Mathematical Modelling for the Transmission Dynamics of blinding Trachoma

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Abstract

Trachoma is an eye infectious disease caused by Chlamydia Trachomatis bacterium, which may lead to irreversible blindness. The disease is spread directly or indirectly by contacting a contaminated material. It can also be transmitted through the disease vector known as “Musca sorbens” or “Bazaar fly”. To curtail the spread of the disease in a population, meaningful information on the spread and possible control of the disease is required. Mathematical modeling provides efficient tools that can be used to understand and analyze the dynamics of the disease and its control. Several compartmental epidemic models have been proposed in the literature to study the dynamics of trachoma; including SI, SIR and SEIR. However, majority of the existing trachoma models consider only person to person transmission. Thus, the information provided by such models is insufficient since they did not capture the disease vector transmission. The current study proposed a novel SEIR-SEI model that consider both person-person and vector transmission dynamics. The threshold quantity, basic reproduction number $R_0$ is obtained using the next generation matrix, and it was proved that the disease-free equilibrium is asymptotically stable when $R_0 < 1$, and the endemic equilibrium is globally asymptotically stable when $R_0 > 1$. Some simulation results with the aid of mesh plots for the reproductive number as a function of two different biological parameters were obtained. Furthermore, a comprehensive sensitivity analysis is conducted to identify the influence of the individual parameters on the $R_0$. Numerical results show that the vector contact rate $\sigma_f$ has the highest sensitivity with respect to $R_0$, and the value of $R_0$ increases with increase in $\sigma_f$, hence, the disease can be controlled by decreasing the vector contact rate. Similarly, improving the rate of environmental hygiene and facial cleanliness will decrease the size of $R_0$ and result in the declination of the disease transmission. Moreover, a detailed parameter estimation of the model parameters and model fitting was presented with the use of field data cases from Northern Nigeria using least-square fitting method. The study provides alternative tools that can be used for planning trachoma control program to achieve global eradication of trachoma as a public health challenge as targeted by WHO in 2030.

Keywords: Trachoma Model, Reproduction number, Chlamydia Trachomatis, Stability, sensitivity analysis, Estimation, Model fitting.


1 Introduction

Trachoma has been one of the 17 neglected tropical diseases (NTDs) given priority for surveillance and elimination by the World Health Organization (WHO) via protective therapies or improved prevention and treatment approaches [20], [20, 11]. Spread of tropical disease is enabled by poverty-related lifestyle, like poor housing conditions, inadequate hygiene, access to insufficient safe drinking water access and basic medical facilities [9].

The blinding trachoma has been a primary source of visual impairment which reportedly affects millions of people in 51 endemic settings [19]. It affects approximately 2.2 million individuals with visual impairment, of which nearly 1.2 million remain permanently blind [19, 21]. Active Trachoma, as reported in the simplified scheme of the world health organization [15], arises only when infection with the Chlamydia trachomatis bacterium occurs. Prolonged exposure to these organisms result in an immunopathological reaction identified through redness or scaring of tarsal conjunctiva, and subsequent twirling of the eyelashes that damage the surface of the cornea. And this may contribute to trachomatous trichiasis (TT), opacity of the cornea (CO), as well as permanent and irreversible blindness. Trachoma is also found to be significantly associated with morbidity and mortality [11]. The Disability Adjusted Life Years “DALY” estimates that were attributable to trachoma seem to be dynamic. The 1990 Global Burden of Disease (GBD) relevant literature reveal that the trachoma prevalence (all age groups) of about 144000.00 [95 percent interval (95 percent U.I), 104000.00 to 189000.00] DALY. While 2010 “GBD” research recorded about 334000.00 [95 percent U.I 243000.00 to 438000.00] [12]. Some scholars determine the number as minimum DALY of 1 million [6]. Sub-Saharan Africa produced as much as 3.6 million, with the highest percentage (72 percent) [7]. In 2010, Trachoma accounted for 5.2 percent Africa’s cause of visual impairment [13]. Nonetheless, a reliable quantitative estimation of the trachoma load persists and is attributed to a numerous of factors, such as the limited data that restricts the ability to get reliable estimation on the number of infected individuals and the unanswered question about the status of trichiasis, “a sequela for debilitating disease?” [4].

Chlamydia T. can be transmitted via 2 main ways. One of those is direct physical contacts with infectious person or by interaction with clothes that encountered contaminated eye discharges [4]. Other path includes transmission through an eye-seeking fly [musca sobens] wich touched the discharge from the eyes or nose of an infected individual [5]. In order to sustain infection transmission it shall constantly be transmitted between individuals. The conditions of untreated patents depends on such factors like aging as well as the length of exposure to the infection [2, 38]. The rate and the spread of the C. Trachomatis is also age-dependent; thus, at the childhood stage infections are higher [2, 38]. Repetitive aged-related inflammation contributes to scar on the tarsal conjunctiva and eventually to tracasis, then Opacities of the cornea and total blind conditions as described earlier [4, 18]. So many epidemiology studies indicated that extreme sequela mostly T. tracasis and C. opacity involve nursing mothers and predominantly relative to that of men because of their much exposures to infectious reservoirs [18, 17]. Wide range of trachoma transmission influences were classified, these include: (i). Eye secretions with which other members of society could come into direct contact and that it may as well spontaneously ignite flies that enhance the spread [5, 14]; (ii). Crowded household that increases a likelihood of contact between members of society that may give rise to much trachoma incidences [1, 14], (iii). Insufficient of clean drinking water which result in inadequate facial wash, overall unhygienic habits and limited or inconvenient access to toilet facilities, which can result in the build-up of fecal contamination in the area attracting eye-seeking flies [5, 14]. Trachoma spread frequency among such communities is categorized in 1-9-year-olds based on active trachoma rates. Societies are considered hyperendemic when infectious disease burden becomes
greater than 20 percent in this age category, mesoendemic when the incidence reaches more than ten percent but not up to 20 percent, and hypoendemic when prevalence is below ten percent [22, 10].

The WHO set to promote trachoma eradication program before 2020 and the Global Alliance for the eradication of Trachoma in 2020 (GET 2020) has been formed for creating guidelines so as to assist the accomplishment of the goal. Such conditions includes: (i) helping to reduce the incidence rates of [TF/TI] (active trachoma) throughout the endemic populations to below five percent among 1- 9-years age group; (ii) decreasing the incidence of TT less than 1 per 1000 persons and (iii) improving use of the components of the SAFE policy (F) and (E) [16]. World Health Organization fully supports a proper realization of the SAFE strategy for treating and controlling trachoma. As stated earlier, it includes four main components: (1) correction of trachomatous trichiasis through surgeries; (2) proper administration of antibiotic excessively (azithromycin for adults) or (tropical tetracycline on pediatrics) for the clearance of active trachoma. (3) promoting facial cleanliness with a view to reducing the spread mostly through eye discharge, and, (4) improving environmental changes with a view to improving living standards; this can be made achievable only through some kind of arenas, like supporting a very good water supply, providing access to the well-designed toilet facilities, helping to reduce the fly density and too much crowding [18, 16].

Modeling and simulation have been used to capture the dynamics of the neglected tropical diseases (specifically, Blinding Trachoma). These models described not only the infectious disease mathematical process but this do provide meaningful information on the possible control and spread of the disease[31]. However, majority of the existing models found in the current literature concentrate their studies only on simple SI, SEI, SEIR (human – human transmission) compartmental models [23, 24, 29, 37], and none of the model has ever considered a Host-Vector transmission dynamic of trachoma, where the contact rate of the disease vector plays a significant role in curtailing the prevalence of trachoma in the populace. This might have made their analysis trivial and thus, not reliable enough to judge the situation. Therefore, it remains a challenging task designing more sophisticated models that can provide useful methods to analyze and describe the nature and control of trachoma. Nevertheless, it is tedious to design a complicated model of this kind to highlight management strategies for trachoma epidemic.

The current study proposed a novel multi-strain SEIR-SEI model that consider both person-person and disease vector transmission to provide some insights on the dynamics characteristics of the trachoma and propose suitable control interventions toward achieving the 2030 goals set by World Health Organization to walkaway trachoma as world’s public health challenge. we have also performed parameter estimation of the model parameters and model fitting with the use of field data cases from Northern Nigeria using least-square fitting method, Some simulation results produced with the aid of mesh plots for the reproductive number as a function of two different biological parameters was obtained. Our new research article complimented some of the earlier mentioned studies in the literature (specifically, [23, 24, 29, 37] Where, the novel $SEI_{(h,c)}R – SEI$ model incorporates the following extensions: (i) Multi strain infectious classes (ii) Disease-Vector population dynamic (iii) Model Fitting and parameter estimation with real cases (iv) The sensitivity analysis, to highlight the influence of each parameter in controlling the epidemic.
2 Formulation and description of the Trachoma model

The model is considered to be a compartmental model comprising of two groups population with two strain of infectious stages: Population of human and population of eye-seeking flies. The human population at time $t$, $N_h(t)$ is sub divided into five classes. viz; $S_h(t)$ Class of Susceptible human who are healthy but can become infected through direct or indirect contact with infectious individual and infectious flies respectively. This class was increased through the recruitment of individuals at the rate $\Pi_h$ and by the loss of infection-acquired temporary immunity at the rate $\phi$ and it was decreased by progression to exposed class at the rate $\lambda_h$ and by natural death at $\mu_h$. $E_h(t)$ the exposed class of human population which was generated through the infection of the susceptible individuals at the rate $\lambda_h$ and decreased by progression to show the symptoms of active trachoma at the rate $\delta_1$ and decreased by progression to the recovered class at the rate $\psi_1$ and by natural death at the rate $\mu_h$. $I_h(t)$ the atage one of infectious class of human population which comprises (i) trachomatous inflammation follicle/intense stage of the trachoma (TF/TI), and (ii) trachomatous scarring stage (TS), this class is generated by population of exposed individuals who develop symptoms of the chlamydia trachomatis at $\delta_1$ and decreased by progression to the next infectious stage at the rate $\delta_2$ and by natural death $\mu_h$ its also decreased through progression to the removed class at rate $\psi_2$. $R_h(t)$ the population of recovered individuals which was generated by the removal of individuals from infectious stages at the end of infectiousness due to the application of one of the control measures mentioned above (SAFE) at rates $\psi_1$, $\psi_2$, and $\psi_3$. This class is decreased by loss of temporary immunity at rate $\phi$ and natural death at rate $\mu_h$. Assuming the infection does not confer permanent immunity to re-infection in the recovered class (i.e $\phi \neq 0$).

Similarly the flies population $N_f(t)$ was sub-divided into three disjoint compartment namely; Susceptible flies $S_f(t)$ generated by the recruitment rate $\Pi_f$ and decreased by progression to exposed class at the rate $\lambda_f$ and natural death at $\mu_f$, the exposed flies $E_f(t)$ which is increased by susceptible flies population at the rate $\lambda_f$ and decreased by progression to infectious stage and natural death at the rates $\tau$ and $\mu_f$ respectively. Infectious flies $I_f(t)$ this produced when the exposed class shows clinical symptoms of Chlamydia trachomatis and reduced by natural death at the rate $\tau$ and $\mu_f$ respectively. the period of infection of flies usually ends with their natural death due to nature of their petite life-cycle(i.e they dos’int recover from infections), the reason why immune class does not appear in the flies population.

The total human and flies population of this model is presented as:

$$N_h(t) = S_h(t) + E_h(t) + I_{hs}(t) + I_{hc}(t) + R_h(t)$$

and

$$N_f(t) = S_f(t) + E_f(t) + I_f(t)$$

respectively.

Using the above description and assumptions the model for the transmission dynamic of blinding trachoma, the model’s associated deterministic system of non-linear ordinary differential equation is
Table 1: The state variables and parameters values used in the Trachoma model

| Variable | Interpretation |
|----------|----------------|
| $S_h(t)$ | Population of susceptible humans |
| $E_h(t)$ | Population of exposed humans |
| $I_{hs}(t)$ | Class of infective individuals with early stages of trachoma |
| $I_{hc}(t)$ | Class of infective individuals with TT and CO stages of trachoma |
| $R_h(t)$ | Population of Recovered humans |
| $S_f(t)$ | Class of susceptible flies |
| $E_f(t)$ | Population of exposed flies |
| $I_f(t)$ | Class of infective flies |

| Parameter | Description |
|-----------|-------------|
| $\Pi_h$  | Human recruitment rate |
| $\mu_h$  | Natural death rate of human |
| $\mu_f$  | Natural death rate of eye-seeking fly (musca sorvens) |
| $\Pi_f$  | Flies recruitment rate |
| $\beta_h$ | Rate of transmission from vector to host |
| $\beta_f$ | Rate of transmission from host to vector |
| $\delta_1$ | Progression rate from $E_h$ to $I_{hs}$ |
| $\delta_2$ | Progression rate from $I_{hs}$ to $I_{hc}$ |
| $\tau$   | Progression rate from $E_f$ to $I_f$ |
| $\varphi$ | Rate at which human looses immunity |
| $\sigma_f$ | Contact Rate of the infected fly |
| $\psi_1$ | Human recovery rate from $E_h$ |
| $\psi_2$ | Human recovery rate from $I_{hs}$ |
| $\psi_3$ | Human recovery rate from $I_{hc}$ |
presented as:

\[
\begin{align*}
\frac{dS_h}{dt} &= \Pi_h - \lambda_h S_h + \varphi R_h - \mu_h S_h \\
\frac{dE_h}{dt} &= \lambda_h S_h - \delta_1 E_h - \mu_h E_h - \psi_1 E_h \\
\frac{dI_{hs}}{dt} &= \delta_1 E_h - \delta_2 I_{hs} - \mu_h I_{hs} - \psi_2 I_{hs} \\
\frac{dI_{hc}}{dt} &= \delta_2 I_{hs} - \psi_3 I_{hc} - \mu_h I_{hc} \\
\frac{dR_h}{dt} &= \psi_1 E_h + \psi_2 I_{hs} + \psi_3 I_{hc} - \varphi R_h - \mu_h R_h \\
\frac{dS_f}{dt} &= \Pi_f - \lambda_f S_f - \mu_f S_f \\
\frac{dE_f}{dt} &= \lambda_f S_f - \tau E_f - \mu_f E_f \\
\frac{dI_f}{dt} &= \tau E_f - \mu_f I_f
\end{align*}
\]

(1)

where, the forces of infection

\[
\lambda_h = (\beta_h \sigma_f \frac{I_f}{N_h}), \quad \lambda_f = \sigma_f \beta_f \left(\frac{I_{hs} + I_{hc}}{N_h}\right)
\]

3 Basic property of the model

Model (1) is monitoring human and flies population. Therefore every parameter and state variable associated with the model are considered to be non-negative for each \( t > 0 \). It is therefore instructive to show that all the state variables belonging to the model are non-negative for all non-negative initial condition before analysing the model.

**Lemma 3.1.** suppose \((S_h, E_h, I_{hs}, I_{hc}, R_h, S_f, E_f, I_f)\) is the solution of the system (1) together with the initial condition, \( S_h(0) \geq 0, E_h(0) \geq 0, I_{hs}(0) \geq 0, I_{hc}(0) \geq 0, R_h(0) \geq 0, S_f(0) \geq 0, E_f(0) \geq 0, I_f(0) \geq 0 \), the closed set;

\[
\Omega = \left\{(S_h, E_h, I_{hs}, I_{hc}, R_h, S_f, E_f, I_f) \in \mathbb{R}^8_+ \mid S_h + E_h + I_{hs} + I_{hc} + R_h = \frac{\Pi_h}{\mu_h}, S_f + E_f + I_f = \frac{\Pi_f}{\mu_f} \right\}
\]

is positively-invariant and attractive with respect to the model (1)

**Proof.** Adding the first five equations in (1) we’ve

\[
\frac{dN_h}{dt} = \Pi_h - \mu_h N_h
\]

(2)

Where,

\[
N_h = S_h + E_h + I_{hs} + I_{hc} + R_h
\]
similarly adding the last three equations in (1) we’ve
\[
\frac{dN_f}{dt} = \Pi_f - \mu_f N_f
\]  
where,
\[
N_f = S_f + E_f + I_f
\]
since, \(\frac{dN_h}{dt} = \pi_h - \mu_h N_h\) it follows that \(\frac{dN_h}{dt} \leq 0\) if \(N_h(t) \geq \frac{\Pi_h}{\mu_h}\), it can be seen that the solution \(N_f\) of (3) approaches the equilibrium \(\frac{\Pi_f}{\mu_f}\) as \(\mu_f \to \infty\). By employing the idea of comparison theorem (see [26]) we’ve, \(N_h(t) \leq N_h(0) e^{-\mu_h t} + \frac{\Pi_h}{\mu_h} (1 - e^{-\mu_h t})\) particularly \(N_h(t) \leq \frac{\Pi_h}{\mu_h}\) if \(N_h(0) \leq \frac{\Pi_h}{\mu_h}\), this implies the set \(\Omega\) is positively-invariant. Thus if \(N_h(t) > \frac{\Pi_h}{\mu_h}\) then the solution either enters \(\Omega\) in finite time or \(N_h(t)\) approaches \(\frac{\Pi_h}{\mu_h}\). Hence all the solutions in \(\mathbb{R}_+^8\) approaches, enter or stay in \(\Omega\). this implies \(\Omega\) is attracting. Consequently the model is considered to be mathematically well-posed and epidemiologically sensible, as all the variables used throughout the model happen to be non-negative for every \(t > 0\). Thus it is adequate and enough to consider the dynamics of the model (1) in \(\Omega\) [27].

4 Existence of the Equilibria and Stability Analysis

In any research of epidemiological models there exist a threshold value usually known as basic reproduction number \(R_0\) which is described as the average number of secondary infections generated by a single infected individuals during the epidemic in a completely susceptible population.[27, 33, 36]

4.1 The Disease-free equilibrium point

To obtain the disease-free equilibrium point (DFE) for the model (1) we set the (RHS) of the equations in (1) to zero, and we’ve
\[
\varepsilon_0 = (S_h, E_h, I_{hs}, I_{hc}, R_h, S_f, E_f, I_f) = (S_h, 0, 0, 0, S_f, 0, 0) = \left(\frac{\Pi_h}{\mu_h}, 0, 0, 0, \frac{\Pi_f}{\mu_f}, 0, 0\right)\]  
that is when there is no infection \(E_h = I_{hs} = I_{hc} = E_f = I_f = 0\) the model has a unique disease-free equilibrium point
\[
\varepsilon_0 = \left(\frac{\Pi_h}{\mu_h}, 0, 0, 0, \frac{\Pi_f}{\mu_f}, 0, 0\right)
\]  

4.2 Local Stability of the Disease-free Equilibrium

The technique of next-generation operator as presented by [35] is employed to determine a critical parameter called the Basic reproduction number \(R_0\) which represent the average number of secondary cases produced by one infected agent throughout the duration of infectious period in a complete susceptible population. The authors used the recipe developed by Van den Driessche and Wanmough, J [28, 34], we can re-write the equations of the model(1) starting with the newly infective
To investigate the linear stability of the diseases-free equilibrium (DFE), we apply the technique of next generation matrix operator to the system (5) [28, 34]. The jacobian matrices $f$ and $v$ which are evaluated at disease-free equilibrium (DFE) denoted ($F$) the matrix of new infection terms and ($V$) the matrix for the remaining transition terms, in relation to the model are given by:

$$
\begin{align*}
\frac{dE_h}{dt} &= \lambda_h S_h - \delta_1 E_h - \mu_h E_h - \psi E_h \\
\frac{dI_{hs}}{dt} &= \delta_1 E_h - \delta_2 I_{hs} - \mu_h I_{hs} - \psi I_{hs} \\
\frac{dI_{hc}}{dt} &= \delta_2 I_{hs} - \psi I_{hc} - \mu_h I_{hc} \\
\frac{dE_f}{dt} &= \lambda_f S_f - \tau E_f - \mu_f E_f \\
\frac{dI_f}{dt} &= \tau E_f - \mu_f I_f \\
\frac{dS_h}{dt} &= \Pi_h - \lambda_h S_h + \varphi R_h - \mu_h S_h \\
\frac{dS_f}{dt} &= \Pi_f - \lambda_f S_f - \mu_f S_f
\end{align*}
$$

(5)

$$
\begin{align*}
F &= \begin{bmatrix}
\beta h \sigma_f \frac{I_{f}}{N_h} S_h \\
0 \\
0 \\
\sigma_f \beta_f \frac{(I_{hs} + I_{hc}) S_f}{N_h} \\
0
\end{bmatrix}, \quad v = \begin{bmatrix}
(\delta_1 + \mu_h + \psi) E_h \\
-\delta_1 E_h + (\delta_2 + \psi + \mu_h) I_{hs} \\
-\delta_2 I_{hs} + (\psi + \mu_h) I_{hc} \\
(\tau + \mu_f) E_f \\
-\tau E_f + \mu_f I_f
\end{bmatrix}
\end{align*}
$$

(6)

$$
\begin{align*}
F &= \begin{bmatrix}
0 & 0 & 0 & 0 & \beta h \sigma_f \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & \sigma_f \beta_f \mu_h S_f & \sigma_f \beta_f \mu_h \pi_f & 0 & 0 \\
0 & 0 & 0 & 0 & 0
\end{bmatrix}, \quad V = \begin{bmatrix}
P_1 & 0 & 0 & 0 & 0 \\
-\delta_1 & P_2 & 0 & 0 & 0 \\
0 & -\delta_2 & P_3 & 0 & 0 \\
0 & 0 & 0 & P_4 & 0 \\
0 & 0 & 0 & -\tau & \mu_f
\end{bmatrix}
\end{align*}
$$

(8)
4.3 Endemic Equilibrium Point

and the trachoma can be eliminated from the population). we summarized the following:

\[ R \]

The following result is then established by applying theorem (2) of [28] (i.e if the basic reproduction number of the model is less than unity, it means the disease-free equilibrium is locally asymptotically stable and the trachoma can be eliminated from the population). we summarized the following:

\[ FV^{-1} = \begin{pmatrix} 0 & 0 & 0 & \frac{\beta h \sigma f \tau}{P_4 \mu f} & \frac{\beta h \sigma f}{\mu f} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ \frac{\beta f \sigma f \mu h \Pi f \delta_1}{\mu f \Pi f P_2} & \frac{\beta f \sigma f \mu h \Pi f \delta_2 \delta_1}{\mu f \Pi f P_2 P_3} & \frac{\beta f \sigma f \mu h \Pi f \beta h \delta_1 \mu h (P_3 + \delta_2) \sigma f}{\mu f \Pi f P_4} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix} \]

where:

\[ P_1 = \delta_1 + \mu h + \psi, \quad P_2 = \delta_2 + \mu h + \psi, \]
\[ P_3 = \mu h + \psi, \quad P_4 = \tau + \mu f, \quad P_5 = \mu h + \varphi, \]

It can be seen that the basic reproduction number of the model is \( R_0 = \rho(FV^{-1}) \), \( \rho \) stands for the spectral radius of the matrix, \( FV^{-1} \). And therefore the basic reproduction number is given by:

\[ R_0 = \frac{\sqrt{P_2 P_1 P_3 P_4 \Pi f \beta f \beta h \delta_1 \mu h (P_3 + \delta_2) \sigma f}}{P_2 P_1 P_3 P_4 \Pi h \mu f} \]

The following result is then established by applying theorem (2) of [28] (i.e if the basic reproduction number \( R_0 \) is less than unity, it means the disease-free equilibrium is locally asymptotically stable and the trachoma can be eliminated from the population). we summarized the following:

**Theorem 4.1.** The disease-free equilibrium "DFE" of the trachoma model(1) is locally asymptotically stable "LAS" if \( R_0 < 1 \) and unstable if \( R_0 > 1 \).

4.3 Endemic Equilibrium Point

Suppose \( \varepsilon^* = \left( S_h^*, E_h^*, I_h^*, I_{hc}, R_h, S_f, E_f, I_f \right) \) is the steady state of \( \varepsilon_0 = (S_h, E_h, I_{hs}, I_{hc}, R_h, S_f, E_f, I_f) \). the endemic equilibrium point the model (1) in terms of the forces of infection, \( \lambda_h^* = (\beta h \sigma f \frac{I_f}{N_h}) \) and \( \lambda_f^* = \sigma f \beta f (\frac{I_{hs} + I_{hc}}{N_h}) \) is obtained by setting the system (1) to zero and solve in terms of the forces of infection \( \lambda_h^* \) and \( \lambda_f^* \) as follows:
\[ S_h^* = \frac{\Pi_h P_5 P_3 P_2 P_1}{(\lambda_h^* + \mu_h) P_1 P_2 P_3 P_5 - K \phi \psi \lambda_h^*} \]

\[ E_h^* = \frac{\lambda_f^* \Pi_h P_5 P_3 P_2}{(\lambda_h^* + \mu_h) P_1 P_2 P_3 P_5 - K \phi \psi \lambda_h^*} \]

\[ I_{hs}^* = \frac{\lambda_f^* \delta_1 \Pi_h P_5 P_3}{(\lambda_h^* + \mu_h) P_1 P_2 P_3 P_5 - K \phi \psi \lambda_h^*} \]

\[ I_{hc}^* = \frac{\lambda_f^* \delta_2 \delta_1 \Pi_h P_5}{(\lambda_h^* + \mu_h) P_1 P_2 P_3 P_5 - K \phi \psi \lambda_h^*} \]

\[ R_h^* = \frac{\lambda_f^* \Pi_h \psi K}{(\lambda_h^* + \mu_h) P_1 P_2 P_3 P_5 - K \phi \psi \lambda_h^*} \]

\[ S_f^* = \frac{\Pi_f}{(\lambda_f^* + \mu_f)} \]

\[ E_f^* = \frac{\lambda_f^* \Pi_f}{(\lambda_f^* + \mu_f) P_4} \]

\[ I_f^* = \frac{\tau \lambda_f^* \Pi_f}{\mu_f (\lambda_f^* + \mu_f) P_4} \]

where:

\[ P_1 = \delta_1 + \mu_h + \psi, P_2 = \delta_2 + \mu_h + \psi, P_3 = \mu_h + \psi, \]

\[ P_4 = \tau + \mu_f, P_5 = \mu_h + \varphi, K = (P_2 P_3 + P_3 \delta_1 + \delta_1 \delta_2) \]

By substituting putting the expression for \( I_f^* \) into \( \lambda_h^* \) and that of \((I_{hs}^* + I_{hc}^*)\) into \( \lambda_f^* \), we obtained:

\[ \lambda_h^* = \frac{\beta_h \sigma_f \mu_h \tau \lambda_f^* \Pi_f}{\Pi_h \mu_f P_4 (\lambda_f^* + \mu_f)} \]

and

\[ \lambda_f^* = \frac{\beta_h \sigma_f \mu_h \lambda_h \delta_1 P_5 P_6}{(\lambda_h + \mu_h) P_1 P_2 P_3 P_5 - K \phi \psi \lambda_h} \]

where:

\[ P_6 = P_3 + \delta_2 \]

Substituting (14) into (13), we can easily see that the equilibria of the model satisfy the following linear equation in terms of \((\lambda_h^*)\).

\[ a_0 (\lambda_h^*) + b_0 = 0 \]

where \( a_0 = \mu_f P_5 P_6 / \beta_h \delta_1 \mu_h \sigma_f P_3 + \mu^2_f P_4 \Pi_h P_1 P_2 P_3 P_5 - \mu^2_f P_4 \Pi_h \phi \psi K \) and

\[ b_0 = \omega (R_0^2 - 1), \omega = \frac{\rho_5 \mu_h}{\Pi_f \delta_1 \tau \mu_h \sigma_f \lambda_f^*} \]

It is clear that \( a_0 \) is positive, and \( b_0 > 0 \) provided \( R_0 > 1 \). Hence there exist an endemic equilibrium when \( R_0 > 1 \). To summarize the above results we have the following lemma.
Lemma 4.2. The model (1) usually has a disease-free equilibrium and a unique endemic equilibrium whenever $R_0 > 1$.

4.4 Global Stability of the Endemic Equilibrium ($\varepsilon^*$)

**Theorem 4.3.** When $R_0 > 1$ the endemic equilibrium ($\varepsilon^*$) is "GAS" globally asymptotically stable and not stable if $R_0 < 1$.

**Proof.**

Let us define a Lyapunov function of the form

$$U = c_1 \left[ S_h - S_h^* - S_h^* \ln \left( \frac{S_h}{S_h^*} \right) \right] + c_1 \left[ E_h - E_h^* - E_h^* \ln \left( \frac{E_h}{E_h^*} \right) \right] + c_2 \left[ I_{hs} - I_{hs}^* - I_{hs}^* \ln \left( \frac{I_{hs}}{I_{hs}^*} \right) \right]$$

$$+ c_3 \left[ I_{hc} - I_{hc}^* - I_{hc}^* \ln \left( \frac{I_{hc}}{I_{hc}^*} \right) \right] + c_4 \left[ S_f - S_f^* - S_f^* \ln \left( \frac{S_f}{S_f^*} \right) \right] + c_4 \left[ E_f - E_f^* - E_f^* \ln \left( \frac{E_f}{E_f^*} \right) \right]$$

$$+ c_5 \left[ I_f - I_f^* - I_f^* \ln \left( \frac{I_f}{I_f^*} \right) \right]$$

where $c_1, c_2, c_3, c_4, c_5$ are some determining constants. Then computing the derivative of $U$ along the solutions of the system (1), we've

$$\dot{U} = c_1 \left( 1 - \frac{S_h^*}{S_h} \right) \dot{S}_h + c_1 \left( 1 - \frac{E_h^*}{E_h} \right) \dot{E}_h + c_2 \left( 1 - \frac{I_{hs}^*}{I_{hs}} \right) \dot{I}_{hs} + c_3 \left( 1 - \frac{I_{hc}^*}{I_{hc}} \right) \dot{I}_{hc} + c_4 \left( 1 - \frac{S_f^*}{S_f} \right) \dot{S}_f$$

$$+ c_4 \left( 1 - \frac{E_f^*}{E_f} \right) \dot{E}_f + c_5 \left( 1 - \frac{I_f^*}{I_f} \right) \dot{I}_f$$

from (17) we have that

$$c_1 \left( 1 - \frac{S_h^*}{S_h} \right) \dot{S}_h = c_1 \left( \lambda_h^* S_h^* + \mu_h S_h^* - \lambda_h S_h - \mu_h S_h \right)$$

$$= c_1 \left( 1 - \frac{S_h^*}{S_h} \right) \left[ \left( \lambda_h^* S_h^* - \lambda_h S_h \right) - \left( \mu_h S_h - \mu_h S_h^* \right) \right]$$

$$= c_1 \lambda_h S_h^* \left( 1 - \frac{S_h}{S_h^*} \right) \left( 1 - \frac{\lambda_h S_h}{\lambda_h^* S_h^*} \right) - \frac{\mu_h}{S_h} (S_h - S_h^*)^2$$

$$\leq c_1 \lambda_h S_h^* \left( 1 - \frac{S_h}{S_h^*} \right) \left( 1 - \frac{\lambda_h S_h}{\lambda_h^* S_h^*} \right)$$

and

$$c_1 \left( 1 - \frac{E_h^*}{E_h} \right) \dot{E}_h = c_1 \left( 1 - \frac{E_h^*}{E_h} \right) \left( \lambda_h S_h - \lambda_h S_h^* \frac{E_h}{E_h^*} \right)$$

$$= c_1 \lambda_h S_h^* \left( 1 - \frac{E_h}{E_h^*} \right) \left( \frac{\lambda_h S_h}{\lambda_h S_h^*} - \frac{E_h}{E_h^*} \right)$$

(18)
Likewise
\[
c_2 \left( 1 - \frac{I_{hs}^*}{I_{hs}} \right) \dot{I}_{hs} = c_2 \left( 1 - \frac{I_{hs}^*}{I_{hs}} \right) \left( \delta_1 E_h - \delta_1 E_{hs}^* \frac{I_{hs}}{I_{hs}^*} \right) 
\]
\[
= c_2 \delta_1 E_h \left( 1 - \frac{I_{hs}^*}{I_{hs}} \right) \left( \frac{E_h}{E_h} - \frac{I_{hs}}{I_{hs}^*} \right) 
\]
and
\[
c_3 \left( 1 - \frac{I_{hc}^*}{I_{hc}} \right) \dot{I}_{hc} = c_3 \left( 1 - \frac{I_{hc}^*}{I_{hc}} \right) \left( \delta_2 I_{hs} - \delta_2 I_{hs}^* \frac{I_{hs}}{I_{hs}^*} \right) 
\]
\[
= c_3 \delta_2 I_{hs} \left( 1 - \frac{I_{hs}^*}{I_{hs}} \right) \left( \frac{I_{hs}}{I_{hs}^*} - \frac{I_{hc}}{I_{hc}^*} \right) 
\]

In similar approach to the equations (18-21) above we get
\[
c_4 \left( 1 - \frac{S_{f}^*}{S_{f}} \right) \dot{S}_{f} = c_4 \left( 1 - \frac{S_{f}^*}{S_{f}} \right) \left( \lambda_f^* S_{f} - \lambda_f S_f - \mu_f S_f \right) 
\]
\[
= c_4 \left( 1 - \frac{S_{f}^*}{S_{f}} \right) \left( \lambda_f^* S_{f} - \lambda_f S_f \right) - \left( \mu_f S_f - \mu_f S_f^* \right) 
\]
\[
= c_4 \left( 1 - \frac{S_{f}^*}{S_{f}} \right) \left( 1 - \frac{\lambda_f S_f}{\lambda_f S_f^*} \right) - \mu_f S_f \left( S_f - S_f^* \right)^2 
\]
\[
\leq c_4 \lambda_f^* S_f \left( 1 - \frac{S_{f}^*}{S_{f}} \right) \left( 1 - \frac{\lambda_f S_f}{\lambda_f S_f^*} \right) 
\]
and
\[
c_4 \left( 1 - \frac{E_{f}^*}{E_{f}} \right) \dot{E}_{f} = c_4 \left( 1 - \frac{E_{f}^*}{E_{f}} \right) \left( \lambda_f S_f - \lambda_f^* S_f E_{f} \frac{E_{f}}{E_{f}^*} \right) 
\]
\[
= c_4 \lambda_f^* S_f \left( 1 - \frac{E_{f}^*}{E_{f}} \right) \left( \frac{\lambda_f S_f}{\lambda_f S_f^*} - \frac{E_{f}}{E_{f}^*} \right) 
\]
also
\[
c_5 \left( 1 - \frac{I_{f}^*}{I_{f}} \right) \dot{I}_{f} = c_5 \left( 1 - \frac{I_{f}^*}{I_{f}} \right) \left( \tau E_f - \tau E_{f}^* \frac{I_{f}}{I_{f}^*} \right) 
\]
\[
= c_5 \tau E_{f}^* \left( 1 - \frac{I_{f}^*}{I_{f}} \right) \left( \frac{E_{f}}{E_{f}^*} - \frac{I_{f}}{I_{f}^*} \right) 
\]
From equations (18-24) we obtain

\[
\dot{U} \leq c_1 \lambda_h S_h^* \left(1 - \frac{S_h^*}{S_h} \right) \left(1 - \frac{\lambda_h S_h}{\lambda_h S_h^*} \right) + c_1 \lambda_h S_h^* \left(1 - \frac{E_h^*}{E_h} \right) \left(\frac{\lambda_h S_h}{\lambda_h S_h^*} - \frac{E_h}{E_h^*} \right) + c_2 \delta_1 E_h^* \left(1 - \frac{I_h^*}{I_h} \right) \left(\frac{E_h}{E_h^*} - \frac{I_h^*}{I_h} \right) + c_3 \delta_2 I_h^* \left(1 - \frac{I_h^*}{I_h} \right) \left(\frac{E_h}{E_h^*} - \frac{I_h^*}{I_h} \right) + c_4 \lambda_f S_f^* \left[\left(1 - \frac{S_f^*}{S_f} \right) \left(1 - \frac{\lambda_f S_f}{\lambda_f S_f^*} \right) + c_4 \lambda_f S_f^* \left(1 - \frac{E_f^*}{E_f} \right) \left(\frac{\lambda_f S_f}{\lambda_f S_f^*} - \frac{E_f}{E_f^*} \right) + c_5 \tau E_f^* \left(1 - \frac{I_f^*}{I_f} \right) \left(\frac{E_f}{E_f^*} - \frac{I_f^*}{I_f} \right) \right] 
\]

(25)

Considering the fact that \(x - 1 > ln(x)\) whenever \(x > 0\) and \(x - 1 = ln(x)\) for \(x = 1\). from (25) we obtained

\[
\frac{2 - S_h^*}{S_h} - \frac{E_h}{E_h^*} - \frac{E_h^* \lambda_h S_h}{E_h \lambda_h S_h^*} + \frac{S_h^* \lambda_h S_h}{S_h \lambda_h S_h^*} \geq -\left(1 - \frac{\lambda_h S_h}{\lambda_h S_h^*} \right) \left(1 - \frac{I_h^*}{I_h} \right) \left(\frac{E_h}{E_h^*} - \frac{I_h^*}{I_h} \right) + 3 \left(\frac{S_h^*}{S_h} - 1 \right) - \left(\frac{I_h^*}{I_h} \lambda_h S_h \right) - \left(\frac{E_h^* \lambda_h S_h}{E_h \lambda_h S_h^*} \right) - \left(\frac{S_h^* \lambda_h S_h}{S_h \lambda_h S_h^*} \right) \leq \frac{I_h}{I_h^*} - ln\left(\frac{I_h^*}{I_h} \right) - \frac{E_h}{E_h^*} + ln\left(\frac{E_h}{E_h^*} \right)
\]

(26)

and

\[
\frac{E_h - I_h^* E_h}{E_h^* - I_h^* E_h} - \frac{I_h}{I_h^*} + 1 \leq \left(\frac{I_h^* E_h}{E_h^* - I_h^* E_h} - 1 \right) + \frac{E_h}{E_h^*} - \frac{I_h}{I_h^*} - \frac{E_h}{E_h^*} + \frac{I_h}{I_h^*} + ln\left(\frac{I_h}{I_h^*} \right)
\]

(27)
similarly

\[
\begin{bmatrix}
I_h & I_h^* - I_h c \frac{I_h^* I_h}{I_h c I_h} + 1
\end{bmatrix} = - \left( I_h \frac{I_h I_h^*}{I_h^*} - 1 \right) + I_h \frac{I_h}{I_h} - I_h \frac{I_h}{I_h^*} \leq -\ln \left( I_h \frac{I_h}{I_h} + I_h \frac{I_h}{I_h} \right) = \left( I_h - \ln \left( I_h \frac{I_h}{I_h} + I_h \frac{I_h}{I_h} \right) \right)
\]

we also compute

\[
\left[ 2 - \frac{S_f^*}{S_f} - \frac{E_f I_f}{E_f^*} - \frac{I_f^* E_f}{I_f^* E_f^*} - \frac{I_f}{I_f^*} + 1 \right] = - \left( \frac{I_f^* E_f}{I_f^* E_f^*} - 1 \right) + \frac{E_f^*}{E_f^*} - \frac{I_f}{I_f^*} \leq -\ln \left( \frac{I_f^* E_f}{I_f^* E_f^*} + \frac{E_f^*}{E_f^*} - \frac{I_f}{I_f^*} \right) = \left( \frac{E_f^*}{E_f^*} - \ln \left( \frac{E_f^*}{E_f^*} + \frac{I_f}{I_f^*} \right) \right)
\]

and

\[
\left[ \frac{E_f - I_f^* E_f}{I_f^* E_f^*} - \frac{I_f}{I_f^*} + 1 \right] = - \left( \frac{I_f^* E_f}{I_f^* E_f^*} - 1 \right) + \frac{E_f^*}{E_f^*} - \frac{I_f}{I_f^*} \leq -\ln \left( \frac{I_f^* E_f}{I_f^* E_f^*} + \frac{E_f^*}{E_f^*} - \frac{I_f}{I_f^*} \right) = \left( \frac{E_f^*}{E_f^*} - \ln \left( \frac{E_f^*}{E_f^*} + \frac{I_f}{I_f^*} \right) \right)
\]

Putting equations (26-30) into (25) we’ve

\[
\dot{U} \leq c_1 \lambda_h S_h \left[ I_h \frac{I_h}{I_h} - \ln \left( I_h \frac{I_h}{I_h} \right) - E_h \frac{E_h}{E_h} + \ln \left( E_h \frac{E_h}{E_h} \right) + c_2 \delta_1 I_h^* \left[ E_h \frac{E_h}{E_h} - \ln \left( E_h \frac{E_h}{E_h} \right) - I_h \frac{I_h}{I_h} + \ln \left( I_h \frac{I_h}{I_h} \right) \right] \right]
\]

By taking the constants \( C_1 = \delta_2 I_h^* \), \( C_2 = \frac{\delta_2 I_h^* S_h}{\delta_1 E_h} \), \( C_3 = \lambda_h S_h \), \( C_4 = \tau E_f^* \), and \( C_5 = \lambda_f S_f^* \) and further simplifying (31) we can simply obtain \( \dot{U} \leq 0 \).while strictly \( \dot{U} = 0 \) is true only when \( S_h = S_h^*, E_h = E_h^*, I_h = I_h^*, I_h = I_h^* \) and \( S_f = S_f^*, E_f = E_f^*, I_f = I_f^* \). then the only invariant set of the model (1) is the endemic equilibrium point \( \varepsilon^* \). Hence Applying Lasalle invarinace principle [32], the endemic equilibrium \( \varepsilon^* \) of the trachoma model is globally asymptotically stable "GAS".This completes the proof.
5 Parameter Estimation

This section discusses how to fit the parameters in the proposed Trachoma model using real-life trachoma cases from Northern Nigeria. The epidemic cases were reported between May and July of 2013, [25] at the time when this research paper was prepared. The population of Northern Nigeria is estimated to be \( N_h(0) = 90.3\)M for initial conditions, with an initial exposed population of \( E_h(0) = 300000 \) and Using the relation \( N_h(0) = S_h(0) + E_h(0) + I_{ha}(0) + I_{hc}(0) + R_h \), we can estimate the rest of the initial values for the state variables \( N_f(0) = S_f(0) + E_f(0) + I_f(0) \). In this case, \( S_h(0) = 6140400, I_{ha}(0) = 410, I_{hc}(0) = 270, \) and \( R_h(0) = 150 \) were obtained, with \( S_f(0) = 341000 \) and \( I_f(0) = 3000 \). As shown in Figure 1, there are 14 biological parameters that have been predicted using the least-square fitting method, coming up with the best fit of the Trachoma model’s solution to actual epidemic cases. The best values of the biological parameters are obtained by reducing the average absolute relative error between the actual Trachoma cases and the model solution. With a value of \( 9.8748e-02 \), the objective function produces a relatively small error. Figure 1 depicts actual Trachoma cases as solid circles, while the best-fitting curve of the model is depicted as a solid line. Table 2 lists the biological parameters used in the model, along with their best approximate values obtained using the least-squares method. For the real Trachoma cases in Northern Nigeria from May to July 2013, these parameters finally provided the value of the basic reproduction number equal to \( R_0 = 1.66 \).

Table 2: Baseline values of the parameters used in the trachoma model (1)

| Parameter | Value       | Units/Remarks | Sources  |
|-----------|-------------|---------------|----------|
| \( N_h(0) \) | 90.3\text{million} | Constant       | [39]     |
| \( N_f(0) \) | 11\text{million} | Constant       | [40]     |
| \( S_h(0) \) | 0.68 \times N(0) | Constant       | Assumed  |
| \( S_f(0) \) | 0.31 \times N(0) | Constant       | Assumed  |
| \( \beta_f \) | 0.07258 \text{day}^{-1} | Assumed       |          |
| \( \tau \) | 2.213 \text{day}^{-1} | Estimated by [41] |          |
| \( \beta_h \) | 0.08353 \text{day}^{-1} | Estimated by [30] |          |
| \( \varphi \) | 0.501 \text{day}^{-1} | Estimated by [37] |          |
| \( \Pi_h \) | 24.9991 \text{day}^{-1} | Fitted       |          |
| \( \Pi_f \) | 1.5 \times 10^6 \text{day}^{-1} | Fitted       |          |
| \( \mu_h \) | 0.0014 \text{day}^{-1} | Fitted       |          |
| \( \mu_f \) | 1.354 \text{day}^{-1} | Fitted       | [40]     |
| \( \delta_1 \) | 0.01212 \text{day}^{-1} | Fitted       |          |
| \( \delta_2 \) | 0.012003 \text{day}^{-1} | Fitted       |          |
| \( \sigma_f \) | 0.2903 \text{day}^{-1} | Fitted       |          |
| \( \psi_1 \) | 0.3010 \text{day}^{-1} | Fitted       |          |
| \( \psi_2 \) | 0.1121 \text{day}^{-1} | Fitted       |          |
| \( \psi_3 \) | 0.1428 \text{day}^{-1} | Fitted       |          |
Figure 1: Data fitting for the real Trachoma cases in Northern Nigeria.
6 The Normalized Sensitivity Analysis

In this section we employed the method of local sensitivity analysis to highlight the sensitivity of the basic reproduction number $R_0$ to some key associated parameters for a trachoma model earlier developed and rigorously analysed in sections (3-4). The basic reproduction number was obtained and described as a parameter dependent output of the model and the severity indicator of the (chlamydia trachomatis/ocular chlamydia) of which lowering the number below a critical figure (i.e. less than unity) is considered as the Major way of curtailing and aborting the spread of the infectious trachoma in the population, and saving individuals from corneal opacifications or even perpetual and irreversible blindness. In this direction, investigating the monotonicity between the model parameters and the basic reproduction number became crucially interested and motivated. The basic properties of the model have been demonstrated earlier in section (3), detailing the boundedness, positivity and well-posedness of the solutions. Our main interest here is just to understand the sensitivity and impressionability of the basic reproduction number in relation to the crucial parameters used throughout the model. The computation of the expression of basic reproduction number was carried out using the technique of next generation matrix, see (4) above, and its denoted by

$$R_0^T = \sqrt{\Pi_h (\mu_h + \psi_3) (\delta_2 + \mu_h + \psi_2) (\delta_1 + \mu_h + \psi_1) (\mu_h + \tau) \tau \Pi_f \beta_f \beta_h \delta_1 \mu_h (\mu_h + \psi_3 + \delta_2) \sigma_f \Pi_f (\mu_h + \psi_3) (\delta_2 + \mu_h + \psi_2) (\delta_1 + \mu_h + \psi_1) (\mu_h + \tau) \mu_f}$$

We will now proceed to computation of the normalized local sensitivity indices of $R_0^T$ proportional the parameters in connection the trachoma model (1). Here the series of input parameters relative to $R_0^T$ is

$$\rho = \{\Pi_h, \mu_h, \psi_3, \delta_2, \psi_2, \delta_1, \psi_1, \tau, \Pi_f \beta_f, \beta_h, \sigma_f, \mu_f\}$$

We then Scrutinize the significance on the normalized forward sensitivity or (Elasticity index) of $R_0^T$ due to parameter discrepancy. Usually if a system is of various parameters, changes in parameters may not equally change the results due to variation in the sensitivity of the parameters, some have high sensitivity while others are slightly sensitive and some are neutrally sensitive (having zero relative sensitivity). The optimization of the output is achieved by determining the sensitivity status of each parameter[42, 44]. We denote by $\Gamma_R^T$ the normalized local sensitivity index of the output $R_0^T$ with respect to a parameter ($\omega$), where $\omega \in \rho$, and is defined as

$$\Gamma_R^T = \frac{\partial \ln(R_0^T)}{\partial \ln(\omega)}$$
[43, 45, 46, 47]. Using the above definition we compute the following indices for the output $R_0^T$ with respect to every parameter presented in (33).

$$\Gamma_{\sigma_f}^{R_T} = \frac{\sigma_f}{R_0^T} \times \frac{\partial R_0^T}{\partial \sigma_f} = 1$$

$$\Gamma_{\beta_h}^{R_T} = \frac{\beta_h}{R_0^T} \times \frac{\partial R_0^T}{\partial \beta_h} = \frac{1}{2}$$

$$\Gamma_{\beta_f}^{R_T} = \frac{\beta_f}{R_0^T} \times \frac{\partial R_0^T}{\partial \beta_f} = \frac{1}{2}$$

$$\Gamma_{\Pi_h}^{R_T} = \frac{\Pi_h}{R_0^T} \times \frac{\partial R_0^T}{\partial \Pi_h} = -\frac{1}{2}$$

$$\Gamma_{\Pi_f}^{R_T} = \frac{\Pi_f}{R_0^T} \times \frac{\partial R_0^T}{\partial \Pi_f} = \frac{1}{2}$$

$$\Gamma_{\tau}^{R_T} = \frac{\tau}{R_0^T} \times \frac{\partial R_0^T}{\partial \tau} = \frac{1}{2} \frac{\mu_h}{\mu_h + \tau}$$

$$\Gamma_{\delta_1}^{R_T} = \frac{\delta_1}{R_0^T} \times \frac{\partial R_0^T}{\partial \delta_1} = \frac{1}{2} \frac{\mu_h + \psi_1}{\delta_1 + \mu_h + \psi_1}$$

$$\Gamma_{\delta_2}^{R_T} = \frac{\delta_2}{R_0^T} \times \frac{\partial R_0^T}{\partial \delta_2} = \frac{1}{2} \frac{(\psi_2 - \psi_3)\delta_2}{(\delta_2 + \mu_h + \psi_2)(\mu_h + \psi_3 + \delta_2 + \psi_2)}$$

$$\Gamma_{\psi_1}^{R_T} = \frac{\psi_1}{R_0^T} \times \frac{\partial R_0^T}{\partial \psi_1} = -\frac{1}{2} \frac{\mu_h + \psi_1}{\psi_1}$$

$$\Gamma_{\psi_2}^{R_T} = \frac{\psi_2}{R_0^T} \times \frac{\partial R_0^T}{\partial \psi_2} = -\frac{1}{2} \frac{\mu_h + \psi_2}{(\delta_2 + \mu_h + \psi_2)}$$

$$\Gamma_{\psi_3}^{R_T} = \frac{\psi_3}{R_0^T} \times \frac{\partial R_0^T}{\partial \psi_3} = -\frac{1}{2} \frac{\mu_h + \psi_3}{(\delta_2 \psi_3)}$$

$$\Gamma_{\mu_f}^{R_T} = \frac{\mu_f}{R_0^T} \times \frac{\partial R_0^T}{\partial \mu_f} = -1$$

$$\Gamma_{\sigma_f}^{R_T} = 1, \quad \Gamma_{\beta_h}^{R_T} = \frac{1}{2}, \quad \Gamma_{\beta_f}^{R_T} = \frac{1}{2}, \quad \Gamma_{\Pi_h}^{R_T} = -\frac{1}{2}, \quad \Gamma_{\Pi_f}^{R_T} = \frac{1}{2}, \quad \Gamma_{\tau}^{R_T} = \frac{1}{2} \frac{\mu_h}{\mu_h + \tau}, \quad \Gamma_{\delta_1}^{R_T} = \frac{1}{2} \frac{\mu_h + \psi_1}{\delta_1 + \mu_h + \psi_1},$$

$$\Gamma_{\delta_2}^{R_T} = \frac{1}{2} \frac{(\psi_2 - \psi_3)\delta_2}{(\delta_2 + \mu_h + \psi_2)(\mu_h + \psi_3 + \delta_2 + \psi_2)}, \quad \Gamma_{\psi_1}^{R_T} = -\frac{1}{2} \frac{\psi_1}{(\delta_1 + \mu_h + \psi_1)}, \quad \Gamma_{\psi_2}^{R_T} = -\frac{1}{2} \frac{\psi_2}{(\delta_2 + \mu_h + \psi_2)},$$

$$\Gamma_{\psi_3}^{R_T} = -\frac{1}{2} \frac{(\delta_2 \psi_3)}{(\mu_h + \psi_3)(\mu_h + \psi_3 + \delta_2)}, \quad \Gamma_{\mu_f}^{R_T} = -1$$

$$\Gamma_{\mu_h}^{R_T} = \frac{1}{2} \frac{(A\psi_3 - B\mu_h^2)\delta_2^2 + 2(A\psi_3 - B\mu_h^2)(D)\delta_2 + (\mu_h + \psi_3)^2(A\psi_2 - B\mu_h^2)}{(\mu_h + \psi_3)(\delta_2 + \mu_h + \psi_2)(\delta_1 + \mu_h + \psi_1)(\mu_h + \tau)(\mu_h + \psi_3 + \delta_2)}$$

where; $A = \tau \delta_1 + \tau \psi_1 - \mu_h^2$, $B = \tau + \delta_1 + 2\mu_h + \psi_1$, $D = \frac{1}{2} \psi_3 + \mu_h + \frac{1}{2} \psi_2$
Remark 6.1. We can see from the above expressions, the local sensitivity indices reveal the following facts:

(i) \( \Gamma_{\sigma_f}^{R_0^T} = 1 \)

(ii) \( 0 < \Gamma_{\beta_h}^{R_0^T}, \Gamma_{\beta_f}^{R_0^T}, \Gamma_{\pi_f}^{R_0^T}, \Gamma_{\delta_1}^{R_0^T}, \Gamma_{\tau}^{R_0^T} < 1 \)

(iii) \( \Gamma_{\beta_h}^{R_0^T} = \Gamma_{\beta_f}^{R_0^T} = \Gamma_{\pi_f}^{R_0^T} = \frac{1}{2} \)

(iv) \( -1 < \Gamma_{\pi_h}^{R_0^T} < \Gamma_{\mu_h}^{R_0^T} < \Gamma_{\psi_1}^{R_0^T} < \Gamma_{\psi_2}^{R_0^T} < \Gamma_{\psi_3}^{R_0^T} < \Gamma_{\delta_2}^{R_0^T} < 0 \)

(v) \( |\Gamma_{\sigma_f}^{R_0^T}| = |\Gamma_{\mu_f}^{R_0^T}|, |\Gamma_{\beta_h}^{R_0^T}| = |\Gamma_{\beta_f}^{R_0^T}| = |\Gamma_{\pi_f}^{R_0^T}| = |\Gamma_{\pi_h}^{R_0^T}| \)

(vi) \( |\Gamma_{\tau}^{R_0^T}| < |\Gamma_{\psi_1}^{R_0^T}|. \)

On the basis of the elasticity analysis above, we can easily see that whenever we increase \( \sigma_f \) by 5% while fixing the remaining parameters, this will consequently attract a 5% increase in \( R_0^T \), which apparently shows a strong connection between the basic reproduction number \( R_0^T \) and the individual parameters. Similarly, it reveals that, increasing the transmission rates, \( \beta_f, \beta_h \), and flies recruitment rate \( \Pi_f \) each by 10% and keeping the remaining parameters unchanged will equally produce a 5% increment in the basic reproduction number and this will boost the trachoma prevalence in the population. Likewise, raising the values of the parameters \( \tau \) and \( \delta_1 \) by 1.0% (one percent) will correspondingly gain less than 1% increment in the basic reproduction number \( R_0^T \). Meanwhile, raising the values of the parameters \( \mu_f \) and \( \Pi_h \) by 10% each will consequently attract a corresponding 10% and 5% decrements in \( R_0^T \) respectively. In a similar manner, whenever we introduce an increments in each of \( \delta_1, \psi_1, \psi_2, \psi_3 \) and \( \mu_h \) by a 1.0%, it will provide us a consequential decrease in the basic reproduction number with less than 1.0%. Therefore, later are the most sensitive parameters that should be targeted to attain the goal of Global Trachoma Elimination as a Public Health Problem, as targeted by the World Health Organization (WHO).

It is now quite enough to position our selves to use the generated coefficients of the normalized local sensitivity indices to highlight the relative influences of each of the parameters on \( R_0^T \).
From figure 2, we can interpret the elasticity indices recorded in the chart based on the values presented earlier in table 2. It reveals that the most sensitive and influential parameter in changing the size of the basic reproduction number $R_0^T$ is $\sigma_f$ (the contact rate of the eye-seeking flies or the musca sovens) whose index value is +1.0000 as earlier explained in the computations of the elasticity expressions. Thus the discrepancies with this parameter will give much more variation in the basic reproduction number $R_0^T$ and consequently, the general model output. Particularly, whenever $\sigma_f$ is variably increase(respectively decrease) by 20%, then the basic reproduction number $R_0^T$ will equally increase(respectively decrease) by 20%. Followed by the parameters ($\beta_h$, $\beta_f$, and $\Pi_f$), the rate of transmission of the chlamydia trachomatis from vector to host, the rate of transmission of chlamydia trachomatis from the eye-seeking flies to human and the rate at which flies are recruited into the vector population respectively. The approximate elasticity sensitivity index is +0.5000, therefore, changing any of them by 20% will correspondingly attract a 10% change in $R_0^T$. Another set of influential parameters that shows more sensitivity to $R_0^T$ are $\mu_f$, $\delta_1$, and $\tau$, the natural death rate of flies that facilitate the spread of Trachoma with elasticity index (−1.0000), the progression rate at which the exposed individuals proceed to show the clinical symptoms of active trachoma(Trachomatous follicular/Intense TF/TI or even begin to develop scars on the tarsal conjunctiva (TS)) with the index of about (0.4641), and $\tau$ the corresponding progression rate at which the exposed eye-seeking flies proceed to infective stage with elasticity index (0.1109) respectively. Among these three, $\mu_f$ is the appropriate parameter to concentrate on, so as to achieve (Global Elimination of Blinding Trachoma as a public Health Problem) possibly by the year 2030. This is because reducing the population of flies that transmit the disease through proper environmental sanitation and or insecticide spraying by 20% will certainly bring about a 20% decrease in the size of basic reproduction number ($R_0^T$) and this will consequently results in curtailing the prevalence of the trachoma in the targeted population by a significant percentage.

The remaining parameters together with their respective indices in a hierarchical curtailing significance include; $\delta_2$, the progression rate of an infected individual from active trachoma stage (i.e Trachomatous Folicular/Intense TF/TI) to the severe stage (i.e Trachomatous Trachiasis or Corneal opacity), $\psi_3$, the recovery rate from severe stage of trachoma(TT) through surgery (the S-component of SAFE strategy), $\psi_2$, the recovery rate of individual from early stages of trachoma(TF/TI or TS) which is achieved by Antibiotic Administration(Azithromycin or Tropical tetracyclin) the A-component of SAFE strategy, $\psi_1$, the recovery rate of exposed individuals who are yet to show any symptom of trachoma(F-component of SAFE strategy), $\mu_h$, the death rate of human population, and finally, $\Pi_h$, the rate at which individuals are recruited into the human population.

Clearly, if a proper concentration is made on the most sensitive parameters for possible adjustment, it will cut down the size of the basic reproduction number ($R_0^T$), and hence the Global Elimination of Blinding Trachoma as a Public Health Problem will be made achievable as targeted by WHO by the year 2030. Precisely, there should be a means of minimizing the rate of contact between the infected flies and susceptible human($\sigma_f$) as low as possible, the transmission of trachoma from flies to human and vice-vasa should also be terminated. Also the means by which the flies are being recruited should be blocked through proper personal and environmental hygiene; and amplifying the death rate of the eye-seeking flies(the musca-sovens) to curtail or truncate the spread of trachoma in a susceptible population requires a consideration.
Figure 2: The local elasticity indices of $R_o$ with respect to parameters of the model (1) as presented in table 2.
Figure 3: The sensitivity of individual parameters in relation to $R_T^0$. (3a.) a plots for $R_T^0$ against parameter $\psi_1$, with $\psi_1$ from 0.01 – 2.00 and other parameters remained unchanged. (3b.) a plots for $R_T^0$ against parameter $\psi_2$, with $\psi_1$ from 0.100 – 0.700 and other parameters remained unchanged.
Figure 4: The sensitivity of individual parameters in relation to $R_T^0$. (4a.) a plots for $R_T^0$ against parameter $\tau$, with $\tau$ from $0.1 - 0.70$ and other parameters remained unchanged. (4b.) a plots for $R_T^0$ against parameter $\sigma_f$, with $\sigma_f$ from $0.100 - 0.700$ and other parameters remained unchanged
Figure 5: The sensitivity of individual parameters in relation to $R^T_0$. (5a.) a plots for $R^T_0$ against parameter $\beta_h$, with $\beta_h$ from 0.01 – 0.10 and other parameters remained unchanged. (5b.) a plots for $R^T_0$ against parameter $\beta_f$, with $\beta_f$ from 0.01 – 0.10 and other parameters remained unchanged.
7 Numerical simulations

Since the basic reproductive number $R_0$ is the most important quantity to comprehend the extent for the spread of an epidemic. It can be seen in figure (6 and 7) that displayed the behaviour of the trachoma model when $R_0 > 1$, and the behaviour of the trachoma model when $R_0 < 1$. It shows that the effective use of WHO adopted control scheme (SAFE) plays a significant role toward achieving the WHO targeted Trachoma eradication as challenge in the public health. $R_0$ has also been investigated by varying different kinds of biological parameters of the proposed Trachoma model. Using mesh plot and the parameter values in Table 2, we obtained some numerical results. The result as depicted in Figure 8, shows a significant increase with the variation in the transmission rates ($\beta_h, \beta_f$) and that of vector contact rate $\sigma_f$ while in Figure 9, $R_0$ decreases/increases with the decreasing/increasing value of progression rate from exposed flies to infected flies $\tau$, progression rates from class of exposed individuals to active trachoma class ($\delta_1$), from active trachoma to severe infection class ($\delta_2$) and that of vector contact rate $\sigma_f$, and Figure 10, shows $R_0$ increases with decrease of natural death rates of both human and flies populations ($\mu_h, \mu_f$), while in figure 11. $R_0$ decreases/increases with the decreasing/increasing value progression rates from class of exposed individuals to active trachoma class ($\delta_1$), from active trachoma to severe infection class ($\delta_2$) and that of vector contact rate $\sigma_f$.

![Figure 6](image.png)

(a)

(b)

**Figure 6:** Effect of SAFE control strategy on human population. (1a.) displays the behaviour of the trachoma model without control (i.e when $R_0 > 1$), (1b.) displays the behaviour of the trachoma model with control (i.e when $R_0 < 1$)
Figure 7: Effect of SAFE control strategy on flies population. (2a.) displays the behaviour of the trachoma model without control (i.e when $R_0 > 1$), (2b.) displays the behaviour of the trachoma model with control (i.e when $R_0 < 1$).

Figure 8: Profile of reproductive number with variation in the contact rate of the disease vector $\sigma_f$, and that of trachoma transmission rates ($\beta_h, \beta_f$).

Figure 9: Profile of reproductive number with variation in (9a.) the contact rate of the disease vector $\sigma_f$, and that of vector progression rate from exposed to infectious flies $\tau$, (9b.) the human transmission rates from exposed individual class to early stage of trachoma ($\delta_1$) to severe stage ($\delta_2$).
Figure 10: Profile of reproductive number with variation in (10a.) the contact rate of the disease vector $\sigma_f$, and that of the natural death rate of human $\mu_h$, (10b.) the contact rate of the disease vector $\sigma_f$, and the natural death rate of the eye-seeking fly $\mu_f$.

Figure 11: Profile of reproductive number with variation in the contact rate of the disease vector $\sigma_f$, and the human transmission rates from exposed individual class to early stage of trachoma ($\delta_1$) to severe stage ($\delta_2$)
8 Discussions/Conclusions

we have developed and rigorously analyzed a compartmental model of (Kermack-Mckenderick) type to monitor the transmission dynamics of chlamydia trachomatis in a multi-stains structured format. It is confirmed that the local asymptotic stability of the disease-free equilibrium and the global asymptotic stability of the unique endemic equilibrium is guaranteed whenever the computed basic reproduction number $R_0$ is below unity and $R_0$ greater than one respectively. We have also estimated the model parameters and fit the model with the use of field data cases from Northern Nigeria using least-square fitting method.

Moreover, we displayed the significance of the model parameters in changing the size of the basic reproduction number $R_0^T$ which can be seen in figure 2. it is confirmed from computed elasticity indices that the most sensitive parameter to basic reproduction number is $\sigma_f$ (vector contact rate), followed by rates of transmission ($\beta_h$ and $\beta_f$) then the rest follows. The elasticity expressions of the model parameters and their respective indices shown in section 6. reveal that those parameters whose indices are other than 1 change with the change in some of the parameters associated with their computed elasticity expression. However, investigating how manifested is the variation when a particular parameter is adjusted either (increase or decreased) is becoming of interest, starting with $\psi_1$ and leaving other parameters unchanged, a plot against $\psi_1$ of $R_0^T$ is shown in figure 3a., similar figure (3b) shows the relationship between the basic reproduction number $R_0^T$ against the parameter $\psi_2$, and figure 4. displayed the sensitivity of the trachoma transmission rates $\beta_h$ and $\beta_f$ to $R_0^T$. From our graphs in figure 3-5. we can see that while keeping the parameters unchanged if not for $\mu_h$ which is adjusted from 0.0014 - 0.03, whenever the non clinical control $\psi_1$ (i.e the E and F components of SAFE strategy) is adjusted from its normal 0.10 to 2.00 the model associated basic reproduction number $R_0^T$ decreases from its endemic value of 1.6 to a disease-free state 0.78. This is true because, improving environmental sanitation and adequate supply of pure water give rise to regular facial cleanliness which plays a significant role in reducing the level exposures to trachoma in the society. In similar way the graphs show the remaining parameters ($\psi_2, \tau, \sigma_f, \beta_h$ and $\beta_f$) displaying their respective influence in changing the basic reproduction number $R_0^T$, see figure (4-5). Furthermore, figure (6 and 7) displaying the behaviour of the trachoma model when $R_0 > 1$, and when $R_0 < 1$. It is revealed that the effective use of WHO adopted control scheme (SAFE) plays a significant role toward achieving the WHO targeted Trachoma eradication as challenge in the public health in the next 10years. We have also obtained some simulation results with the aid of mesh plots for the reproductive number $R_0$ as a function of two different biological parameters in figure (8-11).

Furthermore, it should also be emphasized that the present study will be strengthened in future research work by analyzing and investigating the modern fractional operators and optimal control system. The control strategies are indeed a significant step in drastically curtailing the unknown characteristics and other features of this epidemic.

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