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Research article

The influence of sexual structure and dual incubation delays on spreading dynamics and optimal control of a gonorrhea model

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Abstract: An SIRS (Susceptible-Infectious-Recovered-Susceptible) model with sexual structure and dual incubation delays was proposed to characterize the effects of homosexual and heterosexual behaviors on the transmission dynamics and optimal control of gonorrhea. First, the nonnegativity and boundedness of solutions were obtained, and the basic reproduction number $\mathcal{R}_0$ was calculated as well. Second, local and global asymptotical stability of the disease-free equilibrium was established if $\mathcal{R}_0 < 1$. If $\mathcal{R}_0 > 1$ the disease was uniformly persistent, and there existed at least one endemic equilibrium. An optimal control strategy was derived based on sensitivity analysis and practical intervention policy. Finally, the theoretical findings were illustrated through numerical simulations, revealing that targeted management of male infected individuals can markedly diminish gonorrhea prevalence, while disregarding the incubation periods tended to substantially overestimate the epidemic scale.

Keywords: gonorrhea; incubation delay; sexual structure; uniform persistence; optimal control strategy

1. Introduction

Gonorrhea, a globally endemic sexually transmitted infection (STI), arises from Neisseria gonorrhoeae — a facultative intracellular Gram-negative diplococcus [1], with approximately 82 million annual cases reported by the World Health Organization (2023) [2]. This bacterium poses substantial public health challenges due to its high contagiousness [3, 4] (often asymptomatic [5, 6], affecting over 50% infected women [7, 8]), severe reproductive complications (e.g., pelvic inflammatory disease in females, leading to infertility [9]; epididymitis in males, though less frequent,

requiring clinical attention [10]), and evolving epidemiological trends. Notably, male infection rates increased by 27.9% from 2015 to 2020 [2], driven by the expanding core subpopulations of MSM (men who have sex with men) and bisexual individuals [11], while female infection rates declined by 4.1% [11, 12]. This gender disparity, rooted in anatomical and immunological differences [13, 14], underscores the critical need to model sexual structure (homosexual and heterosexual interaction patterns) and incubation period delays (differential latency in distinct transmission pathways) to explore the complex dynamics of gonorrhea transmission and targeted interventions.

Recently, many mathematical models have emerged as key for unpacking STI transmission dynamics (see [15–19]). Empirical evidence shows that STI transmission rates from males to females exceed those between females [16]; meanwhile, MSM face an infection risk 19.3 times greater than the heterosexual spread [17]. Significantly, bisexual MSM could accelerate STI spread by bridging high-risk and heterosexual networks [18]. As an example, McCluskey et al. [19] constructed a multigroup SIS (Susceptible-Infectious-Susceptible) model for gonorrhoea transmission among the Arya population in northern Kenya as follows:

$$\begin{cases} \dot{S}_{M1} = -\Gamma_{M1} S_{M1} - d_{M1} S_{M1} + \gamma_{M1} I_{M1}, \\ \dot{I}_{M1} = \Gamma_{M1} S_{M1} - (d_{M1} + \gamma_{M1}) I_{M1}, \\ \dot{S}_{S1} = -\Gamma_{S1} S_{S1} - d_{S1} S_{S1} + \gamma_{S1} I_{S1}, \\ \dot{I}_{S1} = \Gamma_{S1} S_{S1} - (d_{S1} + \gamma_{S1}) I_{S1}, \\ \dot{S}_{M2} = -\Gamma_{M2} S_{M2} - d_{M2} S_{M2} + \gamma_{M2} I_{M2}, \\ \dot{I}_{M2} = \Gamma_{M2} S_{M2} - (d_{M2} + \gamma_{M2}) I_{M2}, \\ \dot{S}_{S2} = -\Gamma_{S2} S_{S2} - d_{S2} S_{S2} + \gamma_{S2} I_{S2}, \\ \dot{I}_{S2} = \Gamma_{S2} S_{S2} - (d_{S2} + \gamma_{S2}) I_{S2}, \end{cases} \tag{1.1}$$

where the population was divided into four subgroups: married men ($M1$), married women ($M2$), single men ($S1$), and single women ($S2$), and $\Gamma_{ij} = c_{ij,S2}\beta_{S2,ij} I_{S2} + c_{ij,M2}\beta_{M2,ij} I_{M2}$ represents the strength of infection. They analyzed the sexual partner patterns on disease transmission, suggesting that gonorrhoea cannot be transmitted consistently without a core group or other unincorporated factors.

In addition, Castillo-Chavez et al. [20, 21] and Hsieh et al. [22, 23] further investigated some important STI epidemic models with cross-sexual transmission. Benjamin et al. [24] proposed an SIS/SAS (Susceptible-Asymptomatic-Susceptible) model for gonorrhea in high-risk MSM, evaluating safe behaviors' impact and deriving stability thresholds. Saldana et al. [25] integrated partner duration into an SIS model, showing treatment delays could induce backward bifurcation and defining prevalence thresholds via recovery periods. Bonyah et al. [26] established an alcohol-related gonorrhea epidemic model, identifying its epidemiological features and highlighting the need for integrated preventive-therapeutic strategies. Terefe et al. [27] incorporated the incubation period of gonorrhea into a drug-resistant model, demonstrating its central role in multi-strain spread. Meanwhile, interventions targeting pre-symptomatic stages for MSM are vital to curb epidemics [28], which could effectively confirm sexual structure and incubation delays as core drivers of gonorrhea dynamics.

Based on the aforementioned studies, we develop an epidemiological model for gonorrhea transmission that incorporates both homosexual and heterosexual transmission routes, under the

following considerations. First, since female-to-female transmission of gonorrhea has been shown to be significantly less prevalent compared to male-to-male transmission [29], we mainly focus specifically on male homosexual transmission pathways. Second, we explicitly integrate the gonorrhea latency delay, which typically spans two to ten days [30]. Therefore, we establish the following epidemic model of gonorrhea with MSM and heterosexual spreads, and incubation delays:

$$\begin{cases} \dot{S}_m(t) = \Lambda_m - \left[\beta_1 I_f(t) + \beta_2 I_m(t)\right] S_m(t) - \mu_m S_m(t) + \gamma_m R_m(t), \\ \dot{I}_m(t) = e^{-\mu_m \tau_1} \left[\beta_1 I_f(t-\tau_1) + \beta_2 I_m(t-\tau_1)\right] S_m(t-\tau_1) - (\kappa_m + \mu_m) I_m(t), \\ \dot{R}_m(t) = \kappa_m I_m(t) - (\gamma_m + \mu_m) R_m(t), \\ \dot{S}_f(t) = \Lambda_f - \beta_3 I_m(t) S_f(t) - \mu_f S_f(t) + \gamma_f R_f(t), \\ \dot{I}_f(t) = e^{-\mu_f \tau_2} \beta_3 I_m(t-\tau_2) S_f(t-\tau_2) - (\kappa_f + \mu_f) I_f(t), \\ \dot{R}_f(t) = \kappa_f I_f(t) - (\gamma_f + \mu_f) R_f(t), \end{cases} \tag{1.2}$$

where, $S_m, I_m, R_m, S_f, I_f, R_f$ denote the numbers of susceptible males, infected males, recovered males, susceptible females, infected females, and recovered females, respectively. Consistent with the sex-structured transmission dynamics of gonorrhea (male-to-male and male-female interactions), all parameters in model (1.2) are positive constants, with detailed descriptions provided in Table 1.

Table 1. Description of parameters in model (1.2).

Parameter	Description
τ_1, τ_2	Incubation periods of Neisseria gonorrhoeae in male and female hosts
$e^{-\mu_m \tau_1}$	Survival probability of males from $t-\tau_1$ to t
$e^{-\mu_f \tau_2}$	Survival probability of females from $t-\tau_2$ to t
Λ_m, Λ_f	Recruitment rates for males and females
μ_m, μ_f	Natural mortality rates for males and females
β_1	Transmission rate from infected females to susceptible males
β_2	Transmission rate from infected males to susceptible males
β_3	Transmission rate from infected males to susceptible females
κ_m, κ_f	Recovery rates for infected males and females
γ_m, γ_f	Immunity waning rates for recovered males and females

The paper is arranged in the following framework. In Section 2, the positivity and boundedness of the model solutions are analyzed. In Section 3, the basic reproduction number $\mathcal{R}_0$ undergoes derivation, alongside the establishment of criteria for the local and global asymptotical stability of the disease-free equilibrium E_0 and gonorrhea's uniform persistence. In Section 4, an optimal control strategy is proposed based on sensitivity analysis. In Section 5, the theoretical findings are illustrated through numerical simulations. The final section summarizes the key findings.

2. Basic properties

Denote $\tau = \max\{\tau_1, \tau_2\}$. We denote the relevant state space as the Banach space $C := C([-\tau, 0], \mathbb{R}^6)$, equipped with the norm

$$\|\psi\| = \sum_{j=1}^{6} \|\psi_j\|_\infty, \text{ where } \|\psi_j\|_\infty = \sup_{s\in[-\tau,0]} |\psi_j(s)|.$$

Its positive cone is given by $C^+ := \{\psi \in C : \psi(s) \in \mathbb{R}^6_+ \text{ for all } s \in [-\tau, 0]\}$.

The initial conditions of model (1.2) are specified by continuous functions belonging to the space C^+, defined as:

$$\begin{cases} S_m(s) = \psi_1(s),\ I_m(s) = \psi_2(s),\ R_m = \psi_3(s), \\ S_f(s) = \psi_4(s),\ I_f(s) = \psi_5(s),\ R_f(s) = \psi_6(s), \end{cases} \tag{2.1}$$

where $s \in [-\tau, 0]$, $\psi = (\psi_1(s), \psi_2(s), \psi_3(s), \psi_4(s), \psi_5(s), \psi_6(s)) \in C([-\tau, 0], \mathbb{R}^6_+)$, $\psi_j(0) > 0$, $j = 1, 2, \ldots, 6$.

Theorem 2.1. *Under the initial condition* (2.1)*, the solution* $\phi(t, \psi) \in C^+$ *to model* (1.2) *is nonnegative and ultimately bounded for all* $t \in [0, \infty)$.

Proof. For each $\psi \in C^+$

$$f(t,\psi) = \begin{pmatrix} \Lambda_m - [\beta_1\psi_5(0) + \beta_2\psi_2(0)]\,\psi_1(0) - \mu_m\psi_1(0) + \gamma_m\psi_3(0) \\ e^{-\mu_m\tau_1}\,[\beta_1\psi_5(-\tau_1) + \beta_2\psi_2(-\tau_1)]\,\psi_1(-\tau_1) - (\kappa_m + \mu_m)\psi_2(0) \\ \kappa_m\psi_2(0) - (\gamma_m + \mu_m)\psi_3(0) \\ \Lambda_f - \beta_3\psi_2(0)\psi_4(0) - \mu_f\psi_4(0) + \gamma_f\psi_6(0) \\ e^{-\mu_f\tau_2}\beta_3\psi_2(-\tau_2)\psi_4(-\tau_2) - (\kappa_f + \mu_f)\psi_5(0) \\ \kappa_f\psi_5(0) - (\gamma_f + \mu_f)\psi_6(0) \end{pmatrix}.$$

Given that $f(t, \psi)$ is continuous and satisfies the Lipschitz condition, model (1.2) has a unique solution $\phi(t, \psi)$ defined on its maximal interval $[0, \sigma_\psi)$. Let $f_j(t, \psi)$ denote the j-th component of $f(t, \psi)$ for $j \in 1, 2, \ldots, 6$. According to [31], the condition $\psi_j(0) = 0$ implies $f_j(t, \psi) \geq 0$, which ensures that $\phi_t(\psi)$ remains nonnegative.

Next, we demonstrate ultimate boundedness. Define $N_m(t) = S_m(t) + I_m(t) + R_m(t)$. Then

$$\begin{aligned} \dot{N}_m(t) &= \Lambda_m - (\beta_1 I_f(t) + \beta_2 I_m(t))S_m(t) - \mu_m S_m(t) + \gamma_m R_m(t) \\ &\quad + e^{-\mu_m\tau_1}(\beta_1 I_f(t-\tau_1) + \beta_2 I_m(t-\tau_1))S_m(t-\tau_1) - (\kappa_m + \mu_m)I_m(t) \\ &\quad + \kappa_m I_m(t) - (\gamma_m + \mu_m)R_m(t) \\ &= \Lambda_m - (\beta_1 I_f(t) + \beta_2 I_m(t))S_m(t) - \mu_m N_m(t) \\ &\quad + e^{-\mu_m\tau_1}(\beta_1 I_f(t-\tau_1) + \beta_2 I_m(t-\tau_1))S_m(t-\tau_1). \end{aligned}$$

Define

$$F_m(t) = \int_{t-\tau_1}^{t} e^{-\mu_m(t-s)}(\beta_1 I_f(s) + \beta_2 I_m(s))S_m(s)ds.$$

Then

$$\begin{aligned} \dot{F}_m(t) &= (\beta_1 I_f(t) + \beta_2 I_m(t))S_m(t) - e^{-\mu_m\tau_1}(\beta_1 I_f(t-\tau_1) + \beta_2 I_m(t-\tau_1))S_m(t-\tau_1) \\ &\quad - \mu_m \int_{t-\tau_1}^{t} e^{-\mu_m(t-s)}(\beta_1 I_f(s) + \beta_2 I_m(s))S_m(s)ds. \end{aligned}$$

Let $U_m(t) = N_m(t) + F_m(t)$. Then

$$\begin{aligned}\dot{U}_m(t) &= \Lambda_m - \mu_m N_m(t) - \mu_m F_m(t)\\ &= \Lambda_m - \mu_m U_m(t),\end{aligned}$$

which yields $\limsup_{t\to\infty} U_m(t) \le \frac{\Lambda_m}{\mu_m}$.

Similarly, define $N_f(t) = S_f(t)+I_f(t)+R_f(t)$ and $F_f(t) = \int_{t-\tau_2}^{t} e^{-\mu_f(t-s)}\beta_3 I_m(s)S_f(s)ds$. Then we have $\limsup_{t\to\infty} U_f(t) \le \frac{\Lambda_f}{\mu_f}$.

Since $N(t) = S_m(t)+I_m(t)+R_m(t)+S_f(t)+I_f(t)+R_f(t) = N_m(t)+N_f(t) \le U_m(t)+U_f(t)$, $N(t)$ remains bounded on $[0,\sigma_\psi)$. Consequently, all components of $\phi(t,\psi)$ are bounded on $[0,\sigma_\psi)$. By the extension theorem for delay differential equations [32], $\sigma_\psi = \infty$. Thus, the solution $\phi(t,\psi)$ to model (1.2) is nonnegative and ultimately bounded for all $t \in [0,\infty)$.

Define the following set

$$\Gamma = \left\{ \psi = (\psi_1,\dots,\psi_6) \in C^+ : \begin{array}{l} \psi_1(0)+\psi_2(0)+\psi_3(0)+\int_{-\tau_1}^{0} e^{\mu_m s}[\beta_1\psi_5(s)+\beta_2\psi_2(s)]\psi_1(s)\,ds \le \frac{\Lambda_m}{\mu_m};\\ \psi_4(0)+\psi_5(0)+\psi_6(0)+\int_{-\tau_2}^{0} e^{\mu_f\tau_2}\beta_3\psi_2(s)\psi_4(s)\,ds \le \frac{\Lambda_f}{\mu_f} \end{array} \right\}, \quad (2.2)$$

which is positively invariant and attractive in regard to model (1.2).

3. Stability of equilibria

In this section, based on the basic reproduction number $\mathcal{R}_0$, we discuss the existence and stability of equilibria of model (1.2).

The model (1.2) possesses a unique disease-free equilibrium (DFE) $E_0 = (S_{m0}, 0, 0, S_{f0}, 0, 0)$, where $S_{m0} = \frac{\Lambda_m}{\mu_m}, S_{f0} = \frac{\Lambda_f}{\mu_f}$. Using the next generation matrix method for functional differential equations [33], $\mathcal{R}_0$ of model (1.2) is defined as:

$$\mathcal{R}_0 = \frac{e^{-\mu_m\tau_1}\beta_2\Lambda_m}{(\kappa_m+\mu_m)\mu_m} + \frac{e^{-\mu_m\tau_1}e^{-\mu_f\tau_2}\beta_1\beta_3\Lambda_m\Lambda_f}{(\kappa_m+\mu_m)(\kappa_f+\mu_f)\mu_m\mu_f}.$$

Theorem 3.1. *If $\mathcal{R}_0 < 1$, the DFE E_0 of model* (1.2) *is locally asymptotically stable (LAS).*

Proof. For model (1.2), the Jacobi matrix corresponding to E_0 is presented as follows

$$\mathcal{J}(E_0) = \begin{bmatrix} -\mu_m & -\beta_2 S_{m0} & \gamma_m & 0 & -\beta_1 S_{m0} & 0\\ 0 & e^{-(\mu_m+\lambda)\tau_1}\beta_2 S_{m0} - a_1 & 0 & 0 & e^{-(\mu_m+\lambda)\tau_1}\beta_1 S_{m0} & 0\\ 0 & \kappa_m & -b_1 & 0 & 0 & 0\\ 0 & -\beta_3 S_{f0} & 0 & -\mu_f & 0 & \gamma_f\\ 0 & e^{-(\mu_f+\lambda)\tau_2}\beta_3 S_{f0} & 0 & 0 & -a_2 & 0\\ 0 & 0 & 0 & 0 & \kappa_f & -b_2 \end{bmatrix},$$

where $a_1 = \kappa_m + \mu_m$, $a_2 = \kappa_f + \mu_f$, $b_1 = \gamma_m + \mu_m$, $b_2 = \gamma_f + \mu_f$. Hence the characteristic equation is

$$0 = \begin{vmatrix} \lambda + \mu_m & \beta_2 S_{m0} & -\gamma_m & 0 & \beta_1 S_{m0} & 0 \\ 0 & \lambda - e^{-(\mu_m+\lambda)\tau_1}\beta_2 S_{m0} + a_1 & 0 & 0 & -e^{-(\mu_m+\lambda)\tau_1}\beta_1 S_{m0} & 0 \\ 0 & -\kappa_m & \lambda + b_1 & 0 & 0 & 0 \\ 0 & \beta_3 S_{f0} & 0 & \lambda + \mu_f & 0 & -\gamma_f \\ 0 & -e^{-(\mu_f+\lambda)\tau_2}\beta_3 S_{f0} & 0 & 0 & \lambda + a_2 & 0 \\ 0 & 0 & 0 & 0 & -\kappa_f & \lambda + b_2 \end{vmatrix}.$$

Clearly, the characteristic equation (1.2) has characteristic roots $\lambda_1 = -\mu_m, \lambda_2 = -b_1, \lambda_3 = -\mu_f, \lambda_4 = -b_2$, and the other eigenvalues are determined by the following transcendental equation.

$$(\lambda + a_1)(\lambda + a_2) - e^{-(\mu_m+\lambda)\tau_1}\beta_2 S_{m0}(\lambda + a_2) - e^{-(\mu_m+\lambda)\tau_1}e^{-(\mu_f+\lambda)\tau_2}\beta_1 S_{m0}\beta_3 S_{f0} = 0. \tag{3.1}$$

When $\tau_1 = \tau_2 = 0$, we have $\mathcal{R}_0 = \frac{\beta_2\Lambda_m}{a_1\mu_m} + \frac{\beta_1\beta_3\Lambda_m\Lambda_f}{a_1a_2\mu_m\mu_f}$. Thus, Eq (3.1) can be written as follows:

$$\lambda^2 + D_1\lambda + D_2 = 0, \tag{3.2}$$

where

$$\begin{aligned} D_1 &= a_1 + a_2 - \beta_2 S_{m0} = a_1(1 - \frac{\beta_2 S_{m0}}{a_1}) + a_2 > 0, \\ D_2 &= a_1a_2 - a_2\beta_2 S_{m0} - \beta_1\beta_3 S_{m0}S_{f0} = a_1a_2(1 - \mathcal{R}_0). \end{aligned}$$

When $\mathcal{R}_0 < 1$, $D_2 > 0$ and $D_1D_2 > 0$. It follows from the Routh–Hurwitz criterion that every root of (3.2) has a negative real part.

Thus, when $\tau_1 = \tau_2 = 0$, the DFE E_0 of model (1.2) is LAS.

Whenever $\tau_1 > 0$ and $\tau_2 > 0$, the characteristic rootsλ_5, λ_6 conform to the equation given below

$$(\lambda + a_1)(\lambda + a_2) = e^{-(\mu_m+\lambda)\tau_1}e^{-(\mu_f+\lambda)\tau_2}\beta_1 S_{m0}\beta_3 S_{f0} + e^{-(\mu_m+\lambda)\tau_1}\beta_2 S_{m0}(\lambda + a_2).$$

Define $\lambda^* = x^* + y^*i$. On the assumption that $x^* \geq 0$, it follows that $|\lambda + a_1| \geq x^* + a_1 \geq a_1$. Then

$$\begin{aligned} 1 &= \left| \frac{e^{-(\mu_m+\lambda^*)\tau_1}\beta_2 S_{m0}}{(\lambda^* + a_1)} + \frac{e^{-(\mu_m+\lambda^*)\tau_1}e^{-(\mu_f+\lambda^*)\tau_2}\beta_1 S_{m0}\beta_3 S_{f0}}{(\lambda^* + a_1)(\lambda^* + a_2)} \right| \\ &\leq \frac{e^{-\mu_m\tau_1}\beta_2 S_{m0}}{a_1} + \frac{e^{-\mu_m\tau_1}e^{-\mu_f\tau_2}\beta_1 S_{m0}\beta_3 S_{f0}}{a_1a_2} \\ &= \mathcal{R}_0, \end{aligned} \tag{3.3}$$

which leads to a contradiction, therefore λ_5, λ_6 has negative real part.

Thus, when $\tau_1 > 0, \tau_2 > 0$, the DFE E_0 of model (1.2) is LAS.

When $\tau_1 > 0, \tau_2 = 0$ and $\tau_1 = 0, \tau_2 > 0$, the proofs are the same as the above discussion, so we omit them here.

Thus, if $\mathcal{R}_0 < 1$, the DFE E_0 of model (1.2) is LAS.

Theorem 3.2. *If $\mathcal{R}_0 < 1$, the DFE E_0 of model* (1.2) *is globally asymptotically stable (GAS).*

Proof. By Theorem 3.1, it suffices to show that E_0 is globally attractive in Γ. Let $(S_m(t), I_m(t), R_m(t), S_f(t), I_f(t), R_f(t))$ be a solution with initial conditions (2.1). Then, $S_m(t) \leq \frac{\Lambda_m}{\mu_m} = S_{m0}$ and $S_f(t) \leq \frac{\Lambda_f}{\mu_f} = S_{f0}$. It follows that

$$\begin{cases} \dot{I}_m(t) \leq e^{-\mu_m \tau_1}(\beta_1 I_f(t-\tau_1) + \beta_2 I_m(t-\tau_1))S_{m0} - (\kappa_m + \mu_m)I_m(t), \\ \dot{I}_f(t) \leq e^{-\mu_f \tau_2}\beta_3 I_m(t-\tau_2)S_{f0} - (\kappa_f + \mu_f)I_f(t). \end{cases}$$

Consider the following comparison model:

$$\begin{cases} \dot{i}_1(t) = e^{-\mu_m \tau_1}(\beta_1 i_2(t-\tau_1) + \beta_2 i_1(t-\tau_1))S_{m0} - (\kappa_m + \mu_m)i_1(t), \\ \dot{i}_2(t) = e^{-\mu_f \tau_2}\beta_3 i_1(t-\tau_2)S_{f0} - (\kappa_f + \mu_f)i_2(t). \end{cases}$$

We have shown in the proof of Theorem 3.1 that all the eigenvalues of the characteristic equation of the above system have negative real parts. Thus,

$$(i_1(t), i_2(t)) \to (0,0), \text{when } t \to \infty.$$

By the comparison principle, $\lim\limits_{t\to\infty} I_m(t) = 0$ and $\lim\limits_{t\to\infty} I_f(t) = 0$.

According to the equation $\dot{R}_m(t) = \kappa_m I_m(t) - (\gamma_m + \mu_m)R_m(t)$, we can obtain $\lim\limits_{t\to\infty} R_m(t) = 0$. Similarly, we further have $\lim\limits_{t\to\infty} R_f(t) = 0$. Therefore, we get $\phi(t,\psi) \to (S_{m0}, 0, 0, S_{f0}, 0, 0)$. This completes the proof.

Theorem 3.3. *If $\mathcal{R}_0 > 1$, a positive constant $k > 0$ exists such that every solution of model* (1.2) *subject to the initial conditions* (2.1)*, with $I_m(0) + I_f(0) > 0$, fulfills*

$$\min\left\{\liminf_{t\to\infty} I_m(t), \liminf_{t\to\infty} I_f(t)\right\} > k,$$

i.e., the disease in model (1.2) *is uniformly persistent.*

Proof. Define

$$X = \{(S_m, I_m, R_m, S_f, I_f, R_f) \in \Gamma : I_m > 0,\ I_f > 0\}, \partial X = \{(S_m, I_m, R_m, S_f, I_f, R_f) \in \Gamma : I_m = 0 \text{ or } I_f = 0\}.$$
$$M_\partial = \{\psi \in \Gamma : \phi(t,\psi) \in \partial X \text{ for all } t \geq 0\},\ M_0 = \{E_0\}.$$

First, we prove that $M_\partial = M_0$. Clearly, $M_0 \subseteq M_\partial$. Now we show $M_\partial \subseteq M_0$. If not, there exist $x_0 \in M_\partial$, and $t_1 > 0$ such that $I_m(t_1) > 0$ and $I_f(t_1) > 0$. Notice that

$$\begin{cases} \dot{I}_m(t) \geq -(\kappa_m + \mu_m)I_m(t), \\ \dot{I}_f(t) \geq -(\kappa_f + \mu_f)I_f(t). \end{cases}$$

Given the existence of a constant $t_1 > 0$ satisfying$I_m(t_1) > 0$ and $I_f(t_1) > 0$, it holds that $I_m(t) > 0$ and $I_f(t) > 0$ for all $t > t_1$. Consequently, $\phi(t,\psi) \notin \partial X$, which yields a contradiction. Therefore $M_\partial \subseteq M_0$. The set M_0 is isolated and acyclic. Based on [34], it suffices to demonstrate that $W^s(E_0) \cap X = \emptyset$. Assume that there exists a solution $\psi_t(x_0) \in X$ for which $\lim\limits_{t\to\infty} \psi_t(x_0) \in M_0$, when $t \geq 0$. In other words, given any arbitrarily small constant $\varepsilon > 0$, a constant $t_2 > 0$ exists such that for all $t \geq t_2$, we have

$$\begin{cases} S_{m0} - \varepsilon < S_m(t) < S_{m0} + \varepsilon,\ 0 < I_m(t) \leq \varepsilon,\ 0 < R_m(t) \leq \varepsilon, \\ S_{f0} - \varepsilon < S_f(t) < S_{f0} + \varepsilon,\ 0 < I_f(t) \leq \varepsilon, 0 < R_f(t) \leq \varepsilon. \end{cases}$$

For large enough $t > t_2$, we have

$$\begin{cases} \dot{I}_m(t) \geq e^{-\mu_m \tau_1}(\beta_1 I_f(t-\tau_1) + \beta_2 I_m(t-\tau_1))(S_{m0} - \varepsilon) - (\kappa_m + \mu_m) I_m(t), \\ \dot{I}_f(t) \geq e^{-\mu_f \tau_2}\beta_3 I_m(t-\tau_2)(S_{f0} - \varepsilon) - (\kappa_f + \mu_f) I_f(t). \end{cases}$$

Consider the following comparison system

$$\begin{cases} \dot{I}_{m1}(t) = e^{-\mu_m \tau_1}(\beta_1 I_{f1}(t-\tau_1) + \beta_2 I_{m1}(t-\tau_1))(S_{m0} - \varepsilon) - (\kappa_m + \mu_m) I_{m1}(t), \\ \dot{I}_{f1}(t) = e^{-\mu_f \tau_2}\beta_3 I_{m1}(t-\tau_2)(S_{f0} - \varepsilon) - (\kappa_f + \mu_f) I_{f1}(t). \end{cases} \tag{3.4}$$

The characteristic equation of system (3.4) is the transcendental equation

$$\left(\lambda + a_1 - e^{-(\mu_m+\lambda)\tau_1}\beta_2(S_{m0} - \varepsilon)\right)(\lambda + a_2) - e^{-(\mu_m+\lambda)\tau_1}e^{-(\mu_f+\lambda)\tau_2}\beta_1\beta_3(S_{m0} - \varepsilon)(S_{f0} - \varepsilon) = 0.$$

When $\mathcal{R}_0 > 1$, for sufficiently small $\varepsilon > 0$, this characteristic equation has a positive real root $\lambda_\varepsilon > 0$ (by continuity of roots and the fact that at $\varepsilon = 0$, the equation has a positive root due to $\mathcal{R}_0 > 1$). Moreover, the corresponding eigenvector for the linearized system can be chosen positive. This implies that the comparison system (3.4) admits an exponentially growing solution, i.e., $\lim_{t\to\infty}(I_{m1}(t), I_{f1}(t)) = (\infty, \infty)$. By the comparison principle for delay differential equations, the original solutions satisfy $\lim_{t\to\infty}(I_m(t), I_f(t)) = (\infty, \infty)$, which yields a contradiction. Therefore, $W^s(E_0) \cap X = \emptyset$, which means the uniform persistence of both $I_m(t)$ and $I_f(t)$.

According to the first equation associated with model (1.2), we have

$$\begin{aligned} \dot{S}_m(t) &= \Lambda_m - (\beta_1 I_f(t) + \beta_2 I_m(t))S_m(t) - \mu_m S_m(t) + \gamma_m R_m(t) \\ &\geq \Lambda_m - (\beta_1 I_f(t) + \beta_2 I_m(t) + \mu_m)S_m(t) \\ &\geq \Lambda_m - (\max\{\beta_1, \beta_2\}\frac{\Lambda_m}{\mu_m} + \mu_m)S_m(t). \end{aligned} \tag{3.5}$$

Applying the comparison principle yields

$$\liminf_{t\to\infty} S_m(t) \geq \frac{\Lambda_m}{\max\{\beta_1, \beta_2\}\frac{\Lambda_m}{\mu_m} + \mu_m}.$$

Similarly,

$$\liminf_{t\to\infty} S_f(t) \geq \frac{\Lambda_f}{\beta_3\frac{\Lambda_f}{\mu_f} + \mu_f}.$$

Thus, we obtain the uniform persistence of both $S_m(t)$ and $S_f(t)$.

According to the third equation associated with model (1.2), we have

$$\dot{R}_m(t) = \kappa_m I_m(t) - (\gamma_m + \mu_m)R_m(t).$$

Since $I_m(t)$ is uniformly persistent, there exist $T > 0$ and $k > 0$ such that $I_m(t) \geq k$ for all $t \geq T$. Then for $t \geq T$, and it follows that

$$\dot{R}_m(t) \geq \kappa_m k - (\gamma_m + \mu_m)R_m(t).$$

Thus, $\liminf_{t\to\infty} R_m(t) \geq \frac{\kappa_m k}{\gamma_m + \mu_m} > 0$, which implies the uniform persistence of $R_m(t)$. In a similar vein, the uniform persistence of $R_f(t)$ is achievable. Theorem 2.1 manifests that $\psi_t(x_0)$ is point dissipative. Drawing on Theorem 4.6 in [35], model (1.2) attains uniform persistence with respect to $(X, \partial X)$.

Corollary 3.4. *If $\mathcal{R}_0 > 1$, model (1.2) exhibits a unique endemic equilibrium (EE).*

Proof. Based on Theorems 3.3 and 2.4 in [36], there exists at least one EE. Now we show the uniqueness of the EE. An EE $E^* = (S_m^*, I_m^*, R_m^*, S_f^*, I_f^*, R_f^*)$ satisfies

$$\begin{cases} \Lambda_m - (\beta_1 I_f^* + \beta_2 I_m^*) S_m^* - \mu_m S_m^* + \gamma_m R_m^* = 0, \\ e^{-\mu_m \tau_1}(\beta_1 I_f^* + \beta_2 I_m^*) S_m^* - (\kappa_m + \mu_m) I_m^* = 0, \\ \kappa_m I_m^* - (\gamma_m + \mu_m) R_m^* = 0, \\ \Lambda_f - \beta_3 I_m^* S_f^* - \mu_f S_f^* + \gamma_f R_f^* = 0, \\ e^{-\mu_f \tau_2} \beta_3 I_m^* S_f^* - (\kappa_f + \mu_f) I_f^* = 0, \\ \kappa_f I_f^* - (\gamma_f + \mu_f) R_f^* = 0. \end{cases} \tag{3.6}$$

A direct computation gives

$$S_m^* = \frac{(\kappa_m + \mu_m) I_m^*}{e^{-\mu_m \tau_1}(\beta_1 I_f^* + \beta_2 I_m^*)}, \; R_m^* = \frac{\kappa_m I_m^*}{(\mu_m + \gamma_m)}, \; S_f^* = \frac{(\kappa_f + \mu_f) I_f^*}{e^{-\mu_f \tau_2} \beta_3 I_m^*},$$

$$I_f^* = \frac{e^{-\mu_f \tau_2} \Lambda_f (\gamma_f + \mu_f) \beta_3 I_m^*}{(\kappa_f + \mu_f + \gamma_f)\mu_f e^{-\mu_f \tau_2} \beta_3 I_m^* + \mu_f (\mu_f + \gamma_f)(\kappa_f + \mu_f)}, \; R_f^* = \frac{\kappa_f I_f^*}{(\mu_f + \gamma_f)},$$

where I_m^* satisfies

$$E (I_m^*)^2 + F I_m^* + G = 0, \tag{3.7}$$

with

$$\begin{aligned} E &= (\gamma_m \kappa_m e^{-\mu_m \tau_1} - a_1 b_1)(a_2 b_2 - \gamma_f \kappa_f e^{-\mu_f \tau_2}) \beta_2 \beta_3 < 0, \\ F &= (a_2 b_2 - e^{-\mu_f \tau_2} \gamma_f \kappa_f) \beta_3 b_1 (e^{-\mu_m \tau_1} \Lambda_m \beta_2 - \mu_m a_1) \\ &\quad + (e^{-\mu_m \tau_1} \kappa_m \gamma_m - a_1 b_1) b_2 (e^{-\mu_f \tau_2} \Lambda_f \beta_1 \beta_3 + \beta_2 \mu_f a_2), \\ G &= \Lambda_m \Lambda_f \beta_1 \beta_3 b_1 b_2 e^{-\mu_m \tau_1} e^{-\mu_f \tau_2} + (\Lambda_m \beta_2 e^{-\mu_m \tau_1} - \mu_m a_1) \mu_f a_2 b_1 b_2 \\ &= \left(\frac{e^{-\mu_m \tau_1} \beta_2 \Lambda_m}{(\kappa_m + \mu_m)\mu_m} + \frac{e^{-\mu_m \tau_1} e^{-\mu_f \tau_2} \beta_1 \beta_3 \Lambda_m \Lambda_f}{(\kappa_m + \mu_m)(\kappa_f + \mu_f)\mu_m \mu_f} - 1 \right) \mu_m \mu_f a_1 a_2 b_1 b_2 \\ &= (\mathcal{R}_0 - 1) \mu_m \mu_f a_1 a_2 b_1 b_2. \end{aligned}$$

Since $\mathcal{R}_0 > 1$, we have $G > 0$. By Vieta's theorem, (3.7) has a positive root and a negative root. This shows that (1.2) has a unique EE.

4. Sensitivity analysis and optimal control

This section first conducts a global sensitivity analysis to quantify the influence of parameters on model (1.2). Subsequently, based on the insights derived from this analysis, an optimal control model is formulated and analyzed.

As demonstrated in Figure 1, the parameters μ_m, μ_f , κ_m, and κ_f exhibit negative correlations with $\mathcal{R}_0$. Conversely, Λ_m, Λ_f, and the transmission rates β_1, β_2, and β_3 show positive correlations with $\mathcal{R}_0$, which could highlight two critical intervention pathways:

- The strong positive correlation of $\mathcal{R}_0$ with β_1,β_2, and β_3 suggests that targeted measures effectively decreasing sexual transmission rates, such as promoting condom use, enhancing public awareness campaigns about gonorrhea prevention, and discouraging high-risk sexual behaviors, could significantly reduce infection prevalence.

- The negative correlation between κ_m, κ_f, and $\mathcal{R}_0$ implies that improving access to rapid diagnostic tools and effective antibiotic treatments (while considering antimicrobial resistance trends) would greatly suppress disease propagation.

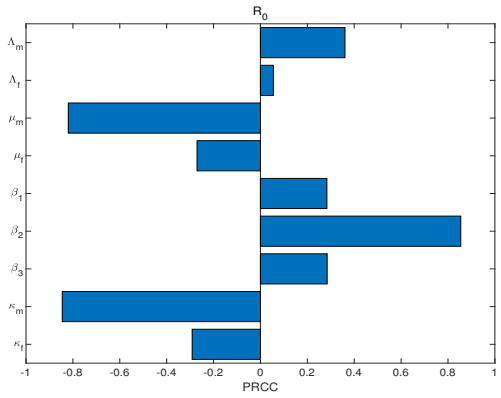


Figure 1. Sensitivity indicators of $\mathcal{R}_0$ for different parameters.

Merging sensitivity analysis outcomes with real-world countermeasures, we first propose the following four targeted control measures:
$\eta_1(t)$: Prevention enhancement and awareness campaigns targeting the male population (e.g., condom promotion, sexual health education);
$\eta_2(t)$: Therapeutic intervention strategy for infected males (e.g., rapid antibiotic treatment, contact tracing);
$\eta_3(t)$: Prevention enhancement and awareness campaigns targeting the female population (e.g., sexual health literacy programs, peer education networks);
$\eta_4(t)$: Therapeutic intervention strategy for infected females (e.g., test-and-treat protocols, resistance monitoring).

Further, we introduce the following control model.

$$\begin{cases} \dot{S}_m(t) = \Lambda_m - ((1-\eta_3(t))\beta_1 I_f(t) + (1-\eta_1(t))\beta_2 I_m(t))S_m(t) - \mu_m S_m(t) + \gamma_m R_m(t), \\ \dot{I}_m(t) = e^{-\mu_m \tau_1}((1-\eta_3(t))\beta_1 I_f(t-\tau_1) + (1-\eta_1(t))\beta_2 I_m(t-\tau_1))S_m(t-\tau_1) \\ \qquad\quad - (\kappa_m + \mu_m + \eta_2(t))I_m(t), \\ \dot{R}_m(t) = (\kappa_m + \eta_2(t))I_m(t) - (\gamma_m + \mu_m)R_m(t), \\ \dot{S}_f(t) = \Lambda_f - (1-\eta_1(t))\beta_3 I_m(t)S_f(t) - \mu_f S_f(t) + \gamma_f R_f(t), \\ \dot{I}_f(t) = e^{-\mu_f \tau_2}(1-\eta_1(t))\beta_3 I_m(t-\tau_2)S_f(t-\tau_2) - (\kappa_f + \mu_f + \eta_4(t))I_f(t), \\ \dot{R}_f(t) = (\kappa_f + \eta_4(t))I_f(t) - (\gamma_f + \mu_f)R_f(t). \end{cases} \tag{4.1}$$

Taking into account the constraints imposed by medical technology and cost control imperatives, it is assumed that the control function has bounds and conforms to the following set of admissible controls:

$$\Omega = \{\eta(t) = (\eta_1(t), \eta_2(t), \eta_3(t), \eta_4(t)) \in L^\infty[0, t_f],\ 0 \le \eta_j(t) \le \eta_j^{\max}\},$$

in which t_f is the prespecified final time. Let $\eta_j^{\max}$ denote the maximum attainable value of $\eta_j(t)$ for $j = 1, 2, 3, 4$. The primary objective of this study is to identify an optimal control $\eta^*(t)$ that minimizes

the objective function $J[\eta(t)]$, that is

$$J[\eta^*(t)] = \min_{\eta(t)\in\Omega} J[\eta(t)] = \min_{\eta(t)\in\Omega} \int_0^{t_f} [I_m(t) + I_f(t) + \frac{1}{2}\sum_{j=1}^{4} A_j\eta_j^2(t)]dt. \tag{4.2}$$

Here A_j signifies the cost weight assigned to $\eta_j(t)$. Owing to the nonlinear nature of cost, we employ a quadratic objective function to quantify the control cost, which is given by $\frac{1}{2}\sum_{j=1}^{4} A_j\eta_j^2(t)$. The objective of the optimization problem is to minimize the objective function, which takes into account both the quantities of $I_m(t)$ and $I_f(t)$ and the cost corresponding to the control variables $\eta_j(t)$.

For the determination of the optimal control, we first establish a Lagrangian function corresponding to the optimal control framework (4.1) and (4.2),

$$L(I_m(t), I_f(t), \eta(t)) = I_m(t) + I_f(t) + \frac{1}{2}\left(A_1\eta_1^2(t) + A_2\eta_2^2(t) + A_3\eta_3^2(t) + A_4\eta_4^2(t)\right). \tag{4.3}$$

To apply Pontryagin's minimum principle with delay [37], the Hamiltonian function is formally established as

$$H(t) = H(I_m(t),\, I_f(t),\, \eta(t),\, \lambda_j(t)) = I_m(t) + I_f(t) + \frac{1}{2}\sum_{j=1}^{4} A_j\eta_j^2(t) + \sum_{j=1}^{6} \lambda_j g_j,$$

where $\lambda_j\,(j = 1, 2, 3, 4, 5, 6)$ denote the adjoint variables to be properly specified, and g_j corresponds to the righthand side of the $j-$th state variable in model (4.1).

We have the following result about the optimal control strategy.

Theorem 4.1. *For any $\eta(t) = (\eta_1(t), \eta_2(t), \eta_3(t), \eta_4(t)) \in \Omega$, given that the cost functional $J[\eta(t)]$ is subject to the dynamical constraints specified in model* (4.1)*, an optimal control $\eta^*(t) \in \Omega$ exists that minimizes the objective functional, i.e.,*

$$J[\eta^*(t) = (\eta_1^*(t),\, \eta_2^*(t),\, \eta_3^*(t),\, \eta_4^*(t)] = \min_{\eta(t)\in\Omega} J[\eta(t)]. \tag{4.4}$$

Proof. The existence of an optimal control is established by verifying the following five conditions required by the Filippov–Cesari Existence Theorem [38] as outlined in [39].

i. The set of admissible control-state pairs is nonempty.

ii. The control set Ω is convex, closed, and bounded in L^∞, hence compact in an appropriate topology.

iii. The righthand side g_j of system (4.1) satisfies a linear growth condition in the state and control variables.

iv. $L(I_m(t), I_f(t), \eta(t))$ is convex on Ω.

v. There exist constants $\omega_1 > 0, \omega_2 > 0$, and $\rho > 1$ such that $L(I_m(t), I_f(t), \eta(t))$ satisfies:

$$L(I_m(t), I_f(t), \eta(t)) \geq \omega_1 + \omega_2\left(|\eta_1(t)|^2 + |\eta_2(t)|^2 + |\eta_3(t)|^2 + |\eta_4(t)|^2\right)^{\frac{\rho}{2}}.$$

Therefore, according to Fleming and Rishel [39], the existence of optimal control can be directly obtained.

Theorem 4.2. *The model* (4.1) *has an optimal control* $\eta^* = (\eta_1^*, \eta_2^*, \eta_3^*, \eta_4^*)$*, where*

$$\begin{aligned}
\eta_1^* &= \min\left\{\eta_1^{\max}, \max\left\{\frac{\beta_2 I_m^*(t)S_m^*(t)\left[e^{-\mu_m\tau_1}\lambda_2(t)-\lambda_1(t)\right]+\beta_3 I_m^*(t)S_f^*(t)\left[e^{-\mu_f\tau_2}\lambda_5(t)-\lambda_4(t)\right]}{A_1}, 0\right\}\right\},\\
\eta_2^* &= \min\left\{\eta_2^{\max}, \max\left\{\frac{I_m^*(t)(\lambda_2(t)-\lambda_3(t))}{A_2}, 0\right\}\right\},\\
\eta_3^* &= \min\left\{\eta_3^{\max}, \max\left\{\frac{\beta_1 I_f^*(t)S_m^*(t)\left[e^{-\mu_m\tau_1}\lambda_2(t)-\lambda_1(t)\right]}{A_3}, 0\right\}\right\},\\
\eta_4^* &= \min\left\{\eta_4^{\max}, \max\left\{\frac{I_f^*(t)(\lambda_5(t)-\lambda_6(t))}{A_4}, 0\right\}\right\}.
\end{aligned} \tag{4.5}$$

Proof.

$$\begin{aligned}
\frac{d\lambda_1(t)}{dt} &= -\frac{\partial}{\partial S_m}\left\{I_m(t)+I_f(t)+\frac{1}{2}\sum_{j=1}^{4}A_j\eta_j^2(t)+\sum_{j=1}^{6}\lambda_j g_j\right\}\\
&= -\left\{-\lambda_1(t)\left((1-\eta_3^*(t))\beta_1 I_f^*(t)+(1-\eta_1^*(t))\beta_2 I_m^*(t)+\mu_m\right)\right\}\\
&\quad -\left\{-\chi_{[0,t_f-\tau_1]}(t)\lambda_2(t+\tau_1)e^{-\mu_m\tau_1}\left[(1-\eta_3^*)(t+\tau_1)\beta_1 I_f^*(t)+(1-\eta_1^*(t+\tau_1))\beta_2 I_m^*(t)\right]\right\}\\
&= \lambda_1(t)\left((1-\eta_3^*(t))\beta_1 I_f^*(t)+(1-\eta_1^*(t))\beta_2 I_m^*(t)+\mu_m\right)\\
&\quad -\chi_{[0,t_f-\tau_1]}\lambda_2(t+\tau_1)e^{-\mu_m\tau_1}\left[(1-\eta_3^*(t+\tau_1))\beta_1 I_f^*(t)+(1-\eta_1^*(t+\tau_1))\beta_2 I_m^*(t)\right].
\end{aligned}$$

Similarly, it is derived that

$$\begin{aligned}
\frac{d\lambda_2(t)}{dt} &= \lambda_1(t)\beta_2(1-\eta_1^*(t))S_m^*(t)-\chi_{[0,t_f-\tau_1]}e^{-\mu_m\tau_1}(1-\eta_1^*(t+\tau_1))\beta_2 S_m^*(t)\lambda_2(t+\tau_1)+\lambda_2(t)(\kappa_m\\
&\quad +\mu_m+\eta_2^*(t))-(\kappa_m+\eta_2^*(t))\lambda_3(t)-\chi_{[0,t_f-\tau_2]}e^{-\mu_f\tau_2}(1-\eta_1^*(t+\tau_2))\beta_3 S_f^*(t)\lambda_5(t+\tau_2)-1,\\
\frac{d\lambda_3(t)}{dt} &= \left[\lambda_3(t)-\lambda_1(t)\right]\gamma_m+\lambda_3(t)\mu_m,\\
\frac{d\lambda_4(t)}{dt} &= \left((1-\eta_1^*(t))\beta_3 I_m^*(t)+\mu_f\right)\lambda_4(t)-\chi_{[0,t_f-\tau_2]}e^{-\mu_f\tau_2}(1-\eta_1^*(t+\tau_2))\beta_3 I_m^*(t)\lambda_5(t+\tau_2),\\
\frac{d\lambda_5(t)}{dt} &= (1-\eta_3^*(t))\beta_1\lambda_1(t)S_m^*(t)-\chi_{[0,t_f-\tau_1]}e^{-\mu_m\tau_1}(1-\eta_3^*(t+\tau_1))\beta_1 S_m^*(t)\lambda_2(t+\tau_1)\\
&\quad +(\kappa_f+\mu_f+\eta_4^*(t))\lambda_5(t)-(\kappa_f+\eta_4^*(t))\lambda_6(t)-1,\\
\frac{d\lambda_6(t)}{dt} &= \left[\lambda_6(t)-\lambda_4(t)\right]\gamma_f+\lambda_6(t)\mu_f,
\end{aligned}$$

where

$$\chi_{[0,t_f-\tau_1]}(t)=\begin{cases}1, & t\in[0,t_f-\tau_1],\\ 0, & \text{otherwise},\end{cases},\quad \chi_{[0,t_f-\tau_2]}(t)=\begin{cases}1, & t\in[0,t_f-\tau_2],\\ 0, & \text{otherwise},\end{cases}. \tag{4.6}$$

Let $\frac{\partial H(t)}{\partial\eta_1(t)}=\frac{\partial H(t)}{\partial\eta_2(t)}=\frac{\partial H(t)}{\partial\eta_3(t)}=\frac{\partial H(t)}{\partial\eta_4(t)}=0$, to get

$$\frac{\partial H(t)}{\partial \eta_1(t)} = A_1\eta_1(t) - \beta_2 I_m^*(t)S_m^*(t)[e^{-\mu_m\tau_1}\lambda_2(t) - \lambda_1(t)] - \beta_3 I_m^*(t)S_f^*(t)[e^{-\mu_f\tau_2}\lambda_5(t) - \lambda_4(t)] = 0,$$
$$\frac{\partial H(t)}{\partial \eta_2(t)} = A_2\eta_2(t) + I_m^*(t)(\lambda_3(t) - \lambda_2(t)) = 0,$$
$$\frac{\partial H(t)}{\partial \eta_3(t)} = A_3\eta_3(t) - \beta_1 I_f^*(t)S_m^*(t)[e^{-\mu_m\tau_1}\lambda_2(t) - \lambda_1(t)] = 0,$$
$$\frac{\partial H(t)}{\partial \eta_4(t)} = A_4\eta_4(t) + I_f^*(t)(\lambda_6(t) - \lambda_5(t)) = 0.$$

We obtain that

$$\eta_1(t) = \frac{\beta_2 I_m^*(t)S_m^*(t)\,[e^{-\mu_m\tau_1}\lambda_2(t) - \lambda_1(t)] + \beta_3 I_m^*(t)S_f^*(t)\,[e^{-\mu_f\tau_2}\lambda_5(t) - \lambda_4(t)]}{A_1},$$
$$\eta_2(t) = \frac{I_m^*(t)(\lambda_2(t) - \lambda_3(t))}{A_2},\ \eta_3(t) = \frac{\beta_1 I_f^*(t)S_m^*(t)\,[e^{-\mu_m\tau_1}\lambda_2(t) - \lambda_1(t)]}{A_3},$$
$$\eta_4(t) = \frac{I_f^*(t)(\lambda_5(t) - \lambda_6(t))}{A_4}.$$

In addition, we have $\eta^* = (\eta_1^*,\ \eta_2^*,\ \eta_3^*,\ \eta_4^*)$,

$$\begin{aligned}
\eta_1^* &= \min\left\{\eta_1^{\max},\ \max\left\{\frac{\beta_2 I_m^*(t)S_m^*(t)\,[e^{-\mu_m\tau_1}\lambda_2(t) - \lambda_1(t)] + \beta_3 I_m^*(t)S_f^*(t)\,[e^{-\mu_f\tau_2}\lambda_5(t) - \lambda_4(t)]}{A_1}, 0\right\}\right\},\\
\eta_2^* &= \min\left\{\eta_2^{\max},\ \max\left\{\frac{I_m^*(t)(\lambda_2(t) - \lambda_3(t))}{A_2}, 0\right\}\right\},\\
\eta_3^* &= \min\left\{\eta_3^{\max},\ \max\left\{\frac{\beta_1 I_f^*(t)S_m^*(t)\,[e^{-\mu_m\tau_1}\lambda_2(t) - \lambda_1(t)]}{A_3}, 0\right\}\right\},\\
\eta_4^* &= \min\left\{\eta_4^{\max},\ \max\left\{\frac{I_f^*(t)(\lambda_5(t) - \lambda_6(t))}{A_4}, 0\right\}\right\}.
\end{aligned} \tag{4.7}$$

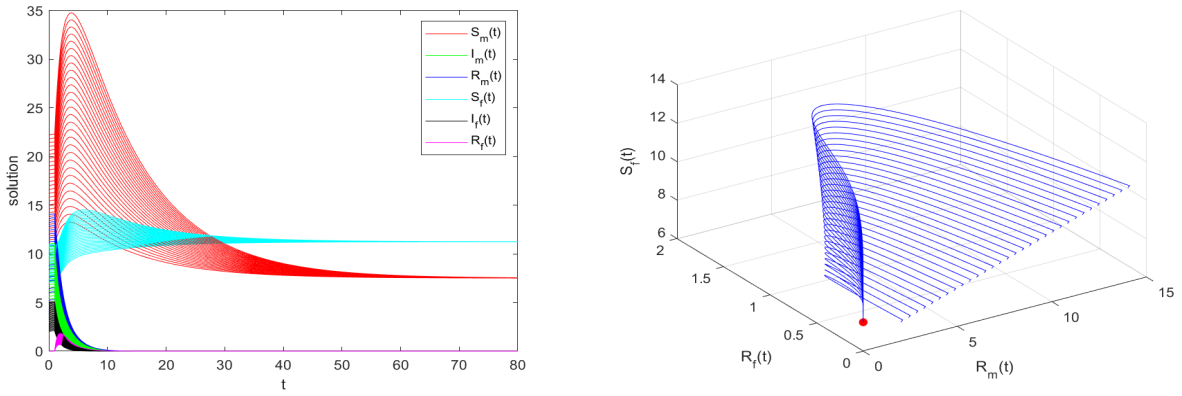
5. Numerical simulations

In this section, the following numerical examples are presented to corroborate the primary conclusions.

Table 2. Values of parameters in model (1.2).

Parameter	Case 1	Case 2	Source	Parameter	Case 1	Case 2	Source
Λ_m	0.3	0.3	[26]	Λ_f	0.45	0.45	[26, 40]
μ_m	0.4	0.4	[26]	μ_f	0.4	0.4	[26, 40]
γ_m	0.6	0.15	fitted	γ_f	0.6	0.15	fitted
κ_m	0.5	0.04	fitted	κ_f	0.5	0.04	fitted
β_1	0.002	0.072	fitted	β_2	0.005	0.075	fitted
β_3	0.005	0.075	fitted				

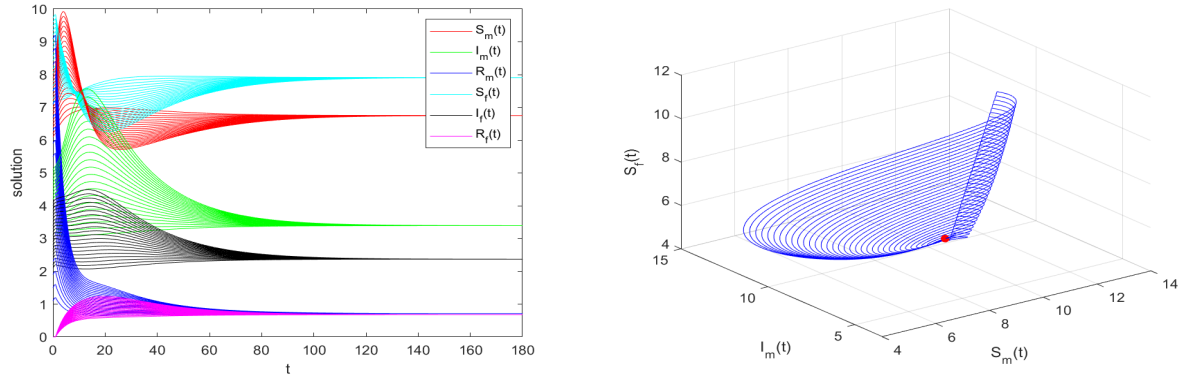
Example 1. The parameter values corresponding to Case 1 in Table 2 are adopted, with the remaining parameters specified as $\tau_1 = 2, \tau_2 = 2$. Initial functions are taken as $(S_m(i), I_m(i), R_m(i), S_f(i), I_f(i), R_f(i))$ =$(11 + 0.4 \times k + 0.1 \times \sin(i \times h), 2 + 0.3 \times k + 0.1 \times \sin(i \times h), 2 + 0.4 \times k + 0.1 \times \sin(i \times h), 7 + 0.13 \times k + 0.1 \times \sin(i \times h), 2 + 0.1 \times k + 0.1 \times \sin(i \times h), 2 + 0.1 \times k + 0.1 \times \sin(i \times h))$, where $i \in [-2, 0], k = 1, ..., 30$. By calculation, it is obtained that $\mathcal{R}_0 = 0.063$. As shown in Figure 2, the DFE $E_0 = (7.521, 0, 0, 11.256, 0, 0)$ of model (1.2) is GAS, which supports that Theorem 3.2.



(a) (b)

Figure 2. (a) and (b) Time series diagram and three-dimensional phase diagram of disease-free equilibrium of system (1.2).

Example 2. In model (1.2), we take the values of parameters in Table 2 Case 2 and set the surplus parameters $\tau_1 = 2.1, \tau_2 = 2.1$, initial functions are taken as $(S_m(i), I_m(i), R_m(i), S_f(i), I_f(i), R_f(i)) = (6 + 0.14 \times k + 0.1 \times \sin(i \times h), 3 + 0.14 \times k + 0.1 \times \sin(i \times h), 0.7 + 0.4 \times k + 0.1 \times \sin(i \times h), 7 + 0.1 \times k + 0.1 \times \sin(i \times h), 2 + 0.1 \times k + 0.1 \times \sin(i \times h), 10 + 0.1 \times k + 0.1 \times \sin(i \times h))$, where $i \in [-2.1, 0], k = 1, ..., 30$. By calculation, $\mathcal{R}_0 = 1.884$. Figure 3 indicates that model (1.2)) is uniformly persistent, which validates Theorem 3.3.



(a) (b)

Figure 3. (a) and (b) Time series diagram and three-dimensional phase diagram of endemic equilibrium of system (1.2).

Example 3. In model (1.2), we take parameters in Table 2 Case 2 and set the surplus parameters $\tau_1 = 2.1, \tau_2 = 2.1$. By calculation, it is obtained that $\mathcal{R}_0 = 1.884$. By Figure 4, model (1.2) exhibits a unique endemic equilibrium, which demonstrates Corollary 3.4 holds.

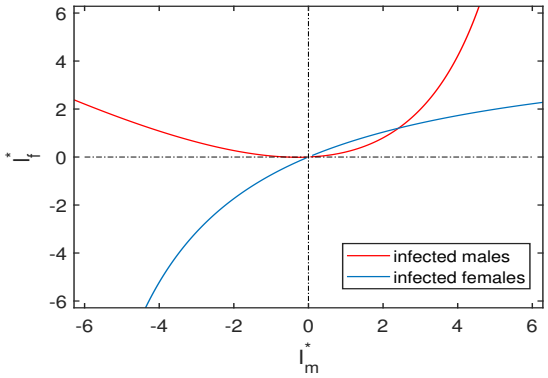


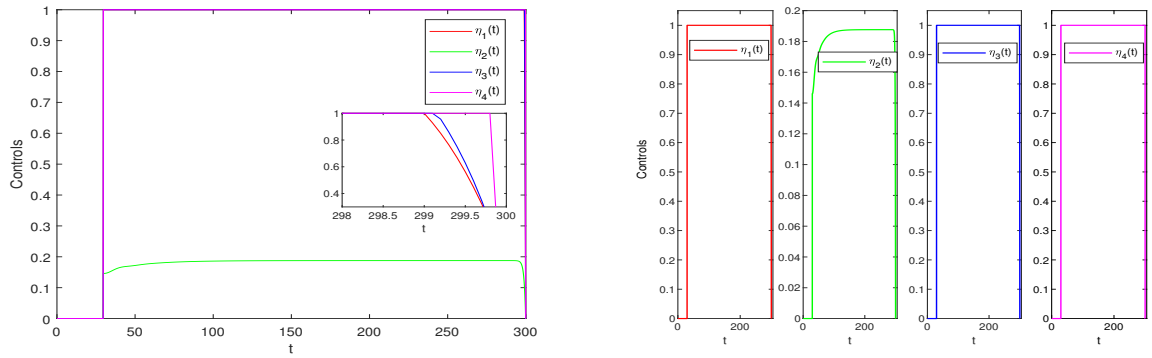
Figure 4. The positive equilibrium exists uniquely.

Example 4. The threshold dynamics of gonorrhea transmission are determined by $\mathcal{R}_0$, whose sensitivity to model parameters was evaluated via Latin Hypercube Sampling (LHS) and Partial Rank Correlation Coefficient (PRCC) analysis [41]. Figure 1 and Table 3 reveal that the parameters μm, μ_f, κ_m, and κ_f exhibit negative correlations with $\mathcal{R}_0$. Conversely, Λ_m, Λ_f, and the transmission rates β_1, β_2, and β_3 show positive correlations with $\mathcal{R}_0$. These findings highlight infection control as the key intervention strategy.

Table 3. Base values and ranges of parameters.

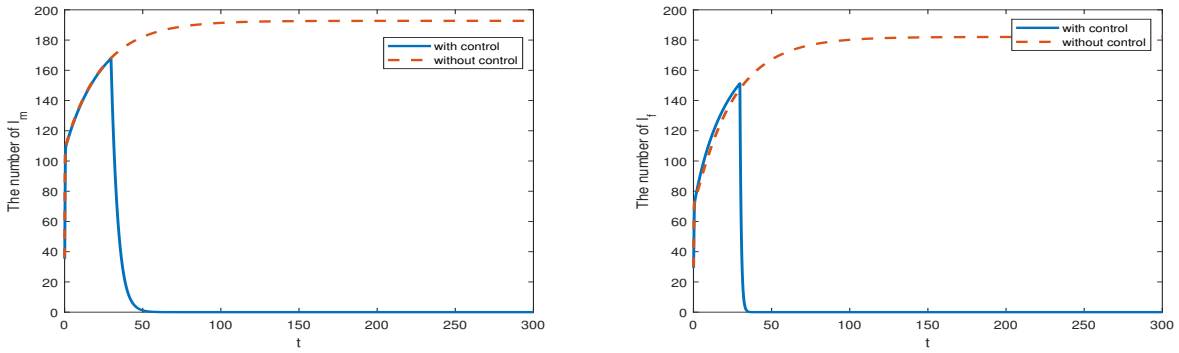
Parameter	Baseline	Range	PRCC	Parameter	Baseline	Range	PRCC
Λ_m	0.3	U[0.2, 0.4]	0.12	Λ_f	0.45	U[0.35, 0.55]	0.08
μ_m	0.04	U[0.01, 0.06]	-0.25	μ_f	0.04	U[0.01, 0.06]	-0.18
κ_m	0.5	U[0.01, 0.6]	-0.3	κ_f	0.5	U[0.01, 0.6]	-0.22
τ_1	1	U[0.1, 10]	-0.15	τ_2	2	U[0.1, 10]	-0.1
β_1	0.002	U[0.001, 0.03]	0.35	β_2	0.005	U[0.001, 0.09]	0.42
β_3	0.005	U[0.001, 0.5]	0.38				

Example 5. For weight constants $A_1 = 10, A_2 = 10.1, A_3 = 1, A_4 = 1$ in the cost functional, the remaining parameters are taken from Table 2 Case 2. Figure 5 demonstrates the effectiveness of the optimal control strategy $(\eta_1(t), \eta_2(t), \eta_3(t), \eta_4(t))$, while Figure 6 illustrates the temporal variations of each compartment over time in the presence or absence of optimal control.



(a) (b)

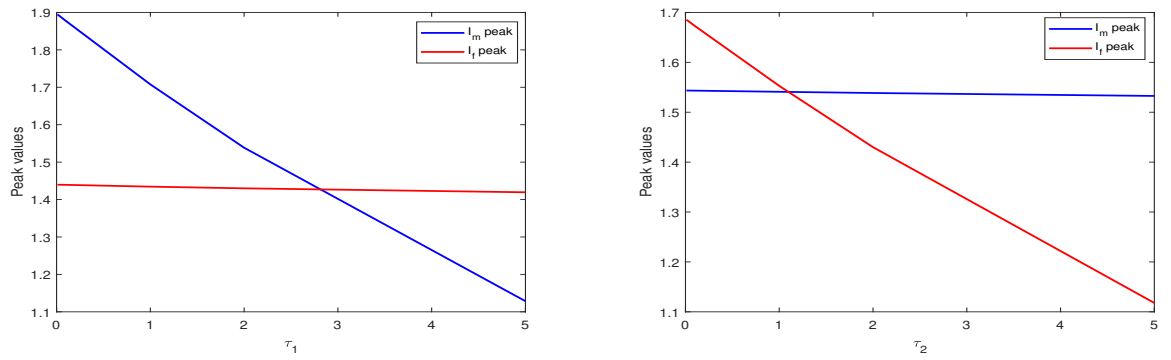
Figure 5. (a) and (b) Time evolution of the four control measures.



(a) (b)

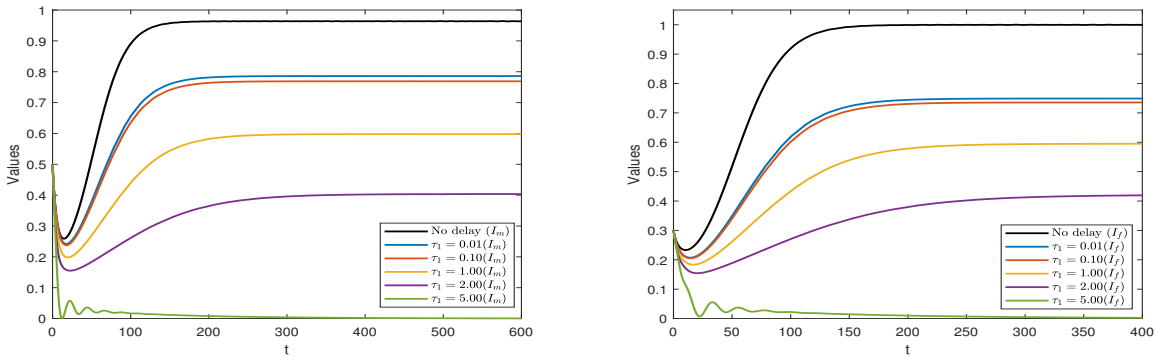
Figure 6. (a) and (b) Comparison of infected populations with and without control.

Example 6. As shown in Figure 7(a), when $\tau_2 = 2$ and τ_1 increases from 0 to 5, the peak value of I_m declines sharply, while that of I_f decreases at a much slower rate. The temporal evolution of I_m and I_f with respect to τ_1 is further illustrated in Figure 8. And Figure 7(b) shows that, for $\tau_1 = 2$, increasing τ_2 from 0 to 5 leads to a monotonic decrease in the peak values of both I_m and I_f, with I_f exhibiting a steeper decline. The corresponding dynamics as functions of τ_2 are depicted in Figure 9.



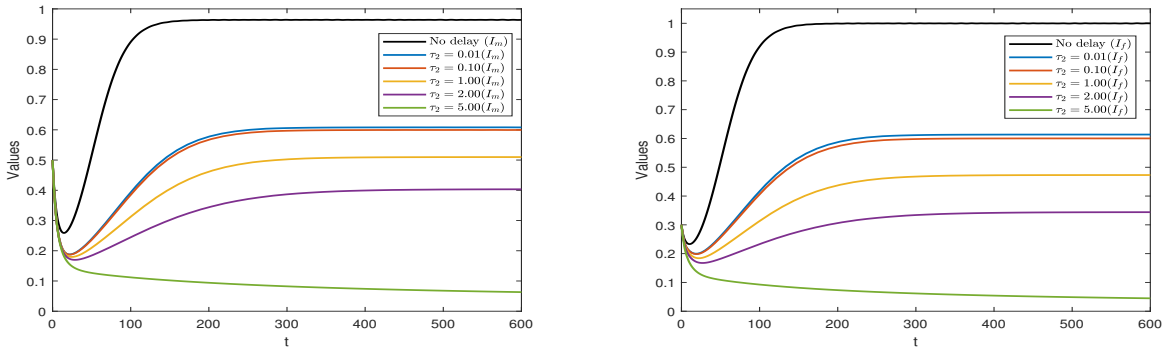
(a) (b)

Figure 7. (a) Peak values of $I_m(t)$ and $I_f(t)$ as functions of τ_1 (with $\tau_2 = 2$); (b) Peak values of $I_m(t)$ and $I_f(t)$ as functions of τ_2 (with $\tau_1 = 2$).



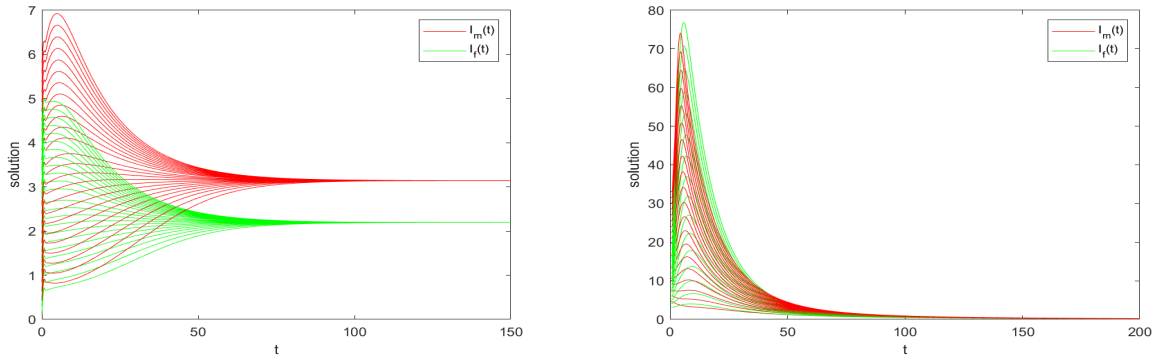
(a) (b)

Figure 8. (a) and (b) Effect of τ_1 on $I_m(t)$ and $I_f(t)$ when $\tau_2 = 2$.



(a) (b)

Figure 9. Effect of τ_2 on $I_m(t)$ and $I_f(t)$ when $\tau_1 = 2$.



(a) (b)

Figure 10. (a) and (b) Comparison of transmission dynamics: with and without homosexual behavior.

Example 7. In model (1.2), we take the values of parameters in Table 2 Case 2 and set the surplus parameters $\tau_1 = 2, \tau_2 = 2$. As shown in Figure 10, infection dynamics differ markedly between transmission scenarios. In the model incorporating homosexual transmission in Figure 10(a), infections rise rapidly to a high peak and stabilize at an elevated endemic level. In contrast, under heterosexual-only transmission in Figure 10(b), the epidemic peak is significantly lower and is followed by eventual

disease extinction.

6. Conclusions

In this paper, we developed and analyzed an SIRS gonorrhea transmission model with dual incubation period delays and sex-structured populations. We computed $\mathcal{R}_0$ and derived the following: (i) when $\mathcal{R}_0 < 1$, the DFE is GAS; (ii) when $\mathcal{R}_0 > 1$, the model shows uniform persistence with a unique endemic equilibrium. These results demonstrate the role of $\mathcal{R}_0$ as the decisive threshold governing disease dynamics. Furthermore, sensitivity analysis reveals that decreasing the sexual transmission rate and enhancing the recovery rate are pivotal strategies for reducing $\mathcal{R}_0$. Based on these findings, an optimal control problem was formulated, indicating that implementing enhanced precautionary measures and promoting sex education could be highly effective in curbing the spread of gonorrhea.

Our comparative analysis showed that the model incorporating homosexual transmission led to a significantly higher infection peak and sustained endemic level, while the heterosexual-only scenario resulted in a lower peak and eventual disease extinction in Figure 10. This indicated that homosexual transmission substantially amplified both the peak and long-term burden of gonorrhea. Therefore, models omitting this component risk underestimating epidemic potential. From a public health perspective, these results emphasized the need for tailored interventions in high-risk populations such as MSM, including enhanced screening and targeted prevention measures, to mitigate the elevated disease burden predicted in our simulations.

In addition, by incorporating the time delay parameter to characterize the incubation period, we conducted a rigorous analysis of the dynamics of infected individuals. As depicted in Figures 8 and 9, an increase in the delay may induce a monotonic decline in the peak magnitude of infected cases and prolong the time required to reach this peak. These findings underscore that overlooking the incubation period in epidemiological models of gonorrhea transmission could lead to an overestimation of the disease's propagation risk. While this study addresses the human incubation period, incorporating the antibiotic-induced latency phase into future models is essential for capturing the full complexity of disease transmission.

Use of AI tools declaration

The authors declare they have not used Artificial Intelligence (AI) tools in the creation of this article.

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Conflict of interest

The authors declare there are no conflicts of interest.

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