Published by
NIGERIAN SOCIETY OF PHYSICAL SCIENCES
Available online @ https://journal.nsps.org.ng/index.php/jnsps

J. Nig. Soc. Phys. Sci. 7 (2025) 2618

Journal of the Nigerian Society of Physical Sciences

Mathematical model analysis on the significance of surveillance and awareness on the transmission dynamics of diphtheria

James Andrawus[a,*], Kayode Isaac Omotoso[a], Agada Apeh Andrew[b], Felix Yakubu Eguda[a], Sunday Babuba[a], Kabiru Garba Ibrahim[a]

[a]*Department of Mathematics, Federal University Dutse, 7156, Jigawa, Nigeria*
[b]*Department of Mathematics, Prime University, Abuja, Nigeria*

Abstract

Corynebacterium diphtheriae is a respiratory pathogen. Diphtheria was a major source of disease and mortality, particularly in children under five years of age and those over forty years of age. However, due to the lack of vaccinations, the disease is still widespread in several countries, particularly after the COVID-19 pandemic. In light of the above reason, we propose a deterministic mathematical model to characterize the dynamics of diphtheria transmission, evaluating the effects of awareness and surveillance that other authors have not considered. The boundedness and positivity of the solution have been established. In addition, it has been investigated that if $\mathcal{R}_c < 1$, the model shows a diphtheria-free equilibrium that is stable both locally and globally. According to the theoretical study, there is a distinct positive endemic equilibrium, and the corresponding control reproduction number is greater than one. The endemic equilibrium has also been shown to be globally asymptotically stable when the disease induces mortality, vaccination, booster vaccination, and isolation are zero. Both the diphtheria-free and the endemic equilibrium global stability are numerically justified. Model fitting and parameter estimation are obtained using the least-squares method. Numerical simulation reveals that the development of surveillance and awareness is effective in curtailing the spread of diphtheria infection. Finally, the theoretical and numerical result shows that with surveillance and awareness, the disease can be eradicated in the population in less than ten years.

DOI:10.46481/jnsps.2025.2618

Keywords: Mathematical model, Analysis, Surveillance, Transmission, Diphtheria

Article History :
Received: 05 January 2025
Received in revised form: 11 June 2025
Accepted for publication: 11 June 2025
Available online: 23 June 2025

Communicated by: J. Ndam

1. Introduction

Human evolution has been coping with infectious diseases for decades. The mechanism of transmission is known for most diseases [1]. Characterized as "The Strangling Angel of Children". Corynebacterium diphtheria, recognized to cause the highly contagious and potentially fatal bacterial diseases known as endemic and epidemic diphtheria disease (Museum of Healthcare) [2]. Due to the exotoxin it creates, it causes respiratory blockages that are accompanied by coughing or sneezing. It is also characterized by difficulties breathing, problems with heart rhythm, heart failure, and even death [3]. Because it is transferred by respiratory droplets, diphtheria is highly contagious, particularly in crowded and unsanitary settings. Globally, 5,000 cases of diphtheria were reported per year [4].

After being exposed to the bacteria, the symptoms often appear two to five days later. Fever, enlarged neck glands, weakness, and sore throat are some of the infection's external symptoms. Dead tissue in the respiratory system can cover tissues in the nose, tonsils, and throat in two to three days, creating a thick, gray layer that makes breathing and swallowing difficult

*Corresponding author Tel. No.: +234-806-783-6348.
Email address: james.a@fud.edu.ng (James Andrawus)

[5]. Lack of vaccine coverage is the main factor influencing the fatality rate from diphtheria, which has been found to range between 5 and 10% worldwide. Adults over 40 and children under five may have up to 20% higher mortality rates; however, among immunocompromised individuals and a large number of unvaccinated, unaware people without an adequate system for monitoring, cases may be high among those over 40 in nations with lower diphtheria prevalence [5]. It is well acknowledged that vaccination is the most efficient way to prevent diphtheria. The diphtheria vaccination, which is frequently given in conjunction with the pertussis and tetanus (DPT) vaccine, has been crucial in lowering the disease's prevalence throughout the world [6].

Public health professionals must educate people and communities about disease awareness and monitoring as an extra precaution to maintain protection following the early recommendation of a booster shot during adolescence or maturity. Over the past 37 years, vaccination rates have dramatically increased in affluent nations; nonetheless, high rates of diphtheria persist in many nations with inadequate access to healthcare and immunizations [7, 8].

In the past, Nigeria has had few outbreaks of diphtheria; the most notable one occurred in 2011, when 98 cases were documented, with 21% of those infections leading to later fatality [9]. Nigeria is now ranked sixth in terms of population, with approximately 42.54% of its population under the age of 14 being afflicted [10]. The nation is more susceptible to the disease because, according to Ref. [11], just 41.7% of children under the age of 15 had received all recommended vaccinations, even though the antitoxin vaccine was available in the nation. A study by Ref. [12] developed an optimal control of the diphtheria epidemic model with prevention and treatment. Their optimal control analysis shows that vaccination, treatment, and quarantine can be efficiently controlled to control the spread of the diphtheria outbreak. Ref. [13] proposed a mathematical model incorporating vaccination parameters in the susceptible compartment for the instance of a diphtheria outbreak. Their results show that the vaccination is helpful in stopping the spread of diphtheria, but it is just meant to be a preventative measure.

The results of Ref. [12], using parameters from Ref. [14] indicated that the dynamics of the model are influenced by the natural immunity rate of the exposed population and the full coverage of basic immunizations. A study by Ref. [15] proposed a mathematical model to simulate the dynamic interaction between two fictitious C. diphtheriae strains in a host population with varying levels of immunity. They concluded that if the interaction between strains is taken into consideration, the use of toxoid vaccinations could result in the eradication of sickness. A study by Ref. [16] proposed a deterministic mathematical model for optimal control of diphtheria disease with a booster vaccination. Their result indicated that treating asymptomatic infected individuals, disinfecting the environment, and immunizing the population as a whole are the most effective combinations.

The most recent outbreak occurred in 2023, following the general breakouts in 2018. According to the most current report from the Nigeria Centre for Disease Control (NCDC), there has been an increase in suspected cases (733), 89 deaths, and a case fatality rate (CFR) of 12.3% that affects children aged 5 to 18 [5]. Nigeria's current diphtheria outbreak is a major public health threat to the neighboring countries and the world in general, being the most populous nation in Africa. Vaccination coverage of at least 90% has been known to give protection to about 60% of the population at risk. However, there is limited coverage of vaccination in the country, [3].

There have been studies on the diphtheria model; [5, 17–19] provided a model for the transmission of the disease by taking into account asymptomatic infectious, full national-acquired immunity, and partial natural-acquired immunity of carriers, individuals with full vaccine-induced immunity, and individuals with partial vaccine-induced immunity; other studies examined the global stability and parameter estimation [20, 21]. The construction and development of a model for the spread of diphtheria that takes into account handwashing practices, quarantine for both exposed and infected individuals, progression for vaccinated exposed individuals, and transmission through contact with an exposed or infected person [22].

Ref. [23] studied mathematical analysis of diphtheria transmission and control with a focus on the effectiveness of diphtheria antitoxins; Recognizing the pattern of the Nigerian diphtheria outbreak between 1941 and 2023 [3]. In order to develop efficient methods for managing and averting diphtheria outbreaks, Ref. [24] recently carried out an analysis on transmission dynamics; Ref. [2], evaluating the effect of booster vaccination on the spread of diphtheria, which also has limitations in terms of data availability for additional measures; In order to describe the SEIR model, Ref. [14] took into account the rate of natural immunity of those exposed to diphtheria. The authors' consideration of the model's fundamental features emphasizes the significance of vaccination coverage, declining immunity, and disease vulnerability in the spread of diphtheria. Additionally, the models indicated that a combination of therapy, immunization, and contact tracing may be useful in controlling diphtheria epidemics. In many regions of the world, diphtheria remained a serious public health concern even after effective vaccinations became available. Based on these considerations, the goal of this work is to investigate the significance and awareness of the transmission dynamics of diphtheria by mathematical model analysis.

For many years, mathematical models have been used to study the dynamics of epidemic disease. In recent years, the use of mathematics in epidemiology has expanded significantly. A model can predict if a disease will spread through the population or die out by predicting model variables, production number, transmission rate, and other characteristics. Additionally, a model can calculate the impact of an intervention and give public administrators the essential direction for future disease eradication efforts [25]. In light of the context in Ref. [22] and the previously described discussions, the goal of this study is to provide a comprehensive model for diphtheria that includes both symptomatic and asymptomatic infectious diseases [25], as well as different control methods.

Our study offers a strong method for preventing the spread of diphtheria by raising awareness and using effective surveil-

lance techniques as recommended by Ref. [26]. The goal of these tactics is to decrease contact between asymptomatic infected people and those who have received vaccinations. In order to raise public awareness and prevent infection through increased awareness efforts, this includes extensive educational initiatives, awareness campaigns, and surveillance programs aimed at educating the public about the significant hazards of diphtheria. In order to reduce the spread of the disease overall, this involves encouraging the use of available vaccines, recommending afflicted individuals to seek medical attention, and stressing the need to avoid close contact with infected individuals through knowledge, good hygiene, and public health surveillance. This research aims to use mathematical model analysis to the significance of awareness and surveillance on the dynamics of diphtheria transmission.transmission of illness.

The paper is organized as follows, the introduction and relevant literature are covered in section 1, model formulation is covered in section 2, analysis of the model covered in section 3, numerical simulations and a discussion of the simulated figures are covered in section 4, and the conclusion is covered in section 5.

2. Model description

To unriddle the mechanism of diphtheria transmission in the human population, we divide the entire population into different compartments based on the status of the disease. The model of the total population is subdivided into eight compartments, which include Susceptible individuals (S), Exposed aware individuals (E_a), Exposed unaware individuals (E_u), Symptomatic individuals (I), Asymptomatic individuals (A), Individuals under surveillance (P), Isolated individuals (J), Recovered and immunized individuals (R). A series of differential equations that take into account a number of parameters, including infection rates, control measures, and recovery rates, including awareness, surveillance, vaccination, treatment, exposure, latency, isolation, recovery, and progression rates, define the mathematical model. In Susceptible compartment (S): (λ), which indicates the emergence of susceptible, i.e., aware and unaware individuals, causes the rate of susceptible individuals to increase, while (μ), which represents the natural death rate, causes this number to decrease. The "S" compartment represents the number of susceptible individuals. meanwhile, there is a reduction caused by $a_1\lambda$, which accounts for individuals aware of the disease but contracted the disease through person-to-person contact, and $(1 - a_1)\lambda$, reflecting those contracting the virus unaware of where $\lambda = \frac{\beta(1-\theta)(A+\eta I)}{N}$ and $N = S + E_a + E_u + I + A + P + J + R$. Compartment ($E_a$): The "$E_a$" compartment represents the aware individuals of the virus who were kept under surveillance and vaccinated against the disease. The number also decreases due to the natural death rate, represented by μ. Compartment "E_u": Unaware people who are catching the virus from asymptomatic infected contacts and those who have been infected by coming into contact with symptomatic people make up the "E_u" compartment. The natural death rate, denoted by μ, can likewise be used to decrease the population in this area.

Compartment (I): "I" symptomatic individual increases through the progression from the exposed, unaware compartment with the progressive rate σ_1, and it reduces due to induced and natural death. ($\mu + \delta_1$) Compartment (A): Corresponding to asymptomatic infected individuals, unaware of the disease, who progress from the main population, natural mortality also occurs due to diphtheria-induced death rate ($\mu + \delta_2$), This compartment is more dangerous in the transmission of the disease. They live in a society without anyone's notice, even though they don't know that they are infected, and some of them are recognized by testing through awareness. Compartment (P): The "P" compartment comprises aware individuals under surveillance of the disease with the surveillance rate ϕ. This compartment sees a reduction due to the Natural death rate, μ. Compartment (J); This compartment represents the isolated hospitalized individual; the number here increases due to $\gamma_1, \gamma_2, \gamma_3$ accounting for symptomatic, asymptomatic, and individuals under surveillance, respectively. The isolated individuals reduced the death rate at ($\mu + \delta_3$). Compartment (R): With a ν and χ recovery rate from diphtheria and immunization, "R" is recruited. Specifically, the recovery rate and post-vaccination rate, respectively, and all classes decrease as a result of the natural death rate, which is μ. The flow between the compartments is described in the figure below. Figure 1 shows the schematic diagram of diphtheria as described using the assumptions above and equation (1) shows the appropriate model equations that are derived from Figure 1. Table 1 provides the interpretations of the variables and parameters.

$$
\begin{aligned}
\frac{dS}{dt} &= \pi - a_1\lambda S - (1 - a_1)\lambda S - \mu S,\\
\frac{dE_a}{dt} &= a_1\lambda S + a_2 E_u - (\psi + \mu)E_a,\\
\frac{dE_u}{dt} &= (1 - a_1)\lambda S - (\mu + a_2 + \sigma_1 + \sigma_2)E_u,\\
\frac{dI}{dt} &= \sigma_1 E_u - (\mu + \delta_1 + \gamma_1)I,\\
\frac{dA}{dt} &= \sigma_2 E_u - (\mu + \delta_2 + \gamma_2)A,\\
\frac{dP}{dt} &= \psi E_a - (\mu + \gamma_3 + \nu)P,\\
\frac{dJ}{dt} &= \gamma_1 I + \gamma_2 A + \gamma_3 P - (\chi + \mu + \delta_3)J,\\
\frac{dR}{dt} &= \chi J + \nu P - \mu R,
\end{aligned}
\tag{1}
$$

where

$$
\lambda = \frac{\beta(1 - \theta)(A + \eta I)}{N}. \tag{2}
$$

3. Basic properties of the model

This section gives the positivity and boundedness of solutions, computation of the basic reproduction number, and all the classical analysis of the system of the model (1).

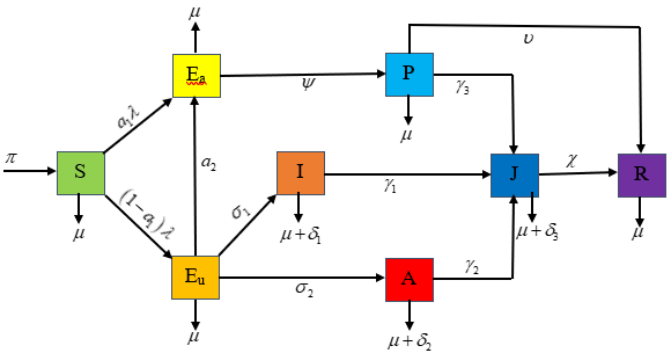

Figure 1: Schematized diagram of diphtheria.

Table 1: Variable and parameter of system equation (1).

Variable	Description
N	Total population
S	Susceptible individuals
E_a, E_u	Exposed aware and unaware individuals
I, A	Symptomatic and Asymptomatic infectious individuals
P	Individuals under Surveillance
J	Isolated individuals
R	Recovered individuals
Parameter	
π	Recruitment rate
a_1, a_2	Awareness rates
μ	Natural death rate
β	effective risk rate
ψ	Surveillance rate
$\gamma_1, \gamma_2, \gamma_3$	Rates of isolation
σ_1, σ_2	Rates of Progression
$\delta_1, \delta_2, \delta_3$	Diphtheria induced death rates
ν	Post-vaccination rates
θ	Personal hygiene
λ	Force of infection
χ	Recovery rate
η	Less infectiousness modification parameter of symptomatic individuals

3.1. Solutions uniqueness and existence

The uniqueness and existence of the solution of model (1) will be tested to ascertain whether the solution exists, and if confirmed to exist, then it is necessary to show that the solution of model (1) is unique. Using the Lipschitz Criteria, we have the solutions of equilibrium as follows:

$$\begin{aligned}
u_1 &= \pi - a_1\lambda S - (1-a_1)\lambda S - \mu S,\\
u_2 &= a_1\lambda S + a_2 E_2 - (\mu+\psi)E_a,\\
u_3 &= (1-a_1)\lambda S - (a_2+\sigma_1+\sigma_2+\mu)E_u,\\
u_4 &= \sigma_1 E_u - (\mu+\delta_1+\gamma_1),\\
u_5 &= \sigma_2 E_a - (\gamma_2+\mu+\delta_2)A,\\
u_6 &= \psi E_a - (\mu+\gamma_3+\nu)P,\\
u_7 &= \gamma_1 I + \gamma_2 A + \gamma_3 P - (\chi+\mu+\delta_3)J,
\end{aligned}$$

$$u_8 = \chi J + \nu P - \mu R. \tag{3}$$

The above equation (3) shows that the system of equations (1) exists using this method. We therefore need to show the uniqueness of the system in the region or an interval containing the solution sets. The boundedness of the solution of model (1) will be checked in a region $0 < P < R$ in which its partial derivatives are within $\delta_1 < P < R$, whereby δ_1 and R are constants greater than zero.

Theorem 3.1. The region $0 < P < R$ in Ω contains a unique solution for model (1), given that

$$\frac{du_i}{dt_j} = 1, 2, 3, 4, 5, 6, 7, 8. \tag{4}$$

The region Ω is continuous and bounded.

Proof. By partially differentiating u_i in relation to each state variable in (1), we obtain:

$$\begin{aligned}
\left|\frac{\partial u_1}{\partial S}\right| &= \left|-\left(\frac{a_1\beta(1-\theta)(A+\eta I)}{N}\right)\right.\\
&\quad \left.-(1-a_1)\left(\frac{\beta(1-\theta)(A+\eta I)}{N}\right)-\mu\right| < \infty\\
\left|\frac{\partial u_1}{\partial E_a}\right| &= \left|\frac{\partial u_1}{\partial E_u}\right| = \left|\frac{\partial u_1}{\partial P}\right| = \left|\frac{\partial u_1}{\partial J}\right| = \left|\frac{\partial u_1}{\partial R}\right| = 0 < \infty,\\
\left|\frac{\partial u_1}{\partial I}\right| &= \left|-\frac{a_1\beta(1-\theta)}{N}\eta S - \frac{\eta(1-a_1)(\beta(1-\theta))}{N}S\right| < \infty,\\
\left|\frac{\partial u_1}{\partial A}\right| &= \left|-\frac{a_1\beta(1-\theta)}{N}S - \frac{(1-a_1)(\beta(1-\theta))}{N}S\right| < \infty.
\end{aligned} \tag{5}$$

As seen in equation (5), similarly, the same thing can be done for $u_2, u_3, u_4, u_5, u_6, u_7$, and u_8, therefore, since all partial derivatives are less than infinity, model (1) exists and has a unique solution in Ω.

Proving the existence of a solution for a system (like a differential equation or a set of equations) is crucial because it demonstrates that a solution is possible under the given conditions. It ensures that the assumptions made about the system are consistent and that a solution can be found, if it exists. Moreover, proving existence can pave the way for understanding the system's behaviour and finding unique solutions, which is fundamental in many fields. □

3.2. Boundedness and positivity of solution

The positivity and boundedness of the system (1) must be demonstrated in this subsection. For the model (1), let Ω be a biologically viable appropriate zone that is specified by:

$$\Omega = (S, E_a, E_u, I, A, P, J, R) \in R_+^8 : N \le \frac{\pi}{\mu}. \tag{6}$$

Theorem 3.2. Suppose that the model's initial values are as follows:
$\{S(0), E_a(0), E_u(0), I(0), A(0), P(0), J(0), R(0) \ge 0\} \in \Omega$.
Consequently, for any $t > 0$, the solution set $\{S(t), E_a(t), E_u(t), I(t), A(t), P(t), J(t), R(t)\}$ of model (1) is positive.

Proof. In order to show that $S(0) > 0$, $E_a(0) > 0$, $E_u(0) > 0$, $I(0) > 0$, $A(0) > 0$, $P(t) > 0$, $J(t) > 0$, and $R(t) > 0$ are not negative, it is necessary to show that the solution variables $S(t), E_a(t), E_u(t), I(t), A(t), P(t), J(t), R(t)$ of model (1) conform to the initial conditions. Let $t_1 = sup(t > 0)$. $u\epsilon[0, 1]$, $I(u) \geq 0$, $A(u) \geq 0$, $P(u) \geq 0$, $S(u) \geq 0$, $E_a(u) \geq 0$, $E_u(u) \geq 0$, $R(u) \geq 0.$, $J(u) \geq 0$, and $J(u) \geq 0$ In order to guarantee the existence of t_1, the aforementioned initial conditions additionally guarantee the continuity of all functions $S, E_a, E_u, I, A, P, J, R$. The solution of model (1) is positive if $t_1 = 0$. There is at least one solution $S(t), E_a(t), E_u(t), I(t), A(t), P(t), J(t), R(t)$ that equals zero at value t_1 if $t_1 < \infty$, that is, t_1, is finite. Considering that t_1 is a supremum by definition. Assuming that $S(t_1) = 0$, from the model's first equation (1).

$$\frac{dS(t)}{dt} = \pi - a_1\lambda S - (1 - a_1)\lambda S - \mu S(t). \tag{7}$$

We know that for all $t \in [0, t_1]$, $\pi - a_1\lambda S \geq 0$. It follows that:

$$\frac{dS(t)}{dt} + (\lambda + \mu)S \geq 0, \tag{8}$$

therefore, the integrating factor of equation (8) is given as:

$$IF = e^{\int(\lambda+\mu)dt} = e^{(\lambda+\mu)t}, \tag{9}$$

using equation (9) to integrate equation (8), gives:

$$S(t)e^{(\lambda+\mu)t} - S(o)e^{(\lambda+\mu)0}, \tag{10}$$

therefore, equation (10) can be written as:

$$S(t)e^{(\lambda+\mu)t} - S(0) \geq 0, \tag{11}$$

equation (11) becomes

$$S(t)e^{(\lambda+\mu)t} \geq S(0), \tag{12}$$

therefore, equation (12) becomes:

$$S(t) \geq S(0)e^{-(\lambda+\mu)t}. \tag{13}$$

Therefore, considering the result in equation (13), it is easy to say that they are in conflict with the fact that $S(t_1) = 0$. In the remaining instances, $S(t_1) = 0$, $E_a(t_1) = 0$, $E_u(t_1) = 0$, $I(t_1) = 0$, and $A(t_1) = 0$. $J(t_1) = 0$, $P(t_1) = 0$ and the identical contradiction results from $R(t_1) = 0$. Consequently, for all $t > 0$, $S(t) > 0$, $E_a(t) > 0$, $E_u(t) > 0$, $I(t) > 0$, $A(t) > 0$, $P(t) > 0$, $J(t) > 0$, and $R(t) > 0$.

Epidemiological implication of Theorem 3.2: Proving the positivity of solutions in a mathematical model is crucial because it ensures the model's results are physically meaningful and accurate in representing real-world phenomena. In many applications, such as epidemiology or biology, negative quantities would be nonsensical, so positivity guarantees the model's output aligns with reality. □

Theorem 3.3. The model defined by equation (1) has a closed physiologically viable region Ω. Since $\Omega = (S, E_a, E_u, I, A, P, J, R) \in R_+^8 : N \leq \frac{\pi}{\mu}$, Ω is positively invariant and attracts all of the model's positive solutions.

Proof. The fact that any solutions on Ω do not depart Ω must be demonstrated. Consequently, the rate at which the entire population is changing is provided by:

$$\frac{dN}{dt} = \pi - \mu N - \delta_1 I - \delta_2 A - \delta_3 J, \tag{14}$$

using the comparison Theorem results on equation (14), we have

$$\frac{dN}{dt} = \pi - \mu N. \tag{15}$$

Using (15) and the integrating factor technique results in

$$N(t) \leq \frac{\pi}{\mu} + \left[N(0) - \frac{\pi}{\mu}\right]e^{-\mu t}, \forall t \geq 0. \tag{16}$$

According to equation (16), is positively invariant and attracts all positive solutions of the system (1) because t, N(t) N(0), N(t) $\leq \frac{\pi}{\mu}$, and as t, N(t) $\frac{\pi}{\mu}$. □

3.3. Existence of equilibrium

At each equilibrium, let $(S^*, E_a^*, E_u^*, I^*, A^*, P^*, J^*, R^*)$ represent the solution of the system (1). We set the rate of change to zero at equilibrium. That is, setting all of the equations in the system (1) to zero and doing so in terms of λ at the same time.

$$\begin{aligned}
S &= \frac{\pi}{\lambda + \mu}, \\
E_a &= \frac{a_1\pi\lambda}{k_1(\lambda + \mu)} + \frac{a_2(1 - a_1)\pi\lambda}{k_2(\lambda + \mu)}, \\
E_u &= \frac{(1 - a_1)\pi\lambda}{k_2(\lambda + \mu)}, \\
I &= \frac{\sigma_1(1 - a_1)\pi\lambda}{k_2k_3(\lambda + \mu)}, \\
A &= \frac{\sigma_2(1 - a_1)\pi\lambda}{k_2k_4(\lambda + \mu)}, \\
P &= \frac{\psi}{k_5}\frac{a_1\pi\lambda + a_2(1 - a_1)\pi\lambda}{k_1k_2(\lambda + \mu)}, \\
J &= \frac{\gamma_1(\sigma_1(1 - a_1)\pi\lambda + \gamma_2\sigma_2(1 - a_1)\pi\lambda + \gamma_3\Psi}{k_1k_2k_3k_4k_5k_6(\lambda + \mu)},
\end{aligned} \tag{17}$$

where $\Psi = \psi(a_1\pi\lambda + a_2(1 - a_1)\pi\lambda)$ and

$$\begin{aligned}
k_1 &= \mu + \psi, \\
k_2 &= a_2 + \sigma_1 + \sigma_2 + \mu, \\
k_3 &= \mu + \delta_1 + \gamma_1, \\
k_4 &= \gamma_2 + \mu + \delta_2, \\
k_5 &= \mu + \gamma_3 + \nu, \\
k_6 &= \chi + \mu + \delta_3.
\end{aligned} \tag{18}$$

3.4. Disease free-equilibrium

A disease-free equilibrium is reached in the model system (1) when infectious classes are zero. By putting the right-hand sides of each equation in system (1) to zero, the disease-free equilibrium can be determined. Equations solved yield the disease-free equilibrium in equation (19)

$$\begin{aligned}\Omega^0 &= (S^0, E_a^0, E_u^0, I^0, A^0, P^0, J^0, R^0)\\ &= \left(\frac{\pi}{\mu}, 0, 0, 0, 0, 0, 0, 0\right).\end{aligned} \tag{19}$$

3.5. Basic reproduction number

The following subsection uses the next generation matrix operator method, as described by, to determine the local stability of Ω^0 using the idea in Refs. [24, 27–34]. The production number is obtained by using the matrices F and V for the new infection terms and the remaining transfer terms.

$$F = \begin{bmatrix} 0 & 0 & a_1\eta\beta\,(1-\theta) & a_1\beta\,(1-\theta) & 0 & 0\\ 0 & 0 & (1-a_1)\,\eta\beta\,(1-\theta) & (1-a_1)\beta\,(1-\theta) & 0 & 0\\ 0 & 0 & 0 & 0 & 0 & 0\\ 0 & 0 & 0 & 0 & 0 & 0\\ 0 & 0 & 0 & 0 & 0 & 0\\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}, \tag{20}$$

and

$$V = \begin{bmatrix} k_1 & -a_2 & 0 & 0 & 0 & 0\\ 0 & k_2 & 0 & 0 & 0 & 0\\ 0 & -\sigma_1 & k_3 & 0 & 0 & 0\\ 0 & -\sigma_2 & 0 & k_4 & 0 & 0\\ -\psi & 0 & 0 & 0 & k_5 & 0\\ 0 & 0 & -\gamma_1 & -\gamma_2 & -\gamma_3 & k_6 \end{bmatrix}. \tag{21}$$

Next, the inverse of V in equation (21) is calculated as

$$V^{-1} = \begin{bmatrix} \frac{1}{k_1} & \frac{a_2}{k_1k_2} & 0 & 0 & 0 & 0\\ 0 & \frac{1}{k_2} & 0 & 0 & 0 & 0\\ 0 & \frac{\sigma_1}{k_2k_3} & \frac{1}{k_3} & 0 & 0 & 0\\ 0 & \frac{\sigma_2}{k_2k_4} & 0 & \frac{1}{k_4} & 0 & 0\\ \frac{\psi}{k_1k_5} & \frac{a_2\psi}{k_1k_2k_5} & 0 & 0 & \frac{1}{k_5} & 0\\ \frac{\gamma_3\psi}{k_1k_5k_6} & \Upsilon & \frac{\gamma_1}{k_3k_6} & \frac{\gamma_2}{k_4k_6} & \frac{\gamma_3}{k_5k_6} & \frac{1}{k_6} \end{bmatrix}, \tag{22}$$

where $\Upsilon = \frac{\gamma_1\sigma_1k_1k_4k_5+\gamma_2\sigma_2k_1k_3k_5+\gamma_3\psi\sigma_2k_3k_4}{k_1k_2k_3k_4k_5k_6}$. Multiplying equation (20) with equation (22), that is FV^{-1} and considering the dominant eigenvalues, gives the effective reproduction number of the model (1):

$$\rho\left(FV^{-1}\right) = \mathcal{R}_{ef} = \frac{(1-a_1)\,\eta\beta\,(1-\theta)\,\sigma_1}{k_2k_3} + \frac{(1-a_1)\beta\,(1-\theta)\,\sigma_2}{k_2k_4}, \tag{23}$$

where (R_c) is the effective reproductive number associated with asymptomatic infectious and symptomatic infectious individuals as given in equation (23). That is when there is an absence of measure to control the disease ($a_1 = a_2 = \gamma_1 = \gamma_2 = \gamma_3 = \theta = 0$),(21) reduces to the basic reproduction number ($\mathcal{R}_0$) given by:

$$\mathcal{R}_0 = \frac{(1-a_1)\,\eta\beta\sigma_1}{k_2^*k_3^*} + \frac{(1-a_1)\beta\sigma_2}{k_2^*k_4^*}, \tag{24}$$

where

$$\begin{aligned} k_2^* &= a_2 + \sigma_1 + \sigma_2 + \mu,\\ k_3^* &= \mu + \delta_1 + \gamma_1,\\ k_4^* &= \gamma_2 + \mu + \delta_2. \end{aligned} \tag{25}$$

Epidemiological Interpretation of $\mathcal{R}_{ef}$: The number of new infections caused by diphtheria-infected individuals in a community of susceptible and immunized persons is known as the effective reproduction number $\mathcal{R}_{ef}$. In a specific sense, it is the quantity generated by an infected person in a community in the presence of controls. Hence, using Theorem 2 in Ref. [31], we claim the following result.

Theorem 3.4. The DEF of model (equation) (1) (disease-free equilibrium) is LAS (locally asymptotically stable) if the effective reproduction number is less than or equal to one (1) ($\mathcal{R}_{ef} \leq 1$).

3.6. Global stability of the disease-free equilibrium

In this subsection, we proved the global asymptotic stability (GAS) of DFE using the linear Lyapunov function, as expressed in Refs. [35–37].

Theorem 3.5. The disease-free equilibrium (Ω^0) of model (equation) (1) is globally asymptotically stable in Ω if $\mathcal{R}_c \leq 1$.

Proof. Let equation (26) be a linear Lyapunov function:

$$\mathcal{L} = B_1E_a + B_2E_u + B_3I + B_4A, \tag{26}$$

it is evident that $\mathcal{L} > 0$, except at disease free equilibrium. Differentiating $\mathcal{L}$ with respect to time, yields

$$\dot{\mathcal{L}} = B_1\dot{E}_a + B_2\dot{E}_u + B_3\dot{I} + B_4\dot{A}. \tag{27}$$

Substituting the values of the state variables concerned of system (1) into equation (27), we have

$$\begin{aligned} \dot{\mathcal{L}} &= B_1\left(\frac{\alpha_1\beta(1-\theta(A+\eta I))S}{N}\right)\\ &+ B_1\left(\alpha_2E_u - (\psi+\mu)E_a\right)\\ &+ B_2\left(\frac{(1-\alpha_1)\beta(1-\theta(A+\eta I))S}{N}\right)\\ &- B_2k_2E_u + B_3\left(\sigma_1E_u - k_3I\right)\\ &+ B_4\left(\sigma_2E_u - k_4A\right), \end{aligned} \tag{28}$$

expanding equation (28), we got

$$\begin{aligned}\dot{\mathcal{L}} = {} & B_1\frac{\alpha_1\beta(1-\theta(A+\eta I))S}{N} \\ & + B_1\alpha_2E_u - B_1(\psi+\mu)E_a \\ & + B_2\frac{(1-\alpha_1)\beta(1-\theta(A+\eta I))S}{N} \\ & - B_2k_2E_u + B_3\sigma_1E_u \\ & - B_3k_3I + B_4\sigma_2E_u - B_4k_4A. \end{aligned} \tag{29}$$

Collecting the like terms of equation (29) in terms of the state variables and with in mind that at disease-free equilibrium $S^0 \leq N^0$, equation (29) becomes

$$\begin{aligned}\dot{\mathcal{L}} \leq {} & -B_1(\psi+\mu)E_a \\ & + (B_1\alpha_2 - B_2k_2 + B_3\sigma_1 + B_4\sigma_2)\,E_u \\ & + (B_1\alpha_1\beta(1-\theta)\eta)\,I \\ & + (B_2(1-\alpha_1)\beta(1-\theta)\eta - B_3k_3)\,I \\ & + (B_1\alpha_1\beta(1-\theta))\,A \\ & + (B_2(1-\alpha_1)\beta(1-\theta) - B_4k_4)\,A. \end{aligned} \tag{30}$$

Obviously from equation (30), it is not hard to say that $B_1 = 0$ and let

$$B_2 = k_3k_4, \tag{31}$$

so that

$$\begin{aligned} B_3 &= (1-\alpha_1)\beta(1-\theta)\eta k_4, \\ B_4 &= (1-\alpha_1)\beta(1-\theta)k_3. \end{aligned} \tag{32}$$

Substituting the values of all four positive constants into the equation (30), we realized

$$\begin{aligned}\dot{\mathcal{L}} \leq {} & (\sigma_1(1-\alpha_1)\beta(1-\theta)\eta k_4)\,E_u \\ & + (\sigma_2(1-\alpha_1)\beta(1-\theta)k_3 - k_2k_3k_4)\,E_u. \end{aligned} \tag{33}$$

Simplifying equation (33), becomes

$$\begin{aligned}\dot{\mathcal{L}} \leq {} & k_2k_3k_4\left(\frac{\sigma_1(1-\alpha_1)\beta(1-\theta)\eta k_4}{k_2k_3}\right)E_u \\ & + k_2k_3k_4\left(\frac{\sigma_2(1-\alpha_1)\beta(1-\theta)}{k_2k_4} - 1\right)E_u, \end{aligned} \tag{34}$$

equation (34) can also be written as

$$\dot{\mathcal{L}} \leq k_2k_3k_4\left(\mathcal{R}_{ef} - 1\right)E_u. \tag{35}$$

□

Therefore, using Lassale's invariant principle, we conclude that $\mathcal{L}$ is a Lyapunov function. Since all requirements of a Lyapunov function are met, we deduce that DFE is GAS if $\mathcal{R}_{ef} \leq 1$. The implication of the Theorem (3.5) is that the spread of the disease can be controlled in a community regardless of the number of people who are unconscious of it, as long as the effective number of reproduction is less than or equal to unity ($\mathcal{R}_{ef} \leq 1$).

3.7. Endemic equilibrium point global asymptotic stability

Let

$$\otimes^{**} = \{(S^{**}, E_a^{**}, E_u^{**}, A^{**}, P^{**}, J^{**}, R^{**}) \in \mathcal{E}^{**}\}, \tag{36}$$

be a well stable manifold of $\mathcal{E}^{**}$. Setting $\omega = 0$ for a special case means that, when there are no cases of reversion of unaware individuals in the society, the number of aware and vaccinated individuals will increase, leading to endemicity in the society, as long as the effective number of reproductions is greater than unity (that is, $\mathcal{R}_{ef} > 1$).

Theorem 3.6. Model (1)'s EEP (Endemic equilibrium point) is a GAS (globally asymptotically stable) in $\mathcal{D}$ whenever $\mathcal{R}_{ef} > 1$.

Proof. We start by building a Goh-Volterra type Lyapunov function, which is provided by:

$$\begin{aligned}\mathcal{F} = {} & \left(S - S^{**} - S^{**}ln\frac{S}{S^{**}}\right) \\ & + \left(E_a - E_a^{**} - E_a^{**}ln\frac{E_a}{E_a^{**}}\right) \\ & + \left(E_u - E_u^{**} - E_u^{**}ln\frac{E_u}{E_u^{**}}\right) \\ & + B_1\left(A - A^{**} - A^{**}ln\frac{A}{A^{**}}\right) \\ & + B_2\left(P - P^{**} - P^{**}ln\frac{P}{P^{**}}\right) \\ & + B_3\left(J - J^{**} - J^{**}ln\frac{J}{J^{**}}\right) \\ & + B_4\left(R - R^{**} - R^{**}ln\frac{R}{R^{**}}\right). \end{aligned} \tag{37}$$

When we differentiate equation (37) in relation to time, we obtain:

$$\begin{aligned}\dot{\mathcal{F}} = {} & \left(1 - \frac{S^{**}}{S}\right)\dot{S} + \left(1 - \frac{E_a^{**}}{E_a}\right)\dot{E}_a \\ & + \left(1 - \frac{E_u^{**}}{E_u}\right)\dot{E}_u + B_1\left(1 - \frac{A^{**}}{A}\right)\dot{A} \\ & + B_2\left(1 - \frac{P^{**}}{P}\right)\dot{P} + B_3\left(1 - \frac{J^{**}}{J}\right)\dot{J} \\ & + B_4\left(1 - \frac{R^{**}}{R}\right)\dot{R}. \end{aligned} \tag{38}$$

By changing the model equation in question from equation (1)

to equation (38), we can differentiate (37) in relation to time.

$$\begin{aligned}\dot{\mathcal{F}} &= \left(1-\frac{S^{**}}{S}\right)(\pi - a_1\lambda S - (1-a_1)\lambda S - \mu S)\\ &+\left(1-\frac{E_a^{**}}{E_a}\right)(a_1\lambda S - k_1 E_a)\\ &+\left(1-\frac{E_u^{**}}{E_u}\right)((1-a_1)\lambda S - k_2 E_u)\\ &+\left(1-\frac{A^{**}}{A}\right)B_1(\sigma_2 E_u - k_4 A)\\ &+\left(1-\frac{P^{**}}{P}\right)B_2(\psi_2 E_a - k_5 P)\\ &+\left(1-\frac{J^{**}}{J}\right)B_3(\gamma_3 P - k_6 J)\\ &+\left(1-\frac{R^{**}}{R}\right)B_4(\chi J - \mu R),\end{aligned} \tag{39}$$

where

$$\bar{\beta} = \beta\frac{1}{N}, \tag{40}$$

in order to provide the force of infection by

$$\bar{\lambda} = \bar{\beta}(A_p + \eta A_S). \tag{41}$$

When π from (41) is substituted into (39), it yields

$$\begin{aligned}\dot{\mathcal{F}} &= a_1\lambda S^{**} + (1-a_1)\lambda S^{**} + \mu S^{**} - \mu S\\ &-\frac{a_1\lambda S^{**^2}}{S} - \frac{(1-a_1)\lambda S^{**^2}}{S} - \frac{\mu S^{**^2}}{S}\\ &+a_1\lambda S^{**} + (1-a_1)\lambda S^{**} + \mu S^{**},\\ &-\frac{a_1\lambda S E_u^{**}}{E} + K_1 E_a^{**} - \frac{(1-a_1)\lambda S E_u^{**}}{E_u}\\ &+k_2 E_u^{**} - \frac{k_2 k_4 A}{\sigma_2} - \frac{k_2 E_u A^{**}}{A} + \frac{k_2 k_4 A^{**}}{\sigma_2}\\ &-\frac{k_1 E_a P^{**}}{P} + \frac{k_1 k_5 P^{**}}{\psi} - \frac{k_1 k_5 J^{**} P}{\psi J},\\ &+\frac{k_1 k_5 k_6 J^{**}}{\psi\gamma_3} + \frac{k_1 k_5 k_6 \mu R}{\psi\gamma_3\psi}\\ &-\frac{k_1 k_5 k_6 J R^{**} J}{\psi\gamma_3 R} + \frac{k_1 k_5 k_6 \mu R^{**}}{\psi\gamma_3\chi}.\end{aligned} \tag{42}$$

After simplifying and changing the relations in equation (41) to equation (42), we have:

$$\begin{aligned}\dot{\mathcal{F}} \le{}& \mu S^{**}\left(2 - \frac{S}{S^{**}} - \frac{S^{**}}{S}\right) + a_1\lambda S^{**}\left(6 - \frac{S^{**}}{S} - \frac{S E_a^{**}}{S^{**}E}\right.\\ &\left.-\frac{E_a P^{**}}{E^{**}P} - \frac{P J^{**}}{P^{**}J} + \frac{R}{R^{**}} - \frac{J R^{**}}{R}\right)\\ &+\lambda S^{**}(1-a_1)\left(4 - \frac{S^{**}}{S} - \frac{S E_u^{**}}{S^{**}E_u} - \frac{A}{A^{**}} - \frac{A^{**}E_u}{E_u^{**}}\right).\end{aligned} \tag{43}$$

Given that the geometric mean is smaller than the arithmetic mean, we then have

$$\left(2 - \frac{S}{S^{**}} - \frac{S^{**}}{S}\right) \le 0,$$

Table 2: parameter values of the proposed model (1).

Denotation of parameter	Values	Unit	Reference
μ	0.01865	per year	Ref. [18]
π	1000	persons per year	Assumed
β	0.221	per year	Ref. [18]
a_1, a_2	(0, 1), (0, 1)	per year	Control parameter
η	0.16	Dimensionless	Estimated
ψ	(0, 1)	per year	Control parameter
$\gamma_1, \gamma_2, \gamma_3$	0.6, 0.532, 0.5763	per year	Estimated
σ_1, σ_2	0.09, 0.0899	per year	Control parameter
$\delta_1, \delta_2, \delta_3$	0.0231, 0.0321, 0.0653	per year	Ref. [18]
ν	(0, 1)	per year	Control parameter
θ	(0, 1)	per year	Control parameter
χ	0.446	per year	Ref. [18]

$$\left(6 - \frac{S^{**}}{S} - \frac{S E_a^{**}}{S^{**}E} - \frac{E_a P^{**}}{E_a P} - \frac{P J^{**}}{P^{**}J} + \frac{R}{R^{**}} - \frac{J R^{**}}{R} \le 0,\right.$$
$$\left(4 - \frac{S^{**}}{S} - \frac{S E_a^{**}}{S^{**}E_a} - \frac{A}{A^{**}} - \frac{A^{**}E_u}{E_u^{**}}\right) \le 0. \tag{44}$$

Since all of the variables in the model, including S, E_a, E_u, I, A, P, J, and R, are at steady state (endemic steady state), we thus have $\dot{\mathcal{F}} \le 0$ and $\mathcal{R}_{ef} > 1$. This may be replaced into the concerned variable of (1) to provide. Thus, the endemic equilibrium point is globally asymptotically stable (GAS) according to Lassalle's invariant principle [38–42].

$$\begin{aligned}&\lim_{t\to\infty}(S(t), E_a(t), E_u(t), I(t), A(t), P(t), J(t), R(t))\\ &\quad\to (S, E_a, E_u, I, A, P, J, R).\end{aligned} \tag{45}$$

Thus, the endemic equilibrium point is globally asymptotically stable (GAS) according to Lassalle's invariant principle (Lasalle, 1976). □

4. Numerical simulations

Here, we use the parameter values from Table 2 to show how the state variables of the model 1 can be numerically simulated. The model (1)'s transmission dynamics are being thoroughly understood using a numerical simulation. Time-series graphs are used to illustrate the compartment behaviour and the influence of a few key parameters on the state variables.

4.1. Discussion of simulated figures

Figure 2(a) shows the influence of post-vaccination and Surveillance rate over the population of individuals under surveillance, where increasing the percentage of post-vaccination shows a significant impact on the population of individuals under surveillance. Figure 2(b) shows the influence of post-vaccination over the population of recovered individuals; increasing the percentage of post-vaccination individuals shows little impact on the population of recovered individuals. Figure 2(c) shows the influence of Surveillance rate over

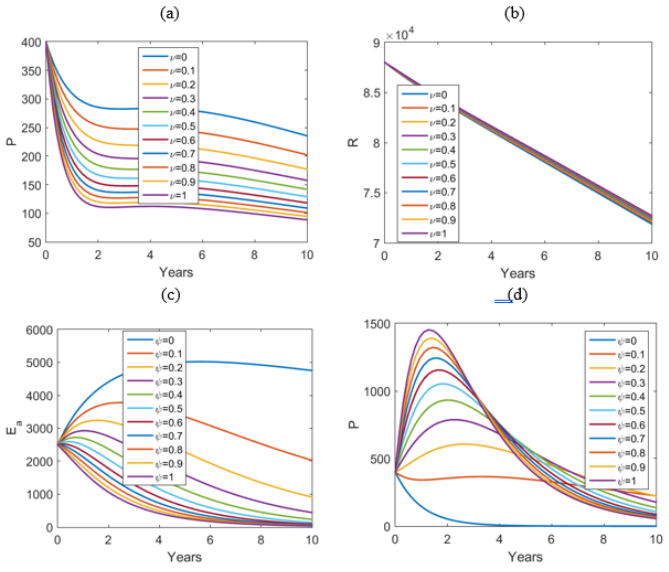

Figure 2: Influence of post vaccination and Surveillance rate over some selected infected compartments.

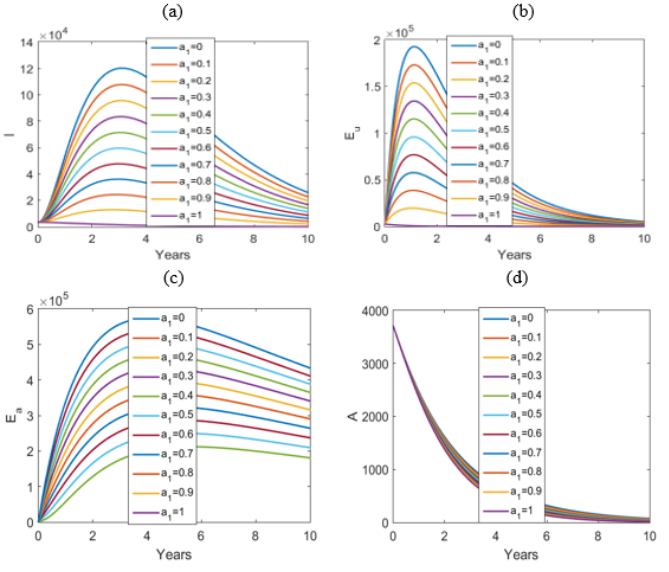

Figure 3: Influence of awareness through contact rate over some selected infected compartments.

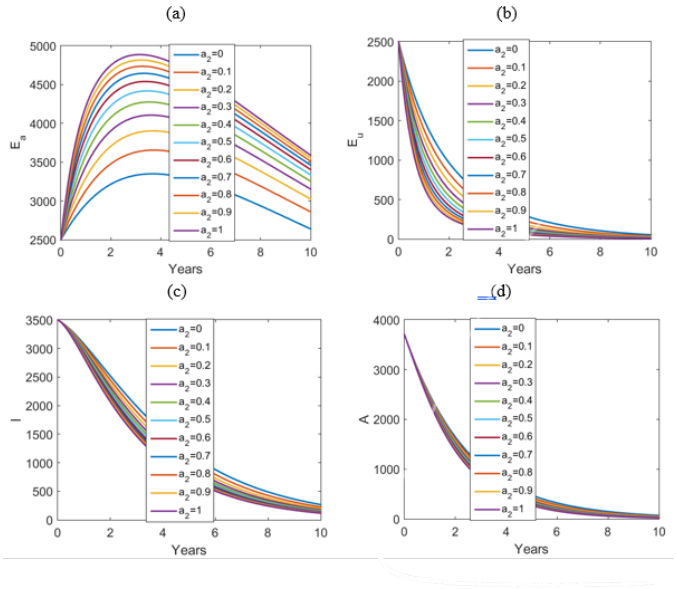

Figure 4: Influence of awareness of unaware exposed over some selected infected compartments.

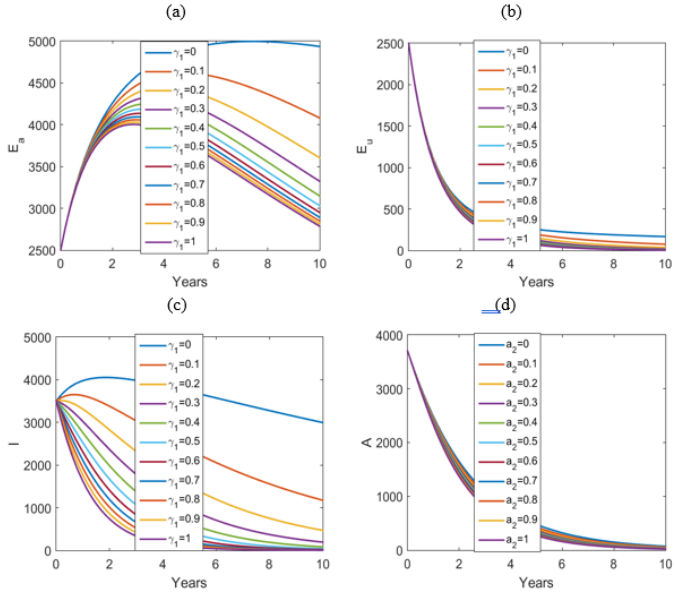

Figure 5: Influence of isolation rate of symptomatic individuals over some selected infected compartments.

the population of exposed individuals, increasing the percentage of Surveillance shows to be very impactful on the population of aware exposed individuals. Figure 2(d) shows the influence of Surveillance rate over the population of individuals under surveillance, increasing the percentage of Surveillance shows to be very impactful on the population of individuals under surveillance.

Figure 3(a) shows the influence of awareness on contact over the population of infectious individuals or symptomatic individuals. Increasing the percentage of awareness on contact shows a significant impact on the population of infectious individuals. Figure 3(b) shows the influence of awareness on contact over the population of unaware latent individuals; increasing the percentage of awareness on contact shows a significant impact on the population of unaware latent individuals. Figure 3(c) shows the influence of awareness on contact over the population of aware latent individuals. Increasing the percentage of awareness on contact shows a great impact on the population of aware latent individuals. Figure 3(d) shows the influence of awareness on contact over the population of asymptomatic individuals; increasing the percentage of awareness on contact shows a great impact on the population of asymptomatic individuals.

Figure 4(a) shows the influence of awareness of unaware la-

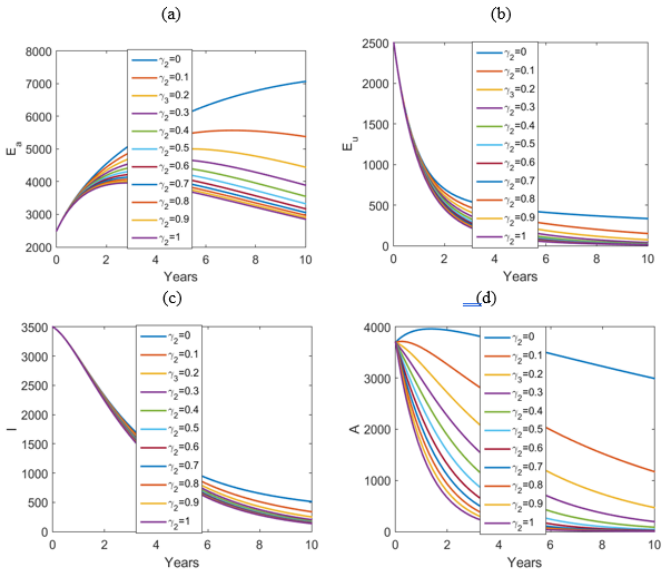

Figure 6: Influence of isolation rate on asymptomatic individuals over some selected infected compartments.

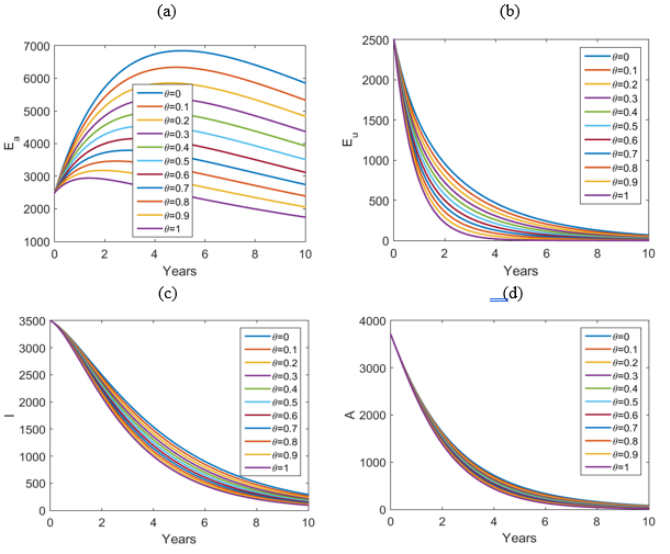

Figure 7: Influence of personal hygiene rate over some selected infected compartments.

tent over the population of aware latent individuals; increasing the percentage of awareness of unaware latent shows a great impact on the population of aware latent individuals. Figure 4(b) shows the influence of awareness of un-aware latent over population of un-aware latent individuals, increasing the percentage of awareness of un-aware latent shows much impact on the population of un-aware latent individuals. Figure 4(c) shows the influence of awareness of un-aware latent over population of infectious individuals or symptomatic individuals, increasing the percentage of awareness of un-aware latent shows much impact on the population of infectious individuals or symptomatic individuals. Figure 4(d) shows the influence of awareness of unaware latent overpopulation of asymptomatic individuals; increasing the percentage of awareness of unaware latent shows little impact on the population of asymptomatic individuals.

Figure 5(a) shows the influence of the isolation rate of symptomatic individuals over the population of aware latent individuals; increasing the isolation rate of symptomatic individuals shows a great impact on the population of aware latent individuals. Figure 5(b) shows the influence of the isolation rate of symptomatic individuals over the population of unaware latent individuals. Increasing the isolation rate of symptomatic individuals shows a significant impact on the population of unaware latent individuals. Figure 5(c) shows the influence of the isolation rate of symptomatic individuals over the population of infectious individuals or symptomatic individuals. Increasing the isolation rate of symptomatic individuals shows a significant impact on the population of infectious individuals or symptomatic individuals. Figure 5(d) shows the influence of an isolation rate of symptomatic individuals over the population of asymptomatic individuals; increasing the isolation rate of symptomatic individuals shows little impact on the population of asymptomatic individuals.

Figure 6(a) shows the influence of the isolation rate of asymptomatic individuals over the population of aware latent individuals; increasing the isolation rate of asymptomatic individuals shows a great impact on the population of aware latent individuals. Figure 6(b) shows the influence of the isolation rate of asymptomatic individuals over the population of unaware latent individuals. Increasing the isolation rate of asymptomatic individuals shows a significant impact on the population of unaware latent individuals. Figure 6(c) shows the influence of the isolation rate of asymptomatic individuals over the population of infectious individuals or symptomatic individuals. Increasing the isolation rate of asymptomatic individuals shows little impact on the population of infectious individuals or symptomatic individuals. Figure 6(d) shows the influence of the isolation rate of asymptomatic individuals over the population of asymptomatic individuals. Increasing the isolation rate of asymptomatic individuals shows the impact on the population of asymptomatic individuals.

Figure 7(a) shows the influence of personal hygiene rate over the population of aware latent individuals; increasing the percentage of personal hygiene rate shows a great impact on the population of aware latent individuals. Figure 7(b) shows the influence of personal hygiene rate over the population of unaware latent individuals; increasing the percentage of personal hygiene rate shows a significant impact on the population of unaware latent individuals. Figure 7(c) shows the influence of personal hygiene rate over the population of infectious individuals or symptomatic individuals; increasing the percentage of personal hygiene rate shows little impact on the population of infectious individuals or symptomatic individuals. Figure 7(d) shows the influence of personal hygiene rate over the population of asymptomatic individuals; increasing the percentage of personal hygiene rate shows little impact on the population of asymptomatic individuals.

5. Conclusion

Our study offers a strong method for preventing the spread of diphtheria by raising awareness and using effective surveillance techniques as recommended by the Centre for Disease Control (2025). In this work, to unriddle the mechanism of diphtheria transmission in the human population, we divide the entire population into different compartments based on the status of the disease. The model has a total population (N), which is subdivided into eight compartments, which include Susceptible individuals (S), Exposed aware individuals (E_a), Exposed unaware individuals (E_u), Symptomatic individuals (I), Asymptomatic individuals (A), Individuals under surveillance (P), Isolated individuals (J), Recovered and immunized individuals (R). The proposed model has been analysed, and both equilibria have been extensively analysed; both are globally asymptotically stable. In the case of endemic equilibrium, it has been shown that it is globally asymptotically stable when the effective reproduction number is greater than unity, while in the case of disease-free equilibrium, it has been shown that it is globally asymptotically stable when the effective reproduction number is less than unity. Numerical simulation has categorically shown that surveillance and awareness play a vital role in controlling diphtheria in the environment. It is therefore recommended that surveillance and awareness should be done in order to control diphtheria in our environment. Further research needs to be done in order to complement this research, especially when it comes to the effect of human mobility on the spread of Diphtheria dynamics.

Data availability

All data used in this work can be found within the manuscript.

References

[1] Centre for academic research (CAR), "CAR 2023", 2023. [Online]. https://www.ijssb.com/index.php/car.

[2] I. S. Fauzi, N. Nural, A. M. Sari, I. B. Wardani, D. Taurustiati, P. Simanullang & B. W. Lestari, "Assessing the impact of booster vaccination on diphtheria transmission: Mathematical modelling and risk zone mapping", Global Impact of Chinese Roots **3** (2024) 234. http://www.keaipublishing.com/idm.

[3] D. Makeri, P. D. Peter & T. Pius, "Understanding the trend of diphtheria outbreak in Nigeria", Diphtheria Outbreak in Nigeria **3** (2023) 42. https://doi.org.10.5958/KJHS-2023-3-2-05.

[4] K. Zakikhany & A. Efstration, "Diphtheria in Europe: current problems and new challenges", Future Microbiology **7** (2012) 595. https://doi.org/10.2217/fmb.12.24.

[5] T. Chao, L. Lu, L. Zhang, R. Huang, Z. Liu, B. Zhou & Y. Liang, "An independent model for specific neutrophil depletion by diphtheria toxin in mice", Science China Life Science **64** (2021) 1227. https://doi.org/10.1007/s/1427-020-1839-3.

[6] A. Skibinski, A. G. David, B. C. Baudner, M. Singh & D. T. O'Hagan, "Combination vaccines", Journal of Global Infectious Diseases **3** (2011) 63. https://doi.org/10.4103/0974-777X.77298.

[7] Mayo Clinic, "Diphtheria 2023", (2023). [Online]. https://www.mayoclinic.org/disease-condition/diphtheria/symptoms-causes/syc-20351897.

[8] Mayo Clinic, "Diphtheria. Causes and symptoms", 2024. [Online]. https://www.mayoclinic.org/disease-conditions/diphtheria/symptoms-cause/syc-20351897.

[9] World Health Organization, "Diphtheria facts sheet", 2023. [Online]. https://www.who.int/news-room/fact-sheets/detail/diphtheria.

[10] A. A. Salim & A. A. Ayoade, "Mathematical modelling of the effect of vaccination on the dynamics of infectious disease", Nepal Journal of Mathematical Sciences (NJMS) **3** (2023) 212. https://doi.org/10.3126/njmathsci.v4/1.53151.

[11] I. Adeifa, "Official statement following the first reported confirmed case of diphtheria in FCT, Abuja", 2023. [Online]. https://ncdc.gov.ng/news/489/officialstatementfollowing/the/firstreported/confirmed/case/ofdiphtheria/inFCT/Abuja.

[12] N. Izzati & A. Andriani, "Dynamical analysis of diphtheria epidemic model with natural immunity rate on exposed individuals", Journal of Physics: Conference Series **1869** (2021) 012117. https://doi.org/10.1088/1742-6596/1869/1/012117.

[13] F. Ilahi & A. Widiana, "The effectiveness of vaccine in the outbreak of diphtheria: mathematical model and simulation", IOP Conference Series: Materials Science and Engineering **434** (2018) 012006. https://doi.org/10.1088/1757-899X/434/1/012006.

[14] N. Izzati, A. Andrinni & R. Robi'aqolbi, "Optimal control of the diphtheria epidemic model with prevention and treatment", Journal of Physics: Conference Series **1663** (2020) 012042. https://do10.1088/1742-6596/1663/1/012042.

[15] F. Lecorvaisier, D. Pontier, B. Soubeyrand & D. Fouchet, "Using a dynamical model to study the impact of a toxoid vaccine on the evolution of a bacterium: The example of diphtheria", Ecological Modelling **487** (2024) 110569. https://doi.org/10.1016/j.ecolmodel.2023.110569.

[16] C. E. Madubueze & K. A. Tijani, "A deterministic mathematical model for optimal control of diphtheria disease with booster vaccination", Healthcare Analytics **3** (2023) 100281. https://doi.org/10.1016/j.health.2023.100281.

[17] L. Valek & I. Tegeder, "Failure of the diphtheria toxin model to induce Parkinson-like behavior in mice", International Journal of Molecular Science **22** (2021) 9494. https://doi.org/10.3390/ijms22179494.

[18] K. Sornbondit, W. Triampo & C. Modchang, "Mathematical modelling of diphtheria transmission in Thailand", Computers in Biology and Medicine **87** (2017) 162. https://doi.org/10.1016/j.compbiomed.2017.05.031.

[19] R. Tosepu, J. Gunawan, D. S. Effendy, O. A. Ahamad & A. Farzan, "The outbreak of diphtheria in Indonesia", The Pan African Medical Journal **31** (2018) 249. https://doi.org/10.11604/pamj.2018.31.24916629.

[20] Z. Islam, S. Ahmed, M. Rahman & M. 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** (2022) 6545179. https://doi.org/10.1155/2022/6545179.

[21] S. O. Adewale, S. O. Ajao, I. A. Olapade, G. A. Adeniran & I. T. Mohammed, "Mathematical analysis of quarantine on the dynamic transmission of diphtheria disease", International Journal of Science and Engineering Investigations **6** (2017) 1. https://arastirmax.com/en/system/files/dergiler/65306/makaleler/6/5/arastirmax-mathematical/analysis/quarantine/dynamical/transmission/diphtheria/disease.pdf.

[22] N. Rahmi & M. Pratama, "Model analysis of diphtheria disease transmission with immunization, quarantine, and hand washing behavior", Journay Teori dan Aplikasi Matematikal (JTAM) **7** (2023) 462. https://doi.org/10.31764/jtam.v7;2.13466.

[23] J. E. Ngozika, E. B. Akponana, E. O. Arierhie & A. M. Okedoye, "Mathematical analysis of spread and control of diphtheria with emphasis on diphtheria antitoxin efficiency", Journal of Theoretical and Applied Sciences **2** (2024) 152. https://doi.org/10.59324/ejtas.

[24] F. A. Oguntolu, O. J. Peter, A. Yusuf, "Mathematical model and analysis of the soil-transmitted helminth infections with optimal control", Model. Earth Syst. Environ. **10** (2024) 883. https://doi.org/10.1007/s40808-023-01815-1.

[25] W. Atkinson, "Diphtheria epidemiology and prevention of vaccine-preventable diseases", Public Health Foundation **12** (2012) 215. https://www.cdc.gov/vaccines/pubs/pinkbook/dip.html.

[26] Centre for Disease Control (CDC), "Diphtheria surveillance and trends", 2025. [Online]. https://www.cdc.gov/diphtheria/php/surveillance/index.html.

[27] J. Andrawus, A. Abubakar, A. Yusuf, "Impact of public awareness on haemo-lyphatic and meningo-encephalitic stage of sleeping sickness using mathematical model approach", Eur. Phys. J. Spec. Top. **2024** (2024) 1. https://doi.org/10.1140/epjs/s11734-024-01417-7.

[28] J. Andrawus, M. A. Iliyasu Muhammad, D. B. Akawu Denue, H. Abdul, A. Yusuf & S. Salahshour, "Unraveling the importance of early awareness strategy on the dynamics of drug addiction using mathematical modeling approach", Chaos: An Interdisciplinary Journal of Nonlinear Science **34** (2024) 1. https://doi.org/10.1063/5.0203892.

[29] C. Castillo-Charvez & B. Song, "Dynamical model of tuberculosis and their applications", Math. Biosci **1** (2004) 361. https://doi.org/10.3934/mbe.2004.1.361.

[30] K. G. Ibrahim, J. Andrawus, A. Abubakar et al., "Mathematical analysis of chickenpox population dynamics unveiling the impact of booster in enhancing recovery of infected individuals", Model. Earth Syst. Environ. **11** (2025) 46. https://doi.org/10.1007/s40808-024-02219-5.

[31] P. Van De Driessche & J. Watmough, "Reproduction number and sub threshold endemic equilibrium for compartmental models of disease transmission", Mathematical Biosciences **180** (2002) 29. https://doi.org/10.1016/s0025-5564(02)00108-6.

[32] O. J. Peter, H. S. Panigoro, A. Abidemi, "Mathematical model of COVID-19 pandemic with double dose vaccination", Acta Biotheor **71** (2023) 9. https://doi.org/10.1007/s10441/023/09460/y.

[33] R. Musa, O. J. Peter & F. A Oguntolu, "A non-linear differential equation model of COVID-19 and seasonal influenza co-infection dynamics under vaccination strategy and immunity waning", Healthcare Analytics **4** (2023) 100240. https://doi.org/10.1016/j.health.2023.100240.

[34] O. Diekmann, J. A. P. Heesterbeek & M. G. Roberts, "The construction of next-generation matrices for computational epidemic models", J R Soc Interface **7** (2010) 873. https://doi.org/10.1098/rsit.2009.0386.

[35] B. A. Eloho, J. E. Ngozika & M. A. Okedoye, "Mathematical modeling of diphtheria transmission dynamics for effective strategies of prevention and control with emphasis on impact of vaccination and waxing immunity", International Journal of Research in Engineering and Science (IJRES) **12** (2024) 1. https://www.ijres.org/papers/Volume-12/Issue-7/1207142153.pdf.

[36] S. M. Surayya & F. M. Hamza, "Biology and life sciences forum. Trends and geographical distribution of diphtheria in Nigeria", Re-Emerging Disease **2023** (2023) 1. https://doi.org/10.3391/ECM2023-16693.

[37] J. Andrawus & F. E. Yakubu, "Mathematical analysis of a model for syphilis endemicity", International Journal of Scientific Engineering and Applied Science (IJSEAS) **3** (2017) 48. https://ijseas.com/volume3/v3i8/ijseas20170804.pdf.

[38] J. P. Lasalle, "The stability of dynamical systems", Regional Conference Series in Applied Mathematics, SIAM, Philadelphia **1976** (1976) 1. https://epubs.siam.org/doi/book/10.1137/1.9781611970432.

[39] Y. U. Ahmad, J. Andrawus, A. Ado, Y. A. Maigoro, A. Yusuf, S. Althobaiti & U. T. Mustapha, "Mathematical modelling and analysis of human-to-human monkeypox virus transmission with post-exposure vaccination", Modeling Earth Systems and Environment **10** (2024) 1. https://doi.org/10.1007/s40808-023-01920-1.

[40] J. Andrawus, A. Yusuf, U. T. Mustapha, A. S. Alshom-rani & D. Baleanu, "Unravelling the dynamics of Ebola virus with contact tracing as control strategy", Fractals **31** (2023) 10. https://doi.org/10.1142/S0218348X2340159X.

[41] J. Andrawus, Y. U. Ahmad, A. A. Andrew, A. Yusuf, S. Qureshe, B. Akawu, H. Abdul & S. Salahshour, "Impact of surveillance in human-to-human transmission of monkeypox virus", The European Physical Journal Special Topics **234** (2024) 1. https://doi.org/10.1140/epjs/s11734-024-01346-5.

[42] U. T. Mustapha, A. I. Muhammad, A. Yusuf, A. Nesreen, A. I. Aliyu & J. Andrawus, "Mathematical dynamics of meningococcal meningitis: Examining carrier diagnosis and prophylaxis treatment", Int. J. Appl. Comput. Math **11** (2025) 77. https://doi.org/10.1007/s40819-025-01890-1.