A co-interaction model of HIV and syphilis infection among gay, bisexual and other men who have sex with men

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A B S T R A C T

We developed a mathematical model to study the co-interaction of HIV and syphilis infection among gay, bisexual and other men who have sex with men (gbMSM). We qualitatively analysed the model and established necessary conditions under which disease-free and endemic equilibria are asymptotically stable. We gave analytical expressions for the reproduction number, and showed that whenever the reproduction numbers of sub-models and co-interaction model are less than unity, the epidemics die out, while epidemics persist when they are greater than unity. We presented numerical simulations of the full model and showed qualitative changes of the dynamics of the full model to changes in the transmission rates. Our numerical simulations using a set of reasonable parameter values showed that: (a) both diseases die out or co-exist whenever their reproduction number is less than or exceed unity. (b) HIV infection impacts syphilis prevalence negatively and vice versa. (c) one possibility of lowering the co-infection of HIV and syphilis among gbMSM is to increase both testing and treatment rates for syphilis and HIV infection, and decrease the rate at which HIV infected individuals go off treatment.

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1. Introduction

HIV is known to be a sexually transmitted and blood-borne infection with a highly variable disease progression in humans (Glance, 2013). People infected with HIV experience immune suppression as a result of continuous destruction of the CD4^+ T lymphocytes, which makes immunosuppressed individuals at risk of acquiring other sexually transmitted infections (such as syphilis, gonorrhea (Bourgeois et al., 2017; Gail Bolan Christopher, 2006; Glance, 2013)). At the end of 2017, approximately 37 million people were living with HIV throughout the world, and over 900,000 reported deaths were attributed to HIV infection (World Health Organization, 2018a, 2018b). In 2016, gay, bisexual and other men who have sex with men (gbMSM) accounted for about half of the new HIV infections in Canada (Public Health Agency of Canada, 2016). Similarly, gbMSM currently accounts for most new and prevalent cases of HIV in Vancouver (British Columbia Centre for Disease Control, 2018a, 2018b) and
San Francisco (Chen, Scheer, Nguyen, Kohn, & Schwartz, 2018). There were about 3320 gbMSM who were newly diagnosed with HIV in the UK in 2015 (Desai et al., 2018). The increase of antiretroviral therapy (ART) coverage to reduce and prevent HIV transmission in British Columbia (BC), Canada, made us observe a positive impact of HAART to prevent HIV transmission and decrease HIV diagnosis per year (Williams, Lima, & Gouws, 2011).

Syphilis is known to be an infection caused by the Treponema pallidum bacteria (Gail Bolan Christopher, 2006; Public Health Agency of Canada, 2017), and progresses from primary → secondary → latent → tertiary stage if left untreated (Public Health Agency of Canada, 2017). Infectious syphilis is more prevalent in males with an increased rate among gbMSM population in BC and Canada (British Columbia Centre for Disease Control, 2018a, 2018b; Public Health Agency of Canada, 2017). In 2017, 5% or more of gbMSM in 22 of 34 reporting countries were infected with syphilis (World Health Organization, 2018a, 2018b). From 2011 to 2015, the rate of reported cases of syphilis per 100,000 population in the United States rose by 58% (from 14.8 to 23.4), with the highest rate observed in San Francisco, where the rates rose by 77% (from 84.3 to 149.6) (Chen et al., 2018). In 2016, gbMSM accounted for about 80.6% of male infectious syphilis in the United States (US Centers for Disease Control and Prevention, 2017). Similarly, in BC, the rate of reported cases of infectious syphilis per 100,000 population in 2016, rose to 16.0 (759 cases) when compared to 4.2 (193 cases) in 2011 (British Columbia Centre for Disease Control, 2018a, 2018b). The highest rate in BC was observed in Vancouver and surrounding regions, where the rates rose from 19.6 (131 cases) in 2011 to 63.7 (428 cases) in 2016 (British Columbia Centre for Disease Control, 2018a, 2018b). In 2016, gbMSM accounted for about 63.5% of infectious syphilis in Vancouver (British Columbia Centre for Disease Control, 2018a, 2018b).

Recent increases in sexually transmitted infections (STIs), especially among gbMSM, brought up about the importance for characterising the co-interaction of HIV and syphilis. Increases in the risk of HIV and STI transmission have been attributed to sexual behaviours over the last decade (Desai et al., 2018; Gail Bolan Christopher, 2006; US Centers for Disease Control and Prevention, 2017). It is estimated that about 43% of gbMSM in BC with syphilis diagnoses and known HIV status in 2016, were HIV positive (British Columbia Centre for Disease Control, 2018a, 2018b). Individuals co-infected with these two diseases are more likely to transmit HIV to their sexual partners, and as well likely to progress to serious disease stages (British Columbia Centre for Disease Control, 2018a, 2018b; Gail Bolan Christopher, 2006), gbMSM living with HIV are about 2 times more likely to be infected with syphilis compared to those that are HIV negative (US Centers for Disease Control and Prevention, 2017).

This paper considers a single class of infectious syphilis since major stages, such as primary, secondary, early latent and infectious neurosyphilis, are generally classified as infectious syphilis, and is of public health concern (Public Health Agency of Canada, 2017). Many mathematical models have been previously used to assess dynamics of the co-infection of HIV and other diseases, such as Hepatitis C virus, gonorrhea, tuberculosis and syphilis (Bhunu, Garira, & Canada, 2017; Mushayabasa, Tchuenche, Bhunu, & Ngarakana-Gwasira, 2011; Nwankwo & Okuonghae, 2018), but only Nwankwo et al. (Nwankwo & Okuonghae, 2018) used a similar approach to study the dynamics of HIV and syphilis. Our study differs from (Nwankwo & Okuonghae, 2018) as we consider the gbMSM population in a setting where treatment of both diseases is readily available. We make simplifying assumptions about the natural history of both diseases and incorporate some epidemiological features of the co-dynamics of HIV and syphilis. From our mathematical analyses and using a set of parameter values from published articles, our model aim to answer the following questions: What effect does syphilis infection have on HIV infected individuals and vice versa? What is the impact of change in transmission rate on the dynamics of both diseases? Can we test and treat mono-infected individuals more to reduce the prevalence of both diseases?

The paper is organised as follows. We develop and describe the model in Section 2, and analyse two sub-models in Sections 3 and 4. We present the analysis of the full co-interaction model and some numerical simulations in Sections 5 and 6 respectively while Section 7 discusses and concludes the paper.

### 2. Model formulation and description

The total gbMSM population at time $t$, denoted by $N(t)$ is divided into 8 mutually exclusive compartments stated in Table (1), so that

| Table 1 |
|---------|
| Model Variables and their Descriptions. |
| Variable | Description |
|---------|-------------|
| $S$ | Susceptible individuals |
| $S_{IS}$ | Individuals mono-infected with syphilis |
| $U_{IS}$ | Individuals mono-infected with HIV and unaware |
| $A_H$ | Individuals mono-infected with HIV and aware |
| $T_H$ | HIV infected individuals on treatment |
| $U_{AH}$ | Co-infected individuals unaware of HIV infection |
| $A_{AH}$ | Co-infected individuals aware of HIV infection |
| $T_{AH}$ | Co-infected individuals on HIV treatment |
We assume that at time $t$, new recruits enter the population at a constant rate $\Pi$. Individuals die in each subclass at a constant natural mortality rate $\mu$. HIV infected individuals ($U_H$, $A_H$, $U_{SH}$, $A_{SH}$) not on treatment have additional HIV induced death rates $d_{U_H}, d_{A_H}, d_{U_{SH}}, d_{A_{SH}}$ respectively. We assume no death from syphilis and that HIV infected individuals on treatment do not transmit HIV infection [24, 40].

Diseases co-dynamics are complicated processes, but for simplicity, we assume that both mono and co-infected individuals can either transmit HIV or syphilis but not both at the same time. Susceptible individuals may acquire syphilis infection when in contact with individuals in $I_S$, $U_{SH}$, $A_{SH}$ and $T_{SH}$ compartments, at a rate $\lambda_S$ (the force of infection associated with syphilis infection), given by $\lambda_S = \beta_S (1 + \varphi_1 U_H + \varphi_2 A_H + \varphi_3 T_H) N$, where $\beta_S$ denotes the transmission rate for syphilis. Parameter $\lambda_S$ is the probability of syphilis transmission from one contact between individuals in $S$ and in other syphilis infected compartments ($I_S$, $U_{SH}$, $A_{SH}$, $T_{SH}$), times the number of contacts per year per individual. Modification parameters $\varphi_1$, $\varphi_2$ and $\varphi_3$ respectively account for the relative infectiousness of syphilis infected individuals with undiagnosed HIV infection ($U_{SH}$), co-infected with HIV and aware ($A_{SH}$), and coinfected with HIV and on HIV treatment ($T_{SH}$), compared to individuals mono-infected with syphilis. We assume that coinfected individuals are about two times as infectious as mono-infected individuals (US Centers for Disease Control and Prevention, 2017). Since it is believed that individuals infected with syphilis recover with temporal immunity (Pourbohloul, Rekart, & Brunham, 2003), we then assume that individuals infected with syphilis recover after treatment and return to the susceptible class at a rate $\sigma_1$.

Susceptible individuals acquire HIV infection from those in the $U_H$, $A_H$, $U_{SH}$ and $A_{SH}$ compartments, at the rate $\lambda_H$ (the force of infection associated with HIV infection), given by $\lambda_H = \beta_H (1 + \varphi_1 U_H + \varphi_2 A_H + \varphi_3 T_H) N$, where $\beta_H$ denotes the transmission rate for HIV. Parameter $\beta_H$ is the probability of HIV transmission from one contact between individuals in $S$ and in other HIV infectious compartments ($U_H$, $A_H$, $U_{SH}$, $A_{SH}$), times the number of contacts per year per individual. Modification parameters $\kappa_1$, $\kappa_2$ and $\kappa_3$ respectively account for the relative infectiousness of individuals mono-infected with HIV and aware ($A_H$), co-infected with HIV and unaware ($U_{SH}$), co-infected with HIV and aware ($A_{SH}$), in comparison with individuals mono-infected with HIV.

Susceptible individuals infected with HIV at rate $\lambda_H$ enter the HIV unaware class $U_H$, where they progress to HIV aware class $A_H$ following testing at a rate $\sigma_1$, and are then placed on treatment at a rate $\rho_2$ to enter the class $T_H$. Individuals in the HIV infected and on treatment classes $T_H$ and $T_{SH}$ can go off treatment at rates $\rho_1$ and $\rho_2$ respectively.

Individuals mono-infected with HIV ($U_H, A_H, T_H$) are infected with syphilis at rates $\eta_1 \lambda_S, \eta_2 \lambda_S, \eta_3 \lambda_S$ to enter classes $U_{SH}, A_{SH}, T_{SH}$ respectively, and modification parameters $\eta_1, \eta_2, \eta_3 > 1$ account for higher risk of syphilis acquisition for people living with HIV.

Individuals mono-infected with syphilis, $I_S$ are infected with HIV at a rate $\gamma \lambda_H$ to enter the class $U_{SH}$, where the modification parameter $\gamma > 1$ due to higher risk of HIV acquisition for people whose immune system are sabotaged by syphilis infection. Co-infected individuals in the class $A_{SH}$ are placed on HIV treatment at a rate $\rho_1$ to enter class $T_{SH}$. Co-infected individuals in the classes $U_{SH}, A_{SH}, T_{SH}$ are tested and treated for syphilis at rates $\sigma_2, \sigma_3, \sigma_4$ to move back into the classes $U_H, A_H, T_H$, respectively. This model assumes uniform and homogeneous mixing population. The model diagram presented in Fig. 1 is described by the following system of non-linear differential equations.}

\[
\begin{align*}
\frac{dS}{dt} &= \Pi + \sigma_1 I_S - (\mu + \lambda_S + \lambda_H) S, \\
\frac{dI_S}{dt} &= \lambda_S S - (\mu + \sigma_1 + \gamma \lambda_H) I_S, \\
\frac{dU_H}{dt} &= \lambda_H S - (\mu + d_{U_H} + \alpha_1 + \eta_1 \lambda_S) U_H, \\
\frac{dA_H}{dt} &= \alpha_1 U_H + \sigma_2 U_{SH} + \sigma_3 A_{SH} + \nu_1 T_H - (\mu + d_{A_H} + \eta_2 \lambda_S + \rho_2) A_H, \\
\frac{dT_H}{dt} &= \rho_2 A_H + \sigma_4 T_{SH} - (\mu + \eta_3 \lambda_S + \nu_1) T_H, \\
\frac{dU_{SH}}{dt} &= \gamma \lambda_H S + \eta_1 \lambda_S U_H - (\mu + d_{U_{SH}} + \sigma_2) U_{SH}, \\
\frac{dA_{SH}}{dt} &= \eta_2 \lambda_S A_H + \nu_2 T_{SH} - (\mu + d_{A_{SH}} + \sigma_3 + \rho_1) A_{SH}, \\
\frac{dT_{SH}}{dt} &= \rho_1 A_{SH} + \eta_3 \lambda_S T_H - (\mu + \nu_2 + \sigma_4) T_{SH}.
\end{align*}
\]
3. Syphilis sub-model

We have the model with syphilis only by setting $U_H = A_H = T_H = U_{SH} = A_{SH} = T_{SH} = 0$ in system (2), and this gives

$$\frac{dS}{dt} = \Pi + \sigma_1 I_S - (\mu + \lambda_S)S,$$

$$\frac{dI_S}{dt} = \lambda_S S - (\mu + \sigma_1)I_S,$$

where $\lambda_S = \beta_S/N_S$, with total population given as $N_S(t) = S(t) + I_S(t)$.

The simple SIS model in (3) ignored syphilis-related death and was extensively discussed in (Pourbohloul et al., 2003) using different stages of syphilis infection to understand the transmission dynamics, and in (Gumel, Lubuma, Sharomi, & Terefe, 2018) to track syphilis dynamics in men and women. Hence, the dynamics of system (3) based on biological consideration in the region $\mathcal{Z}_S = \{ (S, I_S) \in \mathbb{R}_+^2 : N_S \leq \frac{\Pi}{\mu} \}$, is easy to show as being positively invariant with respect to the model (Brauer & Castillo-Chavez, 2012; Brauer et al., 2012; Diekmann, Heesterbeek, & Roberts, 2010). Therefore, model (3) is epidemiologically and mathematically well posed with all variables and parameters being positive for all time series. Model (3) has a disease free equilibrium points given by $E_{0S} = (S_0, I_{0S}) = \left( \frac{\Pi}{\mu}, 0 \right)$.

It is easy to explain the linear stability of disease free equilibrium by the reproduction number which can be derived using the method of next generation matrix in (Diekmann et al., 2010; Van den Driessche James, 2002). Hence, $E_{0S}$ can be explained by $R_{0S}$, where.

$$R_{0S} = \frac{\beta_S}{(\mu + \sigma_1)}$$

is the reproduction number for syphilis dynamics given by the product of the transmission rate of syphilis infection $\beta_S$ and the rate that an infective progresses out of syphilis infectious class $1/(\mu + \sigma_1)$. The biological interpretation of $R_{0S}$ is...
the number of syphilis infections produced by one syphilis infective during the period of infectiousness when introduced in a totally syphilis susceptible population in the presence of treatment.

We can establish the local stability of the disease free equilibrium \( (E_{0S}) \) using Lemma 1 which follows from \cite{(Diekmann et al., 2010)} and Theorem 2 of \cite{(Van den DriesscheJames, 2002)}.

**Lemma 1.** The DFE \( E_{0S} \) of model (3) is locally asymptotically stable (LAS) if \( R_{eS} < 1 \) and unstable if \( R_{eS} > 1 \).

The biological interpretation of \( R_{eS} < 1 \) is that we can eliminate syphilis from the population if the initial sizes of the sub-population of syphilis sub-model are in the attraction region \( E_{0S} \).

To ensure that elimination of syphilis epidemic is independent on the initial sizes of the sub-populations, we establish the global stability of the DFE \( E_{0S} \) by claiming the result in an easily proved Lemma 2.

**Lemma 2.** For any positive solutions \( (S(t), I_S(t)) \) of model system (3), if \( R_{eS} < 1 \), then, the DFE \( E_{0S} \) is a global attractor.

By equating the right-hand side of equation (3) to zero, and solving for \( S^* \) and \( I_S^* \), we have the endemic equilibrium points given by

\[
E_S^* = (S^*, I_S^*) = \left( \frac{\Pi (\mu + \sigma_1)}{\mu (\mu + \sigma_1 + \lambda_5)}, \frac{\lambda_5 S^*}{\mu + \sigma_1} \right)
\]

where \( \Omega = \frac{1}{\mu + \sigma_1} \) denote the mean infective period.

When \( R_{eS} > 1 \), \( E_S^* \) is positive and the epidemic of syphilis persists in the community. We can summarize the uniqueness of the endemic equilibrium in an easily proved Lemma 3.

**Lemma 3.** The endemic equilibrium \( E_S^* \) exists and is unique if and only if \( R_{eS} > 1 \).

**Proof.** It is enough to show that the components of \( E_S^* \) are positive only if \( R_{eS} > 1 \). We have \( I_S^* \) in equation (4) to be non-zero and positive only when \( R_{eS} > 1 \). The same follows for \( S^* \). QED.

The global stability of the endemic equilibrium for syphilis-only model can be easily shown from Chapter 2 in \cite{(Allen, Brauer, Van den Driessche, & Wu, 2008; Brauer, Castillo-Chavez, & Feng, 2018)}, three basic epidemiological models in \cite{(Diekmann et al., 2010)} and by claiming the result in an easily proved Lemma 4.

**Lemma 4.** The endemic equilibrium of syphilis-only model 3 is globally asymptotically stable in \( E_S \) whenever \( R_{eS} > 1 \).

In summary, the syphilis-only model (3) has a globally asymptotically stable disease-free equilibrium whenever \( R_{eS} < 1 \), and a unique endemic equilibrium whenever \( R_{eS} > 1 \).

### 3.1. Sensitivity analysis of \( R_{eS} \)

In this section, we investigate the effect of testing and treating syphilis on the dynamics of syphilis by the elasticity of \( R_{eS} \) with respect to \( \sigma_1 \). From \( R_{eS} = \frac{\beta_5}{\mu + \sigma_1} \), we use the approach in \cite{(Brauer & Castillo-Chavez, 2012; Braueret al., 2012; ChitnisJames and Jim, 2008)} to compute the elasticity \( \left( \text{Caswell, 2001} \right) \) of \( R_{eS} \) with respect to \( \sigma_1 \) as:

\[
\frac{\partial R_{eS}}{\partial \sigma_1} = \frac{\beta_5}{(\mu + \sigma_1)^2} \frac{1}{1 + \frac{\beta_5}{\mu + \sigma_1}}
\]

![Fig. 2. Syphilis reproduction number \( R_{eS} \) as a function of testing and treatment rate \( \sigma_1 \), with all parameters as in Table 2 except \( \beta_5 = 5.0 \). The red dash line indicates the reproduction number \( R_{eS} = 1 \).](image)
\[
\frac{\sigma_1}{R_{es}} \frac{\partial R_{es}}{\partial \sigma_1} - \frac{\sigma_1}{\mu + \sigma_1} \]  
\tag{5}
\]

Equation (5) is used to measure the impact of a change in \(\sigma_1\) on a proportional change in \(R_{es}\). Equation (5) suggests that an increase in the testing and treatment rate of syphilis always leads to decrease of \(R_{es}\), indicating a positive impact on the control of syphilis in the community.

Fig. 2 shows the effect of increasing treatment of syphilis in the community. For the set of parameters used, the figure shows that, by increasing the testing and treatment rate to 5 or more (\(R_{es}/C_0 < 0.99\)) (i.e., test and treat all susceptible males for syphilis every 2.4 months or less), the reproduction number would be below unity, which indicates syphilis eradication in the community.

4. HIV sub-model

We have the model with HIV only by setting \(I_S = U_{SH} = A_{SH} = T_{SH} = 0\) in (2) given by

\[
\begin{align*}
\frac{dS}{dt} &= \Pi - (\mu + \lambda_H)S, \\
\frac{dU_H}{dt} &= \lambda_H S - (\mu + d_{UH} + \alpha_1)U_H, \\
\frac{dA_H}{dt} &= \alpha_1 U_H + \nu_1 T_H - (\mu + d_{AH} + \rho_2)A_H, \\
\frac{dT_H}{dt} &= \rho_2 A_H - (\mu + \nu_1)T_H, \\
\lambda_H &= \frac{\beta_H (U_H + \kappa_1 A_H)}{N_H},
\end{align*}
\]

with the total population given as \(N_H(t) = S(t) + U_H(t) + A_H(t) + T_H(t)\).

Please note that the population is not constant and the equation of \(N_H\) that denotes the total sub-population of HIV-only model follows that

\[
\frac{dN_H}{dt} = \Pi - \mu N - d_{UH} U_H - d_{AH} A_H \leq \Pi - \mu N,
\]

and (8) implies that \(\lim_{t \to \infty} \sup N_H(t) \leq \frac{\Pi}{\mu}\). Therefore the dynamics of system (6) will be studied based on biological consideration in the region \(\mathcal{Z}_H = \{(S, U_H, A_H, T_H) \in \mathbb{R}_+^4 : N_H \leq \frac{\Pi}{\mu}\}\), which is easy to show as being positively invariant with respect to the model. We can similarly consider model (6) to be epidemiologically and mathematically well posed with all variables and parameters being positive for all time series as in (Diekmann et al., 2010).

4.1. Disease free equilibrium point

We have the disease free equilibrium when \(U_H = A_H = T_H = 0\) in model system (6). This gives \(E_0H = \left(\frac{\Pi}{\mu}, 0, 0, 0\right)\).

4.2. Effective reproduction number \(R_{eh}\)

Similarly, using the method of next generation matrix and the approach in (Diekmann et al., 2010; Van den DriesscheJames, 2002), as in \(R_{eh} = \rho(F_{V1})\), we have the reproduction number of HIV infections produced by HIV positive cases to be \(R_{eh}\), which is given as

\[
R_{eh} = \rho(F_{V1}) = \frac{\beta_H (\mu + \nu_1)(\mu + \alpha_1 \kappa_1 + d_{AH}) + \mu \rho_2)}{(\mu + d_{UH} + \alpha_1)(\mu + \nu_1)(\mu + d_{AH}) + \mu \rho_2)},
\]

and we can write \(R_{eh} = B_U + B_A\), where
\[
B_U = \frac{\beta_H}{(\mu + d_{UH} + \alpha_1)}, \\
B_A = \frac{\beta_H \alpha_1 \kappa_1 (\mu + \rho_1)}{(\mu + d_{UH} + \alpha_1)(\mu + \rho_1)(\mu + d_{AH}) + \mu \rho_2}).
\]

\(R_{EH}\) denotes the effective reproduction number for HIV dynamics (the number of HIV infection produced by one HIV case).

**Remark 1.** We can epidemiologically interpret the terms for the expression of \(R_{EH}\) in Equation (10). We have denoted \(B_U\) as the average number of new cases of HIV generated by individuals in the class \(U_H\), and \(B_A\) as the average number of new cases of HIV generated by individuals in the class \(A_H\).

\(B_U\) is interpreted as the product of the transmission rate of HIV infected individuals in the \(U_H\) class \((\beta_H)\) and the average duration spent in the \(U_H\) class \((\frac{1}{\mu + d_{UH} + \alpha_1})\).

Similarly, we can interpret \(B_A\) as the product of the transmission rate of HIV infected individuals in the \(A_H\) class \((\beta_H \kappa_1)\), the fraction that survives the \(U_H\) class \((\frac{1}{\mu + d_{UH} + \alpha_1})\) and the average duration spent in the \(A_H\) class, which include the duration of the fraction that goes off treatment from class \(T_H\) \((\frac{1}{\mu + d_{AH} + \alpha_1})\). Then the reproduction number \(R_{EH}\) is the sum of the expressions for \(B_U\) and \(B_A\), which is the number of HIV infections produced by one HIV infective during the period of infectiousness when introduced in a totally HIV susceptible population in the presence of treatment.

We can establish the local stability of the disease free equilibrium \((E_0H)\) using Lemma 5 which follows from (Diekmann et al., 2010) and Theorem 2 of (Van den DriesscheJames, 2002).

**Lemma 5.** The DFE \(E_0H\) of model (6) is locally asymptotically stable (LAS) if \(R_{EH} < 1\) and unstable otherwise.

The biological interpretation of \(R_{EH} < 1\) means that we can eliminate HIV from the population if the initial sizes of the sub-population of HIV sub-model are in the attraction region \(E_0H\). To be sure that eradication of HIV epidemic is independent of the initial sizes of the sub-populations, it makes sense to show that the disease free equilibrium \(E_0H\) is globally asymptotically stable.

**4.3. Global stability of the disease-free for HIV-only model**

We can rewrite model (6) as,

\[
\frac{dU}{dt} = F(U, V), \\
\frac{dV}{dt} = G(U, V), \quad G(U, 0) = 0,
\]

where \(U = S\) and \(V = (U_H, A_H, T_H)\), with \(U \in \mathcal{R}^1_+\) denoting the number of susceptible individuals and \(V \in \mathcal{R}^3_+\) denoting the number of infected individuals.

We now denote the disease free equilibrium by,

\[E_{0H} = (U^*, 0), \quad \text{where} \quad U^* = \left(\frac{\Pi}{\mu}\right)\]

Conditions S1 and S2 in equation (13) must be satisfied to guarantee local asymptotic stability.

\[
S1 : \frac{dU}{dt} = F(U, 0), \quad U^* \text{ is globally asymptotic stable (g.a.s)}
\]

\[
S2 : G(U, V) = AV - \hat{G}(U, V), \quad \hat{G}(U, V) \geq 0 \text{ for } (U, V) \in \Xi_H,
\]

where \(A = D_V G(U^*, 0)\) denotes the M-matrix (the off diagonal elements of A are non-negative) and \(\Xi_H\) is assumed to be region where the model makes biological sense. Lemma 6 holds if system (11) satisfies the conditions in (13).

**Lemma 6.** The disease free equilibrium point \(E_{0H}\) of HIV-only model is globally asymptotically stable if \(R_{EH} < 1\) and conditions in (13) are satisfied.

**Proof:** We have from Lemma 5 that \(E_{0H}\) is locally asymptotically stable if \(R_{EH} < 1\). Now consider
We can conclude that $E_0$H is globally asymptotically stable for $R_{\text{eff}} < 1$. QED.

4.4. Endemic equilibrium points

We can solve equation (6) in terms of the force of infection $\lambda_H = \beta_H \frac{(U_H + \kappa T_H)}{N_H}$ to find the conditions for the existence of an equilibrium, and for which HIV is endemic in a population.

Equating the right-hand side of equation (6) to zero, solving, substituting and writing in terms of the basic reproduction number $R_{\text{eff}}$ gives the endemic equilibrium point in terms of $R_{\text{eff}}$ as $E^*_H = (S^*, \ U^*_H, \ A^*_H, \ T^*_H)$, where,

\begin{align*}
S^* &= \frac{\Pi \Sigma}{\mu \Sigma + (R_{\text{eff}} - 1)} \quad (16) \\
U^*_H &= \frac{\Pi (R_{\text{eff}} - 1)}{\mu (U_H + \alpha_1)(\mu \Sigma + (R_{\text{eff}} - 1))} \quad (17) \\
A^*_H &= \frac{\alpha_1 \Pi (\mu + r_1)(R_{\text{eff}} - 1)}{(\mu + d_{UH} + \alpha_1)(\mu + \alpha_2 \mu + d_{AH} + \rho_2) + r_1(\mu + d_{AH}) + (\mu + \alpha_2 \mu + d_{AH} + \rho_2)(\mu + (R_{\text{eff}} - 1))} \quad (18) \\
T^*_H &= \frac{\alpha_1 \rho_2 \Pi (R_{\text{eff}} - 1)}{(\mu + d_{UH} + \alpha_1)(\mu + \alpha_2 \mu + d_{AH} + \rho_2) + r_1(\mu + d_{AH}) + (\mu + \alpha_2 \mu + d_{AH} + \rho_2)(\mu + (R_{\text{eff}} - 1))} \quad (19)
\end{align*}

$\lambda_H^* = \frac{(R_{\text{eff}} - 1)}{2}$, and $\Sigma$ denotes the mean infective period given by

$$\Sigma = \frac{1}{\mu (d_{UH} + \alpha_1)} \left(1 + \frac{\alpha_1 \mu}{(\mu + r_1)(\mu + d_{AH} + \mu \rho_2)} + \frac{\alpha_1 \rho_2}{(\mu + r_1)(\mu + d_{AH} + \mu \rho_2)} \right)$$

The endemic equilibrium point $E^*_H$ must be positive since the model in (6) also keeps track of human population. We have from Equations 16–19 that when $R_{\text{eff}} > 1$, $E^*_H$ is positive and HIV is able to attack the population. That is $R_{\text{eff}} > 1$ shows the possibility of HIV to prevail in the community where syphilis infection is not accounted for. The scenario showing the possibility of HIV to prevail in the community where syphilis infection is accounted for will be discussed when analyzing the full model (2).

We can summarize the uniqueness of the endemic equilibrium in Lemma 7.

**Lemma 7.** The endemic equilibrium $E^*_H$ of model (6) exists and is unique if and only if $R_{\text{eff}} > 1$.

**Proof.** It is enough to show that the components of $E^*_H$ are positive only if $R_{\text{eff}} > 1$. We have the numerator and denominator of $U^*_H$ in equation (17) to be positive only when $R_{\text{eff}} > 1$. Therefore, both the numerator and denominator of $U^*_H$ are non-zero and positive when $R_{\text{eff}} > 1$. The same follows for $S^*, A^*_H$ and $T^*_H$. QED.

4.5. Global stability of the endemic equilibrium for HIV-only model

In summary, the HIV-only model (6) is a special case of general staged progression models. For these models, the global asymptotic stability (GAS) of equilibria have been established using Lyapunov functions, see the work of (Bowman et al., 2005; Connell McCluskey, 2010; Guo, Li, & Shuai, 2006; Li & Shuai, 2010).
2005; Connell McCluskey, 2010) and more recent work of (Guo et al., 2006; Li & Shuai, 2010) for details. Hence we conclude that $E_0$ is globally asymptotically stable for $R_{eH} < 1$, while $E_+^H$ is globally asymptotically stable for $R_{eH} > 1$.

4.6. Sensitivity analysis of $R_{eH}$

Firstly, we investigate the effect of treating HIV on the dynamics of HIV by the elasticity of $R_{eH}$ with respect to $r_2$. From Equation (9), we use the approach in (Brauer & Castillo-Chavez, 2012; Brauer et al., 2012; Chitnis James and Jim, 2008) to compute the elasticity ((Caswell, 2001)) of $R_{eH}$ with respect to $r_2$ as:

$$\frac{\rho_2}{R_{eH}} \frac{\partial R_{eH}}{\partial \rho_2} = -\frac{\alpha_1 \kappa_1 \mu \rho_2 (\mu + r_1)}{(\mu + r_1)(\mu + d_{AH} + \mu r_2)/(\mu + \alpha_1 \kappa_1 + d_{AH} + \mu r_2)}.$$  

(20)

Equation (20) is used to measure the impact of a change in $\rho_2$ on a proportional change in $R_{eH}$. Equation (20) suggests that an increase in the rate of treatment of HIV will always lead to decrease of $R_{eH}$, indicating a positive impact on the control of HIV infection in the community.

Fig. 3a shows the effect of increasing treatment of HIV in the community. The figure predicts that even though increasing the number of cases treated can positively impact HIV epidemic by reducing the reproduction number, but elimination may only be achieved with aggressive treatment (i.e $\rho_2 = 50$ means treat all diagnosed cases every week). Note that based on Equation (10), no matter how high we increase $\rho_2$, $B_U$ will not be affected, which indicates that elimination of HIV requires more than increasing the number of cases treated, and may never be achieved by increasing $\rho_2$ if $B_U > 1$.

Secondly, we investigate the effect of testing HIV on the dynamics of HIV by the elasticity of $R_{eH}$ with respect to $\alpha_1$. From Equation (9), we use the approach in (Brauer & Castillo-Chavez, 2012; Brauer et al., 2012; Chitnis James and Jim, 2008) to compute the elasticity ((Caswell, 2001)) of $R_{eH}$ with respect to $\alpha_1$ as:

$$\frac{\alpha_1}{R_{eH}} \frac{\partial R_{eH}}{\partial \alpha_1} = -\frac{\alpha_1 \kappa_1 (\mu + r_1)(\mu + d_{AH}) - \alpha_1 ((\mu + r_1)(\mu + d_{AH} + \mu r_2))}{(\mu + d_{AH} + \alpha_1)((\mu + r_1)(\mu + \alpha_1 \kappa_1 + d_{AH} + \mu r_2))}.$$  

(21)

Equation (21) is used to measure the impact of a change in $\alpha_1$ on a proportional change in $R_{eH}$. Equation (21) suggests that an increase in the rate of testing HIV will have a positive impact in decreasing $R_{eH}$ and reducing HIV burden only if the numerator of Equation (21) is negative, i.e.

$$\alpha_1 (\mu + r_1)(\mu + d_{AH}) - ((\mu + r_1)(\mu + d_{AH} + \mu r_2)) < 0$$

Fig. 3b shows the effect of increasing testing of HIV in the community. The figure predicts that increasing the number of cases tested could positively impact HIV epidemic by reducing the reproduction number, but elimination will never be achieved by testing alone. Note that based on Equation (10), no matter how high we increase $\alpha_1$, there will always be an asymptote of $B_A$ for $\alpha_1 \rightarrow \infty$. This indicates that elimination of HIV requires more than increasing the number of cases tested, and may never be achieved by increasing $\alpha_1$ if the asymptote of $B_A > 1$. [Fig. 3a, 3b, 3c]
Thirdly, we investigate the effect of the rate of treatment failure on the dynamics of HIV by the elasticity of $R_{eH}$ with respect to $v_1$. We compute the elasticity (Caswell, 2001) of $R_{eH}$ with respect to $v_1$ as:

$$\frac{\nu_1}{R_{eH}} \frac{\partial R_{eH}}{\partial v_1} = \frac{\alpha_1 \kappa_1 \mu_2}{((\mu + v_1)(\mu + \alpha_1 \kappa_1 + d_{AH}) + \mu_2)((\mu + v_1)(\mu + d_{AH}) + \mu_2)}$$

Equation (22) is used to measure the impact of a change in $v_1$ on a proportional change in $R_{eH}$. Equation (22) suggests that a decrease in the rate of treatment failure always lead to a decrease of $R_{eH}$, indicating a positive impact on the control of HIV in the community.

Fig. 3c shows the effect of treatment failure on the dynamics of HIV in the community. This Figure predicts that increasing the rate at which people fall out of the treatment program (including failures of treatment) could negatively impact HIV epidemics by increasing the reproduction number and possibly increasing HIV epidemics.

5. Analysis of the HIV-syphilis model

Having analysed the two sub-models, we have the full HIV-syphilis model as in (2). From the equation of $N$ that denotes the total population as in Equation (1), it follows that

$$\frac{dN}{dt} = \Pi - \mu N - d_{UH} U_H - d_{AH} A_H - d_{USS} U_{SH} - d_{ASS} A_{SH} \leq \Pi - \mu N,$$

and (23) implies that $\lim sup N(t) \leq \frac{\Pi}{\mu}$. Therefore the dynamics of system (2) will be studied based on biological consideration in the region $\Xi = \left\{ (S, I_S, U_H, A_H, T_H, U_{SH}, A_{SH}, T_{SH}) \in \mathbb{R}_+^8 : N \leq \frac{\Pi}{\mu} \right\}$, which is easy to show as being positively invariant with respect to the model. Similarly, we can consider model (2) to be epidemiologically and mathematically well posed with all variables and parameters being positive for all time series as in (Diekmann et al., 2010).

Biologically, the full model (2) can have four equilibria, namely, disease free equilibrium $E_0$, HIV free equilibrium $E_1$, syphilis free equilibrium $E_2$ and the interior HIV-syphilis equilibrium $E_3$.

5.1. Disease free equilibrium point (DFE) of the full HIV-syphilis model

We have the disease free equilibrium when $I_S = U_H = A_H = T_H = U_{SH} = A_{SH} = T_{SH} = 0$ in model (2). This gives

$$E_0 = (S_0, I_{0S}, U_{0H}, A_{0H}, T_{0H}, U_{0SH}, A_{0SH}, T_{0SH}) = \left( \frac{\Pi}{\mu}, 0, 0, 0, 0, 0, 0 \right)$$

5.2. Effective reproduction number $R_e$

Using the next generation method in (Diekmann et al., 2010; Van den Driessche James, 2002), we can show that the effective reproduction number for the full HIV-syphilis model (2) is given by

$$R_e = \max \left\{ \frac{\beta_S}{(\mu + \sigma_1)}, \frac{\beta_H((\mu + v_1)(\mu + \alpha_1 \kappa_1 + d_{AH}) + \mu_2)}{(\mu + d_{AH} + \alpha_1)((\mu + v_1)(\mu + d_{AH}) + \mu_2)} \right\}$$

We can establish the local stability of the disease free equilibrium ($E_0$) using Lemma 8 which follows from (Diekmann et al., 2010) and Theorem 2 of (Van den Driessche James, 2002).

Lemma 8. The DFE $E_0$ of model (2) is locally asymptotically stable (LAS) if $R_e < 1$ and unstable otherwise.

Biological interpretation of $R_e < 1$ means that we can eliminate both diseases from the population if the initial sizes of the population are in the attraction region $\Xi$.

In the section below, we show that the elimination of HIV and syphilis epidemics is independent on the initial sizes of the populations by showing the global stability of the DFE $E_0$.

5.3. Global stability of the disease-free of the full HIV-syphilis model

We claim the result in Lemma 9 from Lemmas 2 and 6.

Lemma 9. The DFE $E_0$ of model (2) is globally asymptotically stable (GAS) if $R_e < 1$ and unstable if $R_e > 1$. 

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5.4. Endemic equilibrium point of the full HIV-syphilis model

The computation of the endemic equilibrium of the full HIV-syphilis model is analytically complicated, and therefore the endemic equilibria of model (2) corresponds to:

1. \( E_1 = (S_1, l_{S1}, 0, 0, 0, 0, 0, 0) \), the HIV free equilibrium, where

\[
E_1 = \left( \frac{\Pi}{\mu^2 S} \frac{\Pi(R_{eS} - 1)}{\mu^2 R_{eS}}, 0, 0, 0, 0, 0, 0 \right),
\]

This exists when \( R_{eS} > 1 \). The analysis of the equilibria \( E_1 \) is similar to the endemic equilibria \( E^*_S \) in equation (4).

2. \( E_2 = (S_2, 0, U_{H2}, A_{H2}, T_{H2}, 0, 0, 0) \), the syphilis free equilibrium, where

\[
\begin{align*}
S_2 &= \frac{\Pi S}{\mu^2 S + (R_{eH} - 1)} \\
U_{H2} &= \frac{\Pi (R_{eH} - 1)}{\mu + d_{UH} + \alpha_1 (\mu + d_{AH} + \rho_2) + r_1 (\mu + d_{AH}) (\mu + d_{AH} + (R_{eH} - 1))} \\
A_{H2} &= \frac{\alpha_1 \Pi (R_{eH} - 1)}{(\mu + d_{UH} + \alpha_1) (\mu + d_{AH} + \rho_2) + r_1 (\mu + d_{AH} + (R_{eH} - 1))} \\
T_{H2} &= \frac{\alpha_1 \rho_2 \Pi (R_{eH} - 1)}{(\mu + d_{UH} + \alpha_1) (\mu + d_{AH} + \rho_2) + r_1 (\mu + d_{AH} + (R_{eH} - 1))}
\end{align*}
\]

This exists when \( R_{eH} > 1 \). The analysis of the equilibria \( E_2 \) is similar to the endemic equilibria \( E^*_H \) in equations (16)–(19).

3. \( E_3 = (S_3, l_{S3}, U_{H3}, A_{H3}, T_{H3}, U_{SH3}, A_{SH3}, T_{SH3}) \), the HIV-syphilis co-interaction equilibrium.

We summarize the existence and local stability of the endemic equilibria in the following theorem:

**Theorem 10.** The system of equations in (2) has the following endemic equilibrium points:

1. \( E_1 \) exists for \( R_{eH} < 1 \) and \( R_{eS} > 1 \) which is locally asymptotically stable for \( R_{eS} \) near 1.
2. \( E_2 \) exists for \( R_{eS} < 1 \) and \( R_{eH} > 1 \) which is locally asymptotically stable for \( R_{eH} \) near 1.
3. \( E_3 \) exists when \( R_{eS} > 1 \) and \( R_{eH} > 1 \), i.e. \( R_e > 1 \).

Biological interpretation of the existence of \( E_2 \) (\( R_{eS} < 1 \) & \( R_{eH} > 1 \)) imply that HIV prevail in a community even with the presence of syphilis. Biological interpretation of the existence of \( E_3 \) (\( R_{eS} > 1 \) & \( R_{eH} > 1 \)) imply that both HIV and syphilis prevail in the community.

In general, the instability of the HIV free equilibrium \( E_1 \) and syphilis free equilibrium \( E_2 \) in the direction of the interior implies uniform persistence of the system(2), and hence the existence of the interior HIV-syphilis equilibrium \( E_3 \).

The endemic equilibria \( E_1, E_2, \) and \( E_3 \) will be studied using our numerical simulations.

The PROOF of theorem (10) follows from (Diekmann et al., 2010) Theorem 2 of (Van den Driessche, James, 2002).

6. Numerical simulations of the full model

In order to illustrate the results of the preceding analysis, the full HIV-syphilis model (2) is numerically simulated using R programming language and ggplot2 (Hadley, 2016; Venables SmithR Development Core Team et al., 2009). Unfortunately, we are unable to calibrate the model to data as a result of the complexity of our model and unavailability of data on HIV-syphilis co-interaction, but we make assumptions of parameters for illustrative purposes. Hence the shape of the figures or time of epidemic take-off in our simulations may change if the model is fitted or calibrated to the data of a particular region. We suggest that this theoretical study be seen as a guide for future research and data collection.

Initial conditions used are:

\[
(S(0), I_S(0), U_H(0), A_H(0), T_H(0), U_{SH}(0), A_{SH}(0), T_{SH}(0)) = (5500, 6, 7, 5, 3, 4, 3, 2)
\]
which indicate the presence of both diseases in the community,

\[(S(0), I_S(0), U_H(0), A_H(0), T_H(0), U_{SH}(0), A_{SH}(0), T_{SH}(0)) = (5500, 0, 7, 5, 3, 0, 0, 0)\]  

(28)

which indicate the presence of only HIV infection in the community, and

\[(S(0), I_S(0), U_H(0), A_H(0), T_H(0), U_{SH}(0), A_{SH}(0), T_{SH}(0)) = (5500, 6, 0, 0, 0, 0, 0, 0)\]  

(29)

which indicate the presence of only syphilis infection in the community. Parameters in Table (2) are also used, except otherwise stated.

**Fig. 4** shows the HIV and syphilis epidemics with initial condition (27) and parameters in Table (2). If the reproduction number is less than unity \((R_{SH} = 0.139 < 1, R_{ES} = 0.025 < 1, R_e = 0.139 < 1)\) due to smaller transmission rates of HIV and syphilis \((\beta_H = 0.02, \beta_S = 0.1)\), the number of individuals living with HIV and/or syphilis decreases and converges to the asymptotically stable disease-free equilibrium (Fig. 4a). Biologically, both diseases go to extinction and the epidemics of HIV and syphilis die out in the community. In contrast, if the transmission rates are larger \((\beta_H = 0.4, \beta_S = 5.0)\) and \(R_e > 1 (R_{SH} = 2.780 > 1, R_{ES} = 1.245 > 1, R_e = 2.780 > 1)\), the number of infected individuals converges to the HIV-syphilis endemic equilibrium (Fig. 4b). This biologically means that the epidemics of both HIV and syphilis persist in the community. The simulations are consistent with Lemma 8 and Theorem 10.

Furthermore, **Fig. 5** shows the HIV and syphilis epidemics with initial condition (27). If the reproduction number of syphilis is greater than unity \((R_{SH} = 0.139 < 1, R_{ES} = 1.245 > 1, R_e = 1.245 > 1)\) due to a larger transmission rate of syphilis \((\beta_H = 0.02, \beta_S = 0.5)\), then the reproduction number of the co-infection system is greater than unity. The number of individuals mono-infected and co-infected with HIV persists for a long time and then decreases slowly to zero because of the long life time of people living with HIV (Fig. 5A and B). The number of individuals mono-infected with syphilis increases

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### Table 2

Model parameters and their interpretations.

| Symbol Parameter | Value \(\text{yr}^{-1}\) | Source |
|------------------|------------------|--------|
| \(\Pi\)          | 100              |        |
| \(\mu\)          | 0.017            |        |
| \(\mu\)          | 0.017            |        |
| \(\sigma_1\)     | 4               |        |
| \(\sigma_2\)     | 1/4              |        |
| \(\sigma_3\)     | 4               |        |
| \(\sigma_4\)     | 1/4              |        |
| \(\rho_2\)       | 2.5              |        |
| \(\rho_1\)       | 2.5              |        |
| \(\eta_1\)       | 0.9375           |        |
| \(\eta_2\)       | 0.9375           |        |
| \(\gamma\)       | 2.237, 2.237     |        |
| \(\phi_1, \phi_2\) | 2.867, 2.867    |        |
| \(\phi_3\)       | 2.867, 2.867     |        |
| \(\kappa_1\)     | 1.0              |        |
| \(\kappa_2\)     | 3.5              |        |
| \(\kappa_3\)     | 0.5              |        |
| \(\alpha_1\)     | 1/4              |        |
| \(\alpha_2\)     | 1/4              |        |
| \(\alpha_3\)     | 1/4              |        |
| \(\alpha_4\)     | 1/4              |        |
| \(\beta_S\)      | 0.1              |        |
| \(\beta_H\)      | 0.02             |        |
| \(\beta_S\)      | 5.0              |        |
| \(\beta_H\)      | 0.4              |        |

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Nwankwo and Okuonghae (2018)

Wangleong et al. (2017)

US Centers for Disease Control and Prevention (2017)

Hutchinson, Hook, Shepherd, Verley, and Anne (1994)

James, Banks, and Marchand (2000)
(Fig. 5C) and then becomes stable after about 6 years (the zoomed-in plot of $I_S$ in Fig. 5C) to converge to the asymptotically stable syphilis endemic equilibrium showing one possibility of Theorem 10, (3b). This biologically means that with our choice of parameters and over a long period of time, a community with smaller transmission rate of HIV and larger transmission rate of syphilis will experience syphilis epidemics, while the epidemic of HIV will die out. In this case, the maximum reproduction number of the HIV-syphilis full model will be the reproduction number of the syphilis sub-model.

Fig. 6 similarly shows the HIV and syphilis epidemics with initial condition (27). If the reproduction number of HIV is greater than unity ($R_{eH} = 2.780 > 1$; $R_{eS} = 0.025 < 1$; $R_e = 2.780 > 1$) due to a larger transmission rate of HIV ($b_H = 0.4$, $b_S = 5.0$), then the reproduction number of the co-infection system is greater than unity. The number of individuals mono-infected and co-infected with syphilis decrease to zero (Fig. 6B and C) in less than 2 years (the zoomed-in plot of $I_S$ in Fig. 6C) since syphilis is curable. The number of individuals mono-infected with HIV infection first increase to a maximum value and then decrease to converge to the asymptotically HIV endemic equilibrium (Fig. 6A) showing one possibility of Theorem 10, (3a). This biologically means that with our choice of parameters, a community with larger transmission rate of HIV and smaller transmission rate of syphilis will experience the HIV epidemic, while the syphilis epidemic will die out. In this case, the maximum reproduction number of the HIV-syphilis full model will be the reproduction number of HIV sub-model.

Fig. 7 shows the impact of the presence of one disease on the other in a community where either one or both diseases persist at the initial stage of the epidemic. Fig. 7a shows the number of individuals living with HIV using initial conditions (27)
It is worth noting that the steady state in blue line is about 5% higher than the one in red line, which indicates that, for the same community, the presence of syphilis infection is likely to enhance the HIV prevalence in comparison to no syphilis infection and efforts towards eradicating syphilis infection may in turn decrease HIV prevalence.

Fig. 7a shows the prevalence of HIV with syphilis at the initial stage of the epidemic (initial condition (27), blue dashed line) and without syphilis (initial condition (28), red solid line). (b) Fig. 7b shows the prevalence of syphilis infection with HIV at the initial stage of the epidemic (initial condition (27), blue dashed line) and without HIV (initial condition (29), red solid line).

7. Discussion and conclusion

We presented a mathematical model that rigorously analysed the co-interaction of HIV and syphilis infections in the presence of treatment of both diseases. We carried out the stability analysis of disease-free and endemic equilibria, and showed that...
(a) disease-free equilibria for sub-models and the full model were locally and asymptotically stable whenever their respective reproduction numbers are less than unity.

(b) endemic equilibria for sub-models and the full model were locally and asymptotically stable whenever their respective reproduction numbers are greater than unity.

(c) increasing testing and treatment rate of mono-infected individuals with syphilis may bring the reproduction number of syphilis below unity, and thereby eradicating the disease among mono-infected individuals in the community.

(d) increasing the testing rate, treatment rate and reducing the rate of treatment fallout for mono-infected individuals impact HIV epidemic by lowering the reproduction number of HIV, but may not be able to eradicate the disease in the community.

Despite the limitations of assuming homogeneous mixing populations and using parameter values from published articles, our results and analyses of the reproduction number indicated that:

(a) HIV infection increases syphilis prevalence and vice versa.

(b) we could bring the reproduction number of syphilis below unity if syphilis is tested and treated more, but testing and treating cases of HIV alone may not be sufficient to bring down the prevalence of HIV as this may depend on some other factors, for example, some parameters in Equation (10) (lower HIV-related death, increase time retained on treatment and so on).

Great attention have not been given to the negative effect of the co-interaction of HIV and syphilis globally, and there are not many mathematical models that considered synergistic interactions with treatment of both diseases among gbMSM population. Even though our approach is similar to those considered in the literature (Bhunu et al., 2009; Brauer & Castillo-Chavez, 2012; Braueret al., 2012; Caro-VegaCarlos del Rio et al., 2015; Funke David, 2015; Lih-Ing et al., 2009; Mushayabas et al., 2011; Nwankwo & Okuonghaye, 2018) in terms of the joint dynamics of both diseases, but treatment of both HIV and syphilis infections among gbMSM population is an essential difference that none of those studies examined. Our model can be extended to include general population, and can also be stratified into different age group or risk level.

Declaration of competing interest

None to declare.

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