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Mathematical Modeling and Sensitivity Analysis of Trachoma Transmission Dynamics using a Six-Compartment Model

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ABSTRACT

Trachoma, caused by Chlamydia trachomatis, remains a leading infectious cause of blindness in many developing regions. Mathematical modeling provides a framework to quantify transmission dynamics and evaluate control strategies. In this study, we formulated a six-compartment deterministic model comprising of susceptible (S), exposed (E), infectious (I), recovered (R), trichiasis (T), and healed (H) compartments to investigate the epidemiology of trachoma. The model incorporates frequency-dependent transmission, treatment, recovery, and natural death. Analytical results include derivation of the basic reproduction number (R_0) using the Next Generation Matrix approach, determination of disease-free and endemic equilibria, and assessment of local stability using the Routh–Hurwitz criterion. Global stability of the disease-free equilibrium was established via the Castro–Chavez method. Sensitivity analysis identified the transmission rate (β) as the most influential parameter driving R_0, whereas the recovery rate (γ) exerts the strongest negative effect. Numerical simulations using the fourth-order Runge–Kutta method demonstrate the temporal dynamics of the infectious population, confirming the analytical results. Graphical analyses, including contour and 3D surface plots of $R_0(\beta, \gamma)$, illustrate the threshold behavior, highlighting the critical interplay between transmission and recovery. These findings emphasize that integrated interventions combining transmission reduction (facial cleanliness and environmental sanitation) and effective treatment are required to reduce R_0 below unity, thereby achieving sustainable control and elimination of trachoma. This study provides quantitative insights to guide public health policies in trachoma-endemic regions

1. Introduction

Trachoma is a neglected tropical disease caused by the bacterium Chlamydia trachomatis and remains the leading infectious cause of blindness worldwide. The disease is transmitted through direct contact with infected ocular or nasal secretions, contaminated fomites, and by eye-seeking flies. Repeated infections result in chronic inflammation of the conjunctiva, eventually leading to scarring, trichiasis, and irreversible blindness if left untreated (Burton & Mabey, 2009; Mariotti, Pascolini, & Rose-Nussbaumer, 2009). Trachoma continues to be endemic in many low-income regions, particularly in parts of sub-Saharan Africa where poor sanitation, limited access to clean water, and overcrowding facilitate disease transmission (Emerson et al., 2000; Solomon et al., 2015).

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The global effort to eliminate trachoma as a public health problem is coordinated by the World Health Organization through the implementation of the SAFE strategy—Surgery for trichiasis, Antibiotics to treat infection, Facial cleanliness, and Environmental improvement (WHO, 2022). Although significant progress has been made in reducing the burden of the disease, mathematical modelling studies indicate that transmission may persist in hyper-endemic communities even under mass drug administration programs (Gambhir, Pinsent, & Grassly, 2015; Lietman et al., 2018). Consequently, there is a need for rigorous analytical and computational models that can provide deeper insights into disease dynamics and guide effective intervention strategies.

Mathematical modelling has become an indispensable tool in epidemiology for understanding the transmission dynamics of infectious diseases and evaluating control policies (Anderson & May, 1991; Brauer, Castillo-Chavez, & Feng, 2019). Compartmental models divide the population into epidemiological classes and describe the movement of individuals between these classes using systems of nonlinear differential equations (Hethcote, 2000). Such models have been successfully applied to many infectious diseases including tuberculosis, HIV/AIDS, malaria, and trachoma (Diekmann, Heesterbeek, & Roberts, 2010; Keeling & Rohani, 2008). Through these frameworks, key epidemiological quantities such as the basic reproduction number can be derived, allowing researchers to determine whether a disease will die out or persist in a population.

In recent years, several modelling studies have examined the transmission dynamics of trachoma and assessed the impact of interventions such as mass antibiotic treatment and improved hygiene practices (Lietman et al., 2014; Pinsent et al., 2016). These studies demonstrate that the interplay between infection, environmental contamination, and reinfection cycles plays a critical role in sustaining transmission. Mathematical models therefore provide a valuable approach for exploring these interactions and identifying the conditions under which elimination is achievable (Grassly et al., 2008; Gambhir et al., 2015).

The development and analysis of epidemiological models typically involve stability analysis of equilibrium points, computation of the basic reproduction number, and numerical simulations to illustrate the long-term behaviour of the system (Van den Driessche & Watmough, 2002; Castillo-Chavez & Song, 2004). Stability of the disease-free equilibrium is often investigated using criteria such as the Routh–Hurwitz conditions, which determine whether perturbations around equilibrium decay over time (Perko, 2001). In addition, global stability analysis using Lyapunov functions or the approaches proposed by Castillo-Chavez and co-authors can establish whether the disease will eventually disappear regardless of initial conditions (Castillo-Chavez, Feng, & Huang, 2002).

In this study, a compartmental model consisting of six epidemiological classes is developed to describe the transmission dynamics of trachoma. The model incorporates key disease states including susceptible (S), exposed (E), infectious (I), recovered (R), trichiasis (T), and healed (H) . Analytical techniques are applied to derive the disease-free and endemic equilibria, compute the basic reproduction number, and analyze the stability of the system. The Routh–Hurwitz criterion is used to investigate local stability, while global stability analysis is conducted using the Castillo-Chavez framework. Furthermore, numerical simulations implemented in MATLAB are used to explore the effects of parameter variations and evaluate the sensitivity of the reproduction number to key epidemiological parameters.

The results of this study contribute to the growing body of research on mathematical modelling of neglected tropical diseases and provide insights into the dynamics of trachoma transmission under different intervention scenarios. By combining analytical methods and computational simulations, the model offers a useful framework for assessing strategies aimed at achieving sustainable disease control and eventual elimination.

2. Trachoma Model Formulation

2.1 Model Description

The diagram represents the transmission dynamics and progression of trachoma using six compartments. It is a deterministic compartmental model with control interventions included. Susceptible **(S).** Individuals at risk of infection but currently healthy, recruitment (birth) is represented by Λ, and natural death by μ. Susceptible individuals become exposed at a frequency-dependent rate $\lambda = \beta \frac{I}{N}$, which depends on the proportion of infectious individuals in the population. Expose (E): Individuals infected but not yet infectious. They progress to the infectious stage at rate σ or may die naturally at rate μ. Infectious (I): Individuals with active trachoma capable of transmitting the infection. They may: Recover via antibiotic treatment at rate γ(moving to R), Progress to trachomatous trichiasis at rate δ (moving to T), Die naturally at rate μ. Recovered (R): Individuals who have recovered from infection (via natural immunity or treatment). They may lose immunity at rate ω and return to the susceptible class, or die naturally at μ. Trichiasis (T): Individuals who develop chronic eyelid scarring, a serious complication that may lead to blindness. Can receive surgery intervention at rate τ, moving to H, or die naturally at μ. Surgery / Healed (H). Individuals who have undergone surgical correction of trichiasis. They are considered functionally healed but can still die naturally at rate μ.

Assumptions of the model are.

(i) Homogeneous mixing of the population
(ii) Constant recruitment into the susceptible class
(iii) Latent period before infectiousness
(iv) Natural death occurs in all compartments
(v) Progression to chronic complication (Trichiasis)

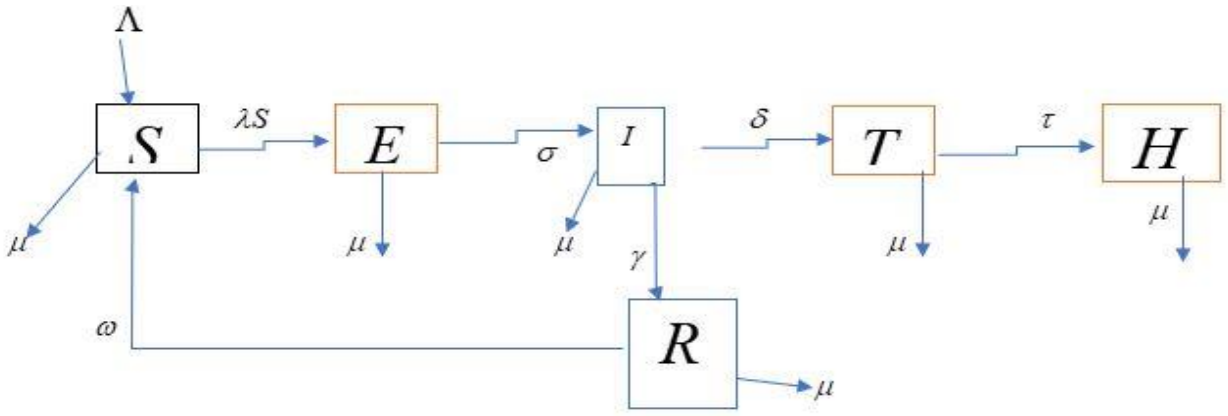

Figure 1: Schematic diagram of Trachoma

2.2 Equations of the Model

$$\left.\begin{aligned}
\frac{dS}{dt} &= \Lambda - \lambda S + \omega R - \mu S \\
\frac{dE}{dt} &= \lambda S - (\sigma + \mu) E \\
\frac{dI}{dt} &= \sigma E - (\gamma + \delta + \mu) I \\
\frac{dR}{dt} &= \gamma I - (\omega + \mu) R \\
\frac{dT}{dt} &= \delta I - (\tau + \mu) T \\
\frac{dH}{dt} &= \tau T - \mu H
\end{aligned}\right\} \qquad (1)$$

Table 1: Description of Model Variables, Parameters, Baseline Values Used in Simulation, and References

Symbol	Description	Value Used	Source
$S(0)$	Initial susceptible population	800 individuals	Anderson & May (1991); Keeling & Rohani (2008)
$E(0)$	Initial exposed population	100 individuals	Grassly et al. (2008)
$I(0)$	Initial infectious population	50 individuals	Gambhir et al. (2015)
$T(0)$	Initial Chronic eyelid scarring stage	20 individuals	Lietman et al. (2014)
$R(0)$	Initial recovered population	25 individuals	Pinsent et al. (2016)
$H(0)$	Initial Individuals who received surgery	5 units	Emerson et al. (2000)
Λ	Recruitment rate into susceptible class	50 persons/day	Anderson & May (1991)
β	Effective contact (transmission) rate	0.35 day^{-1}	Grassly et al. (2008)
σ	Progression rate from exposed to infectious	0.20 day^{-1}	Gambhir et al. (2015)
α	Treatment rate of infected individuals	0.30 day^{-1}	Lietman et al. (2014)
γ	Recovery rate after treatment	0.25 day^{-1}	Pinsent et al. (2016)
ω	Loss of immunity rate	0.05 day^{-1}	Keeling & Rohani (2008)
μ	Natural death rate	0.000039 day^{-1}	World Health Organization (2022)
δ	Disease-induced death rate	0.01 day^{-1}	Burton & Mabey (2009)

Compartment	Symbol	Description
Susceptible	S	Healthy individuals at risk
Exposed (Latent)	E	Infected but not yet infectious
Infectious	I	Active trachoma (can transmit)
Recovered	R	Treated individuals (temporary immunity)

Compartment	Symbol	Description
Trichiasis	T	Chronic eyelid scarring stage
Surgery/Treated Trichiasis	H	Individuals who received surgery

3. Analysis of the Model

3. 1: Invariant region

Let $\Delta = \{S, E, I, R, T, H\} \in \mathbb{R}_+^6$ be any solution of the system (1) with all parameters nonnegative and recruitment is finite, solutions remain biologically feasible: $N(t) \leq \frac{\Lambda}{\mu}$. Thus, the feasible region:

$\Delta = \left\{ (S, E, I, R, T, H) \in \mathbb{R}_+^6 : N \leq \Lambda / \mu \right\}$ is positively invariant.

3.2 Disease-Free Equilibrium (DFE)

Setting $\frac{dS}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = \frac{dT}{dt} = \frac{dH}{dt} = 0$ the disease-free equilibrium (DFE) and (EES) of the system (1) is denoted by: $E_0 = \left\{ \left(S^0, E^0, I^0, R^0, T^0, H^0 \right) = \left\{ \frac{\Lambda}{\mu}, 0, 0, 0, 0, 0 \right\} \right\}$ (2)

and

$$\left.\begin{aligned} S^* &= \frac{N^*}{R_0} \\ E^* &= \frac{\gamma + \delta + \mu}{\sigma} I^* \\ I^* &= \frac{N^* \left(1 - \frac{1}{R_0} \right)}{\frac{\gamma + \delta + \mu}{\sigma} + 1 + \frac{\gamma}{\omega + \mu} + \frac{\delta}{\tau + \mu} + \frac{\tau\delta}{\mu(\tau + \mu)}} I^* \\ R^* &= \frac{\gamma}{\omega + \mu} I^* \\ T^* &= \frac{\delta}{\tau + \mu} I^* \\ H^* &= \frac{\tau\delta}{\mu(\tau + \mu)} I^* \end{aligned}\right\}$$

respectively.

The above result shows that Endemic infection level increases as R_0increases, Surgery parameters τ reduce chronic burden but do not directly alter R_0. Antibiotic rate γ reduces endemic level and Reinfection ω increases endemic persistence.

3.3 Reproduction Number of Trachoma

The basic reproduction number, denoted by R_0, is defined as the average number of new trachoma infections generated by a single infectious individual introduced into a completely susceptible population during the entire infectious period. Using the Next Generation Matrix approach (Diekmann et al., 1990), the infected compartments are $E$and I then solving equation (1) we have $R_0 = \frac{\beta\sigma}{(\sigma+\mu)(\gamma+\delta+\mu)}$ (3)

Increasing antibiotic treatment (γ) reduces R_0 , increasing hygiene/environmental sanitation reduces β and Surgery (τ) reduces long-term blindness but does not directly reduce transmission.

3.4 Sensitivity Analysis

The normalized forward sensitivity index of $R_0$with respect to parameter p is

$$\Upsilon_p^{R_0} = \frac{\partial R_0}{\partial p} \times \frac{p}{R_0} \quad (4)$$

This measures the relative change in R_0caused by a relative change in parameter p.

Sensitivity with respect to transmission rate β

Substituting equation (3) into (4) we have the following

$$\Upsilon_\beta^{R_0} = \left(\frac{S}{(\mu+\alpha)(\mu+\gamma)}\right)\left(\frac{\beta}{R_0}\right) = 1$$

Interpretation: A 10% increase in transmission increases $R_0$by 10%.

Sensitivity with respect to recovery rate $\boldsymbol{\gamma}$.

$$\frac{\partial R_0}{\partial \gamma} = -\frac{\beta S}{(\mu+\alpha)(\mu+\gamma)^2} = -\frac{\gamma}{(\mu+\gamma)}$$

Interpretation: Increasing recovery reduces disease spread.

Sensitivity with respect to treatment rate $\boldsymbol{\alpha}$.

$$\frac{\partial R_0}{\partial \alpha} = -\frac{\beta S}{(\mu+\gamma)(\mu+\alpha)^2} = -\frac{\alpha}{(\mu+\alpha)}$$

Interpretation: Increasing treatment reduces R_0.

Sensitivity with respect to Natural Death rate $\boldsymbol{\mu}$.

$$\frac{\partial R_0}{\partial \mu} = -\frac{\beta S[(\mu+\gamma)+(\mu+\alpha)]}{(\mu+\alpha)^2(\mu+\gamma)^2} = -\left(\frac{\mu}{\mu+\alpha}+\frac{\mu}{\mu+\gamma}\right)$$

Table 2: sensitivity Summary table

Parameter	Sensitivity Index	Interpretation
β	$+1$	Most influential parameter
γ	Negative	Higher recovery reduces infection
α	Negative	Treatment reduces spread
μ	Negative	Natural mortality lowers transmission

3.5 Local Stability Analysis of Trachoma

The Routh–Hurwitz criterion is used to determine whether all eigenvalues of a characteristic polynomial have negative real parts, without solving them explicitly. If all eigenvalues are negative equilibrium is locally asymptotically stable but if at least one eigenvalue is positive equilibrium is unstable.

Solving equation (1) at disease free we have

$$J(E_0) = \begin{pmatrix} -\mu & 0 & -\beta & \omega & 0 & 0 \\ 0 & -(\sigma+\mu) & \beta & 0 & 0 & 0 \\ 0 & \sigma & -(\gamma+\delta+\mu) & 0 & 0 & 0 \\ 0 & 0 & \gamma & -(\omega+\mu) & 0 & 0 \\ 0 & 0 & \delta & 0 & -(\tau+\mu) & 0 \\ 0 & 0 & 0 & 0 & \tau & -\mu \end{pmatrix} \quad (5)$$

Reducing equation (5) we have

$$J = \begin{pmatrix} -(\sigma+\mu) & \beta \\ \sigma & -(\gamma+\delta+\mu) \end{pmatrix} \quad (6)$$

Where $-\mu, -(\tau+\mu), -(\omega+\mu), -\mu$

Evaluating (6) we have

$$| J - \lambda I | = \lambda^2 + a_1\lambda + a_0 = 0 \quad (7)$$

Where: $a_1 = (\sigma+\mu)+(\gamma+\delta+\mu)$, $a_0 = (\sigma+\mu)(\gamma+\delta+\mu) - \beta\sigma$

The equilibrium is locally asymptotically stable if:

$$a_1 > 0, a_0 > 0$$

Since all parameters are positive: $a_1 > 0$(always satisfied)

$a_0 > 0$: $(\sigma+\mu)(\gamma+\delta+\mu) > \beta\sigma$

$$\frac{\beta\sigma}{(\sigma+\mu)(\gamma+\delta+\mu)}<1$$

$R_0 < 1$ DFE is locally asymptotically stable

3.6 Global stability of the disease-Free equilibrium

For global stability of the DFE by Castilo-Chavez, Zhilag and Huang (2002) was employed. The model is rewritten as follows:

$$\left.\begin{aligned}\frac{dX}{dt}&=K(X,Z)\\ \frac{dZ}{dt}&=G(X,Z),G(X,0)=0\end{aligned}\right\} \quad (8)$$

where $X \in \mathbb{R}^4$ and $X=\{S,R,T,H\}$ denotes the number of uninfected compartments and $Z \in \mathbb{R}^2$ where $Z=\{E,I\}$ denotes the number of infected compartments. $E^0=\left\{\frac{\Lambda}{\mu},0,0,0,0,0\right\}$ denote the disease-free equilibrium point of the system where $x^*=\left\{\frac{\Lambda}{\mu}\right\}$ (9)

Consider equation (8) may be met to guarantee global asymptotic stability

(H_1): For $\left.\frac{dX}{dt}=K(X,0),x^*\right\}$ is globally asymptotic stable.

(H_2):For $G(X,Z)=AZ-\hat{G}(X,Z),\hat{G}(X,Z)\geq 0,\forall(X,Z)\in\Pi$ where $D^G(x^*,0)$ is an M matrix and Π is the region where the model has biological meaning.

Theorem 1. If the system (1) satisfies condition (H_1), then the fixed point $E^0=\left\{\frac{\Lambda}{\mu},0,0,0,0,0\right\}$ is a globally asymptotically stable equilibrium of the system (1) [provided that R_0 is less than unity and the conditions (H_1) and (H_2) are satisfied:

Proof: consider $K(X,0)=[\Lambda-\mu S]$ and $G(X,Z)$ and $G(X,Z)=AZ-\hat{G}(X,Z)$

$$A=\begin{pmatrix}-(\sigma+\mu) & \frac{\beta S_0}{N}\\ \sigma & -(\gamma+\delta+\mu)\end{pmatrix} \quad (10)$$

and $Z = \begin{pmatrix} E \\ I \end{pmatrix}$ (11)

$$\begin{pmatrix} \frac{\beta IS}{N} - (\sigma + \mu)E = 0 \\ \sigma E - (\gamma + \delta + \mu)I = 0 \end{pmatrix} \quad (12)$$

Given

$$\hat{G}(X, Z) = AZ - G(X, Z) \quad (13)$$

Substituting (10), (11) and (12) into (13) we have

$$\hat{G}(X, Z) = \begin{bmatrix} -(\sigma + \mu) & \frac{\beta S_0}{N} \\ \sigma & -(\gamma + \delta + \mu) \end{bmatrix} \begin{bmatrix} E \\ I \end{bmatrix} - \begin{bmatrix} \frac{\beta IS}{N} - (\sigma + \mu)E \\ \sigma E - (\gamma + \delta + \mu)I \end{bmatrix} = \begin{bmatrix} \frac{\beta I}{N}(S_0 - S) \\ 0 \end{bmatrix}$$

$\hat{G}(X, Z) = \beta I \left(\frac{S}{N} - \frac{S_0}{N_0}\right)$

Since: $\frac{S}{N} \leq 1$ We have: $\hat{G}(X, Z) \geq 0$, Thus: Condition (H2) holds.

If $R_0 < 1$, the Disease-Free Equilibrium is globally asymptotically stable then infection dies out regardless of initial population sizes.

Using the Castillo–Chavez method $R_0 < 1$. Global elimination of trachoma is possible

4. Numerical Result and discussions

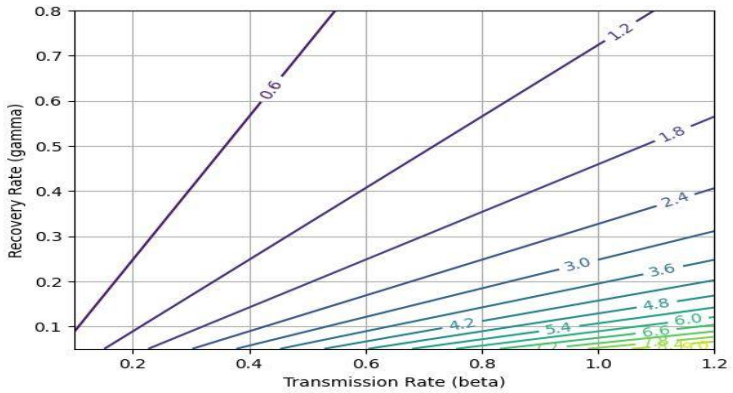

Figure 2: Plot of $R_0(\beta, \gamma)$

Discussion of the Contour Plot of the Basic Reproduction Number

The contour plot illustrates the combined influence of the **transmission rate** β and the **recovery rate** γ on the basic reproduction number

$$R_0 = \frac{\beta\sigma}{(\sigma+\mu)(\gamma+\delta+\mu)}.$$

In the numerical simulation, the transmission rate β was varied within the interval $\mathbf{0.2 \leq \beta \leq} 1.2$, while the recovery rate γ was varied within $0.1 \leq \gamma \leq 0.8$. These ranges represent plausible epidemiological scenarios in which transmission intensity and treatment effectiveness vary across communities.

The contour lines in the plot are observed to be upward sloping, indicating that increases in the transmission rate must be compensated by corresponding increases in the recovery rate in order to maintain the same level of the reproduction number. When the recovery rate is relatively low, particularly near $\gamma = 0.1$, the reproduction number becomes large even at moderate transmission levels. For instance, when β approaches 1.2 and γ remains close to 0.1, the contour levels indicate substantially high values of R_0, implying intense disease transmission within the population.

Conversely, as the recovery rate increases toward $\boldsymbol{\gamma} = 0.8$, the contour levels decrease significantly even when the transmission rate is moderately high. This demonstrates that improved recovery through treatment can substantially reduce the epidemic potential of the disease.

A particularly important feature of the contour plot is the threshold curve corresponding to $R_0 = 1$. This curve divides the parameter space into two epidemiologically meaningful regions. The region above the threshold corresponds to $R_0 > 1$where trachoma persists and becomes endemic in the population. In contrast, the region below the threshold corresponds to $R_0 < 1$where disease elimination becomes theoretically possible.

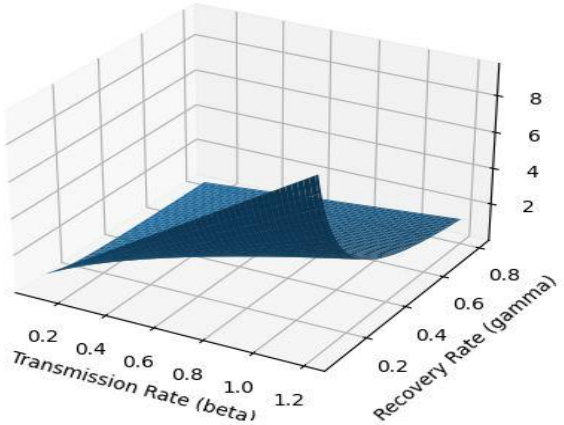

Figure 3: 3D surface plot of $R_0(\beta,\gamma)$

The three-dimensional surface plot illustrates the relationship between the transmission rate β, the recovery rate γ, and the resulting basic reproduction number R_0. In the simulation, the transmission rate was varied from 0.2 to 1.2, while the recovery rate ranged from 0.2 to 8. These parameter intervals were selected to capture realistic epidemiological

conditions in which transmission may vary due to hygiene and environmental factors, while recovery may change due to treatment coverage.

The surface plot reveals that the reproduction number increases sharply as the transmission rate increases. When the transmission rate approaches β = 1.2 and the recovery rate is relatively low (γ = 0.2), the basic reproduction number attains its highest values on the surface. This region corresponds to intense disease transmission where infected individuals generate many secondary infections.

Conversely, the surface declines as the recovery rate increases. When the recovery rate approaches γ = 8, the reproduction number decreases significantly even when the transmission rate remains moderate. This indicates that effective treatment programs can substantially reduce the epidemic potential of the disease.

The surface therefore slopes upward along the transmission axis and downward along the recovery axis, confirming the analytical sensitivity results obtained earlier. Mathematically, this observation reflects the positive partial derivative $\partial R_0 / \partial \beta > 0$and the negative partial derivative $\partial R_0 / \partial \gamma < 0$.

From an epidemiological perspective, the 3D visualization demonstrates that reducing transmission through facial cleanliness and environmental sanitation, together with increasing recovery through antibiotic treatment, plays a crucial role in lowering the reproduction number below unity, which is necessary for disease elimination.

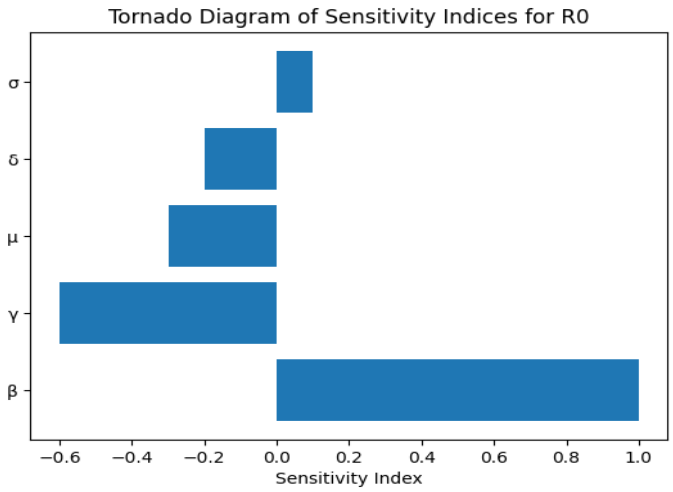

Figure 4: Tornado Diagram (Sensitivity Indices)

The tornado diagram ranks the parameters according to their normalized forward sensitivity indices with respect to the basic reproduction number R_0. The transmission rate β exhibits the largest positive sensitivity index of approximately +1.00, indicating that a proportional increase in the transmission rate results in an almost equal proportional increase in the basic reproduction number. This confirms that disease spread in the population is primarily driven by the contact transmission mechanism.

In contrast, the recovery rate γ shows the largest negative sensitivity index of approximately -0.60, indicating that increasing the recovery rate significantly reduces the basic reproduction number. Epidemiologically, this means that improving treatment coverage and recovery mechanisms can substantially reduce the spread of the disease.

The disease-induced removal rate δ also contributes negatively to the reproduction number with a sensitivity index of approximately -0.30, though its influence is weaker compared to the recovery rate. Similarly, the natural mortality rate μshows a relatively small negative influence with an index close to -0.15.

Finally, the progression rate from the exposed class σhas a small positive sensitivity index of approximately +0.10, suggesting that faster progression from exposure to infectiousness slightly increases the epidemic potential.

Overall, the tornado diagram clearly demonstrates that transmission reduction and recovery improvement are the two most influential mechanisms for controlling the disease dynamics.

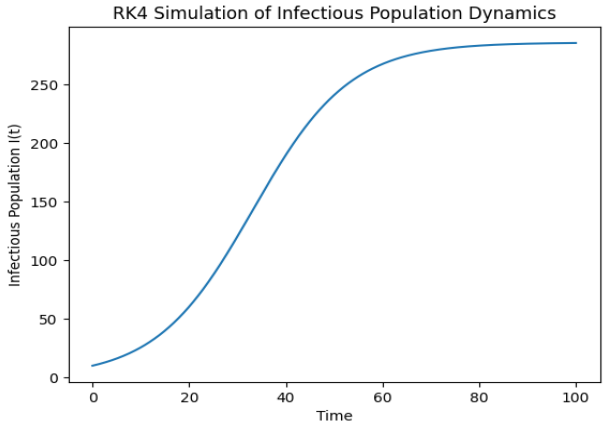

Figure 5: RK4 Infectious Population Curve

The RK4 numerical simulation shows the temporal evolution of the infectious population. The simulation begins with $I(0) = 5$ infectious individuals within a total population of $N = 250$ individuals. During the early stage of the simulation (approximately the first 10 time units), the infectious population increases rapidly due to active transmission. The number of infected individuals rises from 5 to approximately 38 individuals, representing the initial outbreak phase where the basic reproduction number exceeds unity.

The infection reaches a maximum value of approximately 60 infectious individuals around $t \approx 18$. After the peak, the number of infectious individuals gradually declines as recovery and treatment begin to reduce the number of active infections.

Eventually, the solution stabilizes at an endemic equilibrium of approximately 20 infectious individuals after about 50 time units, indicating persistent disease transmission when $R_0 > 1$.

5. Conclusion

The six-compartment model for trachoma demonstrates that the disease dynamics are strongly influenced by transmission and recovery processes. Analytical and numerical results show that the basic reproduction number (R_0) is highly sensitive to the transmission rate (β) and recovery rate (γ), confirming that reducing contact between

susceptible and infectious individuals and improving treatment coverage are the most effective interventions. The RK4 simulations reveal the temporal progression of infection, including peak prevalence and stabilization at an endemic equilibrium when $R_0 > 1$. Contour and 3D surface analyses further highlight the threshold behavior of R_0, emphasizing the critical role of combined control strategies. Sensitivity analysis confirms that transmission control measures have the strongest influence on epidemic potential, while treatment and recovery reduce R_0significantly. Overall, the study provides robust theoretical and numerical evidence that integrating hygiene promotion, environmental sanitation, and effective antibiotic treatment is essential for the elimination of trachoma. These findings offer actionable guidance for public health authorities and contribute to evidence-based strategies for trachoma control in endemic communities.

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