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RESEARCH ARTICLE

AGE-STRUCTURED (SVEIHR) MODEL FOR DIPHTHERIA TRANSMISSION ANALYSIS

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Abstract:
The research investigates the transmission dynamics of diphtheria and the role of vaccination as a prominent control measure. A novel Susceptible-Vaccinated-Exposed-Infectious-Hospitalized-Recovered (SVEIHR) model is developed to analyze the spread of the disease among age-structured populations. The study focuses on the existence and uniqueness of the disease-free equilibrium and conducts stability analyses of both local and global equilibria. Sensitivity analysis of targeted parameters is performed to evaluate their impact on disease transmission dynamics. Numerical simulations utilizing the Laplace Adomian Decomposition Method illustrate the effects of these parameters on the compartments of the model, with results presented graphically. Through this comprehensive analysis, the study aims to provide insights into the effectiveness of vaccination strategies in controlling diphtheria and inform evidence-based public health interventions.

Keywords: Age-structured modeling, Basic Reproduction Number, Stability Analysis, Treatment Rate, Laplace Adomian Decomposition Method

1. Introduction

Diphtheria, caused by the bacterium Corynebacterium diphtheriae, continues to be a significant public health issue, especially in regions with low vaccination coverage [1]. Despite the availability of an effective vaccine, outbreaks still occur, underscoring the need for a deeper understanding of the transmission dynamics and the role of vaccination in controlling the disease [2–6]. Mathematical modeling has long been employed to study infectious diseases, providing insights that are crucial for developing effective control strategies. Traditional models, such as the SEIR (Susceptible-Exposed-Infectious-Recovered) framework, have been instrumental in understanding various aspects of disease spread. However, these models often do not account for age-specific differences in contact patterns and immune responses, which are particularly important in the context of diphtheria [7]. Previous research has highlighted the importance of age structure in infectious disease modeling [8–10]. Age-structured models, which incorporate age-specific contact rates and disease progression probabilities, offer a more realistic representation of disease dynamics [11]. These models have been

applied to various infectious diseases, demonstrating their value in understanding the transmission and impact of interventions across different age groups [12–14]. Diphtheria, several studies have explored the impact of vaccination and age-specific contact patterns. However, there remains a gap in integrating these factors comprehensively into a single model that can inform both the understanding of disease dynamics and the development of targeted intervention strategies [12, 13, 15–20]. This research aims to bridge this gap by introducing an age-structured SVEIHR (Susceptible-Vaccinated-Exposed-Infectious-Hospitalized-Recovered) model for diphtheria transmission analysis [16]. The SVEIHR model extends the traditional SEIR framework by incorporating vaccinated and hospitalized compartments [17–20]. The vaccinated compartment represents individuals who have received the diphtheria vaccine, accounting for partial immunity and reduced infectiousness. The hospitalized compartment includes individuals with severe symptoms requiring medical intervention [21]. By incorporating these compartments, the model captures the critical role of vaccination and hospitalization in diphtheria transmission dynamics. The model considers two distinct age groups, recognizing the differences in contact patterns, disease progression, and vaccination responses between children and adults. This distinction allows for a more nuanced analysis of how vaccination strategies can be tailored to different age groups to maximize their effectiveness in [22]. Through parameter calibration using epidemiological data, the model accurately reflects age-specific incidence rates, vaccination coverage, and contact matrices. Sensitivity analysis identifies the most influential parameters, providing insights into key factors driving diphtheria transmission by [18]. The model's predictions are validated against historical outbreak data, ensuring its reliability and applicability. This research emphasizes the critical role of vaccination as a control tool in managing diphtheria. The findings highlight the need for policymakers and health practitioners to prioritize vaccination coverage and promote public awareness about the benefits of vaccination in [23–26]. By adhering to proper usage and ensuring widespread enlightenment, vaccination can significantly reduce the burden of diphtheria and prevent future outbreaks [27]. This research contributes to infectious disease modeling by presenting an age-structured SVEIHR model that integrates vaccination and age-specific factors. It provides valuable insights for policymakers and health practitioners, guiding effective interventions to combat diphtheria and enhance public health outcomes.

2. Mathematical Formulation

A total population N(t) is considered which is divided into sub-populations of S_1, S_2 of susceptible children and adult population, E(t) exposed , I(t) infected, H(t) hospitalized and R(t) recovered population. The level of individuals migrating into the population at Λ, effective contact rate of an individual β and the fraction of the children recruited into the population at π, the level of the spread induced rate at d. The conversion rate in diphtheria disease between the two population of children and adult being exposed at φ_1, φ_2, . The modification of the disease is at a rate λ, and regular treatment of diphtheria disease is at rate of τ. An exposed individual are subjected to recover at a rate of η_1, η_2 and individuals that are hospitalized having been infected is γ while that of infected are said to recover at a rate of δ. Moreso, set of recovered individual form back into the population occurs at at rate of φ_1, φ_2. Respective individuals across the sub-population are subjected to death naturally by μ. Pictorial illustration of this can be displayed from the figure below

2.1. Existing Model

A proposed compartmental-based model for analyzing the dynamics of the spread of diphtheria transmission disease. The governing model is given by the system of non-linear ordinary differential equations below.
This mathematical model analyses the spread of diphtheria disease, particularly considering the natural immunity rate among exposed individuals within the population; it is often cited as a study

utilizing an (SEIQR) model to explore diphtheria dynamics with a focus on natural immunity in the exposed group.

$$\begin{aligned}
\frac{ds}{dt} &= (1-p)\mu N - \frac{\eta si}{N} - \delta s + \phi e \\
\frac{de}{dt} &= \frac{\eta si}{N} - (\beta + \phi + \delta)e \\
\frac{di}{dt} &= \beta e - (\gamma + \delta + \theta)i \\
\frac{dq}{dt} &= \gamma i - (\varepsilon + \delta)q \\
\frac{dr}{dt} &= p\mu N + \varepsilon q - \delta r
\end{aligned} \tag{2.1}$$

N is the total population as $s(0) = s_o, e(0) = i_o, i(0) = i_o, q(0) = q_o, r(0) = r_o \quad \geq 0$

2.1.1. The Modified Model

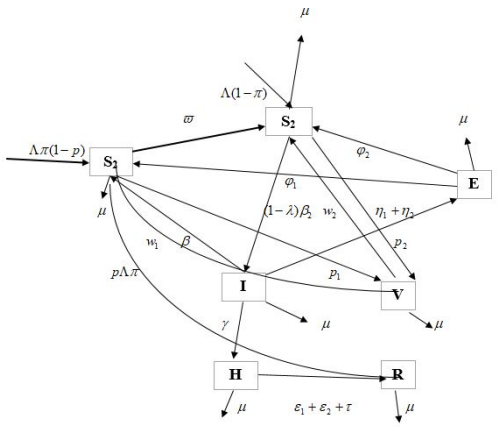

Figure 2.1: The Schematic flow of the SVEIHR Model.

This was extended by that fraction of the children population are newborn who are administered the diphtheria vaccine at a rate p and automatically gain lifelong immunity and moves to recovered class. The model equation is as follows.

$$\begin{aligned}
\frac{dS_1}{dt} &= \Lambda\pi(1-p) - \beta_1 S_1 I - p_1 S_1 + w_1 V + \varphi E - \varpi S s_1 - \mu S_1 \\
\frac{dS_2}{dt} &= \Lambda(1-\pi) - (1-\lambda)\beta S_2 I - p_2 S_2 + w_2 V + \varphi E + \varrho S_1 - \mu S_2 \\
\frac{dV}{dt} &= p_1 S_1 + p_2 S_2 - (w_1 + w_2)V - \mu V \\
\frac{dE}{dt} &= \left(\beta_1 S_1 + (1-\lambda)\beta_2 S_2\right) I - (\eta_1 + \eta_2 + \varphi_1 + \varphi_2 + \mu)E \\
\frac{dI}{dt} &= (\eta_1 + \eta_2)E - (\gamma + \delta + \mu)I \\
\frac{dH}{dt} &= \gamma I - \left(\varepsilon_1 + \varepsilon_2 + \mu + \tau\right) H \\
\frac{dR}{dt} &= p\pi\Lambda + \left(\varepsilon_1 + \varepsilon_2 + \tau\right) H - \mu R
\end{aligned} \tag{2.2}$$

By initial condition that $0 \leq T \leq 1$. When $T = 0$, vulnerable individuals are not immunized or immunization does not affect the vulnerable compartment.

2.2. Tables and Figures

Table numbering follows the subsection in which the table is discussed. For example, in results, the table number starts with 3.1, 3.2, and so on. The same applies to figures. Table captions are placed above the table while figure captions are placed below the figure.

Table 2.1: Description of the parameters and values

Parameter	Description	Values	Units	Refs.
N	Susceptible population	7000		
S_1	Susceptible population of children	4500		
S_2	Susceptible population of Adult	4500		
E	Exposed population	2050		
I	Infected population	142		
H	Hospitalized population	142		
R	Recovered population	306	$perday^{-1}$	[12]
Λ	Recruitment rate into the susceptible children population	0.012	$perday^{-1}$	[5, 8]
τ	Regular treatment rate of hospitalized	0.31	$perday^{-1}$	[19]
d	Diphtheria induced death	0.011	$perday^{-1}$	[20-23]
μ	natural death from the population	0.01	$perday^{-1}$	[18]
π	Children fraction of recruited	0.2102	$perday^{-1}$	[1, 7, 15]
β	Effective contact rate	0.1	$perday^{-1}$	[13]
η_1, η_2	rate of exposed to infected	0.2317	$perday^{-1}$	[2]
γ	Rate of hospitalization	0.31	$perday^{-1}$	[7,18]
$\varepsilon_1, \varepsilon_2$	Recovery rate	0.815	$perday^{-1}$	[17]
δ	Recovery rate from infected	0.3	$perday^{-1}$	[10,16]
λ	Modification parameter	1.7601	$perday^{-1}$	[12, 19]
φ_1, φ_2	Conversion rate exposed children and adults	1.091	$perday^{-1}$	[3]

3. Model Analysis

3.1. Existence and Uniqueness of the Model

Examining the population-related segment of the system of equations, we have
$N(t) = S_1(t) + S_2(t) + V(t) + E(t) + I(t) + H(t) + R(t)$
The derivatives obtained as,

$$\frac{dN(t)}{dt} = \frac{d}{dt}\Big(S_1(t), S_2(t), V(t), E(t), I(t), H(t), R(t)\Big) \tag{3.3}$$

$$\frac{dN(t)}{dt} = \frac{dS_1}{dt} + \frac{dS_2}{dt} + \frac{dV}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dH}{dt} + \frac{dR}{dt} \tag{3.4}$$

$$\begin{aligned}\frac{dN(t)}{dt} &= \Big\{\Lambda\pi(1-p) - \beta_1 S_1 I - p_1 S_1 + w_1 V + \varphi E - \varpi S s_1 - \mu S_1 \\ &+ \Lambda(1-\pi) - (1-\lambda)\beta S_2 I - p_2 S_2 + w_2 V + \varphi E + \varrho S_1 - \mu S_2 + p_1 S_1 + p_2 S_2 - (w_1+w_2)V - \mu V \\ &+ \Big(\beta_1 S_1 + (1-\lambda)\beta_2 S_2\Big) I - (\eta_1 + eta_2 + \varphi_1 + \varphi_2 + \mu)E + (\eta_1 + eta_2)E - (\gamma+\delta+\mu)I + \gamma I \\ &- \Big(\varepsilon_1\varepsilon_2 + \mu + \tau\Big) H \\ &+ p\pi\Lambda + \Big((\varepsilon_1\varepsilon_2) + \tau\Big) H - \mu R\Big\}\end{aligned}$$

$\frac{dN(t)}{dt} \leq \Lambda - \mu N - \delta I(t)$ where no outbreak of diphtheria is observed, $\delta = 0$
$\frac{dN}{dt} + \mu N \leq \Lambda$
$N(t)e^{\mu t} = \frac{\Lambda e^{\mu t}}{\mu} + C$, as where c is a constant of integration
$N(t) = \frac{\Lambda}{\mu} + Ce^{-\mu t}$
By the initial condition at $t = 0$
$C = N(t) - \frac{\Lambda}{\mu}, \quad C = N(0) - \frac{\Lambda}{\mu}$
As time progresses, N(t) is such that;

$$\lim_{t\to\infty} N(t) \leq \lim_{t\to\infty}\left[\frac{\Lambda}{\mu} + \left(N(0) - \frac{\Lambda}{\mu}\right)e^{-\mu t}\right] = \frac{\Lambda}{\mu} \tag{3.5}$$

If $N(0) \leq \frac{\Lambda}{\mu}$, then $N(t) \leq \frac{\Lambda}{\mu}$. Thus, $\Re^5_+$ is a positive invariant set under the flow described by (3.2) so that no solution path leaves through any boundary of $\Re^5_+$. Hence, it is sufficient to consider the dynamics of the model in the domain $\Re^5_+$. In this region, the model can be considered to be mathematically and epidemically well-posed representing a physical problem.
This shows that the total population N(t), i.e., the sub-population $S_1(t), S_2(t), V(t), E(t)I(t), H(t), R(t)$ of the model are bounded and is a unique solution. Hence, it represents a physical problem.

3.2. Positivity and Boundedness of the Model Solution

Theorem 1

Let x, y be distinct points of a normed linear space $(X, \| \cdots \|)$ over $\Re$. Then the map $f : [0,1] \subseteq \Re \to (X, \| \cdot \|)$, such that $f(\lambda) = \lambda x + (1-\lambda)y$ is continuous on $[0,1]$

Proof :

Let $\lambda_0 \in [0,1]$. then $f(\lambda_0) = \lambda_0 x + (1-\lambda_0)y$ for any $\lambda \in [0,1]$,

$$\begin{aligned}\| f(\lambda) - f(\lambda_0) \| &= \| (\lambda - \lambda_0)x + (\lambda_0 - \lambda)y \| \\ &\leq | \lambda - \lambda_0 | (\| x \| + \| y \|).\end{aligned}$$

If $\varepsilon > 0$ is given, let $\delta = \frac{\varepsilon}{\|x\|+\|y\|}$. If $| \lambda - \lambda_0 | < \delta$, then the $\| f(\lambda) - f(\lambda_0) \| < \varepsilon$, . Therefore, f is continuous at λ_0. Since λ_0 is an arbitrary point in $[0,1]$, then f is continuous on $[0,1]$. Let X be a linear space over $\Re$. If x, y are distinct points of X, the set $\lambda x + (1-\lambda)y, 0 \leq \lambda \leq 1$.
Let;

$$f_1 = \Lambda\pi(1-p) - \beta_1 S_1 I - p_1 S_1 + w_1 V + \varphi E - \varpi S s_1 - \mu S_1 \quad (i)$$
$$f_2 = \Lambda(1-\pi) - (1-\lambda)\beta S_2 I - p_2 S_2 + w_2 V + \varphi E + \varrho S_1 - \mu S_2 \quad (ii)$$
$$f_3 = p_1 S_1 + p_2 S_2 - (w_1 + w_2)V - \mu V \quad (iii)$$
$$f_4 = \left(\beta_1 S_1 + (1-\lambda)\beta_2 S_2\right) I - (\eta_1 + eta_2 + \varphi_1 + \varphi_2 + \mu)E \quad (iv)$$
$$f_5 = (\eta_1 + eta_2)E - (\gamma + \delta + \mu)I \quad (v)$$
$$f_6 = \gamma I - \left(\varepsilon_1 + \varepsilon_2 + \mu + \tau\right) H \quad (vi)$$
$$f_7 = p\pi\Lambda + \left(\varepsilon_1 + \varepsilon_2 + \tau\right) H - \mu R \quad (vii)$$

Then,

$$\left.\begin{array}{l} |\frac{df_1}{dS_1}| = |\beta + p + \varpi + \mu| < \infty, \quad |\frac{df_1}{dS_2}| = |0| < \infty, \quad |\frac{df_1}{dV}| = |w_1| < \infty, |\frac{df_1}{dE}| = |\varphi_1| < \infty, \\ |\frac{df_1}{dI}| = |\beta| < \infty, |\frac{df_1}{dH}| = |0| < \infty, |\frac{df_1}{dR}| = |0| < \infty \\ |\frac{df_2}{dS_1}| = |\varphi_1| < \infty, \quad |\frac{df_2}{dS_2}| = |(1-\lambda) + \beta + \mu + p + 2| < \infty, \quad |\frac{df_2}{dV}| = |w_2| < \infty, |\frac{df_2}{dE}| = |\varphi_2| < \infty, \\ |\frac{df_2}{dI}| = |(1-\lambda)\beta| < \infty, |\frac{df_2}{dH}| = |0| < \infty, |\frac{df_2}{dR}| = |0| < \infty \\ |\frac{df_3}{dS_1}| = |p_1| < \infty, \quad |\frac{df_3}{dS_2}| = |p_2| < \infty, \quad |\frac{df_3}{dV}| = |w_1 + w_2 + \mu| < \infty, |\frac{df_3}{dE}| = |0| < \infty, \\ |\frac{df_3}{dI}| = |0| < \infty, |\frac{df_3}{dH}| = |0| < \infty, |\frac{df_3}{dR}| = |0| < \infty \end{array}\right\} \quad (3.6)$$

The bounded solution of the model exist in all the compartments respectively, therefore is well-posed.

3.3. Disease Free Equilibrium

From the above system of equations, at equilibrium when no outbreak of diphtheria is observed in the total population, $I(t), H(t), E(t) = 0$

$$\frac{dS_1}{dt} = \frac{dS_2}{dt} = \frac{dV}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dH}{dt} = \frac{dR}{dt} = 0$$

$$0 = \Lambda\pi(1-p) - \beta_1 S_1 I - p_1 S_1 + w_1 V + \varphi E - \varpi S s_1 - \mu S_1$$
$$0 = \Lambda(1-\pi) - (1-\lambda)\beta S_2 I - p_2 S_2 + w_2 V + \varphi E + \varrho S_1 - \mu S_2$$
$$0 = p_1 S_1 + p_2 S_2 - (w_1 + w_2)V - \mu V$$
$$0 = \left(\beta_1 S_1 + (1-\lambda)\beta_2 S_2\right) I - (\eta_1 + eta_2 + \varphi_1 + \varphi_2 + \mu)E$$
$$0 = (\eta_1 + eta_2)E - (\gamma + \delta + \mu)I$$
$$0 = \gamma I - \left(\varepsilon_1 + \varepsilon_2 + \mu + \tau\right) H$$
$$0 = p\pi\Lambda + \left(\varepsilon_1 + \varepsilon_2 + \tau\right) H - \mu R$$

Lastly, it is obtained from (iv)

$$0 = p\pi\Lambda + \Big(\varepsilon_1 + \varepsilon_2 + \tau\Big)H - \mu R$$

Hence, the disease-free equilibrium
E_1= $(S_{1o}, S_{2o}, V_o, E_o, I_o, H_o, R_o)$ where $S_o \neq 0$ as $I = 0$

$$E_1 = \left\{ S_{1o} = \tfrac{\Lambda\pi(1-p)+w_1w_1}{(\varphi_1+\mu)}, S_{2o} = \tfrac{\Lambda(1-\pi)+w_1\varphi_2}{(\gamma+\delta+\mu)}, V = \tfrac{p_1S_1+p_2S_2}{(\varphi_1+\varphi_2+\mu)}, E = 0, I = 0, H = 0, R = \tfrac{p\Lambda\pi}{\mu} \right\}$$

3.4. Endemic Equilibrium Point

Let $E_e = (S_1^*, S_2^*, V^*, E^*, I^*, H^*, R^*)$ as Endemic equilibrium where $I \neq 0$. Consider the system of equation (3.2) at equilibrium point as:

$0 = \Lambda\pi(1-p) - \beta_1 S_1 I - p_1 S_1 + w_1 V + \varphi E - \varpi S s_1 - \mu S_1$

$0 = \Lambda(1-\pi) - (1-\lambda)\beta S_2 I - p_2 S_2 + w_2 V + \varphi E + \varrho S_1 - \mu S_2$

$0 = p_1 S_1 + p_2 S_2 - (w_1 + w_2)V - \mu V$

$$0 = \Big(\beta_1 S_1 + (1-\lambda)\beta_2 S_2\Big)I - (\eta_1 + eta_2 + \varphi_1 + \varphi_2 + \mu)E$$

$0 = (\eta_1 + eta_2)E - (\gamma + \delta + \mu)I$

$$0 = \gamma I - \Big(\varepsilon_1 + \varepsilon_2 + \mu + \tau\Big)H$$

$$0 = p\pi\Lambda + \Big(\varepsilon_1 + \varepsilon_2 + \tau\Big)H - \mu R$$

$$S_1^* = \frac{\Lambda\pi(1-p) + [w_1 + \beta^2(\eta_1+\eta_2)\sqrt{\beta_1 + (1-\lambda)\beta_2}]}{\beta\varphi_1 + \varphi_1 + (\tau + \eta_2) + \mu} \tag{3.7}$$

from (ii),
$0 = \Lambda(1-\pi) - (1-\lambda)\beta S_2 I - p_2 S_2 + w_2 V + \varphi E + \varrho S_1 - \mu S_2$

$$S_2^* = \frac{\Lambda\beta(1-\pi)\sqrt{\beta_1 + (1-\lambda)\beta_2}}{\mu + \varphi_1 + \varphi_2 + [\eta_1 + \varphi_2 + \varepsilon_2 + \varepsilon_1]\gamma^2(1-\lambda)(\varepsilon_1 + \varepsilon_2)} \tag{3.8}$$

From (iii),
$0 = p_1 S_1 + p_2 S_2 - (w_1 + w_2)V - \mu V$

$$V^* = \frac{p_1 S_1^* + p_1 S_1^*}{(w_1 + w_2 + \mu)} \tag{3.9}$$

From (iv),

$$0 = \Big(\beta_1 S_1 + (1-\lambda)\beta_2 S_2\Big)I - (\eta_1 + eta_2 + \varphi_1 + \varphi_2 + \mu)E$$

$$E^* = \frac{\beta S_1^* + (1-\lambda)\beta_2 S_2^*}{\eta_1 + \eta_2 + \varphi_1 + \varphi_2 + \mu} \tag{3.10}$$

It is obtained from (v) that $0 = (\eta_1 + \eta_2)E - (\gamma + \delta + \mu)I$

$$I^* = \frac{\eta_1 + \eta_2 E^*}{\gamma + \delta + \mu} \tag{3.11}$$

Also obtained that $0 = \gamma I - \left(\varepsilon_1 + \varepsilon_2 + \mu + \tau\right)H$

$$H^* = \frac{\gamma(w_1 + w_[2] + \beta(1-\lambda))I^*}{\varepsilon_1 + \varepsilon_2 + \mu + \tau} \tag{3.12}$$

Lastly it is obtained from the model equations that $0 = p\pi\Lambda + \left((\varepsilon_1 + \varepsilon_2) + \tau\right)H - \mu R$

$$R^* = \frac{\lambda p\pi(\varepsilon_1 + \varepsilon_2) + \tau}{\mu(1-\pi) + \beta\tau\gamma(1-\lambda) + (\eta_1 + \eta_2)} \tag{3.13}$$

3.5. Basic Reproduction Number (R_o)

Lemma

The basic reproduction number denoted as R_0 . It is necessary to quantify the probability of new diphtheria infections resulting from a single carrier or sick person in a population without previous illnesses. We use the next-generation approach to create the system described in System of equation, focusing on the infectious classes E, I, and B. The F and V matrices, which represent the rates of new infections and transitions into and out of the infected compartment, respectively, are computed as part of this methodology. These matrices are obtained using a complex derivation from the equations. There are two disease states but only one way to create a new infection. Hence, exposed, infected enable the diphtheria spread in compartments of the model which are connected from system of equation (2.2). This denotes the number of secondary infections caused as a result of infected individuals in a population. Where $R_o = F \times V^{-1}$.To Obtain R_o from the the spread of diphtheria disease, it is deduced using next generation matrix where at equilibrium, non-infected sub-populations are disease-free. The transition and transmission matrices V and F are obtained from the partial derivatives of f and v to (E, I, H) evaluated at the disease-free equilibrium E_1

$$F_i = \left(\frac{\partial f_i(x_i)}{\partial x_j}\right) \qquad V_i = \left(\frac{\partial \nu_i(x_i)}{\partial x_j}\right) \quad i, j = 1, 2 \ldots 7$$

$$\mathbf{F} = \begin{pmatrix} (\beta_1 S_1 + (1-\lambda)\beta_2 S_2) \\ 0 \\ 0 \end{pmatrix} \qquad \mathbf{V} = \begin{pmatrix} (\eta_1 + \eta_2 + \varphi_1 + \varphi_2 + \mu)E(t) \\ -(\eta_1 + \eta_2)E(t) + (\gamma + \delta + \mu)I(t) \\ (\gamma)I(t) + [\varepsilon_1 + \varepsilon_2 + \mu + \tau]H(t) \end{pmatrix}$$

$$\mathbf{F} = \begin{pmatrix} 0 & (\beta_1 S_1 + (1-\lambda)\beta_2 S_2 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \qquad \mathbf{V} = \begin{pmatrix} (\eta_1 + \eta_2 + \varphi_1 + \varphi_2 + \mu) & 0 & 0 \\ -(\eta_1 + \eta_2) & (\gamma + \delta + \mu) & 0 \\ (\gamma) & \gamma & (\varepsilon_1 + \varepsilon_2 + \mu + \tau) \end{pmatrix}$$

$$\mathbf{F} = \begin{pmatrix} 0 & \frac{\beta_1 \Lambda\pi(1-p) + w_1\varepsilon_1 + \Lambda(1-\pi) + w_1\varphi_2}{(\varphi_1 + \mu)(\gamma + \delta + \mu)} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

The determinant and inverse of V is thus obtained from the adjunct of V thus
$|V| = (\eta_1 + \eta_2 + \varphi_1 + \varphi_2 + \mu)(\gamma + \delta + \mu)(\varepsilon_1 + \varepsilon_2 + \mu + \tau)$
As it is obtained that

$$\mathbf{V_c} = \begin{pmatrix} K_1 & -(\eta_1 + \eta_2 + \mu + \tau) & -\gamma(\eta_1 + \eta_2) \\ 0 & K_2 & 0 \\ 0 & 0 & K_3 \end{pmatrix}$$

where $K_1 = (\varepsilon_1 + \varepsilon_2 + \mu + \tau)(\gamma + \delta + \mu)$, $K_2 = (\eta_1 + \eta_2 + \varphi_1 + \varphi_2 + \mu)(\varepsilon_1 + \varepsilon_2 + \mu + \tau)$, $K_3 = (\eta_1 + \eta_2 + \varphi_1 + \varphi_2 + \mu)(\gamma + \delta + \mu)$

$$V^{-1} = \begin{pmatrix} \frac{1}{(\eta_1+\eta_2+\varphi_1+\varphi_2+\mu)} & -\frac{(\eta_1+\eta_2)}{(\eta_1+\eta_2+\varphi_1+\varphi_2+\mu)(\gamma+\delta+\mu)} & -\frac{(\eta_1+\eta_2)}{(\eta_1+\eta_2+\varphi_1+\varphi_2+\mu)(\gamma+\delta+\mu)(\eta_1+\eta_2+\mu+\tau)} \\ 0 & \frac{1}{(\gamma+\delta+\mu)} & 0 \\ 0 & 0 & \frac{1}{(\eta_1+\eta_2+\mu+\tau)} \end{pmatrix}$$

Given that, $R_o = F \times V^{-1}$ denoting the product of the matrices obtained

$$R_o = \begin{pmatrix} 0 & \frac{\beta_1\Lambda\pi(1-p)+w_1\varepsilon_1+\Lambda(1-\pi)+w_1\varphi_2}{(\varphi_1+\mu)(\gamma+\delta+\mu)} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \begin{pmatrix} A & B & C \\ 0 & \frac{1}{(\gamma+\delta+\mu)} & 0 \\ 0 & 0 & \frac{1}{(\eta_1+\eta_2+\mu+\tau)} \end{pmatrix}$$

Consider $A = \frac{1}{(\eta_1+\eta_2+\varphi_1+\varphi_2+\mu)}$, $B = -\frac{(\eta_1+\eta_2)}{(\eta_1+\eta_2+\varphi_1+\varphi_2+\mu)(\gamma+\delta+\mu)}$,
$C = -\frac{(\eta_1+\eta_2)}{(\eta_1+\eta_2+\varphi_1+\varphi_2+\mu)(\gamma+\delta+\mu)(\eta_1+\eta_2+\mu+\tau)}$

$$R_o = \frac{\beta_1\Lambda\pi(1-p) + w_1\varepsilon_1 + \Lambda(1-\pi) + w_1\varphi_2(\eta_1+\eta_2)}{(\varphi_1+\mu)(\gamma+\delta+\mu)} \tag{3.14}$$

3.6. Local Stability of Disease Free Equilibrium

Theorem 2

The disease-free equilibrium of the model for transmission of diphtheria is locally asymptotically stable if $R_o < 1$ and vice versa.

Proof:

The local stability of disease-free equilibrium at $S_o = \frac{\beta\Lambda}{(\mu+r+\omega)}$
The Jacobian matrix of the system (3.2) as obtained that $|J_{E_1} - \lambda_i I| = 0 i = 1, 2 \ldots 5$
Thus, the disease-free equilibrium is locally asymptotically stable if the eigenvalues λ_i, $i = 1, \ldots, 7$ of the matrix formed satisfies the condition.
The stability criterion of disease-free equilibrium, the general Jacobian matrix has been resolved for as;

$$\mathbf{J_{(E_1)}} = \begin{pmatrix} L & 0 & w_1 & \varphi_1 & -\beta_1 S_1 & 0 & 0 \\ w_1 & M & w_2 & \varphi_2 & N & 0 & 0 \\ p_1 & p_2 & N & 0 & 0 & 0 & 0 \\ \beta_1 I & (1-\lambda)\beta_2 I & 0 & P & [\beta_1 S_1 + (1-\lambda)\beta_2 S_2] & 0 & 0 \\ 0 & 0 & 0 & (\eta_1+\eta_2) & Q & 0 & 0 \\ 0 & 0 & 0 & 0 & \gamma & R & 0 \\ 0 & 0 & 0 & 0 & 0 & (\eta_1+\eta_2+\tau) & -\mu \end{pmatrix} \tag{3.15}$$

Then at disease free equilibrium,

$$\mathbf{J_{(E_1)}} = \begin{pmatrix} L & 0 & w_1 & \varphi_1 & -\beta_1 K_1 & 0 & 0 \\ w_1 & M & w_2 & \varphi_2 & -(1-\lambda)\beta_2 K_2 & 0 & 0 \\ p_1 & p_2 & N & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & P & [\beta_1 K_1 + (1-\lambda)\beta_2 K_2] & 0 & 0 \\ 0 & 0 & 0 & (\eta_1+\eta_2) & Q & 0 & 0 \\ 0 & 0 & 0 & 0 & \gamma & R & 0 \\ 0 & 0 & 0 & 0 & 0 & (\eta_1+\eta_2+\tau) & -\mu \end{pmatrix} \tag{3.16}$$

$$\begin{vmatrix} L & 0 & w_1 & \varphi_1 & -\beta_1 K_1 & 0 & 0 \\ w_1 & M & w_2 & \varphi_2 & -(1-\lambda)\beta_2 K_2 & 0 & 0 \\ p_1 & p_2 & N & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & P & [\beta_1 K_1 + (1-\lambda)\beta_2 K_2] & 0 & 0 \\ 0 & 0 & 0 & (\eta_1+\eta_2) & Q & 0 & 0 \\ 0 & 0 & 0 & 0 & \gamma & R & 0 \\ 0 & 0 & 0 & 0 & 0 & (\eta_1+\eta_2+\tau) & -\mu-\lambda_g \end{vmatrix} = 0 \tag{3.17}$$

As, $L = -(p_1 + w_1 + \mu) - \lambda_a$, $M = -[p_2 + \mu] - \lambda_b$
$N = -(w_1 + w_2 + \mu) - \lambda_c$, $P = -(\eta_1 + \eta_2 + \varphi_1 + \varphi_2 + \mu) - \lambda_d$
$Q = -(\gamma + \delta\mu) - \lambda_e$, $R = -(\eta_1 + \eta_2 + \mu + \tau) - \lambda_f$
As obtained from the previously examined determinant of respective eigenvalues, $\lambda_d = -(\eta_1 + \eta_2 + \varphi_1 + \varphi_2 + \mu)$. Similarly, the last of the eigenvalue is obtained as;

$$\begin{vmatrix} -(w_1+w_2+\mu)-\lambda_c & 0 & 0 & 0 & 0 \\ -(\eta_1+\eta_2+\varphi_1+\varphi_2+\mu)-\lambda_d & [\beta_1 K_1 + (1-\lambda)\beta_2 K_2] & 0 & 0 & \\ (\eta_1+\eta_2) & -(\varepsilon+\delta+\mu+\gamma)-\lambda_e & 0 & 0 & \\ 0 & \gamma & (\eta_1+\eta_2+\mu+\tau)-\lambda_f & 0 & \\ 0 & 0 & (\eta_1+\eta_2) & -\mu-\lambda_g & \end{vmatrix} = 0, \tag{3.18}$$

$$[-(w_1+w_2+\mu)-\lambda_c] \begin{vmatrix} X & [\beta_1 K_1 + (1-\lambda)\beta_2 K_2] & 0 & 0 \\ (\eta_1+\eta_2) & Y & 0 & 0 \\ 0 & \gamma & Z & 0 \\ 0 & 0 & (\eta_1+\eta_2) & -\mu-\lambda_g \end{vmatrix} = 0 \tag{3.19}$$

As $X = -(\eta_1 + \eta_2 + \varphi_1 + \varphi_2 + \mu) - \lambda_d$,
$Y = -(\varepsilon + \delta + \mu + \gamma) - \lambda_e$
$Z = (\eta_1 + \eta_2 + \mu + \tau) - \lambda_f$

Lastly, from the Jacobian matrix earlier stated,$\lambda_c = -(w_1 + w_2 + \mu)$, respectively;

$$\left.\begin{aligned} \lambda_a &= -(p_1 + w + \mu) < 0 \\ \lambda_b &= -(p_2\mu) < 0 \\ \lambda_c &= -(w_1 + w_2 + \mu) < 0 \\ \lambda_d &= -(\eta_1 + \eta_2 + \varphi_1 + \varphi_2 + \mu) < 0 \\ \lambda_e &= -(\gamma + \delta + \mu) < 0 \\ \lambda_f &= -(\varepsilon_1 + \varepsilon_2 + \tau + \mu) < 0 \\ \lambda_g &= -\mu < 0 \end{aligned}\right\} \tag{3.20}$$

Hence, they are negatively invariant in the region $\Re^5_+$, therefore they are locally asymptotically stable.

3.7. Local Stability of Endemic Equilibrium

Theorem 3

Suppose $X = x_n$ is a space of sequence of real number and we define

$$d(x, y) = \left(\sum_{i=1}^{n} \mid x_i \mid^p \right)^{\frac{1}{p}}, \quad p \geq 1 \tag{3.21}$$

X with the metric is called ξ_n^p space. If $\sum_{i=1}^{\infty} \mid x \mid^p < \infty$ or absolutely convergent and $d(x,y) = \left(\sum_{i=1}^{\infty} \mid x_i - y_i \mid^p \right)^{\frac{1}{p}}$, then X with this metric is called an ξ^p space. It can be checked that for each n;

Proof:

$$0 \leq x_1^2 + x_2^2 + x_3^2 + \ldots + x_n^2 \leq (\mid x_1 \mid + \mid x_2 \mid + \mid x_3 \mid + \ldots + \mid x_n \mid)^2$$

This will result to;

$$x_1^2 + x_2^2 \leq (\mid x_1 \mid + \mid x_2 \mid)^2$$

Therefore,

$$0 \leq (x_1^2 + x_2^2 + x_3^2 + \ldots + x_n^2)^{\frac{1}{2}} \leq \mid x_1 \mid + \mid x_2 \mid + \mid x_3 \mid + \ldots + \mid x_n \mid$$

If $\sum_{n=1}^{\infty} \mid x_n \mid$ converges, that is, $\sum_{n=1}^{\infty} \mid x_n \mid$ is absolutely convergent, then

$$0 \leq (x_1^2 + x_2^2 + x_3^2 + \ldots + x_n^2)^{\frac{1}{2}} \leq \mid x_1 \mid + \mid x_2 \mid + \mid x_3 \mid + \ldots + \mid x_n \mid = \sum_{n=1}^{\infty} \mid x_n \mid < \infty$$

Therefore,

$$0 \leq s_n = x_1^2 + x_2^2 + x_3^2 + \ldots + x_n^2 \leq \left[\sum_{n=1}^{\infty} \mid x_n \mid \right]^2 < \infty \tag{3.22}$$

The sequence x_n is monotone increasing and bounded above, it therefore converges. Thus $\sum_{n=1}^{\infty} x_n^2$ converges if $\sum_{n=1}^{\infty} x_n$ converges absolutely i.e if $x_n \in \xi^1$, then $x_n \in \xi^2$ where $\xi^1 \leq \xi^2$.
In case of ξ^1 denote the set of all sequences x_n of real numbers such that $\sum_{n=1}^{\infty} x_n$ is convergent absolutely i.e $\sum_{n=1}^{\infty} \mid x_n \mid < \infty$, whereas ξ^2 denote the set of all sequence x_n of real numbers such that $\sum_{n=1}^{\infty} x_n^2 < \infty$ converges. From the preceding, $x_n \in \xi^1 \longleftrightarrow x_n \in \xi^2$ i.e $\xi^1 \subseteq \xi^2$. Further, if $x_n = \frac{1}{n^{\frac{3}{4}}}$, then $\sum_{n=1}^{\infty} \mid x_n \mid$ diverges, and thus $x_n \notin \xi^1$. But $\sum_{n=1}^{\infty} x_n^2 = \sum_{n=1}^{\infty} \frac{1}{n^{\frac{3}{4}}}$ converges, implying that $x_n \in \xi^2$. We conclude then that $\xi^2 \subseteq \xi^1$ and thus $\xi^1 \neq \xi^2$. If x_n, y_n are sequences of real numbers, then

$$\sum_{i=1}^{n} (x_i = y_i)^2 \leq \sum_{i=1}^{n} x_i^2 + \sum_{i=1}^{n} y_i^2 + 2 \left[\sum_{i=1}^{n} x_i^2 \right]^{\frac{1}{2}} \left[\sum_{i=1}^{n} y_i^2 \right]^{\frac{1}{2}} \tag{3.23}$$

Therefore if $\sum_{i=1}^{\infty} x_i^2 < \infty$ and $\sum_{i=1}^{\infty} y_i^2 < \infty$ then $\sum_{i=1}^{\infty} (x_i - y_i)^2 < \infty$ for all n. The monotone increasing sequence $\left[\sum_{i=1}^{\infty} (x_i - y_i)^2 \right]$ is then bounded above and hence converges i.e $\sum_{i=1}^{\infty} (x_i - y_i)^2 < \infty$. Thus $(x_n - y_n) \in \xi^2$ if x_n, y_n are in ξ^2. The endemic equilibrium of the model outlining the transmission of diphtheria diseases is locally asymptotically stable if $R_o < 1$ and unstable otherwise. Let $S_1 = a + S_1^*, S_2 = b + S_1^*, V = c + V^*, E = d + E^*, I = e + I^*, H = f + H^*, R = f + R^*$ By

linearizing each state variable of the model formulation, it is obtained that,

$$\begin{aligned}
\frac{da}{dt} &= \Lambda\pi(1-p) - \beta_1(a+S^*)(e+I^*) - p_1(a+S^*) + w_1(c+V^*) + \varphi(d+E^*) - (\varpi-\mu)(a+S^*)\\
\frac{db}{dt} &= \Lambda(1-\pi) - (1-\lambda)\beta(b+S^*)(e+I^*) - p_2(b+S^*) + w_2(c+V^*) + \varphi(d+E^*) + \varrho(a+S^*) - \mu S_2^*\\
\frac{dc}{dt} &= p_1(a+S^*) + p_2(b+S^*) - (w_1+w_2)(c+V^*) - \mu(c+V^*)\\
\frac{dd}{dt} &= \left(\beta_1(a+S^*) + (1-\lambda)\beta_2(b+S^*)\right)(e+S^*) - (\eta_1 + eta_2 + \varphi_1 + \varphi_2 + \mu)(d+E^*)\\
\frac{de}{dt} &= (\eta_1 + eta_2)(d+E^*) - (\gamma+\delta+\mu)(e+I^*)\\
\frac{df}{dt} &= \gamma(e+I^*) - \left(\varepsilon_1\varepsilon_2 + \mu + \tau\right)(f+H^*)\\
\frac{dg}{dt} &= p\pi\Lambda + \left((\varepsilon_1\varepsilon_2) + \tau\right)(f+H^*) - \mu(g+R^*)
\end{aligned}$$

Hence,

$$\begin{aligned}
\frac{da}{dt} &= -\beta_1 ae - p_1 a + w_1 c + \varphi d - (\varpi-\mu)a + higherorder + non - linear + \ldots\\
\frac{db}{dt} &= -(1-\lambda)\beta be - p_2 b + w_2 c + \varphi d + \varrho a - \mu b\\
\frac{dc}{dt} &= p_1 a + p_2 b - (w_1+w_2)c - \mu c\\
\frac{dd}{dt} &= \left(\beta_1 a + (1-\lambda)\beta_2 b\right)e - (\eta_1 + eta_2 + \varphi_1 + \varphi_2 + \mu)d\\
\frac{de}{dt} &= (\eta_1 + eta_2)d - (\gamma+\delta+\mu)e\\
\frac{df}{dt} &= \gamma e - \left(\varepsilon_1\varepsilon_2 + \mu + \tau\right)f\\
\frac{dg}{dt} &= \left((\varepsilon_1\varepsilon_2) + \tau\right)f - \mu g
\end{aligned}$$

The Jacobian matrix of the system

$$\mathbf{J}_{(\mathbf{E}^*)} = \begin{pmatrix}
A & 0 & w_1 & \varepsilon_1 & -\beta_1 a & 0 & 0\\
\varrho & B & w_2 & \varepsilon_2 & (1-\lambda)\beta b & 0 & 0\\
p_2 & p_2 & C & 0 & 0 & 0 & 0\\
\beta_1 e & (1-\lambda)\beta_2 e & 0 & -D & (\beta_1 e + (1-\lambda)\beta_2 e) & 0 & 0\\
0 & 0 & 0 & (\eta_1 + eta_2) & E & 0 & 0\\
0 & 0 & 0 & 0 & \gamma & F & 0\\
0 & 0 & 0 & 0 & 0 & (\varepsilon_1\varepsilon_2 + \mu + \tau) & G
\end{pmatrix} \tag{3.24}$$

From the characteristic equation of $|J_(E^*) - \lambda I| = 0$

$$\mathbf{J}_{(\mathbf{E}^*)} = \begin{pmatrix} A-\lambda & 0 & w_1 & \varepsilon_1 & -\beta_1 a & 0 & 0 \\ \varrho & B-\lambda & w_2 & \varepsilon_2 & (1-\lambda)\beta b & 0 & 0 \\ p_2 & p_2 & C-\lambda & 0 & 0 & 0 & 0 \\ \beta_1 e & (1-\lambda)\beta_2 e & 0 & D-\lambda & 0 & 0 & \\ 0 & 0 & 0 & (\eta_1 + eta_2) & E-\lambda & 0 & 0 \\ 0 & 0 & 0 & 0 & \gamma & F-\lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & (\varepsilon_1\varepsilon_2 + \mu + \tau) & G-\lambda \end{pmatrix} \tag{3.25}$$

Respective eigenvalues become

$$\left\{ A-\lambda)(B-\lambda)(D-\lambda)(E-\lambda)(F-\lambda)(G-\lambda) = 0 \right\}$$

where A =$-[\beta_1 e + p_1 + (\varpi - \mu)]$, B = $-[(1-\lambda)\beta e + p_2 + \mu]$, C = $-(w_1 + w_2 + \mu)$,
D = $-(\eta_1 + eta_2 + \varphi_1 + \varphi_2 + \mu)$
E = $-(\gamma + \delta + \mu)$ F = $-(\varepsilon_1\varepsilon_2 + \mu + \tau)$
G = $-\mu$
It is obtained that,

$$(A-\lambda)(B-\lambda)(C-\lambda)(D-\lambda)(E-\lambda)(F-\lambda)(G-\lambda) = 0 \tag{3.26}$$

Hence, the trace of $J_(E_e) < 0$. Thus, the Jacobian matrix $J_(E_e) < 0$ has eigenvalues that contain negative real roots parts. Therefore, we conclude that the endemic equilibrium point is locally asymptotically stable. Therefore, they are locally asymptotically stable.

3.8. Global Stability of Disease Free Equilibrium

Considering the use of the Lyapunov algorithm for the system of equation (2.2), which is rapidly tilting to the variance of zero neighborhood is said to be asymptotically stable as $t > 0$. Hence, taken $v(t, S_1, S_2, V, E, I, H, R) = C_1 I_1 + C_2 I_2 + C_3 I_3$

$$\frac{dV}{dt} = C_1 I_1' + C_2 I_2' + C_3 I_3' \tag{3.27}$$

$$\begin{aligned} \frac{dV}{dt} &= C_1\left\{\left(\beta_1 S_1 + (1-\lambda)\beta_2 S_2\right) I - (\eta_1 + eta_2 + \varphi_1 + \varphi_2 + \mu)E\right\} + C_2\left((\eta_1 + eta_2)E - (\gamma + \delta + \mu)I\right) \\ &\quad + C_3\left\{\gamma I - \left(\varepsilon_1\varepsilon_2 + \mu + \tau\right)H\right\} \\ \frac{dV}{dt} &\leq C_1\left(\beta_1 S_1 + (1-\lambda)\beta_2 S_2\right) IC_1 - (\eta_1 + eta_2 + \varphi_1 + \varphi_2 + \mu)EC_1 + C_2(\eta_1 + eta_2)E - (\gamma + \delta + \mu)IC_2 \\ &\quad + C_3\gamma I - \left(\varepsilon_1\varepsilon_2 + \mu + \tau\right)HC_3 \\ \frac{dV}{dt} &\leq C_1\left\{\left(\beta_1 S_1 + (1-\lambda)\beta_2 S_2\right) I_2 - (\eta_1 + eta_2 + \varphi_1 + \varphi_2 + \mu)I_1\right\} + C_2\left((\eta_1 + eta_2)I_1 - (\gamma + \delta + \mu)I_2\right) \\ &\quad + C_3\left\{\gamma I_2 - \left(\varepsilon_1\varepsilon_2 + \mu + \tau\right)I_3\right\} \end{aligned}$$

Subjecting $C_1 < C_2 < C_3$ as $C_3 \leq 0$

$$\begin{aligned}\frac{dV}{dt} \leq & \left\{C_2(\eta_1+\eta_2) - C_1(\varphi_1+\varphi_2+\varepsilon_1+\varepsilon_2+\mu)\right\}I_1 + \left\{C_1\beta S_1 + (1-\lambda)\beta_2 S_2 + C_3\gamma - C_2(\gamma+\delta+\mu)\right\}I_2 \\ & -C_3(\eta_1+\eta_2+\mu+\tau)I_3\end{aligned}$$

Let

$$\begin{aligned}C_2 &= \frac{\beta_1\Lambda\pi(1-p) + w_1\varepsilon_1 + \Lambda(1-\pi) + w_1\varphi_2}{(\varphi_1+\mu)(\gamma+\delta+\mu)}, \\ C_1 &= \frac{1}{(\eta_1+\eta_2+\varphi_1+\varphi_2+\mu)}\end{aligned}$$

$$\begin{aligned}\frac{dV}{dt} \leq & \left(\frac{\beta_1\Lambda\pi(1-p) + w_1\varepsilon_1 + \Lambda(1-\pi) + w_1\varphi_2(\eta_1+\eta_2)}{(\varphi_1+\mu)(\gamma+\delta+\mu)} - \frac{1}{(\eta_1+\eta_2+\varphi_1+\varphi_2+\mu)}\right) \\ & + \left(\frac{\beta_1 S_1 + (1-\lambda)S_2\beta_2}{(\eta_1+\eta_2+\varphi_1+\varphi_2+\mu)(\gamma+\delta+\mu)} - \frac{\beta_1 S_1 + (1-\lambda)S_2\beta_2}{(\eta_1+\eta_2+\varphi_1+\varphi_2+\mu)(\gamma+\delta+\mu)}\right)\end{aligned}$$

$$\frac{dV}{dt} \leq (R_0 - 1) \tag{3.28}$$

It is crucial to keep in mind that when at $\frac{dV}{dt} = 0$. Equation (1) can be substituted to find that, according to LaSalle's invariance principle, is globally asymptotically stable whenever $R_0 > 1$

3.9. Global Stability of Endemic Equilibrium

Theorem 4

The model of has no periodic orbits

Proof:

Employing the Dulac's criterion as adopted by (Ahmed *et al* 2021). Let $X = (S_1, S_2, V, E, I, H, R)$. Define the Dulac's function as $G = \frac{1}{SE}$

$$\begin{aligned}G\frac{dS_1}{dt} &= \frac{1}{SE}\left\{\Lambda\pi(1-p) - \beta_1 S_1 I - p_1 S_1 + w_1 V + \varphi E - \varpi S_1 - \mu S_1\right\} \\ &= \frac{\Lambda\pi(1-p)}{SE} - \frac{\beta_1}{I} - \frac{p_1}{I} + \frac{w_1 V}{SE} + \frac{\varphi}{S} - \frac{\varpi}{E} - \frac{\mu}{E} \\ G\frac{dS_2}{dt} &= \frac{1}{SE}\left\{\Lambda(1-\pi) - (1-\lambda)\beta S_2 I - p_2 S_2 + w_2 V + \varphi E + \varrho S_1 - \mu S_2\right\} \\ &= \frac{\Lambda(1-\pi) - (1-\lambda)\beta}{E} - \frac{p_2}{E} + \frac{w_2 V}{SE} + \frac{\varphi}{S} + \frac{\varrho}{E}\end{aligned}$$

$$
\begin{aligned}
G\frac{dV}{dt} &= \frac{1}{SE}\left\{p_1S_1 + p_2S_2 - (w_1+w_2)V - \mu V\right\} \\
&= \frac{p_1}{E} + \frac{p_2}{E} - \frac{(w_1+w_2+\mu}{SE} \\
G\frac{dE}{dt} &= \frac{1}{SE}\left\{\beta_1S_1 + (1-\lambda)\beta_2S_2\right)I - (\eta_1+\eta_2+\varphi_1+\varphi_2+\mu)E\right\} \\
&= \frac{\beta_1S_1+(1-\lambda)\beta_2}{SE} + \frac{(r+\varepsilon)I}{SE} - \frac{(\eta_1+\eta_2+\varphi_1+\varphi_2+\mu)}{S} \\
G\frac{dI}{dt} &= \frac{1}{SE}\left\{(\eta_1+eta_2)E - (\gamma+\delta+\mu)I\right\} \\
&= \frac{(\eta_1+eta_2)}{E} - \frac{(\gamma+\delta+\mu)I}{SE} \\
G\frac{dH}{dt} &= \frac{1}{SE}\left\{\gamma I - \left(\varepsilon_1+\varepsilon_2+\mu+\tau\right)H\right\} \\
&= \frac{\gamma I}{SE} - \frac{(\varepsilon_1+\varepsilon_2+\mu+\tau)}{SE} \\
G\frac{dR}{dt} &= \frac{1}{SE}\left\{p\pi\Lambda + \left((\varepsilon_1\varepsilon_2)+\tau\right)H - \mu R\right\} \\
&= \frac{p\pi\Lambda}{SE} + \frac{(\varepsilon_1\varepsilon_2)+\tau}{SE} - \frac{\mu}{SE}
\end{aligned}
$$

$\frac{d(GX)}{dt}$ is obtained as follows:

$$
\begin{aligned}
\frac{d(GX)}{dt} = \frac{\partial}{\partial S_1}\left\{G\frac{dS_1}{dt}\right\} + \frac{\partial}{\partial S_2}\left\{G\frac{dS_2}{dt}\right\} + \frac{\partial}{\partial V}\left\{G\frac{dV}{dt}\right\} + \frac{\partial}{\partial E}\left\{G\frac{dE}{dt}\right\} + \frac{\partial}{\partial I}\left\{G\frac{dI}{dt}\right\} \\
+\frac{\partial}{\partial H}\left\{G\frac{dH}{dt}\right\} + \frac{\partial}{\partial R}\left\{G\frac{dR}{dt}\right\}
\end{aligned}
$$

$$
\begin{aligned}
\frac{d(GX)}{dt} &= \frac{\partial}{\partial S_1}\left\{\frac{\Lambda\pi(1-p)}{SE} - \frac{(\tau+\eta_1+\eta_2)+\beta I+\mu}{E} + \frac{\varphi_1}{SE}\right\} + \frac{\partial}{\partial S_2}\left\{\frac{p_2(1-\pi)}{E} - \frac{(\delta+\gamma+\mu)}{S}\right\} \\
&+ \frac{\partial}{\partial V}\left\{\frac{\eta_1+\eta_2}{E} + \frac{(\varepsilon_1+\varepsilon_2+\eta_1+\mu)}{S} - \frac{(\varepsilon+\delta+\mu)I}{SE}\right\} \\
&+ \frac{\partial}{\partial E}\left\{\frac{\beta_1+(1-\lambda)\beta_2}{E} + \frac{(\eta_1+\eta_2+\varepsilon_2)I}{SE} - \frac{\mu}{SE}\right\} \\
&+ \frac{\partial}{\partial I}\left\{\frac{\gamma}{SE}\right\} + \frac{\partial}{\partial H}\left\{\frac{\gamma+\delta+\mu}{SE}\right\} + \frac{\partial}{\partial R}\left\{\frac{-\mu}{SE}\right\} \\
\frac{d(GX)}{dt} &= \left\{-\frac{\mu}{SE}\right\} + \left\{-\frac{(\delta+\mu+\tau)}{SE}\right\} + \left\{-\frac{(\eta_1+\eta_2+\mu)}{SE}\right\} + \left\{-\frac{(\varepsilon_1+\varepsilon_2+\mu)}{SE}\right\} + \left\{-\frac{\mu}{SE}\right\} \\
\frac{d(GX)}{dt} &= -\left\{\frac{\mu}{SE} + \frac{(\mu+\tau+\delta)}{SE} + \frac{(\eta_1+\eta_2+\mu)}{SE} + \frac{\mu}{SE}\right\} \\
\frac{d(GX)}{dt} &= -\left\{\frac{2\mu+(\mu+\tau+\delta+)+(\varphi_1+\varphi_2+\mu+\eta)^2-(\delta+\gamma+\mu)}{SE}\right\} < 0
\end{aligned}
$$

This implies that the system has no closed orbit. Epidemiologically, the non-existence of a periodic orbit implies that there are fluctuations in the number of infections, which makes it difficult to allocate resources for the control of the disease.

3.10. Sensitivity Analysis

The test for the sensitivity of R_o is to all the parameters in R_o. The normalized forward sensitivity index is defined as shown below

$$\frac{\partial R_o}{\partial P} = \frac{\partial R_o}{\partial P} \times \frac{P}{\partial R_o} \tag{3.29}$$

Hence,

$$\begin{aligned} \frac{\partial R_o}{\partial \Lambda} \times \frac{\Lambda}{\partial R_o} &= 1.000 \\ \frac{\partial R_o}{\partial \pi} \times \frac{\pi}{\partial R_o} &= 1.000 \\ \frac{\partial R_o}{\partial \beta} \times \frac{\beta}{\partial R_o} &= 1.02101 \\ \frac{\partial R_o}{\partial \delta} \times \frac{\delta}{\partial R_o} &= 1.002190 \\ \frac{\partial R_o}{\partial p_1} \times \frac{p_1}{\partial R_o} &= -1.4200 \end{aligned} \tag{3.30}$$

respectively on each of the sensitive parameters of R_0, result obtained as depicted below. Table (3.2)

Table 3.2: Sensitivity Analysis and Indices of the Disease Threshold

Parameters	Indices
Λ	1.000
π	1.00
β	1.02101
δ	1.002190
p_1	1.4200
μ	-1.010
τ	1.0
ε_1	1.210
ε_2	1.10
φ_1	0.1
φ_2	0.1
η_1	0.133
η_2	0.154
p_2	0.813
w_1	0.2
w_2	0.25

shows that the sensitivity indices of $\beta, \omega, \varepsilon$ are positive, while μ is negative. As the sensitivity indices depend on the values of the other parameters, changes in those values will affect the sensitivity indices. Based on the table, we can conclude that parameters β and ε_1,ε_2,p_1 and p_2 are the most sensitive to the basic reproduction number R_0 in equation (1) of the diphtheria model. Specifically, increasing the value of ε_1,ε_2, will result in a 70.25% increase in R_0, while increasing the value of τ will lead to a 82.76%decrease in R_0.

4. Numerical simulation of the model with Laplace Adomian Decomposition method

4.1. Origin of Laplace Adomian Decomposition Method

The Laplace-Adomian Decomposition Method (LADM) is a powerful mathematical technique used for solving differential equations, particularly nonlinear ones. It combines the classical Laplace transform method with the Adomian polynomials to provide accurate and efficient solutions. The method originated from the work of two mathematicians: Pierre-Simon Laplace and George Adomian. The Laplace-Adomian Decomposition Method emerged as a hybrid technique, combining Laplace transforms with Adomian polynomials. This integration improved the efficiency and accuracy of solving nonlinear differential equations, making it applicable to a wide range of scientific and engineering problems. Over the years, the Laplace-Adomian Decomposition Method has gained popularity due to its simplicity, versatility, and effectiveness in solving nonlinear problems. It has been applied across various fields, including physics, engineering, biology, and finance, and continues to be an active area of research and application in mathematical modeling and analysis. As a crucial component of this study, we will undertake a numerical simulation to examine the effects of vaccination on the temporal behavior of the state variables within our proposed model. To accomplish this, it is necessary to acquire the model solution. However, given the absence of an exact solution for the model, we will employ distinct numerical approximation schemes of the Laplace Adomian Decomposition Method to determine an accurate and precise approximate solution for the model. This chosen solution will subsequently be employed to conduct the desired numerical simulation.

4.2. Laplace Adomian Decomposition Method Algorithm

Consider the system of ordinary differential equations of the first order as follows;

$$\left.\begin{aligned} y_1' &= g_1(x, y_1, y_2, y_3, y_4, \dots y_n) \\ y_2'' &= g_2(x, y_1, y_2, y_3, y_4, \dots y_n) \\ y_3''' &= g_3(x, y_1, y_2, y_3, y_4, \dots y_n) \\ &\vdots \\ y_3^n &= g_n(x, y_1, y_2, y_3, y_4, \dots y_n) \end{aligned}\right\} \tag{4.31}$$

Where each represents the derivative of the first order of one of the unknown functions as a mapping depending on the independent variable x, and n unknown functions $g_1, g_2, g_3, g_4, \dots g_n)$, Since every ordinary differential equation of n order can be written as a system consisting of n ordinary differential equation of order one, we restrict our study to a system of differential equation of the first order.

4.3. Analysis of Adomian's Decomposition Method

Consider the differential equation in the general form

$$Ly(x) + Ry(x) + Ny(x) = f(x)$$

where $L$is the linear operator of the highest-order derivative which is assumed to be invertible easily, R is also a linear operator of order less than L, and $Ny(x)$ indicates the non-linear term and f is the source term. Thus applying the inverse operator L^{-1} to the above equation to obtain

$$y(x) = g_0 + L^{-1}\left(f(x) - Ry(x) - Ny(x)\right)$$

where g_0 is the solution of the homogeneous equation,

$$Ly(x) = 0$$

The constants of integration involved in the solution of homogeneous are to be determined by the initial conditions, according to the problem, whether it is an initial value problem or boundary value problem. According to ADM, the solution of the unknown function $y(x)$ can be expressed by an infinite series of the form

$$y(x) = \sum_{n=0}^{\infty} y_n x$$

and the non-linear term can be decomposed by the infinite series of the form

$$Ny(x) = \sum_{n=0}^{\infty} A_n$$

and,$A_n s$ are called Adomian's Polynomials, which can be determined by the algorithm. By substituting into the above equation to obtain;

$$\sum_{n=0}^{\infty} y_n(x) = g_0 + L^{-1}\left[f(x) - R\sum_{n=0}^{\infty} y_n x - \sum_{n=0}^{\infty} A_n\right]$$

where the components $(y_0, y_1, y_2, y_3, y_4, \ldots y_n)$ are determined by the recursive relation $y_0 = g_0$

$$y_{k+1} = -L^{-1}(Ry_k) - L^{-1}(A_k), \quad k \geq 0$$

Given the model solution via the application of (LADM) for equation (1) and taking the Laplace transform of both sides of the above equation

$$\begin{aligned}
L\left[\frac{dS_1}{dt}\right] &= L[\Lambda\pi(1-p)] - L\left[\beta_1 S_1 I - p_1 S_1 + w_1 V + \varphi E - \varpi S_1 - \mu S_1\right] \\
L\left[\frac{dS_2}{dt}\right] &= L[\Lambda(1-\pi)] - L\left[(1-\lambda)\beta S_2 I - p_2 S_2 + w_2 V + \varphi E + \varrho S_1 - \mu S_2\right] \\
L\left[\frac{dV}{dt}\right] &= L[p_1 S_1 + p_2 S_2] - L\left[(w_1 + w_2)V - \mu V\right] \\
L\left[\frac{dE}{dt}\right] &= L\left[\left(\beta_1 S_1 + (1-\lambda)\beta_2 S_2\right) I\right] - L\left[(\eta_1 + \eta_2 + \varphi_1 + \varphi_2 + \mu)E\right] \\
L\left[\frac{dI}{dt}\right] &= L[(\eta_1 + eta_2)E] - L\left[(\gamma + \delta + \mu)I\right] \\
L\left[\frac{dH}{dt}\right] &= L[\gamma I] - L\left[\left(\varepsilon_1 + \varepsilon_2 + \mu + \tau\right)H\right] \\
L\left[\frac{dR}{dt}\right] &= L[p\pi\Lambda] + L\left[\left(\varepsilon_1 + \varepsilon_2 + \tau\right)H - \mu R\right]
\end{aligned}$$

Following the definition of Laplace Transform of derivatives

$$L\left[f'(t)\right] = mf(t) - f(0) \tag{4.32}$$

Substituting into the above equation yields

$$\begin{aligned}
mL\Big[S_1(t)\Big] &= S_1(0) + \tfrac{\Lambda\pi(1-p)}{m} + \beta_1 L[S_1 I - p_1 S_1] + w_1 L[V] + L\Big[\varphi E - \varpi S_1 - \mu S_1\Big] \\
mL\Big[S_2(t)\Big] &= S_2(0) + \tfrac{\Lambda(1-\pi)}{m} + (1-\lambda)\beta L[S_2 I - p_2 S_2 + w_2 V] + L\Big[\varphi E + \varrho S_1 - \mu S_2\Big] \\
mL\Big[V(t)\Big] &= V(o) + \tfrac{(p_1+p_2)}{m} L[S_1 + S_2] + L\Big[(w_1+w_2)V - \mu V\Big] \\
mL\Big[E(t)\Big] &= E(0) + \tfrac{(1-\lambda)\beta_2}{m} + L\Big[\Big(\beta_1 S_1 + S_2\Big) I\Big] - L\Big[(\eta_1+\eta_2+\varphi_1+\varphi_2+\mu)E\Big] \\
mL\Big[I(t)\Big] &= I(0) + \tfrac{(\eta_1+eta_2)}{m} + L\Big[(\gamma+\delta+\mu)I\Big] \\
mL\Big[H(t)\Big] &= H(0) + \tfrac{\varepsilon_1+\varepsilon_2}{m} + \gamma L[I] - L\Big[\Big(+\mu+\tau\Big)H\Big] \\
mL\Big[R(t)\Big] &= R(0) + \tfrac{p\pi\Lambda}{m} + L\Big[\Big(\varepsilon_1+\varepsilon_2+\tau\Big)H - \mu R\Big]
\end{aligned} \tag{4.33}$$

where $S_1(0) = s_0, S_2(0) = s_0, V(0) = v_0, E(0) = e_0, I(0) = i_0, H(0) = h_0, R(0) = r_0$ Letting the non-linear terms $SI = A$ and substitutes by taking the inverse Laplace Transform of both sides,

$$\left.\begin{aligned}
S_1(t) &= \tfrac{s_0}{m} + L^{-1}\left\{\tfrac{\Lambda\pi(1-p)}{m^2} + \tfrac{1}{m}\beta_1 L[S_1 I - p_1 S_1] + w_1 L[V] + \tfrac{1}{m}L[\varphi E - \varpi S_1 - \mu S_1]\right\} \\
S_2(t) &= \tfrac{s_0}{m} + L^{-1}\left\{\tfrac{\Lambda(1-\pi)}{m^2} + \tfrac{1}{dm}(1-\lambda)\beta L[S_2 I - p_2 S_2 + w_2 V] + \tfrac{1}{m}L[\varphi E + \varrho S_1 - \mu S_2]\right\} \\
V(t) &= \tfrac{v_0}{m} + L^{-1}\left\{\tfrac{(p_1+p_2)}{m^0} L[S_1 + S_2] + \tfrac{1}{m}L[(w_1+w_2)V - \mu V]\right\} \\
E(t) &= \tfrac{v_0}{m} + L^{-1}\left\{\tfrac{(1-\lambda)\beta_2}{m^0} + \tfrac{1}{m}L[\Big(\beta_1 S_1 + S_2\Big) I] - L[(\eta_1+\eta_2+\varphi_1+\varphi_2+\mu)E]\right\} \\
I(t) &= \tfrac{i_0}{m} + L^{-1}\left\{\tfrac{(\eta_1+eta_2)}{m^2} + \tfrac{1}{m}L[(\gamma+\delta+\mu)I]\right\} \\
H(t) &= \tfrac{h_0}{m} + L^{-1}\left\{\tfrac{\varepsilon_1+\varepsilon_2}{m^2} + \tfrac{1}{m}\gamma L[I] - \tfrac{1}{m}L[\Big(+\mu+\tau\Big)H]\right\} \\
R(t) &= \tfrac{r_0}{m} + L^{-1}\left\{\tfrac{p\pi\Lambda}{m^2} + \tfrac{1}{m}L[\Big(\varepsilon_1+\varepsilon_2+\tau\Big)H - \mu R]\right\}
\end{aligned}\right\} \tag{4.34}$$

And the nonlinear term is given the Adomian polynomials if;
$A_0 = S_0 I_0, A_1 = S_0 I_1, A_2 = S_0 I_2, A_3 = S_0 I_3, A_4 = S_0 I_4 \ldots$.
Thus if

$$S_1(t) = \sum_{k=0}^{\infty} S_n(t), S_2(t) = \sum_{k=0}^{\infty} S_n(t), V(t) = \sum_{k=0}^{\infty} V_n(t), E(t) = \sum_{k=0}^{\infty} E_n(t), I(t) = \sum_{k=0}^{\infty} I_n(t) \ldots$$

$$
\begin{aligned}
\sum_{k=0}^{\infty} S_n(t) &= \frac{s_0}{m} + L^{-1}\left\{\frac{\Lambda\pi(1-p)}{m^2} + \frac{1}{m}\beta_1 L[\sum_{k=0}^{\infty} S_n(t)I - p_1 S_1] + w_1 L[V] + \frac{1}{m}L[\varphi E - \varpi \sum_{k=0}^{\infty} S_n(t)S_1 - \mu S_1]\right\} \\
\sum_{k=0}^{\infty} S_n(t) &= \frac{s_0}{m} + L^{-1}\left\{\frac{\Lambda(1-\pi)}{m^2} + \frac{1}{dm}(1-\lambda)\beta L[S_2 I - p_2 \sum_{k=0}^{\infty} S_n(t)S_2 + w_2 V] + \frac{1}{m}L[\varphi E\right. \\
&+ \left. \varrho \sum_{k=0}^{\infty} S_n(t)S_1 - \mu S_2]\right\} \\
\sum_{k=0}^{\infty} V_n(t) &= \frac{v_0}{m} + L^{-1}\left\{\frac{(p_1+p_2)}{m^0}L[S_1+S_2] + \frac{1}{m}L[(w_1+w_2)\sum_{k=0}^{\infty} V_n(t) - \mu \sum_{k=0}^{\infty} V_n(t)]\right\} \\
\sum_{k=0}^{\infty} E_n(t) &= \frac{v_0}{m} + L^{-1}\left\{\frac{(1-\lambda)\beta_2}{m^0} + \frac{1}{m}L[\left(\beta_1 S_1 + S_2\right)I] - L[\sum_{k=0}^{\infty} V_n(t)(\eta_1+\eta_2+\varphi_1+\varphi_2+\mu)E]\right\} \\
\sum_{k=0}^{\infty} I_n(t) &= \frac{i_0}{m} + L^{-1}\left\{\frac{(\eta_1 + eta_2)}{m^2} + \frac{1}{m}L[\sum_{k=0}^{\infty} V_n(t)(\gamma+\delta+\mu)I]\right\} \\
\sum_{k=0}^{\infty} H_n(t) &= \frac{h_0}{m} + L^{-1}\left\{\frac{\varepsilon_1+\varepsilon_2}{m^2} + \frac{1}{m}\gamma L[\sum_{k=0}^{\infty} V_n(t)] - \frac{1}{m}L[\left(\mu+\tau\right)]\right\} \\
\sum_{k=0}^{\infty} R_n(t) &= \frac{r_0}{m} + L^{-1}\left\{\frac{p\pi\Lambda}{m^2} + \frac{1}{m}L[\left(\sum_{k=0}^{\infty} V_n(t)(\varepsilon_1+\varepsilon_2) + \tau\right) - \mu R]\right\}
\end{aligned}
$$

The initial approximations of each class are given by;
$S_1(0) = s_0, S_2(0) = s_0, V(0) = v_0, E(0) = e_0, I(0) = i_0, H(0) = h_0, R(0) = r_0$
Now, comparing the coefficients $n = 1$. Using the recurrence relations, the following are obtained For

$$
\begin{aligned}
S_1(t) &= (\Lambda\pi(1-p) - \beta_1 s_0 I - p_1 s_0)t^2 + w_1 v_0 + \frac{1}{2}(\varphi e_0 - \varpi s_0 - \mu s_0)t + s_0 \\
S_2(t) &= ((\Lambda(1-\pi) - (1-\lambda)\beta s_0 i_0)t^2 - p_2 S_2 + w_2 v_0 + \varphi s_0 e_0 + \varrho s_0 - \mu s_0 s_0)t + s_0 \\
V(t) &= (p_1 s_0 + p_2 s_0 - (w_1+w_2)v_0 - \mu s_0)t + v_0 \\
E(t) &= \left(\beta_1 s_0 + ((1-\lambda)\beta_2 s_0\right)i_0)t^2 - (\eta_1+\eta_2+\frac{1}{2}\varphi_1+\varphi_2+\mu)s_0 + e_0 \\
I(t) &= ((\eta_1 + eta_2)e_0 s_0)t^2 - \frac{1}{6}(\gamma+\delta+\mu)e_0 s_0 + i_0 \\
H(t) &= \gamma i_0 - \lambda\left(\frac{1}{3}(\varepsilon_1+\varepsilon_2+\mu+\tau)\right)s_0 i_0 + h_0 \\
R(t) &= (p\pi\Lambda)r_0 i_0)t^2 + \frac{1}{2}\left((\varepsilon_1+\varepsilon_2)+\tau\right)e_0 h_0 - \frac{1}{6}\mu i_0 s_0 + r_0
\end{aligned}
$$

second iteraive terms of the numerical simulation at $n = 2$ is;

$$
\begin{aligned}
S_2(t) &= \frac{1}{3}(\Lambda\pi(1-p) - \beta_1 s_0 i_0 - p_1 s_0)t^2 + \frac{1}{3}w_1 v_0 + \frac{1}{4}(\varphi e_0 s_0 - \varpi i_0 e_0 - \frac{1}{4}(\tau\mu(1-\pi))s_0)t \\
&+ (\beta\lambda(1-\lambda)w_1 e_0 s_0)t^2 + \frac{1}{3}(\delta+\mu+\gamma)t \\
S_2(t) &= \frac{1}{3}((\Lambda\beta(1-\pi)) - \frac{1}{2}(1-\lambda)\beta s_0 i_0)t^3 - (p_2 S_2 + w_2 v_0 + \varphi s_0 e_0) - \frac{1}{3}(\varrho s_0 - \mu s_0 s_0))t^2 \\
&+ s_0(\eta_1+\eta_2+\varphi_1+\varphi_2)t + (\varepsilon_1+\varepsilon_2)t
\end{aligned}
$$

$$
\begin{aligned}
V_2(t) &= -\frac{1}{3}(p_1 s_0 + p_2 s_0) - ((w_1 + w_2) v_0 s_0 t^2 - \mu s_0)t + v_0(1-\lambda)t \\
E_2(t) &= \left(\beta_1 s_0 - \frac{1}{2}((1-\lambda)\beta_2 s_0\right) i_0)t^3 - \frac{1}{4}(\eta_1 + \eta_2 t^2 + \frac{1}{2}\varphi_1 + \varphi_2 + \mu)s_0 + e_0 + (\frac{(\delta+\gamma+\eta_2)}{(\varepsilon_1+\varepsilon_2+\mu)})t \\
I_2(t) &= -\frac{1}{3}((\eta_1 + eta_2)e_0 s_0)t^3 - \frac{1}{6}(\gamma+\delta+\mu)e_0 s_0 + i_0 + -\frac{1}{2}(1-\lambda)\beta s_0 i_0)t^2 - ((1-p)\Lambda\beta + (1-\pi))t \\
H_2(t) &= \frac{1}{2}\gamma i_0 e_0 - \frac{1}{3})\lambda(1-\pi)\beta_1 + \beta_2)t^2 + \left(\frac{1}{3}(\varepsilon_1+\varepsilon_2+\mu+\tau)\right)s_0 i_0 + ()w_1 + w_2) i_0 S_0 - \frac{1}{2}\left(\varepsilon_1+\varepsilon_2+\mu\right)t \\
R_2(t) &= -\frac{1}{6}(\frac{(1-\pi)\beta_1 + \beta_2 i_0 s_0}{(\delta+\gamma+\mu)})t^3 - \frac{1}{2}(p\pi\Lambda)r_0 i_0)t^2 + \frac{1}{2}\left((\varepsilon_1\varepsilon_2) + \tau\right)e_0 h_0 - \frac{1}{6}\mu i_0 s_0 r_0 + (w_2)v_0 s_0)t
\end{aligned}
$$

and so on. This can be further till desired number of iterations are obtained. Thus, the obtained raw solution to each model compartment is obtained as:

$$S_1(t) = \sum_{k=0}^{3} s_n(t), S_2(t) = \sum_{k=0}^{3} s_n(t), V(t) = \sum_{k=0}^{3} v_n(t), E(t) = \sum_{k=0}^{3} e_n(t), I(t) = \sum_{k=0}^{3} i_n(t) \ldots$$

$$
\begin{aligned}
S_1(t) &= 500.012 - 30.02t + 1.13005t^2 - 0.50709t^3 - 3.500963 \times 10^{-2}t^4 - 5.17t^5 \times 10^{-2} \\
S_2(t) &= 635.6747 - 983.32746t + 1.63525t^2 - 0.576569t^3 - -5.175597t^4 \times 10^{-2} \\
V(t) &= 1625.2 - 12230.42t^2 - 6.47254t^3 - 9.71645256t^{-1} \times 10^{-2} \\
E(t) &= 32.32 - 0.53257461t - 1.0653t^3 - 0.50709635t^3 - 3.676423 \times 10^{-1}t^3 - 5.1797597t^2 \times 10^{-2} \\
I(t) &= 1736.0863 + 3765.87t^2 + 73553.2t^3 + 0.5327^4 + 9.832663 \times 10^3 t^4 - 7.73562t^4 \times 10^3 \\
H(t) &= 8.2346 - 8.3427t + 7.237638t^3 - 0.50709t^2 - 9.12343 \times 10^2 t^2 - 78836t^2 \times 10^2 \\
R(t) &= 4008.126 - 2.836t + 9.2352557t^2 - 0.8236t^3 - 4.253437 \times 10^3 t^2 - 5367t^4 \times 10^2
\end{aligned}
$$

The outcomes of the results are evaluated to obtain the dynamic variations of the population of each state variable over 10 Months and graphical illustration of simulation result is depicted thus;

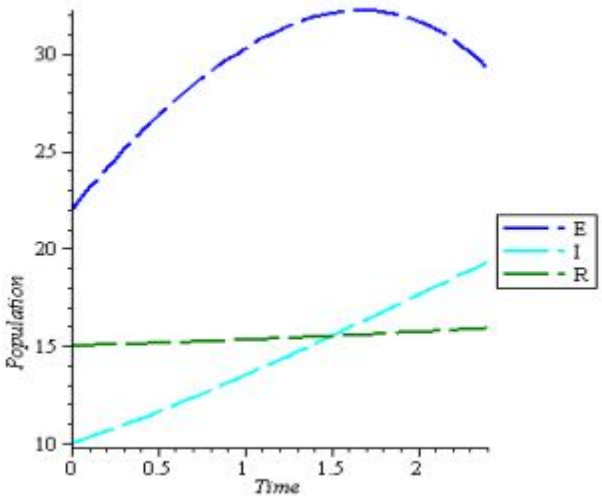

Figure 4.2: Effect of avoiding close contact β with infected individuals in children to adult population.

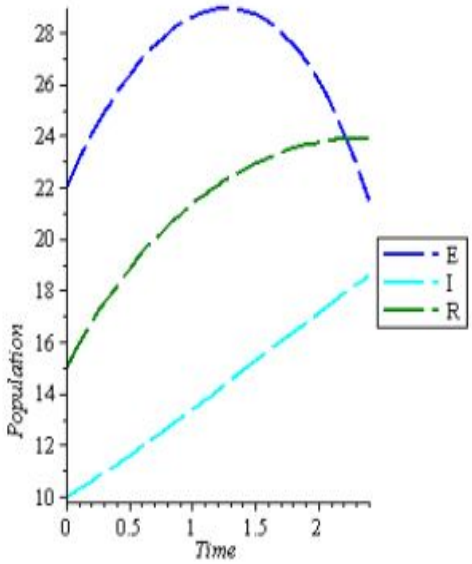

Figure 4.3: The effect of early diagnosis and treatment ε_1 to ε_2 in children to adults of population.

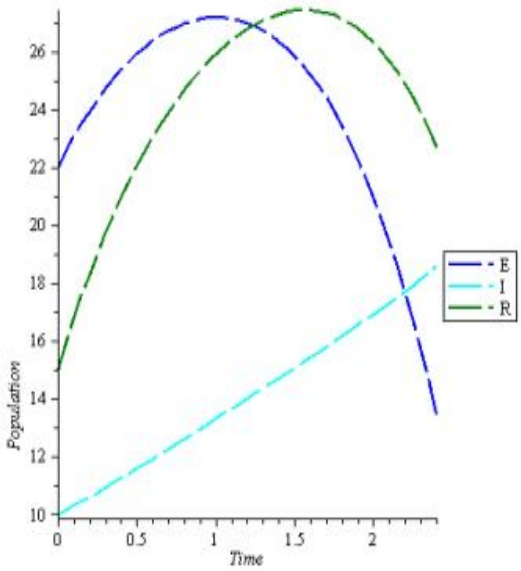

Figure 4.4: Timely vaccination w_1, w_2 of the infected population.

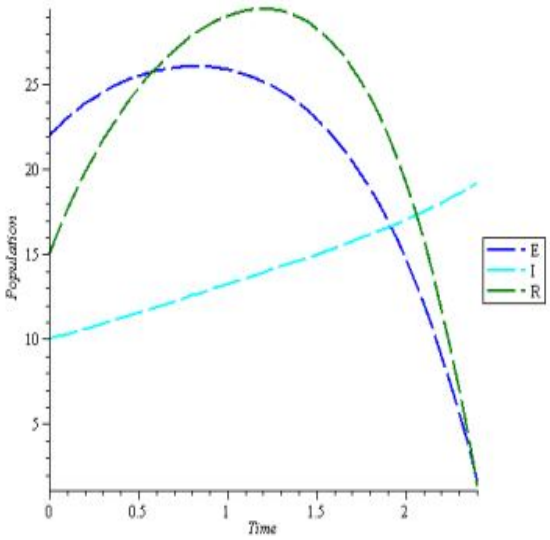

Figure 4.5: Effect of early diagnosis and treatment on the exposed population.

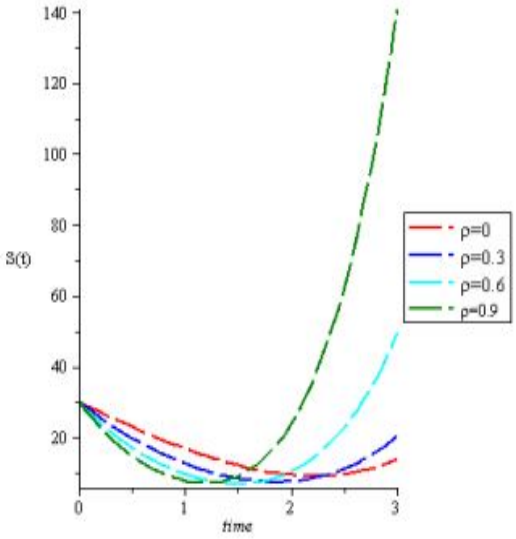

Figure 4.6: Effect of booster doses on recovered children and adult Population.

Result and discussion of simulation iterations

It is obtained that fig. 4.2 Shows the effect of avoiding close contact β with Infected Individuals in the Children to Adult Population. will bring about a fall in the spread of the disease between the two sub-populations. Also from fig. 4.3 Shows the effect of early diagnosis and Treatment ε_1 to ε_2 in Children to adults of population. of diphtheria as the level of the spread reduces in the population. Fig. 4.4 also depicts that the level of timely vaccination w_1, w_2 of the infected population on the set

of vulnerable populations reduces as it brings about a rapid spread in the diphtheria outbreak and vice versa. Fig. 4.5 shows the effect of early Diagnosis and Treatment on the Exposed Population will help in reducing the spread of diphtheria as this brings about a fall in the infected curve. While fig 4.6 shows the effect of booster Doses on recovered Children and Adult populations will increase the population of susceptible individuals as a prominent tool in the control of the disease.

Conclusion

This manuscript has provided an in-depth examination of diphtheria transmission dynamics and the critical role of vaccination in its control among age-structured populations. By developing and analyzing a novel (SVEIHR) model, coupled with rigorous mathematical analyses and numerical simulations, the study has highlighted the nuanced interplay between vaccination coverage, age demographics, and disease dynamics. Investigation into disease-free equilibrium, stability analyses, and sensitivity analysis of targeted parameters has yielded valuable insights into optimal vaccination strategies for diverse settings and age groups. The findings emphasize the importance of vaccination in mitigating diphtheria's public health impact. Moreover, treatment emerges as a prominent control measure for eradication. We recommend that health professionals prioritize treatment practices to combat the spread of diphtheria. By advancing our understanding of transmission dynamics and informing targeted interventions, this thesis contributes to global efforts to combat vaccine-preventable diseases and achieve sustainable health outcomes. Equitable access to vaccination and evidence-based interventions remain crucial for success.

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Declaration of interest

The authors declare that there is no conflict of interest.

References

[1] A. S. Akanda, A. S. jutla, M. Alam, H. de Magny, A. K. Siddique, R. B. Sack, A. Huq, R. R. Colwell, and S. Islam, "Hydroclimatic influences on seasonal and spatial cholera transmission cycles: Implications for public health intervention in the bengal delta," *Water Resources Research*, vol. 5, pp. 33–37, 2011. View online.

[2] M. K. Kolawole, K. A. Odeyemi, A. O. Oladapo, and K. A. Bashiru, "Dynamical analysis and control strategies for capturing the spread of covid-19. tanzania journal of science," *Tanzania Journal of Science*, vol. 48, pp. 680–690, 2022. View online.

[3] M. K. Kolawole, M. O. Olayiwola, H. O. Adekunle, and K. A. Odeyemi, "Conceptual analysis of the combined effects of vaccination, therapeutic actions, and human subjection to physical constraint in reducing the prevalence of covid-19 using the homotopy perturbation method," *Beni-Suef University Journal of Basic and Applied Sciences*, vol. 12, 2023. View online.

[4] A. A. Akindele, V. O. Omoloye, R. O. Ojedele, E. I. Obeagu, and A. O. Hassan, "Antibiotic resistance and virulence genes of pseudomonas aeruginosa isolates in southwest, nigeria," *Journal of Experimental Sciences*, pp. 115–129, 2023. View online.

[5] M. Arifin, "Recommendation of diphtheria antitoxin to different suspects during the diphtheria outbreaks, lumajang district in 2018," *The Indonesian Juornal of Public Health*, vol. 15, p. 356, 2020. View online.

[6] A. Aslan and A. Uzunkaya, "Critical health psychologists' call to recognition and action: Critical health psychology, participatory action research and the situation in turkey," *Klinik Psikoloji Dergisi*, 2021. View online.

[7] M. Kolawole, A. Oluwarotimi, K. Odeyemi, and A. Popoola, "Analysis of corona-virus mathematical model in asymptomatic and symptomatic cases with vaccine using homotopy perturbation method," *Journal of Applied Computer Science and Mathematics*, vol. 17, pp. 20–27, 2023. View online.

[8] P. Bakhache, V. Yewale, R. Parikh, G. Clancey, G. Spickernell, V. Berlaimont, and E. Turriani, "Diphtheria-tetanus-pertussis (dtp) vaccination: understanding the perspectives and expectations of parents and healthcare professionals in france and india," *Human Vaccines and Immunotherapeutics*, vol. 18, 2021. View online.

[9] M. Kolawole, A. Oluwarotimi, K. Odeyemi, and A. Popoola, "Analysis of corona-virus mathematical model in asymptomatic and symptomatic cases with vaccine using homotopy perturbation method," *Journal of Applied Computer Science and Mathematics*, vol. 17, pp. 20–27, 2023. View online.

[10] M. K. Kolawole, A. O. Popoola, K. A. Odeyemi, and K. A. Bashiru, "An approximate solution of fractional order epidemic model of typhoid using the homotopy perturbation method," *UNIOSUN Journal of Engineering and Environmental Sciences*, vol. 5, 2023. View online.

[11] F. Bozkurt, Y. Ali, T. Abdeljawad, K. Adem, and Q. Al Mdallal, "A fractional order model of covid-19 considering the fear effect of the media and social networks on the community," *Chaos Solitons Fractals*, 2021. View online.

[12] F. P. Havers, M. A. Skoff, T. H.and Rench, M. Epperson, G. Rajam, J. Schiffer, S. Hariri, L. S. Swaim, C. J. Baker, and C. M. Healy, "Maternal tetanus toxoid, reduced diphtheria toxoid, and acellular pertussis vaccination during pregnancy: Impact on infant anti-pertussis antibody concentrations by maternal pertussis priming series," *Clinical Infectious Diseases*, vol. 76, pp. 1087–1093, 2022. View online.

[13] A. Hoefer, D. Pampaka, S. Herrera-León, S. Peiró, S. Varona, N. López-Perea, J. Masa-Calles, and L. Herrera-León, "Molecular and epidemiological characterization of toxigenic and nontoxigenic corynebacterium diphtheriae, corynebacterium belfantii, corynebacterium rouxii, and corynebacterium ulcerans isolates identified in spain from 2014 to 2019," *Journal of Clinical Microbiology*, vol. 59, 2021. View online.

[14] F. Hofmann, "Environmentally and occupationally acquired diseases in adolescents and adulthood: Control of diphtheria, tetanus, pertussis, and poliomyelitis through regular booster vaccinations european perspective," *Reviews on Environmental Health*, vol. 20, 2019. View online.

[15] K. Chawla, A. Gupta, M. Gujral, and A. Singh, "Diphtheria resurgence in india: A case study from south karnataka," *Journal of Family Medicine and Primary Care*, vol. 9, p. 5776, 2020. View online.

[16] A. Ejimele and A. Fatusi, "Emergency preparedness in health institutions in nigeria: Current situation and framework for action," *Proceedings of the Nigerian Academy of Science*, vol. 14. View online.

[17] S. D. Elek, "The plate virulence test for diphtheria," *Journal of Clinical Pathology*, vol. 2, pp. 250–258, 2011. View online.

[18] M. M. EL-Hazmi, "Late-onset prosthetic valve endocarditis caused by nontoxigenic corynebacterium diphtheriae," *The Journal of Infection in Developing Countries*, vol. 9, pp. 905–909, 2015. View online.
[19] C. Hailu, G. Fisseha, and A. Gebreyesus, "Determinants of measles vaccination dropout among 12-23 months aged children in pastoralist community of afar, ethiopia," *BMC Infectious Diseases*, vol. 22, 2022. View online.
[20] F. P. Havers, P. L. Moro, P. Hunter, S. Hariri, and H. Bernstein, "Use of tetanus toxoid, reduced diphtheria toxoid, and acellular pertussis vaccines: Updated recommendations of the advisory committee on immunization practices — united states, 2019," *MMWR: Morbidity and Mortality Weekly Report*, vol. 69, pp. 77–83, 2020. View online.
[21] Y. Boucher, "Sustained local diversity of vibrio cholerae o1 biotypes in a previously cholera-free country," *MBio*, vol. 7, 2016. View online.
[22] O. Ibrahim, I. Lawal, B. Mohammed, S. Abdullahi, S. Bello, A. Issa, A. Sanda, B. Suleiman, and M. Ibrahim, "Diphtheria outbreak during covid-19 pandemic in katsina, north-western nigeria: Epidemiological characteristics and predictors of death," *Nigerian Journal of Basic and Clinical Sciences*, vol. 19, p. 59, 2022. View online.
[23] V. Keshav, N. Potgieter, and T. Barnard, "Detection of vibrio cholerae o1 in animal stools collected in rural areas of the limpopo province," *Water SA*, vol. 36, p. 167, 2019. View online.
[24] E. I. Komarovskaya and V. Perelygyna, "Assay of diphtheria vaccine potency by intradermal challenge test," *Epidemiology and Vaccinal Prevention*, vol. 22, pp. 12–27, 2023. View online.
[25] N. Lawal, S. A. Abdullahi, and D. S. Abolude, "Physicochemical characteristics and fish abundance and diversity of mairua reservoir water, funtua, katsina state, north-western nigeria," *Journal of Applied Sciences and Environmental Management*, vol. 27, pp. 125–132, 2023. View online.
[26] B. Liu, "Analysis on the current situation, problems and potential solutions for the chinese precious metals futures market," *BCP Business and Management*, vol. 38, pp. 1239–1244. View online.
[27] V. G. Melnikov, A. Berger, A. Dangel, and A. Sing, "Lateral flow immunoassay-based laboratory algorithm for rapid diagnosis of diphtheria," *Open Research Europe*, vol. 3, 2023. View online.

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