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Model of optimal control strategies for the transmission of diphtheria

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Abstract

This research introduced an extensive mathematical model to capture the dynamics of diphtheria transmission. The study examined the interaction of five control measures viz: routine diphtheria vaccination, often administered with tetanus and pertussis vaccines; interventions for symptomatic to isolated treatment transitions; collaborative efforts addressing asymptomatic to home quarantine transitions; surveillance measures for home quarantine to isolated treatment transitions; and vigilance to detect cases in individuals exposed to symptomatic cases. We established the epidemiological viability of the model by proving, among others, its positivity, equilibrium under endemic conditions, equilibrium in the absence of disease, global and local stability and boundedness. Also the sensitivity analysis of the model highlighted the importance of the important variables in influencing disease occurrence and spread. In addition, the control measures significantly impact virus transmission dynamics, and results from simulations demonstrated that combination of these control strategies effectively flattened the curve of diphtheria transmission. These findings provided healthcare professionals and policymakers with valuable insights into crucial measures for eradicating diphtheria from the population.

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1. Introduction

Diphtheria, caused by Corynebacterium diphtheriae (Figure 1), presents with symptoms such as sore throat, fever, and in severe cases, can lead to airway blockage, barking cough, and heart muscle damage due to toxin production. Complications include nerve inflammation, kidney problems, and bleeding due to low platelets, potentially resulting in abnormal heart rates and paralysis [1, 2]. After receiving medical care, the majority of individuals who experience complications from diphtheria manage to survive, although the recuperation process is frequently gradual. Roughly 5% to 10% of diphtheria cases result in fatality, with biggest mortality rates recorded among children below 5 years of age and adults over 40 years old [3]. Diphtheria is prevalent globally, especially in nations with insufficient vaccination rates, while it is uncommon in developed countries like the United States. As a result of its rarity, any previous patterns of seasonal and geographical distribution are no longer discernible [3]. An occurrence of diphtheria took place in October 2022 at the former Manston Airfield, situated in Kent, England. The airfield, once used by the Ministry of Defense (MoD), had been repurposed into a facility for processing asylum seekers. Although the processing center's intended capacity was 1,000 individuals, approximately 3,000 people were residing there, including some who were housed in tents. The Home Office, the government body overseeing asylum seekers, declined to verify the exact count of cases related to the outbreak [4].

To have deeper insight into the epidemiology of diphtheria, researchers employ a mathematical model to clarify the intricate patterns of disease transmission. This analytical approach, which is similarly employed for ailments such as pertussis and influenza, allows for a

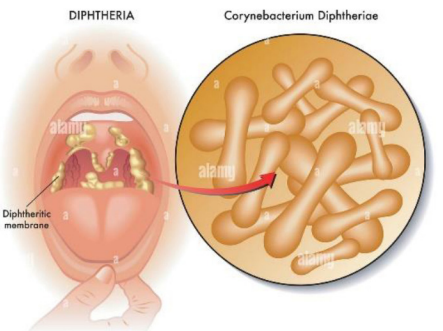

Figure 1

Diphtheria structure

systematic exploration of how diphtheria spreads among populations [5, 6, 7]. Unfortunately, a tailored transmission model for diphtheria is lacking. Existing models focus on diphtheria toxin dynamics and transmission, lacking a comprehensive portrayal of diphtheria spread [5, 9, 10].

In the same manner in which models were developed and used to describe transmission dynamics of diverse infection ranging from covid-19 [11, 12, 13] to others like lasser fever [14, 15, 16, 17], we introduce a comprehensive mathematical model for diphtheria transmission dynamics. Its goal is to predict diphtheria's spread and severity in humans. The main objective is to assess the combined effects of five control measures: 1. Routine diphtheria vaccination, often administered with tetanus and pertussis vaccines. 2. Efforts on symptomatic to isolated treatment transitions. 3. Collaborative efforts on asymptomatic to home-quarantine transitions. 4. Collaborative efforts to survey and promptly detect home quarantine to isolated treatment transitions. 5. Collaborative efforts to survey and promptly detect cases of individuals exposed to symptomatic cases.

2. Method

2.1 *Model description of Diphtheria*

The transmission dynamics of Diphtheria among humans are modeled using a SEIR framework with seven compartments. The total populace, denoted as N, is stratified into seven distinct cohorts: individuals that are susceptible (S), individuals that are exposed (E), individuals that are symptomatic (I_S), asymptomatic individuals (I_A), individuals that are isolated (I), asymptotic individuals that are home-quarantined (Q), and individuals that are recovered (R). In this way, the population is represented as $N = S + E + I_S + I_A + Q + I + R$. It is essential to highlight the different categories of individuals in this model of diphtheria transmission. Susceptible individuals (S) are those who have not been previously infected with diphtheria and have not received vaccination against the disease. Exposed individuals (E) have come into contact with the diphtheria-causing bacteria, but do not display any symptoms yet. Asymptomatic individuals (I_A) refer to individuals who may have been infected with the diphtheria-causing bacteria, Corynebacterium diphtheriae, but do not show any symptoms of the disease. Symptomatic individuals (I_S) are those who are infected with the diphtheria-causing bacteria and exhibit observable symptoms of the disease. Home-

quarantined asymptomatic individuals (Q) are individuals exposed to the diphtheria-causing bacteria but have not yet developed symptoms, and they are required to stay at home to prevent potential transmission to others. Isolated symptomatic individuals (I) are individuals with symptoms of diphtheria who have been placed in isolation to prevent the spread of the bacteria to others. Lastly, recovered individuals are those who have successfully overcome the diphtheria infection and returned to good health. They have developed immunity to diphtheria following their recovery, so we assume they can no longer be infected by the diphtheria-causing bacteria. This model allows us to understand the dynamics of diphtheria transmission and the roles that different groups of individuals play in its spread and containment.

In this model, β represents the recruitment of individuals into the susceptible population (S), while μ represents the natural mortality rate that affects all sub-populations. It is possible for both symptomatic and asymptomatic people to spread diphtheria.

As a result, the force of infection given as $\lambda\left(\frac{\gamma I_a + I_s}{N}\right)$ is defined as the transmission rate, with a modification parameter $0 < \gamma \leq 1$ accounting for the assumed reduction in transmissibility of asymptomatic individuals compared to the symptomatic population.

The exposed individuals move to symptomatic individuals at the rate of $a\phi_2$ or move to the asymptomatic individuals at the rate of $(1-a)\phi_1$, where $0 \leq a \leq 1$ is the percentage or fraction of individuals that are symptomatic, and δ is the diphtheria mortality rate. Also, asymptomatic individuals exposed to diphtheria-causing bacteria but showing no clinical symptoms are moved to the home-quarantined individuals at the rate ω_1 while symptomatic individuals move to isolation individuals for treatment at the rate of ω_2. At the home-quarantine individuals whose case was confirmed positive moved to isolated individuals at $(1-b)\sigma_Q$ while the remaining individuals move to recovered individuals at the rate $b\sigma_2$ where $0 \leq b \leq 1$ the proportion of isolated individuals is. Finally, after the treatment of individuals in isolated center they are moved to recover at the rate σ_I and we assume that they can no longer be infected. Figure 2 shows schematic diagram interaction of each compartment and we assume that they can no longer be infected.

2.1.1. The equations of the model

Based on the description above, the form of the system of equation is given as:

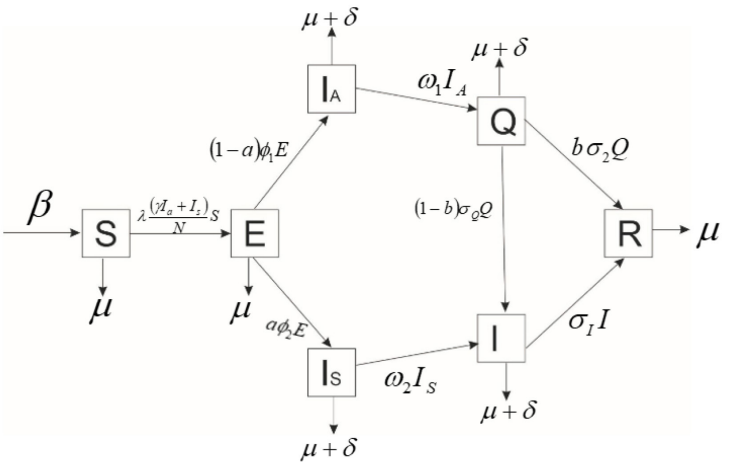

Figure 2

Schematic diagram interaction of each compartment

$$
\begin{aligned}
\frac{dS}{dt} &= \beta - \lambda\frac{(\gamma I_a + I_s)}{N}S - \mu S \\
\frac{dE}{dt} &= \lambda\frac{(\gamma I_a + I_s)}{N}S - (1-a)\phi_1 E - (a\phi_2 + \mu)E \\
\frac{dI_A}{dt} &= (1-a)\phi_1 E - (\omega_1 + \mu + \delta)I_A \\
\frac{dI_S}{dt} &= a\phi_2 E - (\omega_2 + \mu + \delta)I_S \\
\frac{dQ}{dt} &= \omega_1 I_A - (1-b)\sigma_Q Q - (b\sigma_2 + \mu + \delta)Q \\
\frac{dI}{dt} &= \omega_2 I_S + (1-b)\sigma_Q Q - (\sigma_1 + \mu + \delta)I \\
\frac{dR}{dt} &= b\sigma_2 Q + \sigma_I I - \mu R
\end{aligned}
\tag{1}
$$

2.2 *Quantitative Analysis of the Diphtheria Model*

2.2.1 Non-negativity and boundedness of Solution

The differential equation for the population $N = S + E + I_A + I_S + Q + I + R$ is as follows:

$$
\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI_A}{dt} + \frac{dI_S}{dt} + \frac{dQ}{dt} + \frac{dI}{dt} + \frac{dR}{dt} \tag{2}
$$

Substituting the model system of equation (1) into equation (2) and eliminating correctly, we have

$$\frac{dN}{dt} = \beta - \mu N + \delta(I_A + I_S + Q + I + R) \tag{3}$$

Theorem 1: *Let* $(S, E, I_A, I_S, Q_h, I, R)$ *be the solution of equation (1) with the initial conditions in a biologically feasible region* Γ *with:* $\Gamma = (S,\ E,\ I_A,\ I_S,\ Q,\ I,\ R) \in R_+^7 : N_h \leq \frac{\beta}{\mu}$

Then Γ is non-negative invariant [8, 15, 17, 18].

So, Where $\delta = 0$ at DFE, equation (3) becomes

$$\frac{dN}{dt} = \beta - \mu N \tag{4}$$

Solving equation (4) using integrating factor method, we have

$$\therefore \quad \lim_{t \to \infty} N(t) \leq \frac{\beta}{\mu} \tag{5}$$

Here, we verified the non-negativity of the solution which ensures that the diphtheria model's predictions are physically and epidemiologically plausible, and that it does not generate unrealistic scenarios where the number of individuals in a particular compartment becomes negative and also the boundedness of the solution was verified, which shows the diphtheria model do not grow unbounded, but rather, their values remain within a defined range or limit. In the diphtheria transmission, the number of individuals in each compartment does not grow indefinitely but remains within realistic bounds [19, 20, 21]. And also the boundedness ensures that the model's predictions do not lead to unrealistic scenarios where the disease spreads uncontrollably and reaches unattainable levels.

2.2.2 Diphtheria-free steady-state

Here, we check the behavior of the mathematical model where the disease is not present, and the population is in a state where no new diphtheria cases are occurring.

It should be noted that even in the absence disease every individual in the population is vulnerable (i.e. Susceptible).

So, then, $S^0 \neq 0$,

$$\text{For } S_h^0 \neq 0, E^0 = 0, I_A^0 = 0, I_S^0 = 0, Q^0 = 0, I^0 = 0, R^0 = 0,$$

Thus, the diphtheria model's system of equations (1)

$$\beta - \mu S^0 = 0$$

And this gives

$$S^o = \frac{\beta}{\mu} \tag{6}$$

This gives us the Diphtheria-free steady-state for the individuals as,

$$E^0 = (S^0, E^0, I_A^0, I_S^0, Q^0, I, R^0) = \left(\frac{\beta}{\mu}, 0,0,0,0,0,0\right) \tag{7}$$

2.2.3 Basic reproductive ratio

Here, we find the key epidemiological parameter we can used to describe the potential of diphtheria disease to spread within the population. Furthermore, it is imperative to recognize that this metric delineates the mean count of subsequent infections propagated by a solitary infectious individual within a populace entirely susceptible to the ailment [15, 21]. This parameter serves as a pivotal gauge for comprehending the intricacies of diphtheria transmission dynamics and assessing the efficacy of containment strategies.

The foundational reproduction number of the Diphtheria model's system equation (1) is derived employing the next generation matrix technique as introduced by Diekmann and Heesterbeek. [7, 8, 22, 23, 24].

Using

$$R_n = \rho(FV^{-1}) \tag{8}$$

The terminology denoting new infections and transitions in system (1) are expressed as follows;

$$F = \begin{pmatrix} \lambda \frac{(\gamma I_a + I_s)}{N} S \\ 0 \\ 0 \end{pmatrix} \tag{9}$$

And

$$V = \begin{pmatrix} (1-a)\phi_1 E + (a\phi_2 + \mu)E \\ -(1-a)\phi_1 E + (\omega_1 + \mu + \delta)I_A \\ -a\phi_2 E + (\omega_2 + \mu + \delta)I_S \end{pmatrix} \tag{10}$$

Let $E = f_1, I_A = f_2, I_s = f_3$

$$F_* = \begin{bmatrix} \frac{df_1}{dE} & \frac{df_1}{dI_A} & \frac{df_1}{dI_S} \\ \frac{df_2}{dE} & \frac{df_2}{dI_A} & \frac{df_2}{dI_S} \\ \frac{df_3}{dE} & \frac{df_3}{dI_A} & \frac{df_3}{dI_S} \end{bmatrix} \tag{11}$$

Solving equation (11), we have

$$F_* = \begin{bmatrix} 0 & \lambda\gamma & \lambda \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \tag{12}$$

Let $E^* = v_1, I_A = v_2, I_s = v_3,$ then we have

$$V = \begin{bmatrix} \frac{dv_1}{dE^*} & \frac{dv_1}{dI_A} & \frac{dv_1}{dI_s} \\ \frac{dv_2}{dE^*} & \frac{dv_2}{dI_A} & \frac{dv_2}{dI_s} \\ \frac{dv_3}{dE^*} & \frac{dv_3}{dI_A} & \frac{dv_3}{dI_s} \end{bmatrix} \tag{13}$$

Then,

$$V = \begin{bmatrix} ((1-a)\phi_1 + a\phi_2 + \mu) & 0 & 0 \\ -(1-a)\phi_1 & (\omega_1 + \mu + \delta) & 0 \\ -a\phi_2 & 0 & (\omega_2 + \mu + \delta) \end{bmatrix} \tag{14}$$

Now, we try to find the $V^{-1} = \frac{1}{|V|} adjV$

$$|V| = ((1-a)\phi_1 + a\phi_2 + \mu)(\omega_1 + \mu + \delta)(\omega_2 + \mu + \delta) \tag{15}$$

And by cofactor method, we have

$$adjV = \begin{bmatrix} (\omega_1 + \mu + \delta)(\omega_2 + \mu + \delta) & 0 & 0 \\ (1-a)\phi_1(\omega_2 + \mu + \delta) & ((1-a)\phi_1 + a\phi_2 + \mu)(\omega_2 + \mu + \delta) & 0 \\ (\omega_1 + \mu + \delta)a\phi_2 & 0 & ((1-a)\phi_1 + a\phi_2 + \mu)(\omega_1 + \mu + \delta) \end{bmatrix} \tag{16}$$

Then,

$$V^{-1} = \frac{1}{((1-a)\phi_1 + a\phi_2 + \mu)(\omega_1 + \mu + \delta)(\omega_2 + \mu + \delta)}$$

$$\begin{bmatrix} (\omega_1 + \mu + \delta)(\omega_2 + \mu + \delta) & 0 & 0 \\ (1-a)\phi_1(\omega_2 + \mu + \delta) & ((1-a)\phi_1 + a\phi_2 + \mu)(\omega_2 + \mu + \delta) & 0 \\ (\omega_1 + \mu + \delta)a\varphi_2 & 0 & ((1-a)\phi_1 + a\phi_2 + \mu)(\omega_1 + \mu + \delta) \end{bmatrix} \tag{17}$$

Therefore, substitute (17) and (12) in $R_n = \rho(FV^{-1})$, so we have

$$R_n = \frac{1}{((1-a)\phi_1 + a\phi_2 + \mu)(\omega_1 + \mu + \delta)(\omega_2 + \mu + \delta)} \begin{bmatrix} 0 & \lambda\gamma & \lambda \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$
$$\begin{bmatrix} (\omega_1 + \mu + \delta)(\omega_2 + \mu + \delta) & 0 & 0 \\ (1-a)\phi_1(\omega_2 + \mu + \delta) & ((1-a)\phi_1 + a\phi_2 + \mu)(\omega_2 + \mu + \delta) & 0 \\ (\omega_1 + \mu + \delta)a\phi_2 & 0 & ((1-a)\phi_1 + a\phi_2 + \mu)(\omega_1 + \mu + \delta) \end{bmatrix} \tag{18}$$

$$R_n = \frac{(1-a)\phi_1(\omega_2+\mu+\delta)\lambda\gamma}{((1-a)\phi_1+a\phi_2+\mu)(\omega_1+\mu+\delta)(\omega_2+\mu+\delta)} + \frac{a\phi_2(\omega_1+\mu+\delta)\lambda}{((1-a)\phi_1+a\phi_2+\mu)(\omega_1+\mu+\delta)(\omega_2+\mu+\delta)}$$

The fundamental reproductive rate corresponds to the spectral radius of the matrix representing the transmission dynamics of a given pathogen. Subsequently, we denote this as

$$R_n = \frac{1}{((1-a)\phi_1+a\phi_2+\mu)(\omega_1+\mu+\delta)(\omega_2+\mu+\delta)} [(1-a)\phi_1\lambda\gamma(\omega_2+\mu+\delta) + a\phi_2\lambda(\omega_1+\mu+\delta)] \tag{19}$$

2.3 *Stability Property*

2.3.1 Local Stability of the Diphtheria-free steady-state

In this analysis, we investigate the regional resilience of the stable state devoid of diphtheria. This property pertains to how the model's variables evolve over time when there is no presence of diphtheria in the system, referring to the disease-free equilibrium point [7, 8]. It allows us to understand the behavior of the model in the absence of diphtheria and how it responds to small changes or perturbations.

Theorem 1: *In the context of the diphtheria model system (1), the state of disease-free equilibrium is deemed locally asymptotically stable (LAS) provided that all eigenvalues of the Jacobian matrix associated with the system exhibit negative real components.*

Proof: In order to elucidate the aforementioned theorem, we undertake the computation of the Jacobian matrix [8, 22, 25] pertaining to the system's dynamics at the state of disease-free equilibrium (DFE). The Jacobian

matrix, denoted by $J(S,E,I_A,I_S,Q,I,R)$, allows us to calculate and estimate the eigenvalues of the system. The Jacobian matrix is given as follows:

$$J=\begin{bmatrix} -\mu & 0 & -\lambda\gamma & -\lambda & 0 & 0 & 0 \\ 0 & -(1-a)\phi_1-(a\phi_2+\mu) & \lambda\gamma & \lambda & 0 & 0 & 0 \\ 0 & (1-a)\phi_1 & -(\omega_1+\mu+\delta) & 0 & 0 & 0 & 0 \\ 0 & a\phi_2 & 0 & -(\omega_2+\mu+\delta) & 0 & 0 & 0 \\ 0 & 0 & \omega_1 & 0 & -(1-b)\sigma_Q-(b\sigma_2+\mu+\delta) & 0 & 0 \\ 0 & 0 & 0 & \omega_2 & (1-b)\sigma_Q & -(\sigma_1+\mu+\delta) & 0 \\ 0 & 0 & 0 & 0 & b\sigma_2 & \sigma_1 & -\mu \end{bmatrix} \tag{20}$$

Now, we compute the eigenvalue, $|J-\lambda * I|=0$, where $\lambda *$ represent our eigenvalue,

$$|J-\lambda I|=\begin{vmatrix} -\mu-\lambda * & 0 & -\lambda\gamma & -\lambda & 0 & 0 & 0 \\ 0 & -(1-a)\phi_1-(a\phi_2+\mu)-\lambda * & \lambda\gamma & \lambda & 0 & 0 & 0 \\ 0 & (1-a)\phi_1 & -(\omega_1+\mu+\delta)-\lambda * & 0 & 0 & 0 & 0 \\ 0 & a\phi_2 & 0 & -(\omega_2+\mu+\delta)-\lambda * & 0 & 0 & 0 \\ 0 & 0 & \omega_1 & 0 & -(1-b)\sigma_Q-(b\sigma_2+\mu+\delta)-\lambda * & 0 & 0 \\ 0 & 0 & 0 & \omega_2 & (1-b)\sigma_Q & -(\sigma_1+\mu+\delta)-\lambda * & 0 \\ 0 & 0 & 0 & 0 & b\sigma_2 & \sigma_1 & -\mu-\lambda * \end{vmatrix}=0 \tag{21}$$

In the matrix shown above, represented by equation (21), the only entry in the first column that is not zero is $-\mu-\lambda *$. Therefore, $\lambda *_1=-\mu$, then, we proceed by eliminating the row and column that intersect with this particular element. As a result, a modified matrix is obtained as follow:

$$\begin{vmatrix} -(1-a)\phi_1-(a\phi_2+\mu)-\lambda * & \lambda\gamma & \lambda & 0 & 0 & 0 \\ (1-a)\phi_1 & -(\omega_1+\mu+\delta)-\lambda * & 0 & 0 & 0 & 0 \\ a\phi_2 & 0 & -(\omega_2+\mu+\delta)-\lambda * & 0 & 0 & 0 \\ 0 & \omega_1 & 0 & -(1-b)\sigma_Q-(b\sigma_2+\mu+\delta)-\lambda * & 0 & 0 \\ 0 & 0 & \omega_2 & (1-b)\sigma_Q & -(\sigma_1+\mu+\delta)-\lambda * & 0 \\ 0 & 0 & 0 & b\sigma_2 & \sigma_1 & -\mu-\lambda * \end{vmatrix}=0 \tag{22}$$

Also, in the above matrix (22), $-\mu-\lambda *$ is the only entry in the last column that is not zero; hence $\lambda *_2=-\mu$, then, we eliminate the row and column that intersect with this particular element. As a result we have a new matrix as:

$$\begin{vmatrix} -(1-a)\phi_1-(a\phi_2+\mu)-\lambda * & \lambda\gamma & \lambda & 0 & 0 \\ (1-a)\phi_1 & -(\omega_1+\mu+\delta)-\lambda * & 0 & 0 & 0 \\ a\phi_2 & 0 & -(\omega_2+\mu+\delta)-\lambda * & 0 & 0 \\ 0 & \omega_1 & 0 & -(1-b)\sigma_Q-(b\sigma_2+\mu+\delta)-\lambda * & 0 \\ 0 & 0 & \omega_2 & (1-b)\sigma_Q & -(\sigma_1+\mu+\delta)-\lambda * \end{vmatrix}=0 \tag{23}$$

Also, in the above matrix, we have $-(\sigma_1+\mu+\delta)-\lambda^*$ as the only non-zero entry in the last column; hence $\lambda^*_3=-(\sigma_1+\mu+\delta)$, then, we also eliminate the row and column that intersect with this particular element. So, we have a new matrix as

$$\begin{vmatrix} -(1-a)\phi_1-(a\phi_2+\mu)-\lambda^* & \lambda\gamma & \lambda & 0 \\ (1-a)\phi_1 & -(\omega_1+\mu+\delta)-\lambda^* & 0 & 0 \\ a\phi_2 & 0 & -(\omega_2+\mu+\delta)-\lambda^* & 0 \\ 0 & \omega_1 & 0 & -()1-b\sigma_Q-(b\sigma_2+\mu+\delta)-\lambda^* \end{vmatrix}=0$$

(24)

Also, in the above matrix in equation (24), $-(1-b)\,\sigma_Q-(b\sigma_2+\mu+\delta)-\lambda^*$ is the only non-zero entry (diagonal term) in the last column; hence $\lambda^*_4=-[(1-b)\sigma_Q+(b\sigma_2+\mu+\delta)]$, then, we similarly eliminate the row and column that intersect with this particular element. So, the new matrix is

$$\begin{vmatrix} -(1-a)\phi_1-(a\phi_2+\mu)-\lambda & \lambda\gamma & \lambda \\ (1-a)\phi_1 & -(\omega_1+\mu+\delta)-\lambda & 0 \\ a\phi_2 & 0 & -(\omega_2+\mu+\delta)-\lambda \end{vmatrix}=0 \qquad (25)$$

We divide the matrix presented in equation (25) into segments and transform the Jacobian matrix into echelon form [26]. Subsequently, we determine the eigenvalue and identify the remaining eigenvalues through this process. This leads us to the following results:

$$\begin{bmatrix} -(1-a)\phi_1-(a\phi_2+\mu)-\lambda^* & \lambda\gamma & \lambda \\ 0 & -[(1-a)\phi_1+(a\phi_2+\mu)](\omega_1+\mu+\delta)+(1-a)\phi_1\lambda\gamma & (1-a)\phi_1\lambda \\ 0 & a\phi_2\lambda\gamma & -[(1-a)\phi_1+(a\phi_2+\mu)](\omega_2+\mu+\delta)+a\phi_2\lambda \end{bmatrix}-\begin{bmatrix} \lambda^* & 0 & 0 \\ 0 & \lambda^* & 0 \\ 0 & 0 & \lambda^* \end{bmatrix}$$

$$\begin{vmatrix} -(1-a)\phi_1-(a\phi_2+\mu)-\lambda^* & \lambda\gamma & \lambda \\ 0 & -[(1-a)\phi_1+(a\phi_2+\mu)](\omega_1+\mu+\delta)+(1-a)\phi_1\lambda\gamma-\lambda^* & (1-a)\phi_1\lambda \\ 0 & 0 & Z-\lambda^* \end{vmatrix}=0$$

$$Z=\{-[(1-a)\phi_1+(a\phi_2+\mu)](\omega_1+\mu+\delta)+(1-a)\phi_1\lambda\gamma\}\{-[(1-a)\phi_1+(a\phi_2+\mu)](\omega_2+\mu+\delta)+a\phi_2\lambda\}-a\phi_2\lambda\gamma(1-a)\phi_1\lambda$$

(26)

Then we have the remaining following eigenvalues to be

$$\lambda^*_5=-[(1-a)\phi_1-(a\phi_2+\mu)]$$

$$\lambda^*_6=-\{[(1-a)\phi_1+(a\phi_2+\mu)](\omega_1+\mu+\delta)-(1-a)\phi_1\lambda\gamma\} \qquad (27)$$

$$\lambda^*_7 = Z$$

Where

$$\begin{aligned} Z = \{&-[(1-a)+(a\phi_2+\mu)](\omega_1+\mu+\delta)+(1-a)\phi_1\lambda\gamma\} \\ &\{-[(1-a)\phi_1+(a\phi_2+\mu)](\omega_2+\mu+\delta)+a\phi_2\lambda\} \\ &-a\phi_2\lambda\gamma(1-a)\phi_1\lambda \end{aligned}$$

Evidently, the eigenvalues are discernibly real and not involving complex components. Their respective signs play a pivotal role in establishing the stability of the DFE. In this particular circumstance, it is observed that all eigenvalues manifest negative real components, thereby confirming the local asymptotic stability of diphtheria at the Disease-Free Equilibrium (DFE).

Theorem 2: *In the context of the diphtheria model system, the disease-free equilibrium point (1) manifests local asymptotic stability (LAS) under conditions where the basic reproductive ratio* $R_n < 1.$

Proof: From $\lambda^*_7 = Z$

Where

$$\begin{aligned} Z = \{&-[(1-a)+(a\phi_2+\mu)](\omega_1+\mu+\delta)+(1-a)\phi_1\lambda\gamma\} \\ &\{-[(1-a)\phi_1+(a\phi_2+\mu)](\omega_2+\mu+\delta)+a\phi_2\lambda\} \\ &-a\phi_2\lambda\gamma(1-a)\phi_1\lambda \end{aligned}$$

Lemma 1: *The diphtheria disease-free equilibrium of the human population achieves asymptotic stability under the condition that* $R_n < 1.$

Through the process of simplification, we arrive at the expression for (Z) as follows:

$$\begin{aligned} Z = \{&-[(1-a)+(a\phi_2+\mu)](\omega_1+\mu+\delta)+(1-a)\phi_1\lambda\gamma\}\{-[(1-a)\phi_1+(a\phi_2+\mu)] \\ &(\omega_2+\mu+\delta)+a\phi_2\lambda\}-a\phi_2\lambda\gamma(1-a)\phi_1\lambda \\ &=[(1-a)+(a\phi_2+\mu)](\omega_1+\mu+\delta)[(1-a)\phi_1+(a\phi_2+\mu)](\omega_2+\mu+\delta) \\ &-[(1-a)+(a\phi_2+\mu)](\omega_1+\mu+\delta)a\phi_2\lambda-(1-a)\phi_1\lambda\gamma[(1-a)\phi_1+(a\phi_2+\mu)] \\ &(\omega_2+\mu+\delta)+(1-a)\phi_1\lambda\gamma a\phi_2\lambda-a\phi_2\lambda\gamma(1-a)\phi_1\lambda \end{aligned}$$

$$
\begin{aligned}
Z &= [(1-a)+(a\phi_2+\mu)](\omega_1+\mu+\delta)[(1-a)\phi_1+(a\phi_2+\mu)](\omega_2+\mu+\delta) \\
&\quad -[(1-a)+(a\phi_2+\mu)](\omega_1+\mu+\delta)a\phi_2\lambda \\
&\quad -(1-a)\phi_1\lambda\gamma[(1-a)\phi_1+(a\phi_2+\mu)](\omega_2+\mu+\delta) \\
&= [(1-a)+(a\phi_2+\mu)]\begin{bmatrix}[(1-a)\phi_1+(a\phi_2+\mu)](\omega_1+\mu+\delta)(\omega_2+\mu+\delta) \\ -(1-a)\phi_1\lambda\gamma(\omega_2+\mu+\delta)+(\omega_1+\mu+\delta)a\phi_2\lambda\end{bmatrix}
\end{aligned}
$$

$$
\lambda^*_7\,[(1-a)+(a\phi_2+\mu)]^2(\omega_1+\mu+\delta)(\omega_2+\mu+\delta)\left[1-\frac{(1-a)\phi_1\lambda\gamma(\omega_2+\mu+\delta)+(\omega_1+\mu+\delta)a\phi_2\lambda}{[(1-a)\phi_1+(a\phi_2+\mu)](\omega_1+\mu+\delta)(\omega_2+\mu+\delta)}\right] \quad (28)
$$

$$
R_n = \frac{1}{((1-a)\phi_1+a\phi_2+\mu)(\omega_1+\mu+\delta)(\omega_2+\mu+\delta)}\begin{bmatrix}(1-a)\phi_1\lambda\gamma(\omega_2+\mu+\delta) \\ +a\phi_2\lambda(\omega_1+\mu+\delta)\end{bmatrix} \text{ in equation (29)}
$$

We have

$$
\begin{aligned}
\lambda^*_7 &= [(1-a)+(a\phi_2+\mu)]^2(\omega_1+\mu+\delta)(\omega_2+\mu+\delta) \\
&\quad \left[1-\frac{(1-a)\phi_1\lambda\gamma(\omega_2+\mu+\delta)+(\omega_1+\mu+\delta)a\phi_2\lambda}{[(1-a)\phi_1+(a\phi_2+\mu)](\omega_1+\mu+\delta)(\omega_2+\mu+\delta)}\right] \\
&\le [(1-a)+(a\phi_2+\mu)]^2(\omega_1+\mu+\delta)(\omega_2+\mu+\delta)[1-R_n] \qquad (29)
\end{aligned}
$$

The inequality (29) stated above maintains its negative (stable) status if and only if the conditions $R_n < 1$. This concludes the demonstration of Lemma 1.

In this scenario, the regional asymptotic stability of the diphtheria disease-free equilibrium (DFE) is affirmed owing to the basic reproductive ratio being less than unity. This indicates that, on average, each infected individual infects less than one susceptible individual. As a result, the diphtheria disease exhibits an inability to sustain itself within the population, resulting in a progressive decline in the number of infected individuals until eventual eradication.

The local asymptotic stability confirms that if the system begins in proximity to the disease-free equilibrium point, it will gradually approach and converge to this equilibrium point over time. This substantiates the validity of Theorem 2.

2.4 *Global Stability of the Diphtheria-free steady-state*

Theorem 3: *When the basic reproduction number is* $R_n < 1$, *the diphtheria-free equilibrium of the model system (1) demonstrates global asymptotic stability (GAS). In other words, when* $R_n < 1$, *the diphtheria-free steady-state will attract*

all nearby trajectories of the system, indicating that the disease will be eradicated and not persist in the population.

In order to conduct a comprehensive assessment of the disease-free equilibrium's global stability, we will employ the methodology outlined in previous studies [8, 27]. This approach facilitates an examination of the stability characteristics of the disease-free state across a global context, offering valuable perspectives on the enduring dynamics of the system and its capacity for diphtheria eradication.

$$J(S,E,I_A,I_S,Q,I,R)$$

Let the respective class of un-infected and infected populations be represented by $O=(S,R)$ and $P=(E,I_A,I_S,Q,I)$

Point $E^0=(X^0,0)$ is globally asymptotically stable if $R_n<1$ and also if the two conditions (i) and (ii) below hold:

i. Given $\frac{dU}{dt}=H(U,0)$, E_0 is globally asymptotically stable
ii. There is $G(U,V)=XY-G^0(U,V)$ such that $G^0(U,V)\geq 0\forall(U,V)\in R_+^7$

Proof: **1**st Condition

$$\frac{dU}{dt}=\frac{dU}{dt}\Big|_{N=0}=H(U,0)$$

$$H(U,0)=\begin{pmatrix}\beta-\mu S\\ \mu\end{pmatrix} \tag{30}$$

Clearly, $E_0=\left(\frac{\beta}{\mu},0,0,0,0,0,0\right)$ is globally asymptotically stable for $\frac{dU}{dt}=H(U,0)$, by the following proof

$$\frac{dS}{dt}=\beta-\mu S$$

Using the method of integrating factor, we have

$$IF=\ell^{\int \mu dt}$$

$$\left(\frac{dS}{dt}+\mu S\right)\ell^{\mu t}=\beta\ell^{\mu t}$$

$$\frac{d(S\ell^{\mu t})}{dt}=\beta\ell^{\mu t}$$

Thus, $$S=\frac{\beta}{\mu}+\frac{1}{\ell^{\mu t}}\int \ell^{\mu t}dt \tag{31}$$

Accordingly, as $t\to\infty$, $S(t)\to\frac{\beta}{\mu}$ for the model, showing the Global convergence of $H(U,0)$ in R_+^7.

2nd Condition

$$G(U,V)=\begin{cases}\lambda\frac{(\gamma I_a+I_s)}{N}S-(1-a)\phi_1E-(a\phi_2+\mu)E\\(1-a)\phi_1E-(\omega_1+\mu+\delta)I_A\\a\phi_2E-(\omega_2+\mu+\delta)I_S\\\omega_1I_A-(1-b)\sigma_QQ-(b\sigma_2+\mu+\delta)Q\\\omega_2I_S+(1-b)\sigma_QQ-(\sigma_1+\mu+\delta)I\end{cases} \tag{32}$$

$G(U,V)$ can be written as:

$$G(U,V)=\begin{bmatrix}\lambda\frac{(\gamma I_a+I_s)}{N}S\\0\\0\\0\\0\end{bmatrix}$$

$$-\begin{bmatrix}(1-a)\phi_1+(a\phi_2+\mu) & 0 & 0 & 0 & 0\\(1-a)\phi_1 & (\omega_1+\mu+\delta) & 0 & 0 & 0\\a\phi_2 & 0 & (\omega_2+\mu+\delta) & 0 & 0\\0 & \omega_1 & 0 & (1-b)\sigma_Q-(b\sigma_2+\mu+\delta) & 0\\0 & 0 & \omega_2 & -(1-b)\sigma_QQ & (\sigma_1+\mu+\delta)\end{bmatrix}\begin{bmatrix}E\\I_A\\I_S\\Q\\I\end{bmatrix} \tag{33}$$

Thus, we must establish that:

$$G(U,V)=AB-G^0(U,V)$$

In equation (35) above, matrix B is given by $B=[E\quad I_A\quad I_S\quad Q\quad I]$ while $G^0(U,V)$ is the steady-state matrix which determines the 2nd condition of the system's global stability.

$$\therefore A=-\begin{bmatrix}(1-a)\phi_1+(a\phi_2+\mu) & 0 & 0 & 0 & 0\\(1-a)\phi_1 & (\omega_1+\mu+\delta) & 0 & 0 & 0\\a\phi_2 & 0 & (\omega_2+\mu+\delta) & 0 & 0\\0 & \omega_1 & 0 & (1-b)\sigma_Q-(b\sigma_2+\mu+\delta) & 0\\0 & 0 & \omega_2 & -(1-b)\sigma_QQ & (\sigma_1+\mu+\delta)\end{bmatrix} \tag{34}$$

And

$$-G(U,V)=\begin{bmatrix}\lambda\frac{(\gamma I_a+I_s)}{N}S\\0\\0\\0\\0\end{bmatrix} \tag{35}$$

The above equation (37) consequently implies that;

$$G(U,V)=\begin{bmatrix}\lambda\frac{(\gamma I_a+I_s)}{N}S\\0\\0\\0\\0\end{bmatrix}\leq 0 \tag{36}$$

Since $G^0(U,V)\leq 0,$ then condition (2) is not satisfied. Thus $E^0=(A^0,0)$ may not be globally asymptotically stable for $R_n<1.$ Hence, the following theorem is rooted [8].

Theorem 4: *The model's state of diphtheria eradication, as described by Equation (1), demonstrates global asymptotic stability in the biologically feasible region if* $R_n<1$ *and unstable if* $R_n>1.$

2.5 *The presence of the endemic equilibrium points (Diphtheria present state)*

In this study, we investigate the presence of endemic equilibrium states, denoting stable solutions within the model, wherein diphtheria persists within the population over time. These equilibrium points represent the stable disease states where the number of infected individuals and other compartments reaches a steady state.

In epidemiological analysis, the endemic equilibrium points are determined by equating the rates of change of model variables to zero and subsequently solving the resultant system of equations. These equilibrium points represent the stable solutions of the model where the disease persists over time, and they provide important insights into the long-term dynamics of the diphtheria infection in the community.

The endemic equilibrium points are defined as $(S_h^*(t),0,0,0,0,0,0)$ satisfying $S'=E'=I_S'=I_A'=Q'=I'=R'=0,$ by equating equation (1) to 0, we have

$$\beta-\lambda\varphi S^*-\mu S^*=0$$

$$\lambda\varphi S^*-[(1-a)\phi_1+(a\phi_2+\mu)]E^*=0$$

$$(1-a)\phi_1E^*-(\omega_1+\mu+\delta)I_A{}^*=0$$

$$a\phi_2E^*-(\omega_2+\mu+\delta)I_S{}^*=0$$

$$\omega_1I_A{}^*-[(1-b)\sigma_Q+(b\sigma_2+\mu+\delta)]Q^*=0 \tag{37}$$

$$\omega_2 I_S^* + (1-b)\sigma_Q Q^* - (\sigma_1 + \mu + \delta) I^* = 0$$

$$b\sigma_2 Q^* + \sigma_I I^* - \mu R^* = 0$$

Where $\varphi = \frac{(\gamma I_a + I_s)}{N}$

$$S^* = \frac{\beta}{\lambda\phi + \mu};\ E^* = \frac{\lambda\varphi}{[(1-a)\phi_1 + (a\phi_2 + \mu)]} \frac{\beta}{\lambda\varphi + \mu};$$

$$I_A^* = \frac{(1-a)\phi_1 \lambda\beta\varphi}{(\omega_1 + \mu + \delta)[(1-a)\phi_1 + (a\phi_2 + \mu)](\lambda\varphi + \mu)};$$

$$I_S^* = \frac{a\phi_2 \lambda\beta\varphi}{(\omega_2 + \mu + \delta)[(1-a)\phi_1 + (a\phi_2 + \mu)](\lambda\varphi + \mu)};$$

$$Q^* = \frac{\omega_1(1-a)\phi_1 \lambda\beta\varphi}{[(1-b)\sigma_Q + (b\sigma_2 + \mu + \delta)](\omega_1 + \mu + \delta)[(1-a)\phi_1 + (a\phi_2 + \mu)](\lambda\varphi + \mu)}; \tag{38}$$

$$I^* = \frac{\omega_2 a\phi_2 \lambda\beta\varphi}{(\sigma_1 + \mu + \delta)(\omega_2 + \mu + \delta)[(1-a)\phi_1 + (a\phi_2 + \mu)](\lambda\varphi + \mu)} - \frac{(1-b)\sigma_Q}{(\sigma_1 + \mu + \delta)} \frac{\omega_1(1-a)\phi_1 \lambda\beta\varphi}{[(1-b)\sigma_Q + (b\sigma_2 + \mu + \delta)](\omega_1 + \mu + \delta)[(1-a)\phi_1 + (a\phi_2 + \mu)](\lambda\phi + \mu)};$$

$$R^* = \frac{-b\sigma_2 \omega_1(1-a)\phi_1 \lambda\beta\varphi}{\mu[(1-b)\sigma_Q + (b\sigma_2 + \mu + \delta)](\omega_1 + \mu + \delta)[(1-a)\phi_1 + (a\phi_2 + \mu)](\lambda\varphi + \mu)} - \left[\frac{\sigma_I}{\mu} \frac{\omega_2 a\phi_2 \lambda\beta\varphi}{(\sigma_1 + \mu + \delta)(\omega_2 + \mu + \delta)[(1-a)\phi_1 + (a\phi_2 + \mu)](\lambda\varphi + \mu)} - \frac{(1-b)\sigma_Q}{(\sigma_1 + \mu + \delta)} \frac{\omega_1(1-a)\phi_1 \lambda\beta\varphi}{[(1-b)\sigma_Q + (b\sigma_2 + \mu + \delta)](\omega_1 + \mu + \delta)[(1-a)\phi_1 + (a\phi_2 + \mu)](\lambda\varphi + \mu)} \right]$$

Utilizing conventional methodologies, the model exhibits disease-free dynamics at equilibrium point E^0.

2.6 *Sensitivity analysis of the reservoirs-human model*

This section focuses on sensitivity analysis of the diphtheria model, studying how parameter changes affect model predictions. The aim is to identify influential parameters, understand their impact, and enhance the model's robustness.

In sensitivity analysis, a broad spectrum of parameters undergoes systematic variation within their feasible ranges, while the resulting behavior of the model is rigorously scrutinized. This variation can be conducted either in isolation (referred to as one-at-a-time sensitivity

analysis) or collectively (referred to as global sensitivity analysis) for multiple parameters concurrently.

We conducted an examination of the model's reproductive ratio R_n to assess variations and the influence of parameter alterations.

Definition: The delineation of the Normalized Forward-Sensitivity Index for a variable U, characterized by varying dependency on parameter T, is explicated as follows

$$X_T^U = \frac{\partial U}{\partial T} \cdot \frac{T}{U} \tag{42}$$

Concerning the model parameters, we will proceed to calculate the sensitivity indices for the basic reproductive ration R_n denoted as 1.

Sensitivity index for λ

The calculated metric denoted as the Normalized Forward-Sensitivity Index for λ is expressed as:

$$X_\lambda^{R_n} = \frac{\partial R_n}{\partial \lambda} \cdot \frac{\lambda}{R_n} \tag{43}$$

$$R_n = \frac{1}{((1-a)\phi_1 + a\phi_2 + \mu)(\omega_1 + \mu + \delta)(\omega_2 + \mu + \delta)} \begin{bmatrix} (1-a)\phi_1 \lambda \gamma(\omega_2 + \mu + \delta) \\ + a\phi_2 \lambda(\omega_1 + \mu + \delta) \end{bmatrix}$$

Assessing the derivatives in (43), we have;

$$\frac{\partial R_n}{\partial \lambda} = \frac{1}{\lambda} R_n \tag{44}$$

Then,

$$X_\lambda^{R_n} = \frac{\partial R_n}{\partial \lambda} \cdot \frac{\lambda}{R_n} = \frac{1}{\lambda} R_n \cdot \frac{\lambda}{R_n}$$

$$\therefore \quad X_\lambda^{R_n} = +1 \tag{45}$$

This gives us the sensitivity index λ.

The sensitivity measures for the remaining parameters within the basic reproductive ratio are computed employing a uniform procedure, ensuring consistency across calculations. Hence, the sensitivity measures for these parameters are provided Table 1, Figure 3 shows the visual depiction of parameter sensitivity and Figure 4 shows the effect of omega1 and omega2 on Rn. Hence, the sensitivity measures for these parameters are provided as follows:

Table 1

Indices of sensitivity concerning supplementary parameters in the framework of the fundamental reproductive rate.

Variables	Values	Source	Index Indicator	Sensitivity Estimation
λ	0.000097	Islam, Z., et-al, (2022)	+	1
γ	0.7	Kanchanarat, S., et-al (2022)	+	0.3641618497
ϕ_1	0.95	Islam, Z., et-al, (2022)	-	-0.08489277216
ϕ_2	0.95	Islam, Z., et-al, (2022)	+	0.08699361284
ω_1	0.5	Assumed	-	-0.3588508570
ω_2	0.5	Assumed	-	-0.6265649886
μ	0.002	Islam, Z., et-al, (2022)	-	-0.006042503718
δ	0.0054	Islam, Z., et-al, (2022)	-	-0.01064249114
a	0.55	Kanchanarat, S., et-al (2022)	+	0.1907514451

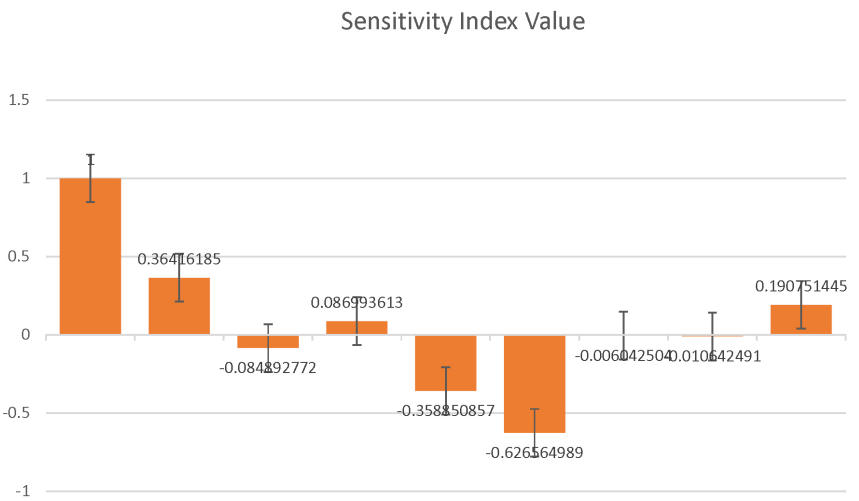

Figure 3

Visual depiction of parameter sensitivity

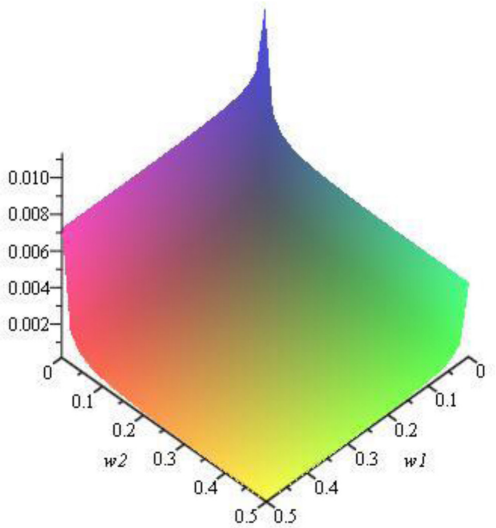

Figure 4

Effect of ω_1 and ω_2 on R_n

2.7 *Optimal Control Strategies for Diphtheria*

The objective is to mitigate disease transmission and its associated effects, accounting for resource limitations and optimizing the deployment of existing interventions. These strategies typically involve a combination of preventive measures, surveillance, vaccination campaigns, and prompt case management. Several critical elements contribute to effective diphtheria control measures. These include the modulation of transmission dynamics through the reduction of transmission rates by $(1-\tau_1)$, where τ_1 denotes the routine vaccination with the diphtheria vaccine, which is typically administered as part of a combination vaccine with tetanus and pertussis. The efforts of health facilities, laboratories, and health care providers on surveillance and early detection of diphtheria in the transition of symptomatic to isolated treatment, asymptomatic to home quarantine, home quarantine to Isolated treatment, and exposed to symptomatic are denoted as τ_2, τ_3, τ_4, and τ_5, respectively. Utilizing these premises as a basis, the subsequent array of novel equations is formulated:

$$\frac{dS}{dt} = \beta - (1-\tau_1)\lambda\frac{(\gamma I_a + I_s)}{N}S - \mu S$$

$$\frac{dE}{dt} = (1-\tau_1)\lambda\frac{(\gamma I_a + I_s)}{N}S - (1-a)\phi E - (a\tau_5 + \mu)E$$

$$\begin{aligned}
\frac{dI_A}{dt} &= (1-a)\phi E - (\tau_3 + \mu + \delta)I_A \\
\frac{dI_S}{dt} &= a\tau_5 E - (\tau_2 + \mu + \delta)I_S \\
\frac{dQ}{dt} &= \tau_3 I_A - (1-b)\tau_4 Q - (b\sigma_2 + \mu + \delta)Q \\
\frac{dI}{dt} &= \tau_2 I_S + (1-b)\tau_4 Q - (\sigma_I + \mu + \delta)I \\
\frac{dR}{dt} &= b\sigma_2 Q + \sigma_I I - \mu R
\end{aligned} \tag{46}$$

2.8 *Examination of the model integrating preventive interventions*

In this section, a model was developed based on a systematic framework, emphasizing the potential for manipulation using Pontryagin's Maximum Principle. By focusing on the optimal solution described in equation set (46), a significant concern regarding control has been identified and subsequently explained before exploring its comprehensive global optimization. The complex task of selecting the most effective strategies is encapsulated by the objective function labeled as F. The primary goal is to minimize the population susceptible to, exposed to, and affected by the disease, whether asymptomatic or symptomatic, within a specified time frame [0, T].

Let $V = \{(\tau_1, \tau_2, \tau_3, \tau_4, \tau_5) \in V\}$ define over a Lebesgue measurable set on [0, 1],

For $0 \le \tau_i(t) \le 1 \in [0,1], i = 1, 2, 3, 4, 5$

Next, the objective function, denoted as F, is established.

$$\begin{aligned}
F(\tau_1, \tau_2, \tau_3, \tau_4, \tau_5) = \int_0^T (P_1 I + P_2 Q + P_3 I_S + P_4 I_A + P_5 E \\
+ \frac{1}{2}(U_1 \tau_1^{\,2} + U_2 \tau_2^{\,2} + U_3 \tau_3^{\,2} + U_4 \tau_4^{\,2} + U_5 \tau_5^{\,2}))\, dt
\end{aligned} \tag{47}$$

Constraint to

$$\begin{aligned}
\frac{dS}{dt} &= \beta - (1-\tau_1)\lambda \frac{(\gamma I_a + I_s)}{N} S - \mu S \\
\frac{dE}{dt} &= (1-\tau_1)\lambda \frac{(\gamma I_a + I_s)}{N} S - (1-a)\phi E - (a\tau_5 + \mu)E \\
\frac{dI_A}{dt} &= (1-a)\phi E - (\tau_3 + \mu + \delta)I_A \\
\frac{dI_S}{dt} &= a\tau_5 E - (\tau_2 + \mu + \delta)I_S \\
\frac{dQ}{dt} &= \tau_3 I_A - (1-b)\tau_4 Q - (b\sigma_2 + \mu + \delta)Q
\end{aligned}$$

$$\frac{dI}{dt} = \tau_2 I_S + (1-b)\tau_4 Q - (\sigma_I + \mu + \delta)I$$
$$\frac{dR}{dt} = b\sigma_2 Q + \sigma_I I - \mu R$$

The final time point is denoted by the parameter T, with coefficients P_1 through P_5 signifying the weight coefficients assigned to the virus across various demographic categories, including isolated persons, home-quarantined individuals, symptomatic cases, asymptomatic cases, and exposed individuals.

The principal aim of this section revolves around diminishing operational costs, as delineated by equation (47). Additionally, our inquiry expands to include an examination of the social and economic ramifications $U_1\tau_1^2$, $U_2\tau_2^2$, $U_3\tau_3^2$, $U_4\tau_4^2$ and $U_5\tau_5^2$ linked to the outlined scenario.

To achieve the objective of tackling the issue of control, our efforts are directed towards discerning the functionalities.

$(\tau_1^*(t), \tau_2^*(t), \tau_3^*(t), \tau_4^*(t), \tau_5^*(t))$ such that

$$F(\tau_1^*(t), \tau_2^*(t), \tau_3^*(t), \tau_4^*(t), \tau_5^*(t)) = \min\{F(\tau_1, \tau_2, \tau_3, \tau_4, \tau_5), (\tau_1, \tau_2, \tau_3, \tau_4, \tau_5) \in V\} \quad (48)$$

2.9 *The Presence of an Optimal Control Solution*

Theorem: *In accordance with equation (48), it is pertinent to consider* $F(\tau_1, \tau_2, \tau_3, \tau_4, \tau_5)$ *subject under the constraints outlined in (46), with t=0 denoting the initial condition. Subsequently, when determining the optimal control, it is imperative to ascertain the aforementioned condition to be* $\tau^* = (\tau_1^*(t), \tau_2^*(t), \tau_3^*(t), \tau_4^*(t), \tau_5^*(t))$ *such that*

$$F(\tau_1^*(t), \tau_2^*(t), \tau_3^*(t), \tau_4^*(t), \tau_5^*(t)) = \min\{F(\tau_1, \tau_2, \tau_3, \tau_4, \tau_5), (\tau_1, \tau_2, \tau_3, \tau_4, \tau_5) \in V\}$$

Proof: Due to the convexity exhibited by the integrand F regarding control measures $\tau_1, \tau_2, \tau_3, \tau_4, \tau_5$, the presence of an optimal control solution is guaranteed.

Subsequently, it is imperative to illustrate the most effective remedy. The Lagrangian function is articulated in the subsequent manner:

$$L = P_1 I + P_2 Q + P_3 I_S + P_4 I_A + P_5 E + \frac{1}{2}(U_1\tau_1^2 + U_2\tau_2^2 + U_3\tau_3^2 + U_4\tau_4^2 + U_5\tau_5^2) \quad (49)$$

The Hamiltonian function is given as;

$$
\begin{aligned}
H_A &= P_1 I + P_2 Q + P_3 I_S + P_4 I_A + P_5 E + \frac{1}{2}(U_1 \tau_1^{\,2} + U_2 \tau_2^{\,2} + U_3 \tau_3^{\,2} + U_4 \tau_4^{\,2} + U_5 \tau_5^{\,2}) \\
&+ \Omega_S \left[\beta - (1-\tau_1)\lambda \frac{(\gamma I_a + I_s)}{N} S - \mu S \right] \\
&+ \Omega_E \left[(1-\tau_1)\lambda \frac{(\gamma I_a + I_s)}{N} S - (1-a)\phi E - (a\tau_5 + \mu) E \right] \\
&+ \Omega_{I_A} [(1-a)\phi E - (\tau_3 + \mu + \delta) I_A] + \Omega_{I_S} [a\tau_5 E - (\tau_2 + \mu + \delta) I_S] \\
&+ \Omega_Q [\tau_3 I_A - (1-b)\tau_4 Q - (b\sigma_2 + \mu + \delta) Q] \\
&+ \Omega_I [\tau_2 I_S + (1-b)\tau_4 Q - (\sigma_I + \mu + \delta) I] + \Omega_R [b\sigma_2 Q + \sigma_I I - \mu R]
\end{aligned} \tag{50}
$$

Given $\Omega_k, k \in \{S, E, I_A, I_S, Q, I, R\}$ are distinct and non-overlapping variables.

Currently, we are in a position to apply the requisite parameters to the Hamiltonian (H) for examination.

In the pursuit of elucidating the adjoint equation and satisfying the transversality condition, we employ the Hamiltonian H as our analytical tool. By engaging in differential calculus, we determine the derivatives of the variables S, E, I_A, I_S, Q, I, R relative to the Hamiltonian. This systematic approach culminates in the derivation of the adjoint equation, presented as follows:

$$
\frac{d\Omega_S}{dt} = -\frac{\partial H}{dS} = \begin{bmatrix} \left((1-\tau_1)\lambda \frac{(\gamma \Omega_{I_A} + \Omega_{I_S})}{N} + (1-\tau_1)\lambda \frac{(\gamma I_A + I_S)}{N^2} S + \mu \right) \Omega_S + \\ + \left((1-\tau_1)\lambda \frac{(\gamma \Omega_{I_A} + \Omega_{I_S})}{N} + (1-\tau_1)\lambda \frac{(\gamma I_A + I_S)}{N^2} S + \mu \right) \Omega_E \end{bmatrix}
$$

$$
\frac{d\Omega_E}{dt} = \frac{-\partial H}{dE} = \begin{bmatrix} -P_5 + \left((1-\tau_1)\lambda \frac{(\gamma I_a + I_s)}{N^2} S + (1-a)\phi - (a\tau_5 + \mu) \right) \Omega_E \\ + (1-\tau_1)\lambda \frac{(\gamma I_a + I_s)}{N^2} \Omega_S - (1-a)\phi \Omega_{I_A} - a\tau_5 \Omega_{I_S} \end{bmatrix}
$$

$$
\frac{d\Omega_{I_A}}{dt} = -\frac{\partial H}{dI_A} = \begin{bmatrix} -P_4 + (\tau_3 + \mu + \delta)\Omega_{I_A} + (1-\tau_1)\lambda \left(\frac{(\gamma I_a + I_s)}{N^2} - \frac{\gamma}{N} \right) \Omega_S \\ + (1-\tau_1)\lambda \left(\frac{(\gamma I_a + I_s)}{N^2} - \frac{\gamma}{N} \right) \Omega_E + \tau_3 \Omega_Q \end{bmatrix} \tag{51}
$$

$$
\frac{d\Omega_{I_S}}{dt} = -\frac{\partial H}{dI_S} = \begin{bmatrix} -P_3 + (\tau_2 + \mu + \delta)\Omega_{I_S} + (1-\tau_1)\lambda \left(\frac{(\gamma I_a + I_s)}{N^2} - \frac{1}{N} \right) \Omega_S \\ + (1-\tau_1)\lambda \left(\frac{(\gamma I_a + I_s)}{N^2} - \frac{\gamma}{N} \right) \Omega_E + \tau_2 \Omega_I \end{bmatrix}
$$

$$\frac{d\Omega_Q}{dt} = -\frac{\partial H}{dQ} = \begin{bmatrix} -P_2 + ((1-b)\tau_4 + (b\sigma_2 + \mu + \delta))\Omega_Q - (1-b)\tau_4\Omega_I - b\sigma_Q\Omega_R \\ +(1-\tau_1)\lambda\frac{(\gamma I_a + I_s)}{N^2}\Omega_S + (1-\tau_1)\lambda\left(\frac{(\gamma I_a + I_s)}{N^2} - \frac{\gamma}{N}\right)\Omega_E \end{bmatrix}$$

$$\frac{d\Omega_I}{dt} = -\frac{\partial H}{dI} = \begin{bmatrix} -P_1 + (\sigma_I + \mu + \delta)\Omega_I - \sigma_I\Omega_R + (1-\tau_1)\lambda\frac{(\gamma I_a + I_s)}{N^2}\Omega_S \\ +(1-\tau_1)\lambda\left(\frac{(\gamma I_a + I_s)}{N^2} - \frac{\gamma}{N}\right)\Omega_E \end{bmatrix}$$

$$\frac{d\Omega_R}{dt} = -\frac{\partial H}{dR} = \left[\mu\Omega_R - (1-\tau_1)\lambda\frac{(\gamma I_a + I_s)}{N^2}\Omega_S - (1-\tau_1)\lambda\left(\frac{(\gamma I_a + I_s)}{N^2} - \frac{\gamma}{N}\right)\Omega_E\right]$$

Given the conditions of transversally to be $\Omega_k(T) = 0, k \in \{S, E, I_A, I_S, Q, I, R\}$.

In the quest for minimizing the Hamiltonian, symbolized as H, concerning the optimal control variables, we engage in the differentiation process with respect to $\tau_1, \tau_2, \tau_3, \tau_4, \tau_5$. This yields a set of equations, which we subsequently equate to zero to determine the optimal control configuration. This methodology culminates in the attainment of the desired optimal control solution.

With $S = S^*, E = E^*, I_A = I_A{}^*, I_S = I_S{}^*, Q = Q^*, I = I^*, R = R^*$

Then, we have

$$\begin{aligned} \frac{dH}{d\tau_1} &= U_1\tau_1{}^* - \lambda\frac{(\gamma I_a + I_s)}{N}(\Omega_S - \Omega_E)S = 0 \\ \frac{dH}{d\tau_2} &= U_2\tau_2{}^* - (\Omega_{I_S} - \Omega_I)I_S = 0 \\ \frac{dH}{d\tau_3} &= U_3\tau_3{}^* - (\Omega_{I_A} - \Omega_Q)I_A = 0 \\ \frac{dH}{d\tau_4} &= U_4\tau_4{}^* - (\Omega_Q - \Omega_I)(1-b)Q = 0 \\ \frac{dH}{d\tau_5} &= U_5\tau_5{}^* - (\Omega_E - \Omega_{I_S})a = 0 \end{aligned} \tag{52}$$

Through simplification, a solution is derived for the optimal control strategy.

$$\begin{aligned} \tau_1{}^* &= \lambda\frac{(\gamma I_a + I_s)}{U_1 N}(\Omega_S - \Omega_E)S \\ \tau_2{}^* &= \frac{(\Omega_{I_S} - \Omega_I)I_S}{U_2} \\ \tau_3{}^* &= \frac{(\Omega_{I_A} - \Omega_Q)I_A}{U_3} \end{aligned} \tag{53}$$

$$\tau_4^* = \frac{(\Omega_Q - \Omega_I)(1-b)Q}{U_4}$$
$$\tau_5^* = \frac{(\Omega_E - \Omega_{I_S})a}{U_5}$$

Utilizing the boundary conditions, the solution is provided as follows.

$$\begin{aligned}
\tau_1 &= \min\left\{1, \max\left\{0, \lambda \frac{(\gamma I_a + I_s)}{U_1 N}(\Omega_S - \Omega_E)S\right\}\right\},\\
\tau_2 &= \min\left\{1, \max\left\{0, \frac{(\Omega_{I_S} - \Omega_I)I_S}{U_2}\right\}\right\},\\
\tau_3 &= \min\left\{1, \max\left\{0, \frac{(\Omega_{I_A} - \Omega_Q)I_A}{U_3}\right\}\right\},\\
\tau_4 &= \min\left\{1, \max\left\{0, \frac{(\Omega_Q - \Omega_I)(1-b)Q}{U_4}\right\}\right\},\\
\tau_5 &= \min\left\{1, \max\left\{0, \frac{(\Omega_E - \Omega_{I_S})a}{U_5}\right\}\right\},
\end{aligned} \tag{54}$$

Proved.

3. Results

3.1. *Numerical Simulation*

In this simulation we provide a way to observe how the disease spreads over time, how different variables change, and how the impact of interventions can be assessed and this allows researchers and public health officials to gain insights into the behavior of the disease under various scenarios and to test the effectiveness of different control strategies.

Table 2

Parameters with their values

Parameters	Values	Source
β	200	Islam, Z., et-al, (2022)
b	0.55	Kanchanarat, S., et-al (2022)
σ_2	2.1429	Kanchanarat, S., et-al (2022)
σ_1	2.1429	Kanchanarat, S., et-al (2022)
σ_Q	0.5	Assumed

The state variables' initial conditions are as follows; $S(0)=1500$, $E(0)=1400$, $I_A(0)=880$, $I_S(0)=550$, $Q(0)=500$, $I(0)=800$, $R(0)=1100$. Also, $\beta=200$, $b=0.55$, $\sigma_I=2.1429$, $\sigma_2=2.1429$. Likewise $\tau_1=k_1$, $\tau_2=k_2$, $\tau_3=k_3$, $\tau_4=k_4$, $\tau_5=k_5$. The parameter values needed for the simulation are displayed in **Table 1** and **Table 2**.

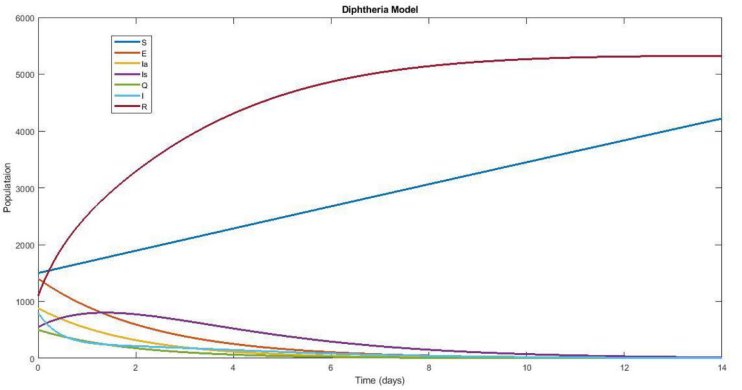

Figure 5

Solution of the Diphtheria Model equations (46) with the values of the parameters in Table 1 and for $\tau_1=0.5, \tau_2=\omega_2, \tau_3=\omega_1, \tau_4=\sigma_Q=0.5, \tau_5=0.95.$

$$\tau_1=k_1, \tau_2=k_2, \tau_3=k_3, \tau_4=k_4, \tau_5=k_5.$$

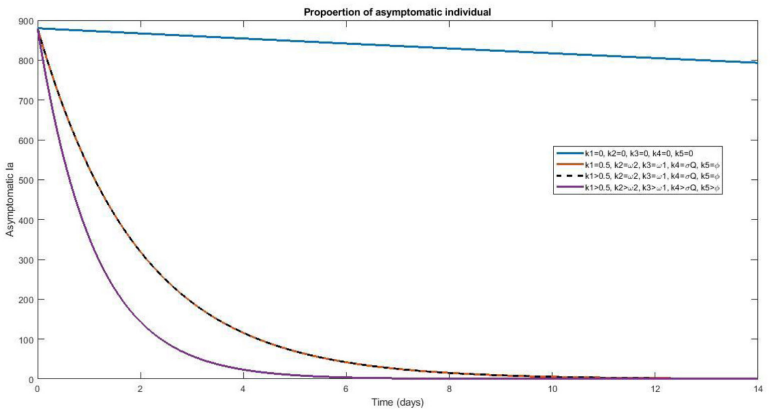

Figure 6

Trajectories solution for the optimal control of asymptomatic individual

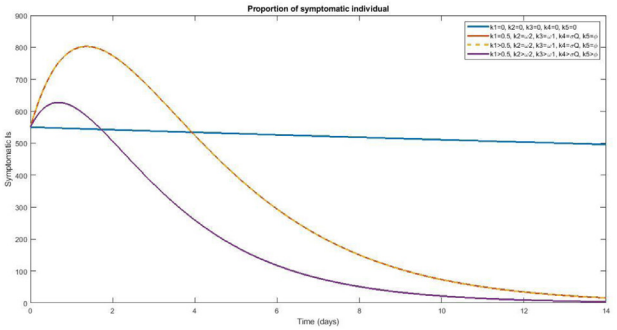

Figure 7

Trajectories solution for the optimal control of symptomatic individual

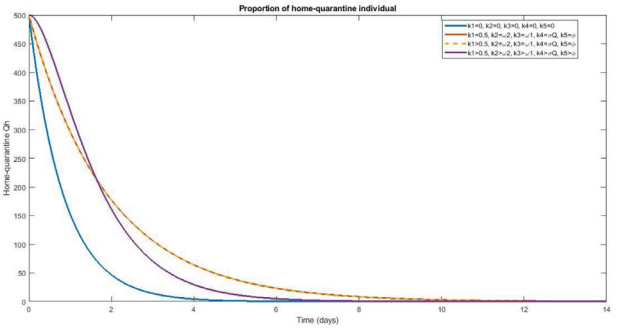

Figure 8

Trajectories solution for the optimal control of home-quarantine individual

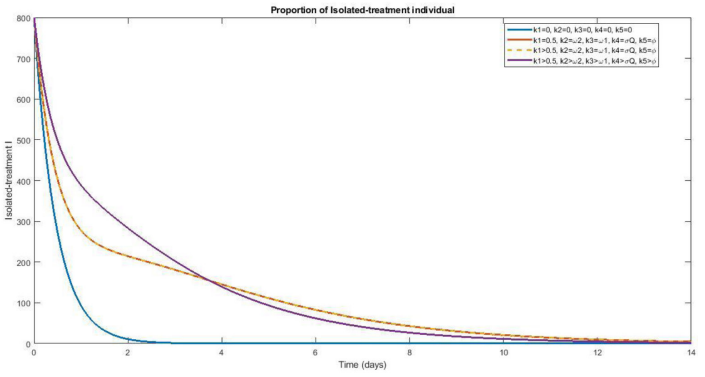

Figure 9

Trajectories solution for the optimal control of Isolated-treatment individual

3.2 *Discussion*

Figure 5 depicts the comprehensive compartments of the diphtheria model, crucial for elucidating and scrutinizing the dynamics of diphtheria disease transmission. This visualization augments the model's elucidative capacity, facilitates comprehension of diphtheria disease transmission mechanisms, and furnishes valuable insights for formulating strategic interventions aimed at disease control and prevention.

In **Figure 6**, the figure show that when optimal control measures are applied, there is a noticeable decrease in the size of the asymptomatic compartment. This suggests that these optimal measures are effective in reducing the number of individuals who are carriers of the diphtheria bacteria without showing symptoms. **Figure 6** also demonstrate that routine vaccination alone doesn't lead to a significant decrease in the size of the asymptomatic compartment. This finding imply that while routine vaccination is important for preventing severe cases of diphtheria, it might not be as effective at directly reducing the carrier population. This could be due to factors such as incomplete coverage, waning immunity, or the specific dynamics of diphtheria transmission. The **figure 6** highlight the significance of a comprehensive approach to diphtheria control. Combining routine vaccination with efforts from health facilities, laboratories, and healthcare providers for surveillance and early detection, as well as implementing measures to transition symptomatic individuals to isolated treatment, asymptomatic individuals to home quarantine, and exposed individuals to symptomatic, leads to a noticeable decrease in the size of the asymptomatic compartment. This suggests that a combination of interventions targeting different stages of the

The **figure 7** indicate that implementing optimal control measures results in a noticeable decrease in the size of the symptomatic compartment. This observation suggests that these optimal measures are highly effective in reducing the number of individuals who develop symptoms due to diphtheria. When only routine vaccination is employed as a control measure, there is no significant decrease in the size of the symptomatic compartment. This suggests that routine vaccination alone might not be enough to substantially reduce the number of individuals displaying symptoms. However, when a combination of control measures is used, including routine vaccination along with efforts from health facilities, laboratories, and healthcare providers focused on surveillance, early detection, and timely intervention, there is a noticeable decrease in the size of the symptomatic compartment. This implies that a comprehensive

strategy that involves both vaccination and active measures to identify, isolate, and treat symptomatic cases can lead to a more significant reduction in symptomatic individuals. The analysis also highlights the impact of interventions at various transition stages of the disease. These stages include the transition from symptomatic to isolated treatment, asymptomatic to home quarantine, home quarantine to isolated treatment, and exposed to symptomatic. The efforts made at each of these stages contribute to reducing the number of symptomatic cases and subsequently the size of the symptomatic compartment.

Figure 8 describes how the size of the home quarantine compartment changes over time based on various control measures applied in a diphtheria context. When optimal control measures are applied, there's a noticeable increase in the size of the home quarantine compartment. This suggests that these measures are leading to more individuals being placed in home quarantine as a preventive measure. Implementing all recommended control measures from the initial day through the subsequent day leads to a rise in the populace undergoing home quarantine. This indicates that the measures taken from the early stages of the outbreak lead to more individuals being identified and isolated. Interestingly, when only vaccination control measures are applied, the number of individuals in home quarantine increases more than when all optimal control measures are applied from day 2 onwards. This might indicate that vaccination efforts alone are effective in identifying and isolating individuals who are potentially exposed to diphtheria.

Finally, from **Figure 9**, which represent the changes in the size of the isolated treatment compartment over time based on various control measures applied. When optimal control measures are applied, there is a noticeable increase in the size of the isolated treatment compartment. This suggests that these measures are effective in increasing the number of individuals who are receiving isolated treatment. Applying all the optimal control measures from day 1 to day 4 results in an increase in the number of isolated treatment individuals. This indicates that implementing these measures early in the outbreak helps in effectively isolating and treating affected individuals. Interestingly, when only the vaccination control is applied, the number of isolated treatment individuals increases more than when using all the optimal controls from day 4. This could imply that vaccination alone can lead to an increased identification and isolation of cases, possibly due to the reduction in the overall disease burden in the population.

4. Conclusion

The analysis underscores the significance of adopting a comprehensive approach when dealing with the control of diphtheria, extending well beyond mere vaccination. It emphasizes the need to address multiple dimensions, such as swift and accurate diagnosis, the effective isolation of affected individuals, the implementation of quarantine measures, and the provision of appropriate treatment for those showing symptoms. This combined strategy holds the potential to significantly reduce the entire burden of the disease and its propensity to spread.

While routine vaccination certainly plays a crucial role in preventive efforts, the study reveals that a more holistic combination of interventions is required. This involves tackling various stages of the disease's progression through timely detection, isolating both asymptomatic and symptomatic cases, and providing appropriate treatment. Such a comprehensive approach emerges as pivotal for significantly curtailing the prevalence of both asymptomatic carriers and symptomatic cases.

These findings hold significant implications for the formulation of public health policies and interventions geared towards diphtheria control. It underscores the importance for governments and healthcare organizations to adopt a multi-faceted strategy. This strategy should encompass not only vaccination but also encompass vigilant surveillance, early identification of cases, the isolation of affected individuals, and the implementation of quarantine protocols. By embracing such a holistic strategy, the spread of diphtheria can be effectively managed, and its impact on the population's health minimized.

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Authors' contribution statement

The authors, Adedapo Loyinmi and Sunday Gbodogbe, contributed equally to the manuscript

Competing interest

The authors declare that they have no competing interest.

Data Availability

There are no dataset generated during and/or analyzed during the current study.

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References

[1] WHO, "Diphtheria - Questions and Answers," (2017). [Online]. Available: https://www.who.int/news-room/questions-and-answers/item/diphtheria. [Accessed: Dec. 7, 2024].

[2] Mayo Clinic Staff, "Diphtheria: Symptoms and Causes," (2022). [Online]. Available: https://www.mayoclinic.org/diseases-conditions/diphtheria/symptoms-causes/syc-20351897. [Accessed: Dec. 7, 2024].

[3] Anna M. Acosta, Pedro L. Moro, Susan Hariri, and Tejpratap S.P. Tiwari, "Diphtheria," CDC, (2021). [Online]. Available: https:// www.cdc.gov / vaccines / pubs/ pinkbook / dip.html#:~:text=Occurrence. [Accessed: Dec. 7, 2024].

[4] Wikipedia, "Diphtheria," (2022). [Online]. Available: https://en.m.wikipedia.org/wiki/Diphtheria#:~:text=In%201735%2C%20a%20diphtheria%20epidemic. [Accessed: Dec. 7, 2024].

[5] K. Sornbundit, W. Triampo, and C. Modchang, "Mathematical modeling of diphtheria transmission in Thailand," *Computers in Biology and Medicine*, vol. 87, pp. 162–168 (2017).

[6] K. T. Akinfe and A. C. Loyinmi, "Stability analysis and semi-analytic solution to a SEIR–SEI Malaria transmission model using He's variational iteration method," *Preprints* (2022).

[7] J. O. Agbomola and A. C. Loyinmi, "Modelling the impact of some control strategies on the transmission dynamics of Ebola virus in human–bat population: An optimal control analysis," *Heliyon*, vol. 8, no. 12 (2022), Art. no. e12121. [Online]. Available: https://doi.org/10.1016/j.heliyon.2022.e12121.

[8] A. C. Loyinmi, T. K. Akinfe, and A. A. Ojo, "Qualitative analysis and dynamical behavior of a Lassa haemorrhagic fever model with exposed rodents and saturated incidence rate," *Scientific Afri-*

can, vol. 14 (2021), Art. no. e01028. [Online]. Available: https://doi.org/10.1016/j.sciaf.2021.e01028.

[9] Z. Islam, S. Ahmed, M. M. Rahman, M. F. Karim, and M. R. Amin, "Global stability analysis and parameter estimation for a diphtheria model: A case study of an epidemic in Rohingya refugee camp in Bangladesh," *Computational and Mathematical Methods in Medicine* (2022).

[10] S. Kanchanarat, S. Chinviriyasit, and W. Chinviriyasit, "Mathematical assessment of the impact of imperfect vaccination on diphtheria transmission dynamics," *Symmetry*, vol. 14, no. 10 (2022).

[11] V. Singh, R. C. Poonia, S. Kumar, P. Dass, P. Agarwal, V. Bhatnagar, and L. Raja, "Prediction of COVID-19 corona virus pandemic based on time series data using support vector machine," *Journal of Discrete Mathematical Sciences and Cryptography* (2020). DOI: 10.1080/09720529.2020.1784535.

[12] V. Bhatnagar, R. C. Poonia, P. Nagar, S. Kumar, V. Singh, L. Raja, and P. Dass, "Descriptive analysis of COVID-19 patients in the context of India," *Journal of Interdisciplinary Mathematics*, vol. 24, no. 3, pp. 489–504 (2021). DOI: 10.1080/09720502.2020.1761635.

[13] J. Mondal, P. Samui, and A. N. Chatterjee, "Optimal control strategies of non-pharmaceutical and pharmaceutical interventions for COVID-19 control," *Journal of Interdisciplinary Mathematics*, vol. 24, no. 1, pp. 125–153 (2021). DOI: 10.1080/09720502.2020.1833459.

[14] A. C. Loyinmi, S. O. Gbodogbe, and K. O. Idowu, "On the interaction of the human immune system with foreign body: Mathematical modeling approach," *Kathmandu University Journal of Science, Engineering and Technology*, vol. 17, no. 2, pp. 1–17 (2023). [Online]. Available: https://journals.ku.edu.np/kuset/article/view/137.

[15] O. K. Idowu and A. C. Loyinmi, "Qualitative analysis of the transmission dynamics and optimal control of COVID-19," *EDUCATUM Journal of Science, Mathematics and Technology*, vol. 10, no. 1, pp. 54–70 (2023). DOI: 10.37134/ejmst.vol10.1.7.2023.

[16] A. C. Loyinmi and T. K. Akinfe, "Exact solution to the family of Fisher reaction-diffusion equations using Elzaki homotopy transformation perturbation method," *Engineering Reports*, vol. 2 (2020), Art. no. e12084. DOI: 10.1002/eng2.12084.

[17] K. O. Idowu and A. C. Loyinmi, "Impact of contaminated surfaces on the transmission dynamics of coronavirus disease (COVID-19)," *Biomedical Journal of Scientific & Technical Research*, vol. 51, pp. 42280–42290 (2023). DOI: 10.26717/BJSTR.2023.51008046.

[18] A. C. Loyinmi and O. W. Lawal, "The asymptotic solution for the steady variable-viscosity free convection flow on a porous plate," *Journal of Nigerian Association of Mathematical Physics*, vol. 19, pp. 273–276 (2011).

[19] T. K. Akinfe and A. C. Loyinmi, "An improved differential transform scheme implementation on the generalized Allen-Cahn equation governing oil pollution dynamics in oceanography," *Partial Differential Equations in Applied Mathematics*, vol. 6, Art. no. 100416 (2022). DOI: 10.1016/j.padiff.2022.100416.

[20] A. C. Loyinmi and S. O. Gbodogbe, "Mathematical modeling and control strategies for Nipah virus transmission incorporating Bat-to-pig-to-human pathway," *EDUCATUM Journal of Science, Mathematics and Technology*, vol. 10, no. 2 (2023). DOI: 10.37134/ejsmt.vol11.1.7.2024.

[21] T. K. Akinfe, "A reliable analytic technique for the modified prototypical Kelvin-Voigt viscoelastic fluid model by means of the hyperbolic tangent function," *Partial Differential Equations in Applied Mathematics*, vol. 7, Art. no. 10523 (2023). DOI: 10.1016/j.padiff.2023.10523.

[22] A. C. Loyinmi and T. K. Akinfe, "An algorithm for solving the Burgers-Huxley equation using the Elzaki transform," *SN Applied Sciences*, vol. 2, pp. 1–17 (2020). DOI: 10.1007/s42452-019-1652-3.

[23] T. K. Akinfe and A. C. Loyinmi, "A solitary wave solution to the generalized Burgers-Fisher equation using an improved differential transform method: A hybrid approach scheme," *Heliyon*, vol. 7, Art. no. e07001 (2021). DOI: 10.1016/j.heliyon.2021.e07001.

[24] A. C. Loyinmi and S. O. Gbodogbe, "MATLAB code to simulate the effects of combination of control strategies on the transmission of diphtheria [Dataset]," *Dryad* (2023). DOI: 10.5061/dryad.qrfj6q5ng.

[25] A. C. Loyinmi and K. O. Idowu, "Semi-analytical approach to solving Rosenau-Hyman and Korteweg-de Vries equations using integral transform," *Tanzania Journal of Science*, vol. 49, pp. 26–40 (2023). DOI: 10.4314/tjs.v49i1.3.

[26] A. C. Loyinmi, A. I. Oredein, and S. U. Prince, "Homotopy Adomian decomposition method for solving linear and nonlinear partial differential equations," *Tai Solarin University of Education Journal of Pure and Applied Sciences*, vol. 1, pp. 254–260 (2018).

[27] A. C. Loyinmi, O. W. Lawal, and D. O. Sottin, "Reduced differential transform method for solving partial integro-differential equation," *Journal of Nigerian Association of Mathematical Physics*, vol. 43, pp. 37–42 (2017).

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