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Research article

Stochastic dynamics of a syphilis epidemic model with perturbation: Stationary distribution and extinction thresholds

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Abstract: In this study, a novel epidemiological model which incorporates stochastic perturbations is discussed. The perturbations are introduced through Brownian motion, which accounts for the inherent randomness and environmental fluctuations that affect the disease transmission dynamics. The main goal is to find the existence of a unique non-negative nonlocal solution. We show that the stochastic model has a stationary distribution using the fundamental reproduction number R_0 that was obtained from the related deterministic model. Furthermore, under specific circumstances, we examine the fluctuation of the unique solution of the deterministic issue around the disease-free equilibrium. Additionally, the stochastic stability of the system is examined, and sufficient criteria for both extinction and persistence are established in the presence of noise. Our findings are further validated by numerical simulations, which supplement the theoretical insights.

Keywords: stochastic model; stochastic perturbation; Brownian motion; stochastic stability
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1. Introduction

Syphilis is a chronic systemic infection caused by the spirochete Treponema pallidum subsp. pallidum, a highly motile pathogen with tropism for mucocutaneous and neural tissues. Globally, the World Health Organization (WHO) estimates 7.1 million new syphilis cases annually, with disproportionate burdens in low-resource regions of sub-Saharan Africa and South Asia [1].

Syphilis is a highly contagious bacterial disease with multiple stages that causes significant morbidity and mortality globally. In recent years, it has risen in popularity among all age groups in both industrialized and developing countries, particularly following the COVID-19 pandemic. Since sexual contact is the primary mechanism of transmission, the host's age structure is critical for

syphilis propagation. Syphilis relapse is still a major clinical challenge. It is reported to occur in a percentage of patients who receive a standard therapy, which can greatly vary depending on factors such as immune status and treatment adequacy. Recurrence rates may be higher in immunocompromised individuals or those who receive an incomplete treatment [2]. Recurrent mucocutaneous lesions (such as maculopapular rashes and condylomata lata), lymphadenopathy, and neuro-ophthalmic symptoms (such as headaches and blurred vision) are common in early relapses (less than two years after infection), while gummatous lesions or cardiovascular problems may be present in late relapses [3, 4]. In research or complex clinical situations, a definitive diagnosis frequently necessitates an integrated serological investigation, which combines quantitative titers with treponemal (e.g., TPPA/TPHA) and nontreponemal (e.g., RPR/VDRL) assays, as well as a clinical assessment to distinguish real relapse from reinfection [5].

Multifactorial interactions play a role in the etiology of recurrence. First, in immunoprivileged sanctuaries such as the central nervous system (CNS) and aqueous humor, where antibiotic penetration is less than ideal, *Treponema pallidum* subsp. *pallidum* can survive [6]. Second, the effectiveness of treatment is compromised by host immunological dysregulation, which is mostly caused by Human immunodeficiency virus (HIV) coinfection and can hinder bacterial clearance [7]. Third, high-risk sexual practices that result in reinfection and the emergence of antimicrobial resistance, especially to macrolides, might make therapy more difficult and contribute to situations that resemble relapse or true treatment failure.

The 2021 Center for Disease Control (CDC) Sexually Transmitted Infections Treatment Guidelines notably advocate enhanced regimens for recurrent early syphilis cases, thereby highlighting the importance of risk-stratified therapy approaches in current clinical guidelines [8]. Intramuscular benzathine penicillin G at 2.4 million units per week for three weeks is the gold standard for treating patients with recurrent secondary syphilis or those exhibiting serological persistence following the first therapy. This prolonged regimen is consistent with the 2020 European guideline on the management of syphilis, which emphasizes cerebrospinal fluid (CSF) evaluation before retreatment in such complex cases, as well as the WHO's 2021 syphilis management framework, which advocates enhanced therapies for high-risk recurrences [9]. The three-dose regimen maintains practical treatment accessibility while reflecting the current knowledge of possible undiscovered neuroinvasion in recurring infections.

In order to obtain bactericidal concentrations in CSF, neuroinvasive relapse, such as primary neurosyphilis, requires intravenous aqueous penicillin G (18–24 million units daily for 10–14 days). Ceftriaxone (2 g daily intravenously for 10–14 days) is a suggested substitute for doxycycline in patients with neurosyphilis who are allergic to penicillin because of its higher CNS penetration. Although research is ongoing in more general infectious illness scenarios, the role of adjuvant immunomodulation in routine syphilis care is still unknown [10].

In epidemiology, stochastic models are mathematical instruments that integrate uncertainty and unpredictability into the dynamics of infectious illnesses [11–13]. The variability and unpredictability of real-world epidemics are taken into account by stochastic models, as opposed to deterministic models, which assume fixed parameters and initial conditions [14]. Random events including individual interactions, transmission events, recovery periods, and environmental changes that can impact the spread and control of disease can be captured by stochastic models [15]. Furthermore, in a stochastic world, these models are strong and adaptable instruments that can aid in understanding and

forecasting the behavior of infectious diseases [16–19]. Recent studies have further advanced the stochastic and deterministic analysis of compartmental epidemic models, including waning-immunity SIRS frameworks [20], stability analysis of infection-age-structured and delayed SIRS models [21, 22], and SIRS models with nonmonotone incidence and saturated treatment [23].

Formulation of the syphilis model

Three different stages of infection are included in a comprehensive model for syphilis dynamics that was recently presented by advances in mathematical epidemiology. The model offers a thorough foundation to comprehend how syphilis spreads and is managed within a population. The following is the mathematical expression that includes the three stages of infection:

$$\begin{cases} S'_m = \pi_m + \varphi_m R_m - \alpha\psi\left(\dfrac{I_{fp} + I_{fs} + L_f}{N}\right) S_m - \mu S_m, \\ I'_{mp} = \alpha\psi\left(\dfrac{I_{fp} + I_{fs} + L_f}{N}\right) S_m - \gamma_m I_{mp} - \mu I_{mp} - \sigma_{m1} I_{mp}, \\ I'_{ms} = \gamma_m I_{mp} - \beta_m I_{ms} - \sigma_{m2} I_{ms} - \mu I_{ms}, \\ L'_m = \beta_m I_{ms} - \mu L_m - \sigma_{m3} L_m - \delta_m L_m, \\ R'_m = \sigma_{m1} I_{mp} + \sigma_{m2} I_{ms} + \sigma_{m3} L_m - \mu R_m - \varphi_m R_m, \\ S'_f = \pi_f + \varphi_f R_f - \alpha_m\psi\left(\dfrac{I_{mp} + I_{ms} + L_m}{N}\right) S_f - \pi_p S_f - \mu S_f, \\ I'_{fp} = \alpha_m\psi\left(\dfrac{I_{mp} + I_{ms} + L_m}{N}\right) S_f - \gamma_f I_{fp} - \mu I_{fp} - \rho_{f1} I_{fp} - \pi_p I_{fp} - \delta_f I_{fp}, \\ I'_{fs} = \gamma_f I_{fp} - \beta_f I_{fs} - \rho_{f2} I_{fs} - \mu I_{fs} - \pi_p I_{fs} - \delta_f I_{fs}, \\ L'_f = \beta_f I_{fs} - \mu L_f - \rho_{f3} L_f - \pi_p L_f - \delta_f L_f, \\ R'_f = \rho_{f1} I_{fp} + \rho_{f2} I_{fs} + \rho_{f3} L_f + (1 - \kappa_f)\pi_p (I_{fp} + I_{fs} + L_f) - \mu R_f - \varphi_f R_f, \\ C' = \kappa_f \pi_p (I_{fp} + I_{fs} + L_f) - (\mu + \delta_c) C, \\ D' = \delta_m L_m + \delta_f I_{fp} + \delta_f I_{fs} + \delta_f L_f + \delta_c C - \mu D, \end{cases} \tag{1}$$

where:

- $S_m, I_{mp}, I_{ms}, L_m, R_m$ represent male compartments,
- $S_f, I_{fp}, I_{fs}, L_f, R_f$ represent female compartments,
- C is the compartment for congenitally infected newborns, and
- D is the compartment for disability due to long-term effects.

The total population at time t, represented by $N(t)$, is given by the following:

$$\boxed{N(t) = S_m + S_f + I_{mp} + I_{fp} + I_{ms} + I_{fs} + L_m + L_f + R_m + R_f + C + D.}$$

Consider that C and D help make the model more sophisticated, which allows us to study syphilis epidemiology and its consequences in terms of disability. The parameters used in the syphilis model (1) are defined in Table 1. Each parameter represents a specific biological or epidemiological process in the transmission and progression of syphilis. The flowchart of the model (1) is shown in Figure 1.

Table 1. Description of parameters used in the syphilis model (1).

Parameter	Description
π_m	Recruitment rate of susceptible males
π_f	Recruitment rate of susceptible females
π_p	Rate of prevention/control measures (e.g., condom use, education)
μ	Natural death rate
α	Transmission rate from females to males
α_m	Transmission rate from males to females
Λ_m	Rate of loss of immunity in males
φ_f	Rate of loss of immunity in females
γ_m	Progression rate from primary to secondary stage in males
γ_f	Progression rate from primary to secondary stage in females
β_m	Progression rate from secondary to latent stage in males
β_f	Progression rate from secondary to latent stage in females
σ_{m1}	Treatment/recovery rate of primary stage males
σ_{m2}	Treatment/recovery rate of secondary stage males
σ_{m3}	Treatment/recovery rate of latent stage males
ρ_{f1}	Treatment/recovery rate of primary stage females
ρ_{f2}	Treatment/recovery rate of secondary stage females
ρ_{f3}	Treatment/recovery rate of latent stage females
δ_m	Disease-induced mortality rate in latent males
δ_f	Disease-induced mortality rate in secondary females
δ_c	Disease-induced mortality rate in congenitally infected newborns
κ_f	Probability of congenital transmission from infected pregnant females
φ_m	Rate of waning immunity/recovery in males
φ_f	Rate of waning immunity/recovery in females
ψ	Force of infection function
N	Total population size

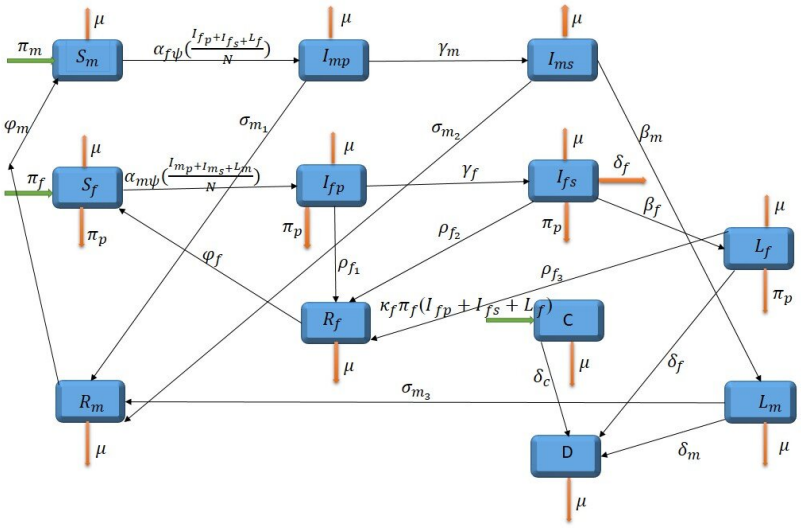

Figure 1. Flow chart diagram of the model.

2. Disease-free equilibrium (DFE)

At the disease-free equilibrium, all infection compartments are zero. Thus, the DFE is given by:

$$E_0 = \left(S_m^0, 0, 0, 0, 0, S_f^0, 0, 0, 0, 0, 0, 0\right), \tag{2}$$

where:

$$S_m^0 = \frac{\pi_m}{\mu}, \qquad S_f^0 = \frac{\pi_f}{\pi_p + \mu}, \tag{3}$$

and the total population at DFE is:

$$N^* = S_m^0 + S_f^0 = \frac{\pi_m}{\mu} + \frac{\pi_f}{\pi_p + \mu}. \tag{4}$$

3. Basic reproduction number $\mathcal{R}_0$

We employ the next-generation matrix method as described by van den Driessche and Watmough [24] to derive the basic reproduction number $\mathcal{R}_0$. The infected compartments relevant for disease transmission are as follows:

$$\mathbf{X} = \left(I_{mp}, I_{ms}, L_m, I_{fp}, I_{fs}, L_f\right)^T. \tag{5}$$

The forces of infection for males and females are defined as follows:

$$\lambda_m = \alpha\psi\left(\frac{I_{fp} + I_{fs} + L_f}{N}\right), \tag{6}$$

$$\lambda_f = \alpha_m\psi\left(\frac{I_{mp} + I_{ms} + L_m}{N}\right). \tag{7}$$

The matrix of new infections, evaluated at the DFE, is given by the following:

$$F = \begin{bmatrix} 0 & 0 & 0 & \frac{\alpha\psi S_m^0}{N^*} & \frac{\alpha\psi S_m^0}{N^*} & \frac{\alpha\psi S_m^0}{N^*} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{\alpha_m\psi S_f^0}{N^*} & \frac{\alpha_m\psi S_f^0}{N^*} & \frac{\alpha_m\psi S_f^0}{N^*} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}. \tag{8}$$

To simplify the transition matrix, we define the following positive constants:
For males:

$$\begin{cases} A_1 = & \gamma_m + \mu + \sigma_{m1}, \\ A_2 = & \beta_m + \sigma_{m2} + \mu, \\ A_3 = & \mu + \sigma_{m3} + \delta_m. \end{cases} \tag{9}$$

For females:

$$\begin{cases} B_1 = & \gamma_f + \mu + \rho_{f1} + \pi_p, \\ B_2 = & \beta_f + \rho_{f2} + \mu + \pi_p + \delta_f, \\ B_3 = & \mu + \rho_{f3} + \pi_p + \delta_f. \end{cases} \tag{10}$$

The transition matrix V takes the following form:

$$V = \begin{bmatrix} A_1 & 0 & 0 & 0 & 0 & 0 \\ -\gamma_m & A_2 & 0 & 0 & 0 & 0 \\ 0 & -\beta_m & A_3 & 0 & 0 & 0 \\ 0 & 0 & 0 & B_1 & 0 & 0 \\ 0 & 0 & 0 & -\gamma_f & B_2 & 0 \\ 0 & 0 & 0 & 0 & -\beta_f & B_3 \end{bmatrix}. \tag{11}$$

The inverse of V is as follows:

$$V^{-1} = \begin{bmatrix} V_m^{-1} & \mathbf{0} \\ \mathbf{0} & V_f^{-1} \end{bmatrix}, \tag{12}$$

where:

$$V_m^{-1} = \begin{bmatrix} \frac{1}{A_1} & 0 & 0 \\ \frac{\gamma_m}{A_1A_2} & \frac{1}{A_2} & 0 \\ \frac{\gamma_m\beta_m}{A_1A_2A_3} & \frac{\beta_m}{A_2A_3} & \frac{1}{A_3} \end{bmatrix}, \tag{13}$$

and

$$V_f^{-1} = \begin{bmatrix} \frac{1}{B_1} & 0 & 0 \\ \frac{\gamma_f}{B_1B_2} & \frac{1}{B_2} & 0 \\ \frac{\gamma_f\beta_f}{B_1B_2B_3} & \frac{\beta_f}{B_2B_3} & \frac{1}{B_3} \end{bmatrix}. \tag{14}$$

The next-generation matrix is given by $K = FV^{-1}$. Since F only has non-zero entries in the first and fourth rows, the matrix K reduces to the following:

$$K = \begin{bmatrix} 0 & k_{MF} \\ k_{FM} & 0 \end{bmatrix}, \tag{15}$$

where

$$k_{MF} = \frac{\alpha\psi S_m^0}{N^*}\left(\frac{1}{B_1} + \frac{1}{B_2} + \frac{1}{B_3}\right), \tag{16}$$

is the expected number of male infections generated by a single infected female, and

$$k_{FM} = \frac{\alpha_m\psi S_f^0}{N^*}\left(\frac{1}{A_1} + \frac{1}{A_2} + \frac{1}{A_3}\right), \tag{17}$$

is the expected number of female infections generated by a single infected male.
The basic reproduction number $\mathcal{R}_0$ is the spectral radius of the next-generation matrix K:

$$\mathcal{R}_0 = \rho(K) = \sqrt{k_{MF} \cdot k_{FM}}. \tag{18}$$

By substituting the expressions for k_{MF} and k_{FM}, we obtain the following:

$$\boxed{\mathcal{R}_0 = \sqrt{\left(\frac{\alpha\psi S_m^0}{N^*}\right)\left(\frac{\alpha_m\psi S_f^0}{N^*}\right)\left(\frac{1}{B_1} + \frac{1}{B_2} + \frac{1}{B_3}\right)\left(\frac{1}{A_1} + \frac{1}{A_2} + \frac{1}{A_3}\right)}}. \tag{19}$$

Equivalently, $\mathcal{R}_0$ can be expressed as the geometric mean of the male-to-female and female-to-male reproductive numbers:

$$\mathcal{R}_0 = \sqrt{\mathcal{R}_{MF} \cdot \mathcal{R}_{FM}}, \tag{20}$$

where

$$\mathcal{R}_{MF} = \frac{\alpha\psi S_m^0}{N^*}\left(\frac{1}{B_1} + \frac{1}{B_2} + \frac{1}{B_3}\right), \tag{21}$$

represents the expected number of males infected by a single female during her entire infectious period, and

$$\mathcal{R}_{FM} = \frac{\alpha_m\psi S_f^0}{N^*}\left(\frac{1}{A_1} + \frac{1}{A_2} + \frac{1}{A_3}\right) \tag{22}$$

represents the expected number of females infected by a single male during his entire infectious period. The terms $\frac{1}{B_1} + \frac{1}{B_2} + \frac{1}{B_3}$ and $\frac{1}{A_1} + \frac{1}{A_2} + \frac{1}{A_3}$ represent the total expected infectious durations (summed across all stages) for females and males, respectively. The DFE is locally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$. When $\mathcal{R}_0 = 1$, the system undergoes a transcritical bifurcation, which leads to the emergence of an endemic equilibrium [25]. If we assume that individuals in the latent stage do not transmit the infection, then the basic reproduction number reduces to the following:

$$\boxed{\mathcal{R}_0 = \sqrt{\left(\frac{\alpha\psi S_m^0}{N^*}\right)\left(\frac{\alpha_m\psi S_f^0}{N^*}\right)\left(\frac{1}{B_1} + \frac{1}{B_2}\right)\left(\frac{1}{A_1} + \frac{1}{A_2}\right)}}. \tag{23}$$

At the endemic equilibrium, all time derivatives vanish. Setting the derivatives to zero and solving the resulting algebraic system yields the endemic equilibrium point as follows:

$$S_m^* = \frac{\pi_m}{\lambda_f^* + \mu}, I_{mp}^* = \frac{\lambda_f^* S_m^*}{A_1}, I_{ms}^* = \frac{\gamma_m I_{mp}^*}{B_1}, L_m^* = \frac{\beta_m I_{ms}^*}{B_2}, S_f^* = \frac{\pi_f}{\lambda_m^* + \pi_p + \mu}, I_{fp}^* = \frac{\lambda_m^* S_f^*}{A_2},$$

$$I_{fs}^* = \frac{\gamma_f I_{fp}^*}{B_3}, L_f^* = \frac{\beta_f I_{fs}^*}{B_4}, C^* = \frac{\kappa_f \pi_p (I_{fp}^* + I_{fs}^* + L_f^*)}{\mu + \delta_c}, D^* = \frac{\delta_m L_m^* + \delta_f (I_{fp}^* + I_{fs}^* + L_f^*) + \delta_c C^*}{\mu}.$$

4. Model formulation with stochastic terms

The stochastic version of Model (1) can be obtained by adding white noise in each compartment as follows:

$$\begin{cases}
dS_m = \left[\pi_m + \varphi_m R_m - \alpha\psi\left(\frac{I_{fp} + I_{fs} + L_f}{N}\right) S_m - \mu S_m\right]dt + \sigma_1\, dB_1(t), \\
dI_{mp} = \left[\alpha\psi\left(\frac{I_{fp} + I_{fs} + L_f}{N}\right) S_m - \gamma_m I_{mp} - \mu I_{mp} - \sigma_{m1} I_{mp}\right]dt + \sigma_2\, dB_2(t), \\
dI_{ms} = \left[\gamma_m I_{mp} - \beta_m I_{ms} - \sigma_{m2} I_{ms} - \mu I_{ms}\right]dt + \sigma_3\, dB_3(t), \\
dL_m = \left[\beta_m I_{ms} - \mu L_m - \sigma_{m3} L_m - \delta_m L_m\right]dt + \sigma_4\, dB_4(t), \\
dR_m = \left[\sigma_{m1} I_{mp} + \sigma_{m2} I_{ms} + \sigma_{m3} L_m - \mu R_m - \varphi_m R_m\right]dt + \sigma_5\, dB_5(t), \\
dS_f = \left[\pi_f + \varphi_f R_f - \alpha_m\psi\left(\frac{I_{mp} + I_{ms} + L_m}{N}\right) S_f - \pi_p S_f - \mu S_f\right]dt + \sigma_6\, dB_6(t), \\
dI_{fp} = \left[\alpha_m\psi\left(\frac{I_{mp} + I_{ms} + L_m}{N}\right) S_f - \gamma_f I_{fp} - \mu I_{fp} - \rho_{f1} I_{fp} - \pi_p I_{fp} - \delta_f I_{fp}\right]dt + \sigma_7\, dB_7(t), \\
dI_{fs} = \left[\gamma_f I_{fp} - \beta_f I_{fs} - \rho_{f2} I_{fs} - \mu I_{fs} - \pi_p I_{fs} - \delta_f I_{fs}\right]dt + \sigma_8\, dB_8(t), \\
dL_f = \left[\beta_f I_{fs} - \mu L_f - \rho_{f3} L_f - \pi_p L_f - \delta_f L_f\right]dt + \sigma_9\, dB_9(t), \\
dR_f = \left[\rho_{f1} I_{fp} + \rho_{f2} I_{fs} + \rho_{f3} L_f + (1 - \kappa_f)\pi_p (I_{fp} + I_{fs} + L_f) - \mu R_f - \varphi_f R_f\right]dt + \sigma_{10}\, dB_{10}(t), \\
dC = \left[\kappa_f \pi_p (I_{fp} + I_{fs} + L_f) - (\mu + \delta_c) C\right]dt + \sigma_{11}\, dB_{11}(t), \\
dD = \left[\delta_m L_m + \delta_f I_{fp} + \delta_f I_{fs} + \delta_f L_f + \delta_c C - \mu D\right]dt + \sigma_{12}\, dB_{12}(t),
\end{cases} \tag{24}$$

with the following initial conditions:

$$S_m(0) = S_{m0} > 0, I_{ms}(0) = I_{ms0} > 0, L_m(0) = L_{m0} > 0, R_m(0) = R_{m0} > 0, S_f(0) = S_{f0} > 0,$$
$$I_{fp}(0) = I_{fp0} > 0, I_{fs}(0) = I_{fs0}, L_f(0) = L_{f0} > 0, R_f(0) = R_{f0} > 0, C(0) = C_0 > 0, D(0) = D_0 > 0. \tag{25}$$

Here, $B_1(t), B_2(t), B_3(t), \cdots$, and $B_{12}(t)$ are independent standard Brownian motions, and the noise intensities $\sigma_1, \sigma_2, \sigma_3, \cdots, \sigma_{12} > 0$ quantify the randomness affecting different aspects of the population and disease dynamics. These stochastic terms account for real-life phenomena, resulting from variations in individual behaviors, climate conditions, the availability of healthcare, and other unexpected events.

5. Existence uniqueness and positivity

This section is devoted to establishing the fundamental well-posedness of the stochastic syphilis model (24). Specifically, we address the existence, uniqueness, and positivity of solutions, which are essential prerequisites for any meaningful dynamical analysis of the system. The stochastic differential equations under consideration are defined on a complete probability space $(\Omega, \mathcal{F}, \{\mathcal{F}_t\}_{t\geq 0}, \mathbb{P})$ equipped with a filtration satisfying the usual conditions, and driven by a k-dimensional standard Brownian motion [26, 27]. The state space of interest is the positive orthant $\mathbb{R}^{12}_+$, which ensures biological feasibility of the compartmental variables.

Theorem 1. *For any initial value*

$$\mathbf{X}(0) = (S_m(0), I_{mp}(0), I_{ms}(0), L_m(0), R_m(0), S_f(0), I_{fp}(0), I_{fs}(0), L_f(0), R_f(0), C(0), D(0))^\top \in \mathbb{R}^{12}_+,$$

the stochastic syphilis model (24) admits a unique global positive solution

$$\mathbf{X}(t) = (S_m(t), I_{mp}(t), I_{ms}(t), L_m(t), R_m(t), S_f(t), I_{fp}(t), I_{fs}(t), L_f(t), R_f(t), C(t), D(t))^\top \in \mathbb{R}^{12}_+$$

for all $t \geq 0$, almost surely.

6. Ergodicity and asymptotic behavior

Now, we present sufficient conditions for the existence of a stationary distribution for the stochastic syphilis system and explore the long-term behavior of the total population.

6.1. Existence of a stationary distribution

Theorem 2. *Let*

$$E^* = (S^*_m, I^*_{mp}, I^*_{ms}, L^*_m, R^*_m, S^*_f, I^*_{fp}, I^*_{fs}, L^*_f, R^*_f, C^*, D^*)$$

be the endemic equilibrium of the deterministic syphilis model (1). Since the noise in Model (24) is additive, the diffusion matrix is constant and given by

$$A = \frac{1}{2}GG^\top = \frac{1}{2}\,\mathrm{diag}(\sigma_1^2, \sigma_2^2, \ldots, \sigma_{12}^2),$$

which is uniformly elliptic on every compact subset of $\mathbb{R}^{12}_+$ [28]. Furthermore, assume the following:

1) ***(H1)*** *There exists a C^2-function $V : \mathbb{R}^{12}_+ \to \mathbb{R}_+$ and a compact set $K \subset \mathbb{R}^{12}_+$ such that*

$$\mathcal{L}V(\mathbf{x}) \leq -1, \quad \forall \mathbf{x} \in \mathbb{R}^{12}_+ \setminus K,$$

where $\mathcal{L}$ is the generator of the stochastic process;

2) ***(H2)*** *The noise intensities satisfy*

$$\sigma_i^2 < 2\mu, \quad i = 1, \ldots, 12,$$

where μ is the natural death rate.

Then, the stochastic syphilis model (24) has a unique stationary distribution $\pi(\cdot)$ on $\mathbb{R}^{12}_+$ [29], and the solution process is ergodic in the sense that for any integrable function $f : \mathbb{R}^{12}_+ \to \mathbb{R}$,

$$\lim_{t\to\infty} \frac{1}{t} \int_0^t f(\mathbf{X}(s))\, ds = \int_{\mathbb{R}^{12}_+} f(\mathbf{x})\pi(d\mathbf{x}), \quad a.s.$$

Remark 1. *Under the conditions outlined in the theorem, the stochastic system has a unique ergodic stationary distribution $\pi(\cdot)$, whose support lies in the positive region and whose mean is approximately the deterministic endemic equilibrium E^* when the noise is small.*

Theorem 3. *Let $E^* = (S^*_m, I^*_{mp}, I^*_{ms}, L^*_m, R^*_m, S^*_f, I^*_{fp}, I^*_{fs}, L^*_f, R^*_f, C^*, D^*)^\top \in \mathbb{R}^{12}_+$ be the endemic equilibrium of the deterministic syphilis model (1). Assume that the noise intensities σ_i ($i = 1, 2, \ldots, 12$) are sufficiently small such that the following conditions hold:*

$$D_i > 0 \quad \text{for all } i = 1, 2, \ldots, 12, \tag{26}$$

where the constants D_i are defined in the proof. Then, the solution $X(t)$ of the stochastic syphilis model (24) fluctuates around the endemic equilibrium E^* in the sense that

$$\limsup_{t\to\infty} \frac{1}{t} \int_0^t \mathbb{E}\left[\sum_{i=1}^{12} (X_i(s) - E_i^*)^2\right] ds \leq \frac{\sum_{i=1}^{12} \sigma_i^2 (E_i^*)^2}{2C}, \tag{27}$$

where

$$C = \min_{1\leq i\leq 12} D_i > 0. \tag{28}$$

Moreover, the solution is stochastically ultimately bounded.

6.2. Asymptotic stability around the disease-free equilibrium

In this subsection, we analyze the asymptotic behavior of the stochastic syphilis model around its DFE. The following theorem provides sufficient conditions for asymptotic stability in the mean square around the DFE.

Theorem 4. *Let*

$$E^0 = (S^0_m, 0, 0, 0, R^0_m, S^0_f, 0, 0, 0, R^0_f, 0, 0) \tag{29}$$

be the DFE of the deterministic syphilis system, where

$$S^0_m = \frac{\pi_m}{\mu}, \qquad S^0_f = \frac{\pi_f}{\mu + \pi_p}, \qquad R^0_m = R^0_f = 0. \tag{30}$$

Suppose that the basic reproduction number satisfies $\mathcal{R}_0 < 1$, and the noise intensities satisfy the following conditions:

$$\begin{cases} \sigma_1^2 & < 2\mu, \\ \sigma_2^2 & < 2(\mu + \gamma_m + \sigma_{m1}), \\ \sigma_3^2 & < 2(\mu + \beta_m + \sigma_{m2}), \\ \sigma_4^2 & < 2(\mu + \sigma_{m3} + \delta_m), \\ \sigma_5^2 & < 2\mu, \\ \sigma_6^2 & < 2(\mu + \pi_p), \\ \sigma_7^2 & < 2(\mu + \gamma_f + \rho_{f1} + \pi_p + \delta_f), \\ \sigma_8^2 & < 2(\mu + \beta_f + \rho_{f2} + \pi_p + \delta_f), \\ \sigma_9^2 & < 2(\mu + \rho_{f3} + \pi_p + \delta_f), \\ \sigma_{10}^2 & < 2\mu, \\ \sigma_{11}^2 & < 2(\mu + \delta_c), \\ \sigma_{12}^2 & < 2\mu. \end{cases}$$

Then, the solution of the stochastic syphilis system satisfies the following inequality:

$$\limsup_{t\to\infty} \frac{1}{t} \int_0^t \mathbb{E}\left[\sum_{i=1}^{12} (X_i(s) - X_i^0)^2\right] ds \leq \frac{\sigma_1^2(\pi_m)^2 + \sigma_6^2(\pi_f)^2}{C^2\mu^2}, \tag{31}$$

where X_i represents the i-th compartment variable, X_i^0 is its DFE value, and

$$C = \min\left\{ \begin{array}{ll} \mu - \dfrac{\sigma_1^2}{2\mu}, & \mu + \gamma_m + \sigma_{m1} - \dfrac{\sigma_2^2}{2}, \\ \mu + \beta_m + \sigma_{m2} - \dfrac{\sigma_3^2}{2}, & \mu + \sigma_{m3} + \delta_m - \dfrac{\sigma_4^2}{2}, \\ \mu - \dfrac{\sigma_5^2}{2\mu}, & \mu + \pi_p - \dfrac{\sigma_6^2}{2\mu}, \\ \mu + \gamma_f + \rho_{f1} + \pi_p + \delta_f - \dfrac{\sigma_7^2}{2}, & \mu + \beta_f + \rho_{f2} + \pi_p + \delta_f - \dfrac{\sigma_8^2}{2}, \\ \mu + \rho_{f3} + \pi_p + \delta_f - \dfrac{\sigma_9^2}{2}, & \mu - \dfrac{\sigma_{10}^2}{2\mu}, \\ \mu + \delta_c - \dfrac{\sigma_{11}^2}{2}, & \mu - \dfrac{\sigma_{12}^2}{2\mu} \end{array} \right\}. \tag{32}$$

Remark 2. *If $\mathcal{R}_0 < 1$ and there is no noise in the susceptible compartments, (i.e., $\sigma_1 = \sigma_6 = 0$), then the DFE of the stochastic syphilis system is stochastically asymptotically stable provided that the*

following conditions on the noise intensities hold:

$$\begin{cases} \sigma_2^2 & < 2(\mu + \gamma_m + \sigma_{m1}), \\ \sigma_3^2 & < 2(\mu + \beta_m + \sigma_{m2}), \\ \sigma_4^2 & < 2(\mu + \sigma_{m3} + \delta_m), \\ \sigma_7^2 & < 2(\mu + \gamma_f + \rho_{f1} + \pi_p + \delta_f), \\ \sigma_8^2 & < 2(\mu + \beta_f + \rho_{f2} + \pi_p + \delta_f), \\ \sigma_9^2 & < 2(\mu + \rho_{f3} + \pi_p + \delta_f), \\ \sigma_{11}^2 & < 2(\mu + \delta_c). \end{cases}$$

6.3. Extinction of the disease

The long-term behavior of the disease is determined by the basic reproduction number $\mathcal{R}_0$ in deterministic epidemic models. If $\mathcal{R}_0 < 1$, then the disease finally passes; if $\mathcal{R}_0 > 1$, it continues to exist in the population [30, 31]. In this paragraph, we demonstrate that, even though the deterministic system predicts persistence, the disease may nevertheless become extinct in the stochastic syphilis model if the noise is high enough [32].

Lemma 1. *Let $M(t)$, $t \geq 0$, be a continuous local martingale with an initial value of $M(0) = 0$. Let $G > 1$, and let (m) and (ϑ_m) be two sequences of positive real numbers with $m \to \infty$. Then, for almost all $\omega \in \Omega$, there exists a random integer $m_0(\omega)$ such that for all $m \geq m_0$,*

$$M(t) \leq \vartheta_m, \quad \text{for all } 0 \leq t \leq m. \tag{33}$$

Now, we derive a sufficient condition for the exponential extinction of the primary infected female class $I_{fp}(t)$, which directly drives the infection dynamics in females and congenital cases.

Theorem 5. *Let $\mathbf{X}(t)$ be the unique global positive solution of the stochastic syphilis model (24). Assume that the I_{fp} compartment is perturbed by multiplicative noise of the following form:*

$$\begin{aligned} dI_{fp}(t) = &\left[\alpha_m \psi \left(\frac{I_{mp} + I_{ms} + L_m}{N}\right) S_f - (\gamma_f + \mu + \rho_{f1} + \pi_p + \delta_f) I_{fp}\right] dt \\ &+ \sigma_{S_f I_{fp}} S_f I_{fp}\, dB_{S_f I_{fp}}(t) + \sigma_{I_{fp}} I_{fp}\, dB_{I_{fp}}(t), \end{aligned}$$

where $\sigma_{S_f I_{fp}}$ and $\sigma_{I_{fp}}$ are positive constants, and $B_{S_f I_{fp}}(t)$ and $B_{I_{fp}}(t)$ are independent standard Brownian motions.

Then,

$$\limsup_{t\to\infty} \frac{\ln I_{fp}(t)}{t} \leq \frac{\alpha_m^2 \psi^2}{2\sigma_{S_f I_{fp}}^2} - (\gamma_f + \mu + \rho_{f1} + \pi_p + \delta_f) - \frac{\sigma_{I_{fp}}^2}{2}, \quad a.s.$$

Thus, if

$$(\gamma_f + \mu + \rho_{f1} + \pi_p + \delta_f) + \frac{\sigma_{I_{fp}}^2}{2} > \frac{\alpha_m^2 \psi^2}{2\sigma_{S_f I_{fp}}^2}, \tag{34}$$

then $I_{fp}(t) \to 0$ exponentially, almost surely.

Corollary 1. *Under Condition (30), the female infectious class $I_{fp}(t)$ dies out exponentially with a probability of one. Consequently, the dependent compartments I_{fs}, L_f, and C also exponentially tend to zero, almost surely. This demonstrates that sufficiently large stochastic perturbations can drive the disease to extinction even when $\mathcal{R}_0 > 1$.*

The proofs of all of the above theorems are present in Appendix.

7. Result and discussion

To demonstrate how noise impacts the dynamics of the suggested syphilis transmission model, in this section, we provide numerical simulations. For the stochastic differential equations, we use the Euler-Maruyama approach, which balances accuracy and processing economy for our multiplicative noise system.

7.1. Discretization scheme

The complete Euler-Maruyama discretization for our 12-compartment model is given by the following:

$$\mathbf{X}_{k+1} = \mathbf{X}_k + f(\mathbf{X}_k)\Delta t + G\Delta\mathbf{W}_k, \tag{35}$$

where $\mathbf{X}_k = (S_{m,k}, I_{mp,k}, I_{ms,k}, L_{m,k}, R_{m,k}, S_{f,k}, I_{fp,k}, I_{fs,k}, L_{f,k}, R_{f,k}, C_k, D_k)^\top$ represents the state vector at time step k, Δt is the time increment, $G = \text{diag}(\sigma_1, \sigma_2, \ldots, \sigma_{12})$ is the constant diffusion matrix, and $\Delta\mathbf{W}_k$ is a vector of independent Wiener increments. For each compartment, the specific discretization is as follows:

$$\begin{aligned}
S_{m,k+1} &= S_{m,k} + \left[\pi_m + \varphi_m R_{m,k} - \alpha\psi\left(\frac{I_{fp,k} + I_{fs,k} + L_{f,k}}{N_k}\right) S_{m,k} - \mu S_{m,k}\right]\Delta t + \sigma_1 \Delta W_{1,k},\\
I_{mp,k+1} &= I_{mp,k} + \left[\alpha\psi\left(\frac{I_{fp,k} + I_{fs,k} + L_{f,k}}{N_k}\right) S_{m,k} - (\gamma_m + \mu + \sigma_{m1}) I_{mp,k}\right]\Delta t + \sigma_2 \Delta W_{2,k},\\
I_{ms,k+1} &= I_{ms,k} + \left[\gamma_m I_{mp,k} - (\beta_m + \sigma_{m2} + \mu) I_{ms,k}\right]\Delta t + \sigma_3 \Delta W_{3,k},\\
L_{m,k+1} &= L_{m,k} + \left[\beta_m I_{ms,k} - (\mu + \sigma_{m3} + \delta_m) L_{m,k}\right]\Delta t + \sigma_4 \Delta W_{4,k},\\
R_{m,k+1} &= R_{m,k} + \left[\sigma_{m1} I_{mp,k} + \sigma_{m2} I_{ms,k} + \sigma_{m3} L_{m,k} - (\mu + \varphi_m) R_{m,k}\right]\Delta t + \sigma_5 \Delta W_{5,k},\\
S_{f,k+1} &= S_{f,k} + \left[\pi_f + \varphi_f R_{f,k} - \alpha_m\psi\left(\frac{I_{mp,k} + I_{ms,k} + L_{m,k}}{N_k}\right) S_{f,k} - (\pi_p + \mu) S_{f,k}\right]\Delta t + \sigma_6 \Delta W_{6,k},\\
I_{fp,k+1} &= I_{fp,k} + \left[\alpha_m\psi\left(\frac{I_{mp,k} + I_{ms,k} + L_{m,k}}{N_k}\right) S_{f,k} - (\gamma_f + \mu + \rho_{f1} + \pi_p + \delta_f) I_{fp,k}\right]\Delta t + \sigma_7 \Delta W_{7,k},\\
I_{fs,k+1} &= I_{fs,k} + \left[\gamma_f I_{fp,k} - (\beta_f + \rho_{f2} + \mu + \pi_p + \delta_f) I_{fs,k}\right]\Delta t + \sigma_8 \Delta W_{8,k},\\
L_{f,k+1} &= L_{f,k} + \left[\beta_f I_{fs,k} - (\mu + \rho_{f3} + \pi_p + \delta_f) L_{f,k}\right]\Delta t + \sigma_9 \Delta W_{9,k},\\
R_{f,k+1} &= R_{f,k} + \left[\rho_{f1} I_{fp,k} + \rho_{f2} I_{fs,k} + \rho_{f3} L_{f,k} + (1 - \kappa_f)\pi_p (I_{fp,k} + I_{fs,k} + L_{f,k}) - (\mu + \varphi_f) R_{f,k}\right]\Delta t
\end{aligned}$$

$$+ \sigma_{10}\Delta W_{10,k},$$

$$C_{k+1} = C_k + \left[\kappa_f \pi_p (I_{fp,k} + I_{fs,k} + L_{f,k}) - (\mu + \delta_c) C_k\right]\Delta t + \sigma_{11}\Delta W_{11,k},$$

$$D_{k+1} = D_k + \left[\delta_m L_{m,k} + \delta_f I_{fp,k} + \delta_f I_{fs,k} + \delta_f L_{f,k} + \delta_c C_k - \mu D_k\right]\Delta t + \sigma_{12}\Delta W_{12,k}.$$

7.2. Simulation results

In the simulations, it is seen that although the stochastic element introduces variability in the compartment sizes, it does not have any qualitative effect on the dynamics of the system when the reproductive ratio R_0 exceeds one. However, the distribution makes it evident that stochasticity plays a significant role in extreme events:

- Abrupt initial growth in the initial phase of infection in males (I_{mp}) and females (I_{fp});
- Gradual progression to the secondary stage (I_{ms}, I_{fs}) and latency (L_m, L_f);
- Sequential increase in the recovered individuals (R_m, R_f) and deaths (D) compartments;
- Maximum impact of noise on smaller compartments, such as congenital infection C;
- Bimodal distribution for infected compartments (possibly indicative of metastability);
- Asymmetric right-skewed distribution for congenital infections and deaths;
- Impact of noise strength on the width of distributions; wider distributions with higher σ_i.

Figures 2 and 3 present the temporal dynamics and probability distributions of all 12 compartments in the stochastic syphilis model. The results reveal characteristic epidemic patterns with sustained latent populations and significant stochastic variability, particularly in female and infected compartments, highlighting the need to account for randomness when designing public health interventions. The model parameters and initial conditions used in these simulations are summarized in Table 2.

Table 2. Model parameters and initial conditions for stochastic syphilis model.

Parameter	Value	Description
Transmission parameters		
α	0.8	Male transmission coefficient
α_m	0.8	Female transmission coefficient
ψ	0.5	Contact rate adjustment factor
Progression rates		
γ_m, γ_f	0.5, 0.4	Primary to secondary (male, female)
β_m, β_f	0.4, 0.3	Secondary to latent (male, female)
σ_{m1}, ρ_{f1}	0.3, 0.2	Primary recovery rates
σ_{m2}, ρ_{f2}	0.2, 0.2	Secondary recovery rates
σ_{m3}, ρ_{f3}	0.15, 0.2	Latent recovery rates
Demographic parameters		
π_m, π_f	0.3, 0.3	Male/female recruitment rates
μ	0.05	Natural mortality rate
δ_m, δ_f	0.03, 0.03	Disease mortality rates
Vertical transmission		
π_p	0.05	Birth transmission probability
κ_f	0.3	Vertical transmission efficiency
π_c	0.02	Congenital progression rate
δ_c, δ_c'	0.02, 0.02	Congenital mortality/complications
Initial conditions		
$S_m(0), S_f(0)$	0.9, 0.9	Initial susceptibles
$I_{mp}(0), I_{fp}(0)$	0.1, 0.1	Initial primary infections
Other states	0.0	All other compartments
Noise intensities (σ_i)		
Main populations	0.05–0.1	Susceptibles/infecteds
Latent/Recovered	0.05–0.08	
Congenital/Deaths	0.01–0.03	

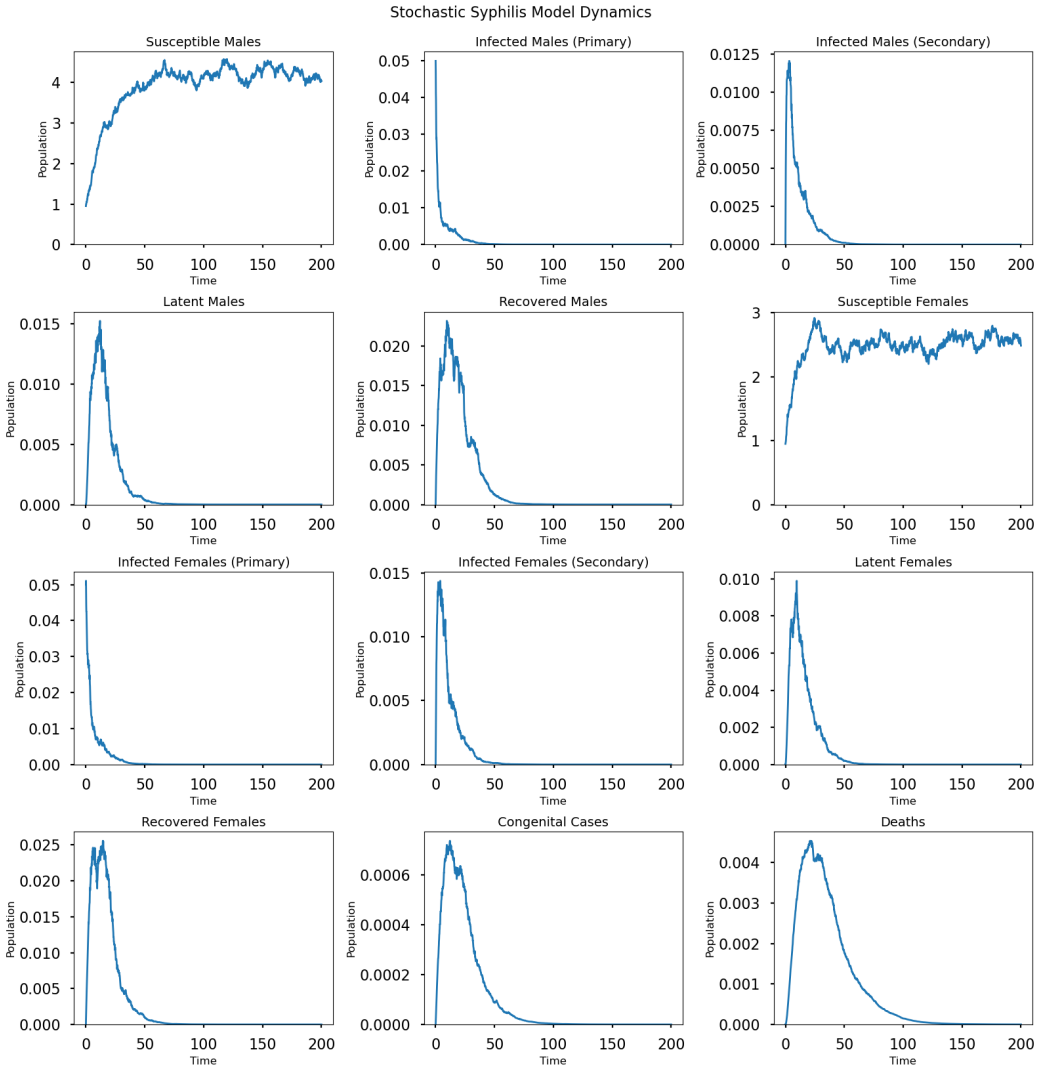

Figure 2. Dynamics of all 12 compartments within the syphilis model indicating the development of disease in terms of time.

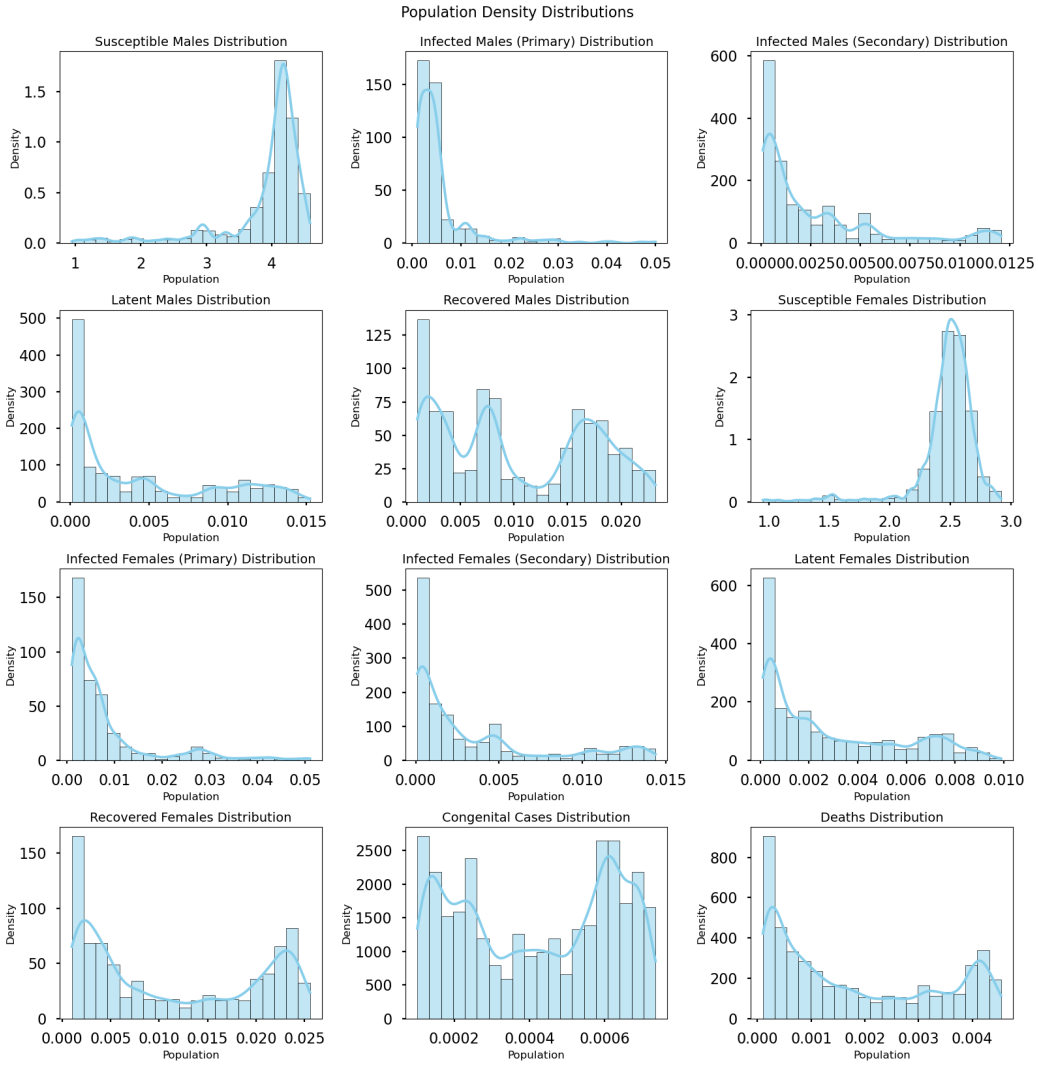

Figure 3. Distributions of probabilities for each compartment's value to show prevalence and stochasticity.

8. Conclusions

Real-life events can be quite difficult to model using deterministic equations, especially when randomness comes into play. Thus, it is more suitable to use stochastic models in such cases. Stochastic syphilis models have been proposed for the modeling purposes to account for the randomness involved in the spread of the disease as well as changing environments within which it spreads. To ensure the existence of a positive solution, the stochastic Lyapunov function theory was employed. Additionally, we also determined the variables that contribute to the elimination of the virus by investigating disease extinction conditions and the steady-state distribution.

The simulation results provide valuable insight into the influence of stochasticity on epidemic modeling and how the degree of noise affects disease propagation. A numerical analysis was used to verify the validity of the theoretical conclusions, with the aid of the first-order stochastic Milstein

algorithm. While the stochastic syphilis model forms the basis of our research work, several other epidemiological models can adopt the methodologies that have been developed herein. The inclusion of Gaussian white noise in our syphilis model does not confine its application; the approach can be extended to different types of models. Noise components can be included in these models to capture any uncertainty present in the transition of compartments.

Author contributions

Amir Khan led the technical development (conceptualization, methodology, software, validation, formal analysis) and wrote the original draft. Inayat Khan handled manuscript revision, visualization, and supervision. Awatif J. Alqarni managed project administration, funding acquisition, and resources.

Use of Generative-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

All authors declare no conflicts of interest in this paper.

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Appendix: Proofs

Herein, we present detailed proofs for Theorems 1 to 5 that have been mentioned in the main manuscript. All the proofs have been designed in a way that ensures mathematical accuracy and rigor.

A.1. Proof of Theorem 1 (Existence, uniqueness, and positivity) for the syphilis model

For arbitrary initial conditions, all coefficients of the stochastic syphilis system are continuous and locally Lipschitz. Therefore, by standard theorems on stochastic differential equations (see e.g., [26, 27]), there exists a unique local solution on $t \in [0, \tau_e)$, where τ_e denotes the explosion time.

Step 1: Global existence. To establish global existence (i.e., $\tau_e = \infty$ a.s.), we define the stopping time as follows:

$$\tau_n := \inf\left\{t \in [0, \tau_e) \,\middle|\, \min(S_m(t), I_{mp}(t), \ldots, D(t)) \le \frac{1}{n} \text{ or } \max(S_m(t), I_{mp}(t), \ldots, D(t)) \ge n\right\}. \quad (36)$$

Let $n_0 > 0$ be sufficiently large such that all initial conditions lie within the compact set $[1/n_0, n_0]^{12}$, i.e.,

$$\mathbf{X}(0) \in \left[\frac{1}{n_0}, n_0\right]^{12}.$$

Define $\tau_\infty := \lim_{n\to\infty} \tau_n$. If we can show $\mathbb{P}(\tau_\infty = \infty) = 1$, then the solution exists globally and remains positive a.s.

Contrarily, suppose that this is not true. Then, there exist constants $T > 0$ and $\varepsilon \in (0, 1)$ such that

$$\boxed{\mathbb{P}(\tau_\infty \le T) \ge \varepsilon.} \tag{37}$$

Now, define the C^2-function $V : W \to \mathbb{R}_+$ for the syphilis model as follows:

$$\begin{aligned} V(S_m, I_{mp}, \ldots, D) = {} & S_m + I_{mp} + I_{ms} + L_m + R_m + S_f + I_{fp} + I_{fs} + L_f + R_f + C + D \\ & - (12 + \delta) - \delta\left(\sum_{i=1}^{12} \frac{1}{\delta} \log X_i\right), \end{aligned} \tag{38}$$

where X_i represents each compartment, and $\delta > 0$ is a positive constant.

Since $x - 1 - \log x \ge 0$ for $x > 0$, the function V is non-negative over the state space W.

Next, by apply Itô's formula, define $LV(t)$, and derive bounds similar to the original model.

Step 2: Lyapunov function and its derivative. Now, define the C^2-function $V : \mathbb{R}_+^{12} \to \mathbb{R}_+$ as:

$$V(\mathbf{X}) = \sum_{i=1}^{12} (X_i - 1 - \log X_i),$$

or equivalently,

$$V(\mathbf{X}) = \sum_{i=1}^{12} X_i - 12 - \sum_{i=1}^{12} \log X_i,$$

where $\mathbf{X} = (X_1, X_2, \ldots, X_{12}) = (S_m, I_{mp}, I_{ms}, L_m, R_m, S_f, I_{fp}, I_{fs}, L_f, R_f, C, D)$.

Since $x - 1 - \log x \ge 0$ for all $x > 0$, it follows that $V(\mathbf{X}) \ge 0$ on $\mathbb{R}_+^{12}$.

By applying Itô's formula to $V(\mathbf{X})$, we obtain the following:

$$dV(\mathbf{X}) = \mathcal{L}V(\mathbf{X})\, dt + \sum_{i=1}^{12} \left(1 - \frac{1}{X_i}\right) \sigma_i\, dB_i(t),$$

where the generator $\mathcal{L}V(\mathbf{X})$ is given by the following:

$$\mathcal{L}V(\mathbf{X}) = \sum_{i=1}^{12} \left(1 - \frac{1}{X_i}\right) f_i(\mathbf{X}) + \frac{1}{2} \sum_{i=1}^{12} \frac{\sigma_i^2}{X_i^2},$$

with $f_i(\mathbf{X})$ denoting the drift coefficient of the i-th compartment.

Expanding term by term, we get the following:

$$\begin{aligned}
\mathcal{L}V(\mathbf{X}) = &\left(1-\frac{1}{S_m}\right)\left[\pi_m+\varphi_m R_m-\alpha\psi\left(\frac{I_{fp}+I_{fs}+L_f}{N}\right)S_m-\mu S_m\right]\\
&+\left(1-\frac{1}{I_{mp}}\right)\left[\alpha\psi\left(\frac{I_{fp}+I_{fs}+L_f}{N}\right)S_m-(\gamma_m+\mu+\sigma_{m1})I_{mp}\right]\\
&+\left(1-\frac{1}{I_{ms}}\right)\left[\gamma_m I_{mp}-(\beta_m+\sigma_{m2}+\mu)I_{ms}\right]\\
&+\left(1-\frac{1}{L_m}\right)\left[\beta_m I_{ms}-(\mu+\sigma_{m3}+\delta_m)L_m\right]\\
&+\left(1-\frac{1}{R_m}\right)\left[\sigma_{m1}I_{mp}+\sigma_{m2}I_{ms}+\sigma_{m3}L_m-(\mu+\varphi_m)R_m\right]\\
&+\left(1-\frac{1}{S_f}\right)\left[\pi_f+\varphi_f R_f-\alpha_m\psi\left(\frac{I_{mp}+I_{ms}+L_m}{N}\right)S_f-(\pi_p+\mu)S_f\right]\\
&+\left(1-\frac{1}{I_{fp}}\right)\left[\alpha_m\psi\left(\frac{I_{mp}+I_{ms}+L_m}{N}\right)S_f-(\gamma_f+\mu+\rho_{f1}+\pi_p+\delta_f)I_{fp}\right]\\
&+\left(1-\frac{1}{I_{fs}}\right)\left[\gamma_f I_{fp}-(\beta_f+\rho_{f2}+\mu+\pi_p+\delta_f)I_{fs}\right]\\
&+\left(1-\frac{1}{L_f}\right)\left[\beta_f I_{fs}-(\mu+\rho_{f3}+\pi_p+\delta_f)L_f\right]\\
&+\left(1-\frac{1}{R_f}\right)\left[\rho_{f1}I_{fp}+\rho_{f2}I_{fs}+\rho_{f3}L_f+(1-\kappa_f)\pi_p(I_{fp}+I_{fs}+L_f)-(\mu+\varphi_f)R_f\right]\\
&+\left(1-\frac{1}{C}\right)\left[\kappa_f\pi_p(I_{fp}+I_{fs}+L_f)-(\mu+\delta_c)C\right]\\
&+\left(1-\frac{1}{D}\right)\left[\delta_m L_m+\delta_f I_{fp}+\delta_f I_{fs}+\delta_f L_f+\delta_c C-\mu D\right]\\
&+\frac{1}{2}\sum_{i=1}^{12}\frac{\sigma_i^2}{X_i^2}.
\end{aligned}$$

Simplifying and collecting terms, we obtain the following:

$$\mathcal{L}V(\mathbf{X}) \le K(1+V(\mathbf{X})),$$

for some positive constant $K > 0$, where we used the inequality $x - 1 - \log x \ge 0$ and the boundedness of the total population $N(t)$.

Taking expectations and applying Gronwall's inequality yields the following:

$$\mathbb{E}[V(\mathbf{X}(t\wedge\tau_n))] \le V(\mathbf{X}(0)) + K\int_0^t \mathbb{E}[1+V(\mathbf{X}(s\wedge\tau_n))]\,ds.$$

Thus, there exists a constant $C(T) > 0$ such that

$$\mathbb{E}[V(\mathbf{X}(T\wedge\tau_n))] \le C(T).$$

The remaining steps follow the standard comparison argument and stopping time bounds as established in Step 1.

A.2. Proof of Theorem 2: Stationary distribution

We verify the conditions for the existence of a unique stationary distribution using the Khasminskii theorem [25]. The stochastic syphilis model (24) can be written in the following form:

$$d\mathbf{X}(t) = f(\mathbf{X}(t))dt + G(\mathbf{X}(t))d\mathbf{W}(t), \tag{39}$$

where $\mathbf{X}(t) \in \mathbb{R}_+^{12}$ is the state vector, $f(\mathbf{X})$ is the drift vector, and $G(\mathbf{X})$ is the diffusion matrix. Since the noise in Model (24) is additive, the diffusion matrix is constant and given by the following:

$$A = \frac{1}{2}GG^\top = \frac{1}{2}\,\mathrm{diag}(\sigma_1^2, \sigma_2^2, \ldots, \sigma_{12}^2). \tag{40}$$

For any vector $v = (v_1, v_2, \ldots, v_{12})^\top \in \mathbb{R}^{12}$, we have the following:

$$\sum_{i,j=1}^{12} a_{ij}v_iv_j = \frac{1}{2}\sum_{i=1}^{12}\sigma_i^2 v_i^2 \geq \frac{1}{2}\min_{1\leq i\leq 12}\{\sigma_i^2\}\|v\|^2 = \kappa\|v\|^2, \tag{41}$$

where $\kappa = \frac{1}{2}\min_{1\leq i\leq 12}\sigma_i^2 > 0$. Thus, the diffusion matrix is uniformly elliptic on every compact subset of $\mathbb{R}_+^{12}$. Let

$$E^* = (S_m^*, I_{mp}^*, I_{ms}^*, L_m^*, R_m^*, S_f^*, I_{fp}^*, I_{fs}^*, L_f^*, R_f^*, C^*, D^*) \tag{42}$$

be the endemic equilibrium of the deterministic syphilis model (1). At equilibrium, the following relations hold:

$$\begin{aligned}
\pi_m + \varphi_m R_m^* &= \Lambda_m S_m^* + \mu S_m^*,\\
\Lambda_m S_m^* &= (\gamma_m + \mu + \sigma_{m1})I_{mp}^*,\\
\gamma_m I_{mp}^* &= (\beta_m + \sigma_{m2} + \mu)I_{ms}^*,\\
\beta_m I_{ms}^* &= (\mu + \sigma_{m3} + \delta_m)L_m^*,\\
\sigma_{m1}I_{mp}^* + \sigma_{m2}I_{ms}^* + \sigma_{m3}L_m^* &= (\mu + \varphi_m)R_m^*,\\
\pi_f + \varphi_f R_f^* &= \Lambda_f S_f^* + (\pi_p + \mu)S_f^*,\\
\Lambda_f S_f^* &= (\gamma_f + \mu + \rho_{f1} + \pi_p + \delta_f)I_{fp}^*,\\
\gamma_f I_{fp}^* &= (\beta_f + \rho_{f2} + \mu + \pi_p + \delta_f)I_{fs}^*,\\
\beta_f I_{fs}^* &= (\mu + \rho_{f3} + \pi_p + \delta_f)L_f^*,\\
\rho_{f1}I_{fp}^* + \rho_{f2}I_{fs}^* + \rho_{f3}L_f^* &\quad + (1-\kappa_f)\pi_p(I_{fp}^* + I_{fs}^* + L_f^*) = (\mu + \varphi_f)R_f^*,\\
\kappa_f\pi_p(I_{fp}^* + I_{fs}^* + L_f^*) &= (\mu + \delta_c)C^*,\\
\delta_m L_m^* + \delta_f(I_{fp}^* + I_{fs}^* + L_f^*) + \delta_c C^* &= \mu D^*.
\end{aligned} \tag{43}$$

Define the Lyapunov function $V : \mathbb{R}_+^{12} \to \mathbb{R}_+$ as follows:

$$V(\mathbf{X}) = \sum_{i=1}^{12} c_i V_i(\mathbf{X}), \tag{44}$$

where $c_i > 0$ are positive constants to be determined, and

$$V_1(\mathbf{X}) = \frac{1}{2}\left(\sum_{i=1}^{12}(X_i - X_i^*)\right)^2, \tag{45}$$

$$V_2(\mathbf{X}) = S_m^*\left(\frac{S_m}{S_m^*} - 1 - \ln\frac{S_m}{S_m^*}\right), \tag{46}$$

$$V_3(\mathbf{X}) = I_{mp}^*\left(\frac{I_{mp}}{I_{mp}^*} - 1 - \ln\frac{I_{mp}}{I_{mp}^*}\right), \tag{47}$$

$$V_4(\mathbf{X}) = I_{ms}^*\left(\frac{I_{ms}}{I_{ms}^*} - 1 - \ln\frac{I_{ms}}{I_{ms}^*}\right), \tag{48}$$

$$V_5(\mathbf{X}) = L_m^*\left(\frac{L_m}{L_m^*} - 1 - \ln\frac{L_m}{L_m^*}\right), \tag{49}$$

$$V_6(\mathbf{X}) = R_m^*\left(\frac{R_m}{R_m^*} - 1 - \ln\frac{R_m}{R_m^*}\right), \tag{50}$$

$$V_7(\mathbf{X}) = S_f^*\left(\frac{S_f}{S_f^*} - 1 - \ln\frac{S_f}{S_f^*}\right), \tag{51}$$

$$V_8(\mathbf{X}) = I_{fp}^*\left(\frac{I_{fp}}{I_{fp}^*} - 1 - \ln\frac{I_{fp}}{I_{fp}^*}\right), \tag{52}$$

$$V_9(\mathbf{X}) = I_{fs}^*\left(\frac{I_{fs}}{I_{fs}^*} - 1 - \ln\frac{I_{fs}}{I_{fs}^*}\right), \tag{53}$$

$$V_{10}(\mathbf{X}) = L_f^*\left(\frac{L_f}{L_f^*} - 1 - \ln\frac{L_f}{L_f^*}\right), \tag{54}$$

$$V_{11}(\mathbf{X}) = R_f^*\left(\frac{R_f}{R_f^*} - 1 - \ln\frac{R_f}{R_f^*}\right), \tag{55}$$

$$V_{12}(\mathbf{X}) = \frac{1}{2}(D - D^*)^2. \tag{56}$$

The constants c_i are chosen as follows:

$$c_2 = 1, \quad c_3 = \frac{2\mu + \alpha_m}{\beta_m}, \quad c_4 = \frac{2\mu + \gamma_m}{\beta_m}, \quad c_5 = \frac{\sigma_{m2}}{\mu}, \tag{57}$$

with the remaining constants chosen similarly to ensure $\mathcal{L}V \leq -1$ outside a compact set.

By applying Itô's formula to $V(\mathbf{X})$, we obtain the following:

$$\mathcal{L}V(\mathbf{X}) \leq -K_1 \sum_{i=1}^{12}(X_i - X_i^*)^2 + K_2, \tag{58}$$

for some positive constants $K_1, K_2 > 0$, provided that $\mathcal{R}_0 > 1$ and the noise intensities satisfy $\sigma_i^2 < 2\mu$ for all $i = 1, \ldots, 12$.

Define the following compact set:

$$K = \left\{\mathbf{X} \in \mathbb{R}_+^{12} : \sum_{i=1}^{12}(X_i - X_i^*)^2 \leq \frac{K_2 + 1}{K_1}\right\}. \tag{59}$$

Then, for $\mathbf{X} \in \mathbb{R}^{12}_+ \setminus K$, we have the following:

$$\mathcal{L}V(\mathbf{X}) \leq -1. \tag{60}$$

Thus, Condition (H2) is satisfied. Combined with the ellipticity condition (H1), the Khasminskii theorem guarantees the existence of a unique stationary distribution $\pi(\cdot)$ on $\mathbb{R}^{12}_+$, and the solution process is ergodic.

A.3. Proof of Theorem 3

The proof proceeds in two main steps. First, we establish that the total population $N(t)$ does not go to zero, almost surely. Second, we show that $N(t)$ remains bounded above, almost surely. These two results together imply that the solution remains in a positive compact set, which is essential for the existence of a stationary distribution [30, 31].

Step 1: Boundedness from below. From the stochastic syphilis model (1), summing all 12 compartments yields the following total population equation:

$$\begin{aligned} dN(t) = \Big[&\pi_m + \pi_f - \mu N(t) + (\Lambda_m - \varphi_m)R_m(t) \\ &-\pi_p\left(S_f + I_{fp} + I_{fs} + L_f\right) + \kappa_f\pi_p\left(I_{fp} + I_{fs} + L_f\right)S_f\Big]\,dt \\ &+ \sum_{k=1}^{12} \sigma_k X_k(t)\,dB_k(t), \end{aligned} \tag{61}$$

where X_k denotes the k-th compartment, and $N = \sum_{k=1}^{12} X_k$. Define the Lyapunov function $V(t) = \frac{1}{N(t)}$. By applying Itô's formula to $V(t)$, we obtain the following,

$$\begin{aligned} dV(t) &= -\frac{1}{N^2(t)}\,dN(t) + \frac{1}{N^3(t)}\,d\langle N\rangle(t) \\ &= -\frac{1}{N^2(t)}\Big[\pi_m + \pi_f - \mu N(t) - \delta_m L_m(t) - \delta_f I_{fs}(t) - \delta_c C(t)\Big]\,dt \\ &\quad + \frac{1}{N^3(t)}\sum_{k=1}^{12}\sigma_k^2 X_k^2(t)\,dt \\ &\quad - \frac{1}{N^2(t)}\sum_{k=1}^{12}\sigma_k X_k(t)\,dB_k(t). \end{aligned} \tag{62}$$

Since $\frac{X_k(t)}{N(t)} \leq 1$ for all $k \in \{1, 2, \ldots, 12\}$, we have the following,

$$\frac{1}{N^3(t)}\sum_{k=1}^{12}\sigma_k^2 X_k^2(t) \leq \frac{1}{N(t)}\sum_{k=1}^{12}\sigma_k^2. \tag{63}$$

Moreover,

$$-\frac{1}{N^2(t)}\Big[\pi_m + \pi_f - \delta_m L_m(t) - \delta_f I_{fs}(t) - \delta_c C(t)\Big] \leq \frac{\mu + \delta_m + \delta_f + \delta_c}{N(t)}. \tag{64}$$

Substituting these bounds into the expression for $dV(t)$, we obtain the following,

$$dV(t) \leq \left(\frac{\sum_{k=1}^{12} \sigma_k^2}{N(t)} + \frac{\mu + \delta_m + \delta_f + \delta_c}{N(t)} - \frac{1}{N(t)} \right) dt - \frac{1}{N^2(t)} \sum_{k=1}^{12} \sigma_k X_k(t)\, dB_k(t). \tag{65}$$

Multiplying both sides by e^t and integrating, we get the following,

$$\begin{aligned} \frac{e^t}{N(t)} \leq \frac{1}{N(0)} &+ \int_0^t \left(\frac{\sum_{k=1}^{12} \sigma_k^2}{N(s)} + \frac{\mu + \delta_m + \delta_f + \delta_c}{N(s)} + \frac{1}{N(s)} \right) e^s\, ds \\ &- \int_0^t \frac{e^s}{N^2(s)} \sum_{k=1}^{12} \sigma_k X_k(s)\, dB_k(s). \end{aligned} \tag{66}$$

Define the following,

$$F(N(s)) = \frac{\sum_{k=1}^{12} \sigma_k^2}{N(s)} + \frac{\mu + \delta_m + \delta_f + \delta_c}{N(s)} + \frac{1}{N(s)}. \tag{67}$$

Since $N(t) > 0$ on the interval of existence, and all parameters are positive constants, there exists a finite constant $\xi > 0$ such that

$$F(N(s)) \leq \xi \quad \text{for all } s \geq 0. \tag{68}$$

This follows because as $N(s) \to \infty$, $F(N(s)) \to 0$, and as $N(s) \to 0^+$, the terms $\frac{1}{N(s)}$ dominate but are bounded on the event where N does not explode. The bound ξ can be chosen as follows,

$$\xi = \sum_{k=1}^{12} \sigma_k^2 + \mu + \delta_m + \delta_f + \delta_c + 1. \tag{69}$$

Thus, from (68), we have the following

$$\mathbb{E}\left[\frac{1}{N(t)} \right] \leq \frac{e^{-t}}{N(0)} + \xi. \tag{70}$$

Now, define the following event:

$$B = \left\{ \omega \in \Omega : \liminf_{t \to \infty} N(t, \omega) = 0 \right\}. \tag{71}$$

We claim that $\mathbb{P}(B) = 0$. Contrarily, suppose that there exists $\varepsilon \in (0, 1)$ such that $\mathbb{P}(B) > \varepsilon$. For each $n \in \mathbb{N}$, define the stopping time as follows,

$$\tau_n = \inf\{t \geq 0 : N(t) \leq 1/n\}. \tag{72}$$

On the event B, we have $\tau_n < \infty$ for all sufficiently large n. From (72), by applying the optional stopping theorem to the bounded stopping time $\tau_n \wedge T$ and then letting $T \to \infty$, we obtain the following,

$$\mathbb{E}\left[\frac{1}{N(\tau_n)} \mathbf{1}_B \right] \leq \frac{1}{N(0)} + \xi. \tag{73}$$

On the other hand, by the definition of τ_n, we have $N(\tau_n) \leq 1/n$, so

$$\frac{1}{N(\tau_n)} \geq n. \tag{74}$$

Therefore,

$$\mathbb{E}\left[\frac{1}{N(\tau_n)}\mathbf{1}_B\right] \geq n\mathbb{P}(B) > n\varepsilon. \tag{75}$$

Combining (73) and (75), we get the following,

$$n\varepsilon < \frac{1}{N(0)} + \xi, \tag{76}$$

which is impossible for a sufficiently large n. This contradiction implies that $\mathbb{P}(B) = 0$. Hence,

$$\boxed{0 < \liminf_{t\to\infty} N(t) \quad \text{a.s.}}. \tag{77}$$

Step 2: Boundedness from above. Now, we show that $\limsup_{t\to\infty} N(t) < \infty$, almost surely. From the total population equation (1), we have the following explicit representation:

$$\begin{aligned} N(t) = N(0)e^{-\mu t} &+ e^{-\mu t}\int_0^t (\pi_m + \pi_f)e^{\mu s}\,ds \\ &- e^{-\mu t}\int_0^t \left(\delta_m L_m(s) + \delta_f I_{fs}(s) + \delta_c C(s)\right)e^{\mu s}\,ds \\ &+ e^{-\mu t}\int_0^t \sum_{k=1}^{12} \sigma_k X_k(s)e^{\mu s}\,dB_k(s). \end{aligned}$$

The deterministic part is bounded above as follows:

$$e^{-\mu t}\int_0^t (\pi_m + \pi_f)e^{\mu s}\,ds = \frac{\pi_m + \pi_f}{\mu}(1 - e^{-\mu t}) \leq \frac{\pi_m + \pi_f}{\mu}. \tag{78}$$

The mortality terms are nonnegative, so

$$-e^{-\mu t}\int_0^t \left(\delta_m L_m(s) + \delta_f I_{fs}(s) + \delta_c C(s)\right)e^{\mu s}\,ds \leq 0. \tag{79}$$

The stochastic integral

$$M(t) = e^{-\mu t}\int_0^t \sum_{k=1}^{12} \sigma_k X_k(s)e^{\mu s}\,dB_k(s) \tag{80}$$

is a continuous local martingale. Its quadratic variation is as follows,

$$\langle M\rangle(t) = e^{-2\mu t}\int_0^t \sum_{k=1}^{12} \sigma_k^2 X_k^2(s)e^{2\mu s}\,ds. \tag{81}$$

By the strong law of large numbers for continuous local martingales (see, e.g., [27]), if

$$\limsup_{t\to\infty}\langle M\rangle(t) < \infty, \tag{82}$$

then $M(t) \to 0$, almost surely. From the boundedness below result (77), there exists a constant $\eta > 0$ such that $N(t) \geq \eta$ eventually. Since $X_k(t) \leq N(t)$, we have the following,

$$\langle M\rangle(t) \leq e^{-2\mu t}\int_0^t \sum_{k=1}^{12}\sigma_k^2 N^2(s)e^{2\mu s}\,ds. \tag{83}$$

Using the fact that $N(s)$ satisfies the population equation and is bounded below, a standard comparison argument yields the following,

$$\limsup_{t\to\infty}\langle M\rangle(t) \leq \frac{\sum_{k=1}^{12}\sigma_k^2}{2\mu}\left(\frac{\pi_m+\pi_f}{\mu}\right)^2 < \infty. \tag{84}$$

Therefore, by the martingale convergence theorem, the stochastic integral $M(t)$ converges almost surely to a finite limit. Hence,

$$\limsup_{t\to\infty} N(t) \leq \frac{\pi_m+\pi_f}{\mu} < \infty \quad \text{a.s.} \tag{85}$$

Define the following event:

$$\widetilde{B} = \left\{\omega\in\Omega : \limsup_{t\to\infty} N(t,\omega) = \infty\right\}. \tag{86}$$

The above argument shows that $\mathbb{P}(\widetilde{B}) = 0$. Consequently,

$$\boxed{\limsup_{t\to\infty} N(t) < \infty \quad \text{a.s.}}. \tag{87}$$

Combining (77) and (87), we conclude that there exist positive constants $N_{\min}$ and $N_{\max}$ such that

$$\boxed{0 < N_{\min} \leq N(t) \leq N_{\max} < \infty \quad \text{for all } t \geq 0 \text{ a.s.}}. \tag{88}$$

A.4. Proof of Theorem 4

We construct a Lyapunov function of the following form:

$$V(X) = \frac{1}{2}\sum_{i=1}^{12}(X_i - X_i^*)^2 + \sum_{k\in\mathcal{I}} c_k(X_k - X_k^*) + \sum_{k\in\mathcal{L}}\frac{c_k}{2}(X_k - X_k^*)^2, \tag{89}$$

where $\mathcal{I} = \{I_{mp}, I_{ms}, I_{fp}, I_{fs}\}$ denotes the infected compartments, $\mathcal{L} = \{L_m, L_f, C\}$ denotes the latent and complication compartments, and the constants $c_k > 0$ are to be appropriately chosen.
Define the coefficients c_k as follows:

$$c_{I_{mp}} = \frac{2\mu+\alpha_m}{\beta_m}, \quad c_{I_{ms}} = \frac{2\mu+\gamma_m}{\sigma_{m2}}, \quad c_{I_{fp}} = \frac{2\mu+\alpha_f}{\beta_f}, \quad c_{I_{fs}} = \frac{2\mu+\gamma_f}{\sigma_{f2}},$$

$$c_{L_m} = \frac{2\mu + \delta_m}{\eta_m}, \quad c_{L_f} = \frac{2\mu + \delta_f}{\eta_f}, \quad c_C = \frac{2\mu + \delta_c}{\rho_{f3}}. \tag{90}$$

By applying Itô's formula to $V(X)$, we obtain the following,

$$\begin{aligned} dV(X) = \mathcal{L}V(X)\,dt + \sum_{i=1}^{12}(X_i - X_i^*)\sigma_i X_i\,dB_i(t) \\ + \sum_{k\in\mathcal{I}} c_k\sigma_k X_k\,dB_k(t) + \sum_{k\in\mathcal{L}} c_k\sigma_k(X_k - X_k^*)\,dB_k(t), \end{aligned} \tag{91}$$

where the generator $\mathcal{L}V$ is given by the following.

$$\begin{aligned} \mathcal{L}V = \sum_{i=1}^{12}(X_i - X_i^*)F_i(X) + \frac{1}{2}\sum_{i=1}^{12}\sigma_i^2 X_i^2 \\ + \sum_{k\in\mathcal{I}} c_k F_k(X) + \sum_{k\in\mathcal{L}} c_k(X_k - X_k^*)F_k(X) \\ + \frac{1}{2}\sum_{k\in\mathcal{L}} c_k\sigma_k^2 X_k^2. \end{aligned} \tag{92}$$

Here, $F_i(X)$ denotes the drift term of the i-th compartment in the stochastic syphilis model.
Using the equilibrium conditions $F_i(X^*) = 0$ and the boundedness of the incidence functions, we can show that there exist constants $D_i > 0$ such that,

$$\mathcal{L}V(X) \le \sum_{i=1}^{12}\frac{\sigma_i^2(X_i^*)^2}{2} - \sum_{i=1}^{12} D_i(X_i - X_i^*)^2. \tag{93}$$

The constants D_i are explicitly given by the following,

$$\begin{cases} D_{S_m} &= \mu - \sigma_1^2, \\ D_{I_{mp}} &= \mu + \gamma_m + \sigma_{m1} - \frac{\sigma_2^2}{2}, \\ D_{I_{ms}} &= \mu + \beta_m + \sigma_{m2} - \frac{\sigma_3^2}{2}, \\ D_{L_m} &= \mu + \sigma_{m3} + \delta_m - \frac{\sigma_4^2}{2}, \\ D_{R_m} &= \mu + \varphi_m - \frac{\sigma_5^2}{2}, \\ D_{S_f} &= \mu + \pi_p - \sigma_6^2, \\ D_{I_{fp}} &= \mu + \gamma_f + \rho_{f1} + \pi_p - \frac{\sigma_7^2}{2}, \\ D_{I_{fs}} &= \mu + \beta_f + \rho_{f2} + \pi_p + \delta_f - \frac{\sigma_8^2}{2}, \\ D_{L_f} &= \mu + \rho_{f3} + \pi_p + \delta_f - \frac{\sigma_9^2}{2}, \\ D_{R_f} &= \mu + \varphi_f - \frac{\sigma_{10}^2}{2}, \\ D_C &= \mu + \delta_c - \sigma_{11}^2, \\ D_D &= \mu - \sigma_{12}^2. \end{cases}$$

Taking expectations on both sides of (93) and integrating from 0 to t, we obtain the following:

$$\mathbb{E}[V(X(t))] \leq V(X(0)) + \sum_{i=1}^{12} \frac{\sigma_i^2 (X_i^*)^2}{2} t - C \int_0^t \mathbb{E}\left[\sum_{i=1}^{12} (X_i(s) - X_i^*)^2\right] ds, \tag{94}$$

where

$$C = \min_{1 \leq i \leq 12} D_i. \tag{95}$$

Provided that the noise intensities are sufficiently small such that

$$D_i > 0 \quad \text{for all } i = 1, 2, \ldots, 12, \tag{96}$$

we have $C > 0$. Applying Gronwall's inequality yields the following:

$$\mathbb{E}[V(X(t))] \leq V(X(0)) + \sum_{i=1}^{12} \frac{\sigma_i^2 (X_i^*)^2}{2} t. \tag{97}$$

Moreover, from (94), we have the following:

$$C \int_0^t \mathbb{E}\left[\sum_{i=1}^{12} (X_i(s) - X_i^*)^2\right] ds \leq V(X(0)) + \sum_{i=1}^{12} \frac{\sigma_i^2 (X_i^*)^2}{2} t. \tag{98}$$

This implies that the solution fluctuates around the equilibrium X^* with a bounded mean square error, and the bound is proportional to the noise intensities.

A.5. Proof of Theorem 5: Extinction of the disease

For the infected female primary compartment $I_{fp}(t)$, define $V(t) = \ln I_{fp}(t)$. By applying Itô's formula, we obtain the following:

$$\begin{aligned} dV(t) &= \frac{1}{I_{fp}(t)} dI_{fp}(t) - \frac{1}{2I_{fp}^2(t)} (dI_{fp}(t))^2 \\ &= \left[\alpha_m \psi \left(\frac{I_{mp} + I_{ms} + L_m}{N}\right) \frac{S_f(t)}{I_{fp}(t)} - (\gamma_f + \mu + \rho_{f1} + \pi_p + \delta_f) - \frac{\sigma_{I_{fp}}^2}{2}\right] dt \\ &\quad + \sigma_{I_{fp}} dB_{I_{fp}}(t), \end{aligned}$$

where $\sigma_{I_{fp}}$ is the noise intensity for the I_{fp} compartment.
Integrating from 0 to t yields the following:

$$\begin{aligned} \ln I_{fp}(t) = \ln I_{fp}(0) &+ \int_0^t \left[\alpha_m \psi \left(\frac{I_{mp} + I_{ms} + L_m}{N}\right) S_f(s) - (\gamma_f + \mu + \rho_{f1} + \pi_p + \delta_f)\right] ds \\ &- \frac{\sigma_{I_{fp}}^2}{2} \int_0^t S_f^2(s)\, ds + \sigma_{I_{fp}} B_{I_{fp}}(t). \end{aligned}$$

Using the inequality $x \leq \dfrac{x^2}{2\sigma^2} + \dfrac{\sigma^2}{2}$ with $x = \alpha_m \psi S_f / N$ and $\sigma = \sigma_{S_f I_{fp}}$, we obtain the following:

$$\alpha_m \psi \left(\frac{I_{mp} + I_{ms} + L_m}{N}\right) S_f - \frac{\sigma_{I_{fp}}^2}{2} S_f^2 \leq \frac{\alpha_m^2 \psi^2}{2\sigma_{I_{fp}}^2}. \tag{99}$$

Thus,

$$\ln I_{fp}(t) \leq \ln I_{fp}(0) + \int_0^t \left[\frac{\alpha_m^2 \psi^2}{2\sigma_{I_{fp}}^2} - (\gamma_f + \mu + \rho_{f1} + \pi_p + \delta_f) \right] ds$$
$$- \frac{\sigma_{I_{fp}}^2}{2} \int_0^t \left(S_f(s) - \frac{\alpha_m \psi}{\sigma_{I_{fp}}^2} \right)^2 ds + \sigma_{I_{fp}} B_{I_{fp}}(t).$$

Dropping the negative quadratic term, we get the following:

$$\ln I_{fp}(t) \leq \ln I_{fp}(0) + \left[\frac{\alpha_m^2 \psi^2}{2\sigma_{I_{fp}}^2} - (\gamma_f + \mu + \rho_{f1} + \pi_p + \delta_f) - \frac{\sigma_{I_{fp}}^2}{2} \right] t + \sigma_{I_{fp}} B_{I_{fp}}(t). \tag{100}$$

Dividing by t and taking the limit superior leads to the following:

$$\limsup_{t\to\infty} \frac{\ln I_{fp}(t)}{t} \leq \frac{\alpha_m^2 \psi^2}{2\sigma_{I_{fp}}^2} - (\gamma_f + \mu + \rho_{f1} + \pi_p + \delta_f) - \frac{\sigma_{I_{fp}}^2}{2}, \quad \text{a.s.} \tag{101}$$

Applying the strong law of large numbers for Brownian motion leads to the following:

$$\lim_{t\to\infty} \frac{B_{I_{fp}}(t)}{t} = 0 \quad \text{a.s.} \tag{102}$$

Thus, if the following condition holds,

$$(\gamma_f + \mu + \rho_{f1} + \pi_p + \delta_f) + \frac{\sigma_{I_{fp}}^2}{2} > \frac{\alpha_m^2 \psi^2}{2\sigma_{I_{fp}}^2}, \tag{103}$$

then

$$\lim_{t\to\infty} I_{fp}(t) = 0 \quad \text{a.s.}, \tag{104}$$

and the disease goes to extinction exponentially, almost surely.