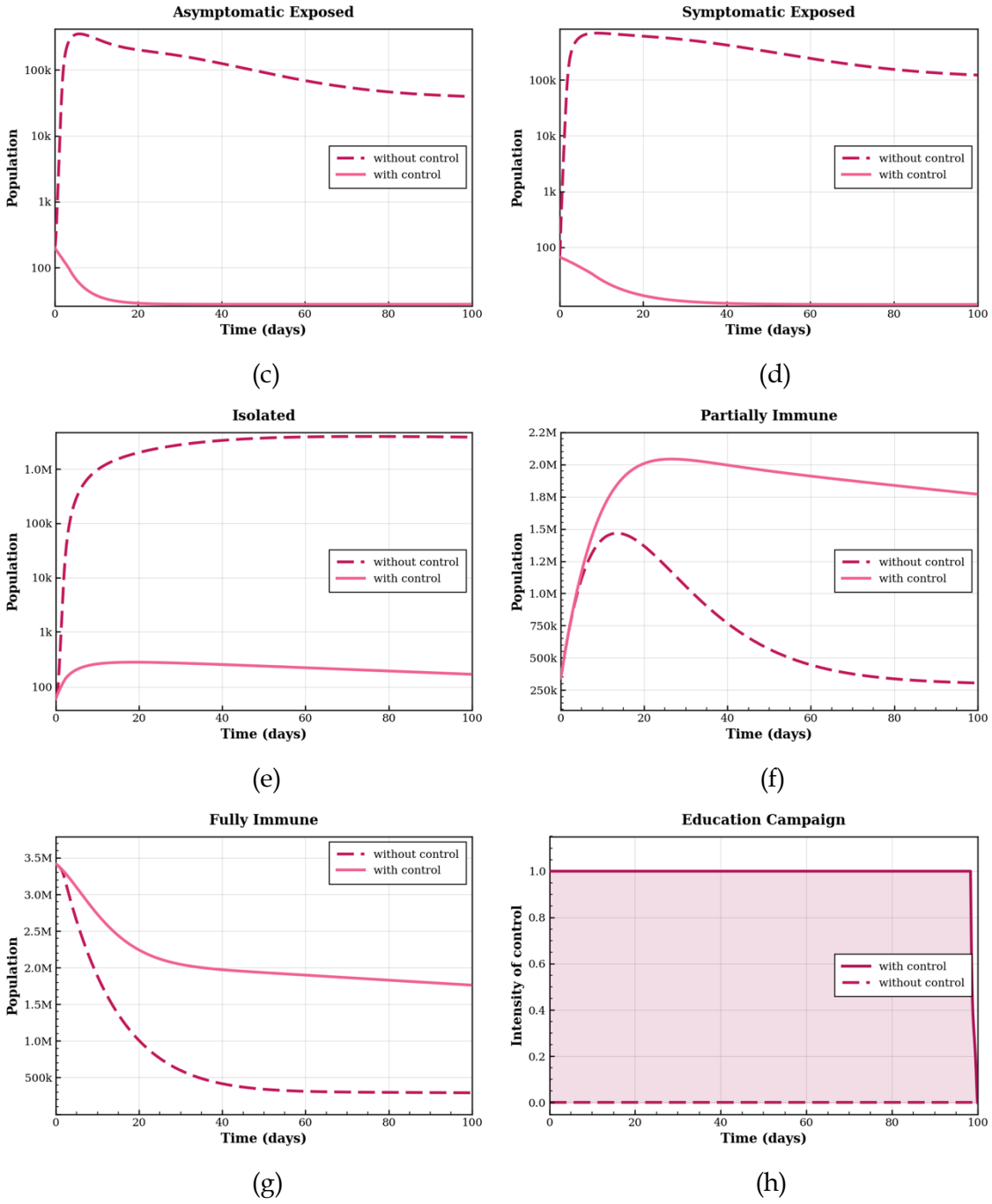
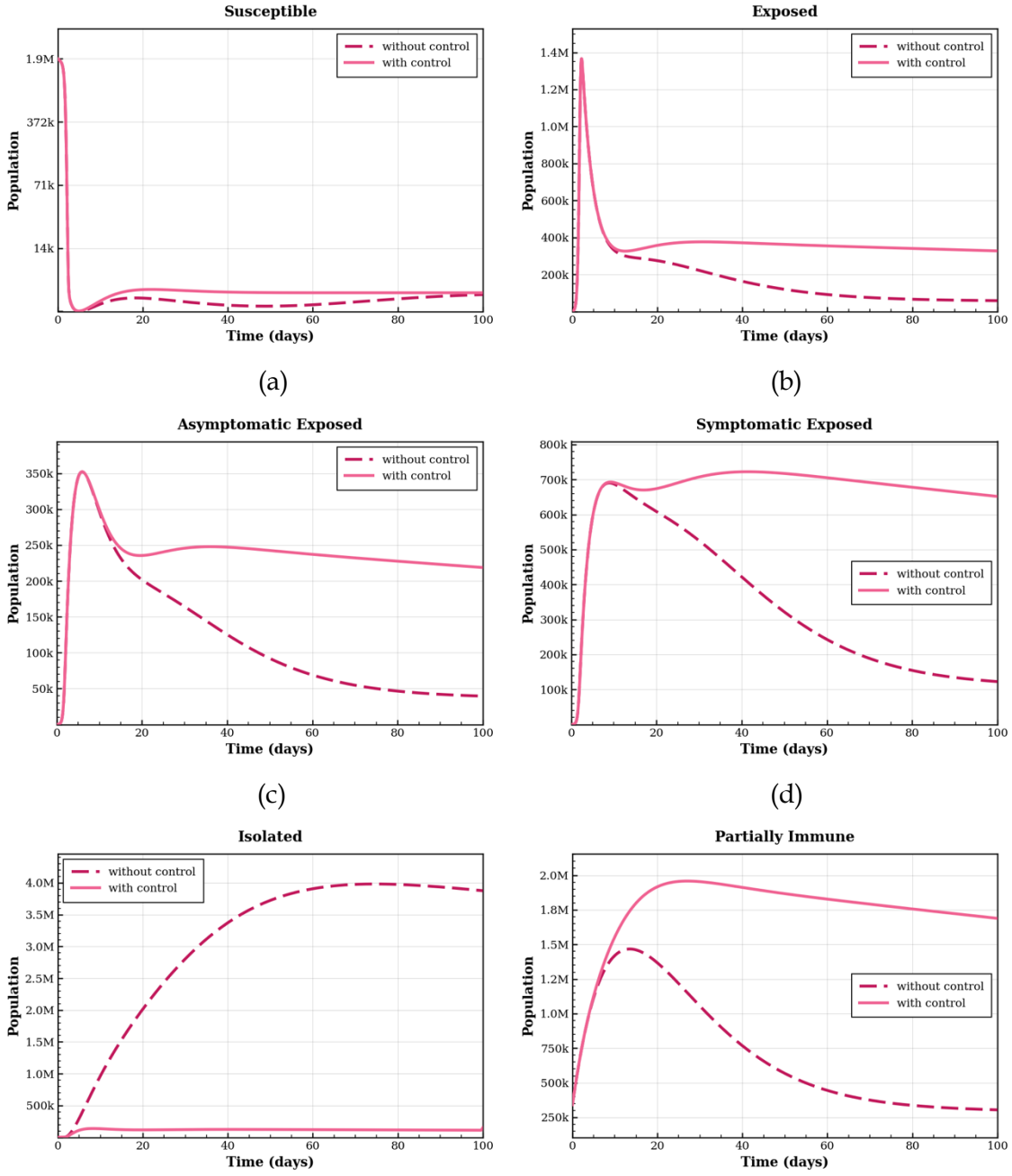


Figure 2. Scenario 1: Model with Education Campaign Control Variable (u_1).

Figure 2 (a)–(h) shows that the implementation of educational campaign controls affects the dynamics of diphtheria transmission over the 100-day simulation period. In Figure (a) shows a curve with higher control measures compared to no control measures, as the educational campaign reduces the risk of exposure, causing more individuals to remain in the susceptible compartment. Figures (b), (c), and (d) shows curves with lower control measures compared to no

control measures, indicating a reduction in new infections due to decreased effective contact. Figure (e) shows a curve with lower control compared to no control because of the reduction in cases requiring isolation. Figures (f) and (g) shows curves with higher control compared to no control, indicating increased protection in the population. Figure (h) shows that the educational campaign control reaches a maximum value throughout the simulation period, whereas the value without control is zero. This indicates that the educational campaign is optimally implemented to suppress the spread of diphtheria.



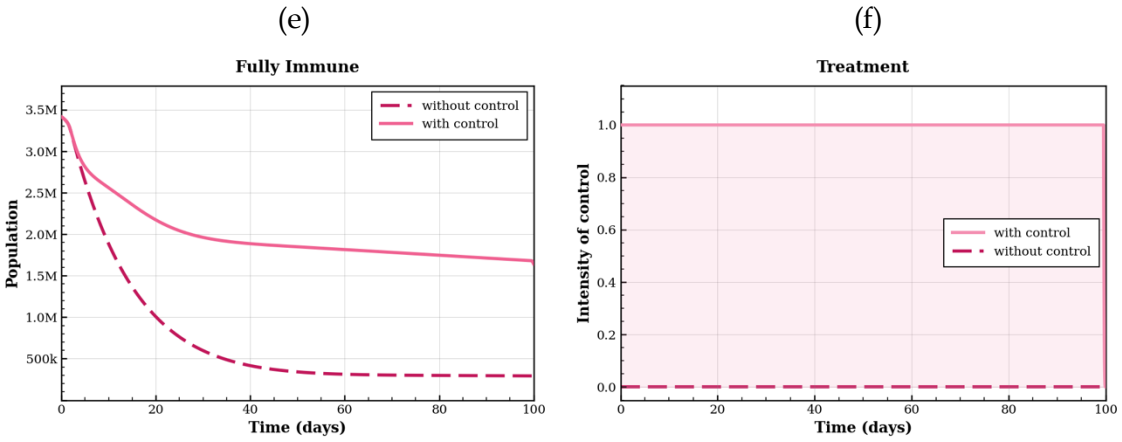
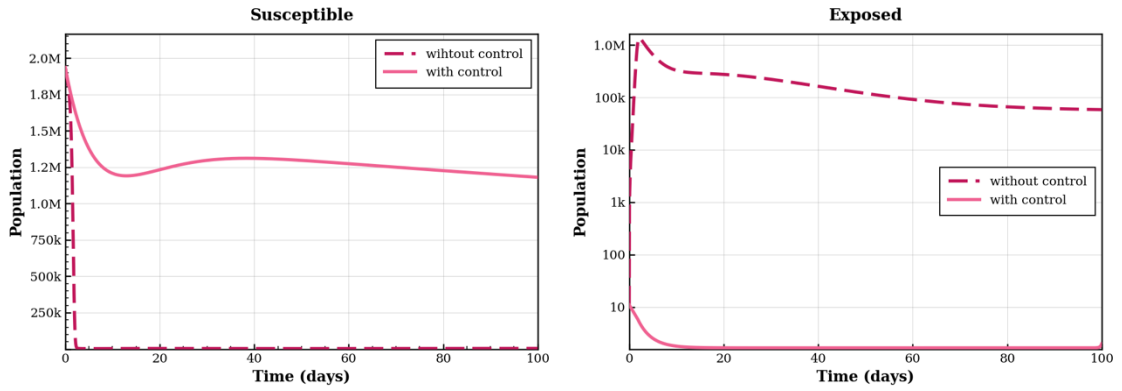


Figure 3. Scenario 2: Model with Treatment Control Variable (u_2).

Figure 3 (a) – (h) shows that the implementation of treatment controls affects the dynamics of diphtheria transmission over the 100-day simulation period. In Figures (a), (b), (c), and (d) shows that the curves with controls are higher than those without controls. This indicates that treatment increases the number of individuals remaining in the susceptible, exposed, and infectious compartments through the recovery process and by slowing the progression of the disease. Furthermore, Figure (e) shows that the curve with treatment is lower than that without treatment because treatment accelerates recovery, thereby reducing the need for isolation. Figures (f) and (g) shows that the curve with treatment is higher than that without treatment, indicating an increase in the number of individuals acquiring immunity due to successful treatment. Figure (h) shows that the curve with treatment control remains near its maximum value throughout the simulation, whereas the curve without control remains at zero. This indicates that treatment is optimally applied to accelerate recovery and reduce the impact of diphtheria spread within the population.



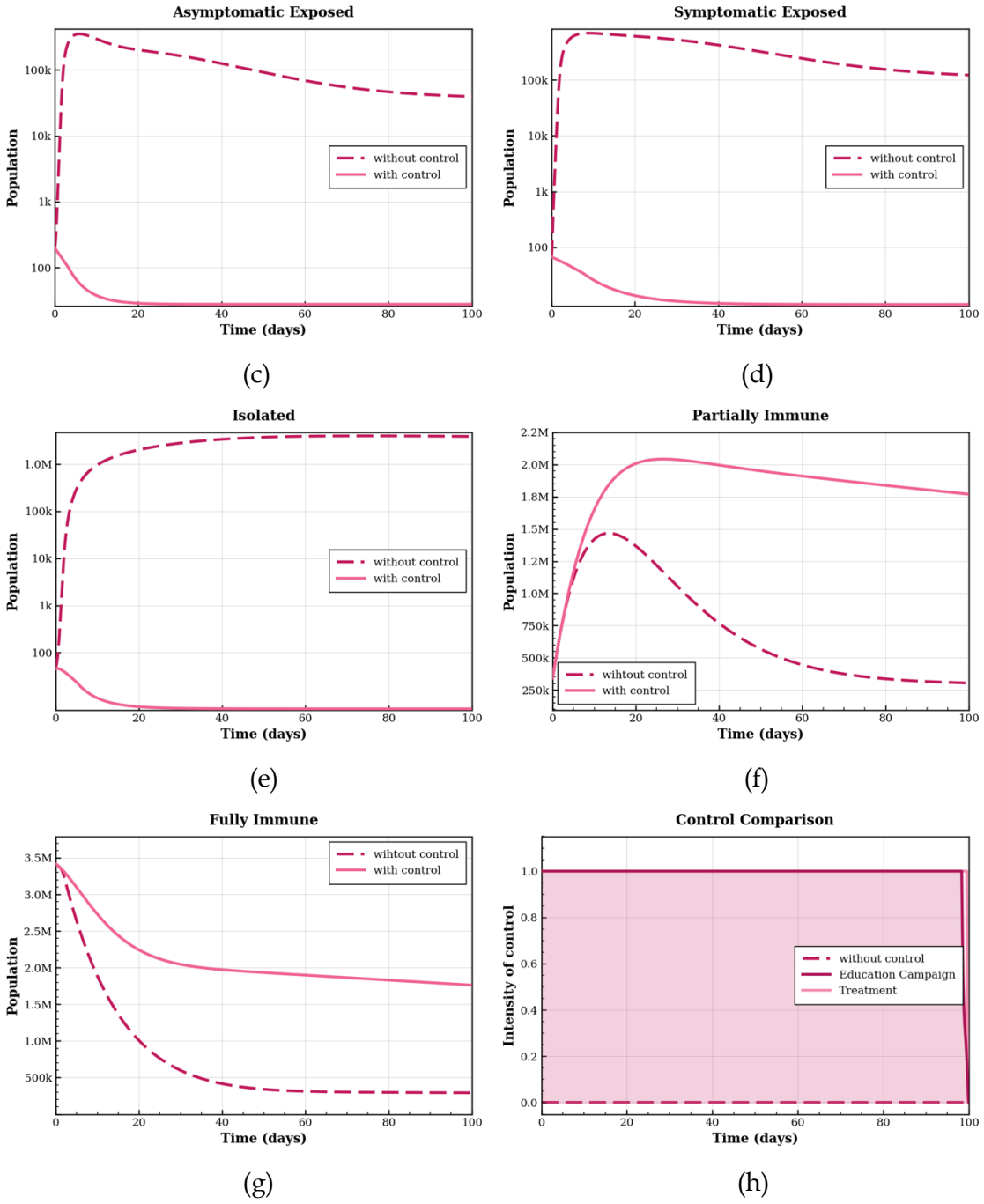


Figure 4. Scenario 3: Model with Two Control Variables (u_1) and (u_2).

Figure 4 (a) − (h) shows that the implementation of combined control measures including educational campaign and treatment affects the dynamics of diphtheria transmission over a 100-day simulation period. In Figure (a) shows a curve with higher control compared to no control because education campaign and treatment reduce the risk of infection, causing more individuals to remain in the susceptible compartment. Figures (b), (c), (d), and (e) show curves with lower

control compared to no control, indicating a decrease in the number of exposed, symptomatic, and isolated individuals due to reduced disease transmission. Furthermore, Figures (f) and (g) show curves with higher control levels compared to no control, indicating an increase in the number of individuals with partial and full immunity. Figure (h) shows that the education campaign and treatment controls were optimally applied throughout the simulation period to suppress the spread of diphtheria and enhance protection within the population.

7. Conclusion

Increases in the natural birth rate (α) and effective contact rate (β) contribute to an increase in the basic reproduction number (R_0) in the diphtheria disease spread model. Based on the results of sensitivity and stability analysis, it was found that when $\alpha = 30$ and $\beta = 0.00003$, the value of $R_0 = 0.9 < 1$ so that the disease-free equilibrium point is asymptotically stable and the disease cannot spread continuously in the population. Conversely, when increased to $\alpha = 35$ and $\beta = 0.000036$, the value of $R_0 = 1.1 > 1$ so that the disease-free equilibrium point becomes unstable and an asymptotically stable endemic equilibrium point emerges, indicating that the disease can persist in the population at a certain endemic level. Thus, the values $\alpha = 32$ and $\beta = 0.00003$ can be considered as thresholds that still satisfy the condition $R_0 < 1$. Numerical simulation results show that applying optimal control in the form of simultaneous education campaign and treatment is the most effective strategy in reducing the number of infected individuals and maintaining system stability. Therefore, as long as these key parameters are maintained below their thresholds and control strategies are applied consistently, the spread of diphtheria can be controlled and will not develop endemically in the population.

Conflicts of Interest: The authors declare that there are no conflicts of interest regarding the publication of this paper.

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