Earthline Journal of Mathematical Sciences
E-ISSN: 2581-8147
Volume 14, Number 3, 2024, Pages 391-404
https://doi.org/10.34198/ejms.14324.391404

Age Structured Deterministic Model of Diphtheria Infection

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Abstract

Age-structured mathematical model of diphtheria infection has been formulated with specific epidemiological classes such as S_1, susceptible infant at time t (0-1years), S_2, susceptible school children population at time t, V, vaccination population at time t, E, exposed population at time t, I_1, asymptomatic infection population at time t, I_2, symptomatic infection population at time t, I_D, detected infectious humans at time t (asymptomatic and symptomatic) population through testing, R, recovered population at time t. It was established through theorems and proofs that the model is epidemiologically meaningful, and that all its state variables are positive (non-negative) at time $t > 0$ in the domain $\wp$, and that the domain $\wp$ is indeed bounded. Using the next generation matrix, the reproduction ratio R_b of the system was determined. Using dynamical system theory, it was established that the system is locally stable. A matrix-theoretic method was used in the construction of an appropriate Lyapunov function for the global stability analysis of the formulated model, and also established that the system is globally asymptotically stable if $R_b \leq 1$ and unstable otherwise.

1. Introduction

Diphtheria is a bacterial infectious disease that can lead to severe complications such as respiratory failure, heart problems and even deaths if it is not detected early. This infection that mostly affects the throat and the nose can be prevented by vaccination.

Received: January 1, 2024; Revised & Accepted: February 5, 2024; Published: February 16, 2024

2020 Mathematics Subject Classification: 92D25.

Keywords and phrases: Lyapunov function, global stability, diphtheria infection, reproduction ratio, vaccination.

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Case-fatality occurs only in places where there is poor sanitation condition and inadequate vaccination coverage as a result of low resources [1,2,3]

There is a report of outbreak of diphtheria infection in Nigeria with confirmed cases in Kano and Lagos but that of Yobe and Osun is under observation. The report indicates that case fatality rate is at 18.5% occurring in areas that were not adequately covered in terms of vaccination [4]. This report has emphasised the role of vaccination and herd immunity, impact of vaccination coverage and sensitisation (media coverage) and effect of complacency in controlling the spread of infectious disease, as demonstrated by [5,6,7]. In countries where there is low income, inadequate vaccination coverage and poor sanitation conditions case fatality rate among untreated and unvaccinated population is high. Diphtheria vaccination coverage estimate in Nigeria have been in the decline. Because of the outbreak of COV-19 and unavailability of vaccines, vaccination rate declined from 86% in 2019 to 81% in 2021 and now 56%, which grossly suboptimal in Nigeria [4].

The control of this infection is based mainly on prevention, by ensuring high population immunity. Secondary infection should be prevented by the rapid investigation of close contacts to ensure prompt treatment of those infected. To avoid a rise in the number of confirmed cases and related death and guarantee community protection, the national vaccination coverage of 80-86% should be sustained [8].

To effectively contend the spread of diphtheria, different control measures, such as isolation of patient, maintenance of one meter between patients, keeping patient care areas with good ventilation, the use mask that is medically prepared and cover any wound/lesions on patient's body by patient who may have to move out of the isolation areas, population subgroup such as young children under five years of age, school children, elderly who are at greater risk and have close contact with diphtheria infection and health workers should highly prioritized with treatment and vaccination, epidemiological surveillance ensuring early detection of diphtheria outbreak, administering antitoxin to neutralize the toxin and antibiotics to kill the bacteria, reducing complication and mortality should be implemented [8].

Mathematical models over the years have become a veritable tool in the hand of mathematicians and epidemiologist in studying, understanding, describing and analysing the dynamics of infectious diseases [9,10,11,12,13,14,15,16]. Apart from understanding the dynamics of infections, mathematical models such as [17,18,19] have given good insight in real life problems, it analysis have been of immense benefit in decision making.

Some mathematical models such as [20,21] has been formulated for the transmission dynamics of diphtheria, which correctly describe the decreasing trend of diphtheria cases and predicted the diphtheria outbreak patterns that may occur in the near future. Finds parameters that gives contribution endemic and non-endemic conditions

The work under consideration is aimed at presenting an age-structure deterministic mathematical model of the transmission dynamics of diphtheria infection in Nigeria in to investigate the impact of some of the control measures recommended by WHO on the spread of diphtheria infection, in targeting the infants, school children and the elderly who are mostly prone to the diphtheria infection, through contact tracing to mitigate and reduce the spread of diphtheria in the population.

The research is expected to discuss the feasibility of using age-structured deterministic model of the transmission dynamics diphtheria to assess the development of control measures adopted in 2023 diphtheria epidemic outbreak in Nigeria.

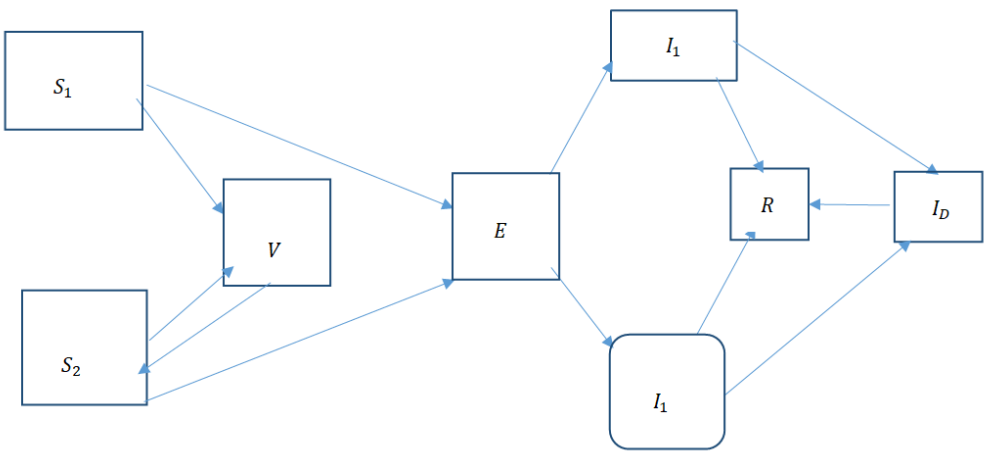

Figure 1: Schematic flowchart of transmission dynamics of diphtheria.

2. Model Formulation

2.1. Assumptions

1. The control of diphtheria is based on primary prevention of disease by ensuring high population immunity of the infant ((0-1 year) and school children by vaccination.

2. Isolation of detected cases (that is confirmed cases are not allowed to interact with the population freely.
3. Epidemiological surveillance ensuring early detection through contact tracing is carried out.
4. Secondary prevention spread by the rapid investigation of close contacts to ensure prompt treatment of the infected.
5. The total population of human at time t under consideration denoted by N_h is split into mutually exclusive sub-population of S_1, susceptible infant at time t (0-1 years), S_2, susceptible school children population at time t, V, vaccination population at time t, E, exposed population at time t, I_1, asymptomatic infection population at time t, I_2, symptomatic infection populationat time t, R, recovered population at time t. I_D, detected infectious humans at time t (asymptomatic and symptomatic) population through testing,

$$H_h = S_1 + S_2 + V + E + I_1 + I_2 + R + I_D.$$

2.2. State variables

N_h −The total population of human at time t under consideration,

S_1 − Susceptible infant at time t (0-1years),

S_2 − Susceptible school children population at time t,

V −Vaccination population at time t,

E − Exposed population at time t,

I_1 − Asymptomatic infection population at time t,

I_2 − Symptomatic infection population at time t,

I_D − Detected infectious humans at time t (asymptomatic and symptomatic) population through testing,

R − Recovered population at time t.

2.3. Parameters

σ_1 − progress rate from exposed to I_1, asymptomatic infection population

$\sigma_2 -$ progress rate efrom exposed to I_2, symptomatic infection population

$\delta -$ fraction of new infection that are I_1, asymptomatic

$1 - \delta -$ fraction of new infection that are I_2, symptomatic

$\beta_1 -$ effective transmission rate from S_1

$\beta_2 -$ effective transmission rate from S_2

$\tau_1 -$ vaccination coverage for S_1

$\tau_2 -$ vaccination coverage for S_2

$\varepsilon -$ vaccine efficacy

$\rho -$ maturity rate from S_1 to S_2

$b -$ per capita birth rate of humans

$\varepsilon\tau_1 -$ immunization rate in S_1

$\varepsilon\tau_2 -$ immunization rate in S_2

$\theta_1 -$ detection rate (via contact tracing) for S_1

$\theta_2 -$ detection rate (via contact tracing) for S_2

$\eta -$ death due to infection

$\alpha -$ natural death rate

$\gamma_1 -$ the recovery rate of I_1

$\gamma_2 -$ the recovery rate of I_2

$\gamma_3 -$ the recovery rate of I_D

$$\lambda_1(t) = \frac{\beta_1(\sigma_1 I_1 + \sigma_2 I_2)}{N_h - I_D}$$

$$\lambda_2(t) = \frac{\beta_2(\sigma_1 I_1 + \sigma_2 I_2)}{N_h - I_D}$$

2.4. Model equations

Using the above described state variables and parameters together with the schematic diagram in Figure 1, the model of the diphtheria infection transmission dynamics results

in the following system of deterministic non-linear first order differential equations

$$\frac{dS_1}{dt} = bN_h - \frac{\beta_1(\sigma_1 I_1 + \sigma_2 I_2)}{N_h - I_D} S_1 - \varepsilon\tau_1 S_1 - \rho S_1 - \alpha S_1 \tag{2.1}$$

$$\frac{dS_2}{dt} = \rho S_1 - \frac{\beta_2(\sigma_1 I_1 + \sigma_2 I_2)}{N_h - I_D} S_2 - \varepsilon\tau_2 S_2 - \alpha S_2 \tag{2.2}$$

$$\frac{dV}{dt} = \varepsilon\tau_1 S_1 + \varepsilon\tau_2 S_2 - \alpha V \tag{2.3}$$

$$\frac{dE}{dt} = \frac{\beta_1(\sigma_1 I_1 + \sigma_2 I_2)}{N_h - I_D} S_1 + \frac{\beta_2(\sigma_1 I_1 + \sigma_2 I_2)}{N_h - I_D} S_2 - \sigma_1\delta E - \sigma_2(1-\delta)E - \alpha E \tag{2.4}$$

$$\frac{dI_1}{dt} = \sigma_1\delta E - \gamma_1 I_1 - \theta_1 I_1 - \alpha I_1 - \eta I_1 \tag{2.5}$$

$$\frac{dI_2}{dt} = \sigma_2(1-\delta)E - \gamma_2 I_2 - \theta_2 I_2 - \alpha I_2 - \eta I_2 \tag{2.6}$$

$$\frac{dR}{dt} = \gamma_1 I_1 + \gamma_2 I_2 + \gamma_3 I_D - \alpha R \tag{2.7}$$

$$\frac{dI_D}{dt} = \theta_1 I_1 + \theta_2 I_2 - \gamma_3 I_D - \alpha I_D - \eta I_D \tag{2.8}$$

3. Analysis of the Model

This section seeks to study qualitatively the dynamical properties of the Diphtheria model (2.1)-(2.8).

3.1. Basic properties of the model

Before the model (2.1)-(2.8) is epidemiologically meaningful, it is important to show that all its state variables are positive (non-negative) at all time $t > 0$ in the domain $\wp$, and that the domain $\wp$ is indeed bounded. This shall be done through the following theorems and proves.

Theorem 3.1. *Let the initial data of the model* (2.1)-(2.8) *be given as* $S_1(0) \geq 0$, $S_2(0) \geq 0, V(0) \geq 0, E(0) \geq 0, I_1(0) \geq 0, I_2(0) \geq 0, R(0) \geq 0, I_D(0) \geq 0$. *Then the solutions* $S_1, S_2, V, E, I_1, I_2, R, I_D$ *of the model* (2.1)-(2.8) *are non-negative* $\forall\ t > 0$.

Proof. Let $t_1 = sup\{t > 0: S_1 > 0,\ S_2 > 0,\ V > 0,\ E > 0,\ I_1 > 0,\ I_2 > 0,\ R > 0,\ I_D > 0\ \in [0, t]\}$. Thus, $t_1 > 0$.

We have from (2.1)

$$\frac{dS_1}{dt} = bN_h - \frac{\beta_1(\sigma_1 I_1 + \sigma_2 I_2)}{N_h - I_D} S_1 - \varepsilon\tau_1 S_1 - \rho S_1 - \alpha S_1$$

$$\frac{dS_1}{dt} = bN_h - (\lambda S_1 + \varepsilon\tau_1 + \rho + \alpha) S_1 \quad \text{where } \lambda_1(t) = \frac{\beta_1(\sigma_1 I_1 + \sigma_2 I_2)}{N_h - I_D}$$

which can be re-written as

$$\frac{dS_1}{dt} + (\lambda S_1 + \varepsilon\tau_1 + \rho + \alpha) S_1 = bN_h$$

so that

$$S_1(t) = e^{-(\lambda S_1 + \varepsilon\tau_1 + \rho + \alpha)t} \int_0^t bN_h \, e^{-(\lambda S_1 + \varepsilon\tau_1 + \rho + \alpha)s} ds > 0.$$

Similarly, it can be shown that: $S_2 > 0, V > 0, E > 0, I_1 > 0, I_2 > 0, R > 0, I_D > 0$.

Theorem 3.2. *The domain* $\wp = \{(S_1, S_2, V, E, I_1, I_2, R, I_D) \in \Re_+^8 : N_h \leq N_h(0)\}$ *is positively-invariant for the model* (2.1)-(2.8) *and attracts all positive solutions of the model.*

Proof. Adding all the equation of the model (2.1)-(2.8), we have

$$\frac{dN_h}{dt} = bN_h - \alpha S_1 - \alpha S_2 - \omega V - \alpha V - \alpha E - \alpha I_1 - \eta I_1 - \alpha I_2 - \eta I_2 - \alpha I_D - \eta I_D - \alpha R$$

$\varphi = \min\{\alpha. \omega, \eta\}$

$$\frac{dN_h}{dt} \leq bN_h - \varphi N_h = \vartheta N_h, \qquad \vartheta = b - \varphi$$

$$\frac{dN_h}{dt} \leq \vartheta N_h,$$

$$\int \frac{dN_h}{N_h} \leq \int \vartheta dt$$

$$N_h(t) = N_h(0) e^{\vartheta t}$$

$N_h(t)$ approaches $N_h(0)$ as $t \to \infty \; \forall \; b < \varphi$.

Hence, the domain $\wp$ attracts all solutions in $\Re_+^8$.

3.2. Local asymptotic stability of the disease free equilibrium of the model (2.1)-(2.8)

By setting the right hand side of the model equation (2.1)-(2.8) to zero (0), the DFE of (2.1)-(2.8) is obtained as follows

$$\chi_0 = (S_1^0, S_2^0, V^0, E^0, I_1^0, I_2^0, R^0, I_D^0)$$
$$= (N_h(0), S_1(0), 0, 0, 0, 0, 0, 0).$$

The local stability equilibrium of the DFE χ_0 can be established by the use of next generation matrix method on the system (2.1)-(2.8) as in [1,2]. Hence, the transmission matrix F and the transition matrix of the system (2.1)-(2.8) is given respectively as follows:

$$F(E, I_1, I_2, I_D) = \begin{pmatrix} 0 & \sigma_1(\beta_1+\beta_2) & \sigma_2(\beta_1+\beta_2) & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

$$V(E, I_1, I_2, I_D) = \begin{pmatrix} \sigma_1\delta + \sigma_2(1-\delta) + \alpha & 0 & 0 & 0 \\ -\sigma_1\delta & \gamma_1 + \theta_1 + \alpha + \eta & 0 & 0 \\ -\sigma_2(1-\delta) & 0 & \gamma_2 + \theta_2 + \alpha + \eta & 0 \\ 0 & -\theta_1 & -\theta_2 & \gamma_1 + \alpha + \eta \end{pmatrix}$$

From [1,2], it follows that the basic reproduction number denoted by R_b is obtained as

$$R_b = \frac{\sigma_1^2\delta(\beta_1+\beta_2)}{((\sigma_1\delta + \sigma_2(1-\delta) + \alpha)(\theta_1+\gamma_1+\alpha+\eta)} + \frac{\sigma_2^2(1-\delta)(\beta_1+\beta_2)}{((\sigma_1\delta + \sigma_2(1-\delta) + \alpha)(\theta_2+\gamma_2+\alpha+\eta)}$$

or

$$R_b = (\beta_1+\beta_2)\left\{\frac{\sigma_1^2\delta}{((\sigma_1\delta + \sigma_2(1-\delta) + \alpha)(\theta_1+\gamma_1+\alpha+\eta)} + \frac{\sigma_2^2(1-\delta)}{((\sigma_1\delta + \sigma_2(1-\delta) + \alpha)(\theta_2+\gamma_2+\alpha+\eta)}\right\}.$$

In the light of the above, the following theorem with proof is hereby claimed.

Theorem 3.3. *The DFE,* χ_0, *of the model* (2.1)-(2.8) *is locally asymptotically* stable (LAS) *if* $R_b < 1$, *and unstable if* $R_b > 1$.

Proof. The local stability of the model (2.1)-(2.8) is analysed by the Jacobian matrix of the system (2.1)-(2.8) evaluated at the disease-free equilibrium $\chi_0 = (N_h(0), S_1(0), 0, 0, 0, 0, 0, 0)$, given by

$$J(S_1, S_2, V, E, I_1, I_2, R, I_D) =$$

$$\begin{pmatrix} -(\varepsilon\tau_1 + \rho + \alpha) & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \rho & -(\varepsilon\tau_2 + \alpha) & 0 & 0 & 0 & 0 & 0 & 0 \\ \varepsilon\tau & \varepsilon\tau_2 & -(\omega + \alpha) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -(\sigma_1 + \alpha) & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \sigma_1\delta & -(\gamma_1 + \theta_1 + \alpha + \eta) & 0 & 0 & 0 \\ 0 & 0 & 0 & \sigma_2(1-\delta) & 0 & -(\gamma_2 + \theta_2 + \alpha + \eta & 0 & 0 \\ 0 & 0 & 0 & 0 & \theta_1 & \theta_2 & -(\gamma_3 + \alpha + \eta) & 0 \\ 0 & 0 & 0 & 0 & \gamma_1 & \gamma_2 & \gamma_3 & -\alpha \end{pmatrix}$$

The eigenvalues are given by $\lambda_1 = -(\varepsilon\tau_1 + \rho + \alpha)$, $\lambda_2 = -(\varepsilon\tau_2 + \alpha)$, $\lambda_3 = -(\omega + \alpha)$, $\lambda_4 = -(\sigma_1 + \alpha)$, $\lambda_5 = -(\gamma_1 + \theta_1 + \alpha + \eta)$, $\lambda_6 = -(\gamma_2 + \theta_2 + \alpha + \eta)$, $\lambda_7 = -(\gamma_3 + \alpha + \eta)$, $\lambda_8 = -\alpha$.

Using methods from dynamical system theory [5, 22, 23], it is established that all the eigenvalues of the system (2.1)-(2.8), have negative real parts, hence the system (2.1)-(2.8) is locally asymptotically stable.

3.3 Global asymptotic stability (GAS) of the disease free equilibrium (DFE) of the model (2.1)-(2.8)

In this section, the Lyapunov function is constructed to study the global stability of (2.1)-(2.8). Following [24], the matrix theoretic method shall be used as a guide in the construction of the Lyapunov function.

Let $p = (E, I_1, I_2, I_D)^T$ be the diseases component and $q = (S_1, S_2, V, R,)^T$ the non-disease component. Given that the initial condition, $S_1(0) \geq 0, S_2(0) \geq 0, V(0) \geq 0, E(0) \geq 0, I_1(0) \geq 0, I_2(0) \geq 0, R(0) \geq 0, I_D(0) \geq 0$ in Theorem 3.1 above, the feasible region $\wp$ is defined as $\wp = \{(S_1, S_2, V, E, I_1, I_2, R, I_D) \in \Re_+^8 : N_h \leq N_h(0)\}$. The disease free equilibrium of the model (2.1)-(2.8) is $\chi_0 = (N_h(0), S_1(0), 0, 0, 0, 0, 0, 0)$. The basic reproduction number is given by the spectral radius of FV^{-1} to be

$$R_b = \rho(FV^{-1}) = (\beta_1 + \beta_2)\left\{\frac{\sigma_1^2\delta}{(\sigma_1\delta + \sigma_2(1-\delta) + \alpha)(\gamma_{1+\theta_1} + \alpha + \eta)} + \frac{\sigma_2^2(1-\delta)}{(\sigma_1\delta + \sigma_2(1-\delta) + \alpha)(\gamma_2 + \theta_2 + \alpha + \eta)}\right\}.$$

Using the left eigenvector of the nonnegative matrix FV^{-1} to obtain W^T, W^TV^{-1} is derived as

$$w^TV^{-1} = \left(\left\{\frac{1}{\sigma_1\delta + \sigma_2(1-\delta) + \alpha}\frac{\sigma_1((\sigma_1\delta + \sigma_2 + \alpha) - \delta)}{(\sigma_1^2 - \sigma_2^2)\delta + \sigma_2^2}R_b\right\} + \frac{\sigma_1\delta}{(\sigma_1\delta + \sigma_2(1-\delta) + \alpha)(\gamma_{1+\theta_1} + \alpha + \eta)}, \frac{1}{\gamma_{1+\theta_1} + \alpha + \eta}\right)$$

which is the Lyapunov function of (2.1)-(2.8), hence the following theorem is needed to establish the Global Asymptotic Stability (GAS) of the system (2.1)-(2.8).

Theorem 3.4. *The disease free equilibrium of the model* (2.1)-(2.8) *is globally asymptotically stable in* $\wp = \{(S_1, S_2, V, E, I_1, I_2, R, I_D) \in \Re_+^8 : N_h \leq N_h(0)\}$, *if* $R_b \leq 1$.

Proof. Let

$$L = w^TV^{-1}p = \left\{\frac{1}{\sigma_1\delta + \sigma_2(1-\delta) + \alpha}\frac{\sigma_1((\sigma_1\delta + \sigma_2 + \alpha) - \delta)}{(\sigma_1^2 - \sigma_2^2)\delta + \sigma_2^2}R_b\right\}E + \frac{\sigma_1\delta}{(\sigma_1\delta + \sigma_2(1-\delta) + \alpha)(\gamma_{1+\theta_1} + \alpha + \eta)}E + \frac{I_1}{\gamma_{1+\theta_1} + \alpha + \eta}$$

be a Lyapunov function of the model (2.1)-(2.8) on $\wp = \{(S_1, S_2, V, E, I_1, I_2, R, I_D) \in \Re_+^8 : N_h \leq N_h(0)\}$, with $R_b < 1$ and $f(p.q) \geq 0$. Then by differentiating along the solution of (2.1)-(2.8), that is, the time derivative gives

$$\dot{L} = w^TV^{-1}\dot{p}\,.$$

When the expressions for the derivatives, $\dot{E}$ and $\dot{I}_1$ from (2.1)-(2.8), is substituted into the Lyapunov derivative, $\dot{L}$, and carrying out some algebraic manipulations, given that $\dot{p} = (V - F)P - f(p, q)$ the following is obtained

$$\dot{L} = w^TV^{-1}\dot{p} = w^TV^{-1}\{(V - F)P - f(p, q)\}$$

$$\dot{L} = w^TV^{-1}\dot{p} = w^TV^{-1}(V - F)P - w^TV^{-1}f(p, q)$$

$$\dot{L} = (R_b - 1)w^T P - w^T V^{-1} f(p,q)$$

$$\dot{L} = (R_b - 1)w^T p \; - \; w^T V^{-1} f(p,q)$$

$$\begin{aligned}\dot{L} = (R_b - 1)(T_2 E + I) \\ - \frac{1}{\sigma_1\delta + \sigma_2(1-\delta) + \alpha} \frac{\sigma_1((\sigma_1\delta + \sigma_2 + \alpha) - \delta)}{(\sigma_1^2 - \sigma_2^2)\delta + \sigma_2^2} R_b \left\{ \frac{\beta_1(\sigma_1 I_1 + \sigma_2 I_2)}{N_h - I_D} S_1 \right. \\ \left. + \frac{\beta_2(\sigma_1 I_1 + \sigma_2 I_2)}{N_h - I_D} S_2 \right\} \\ - \frac{\sigma_1\delta}{(\sigma_1\delta + \sigma_2(1-\delta) + \alpha)(\gamma_{1+\theta_1} + \alpha + \eta)} \left\{ \frac{\beta_1(\sigma_1 I_1 + \sigma_2 I_2)}{N_h - I_D} S_1 \right. \\ \left. + \frac{\beta_2(\sigma_1 I_1 + \sigma_2 I_2)}{N_h - I_D} S_2 \right\} - \frac{\sigma_1\delta E}{\gamma_{1+\theta_1} + \alpha + \eta}.\end{aligned}$$

Thus it follows that $\dot{L} \leq 0$ if $R_b \leq 1$. If $R_b = 1$, then $\dot{L} = 0$ if and only if $I_1 = I_2 = E = 0$. Therefore every solution trajectory of the equations in the model (2.1)-(2.8) converges to the largest compart invariant set $M = \{(S_1, S_2, V, E, I_1, I_2, R, I_D)\}$ and the only point in M is the disease free equilibrium. Then by LaSalle's invariant principle in [23, 25, 26], χ_0 is globally asymptotically stable in $\wp$ if $R_b \leq 1$. That is every solution trajectory of equation in the model (2.1)-(2.8) approaches χ_0 as $t \to \infty$.

4. Summary/Conclusion

This work considered and formulated an age-structured mathematical model of diphtheria infection with specific epidemiological classes such as S_1, susceptible infant at time t (0-1years), S_2, susceptible school children population at time t, V, vaccination population at time t, E, exposed population at time t, I_1, asymptomatic infection population at time t, I_2, symptomatic infection population at time t, I_D, detected infectious humans at time t (asymptomatic and symptomatic) population through testing, R, recovered population at time t. It was established through theorems and proofs that the model (2.1)-(2.8) is epidemiologically meaningful, and that all its state variables are positive (non-negative) at time $t > 0$ in the domain $\wp$, and that the domain $\wp$ is indeed bounded.

Using dynamical system theory, it was established that the system is locally stable. A matrix-theoretic method was used in the construction of an appropriate Lyapunov function

$$L = w^T V^{-1} p = \left\{ \frac{1}{\sigma_1\delta + \sigma_2(1-\delta) + \alpha} \frac{\sigma_1((\sigma_1\delta + \sigma_2 + \alpha) - \delta)}{(\sigma_1^2 - \sigma_2^2)\delta + \sigma_2^2} R_b \right\} E + \frac{\sigma_1\delta}{(\sigma_1\delta + \sigma_2(1-\delta) + \alpha)(\gamma_{1+\theta_1} + \alpha + \eta)} E + \frac{I_1}{\gamma_{1+\theta_1} + \alpha + \eta}$$

for the global stability analysis of the of the formulated model. Using the next generation matrix, the reproduction ratio R_b of the system was determined to be

$$R_b = (\beta_1 + \beta_2)\left\{ \frac{\sigma_1^2\delta}{((\sigma_1\delta + \sigma_2(1-\delta) + \alpha)(\theta_1 + \gamma_1 + \alpha + \eta)} + \frac{\sigma_2^2(1-\delta)}{((\sigma_1\delta + \sigma_2(1-\delta) + \alpha)(\theta_2 + \gamma_2 + \alpha + \eta)} \right\}$$

and also established that the system is globally asymptotically stable if $R_b \leq 1$ and unstable otherwise.

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