Approach Bayesian to estimate the effect of HIV Prevention Programs among Females Sex Workers in Burkina Faso

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Abstract

Keywords :

1 Introduction

More than three decades after the first reported case of AIDS, sub-Saharan Africa remains the most affected region in the world [1]. In 2019, adolescent girls and young women (aged 15-24) in this region alone accounted for 24% of new HIV infections, more than double their proportion of 10% of the population [2]. The epidemic is most prevalent among key populations (KP), including women and men who sell or exchange sex, men who have sex with men, people who inject drugs, transgender women who have sex with men, and individuals in prison [3]. Stigmatised and marginalised, key populations account for the vast majority of new infections in West, North and Central Africa, and about 25% of new infections in East and Southern Africa, although they represent relatively small proportions of all these populations [1]. In 2020, the risk of HIV infection was 25 times higher among men who have sex with men than among heterosexual men; 26 times higher among female sex workers (SWs) than among women in the general population; 34 times higher among transgender women than among other adults; and 35 times higher among people who inject drugs than among people who do not inject drugs [4]. In West Africa, sex workers remain the main group involved in the dynamics of HIV transmission. More than 75% of HIV infections among heterosexual men are attributed to sex with sex workers [5]. The main strategy to combat HIV remains the use of antiretroviral therapy (ART). Nevertheless, HIV prevention methods including pre-exposure prophylaxis (PrEP), circumcision and the use of condoms have been associated with ARVs to contain the disease [2, 6]. Since 2001, HIV incidence has declined by more than 25% in 22 sub-Saharan African countries [7]. However, more needs to be done to reduce HIV transmission among key populations that continue to fuel the spread of HIV, threatening progress in the fight against the disease. Understanding the dynamics of HIV transmission in this specific population would allow for effective and optimal strategies to prevent and control the spread of HIV.

Mathematical modelling is an important tool for understanding epidemics and for providing a rational basis

for evaluating policies to control the spread of a disease [6]. Thus, we find in the literature various mathematical models both to understand the dynamics of the spread of HIV and to measure the effect of intervention programmes on it [8–16]. In Kenya, for example, Omondi et al. developed a mathematical model of HIV transmission between sex workers and injecting drug users to assess the effect of combined PrEP, ARVs on the spread of HIV [6]. In Ivory Coast, Maheu-Giroux et al. developed an age-stratified dynamic model of sexual and vertical transmission of HIV among the general population, TS and men who have sex with men. Their model was calibrated on detailed prevalence and intervention data (ARVs and condoms) [17]. In Benin, Geidelberg et al. looked at the role of pre-exposure prophylaxis on the spread of HIV among PWs in the city of Cotonou. The authors also used a compartmental model involving PrEP and antiretroviral therapy (ARV) in high-risk (HCV and clients) and low-risk populations [18].

In Burkina Faso, the government has made the fight against HIV, AIDS and STIs a major challenge. The 2018 serosurveillance report gives an overall HIV prevalence of 1.2 % [1.0-1.4] compared to 1.3 % [1.0-1.5] among 15-49 year olds at the end of 2017. Among key populations, numerous studies have been set up to study the prevalence and incidence of HIV among MSM and TS [19–25]. Concerning TS, Low et al. [19] develop for the first time in 2015, a deterministic compartmental model to evaluate the impact of condom and ARV use on HIV incidence among TS and their clients. The model used had eight bins to account for ARV treatment of HIV-infected individuals and the study population was stratified by gender. The impact of the interventions was assessed in terms of proportion of infections averted and years of life gained by comparing a status quo (no intervention) group with an intervention group. The status quo group was constituted with reference to the prevalence of the disease between the years 1975 and 1980, the date of the first recorded case of HIV in Burkina Faso. Some parameters of the model were estimated from a quasi-Bayesian framework [19]. Since the introduction of PrEP as an HIV prevention method, there are no detailed mathematical models that take into account HIV transmission between sex workers, their clients and the inter-group mobility of these individuals. However, it is common practice for sex workers to engage in unprotected sex with clients in order to get more money or for the condom to break during sex. It is therefore important to measure the contribution of this new strategy to the spread of HIV.

The main objective of this work was to estimate the impact of the PrEP/Condom strategy on the spread of HIV among HCVs and their clients through a comparative study of this combination against the standard of care that existed at the time. This evaluation was assessed in terms of new infections averted by the strategy. A secondary objective was to explore the impact of the PrEP/Condoms intervention on different categories of STs and clients. A Bayesian supported Susceptible-Infected compartmental model will be developed to answer this question. This model will be built according to the key stages of the evolution of the epidemic and the risk behaviour of the study population. All the parameters of the model have been estimated by the Markov Chain Monte Carlo (MCMC) estimation method, contrary to classical ODE methods; this has the advantage of minimising the uncertainty of the parameters related to the epidemic [26].

The work is organized as follows: In section 2, the model describing HIV transmission between sex workers and their clients is formulated. Section 3 concerns the mathematical study of the proposed model. More precisely, in this section, we proved the well-posedness of the model, we computed the disease-free equilibrium point and the basic reproduction number that has been shown to be the key threshold parameter in investigating the disease dynamics. Moreover, thanks to certain conditions on $\mathcal{R}_0(\mathcal{R}_0 < 1 or \mathcal{R}_0 > 0)$, to the Varga theorem and

to Lyapunov's function principle [27, 28], we have studied the local and global stabilities of the steady states; which ensures the validity and robustness of the model. This model was then used to build the Bayesian models. We devoted Section 4 to the numerical simulations. The paper ends with a conclusion section 5 where we give recommendations and perspectives.

2 Model formulation

The proposed approach is largely inspired by the work of Low et al.[19] and Wang et al.[29]. Disease dynamics are captured in an Ordinary Derivative Equation (ODE) Susceptible-Infected system with Bayesian support. In the first model, the study population is divided into three compartments consisting of those susceptible to each stage of infection or AIDS. S_C refers to HIV susceptible men (respectively S_{SW} HIV susceptible SWs), I_C to HIV infected men (respectively I_{SW} to HIV infected SWs) and A_C to AIDS infected men (respectively A_{SW} to AIDS infected SW) The diagram and the interactions between compartments are described in Figure 2.

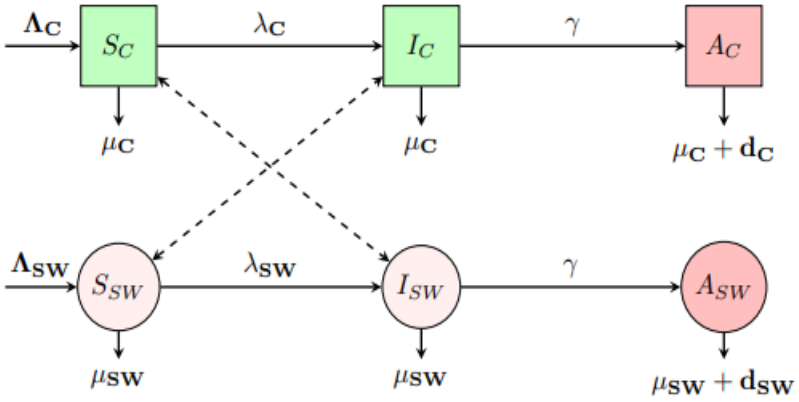

Figure 1: A compartmental representation of the pattern of HIV transmission between TS and their clients. Dotted lines indicate transitions between risk groups, while solid lines indicate movement of individuals in the same risk group.

The population sizes at time t for the two risk groups for the first model are given by :

$$\mathrm{N_C(t) = S_C(t) + I_C(t) + A_C(t), \qquad N_{SW}(t) = S_{SW}(t) + I_{SW}(t) + A_{SW}(t).}$$

The total population size at time t is the sum of the population sizes of the two risk groups at time t, and is given by $\mathrm{N(t) = N_C(t) + N_{SW}(t)}$. In the model, clients and SW are likely to become infected at different infection rates given by :

$$\lambda_C = \beta_C \theta_{SW} p_{SW}, \qquad \lambda_{SW} = \beta_{SW} \theta_C p_C, \qquad \theta_C = \frac{I_C}{N_C}, \qquad \theta_{SW} = \frac{I_{SW}}{N_{SW}}$$

In the case of the use of HIV prevention methods by one and/or both partners, the risk of transmission of the disease when a person at risk meets an infected person is modified as follows :

$$\lambda_C = \pi \beta_C \theta_{SW} p_{SW}, \qquad \lambda_{SW} = \pi \beta_{SW} \theta_C p_C$$

Where π represents the intervention package available to TS.The full list of model parameters and other assumptions are described in the table opposite
(Table 1) :

Table 1: Description of parameters used in the model.

Symbol	Description
N	Population of people entering sexually active population who enter each risk group
μ	Death rate based on life expectency at age 15
α	Population growth factor
Λ	Recruitment rate on each group
p	HIV transmission probabilities through sex
γ	Duration taken to move from asymptomatic stage to AIDS
λ	Force of infection following the sex and risk group
θ	Disease prevalence by the gender
d	AIDS death rates by the gender

Following the above description, the following nonlinear ordinary differential equation are obtained

$$\begin{cases} \frac{\mathrm{d}S_C}{\mathrm{d}t} &= \Lambda_C - (\lambda_C + \mu_C)S_C, \\ \frac{\mathrm{d}I_C}{\mathrm{d}t} &= \lambda_C S_C - (\mu_C + \gamma)I_C, \\ \frac{\mathrm{d}A_C}{\mathrm{d}t} &= \gamma I_C - (\mu_C + d_C)A_C. \end{cases} \tag{1}$$

$$\begin{cases} \frac{\mathrm{d}S_{SW}}{\mathrm{d}t} &= \Lambda_{SW} - (\lambda_{SW} + \mu_{SW})S_{SW}, \\ \frac{\mathrm{d}I_{SW}}{\mathrm{d}t} &= \lambda_{SW} S_{SW} - (\mu_{SW} + \gamma)I_{SW}, \\ \frac{\mathrm{d}A_{SW}}{\mathrm{d}t} &= \gamma I_{SW} - (\mu_{SW} + d_{SW})A_{SW}. \end{cases} \tag{2}$$

The system of equations in (1)–(2) is subject to the following initial conditions, $S_C(0) \geq 0$; $I_C(0) \geq 0$; $A_C(0) \geq 0$; $S_{SW}(0) \geq 0$; $I_{SW}(0) \geq 0$; $A_{SW}(0) \geq 0$.

3 Models Analysis

3.1 Boundedness and Positivity of solutions of the model

Lemma 3.1 *The domain* Δ *defined by*

$$\Delta := \left\{ (S_C, I_C, A_C, S_{SW}, I_{SW}, A_{SW}) \in \mathbb{R}^6_+ : S_C + I_C + A_C + S_{SW} + I_{SW} + A_{SW} \leq \frac{\Lambda_C + \Lambda_{SW}}{\mu} \right\} \tag{3}$$

is bounded

Proof : From eq (1)-(2), we can write

$$\frac{\mathrm{d}N_C}{\mathrm{d}t} = \Lambda_C - \mu_C N_C - d_C A_C \leqslant \Lambda_C - \mu_C N_C \tag{4}$$

$$\frac{\mathrm{d}N_{SW}}{\mathrm{d}t} = \Lambda_{SW} - \mu_{SW} N_{SW} - d_{SW} A_{SW} \leqslant \Lambda_{SW} - \mu_{SW} N_{SW} \tag{5}$$

In doing (4)+(5), we get

$$\frac{\mathrm{d}N}{\mathrm{d}t} = \Lambda_C + \Lambda_{SW} - (\mu_C N_C + \mu_{SW} N_{SW}) \tag{6}$$

If we let $\mu = min\{\mu_C, \mu_{SW}\}$, It can easily be shown that

$$N_C \leqslant \frac{\Lambda_C}{\mu_C} + (N_{C0} - \frac{\Lambda_C}{\mu_C})e^{-\mu_C t} \quad where \quad N_{C0} = N_C(0) \tag{7}$$

$$N_{SW} \leqslant \frac{\Lambda_{SW}}{\mu_{SW}} + (N_{SW0} - \frac{\Lambda_{SW}}{\mu_{SW}})e^{-\mu_{SW} t} \quad where \quad N_{SW0} = N_{SW}(0) \tag{8}$$

From (7) & (8), we observe that as $t \to \infty$, $N(t) \to \frac{\Lambda}{\mu}$. So if $N_0 \leqslant \frac{\Lambda}{\mu}$ then $\lim_{t\to+\infty} N(t) = \frac{\Lambda}{\mu}$. Clearly, $\frac{\Lambda}{\mu}$ is the upper bound of N. On the other hand, if $N_0 > \frac{\Lambda}{\mu}$, then n will decrease to $\frac{\Lambda}{\mu}$ as $t \to \infty$. This means that if $N_0 > \frac{\Lambda}{\mu}$, then the solution $(S_C(t), I_C(t), A_C(t), S_{SW}(t), I_{SW}(t), A_{SW}(t))$ enters Δ or approaches it asymptotically. Hence Δ is positively invariant under the flow induced by system (1)-(2). Which completes the demonstration.

Lemma 3.2 *For any strictly positive initial condition* $\Big(S_C(0), I_C(0), A_C(0), S_{SW}(0), I_{SW}(0), A_{SW}(0)\Big)$*, the system (1)-(2) has positive solutions.*

Proof : To prove this, we will proceed by the absurd.
Let $e(t)$ be such as $e(t) = \min\Big\{S_C(t), I_C(t), A_C(t)\Big\}$, $\forall t \geq 0$
Let assum there exists $t_1 > 0$ such that $e(t_1) \notin \mathbb{R}_+^*$ and $e(t) > 0\ \forall t \in [0, t_1)$.
If $e(t) = I_C(t)$, then from the second equation of the system (1), we have

$$\dot{I}_C > -(\mu_C + \gamma)I_C$$

It follows that

$$0 = I_C(t) > I_C(0)\exp\Big[-(\mu_C + \gamma)t_1\Big] > 0,$$

Which is a contradiction.
If $e(t) = A_C(t)$, then from the third equation of the system (1), we have

$$\dot{A}_C > -(\mu_C + d_C)A_C$$

It follows that

$$0 = A_C(t) > A_C(0)\exp\Big[-(\mu_C + d_C)t_1\Big] > 0,$$

Which is a contradiction.
If $e(t) = S_C(t)$, then from the first equation of the system (1), we have

$$\dot{S}_C > -(\lambda_C + \mu_C)A_C$$

It follows that

$$0 = S_C(t) > S_C(0)\Big[\exp(-\mu_C t_1) + \beta_C \theta_C p_{SW} \exp\Big(-\int_0^{t_1} I_{SW}(t)dt\Big)\Big] > 0,$$

Which is a contradiction.

Thus, proceeding in the same way for system (2) leads to a contradiction.

Therefore, all the solutions (1) – (2) of the dynamic model of differential equations are positive.

3.2 Free Equilibrium point and Endemic Equilibrium

The system (1) – (2) has the following four (4) equilibrium points defined like that

$$\varepsilon_0 = \Big\{S_C^0, 0, 0, S_{SW}^0, 0, 0\Big\}, \tag{9}$$

$$\varepsilon_1 = \Big\{S_C^*, I_C^*, A_C^*, S_{SW}^*, 0, 0\Big\}, \tag{10}$$

$$\varepsilon_2 = \Big\{S_C^*, 0, 0, S_{SW}^*, I_{SW}^*, A_{SW}^*\Big\}, \tag{11}$$

$$\varepsilon_3 = \Big\{S_C^*, I_C^*, A_C^*, S_{SW}^*, I_{SW}^*, A_{SW}^*\Big\}. \tag{12}$$

Note that ε_0 refers to the HIV-free equilibrium whereas ε_1 and ε_2 refer to the first and second boundary endemic equilibria. On the other hand,ε_3 defines the interior endemic equilibrium in the domain Δ. The HIV-free equilibrium ε_1 in the two risk populations is obtained from the systems (1) – (2) which reduces to

$$\begin{cases} \frac{\mathrm{d}S_C}{\mathrm{d}t} &= \Lambda_C - \mu_C S_C \\ \frac{\mathrm{d}S_{SW}}{\mathrm{d}t} &= \Lambda_{SW} - \mu_{SW} S_{SW} \end{cases} \tag{13}$$

Setting the right-hand side of the system (13) to zero and solving, we obtain

$$\begin{aligned} S_C^0 &= \frac{\Lambda_C}{\mu_C}, \\ S_{SW}^0 &= \frac{\Lambda_{SW}}{\mu_{SW}}, \\ S_C^* &= \frac{\Lambda_C - (\mu_C + \gamma) I_C^*}{\mu_C}, \\ S_{SW}^* &= \frac{\Lambda_{SW} - (\mu_{SW} + \gamma) I_{SW}^*}{\mu_{SW}}, \\ A_C^* &= \frac{\gamma}{\mu_C + d_C} I_C^*, \\ A_{SW}^* &= \frac{\gamma}{\mu_{SW} + d_{SW}} I_{SW}^*. \end{aligned}$$

We also make this assumption :

for all **H:** $\mathrm{I_C}, \mathrm{A_C}, \mathrm{I_{SW}}, \mathrm{A_{SW}} \in \mathbb{R}_+$, $\begin{pmatrix} \mathrm{I_C} \\ \mathrm{A_C} \\ \mathrm{I_{SW}} \\ \mathrm{A_{SW}} \end{pmatrix} \leq \begin{pmatrix} \mathrm{I_C^*} \\ \mathrm{A_C^*} \\ \mathrm{I_{SW}^*} \\ \mathrm{A_{SW}^*} \end{pmatrix}$

3.3 The basic reproduction number $\mathcal{R}_0$

The Basic Reproduction Number $\mathcal{R}_0$ for the system (1) – (2) is obtained using the new generation method described in [30]. To find the threshold number $\mathcal{R}_0$ of the system (1) – (2), let $\mathcal{F}$ and $\mathcal{V}$ be the matrices of new infections and transmission, respectively. At the HIV-free equilibrium ε_0 of the system (1) – (2), the matrices $\mathcal{F}$ and $\mathcal{V}$ are given by

$$\mathcal{F} = \begin{pmatrix} 0 & 0 & \frac{\beta_C p_{SW} S_C^*}{S_{SW}^*} & 0 \\ 0 & 0 & 0 & 0 \\ \frac{\beta_{SW} p_C S_{SW}^*}{S_C^*} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \qquad \mathcal{V}^{-1} = \begin{pmatrix} \frac{1}{\mu_C + d_C} & 0 & 0 & 0 \\ -\frac{\gamma}{(\mu_C+\gamma)(\mu_C+d_C)} & \frac{1}{\mu_C+d_C} & 0 & 0 \\ 0 & 0 & -\frac{1}{\gamma} & 0 \\ 0 & 0 & 0 & \frac{1}{\mu_{SW}+d_{SW}} \end{pmatrix}$$

Thus, the $\mathcal{R}_0$ which is the spectral radius of the matrix $\mathcal{F}\mathcal{V}^{-1}$ is given

$$\mathcal{R}_0 = \sqrt{\frac{\beta_C \beta_{SW} p_C p_{SW}}{\gamma(\mu_C + \gamma)}} \tag{14}$$

3.4 Stability Analysis

Lemma 3.3 *The HIV-free equilibrium ε_0 is locally asymptotically stable whenever $\mathcal{R}_0 < 1$, and unstable otherwise.*

Proof : Let us consider the infected classes I_C, A_C, I_{SW}, A_{SW}. By the equations corresponding to these states, we have the linearization system at ε_0 given by:

$$\begin{cases} \frac{dI_C}{dt} &= \lambda_C S_C - (\mu_C + \gamma) I_C, \\ \frac{dA_C}{dt} &= \gamma I_C - (\mu_C + d_C) A_C, \\ \frac{dI_{SW}}{dt} &= \lambda_{SW} S_{SW} - (\mu_{SW} + \gamma) I_{SW}, \\ \frac{dA_{SW}}{dt} &= \gamma I_{SW} - (\mu_{SW} + d_{SW}) A_{SW}. \end{cases} \tag{15}$$

The matrix $\mathcal{M}_1$ associate to the linearised system (15) is given by :

$$\mathcal{M}_1 = \begin{pmatrix} -(\mu_C + \gamma) & 0 & \beta_C p_{SW} \frac{\Lambda_C}{\mu_C N_{SW}} & 0 \\ \gamma & -(\mu_C + d_C) & 0 & 0 \\ \beta_{SW} p_C \frac{\Lambda_{SW}}{\mu_{SW} N_C} & 0 & -(\mu_{SW} + \gamma) & 0 \\ 0 & 0 & \gamma & -(\mu_{SW} + d_{SW}) \end{pmatrix} \tag{16}$$

and the linearization system (15) can be rewrite at follows :

$$\dot{y} \leq V_1 y,$$

where $y = (I_C, A_C, I_{sw}, A_{sw})^t$

Let $V_1 = F_1 + V_1$ such as

$$F_1 = \begin{pmatrix} 0 & 0 & \beta_C p_{SW} \frac{\Lambda_C}{\mu_C N_{SW}} & 0 \\ 0 & 0 & 0 & 0 \\ \beta_{SW} p_C \frac{\Lambda_{SW}}{\mu_{SW} N_C} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix};$$

and

$$V_1 = \begin{pmatrix} -(\mu_C + \gamma) & 0 & 0 & 0 \\ \gamma & -(\mu_C + d_C) & 0 & 0 \\ 0 & 0 & -(\mu_{SW} + \gamma) & 0 \\ 0 & 0 & \gamma & -(\mu_{SW} + d_{SW}) \end{pmatrix}$$

The matrix V_1 is an inversible matrix and V_1^{-1} is given by :

$$V_1^1 = \begin{pmatrix} -\frac{1}{\mu_C + \gamma} & 0 & 0 & 0 \\ -\frac{\gamma}{(\mu_C + \gamma)(\mu_C + d_C)} & -\frac{1}{\mu_C + d_C} & 0 & 0 \\ 0 & 0 & -\frac{1}{\mu_{SW} + \gamma} & 0 \\ 0 & 0 & -\frac{\gamma}{(\mu_{SW} + \gamma)(\mu_{SW} + d_{SW})} & -\frac{1}{\mu_{SW} + d_{SW}} \end{pmatrix}$$

We can also see that $F_1 \geq 0$ and $V_1^{-1} \geq 0$

Thus, $\mathbb{R}_0 = \rho(-F_1 V_1^{-1}) < 1$ and from the theorem of Varga [27] the matrix M1 is asymptotically stable. The eigenvalue of matrix $\mathcal{M}_1$ has negative real part, by a standard comparison theorem [31], when $t \longrightarrow +\infty$, $I_C \longrightarrow 0, A_C \longrightarrow 0, I_{SW} \longrightarrow 0$ and $A_{SW} \longrightarrow 0$ for system (15) and substituting $I_C = 0, A_C = 0, I_{SW} = 0, A_{SW} = 0$, in $(1) - (2)$ we get $S_C \longrightarrow \frac{\Lambda_C}{\mu_C}, S_{SW} \longrightarrow \frac{\Lambda_{SW}}{\mu_{SW}}$, as well as $t \longrightarrow +\infty$.

Thus, $(S_C, I_C, A_C, S_{SW}, I_{SW}, A_{SW}) \longrightarrow (\frac{\Lambda_C}{\mu_C}, 0, 0, \frac{\Lambda_{SW}}{\mu_{SW}}, 0, 0)$ as $t \longrightarrow +\infty$ for system $(1) - (2)$, when $\mathbb{R}_0 < 1$.

Therefore disease- free equilibrium ε_0 is globally asymptotically stable in the positively set Δ when $\mathbb{R}_0 < 1$.

Lemma 3.4 *The endemic equilibrium ε_1 of system $(1) - (2)$ is globally asymptotically stable, when $\mathcal{R}_0 > 1$.*

Proof : Let $\varepsilon_1 = \left\{S_C^*, I_C^*, A_C^*, S_{SW}^*, 0, 0\right\}$ be the endemic equilibrium of system $(1)-(2)$. From system $(1)-(2)$, we have

$$\begin{cases} \Lambda_C & = \lambda_C^* S_C^* + \mu_C S_C^*, \\ \lambda_C^* S_C^* & = (\mu_C + \gamma) I_C^*, \\ (\mu_C + d_C) A_C^* & = \gamma I_C^*, \\ \Lambda_{SW} & = \lambda_{SW}^* S_{SW}^* + \mu_{SW} S_{SW}^*, \\ \lambda_{SW} S_{SW}^* & = (\mu_{SW} + \gamma) I_{SW}^*, \\ (\mu_{SW} + d_{SW}) A_{SW}^* & = \gamma I_{SW}^* \end{cases} \tag{17}$$

Let define the function ψ on $\mathbb{R}_+^*$ by

$$\Psi(x) \quad = \quad x - 1 - \ln(x). \tag{18}$$

The function $\Psi(x)$ is non-negative for all $x \in \mathbb{R}_+^*$. Let us consider the Lyapunov candidate function define by :

$$V = V_C + V_{SW}, \qquad \text{with} \qquad V_C = V_{S_C} + V_{I_C} + V_{A_C} \qquad \text{and} \qquad V_{SW} = V_{S_{SW}} + V_{I_{SW}} + V_{A_{SW}},$$

where

$$\begin{aligned}
\mathrm{V_{S_C}} &= \mathrm{S_C^*}\Psi(\frac{\mathrm{S_C}}{\mathrm{S_C^*}}),\\
\mathrm{V_{I_C}} &= \mathrm{I_C^*}\Psi(\frac{\mathrm{I_C}}{\mathrm{I_C^*}}),\\
\mathrm{V_{A_C}} &= \mathrm{A_C^*}\Psi(\frac{\mathrm{A_C}}{\mathrm{A_C^*}}),\\
\mathrm{V_{S_{SW}}} &= \mathrm{S_{SW}^*}\Psi(\frac{\mathrm{S_{SW}}}{\mathrm{S_{SW}^*}}),\\
\mathrm{V_{I_{SW}}} &= \mathrm{I_{SW}^*}\Psi(\frac{\mathrm{I_{SW}}}{\mathrm{I_{SW}^*}}),\\
\mathrm{V_{A_{SW}}} &= \mathrm{A_{SW}^*}\Psi(\frac{\mathrm{A_{SW}}}{\mathrm{A_{SW}^*}}).
\end{aligned}$$

Now, we have to differentiate the function V with respect to the time.

Let us compute $\mathrm{V_C}$

$$\begin{aligned}
\dot{\mathrm{V}}_{\mathrm{S_C}} &= (1-\frac{\mathrm{S_C^*}}{\mathrm{S_C}})\dot{\mathrm{S}}_{\mathrm{C}}\\
&= (1-\frac{\mathrm{S_C^*}}{\mathrm{S_C}})(\Lambda_{\mathrm{C}}-\lambda_{\mathrm{C}}\mathrm{S_C}-\mu_{\mathrm{C}}\mathrm{S_C})\\
&= (1-\frac{\mathrm{S_C^*}}{\mathrm{S_C}})(\lambda_{\mathrm{C}}^*\mathrm{S_C^*}+\mu_{\mathrm{C}}\mathrm{S_C^*}-\lambda_{\mathrm{C}}\mathrm{S_C}-\mu_{\mathrm{C}}\mathrm{S_C})\\
&= -\mu_C\frac{(S_C-S_C^*)^2}{S_C}+\beta_C p_{SW}\frac{S_C^*I_{SW}^*}{N_{SW}}\Big[(1-\frac{S_C^*}{S_C})(1-\frac{S_CI_{SW}}{S_C^*I_{SW}^*})\Big]\\
&= -\mu_C\frac{(S_C-S_C^*)^2}{S_C}+\beta_C p_{SW}\frac{S_C^*I_{SW}^*}{N_{SW}}\Big[-\frac{S_CI_{SW}}{S_C^*I_{SW}^*}+1+\ln(\frac{S_CI_{SW}}{S_C^*I_{SW}^*})-\frac{S_C^*}{S_C}+1+\ln(\frac{S_C^*}{S_C})\\
&\qquad\qquad +\frac{I_{SW}^*}{I_{SW}}-1-\ln(\frac{I_{SW}^*}{I_{SW}})\Big]\\
\dot{\mathrm{V}}_{\mathrm{S_C}} &= \mu_C\frac{(S_C-S_C^*)^2}{S_C}+\beta_C p_{SW}\frac{S_C^*I_{SW}^*}{N_{SW}}\Big[-\Psi(\frac{S_CI_{SW}}{S_C^*I_{SW}^*})-\Psi(\frac{S_C^*}{S_C})+\Psi(\frac{I_{SW}}{I_{SW}^*})\Big]\\
\dot{\mathrm{V}}_{\mathrm{I_C}} &= (1-\frac{\mathrm{I_C^*}}{\mathrm{I_C}})\dot{\mathrm{I}}_{\mathrm{C}}\\
&= (1-\frac{\mathrm{I_C^*}}{\mathrm{I_C}})\Big[\lambda_{\mathrm{C}}\mathrm{S_C}-(\mu_{\mathrm{C}}+\gamma)\mathrm{I_C}\Big]\\
&= \frac{\beta_{\mathrm{C}}\mathrm{p_{SW}}\mathrm{S_C^*I_{SW}^*}}{\mathrm{N_{SW}}}(1-\frac{\mathrm{I_C^*}}{\mathrm{I_C}})\Big[\frac{\mathrm{I_{SW}S_C}}{\mathrm{I_{SW}^*S_C^*}}-\frac{\mathrm{I_C}}{\mathrm{I_C^*}}\Big]\\
&= \frac{\beta_{\mathrm{C}}\mathrm{p_{SW}}\mathrm{S_C^*I_{SW}^*}}{\mathrm{N_{SW}}}\Big[\frac{\mathrm{I_{SW}S_C}}{\mathrm{I_{SW}^*S_C^*}}-1-\ln(\frac{\mathrm{I_{SW}S_C}}{\mathrm{I_{SW}^*S_C^*}})-\frac{\mathrm{I_C}}{\mathrm{I_C^*}}+1+\ln(\frac{\mathrm{I_C}}{\mathrm{I_C^*}})-\frac{\mathrm{S_CI_C^*I_{SW}}}{\mathrm{S_C^*I_CI_{SW}^*}}+1+\ln(\frac{\mathrm{S_CI_C^*I_{SW}}}{\mathrm{S_C^*I_CI_{SW}^*}})\Big]\\
\dot{\mathrm{V}}_{\mathrm{I_C}} &= \frac{\beta_{\mathrm{C}}\mathrm{p_{SW}}\mathrm{S_C^*I_{SW}^*}}{\mathrm{N_{SW}}}\Big[\Psi(\frac{\mathrm{S_CI_{SW}}}{\mathrm{S_C^*I_{SW}^*}})-\Psi(\frac{\mathrm{I_C}}{\mathrm{I_C^*}})-\Psi(\frac{\mathrm{S_CI_C^*I_{SW}}}{\mathrm{S_C^*I_CI_{SW}^*}})\Big]\\
\dot{\mathrm{V}}_{\mathrm{A_C}} &= (1-\frac{\mathrm{A_C^*}}{\mathrm{A_C}})\dot{\mathrm{A}}_{\mathrm{C}}\\
&= (1-\frac{\mathrm{A_C^*}}{\mathrm{A_C}})\Big[\gamma\mathrm{I_C}-(\mu_{\mathrm{C}}+\mathrm{d_C})\mathrm{A_C}\Big]\\
&= \gamma\mathrm{I_C^*}(1-\frac{\mathrm{A_C^*}}{\mathrm{A_C}})\Big[\frac{\mathrm{I_C}}{\mathrm{I_C^*}}-\frac{\mathrm{A_C}}{\mathrm{A_C^*}}\Big]\\
&= \gamma\mathrm{I_C^*}\Big[\frac{\mathrm{I_C}}{\mathrm{I_C^*}}-1\ln(\frac{\mathrm{I_C}}{\mathrm{I_C^*}})-\frac{\mathrm{A_C}}{\mathrm{A_C^*}}+1+\ln(\frac{\mathrm{A_C}}{\mathrm{A_C^*}})-\frac{\mathrm{A_C^*I_C}}{\mathrm{A_CI_C^*}}+1+\ln(\frac{\mathrm{A_C^*I_C}}{\mathrm{A_CI_C^*}})\Big]\\
\dot{\mathrm{V}}_{\mathrm{A_C}} &= \gamma\mathrm{I_C^*}\Big[\Psi(\frac{\mathrm{I_C}}{\mathrm{I_C^*}})-\Psi(\frac{\mathrm{A_C}}{\mathrm{A_C^*}})-\Psi(\frac{\mathrm{A_C^*I_C}}{\mathrm{A_CI_C^*}})\Big]
\end{aligned}$$

Let $K_1=\max\Big\{\frac{\beta_C p_{SW}S_C^*I_C^*}{N_{SW}};\gamma I_C^*\Big\}$

$$\mathrm{V_C} \leq -\mu_{\mathrm{C}}\frac{(\mathrm{S_C}-\mathrm{S_C^*})^2}{\mathrm{S_C}}+\mathrm{K_1}\Big[-\Psi(\frac{\mathrm{S_C^*}}{\mathrm{S_C}})+\Psi(\frac{\mathrm{I_{SW}}}{\mathrm{I_{SW}^*}})-\Psi(\frac{\mathrm{S_CI_C^*I_{SW}}}{\mathrm{S_C^*I_CI_{SW}^*}})-\Psi(\frac{\mathrm{A_C}}{\mathrm{A_C^*}})-\Psi(\frac{\mathrm{A_C^*I_C}}{\mathrm{A_CI_C^*}})\Big] \qquad (19)$$

Similarly, it is shown that

$$\mathrm{V_{SW}} \leq -\mu_{\mathrm{SW}}\frac{(\mathrm{S_{SW}}-\mathrm{S^*_{SW}})^2}{\mathrm{S_{SW}}}+\mathrm{K_2}\Big[-\Psi(\frac{\mathrm{S^*_{SW}}}{\mathrm{S_{SW}}})+\Psi(\frac{\mathrm{I_C}}{\mathrm{I^*_C}})-\Psi(\frac{\mathrm{S_{SW}I^*_{SW}I_C}}{\mathrm{S^*_{SW}I_{SW}I^*_C}})-\Psi(\frac{\mathrm{A_{SW}}}{\mathrm{A^*_{SW}}})-\Psi(\frac{\mathrm{A^*_{SW}I_{SW}}}{\mathrm{A_{SW}I^*_{SW}}})\Big] \quad (20)$$

With $K_2 = \max\left\{\frac{\beta_{sw}p_C S^*_{SW}I^*_{SW}}{N_C};\gamma I^*_{SW}\right\}$
By using the assumption **H**, we obtain

$$\dot{\mathrm{V}} \leq 0 \quad (21)$$

Also, we have $\mathrm{V} > 0$ for all $\mathrm{I_C}, \mathrm{A_C}, \mathrm{I_{SW}}, \mathrm{A_{SW}} \in \mathbb{R}_+$, and $\dot{\mathrm{V}} = 0$ for $\mathrm{I_C} = \mathrm{I^*_C}$, $\mathrm{A_C} = \mathrm{A^*_C}$, $\mathrm{I_{SW}} = \mathrm{I^*_{SW}}$, $\mathrm{A_{SW}} = \mathrm{A^*_{SW}}$. Then, by the asymptotic stability theorem [28], the endemic equilibrium ε_3 of System (1)-(2) is globally asymptotically stable.

3.5 Bayesian SI Model for the Outbreak of HIV

According to the equations $(1)-(2)$, the cumulative frequency for each state until time t can be expressed as follows each state until time t can be expressed as follows:

$$\mathrm{S_C} = \int_0^{\mathrm{t}} \Big(\Lambda_{\mathrm{C}} - (\lambda_{\mathrm{C}}(\mathrm{s}) + \mu_{\mathrm{C}})\mathrm{S_C}(\mathrm{s})\Big)\mathrm{ds}, \quad (22a)$$

$$\mathrm{I_C} = \int_0^{\mathrm{t}} \Big(\lambda_{\mathrm{C}}\mathrm{S_C}(\mathrm{s}) - (\mu_{\mathrm{C}} + \gamma)\mathrm{I_C}(\mathrm{s})\Big)\mathrm{ds}, \quad (22b)$$

$$\mathrm{A_C} = \int_0^{\mathrm{t}} \Big(\gamma\mathrm{I_C}(\mathrm{s}) - (\mu_{\mathrm{C}} + \mathrm{d_C})\mathrm{A_C}(\mathrm{s})\Big)\mathrm{ds}, \quad (22c)$$

$$\mathrm{S_{SW}} = \int_0^{\mathrm{t}} \Big(\Lambda_{\mathrm{SW}} - (\lambda_{\mathrm{SW}}(\mathrm{s}) + \mu_{\mathrm{SW}})\mathrm{S_{SW}}(\mathrm{s})\Big)\mathrm{ds}, \quad (22d)$$

$$\mathrm{I_{SW}} = \int_0^{\mathrm{t}} \Big(\lambda_{\mathrm{SW}}\mathrm{S_{SW}}(\mathrm{s}) - (\mu_{\mathrm{SW}} + \gamma)\mathrm{I_{SW}}(\mathrm{s})\Big)\mathrm{ds}, \quad (22e)$$

$$\mathrm{A_{SW}} = \int_0^{\mathrm{t}} \Big(\gamma\mathrm{I_{SW}}(\mathrm{s}) - (\mu_{\mathrm{SW}} + \mathrm{d_{SW}})\mathrm{A_{SW}}(\mathrm{s})\Big)\mathrm{ds}. \quad (22f)$$

The data consist of yearly counts $Y_t = (Y_t^{I_C}, Y_t^{A_C}, Y_t^{I_{SW}}, Y_t^{A_{SW}})$ of the number of infected persons, over a time interval T in years. To link the data to the SI dynamics, we can specify the following Poisson observation model:

$$\mathrm{Y_t^{I_C}} \sim \mathrm{Poisson}(\mathrm{I_C}(\mathrm{t})), \quad (23a)$$

$$\mathrm{Y_t^{A_C}} \sim \mathrm{Poisson}(\mathrm{A_C}(\mathrm{t})), \quad (23b)$$

$$\mathrm{Y_t^{I_{SW}}} \sim \mathrm{Poisson}(\mathrm{I_{SW}}(\mathrm{t})), \quad (23c)$$

$$\mathrm{Y_t^{A_{SW}}} \sim \mathrm{Poisson}(\mathrm{A_{SW}}(\mathrm{t})). \quad (23d)$$

Solving the system of differential $(22a)-(22e)$ is subject to the initial values specified by $S_C = N_C - 1$, $I_C = 1$, $A_C = 0$, $S_{SW} = N_{SW} - 1$, $I_{SW} = 1$, $A_{SW} = 0$ respectively.

3.6 Estimation of parameters

The model parameters were estimated using a Bayesian Markov Chain Monte Carlo (MCMC) method for the system of differential equations. This approach allows us to incorporate prior information from the parameters. The table below (**??**) summarises the data, estimated parameters, and posterior distribution of the Bayesian SI model.

3.7 Estimation of the impact of the intervention

The effect of the interventions in terms of person-years of infection averted over a 25-year period since 1980, when HIV infection began in Burkina Faso, is assessed. To do this, we estimate and compare the expected number of HIV infections among sex workers with and without the intervention during our study period.

4 Numerical simulations

The model was coded in R software using Stan's rstan package. We opt for the fourth-order Runge-Kutta (RK4) approximation for a numerical evaluation of the solution. Stan is a toolkit for Bayesian inference containing a programming language for defining Bayesian statistical models, an MCMC sampler based on an algorithm called "Hamiltonian Monte Carlo" (much faster than the Gibbs sampler implemented in BUGS) and a Quasi-Newton type optimizer (for finding ML/MAP estimators).

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