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

African Scientific Reports 5 (2026) 448

African Scientific Reports

Transmission dynamics of community-and hospital-acquired infections: insights from mathematical modelling

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Abstract

Hospital-associated infections (HAIs) remain a critical public health challenge, particularly in resource-limited settings where surveillance and infection control are inadequate. This study develops and analyzes a modified $SEIR$ mathematical model, denoted by $SE_cI_cE_hI_hR$, to capture the transmission dynamics of infectious diseases across hospital and community settings. The model incorporates both community and hospital transmission of infections, which have not been addressed jointly in previous studies. Using the next-generation matrix approach, the basic reproduction number (R_0) was determined. Local and global stability analyses revealed that the disease-free equilibrium is stable when $R_0 < 1$, but unstable otherwise. Sensitivity analysis identified the recruitment rate, community contact rate, and hospital infection rate as the most influential parameters, with community transmission exerting a stronger impact on the overall dynamics than hospital transmission. Numerical simulations further confirmed the dominant role of community transmission over hospital infections. Bifurcation analysis confirmed the possibility of backward bifurcation, indicating the possible coexistence of endemic and disease-free equilibrium states, even when $R_0 < 1$. These findings highlight the complex interplay between hospital and community transmission and indicate that effective control strategies must prioritize reducing community contact rates while strengthening hospital infection-prevention measures. The model provides insights for policymakers and healthcare managers designing evidence-based interventions to mitigate HAIs and community outbreaks, particularly in countries facing systemic healthcare challenges.

DOI:10.46481/asr.2026.5.2.448

Keywords: Community transmission, Local and global stability, Bifurcation analysis, Hospital-acquired infection.

Article History:
Received: 15 January 2026
Received in revised form: 29 April 2026
Accepted for publication: 03 May 2026
Available online: 29 May 2026

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1. Introduction

Notwithstanding advances in medicine and biology, hospital-associated infections (HAIs) have continued to rise in recent decades. The United States Centers for Disease Control and Prevention (CDC) reported that HAIs remain among the most common problems in healthcare, with about 687,000 cases each year, leading to around 72,000 deaths [1], and more than 140,000 deaths recorded annually worldwide [2]. The burden of HAIs is mainly due to the emergence of severe infections and drug-resistant bacterial strains. Other serious concerns include hospital-acquired pneumonia and viruses such as influenza, severe acute respiratory syndrome (SARS), Middle East respiratory syndrome (MERS), COVID-19, and Ebola, which are public health issues in many countries [1].

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In Nigeria, infectious diseases, especially HAIs, constitute a significant challenge to patient safety. These infections can be caused by bacteria, viruses, and fungi, which spread through direct contact, airborne transmission, or environmental contamination. HAIs are particularly risky for patients and healthcare workers, especially during outbreaks such as the COVID-19 pandemic [3]. Despite efforts to control infectious diseases, Nigerian hospitals and other low-income countries still face regular outbreaks, indicating the need for more effective infection-control measures [3–5]. Mathematical modelling has become an essential tool for understanding how these infections spread, but little research has examined how infections are transmitted from hospitals to the community and vice versa, given the country's unique healthcare challenges. As far as we are aware, no mathematical model has been developed to examine the transmission dynamics of community and hospital-associated infections. This study aims to fill this gap by developing a mathematical model to investigate how infectious diseases and HAIs spread in hospitals and communities. The goal is to identify key factors driving transmission and suggest evidence-based policies to reduce the spread of infections in hospitals and communities.

The Nigerian Centre for Disease Control (NCDC) has responded to disease outbreaks by activating emergency operations centres, the highest level of emergency response in Nigeria. However, Abubakar *et al.* [6] noted that the actual impact of outbreaks might be worse than reported because of poor disease tracking and inadequate healthcare quality. HAIs continue to threaten Nigeria's health sector, leading to increased mortality and healthcare costs. Disease spread in hospitals and communities is complex, involving interactions among patients, healthcare workers, and community members. There is a clear need for research that captures the complex dynamics of infectious disease transmission in both hospitals and communities.

Milazzo *et al.* [7] used a model to study healthcare-associated infections through computer simulation, focusing on pathogens such as methicillin-resistant *Staphylococcus aureus* (MRSA) and SARS, and examining how these infections spread. Their model considered factors such as individual mobility, hand hygiene, and other control measures. They found that strict patient separation might be harmful if it is not combined with personal measures. Alimi and Ayoade [8] developed an SEIR model to study the effects of vaccination on infectious diseases. Their findings showed that vaccinated individuals developed immunity and moved into the recovered category, thereby reducing transmission rates and deaths. They emphasized the importance of vaccinating populations before outbreaks to lower the spread of infections. However, their model did not address hospital-associated infections. Assab *et al.* [9] reviewed studies on HAIs from various databases and found that MRSA was the most studied pathogen. Their research considered how models simulated the outbreak of these pathogens and assessed the long-term effectiveness of control measures. Omame and Abbas [10] investigated how COVID-19 and dengue vaccinations influence the spread of the Zika virus using a model that incorporates specific infection rates. They found that co-infections can complicate transmission dynamics and that increased vaccination efforts against COVID-19 and dengue could effectively reduce the spread of Zika. Overall, their study highlights the interconnections between these diseases and the advantages of integrated vaccination strategies.

Adewole *et al.* [11], on the other hand, developed an SEQAIR model, which showed that effective intervention strategies could help contain infections. Their analysis highlighted the importance of isolating infectious individuals and suggested that achieving a virus-free population would take time. Adedire and Ndam [12] proposed a model to study COVID-19 in Plateau State, Nigeria. Their results suggested that COVID-19 tended to become endemic but could reach a disease-free state over time. Akinsola *et al.* [13] formulated another model emphasizing the importance of testing and isolation. Iboi *et al.* [14] designed a model to analyse the community impact of COVID-19 in Nigeria, with the result that the disease could be controlled if the basic reproduction number (R_0) was less than 1. However, their model assumed that recovered individuals had permanent immunity, which is not always accurate. In a similar study, Ndam [15] examined COVID-19 transmission, focusing on community lockdown and isolation as strategies to contain disease spread, and concluded that lockdown was more effective than isolation. Wang *et al.* [16] developed a predictive model for healthcare-associated infections in the intensive care unit using machine learning. They identified key risk factors for HAIs, which helped identify at-risk patients, but noted that their sample size was not large enough to support accurate predictions. Tahir *et al.* [17] created a mathematical model to study how patients move within hospitals and how this movement can lead to the spread of infections acquired in healthcare settings. They proposed a ranking system that should focus on the most connected departments to help design better ways to reduce infection risks.

Overall, while many studies have examined infectious diseases and HAIs, most do not consider the simultaneous effects of infections from both hospital and community settings and instead model them separately. The aim of this study is to develop a comprehensive mathematical model that includes both hospital-associated infections and community transmission to provide effective control measures, given the inevitable relationships between healthcare centres and the community during disease outbreaks. The remainder of the paper is organized as follows: Section 2 presents the model formulation, Section 3 presents model analysis, Section 4 presents sensitivity analysis, Section 5 presents simulation and discussion, and Section 6 presents the conclusion and recommendations.

2. Materials and methods

A compartmental model consisting of six nonlinear differential equations is constructed in this section to examine the transmission dynamics of infectious diseases with HAIs. To develop the model, the total population $N(t)$ is partitioned into six epidemiological compartments: susceptible $S(t)$, community exposed $E_c(t)$, hospital exposed $E_h(t)$, community infected $I_c(t)$, hospital infected $I_h(t)$,

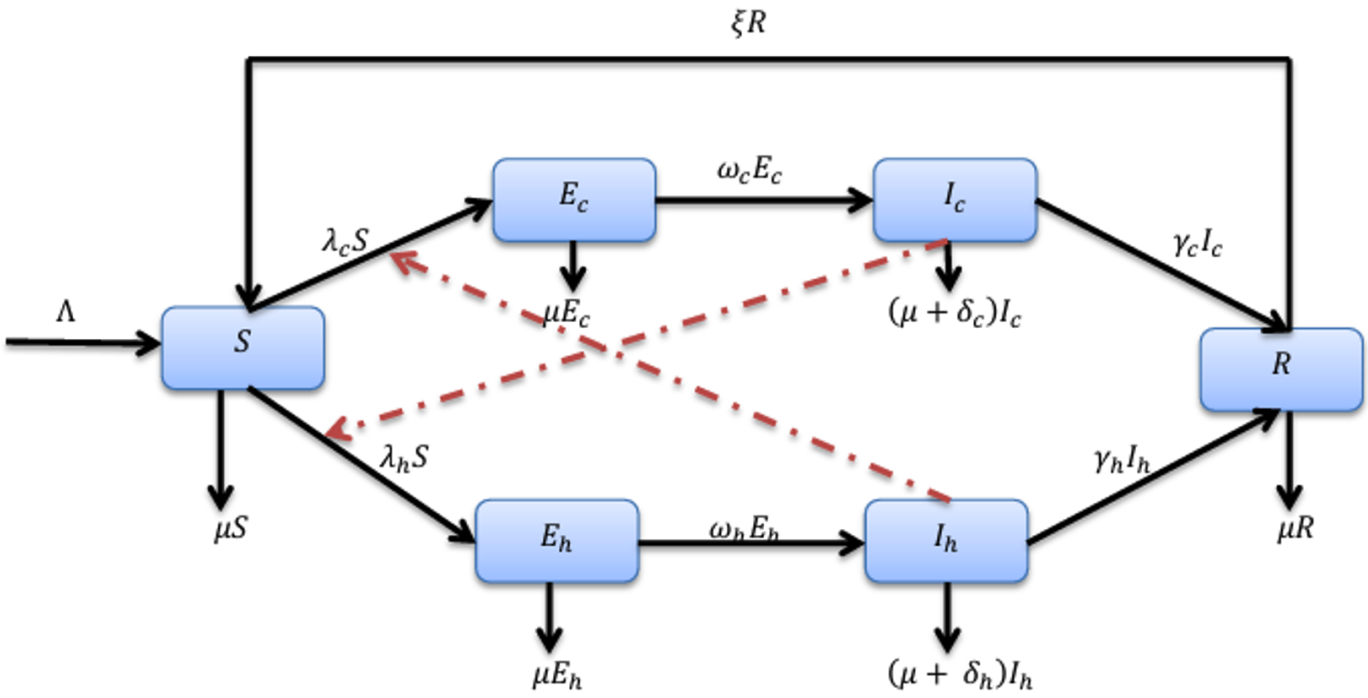


Figure 1: Schematic diagram for the community and hospital infection model.

Table 1: Parameter values for the $S E_c I_c E_h I_h R$ model.

Description	Parameter	Value	Source
Recruitment rate	Λ	0.45	[8]
Contact rate for community	β_c	0.091	[18]
Transition rate of community-exposed to infected	ω_c	0.1923	Assumed
Natural death rate	μ	0.0183	[19]
Recovery rate for community	γ_c	0.03	[20]
Disease-induced death rate for community	δ_c	0.2	Assumed
Disease-induced death rate for hospital	δ_h	0.15	Assumed
Recovery rate for hospital	γ_h	0.07143	[14]
Rate of loss of immunity	ξ	0.0001	[13]
Hospital infection rate	β_h	0.075	Assumed
Transition rate of hospital-exposed to infected	ω_h	0.120	Assumed

and recovered $R(t)$, such that

$$N(t) = S(t) + E_c(t) + I_c(t) + E_h(t) + I_h(t) + R(t).$$

2.1. Model formulation

The governing equations of the model are formulated based on the following assumptions: the population size in the model is differentiable with respect to time, and the epidemic process is deterministic; all susceptible individuals may be infected by infectious individuals; and recovery from the disease does not confer permanent immunity because reinfection can occur. The diagrammatic representation of the model is shown in Figure 1.

In this model, both community and hospital transmission are considered. The model parameters are the recruitment rate Λ, natural death rate μ, contact rate for the community β_c, exposed rate of human transmission for the community ω_c, recovery rate for the community γ_c, disease-induced death rate for the community δ_c, disease-induced death rate for the hospital δ_h, recovery rate for the hospital γ_h, contact rate for the hospital β_h, transition rate from the hospital-exposed to infected class ω_h, and rate at which

a recovered person loses immunity ξ. The governing equations for the proposed model are expressed as the following system of nonlinear differential equations:

$$\begin{aligned}
\frac{\mathrm{d}S}{\mathrm{d}t} &= \Lambda + \xi R - (\beta_c + \beta_h)(I_c + I_h)S - \mu S,\\
\frac{\mathrm{d}E_c}{\mathrm{d}t} &= \beta_c(I_c + I_h)S - (\mu + \omega_c)E_c,\\
\frac{\mathrm{d}I_c}{\mathrm{d}t} &= \omega_c E_c - (\mu + \delta_c + \gamma_c)I_c,\\
\frac{\mathrm{d}E_h}{\mathrm{d}t} &= \beta_h(I_c + I_h)S - (\mu + \omega_h)E_h,\\
\frac{\mathrm{d}I_h}{\mathrm{d}t} &= \omega_h E_h - (\mu + \delta_h + \gamma_h)I_h,\\
\frac{\mathrm{d}R}{\mathrm{d}t} &= \gamma_c I_c + \gamma_h I_h - (\xi + \mu)R.
\end{aligned} \tag{1}$$

The corresponding non-negative initial conditions are $S(0) > 0$, $E_c(0) \geq 0$, $I_c(0) \geq 0$, $E_h(0) \geq 0$, $I_h(0) \geq 0$, and $R(0) \geq 0$, where $\lambda_c = \beta_c(I_c + I_h)$ and $\lambda_h = \beta_h(I_c + I_h)$.

2.2. Invariant region and disease-free and endemic equilibria

The boundedness and feasibility region of solutions of the model, as well as the disease-free and endemic steady states, are determined in this section.

2.2.1. Invariant region

From the model equations (1), the total population is given by

$$N(t) = S(t) + E_c(t) + I_c(t) + E_h(t) + I_h(t) + R(t).$$

Hence,

$$\begin{aligned}
\frac{\mathrm{d}N(t)}{\mathrm{d}t} &= \frac{\mathrm{d}S}{\mathrm{d}t} + \frac{\mathrm{d}E_c}{\mathrm{d}t} + \frac{\mathrm{d}I_c}{\mathrm{d}t} + \frac{\mathrm{d}E_h}{\mathrm{d}t} + \frac{\mathrm{d}I_h}{\mathrm{d}t} + \frac{\mathrm{d}R}{\mathrm{d}t}\\
&= \Lambda - \mu N - \delta_c I_c - \delta_h I_h \leq \Lambda - \mu N.
\end{aligned}$$

Thus,

$$\frac{\mathrm{d}N}{\mathrm{d}t} \leq \Lambda - \mu N \quad \Rightarrow \quad 0 \leq N \leq \frac{\Lambda}{\mu} - Ae^{-\mu t},$$

where A is a constant. Consequently, as $t \to \infty$, $N \leq \Lambda/\mu$. Hence, the region in which the model solutions are bounded and biologically meaningful is given by

$$\Omega = \left\{(S, E_c, I_c, E_h, I_h, R) \in \mathbb{R}^6_+ : S + E_c + I_c + E_h + I_h + R \leq \frac{\Lambda}{\mu}\right\}.$$

2.2.2. Disease-free and endemic equilibrium states

From the system of equation (1), the disease-free equilibrium for the model, E_0, exists in the absence of infection in both hospital and community settings. This implies that $E_c = E_h = I_c = I_h = R = 0$, and we obtain $\Lambda - \mu S = 0 \Rightarrow S = \Lambda/\mu$. Therefore, the disease-free equilibrium is given by

$$E_0 = (S, E_c, I_c, E_h, I_h, R) = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0\right).$$

The disease-endemic equilibrium point is given by

$$E_1 = (S^*, E_c^*, I_c^*, E_h^*, I_h^*, R^*),$$

where

$$S^* = \frac{\Lambda(\mu+\xi)+\xi(\gamma_c I_c^* + \gamma_h I_h^*)}{(\mu+\xi)\left[\mu+(\beta_c+\beta_h)(I_c^*+I_h^*)\right]},$$
$$E_c^* = \frac{\beta_c(I_c^*+I_h^*)\left[\Lambda(\mu+\xi)+\xi(\gamma_c I_c^*+\gamma_h I_h^*)\right]}{(\mu+\xi)(\mu+\omega_c)\left[\mu+(\beta_c+\beta_h)(I_c^*+I_h^*)\right]},$$
$$E_h^* = \frac{\beta_h(I_c^*+I_h^*)\left[\Lambda(\mu+\xi)+\xi(\gamma_c I_c^*+\gamma_h I_h^*)\right]}{(\mu+\xi)(\mu+\omega_h)\left[\mu+(\beta_c+\beta_h)(I_c^*+I_h^*)\right]},$$
$$R^* = \frac{\gamma_c I_c^* + \gamma_h I_h^*}{\mu+\xi}.$$

2.3. Basic reproduction number, R_0

The stability of the infection-free steady state E_0 is governed by the basic reproduction number R_0, which is a mathematical quantity considered paramount in the study of the transmission dynamics of infectious diseases. R_0 is defined as the average number of secondary infections produced by a single infectious individual in a population where everyone is susceptible [21]. It is computed using the next-generation matrix approach described by Diekmann *et al.* [21]. The infected compartments of the model can be expressed as

$$\frac{\mathrm{d}X_i}{\mathrm{d}t} = F_i(x) - V_i(x),$$

where F_i represents the new infections going into compartment i, and V_i represents the transfer of infections into and out of the compartment. From the model equations (1),

$$F_1 = \beta_c(I_c+I_h)S, \quad F_2 = 0, \quad F_3 = \beta_h(I_c+I_h)S, \quad F_4 = 0,$$

while

$$V_1 = (\mu+\omega_c)E_c,$$
$$V_2 = (\mu+\delta_c+\gamma_c)I_c - \omega_c E_c,$$
$$V_3 = (\mu+\omega_h)E_h,$$
$$V_4 = (\mu+\delta_h+\gamma_h)I_h - \omega_h E_h.$$

Hence, finding the Jacobian matrices at the DFE, we have

$$F(E_0) = \begin{pmatrix} 0 & \frac{\beta_c\Lambda}{\mu} & 0 & \frac{\beta_c\Lambda}{\mu} \\ 0 & 0 & 0 & 0 \\ 0 & \frac{\beta_h\Lambda}{\mu} & 0 & \frac{\beta_h\Lambda}{\mu} \\ 0 & 0 & 0 & 0 \end{pmatrix}, \quad V(E_0) = \begin{pmatrix} a_1 & 0 & 0 & 0 \\ -\omega_c & a_2 & 0 & 0 \\ 0 & 0 & a_3 & 0 \\ 0 & 0 & -\omega_h & a_4 \end{pmatrix},$$

where $a_1 = \mu+\omega_c$, $a_2 = \mu+\delta_c+\gamma_c$, $a_3 = \mu+\omega_h$, and $a_4 = \mu+\delta_h+\gamma_h$. The basic reproduction number, denoted by $R_0 = \rho(FV^{-1})$, represents the largest eigenvalue of FV^{-1}. Therefore,

$$R_0 = \frac{\Lambda}{\mu}\left(\frac{\beta_c\omega_c}{a_1a_2} + \frac{\beta_h\omega_h}{a_3a_4}\right) = \frac{\Lambda}{\mu}\left(R_{0c}+R_{0h}\right),$$

where

$$R_{0c} = \frac{\beta_c\omega_c}{(\mu+\omega_c)(\mu+\delta_c+\gamma_c)} \quad \text{and} \quad R_{0h} = \frac{\beta_h\omega_h}{(\mu+\omega_h)(\mu+\delta_h+\gamma_h)}$$

are the community and hospital-associated transmission basic reproduction numbers, respectively. Clearly, the basic reproduction number comprises both community and hospital transmissions. While R_{0c} measures the intensity of infection transmission in the community, R_{0h} determines how fast infections spread in hospital settings.

2.4. Local stability of the disease-free equilibrium

To determine the stability of the disease-free equilibrium, we compute the Jacobian matrix of the system at the disease-free equilibrium and obtain the eigenvalues required for this purpose. The result is summarized in the following theorem.

Theorem 2.1. The infection-free equilibrium E_0 of the model is locally asymptotically stable for $R_0 < 1$ and unstable when $R_0 > 1$.

Proof. To prove local stability of the DFE, we show that the Jacobian matrix $J(E_0)$ of the model equations (1) at the infection-free state has negative real eigenvalues or eigenvalues with negative real parts. The matrix $J(E_0)$ is given by

$$J(E_0) = \begin{pmatrix} -\mu & 0 & -b_1 & 0 & -b_1 & \xi \\ 0 & -a_1 & b_2 & 0 & b_2 & 0 \\ 0 & \omega_c & -a_2 & 0 & 0 & 0 \\ 0 & 0 & b_3 & -a_3 & b_3 & 0 \\ 0 & 0 & 0 & \omega_h & -a_4 & 0 \\ 0 & 0 & \gamma_c & 0 & \gamma_h & -(\mu+\xi) \end{pmatrix}, \tag{2}$$

where

$$b_1 = \frac{(\beta_c+\beta_h)\Lambda}{\mu}, \quad b_2 = \frac{\beta_c\Lambda}{\mu}, \quad b_3 = \frac{\beta_h\Lambda}{\mu}.$$

Some of the eigenvalues of the matrix (2) are $\lambda_1 = -\mu$ and $\lambda_2 = -(\mu+\xi)$, while the remaining four eigenvalues satisfy the characteristic equation

$$\lambda^4 + A\lambda^3 + B\lambda^2 + C\lambda + D = 0, \tag{3}$$

where

$$\begin{aligned} A &= a_1 + a_2 + a_3 + a_4, \\ B &= a_1a_2 + a_1a_3 + a_1a_4 + a_2a_3 + a_2a_4 + a_3a_4 - b_2\omega_c - b_3\omega_h, \\ C &= a_1a_2a_3 + a_1a_2a_4 + a_1a_3a_4 + a_2a_3a_4 - a_3b_2\omega_c - a_4b_2\omega_c - a_1b_3\omega_h - a_2b_3\omega_h, \\ D &= a_1a_2a_3a_4 - a_3a_4b_2\omega_c - a_1a_2b_3\omega_h. \end{aligned}$$

After simplifying D by substituting the values of the a_i's and b_i's, we obtain

$$D = (\mu+\omega_c)(\mu+\omega_h)(\mu+\delta_c+\gamma_c)(\mu+\delta_h+\gamma_h)(1-R_0).$$

Hence, by the Routh–Hurwitz criteria, if $A > 0$, $C > 0$, $D > 0$, and $ABC > C^2 + A^2D$, then $R_0 < 1$ and the disease-free equilibrium is locally asymptotically stable; otherwise, it is unstable. The epidemiological implication of this result is that, for the disease to be eliminated, the basic reproduction number must necessarily be reduced below unity. □

2.5. Global stability

The global stability analysis of the disease-free equilibrium state is important in epidemiological models because it reveals whether eradication of the disease can be achieved irrespective of the magnitude of the initial infection. The result is stated as follows.

Theorem 2.2. The infection-free equilibrium E_0 of the model is globally asymptotically stable for $R_0 < 1$ and unstable when $R_0 > 1$.

Proof. To examine the global stability of the DFE, we define the Lyapunov function

$$L = \alpha_1E_c + \alpha_2I_c + \alpha_3E_h + \alpha_4I_h.$$

The function possesses the basic properties of a Lyapunov function. Hence,

$$\begin{aligned} \frac{\mathrm{d}L}{\mathrm{d}t} &= \alpha_1\frac{\mathrm{d}E_c}{\mathrm{d}t} + \alpha_2\frac{\mathrm{d}I_c}{\mathrm{d}t} + \alpha_3\frac{\mathrm{d}E_h}{\mathrm{d}t} + \alpha_4\frac{\mathrm{d}I_h}{\mathrm{d}t} \\ &\le (\alpha_2\omega_c - \alpha_1(\mu+\omega_c))\,E_c + \left[(\alpha_1\beta_c + \alpha_3\beta_h)\frac{\Lambda}{\mu} - \alpha_2(\mu+\delta_c+\gamma_c)\right]I_c + (\alpha_4\omega_h - \alpha_3(\mu+\omega_h))\,E_h \\ &+ \left[(\alpha_1\beta_c + \alpha_3\beta_h)\frac{\Lambda}{\mu} - \alpha_4(\mu+\delta_h+\gamma_h)\right]I_h. \end{aligned} \tag{4}$$

Choosing

$$\alpha_1 = \frac{\mu+\delta_h+\gamma_h}{\mu+\omega_c}, \quad \alpha_2 = \mu+\delta_h+\gamma_h, \quad \alpha_3 = \frac{(\mu+\delta_c+\gamma_c)\omega_h}{\mu+\omega_h}, \quad \alpha_4 = \mu+\delta_c+\gamma_c,$$

equation (4) reduces to

$$\frac{\mathrm{d}L}{\mathrm{d}t} \le \frac{1}{(\mu+\delta_c+\gamma_c)(\mu+\delta_h+\gamma_h)}(R_0-1)(I_c+I_h).$$

For $R_0 < 1$, $\mathrm{d}L/\mathrm{d}t \le 0$, and by LaSalle's invariance principle, every solution emanating from Ω, the invariant region, converges to the largest singleton in $\{E_0 \in \Omega : \mathrm{d}L/\mathrm{d}t = 0\}$. Hence, the DFE is globally asymptotically stable. The implication of this conclusion is that the disease-free equilibrium can always be attained from any initial conditions. □

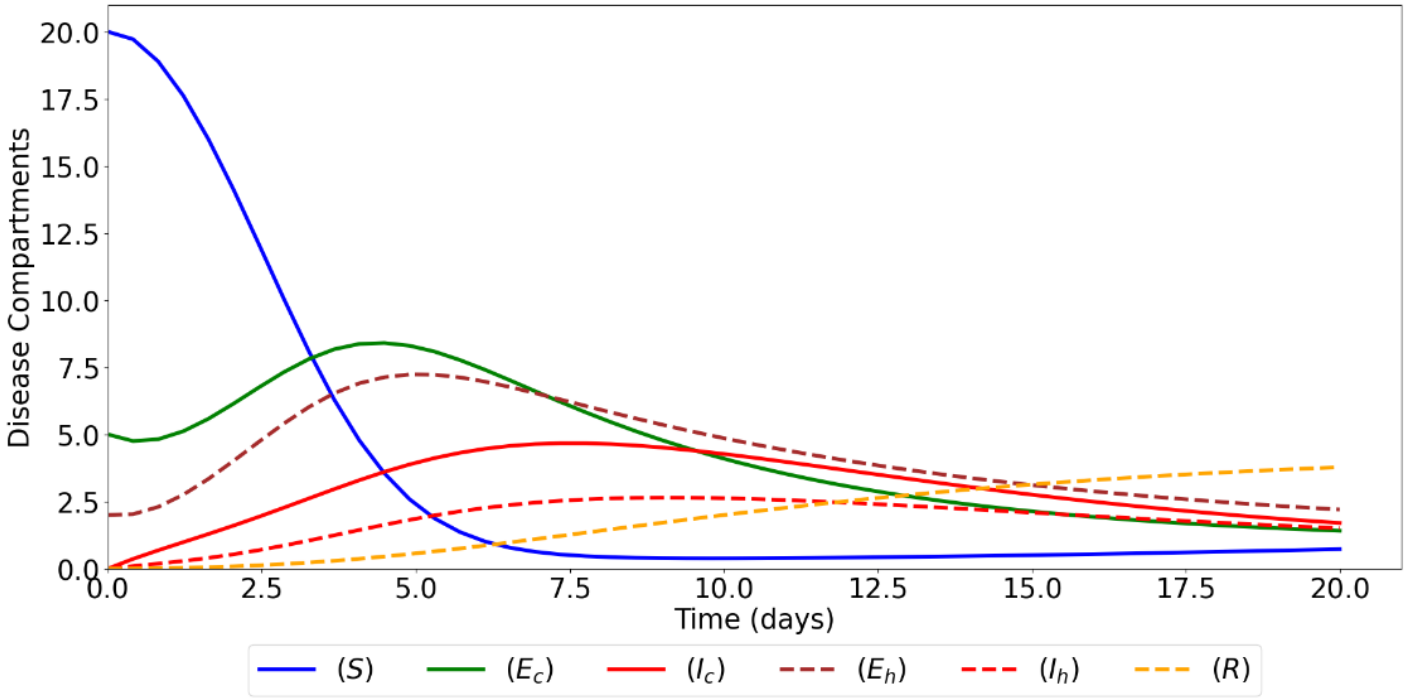


Figure 2: Plot of all compartments when $R_0 > 1$, using $\beta_c = 0.091$, $\beta_h = 0.075$, $\omega_c = 0.1923$, and $\omega_h = 0.120$.

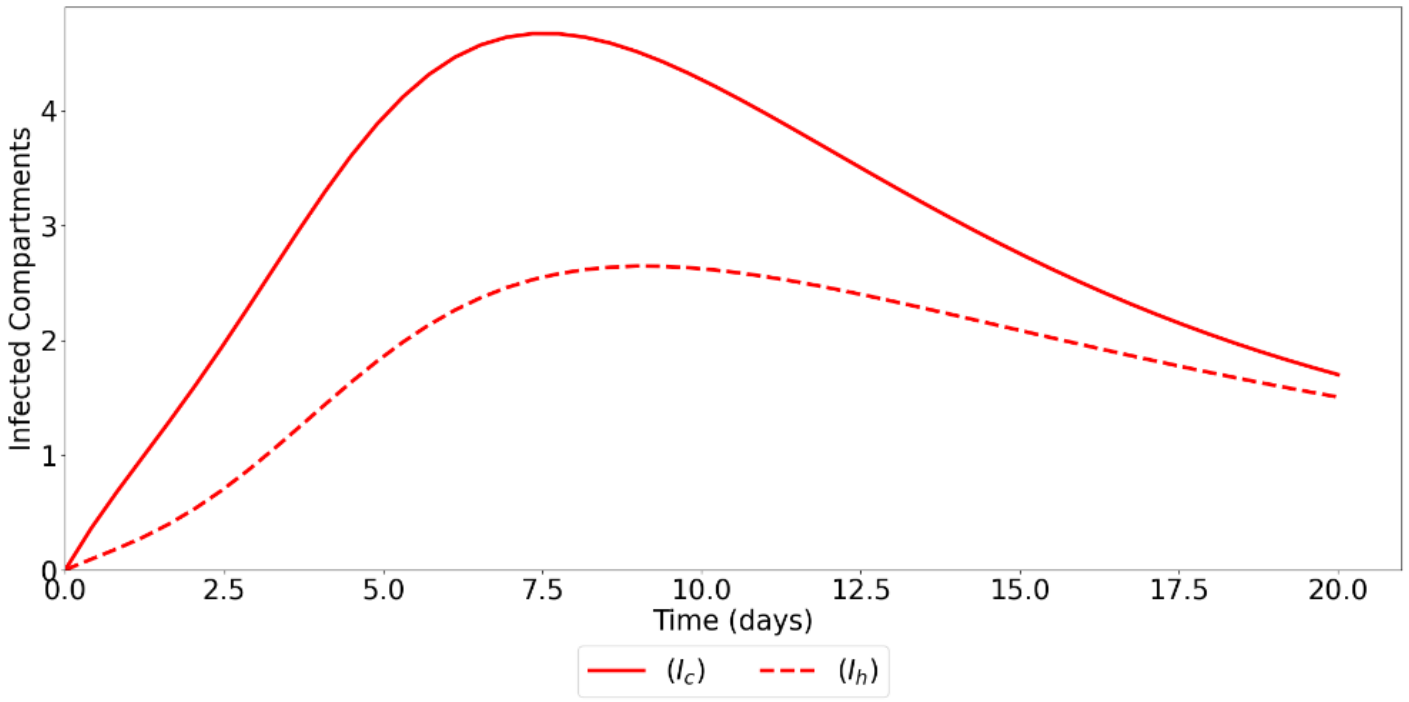


Figure 3: Comparison of community and hospital infections.

2.6. *Bifurcation analysis*

In this section, we examine the nature of bifurcation of the endemic equilibrium state to check for the possible occurrence of backward bifurcation, which is the state in which a stable endemic equilibrium coexists with a stable disease-free equilibrium when the associated reproduction number is less than unity. When this occurs, the condition for disease eradication, $R_0 < 1$, is no longer sufficient. We use the centre manifold theorem of Castillo-Chavez and Song [22] to check for backward bifurcation. The result of the bifurcation analysis is summarized in the following theorem.

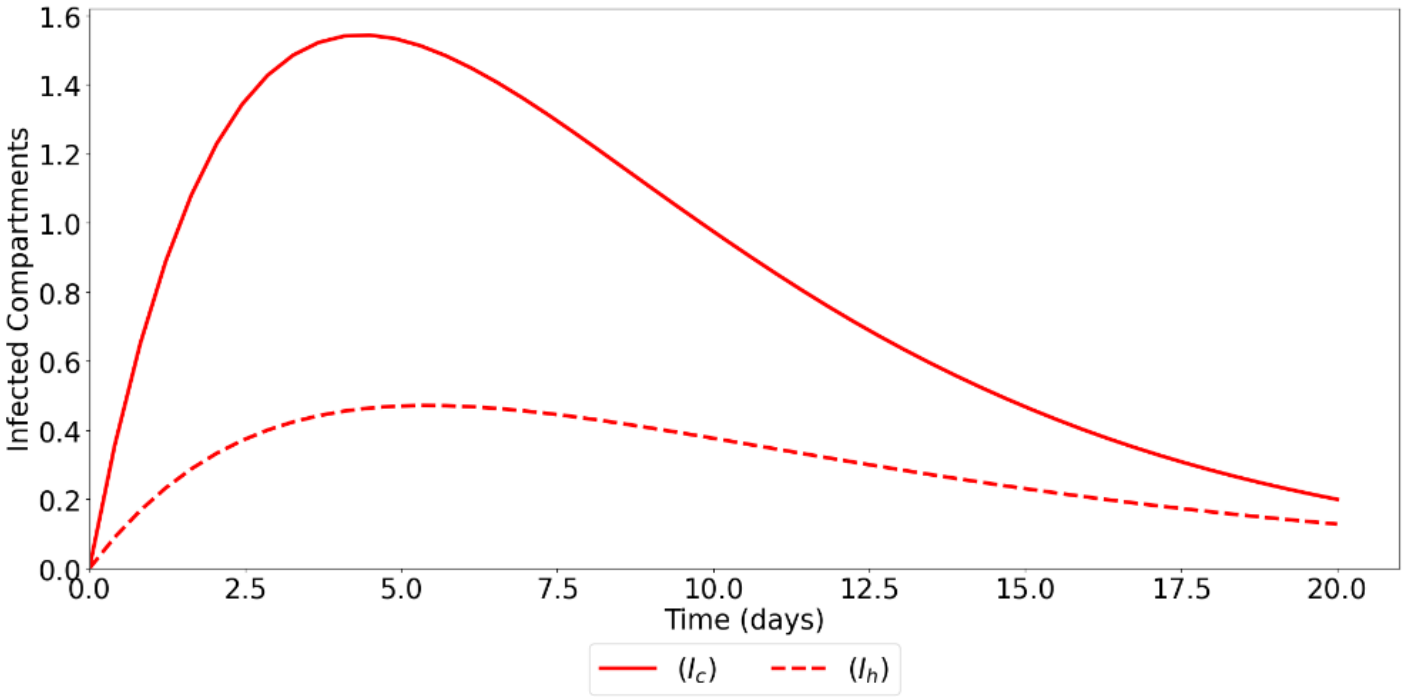


Figure 4: Community and hospital transmission in the absence of new infections ($\beta_c = \beta_h = 0$).

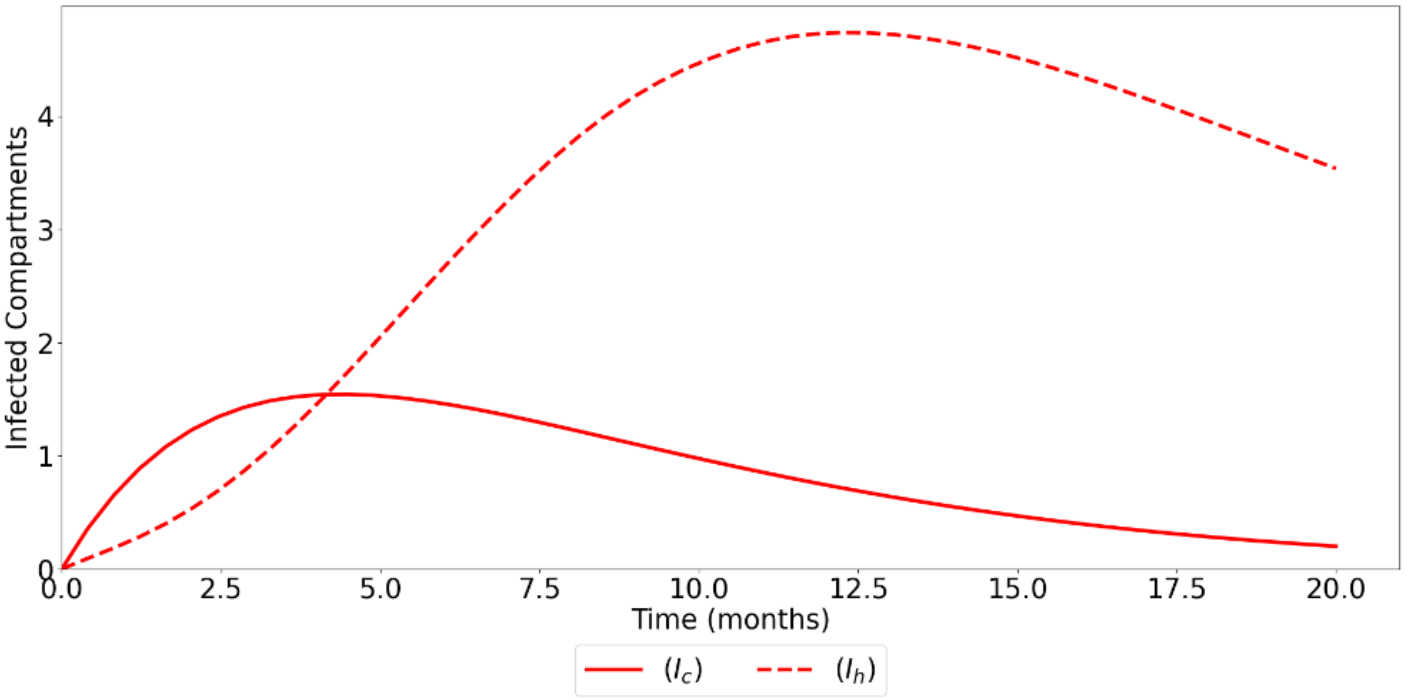


Figure 5: Community and hospital transmission in the absence of community infections ($\beta_c = 0$).

Theorem 2.3. The endemic equilibrium of model equation (1) undergoes a backward bifurcation at $R_0 = 1$ when

$$[\beta_c\Lambda + \mu(\mu+\omega_c)(\mu+\delta_c+\gamma_c)]\chi_1 + \beta_c\omega_c\Lambda\chi_2 > [\beta_c\Lambda + \mu(\mu+\omega_c)(\mu+\delta_c+\gamma_c)]\chi_2 + \beta_c\omega_c\Lambda\chi_1$$

Proof. We express the disease compartments as

$$S = x_1, \quad E_c = x_2, \quad I_c = x_3, \quad E_h = x_4, \quad I_h = x_5, \quad R = x_6.$$

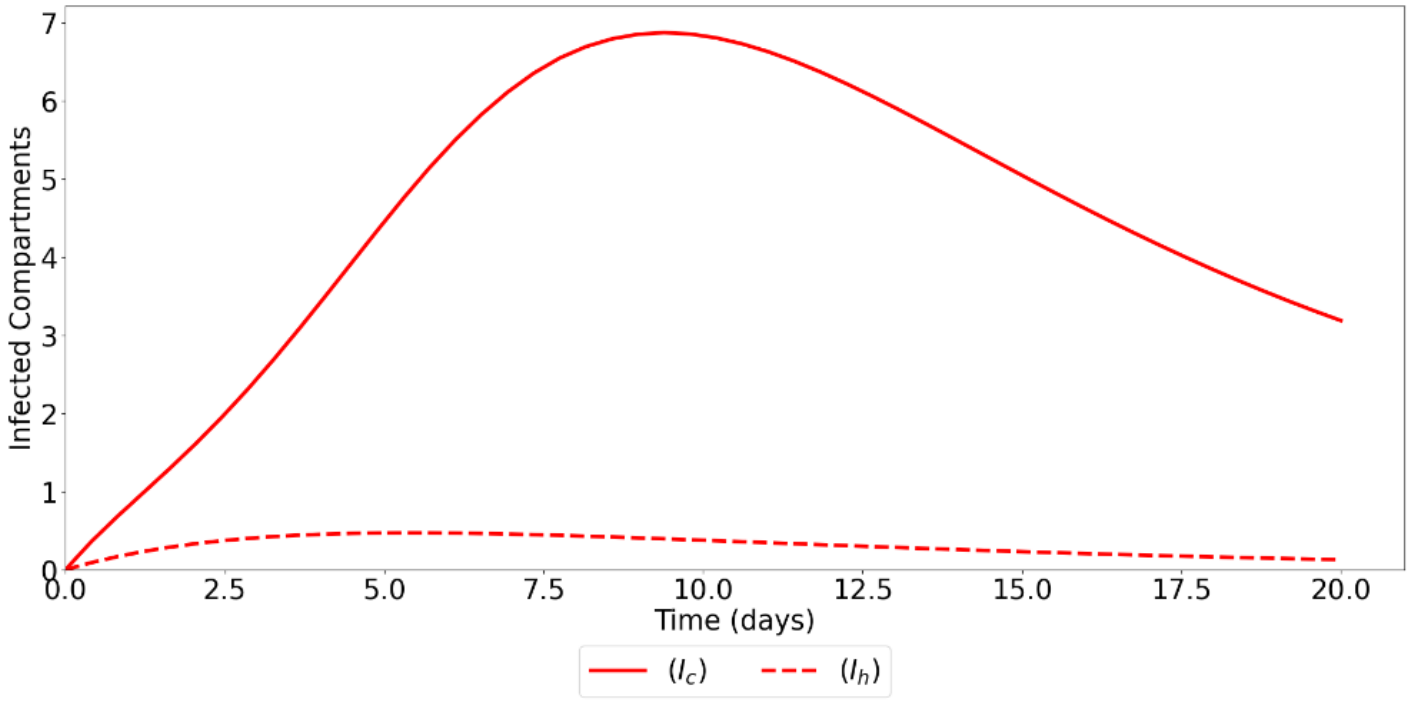


Figure 6: Community and hospital transmission in the absence of hospital infections ($\beta_h = 0$).

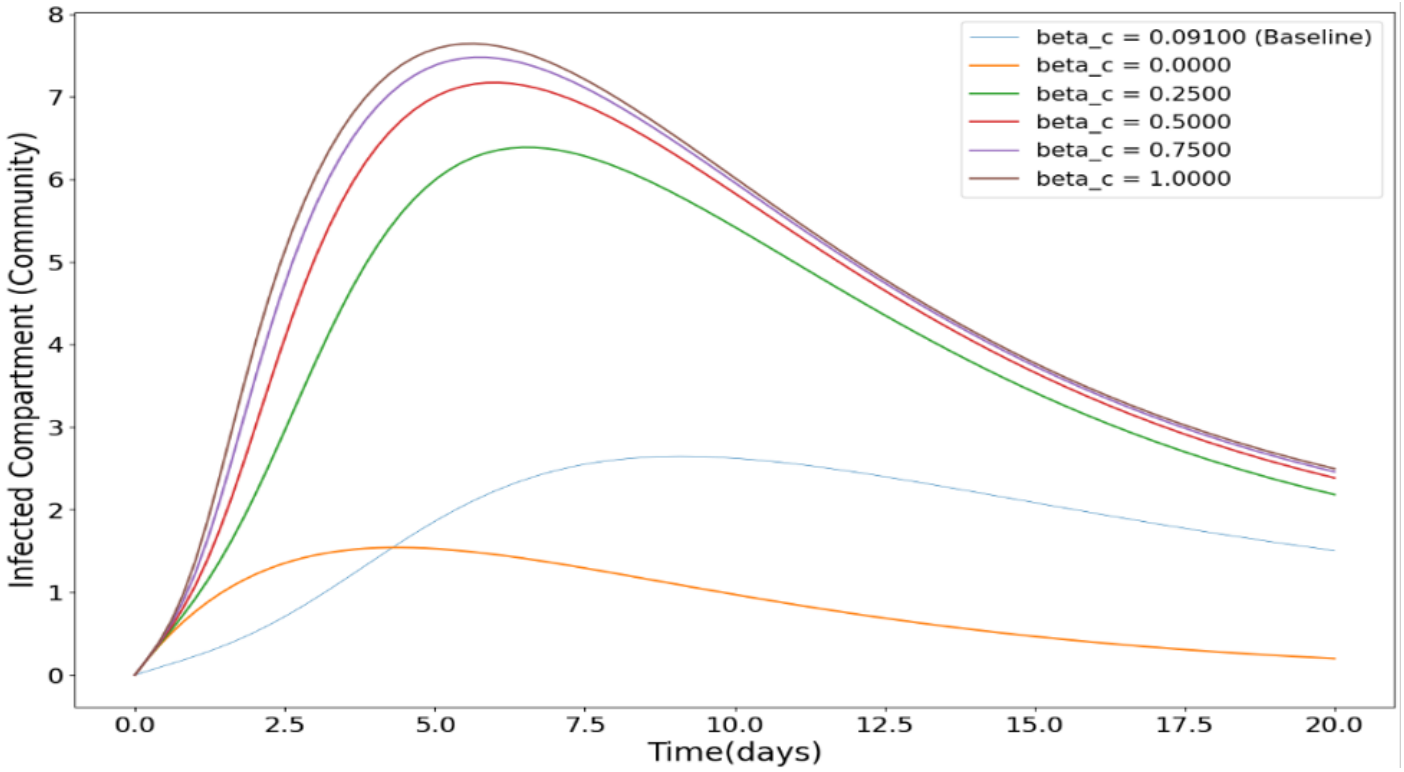


Figure 7: Plots of community infection I_c for various values of β_c.

Now, let $U = (x_1, x_2, x_3, x_4, x_5, x_6)^T$. Then, the governing equation (1) can be written as

$$\frac{dU}{dt} = (f_1, f_2, f_3, f_4, f_5, f_6)^T. \tag{5}$$

We consider β_c as the bifurcation parameter. Thus, the Jacobian matrix of the system equation (5) evaluated at the disease-free

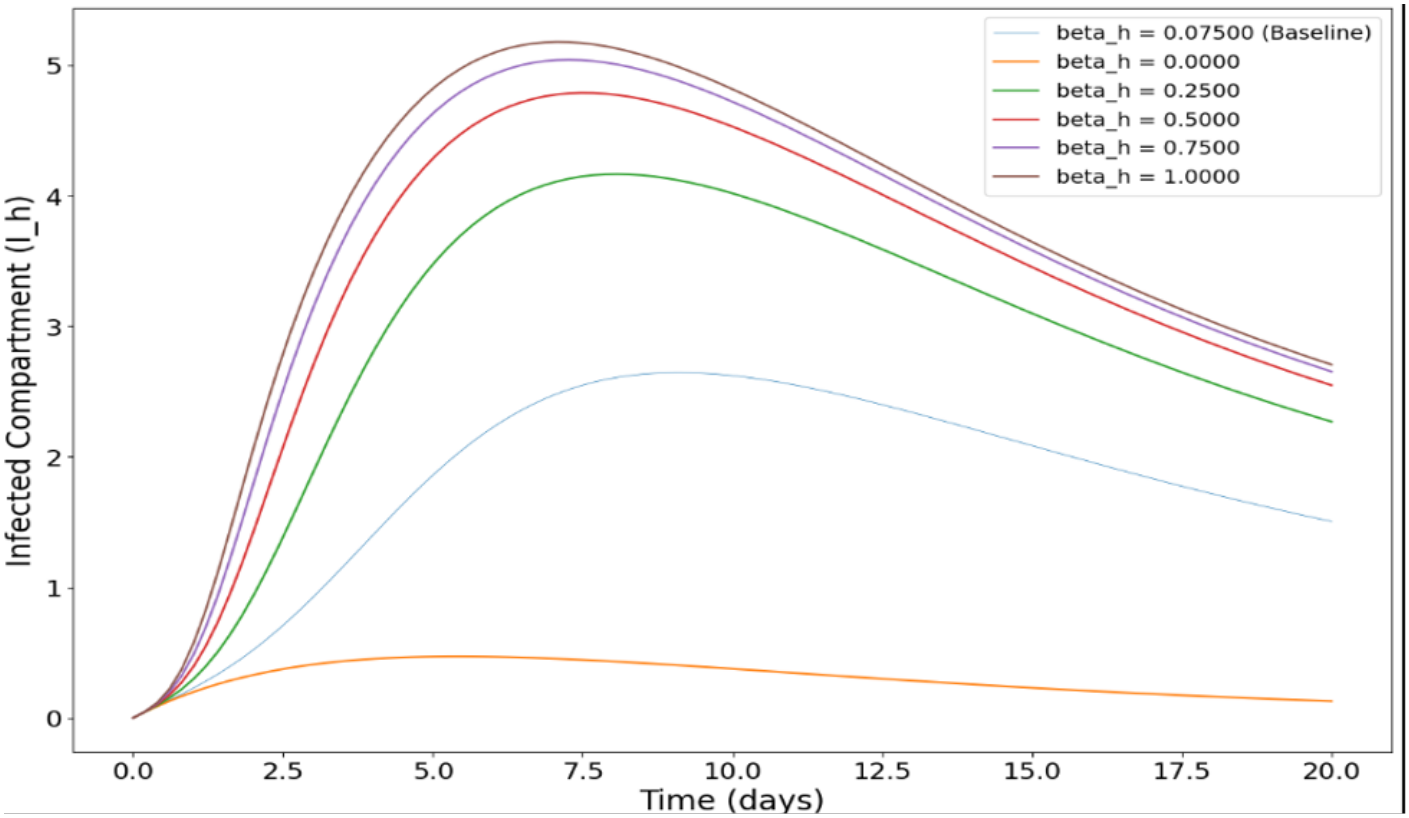


Figure 8: Plots of hospital-associated infection I_h for various values of β_h.

equilibrium state E_0 is given by

$$J(E_0) = \begin{pmatrix} -\mu & 0 & -\frac{(\beta_c+\beta_h)\Lambda}{\mu} & 0 & -\frac{(\beta_c+\beta_h)\Lambda}{\mu} & \xi \\ 0 & -(\mu+\omega_c) & \frac{\beta_c\Lambda}{\mu} & 0 & \frac{\beta_c\Lambda}{\mu} & 0 \\ 0 & \omega_c & -(\mu+\delta_c+\gamma_c) & 0 & 0 & 0 \\ 0 & 0 & \frac{\beta_h\Lambda}{\mu} & -(\mu+\omega_h) & \frac{\beta_h\Lambda}{\mu} & 0 \\ 0 & 0 & 0 & \omega_h & -(\mu+\delta_h+\gamma_h) & 0 \\ 0 & 0 & \gamma_c & 0 & \gamma_h & -(\mu+\xi) \end{pmatrix}. \tag{6}$$

This matrix has a simple zero eigenvalue at $R_0 = 1$. Let $W = (w_1, w_2, w_3, w_4, w_5, w_6)^T$ be the right eigenvector corresponding to the zero eigenvalue. The components of W are given by

$$w_1 = \frac{(\chi_1 - \chi_2)w_2}{\beta_c\Lambda(\mu+\delta_c+\gamma_c)(\mu+\delta_h+\gamma_h)\mu}, \quad w_2 = w_2 > 0, \quad w_3 = \frac{\omega_c w_2}{\mu+\delta_c+\gamma_c},$$

$$w_4 = \frac{\beta_h(\mu+\omega_c)w_2}{\beta_c\Lambda(\mu+\omega_h)}, \quad w_5 = \frac{\mu(\mu+\omega_c)(\mu+\delta_c+\gamma_c) - \beta_c\Lambda\omega_c}{\beta_c\Lambda(\mu+\delta_c+\gamma_c)}w_2,$$

$$w_6 = \frac{\omega_h[\mu(\mu+\omega_c)(\mu+\delta_c+\gamma_c) - \beta_c\Lambda\omega_c]w_2}{\beta_c\Lambda(\mu+\delta_c+\gamma_c)(\mu+\delta_h+\gamma_h)}, \text{ where}$$

$$\chi_1 = \mu(\mu+\omega_c)(\mu+\delta_c+\gamma_c)\xi\omega_h,$$

and

$$\chi_2 = \Big[\beta_c\xi\omega_c\omega_h + (\beta_c+\beta_h)(\mu+\omega_c)(\mu+\delta_c+\gamma_c)(\mu+\delta_h+\gamma_h)\Big]\Lambda.$$

Components of the left eigenvector of equation (6), corresponding to the zero eigenvalue are obtained as

$$v_1 = v_6 = 0, \quad v_3 = \frac{(\mu+\omega_c)w_2}{\omega_c}, \quad v_4 = \frac{1}{\beta_h\Lambda}\Big[\mu(\mu+\omega_c)(\mu+\delta_c+\gamma_c) - \beta_c\Lambda\omega_c\Big]v_2,$$

Table 2: Sensitivity indices of parameters of R_0.

Parameter	Description	Index
Λ	Recruitment rate	1.00000
β_c	Community contact rate	0.55316
β_h	Hospital infection rate	0.44680
ω_c	Transition rate, community exposed to infected	0.04806
ω_h	Transition rate, hospital exposed to infected	0.059126
γ_c	Community recovery rate	-0.06683
γ_h	Hospital recovery rate	-0.13258
μ	Natural death rate	-1.18193
δ_c	Community disease-induced death rate	-0.44556
δ_h	Hospital disease-induced death rate	-0.28028

$$\text{and} \quad v_5 = \frac{(\mu+\omega_h)}{\beta_h\omega_h\Lambda}\Big[\Lambda(\mu+\omega_c)(\mu+\delta_c+\gamma_c) - \beta_c\Lambda\omega_c\Big].$$

The bifurcation parameters p and q are defined by

$$p = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(E_0, \beta_c^*), \qquad q = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta_c}(E_0, \beta_c^*).$$

After substituting the second partial derivatives in the expressions for p and q, we obtain

$$\begin{aligned} p &= w_1(w_3 + w_5)(\beta_c v_2 + \beta_h v_4) \\ &= \frac{(\mu+\omega_c)(c_1 - c_2)v_2 w_2^2}{\beta_c^2\Lambda^3(\mu+\delta_c+\gamma_c)(\mu+\delta_h+\gamma_h)}, \end{aligned}$$

$$q = \frac{1}{\beta_c}(\mu+\omega_c)v_2 w_2 > 0,$$

where

$$c_1 = \Big[\beta_c\Lambda + \mu(\mu+\omega_c)(\mu+\delta_c+\gamma_c)\Big]\chi_1 + \beta_c\omega_c\Lambda\chi_2,$$

$$c_2 = \Big[\beta_c\Lambda + \mu(\mu+\omega_c)(\mu+\delta_c+\gamma_c)\Big]\chi_2 + \beta_c\omega_c\Lambda\chi_1.$$

Therefore, the bifurcation parameter q is positive. When $p > 0$, that is, $c_1 > c_2$ and $q > 0$, the centre manifold theory of Castillo-Chavez and Song [22] shows that backward bifurcation occurs; hence, it is possible to have co-existence of the disease-free equilibrium and endemic equilibrium. However, when $p < 0$, forward bifurcation occurs. □

3. Results

The results of the theoretical analysis and numerical simulation of the model are presented in this section.

3.1. Sensitivity analysis

Since the basic reproduction number R_0 helps predict the future course of disease transmission, sensitivity analysis is performed to understand which parameters involved in the model affect the value of R_0 relatively more. The normalized forward sensitivity index of a parameter v is defined by

$$\Omega_v^{R_0} = \frac{v}{R_0} \times \frac{\partial R_0}{\partial v}.$$

A negative sensitivity index shows that the parameter and R_0 are inversely proportional. A positive sensitivity index denotes that the value of R_0 increases with the value of the parameter concerned. Using the parameter values in Table 1, the sensitivity index is obtained for each parameter of R_0, as shown in Table 2.

From Table 2, we see that Λ, β_c, and β_h have the highest sensitivity indices, implying that they are the most sensitive parameters in this model. Furthermore, β_c has a higher sensitivity index than β_h, indicating that changes in how quickly the disease spreads in the community have a much greater impact on the outbreak's overall dynamics. This means that effectively controlling transmission in the community is essential for managing disease spread. The large sensitivity index of the recruitment rate Λ has wider implications for national and global health policy on immigrants, such as quarantine and vaccination against some common diseases.

3.2. Numerical simulation of the model

Considering the $SE_cI_cE_hI_hR$ model, we use the estimated parameter values in Table 1 to carry out the numerical simulation. The same parameter values are used to generate Figures 2–8, except where otherwise indicated.

4. Discussion

Figure 2 shows the time-course solution for an endemic situation in which the susceptible population decreases as more people become exposed or infected. The disease persists in both the community and the hospital, as $R_0 > 1$. In Figure 3, the solid red line shows community infection, which rises sharply above the hospital-associated infection, peaks around day 7, and then declines, indicating rapid initial spread. The dashed line represents hospital infection, which starts lower and increases more slowly, likely because of the lower infection rate in hospital settings, given the regulated environment. Together, the graphs illustrate the differences in how the disease spreads in the community versus hospitals. This is consistent with the sensitivity analysis, which shows a higher index for the community contact rate than for the hospital infection rate. In Figure 4, both infection rates are zero, implying that no new infections occur in either the community or hospital; hence, infections are generally low in the population compared with Figure 3.

In Figure 5, some community infections rise slightly at first, but since no new cases are entering, this number levels off over time. Hospital infections begin low and increase with time, but this rise eventually stabilizes. Without new community infections, both community and hospital infections stabilize and remain constant. In Figure 6, community infections rise during the first few days and then level off in the absence of hospital infections. However, hospital-associated infections remain low without new cases. Based on the results depicted in Figures 5 and 6, strategies such as isolation, vaccination, personal hygiene, routine surveillance for disease outbreaks, and prompt treatment when detected must be implemented to reduce community and hospital contact rates, depending on the nature of the disease. This is consistent with the findings of Milazzo *et al.* [7] and Omame and Abbas [10].

In Figure 7, as the community infection rate (β_c) increases, the number of infected individuals also rises, showing that faster transmission leads to more infections. Small increases in the infection rate can lead to significant spikes. This emphasizes the need to manage community transmission to reduce the infection's overall impact.

In Figure 8, as the hospital infection rate (β_h) increases, the number of hospital-acquired infections also increases. Even small increases can lead to more infections over time, suggesting the need for strong infection-control measures in hospitals to protect patients and health workers. These results have implications for both local and global health policies, such as quarantining and vaccinating immigrants to curb the spread of infections in communities and healthcare centres. However, inadequate real data on hospital and community infections are a limitation of this study.

5. Conclusion

A model for the transmission of infections in hospital and community settings is considered. Standard analysis showed the infection-free equilibrium to be locally and globally stable for $R_0 < 1$. The results from the normalized sensitivity analysis and numerical simulations showed that community transmission of infections is higher than hospital transmission. However, hospital-transmitted infections can aggravate transmission in the community, and vice versa; hence, strict observance of public hygiene practices such as hand washing, vaccination, use of face masks, and safe waste disposal, among others, is recommended to reduce infections in community and hospital settings. Future research should focus on optimal control and effectiveness analysis to assess the efficacy of control measures against community and hospital transmission of infections.

Data availability

No new data were generated for this study.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this manuscript.

Funding

This research was supported by funding from the Pastor E. A. Adeboye Professorial Chair Endowment in Mathematics at the University of Jos.

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