



Article

HIV/AIDS Mathematical Model of Triangle Transmission.

Cristian Camilo Espitia Morillo¹0000-0001-5500-0460, João Frederico da Costa Azevedo Meyer²0000-0001-8002-7075

¹ Universidad de los Llanos; cristian.espitia@unillanos.edu.co
² Universidade Estadual de Campinas; joni@unicamp.br
* Correspondence: espitiacristian@gmail.com; Tel.: (+57) 3156399888

Abstract: In this paper mathematical analysis of the HIV / AIDS deterministic model exposed in [Espitia, C. et. al. Mathematical Model of HIV / AIDS Considering Sexual Preferences Under Antiretroviral Therapy, a Case Study in San Juan de Pasto, Colombia, Journal of Computational Biology 29 (2022) 483–493] is made. The objective is to gain insight into the qualitative dynamics of the model determining the conditions for the persistence or effective control of the disease in the community through the study of basic properties such as positiveness and boundedness, calculus of basic reproduction number, stationary points such as disease free equilibrium (DFE), boundary equilibrium (BE) and endemic equilibrium (EE) are calculated, local stability (LAS) of disease free equilibrium. It research allow to conclude that the best way to reduce contagion and consequently to reach a DFE is thought to be the reduction of homosexual partners rate as they are the most affected population by the virus, and are therefore the most likely to become infected and to spread the infection. Increasing the departure rate of infected individuals, leads to a decrease in untreated infected heterosexual men and untreated infected women.

Keywords: HIV / AIDS Mathematical Model; Basic Reproduction Number; Stationary Points; Local and Global Stability Analysis.

1. Introduction

Epidemiological evidence shows that HIV is transmitted only through the exchange of body fluids such as blood, semen, vaginal or anal secretions and breast milk. As a result, the highly common means of transmission are: unprotected sex, from mother to child during pregnancy, childbirth or breast feeding, injecting drugs with a needle that has infected blood, infected blood donation or organ transplant, [22]. There are lots of myths and misconceptions about how a person can get HIV. It is not transmitted through body fluids such as sweat, tears or saliva, touching someone who has HIV, mosquito bites or others.

The sexual transmission of HIV is usually considered heterosexual, or homosexual men by anal intercourse. Transmission from female to female is almost null, however this form is possible by sharing toys such as sexual vibrators, [3,12]. Female homosexual contact has not been demonstrated to pose appreciable HIV transmission risk and such transmission appears to be rare, [13,14]. According to communication with the HIV / AIDS infectious disease specialist Dr. Alexandre Naime Barbosa; the sexual transmission between men can occur three mechanisms; exclusive homosexual transmission, exclusive heterosexual transmission or bisexual transmission, while in women the transmission is almost always heterosexual. The Center for Disease Control and Prevention estimates that HIV rates in men who have sex with men (MSM) is higher than rate in heterosexual contacts. In part, these differences reflect the fact that an individual MSM can engage in both insertive and receptive sexual roles (versatility), while exclusively heterosexual men and women each engage in only one of these roles, [2,9].

When discussing transmission the term “Discordant Couples” will be used to represent a couple in which one partner has a sexually transmitted disease, while the other partner

does not. If two participants are infected the transmission could imply co-infection which is not the objective in this investigation. However, if the two participants are susceptible then there is no contagion.

The risk of acquiring HIV is: 22 times higher among men who have sex with men (MSM), 22 times higher among individuals who are injectable drugs users (IDU) that shared needles, 21 times higher for sex workers, and 12 times higher for transgender people compared to the risk of transmission in heterosexual contact [17,23]. One form of measuring how transmissible some disease is the “Basic Reproduction Number”, which describes secondary infections from a first infection, it depends on the contagion form. For example, for HIV/AIDS transmission the basic reproduction number is 4 in homosexual population in the United Kingdom, whereas the basic number is 11 for female prostitutes in Kenya, [11]. As a result of the varying in these statistics, we consider the homosexual transmission to be greater than heterosexual transmission.

In triangle transmission model, it is assumed that the only way to transmit HIV virus is through sexual intercourse, and it commonly considered that the contagion form takes into account heterosexuals and homosexuals in the dynamic of infection, but, Can the population be split into heterosexuals and homosexuals and thus the group of bisexuals be ignored? and, What is the contribution of these group in the HIV transmission? To try to answer these questions we propose a different mathematical model considering HIV infected bisexuals under ART. Several articles have also focused on the whole population of constant size when considering force of infection, although some studies such as [16,19] have stressed the importance of variable population size in epidemic dynamics. All these assumptions such as sexual preference and variable population in force of infection are considered in our model.

In regards sexual contact between homosexual men, heterosexual men and women [18,21] say: "There exist individuals that change their sexual behavior depending on the situation or at different stages in their life. A possibly common and transient example of situational sexuality is the person who self-identifies as heterosexual, but will sexually interact with a member of the same sex when lacking other opportunities. Less transient but also possibly common, a person who self-identifies as gay or lesbian (either at the time, or later) may sexually interact with a member of the opposite sex if a same-sex relationship seems unfeasible". Thus, in our model we consider bisexual contact.

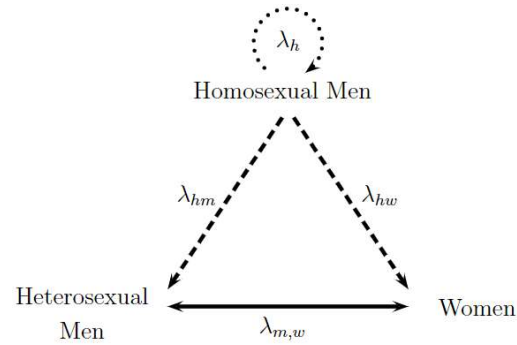
2. Materials and Methods

The epidemiological model under consideration was studied in [8], it contains three population groups: the first men with homosexual preference in men, the second one men with heterosexual preference and the third one for women who may be homosexuals or heterosexuals, but engage in sexual relations with homosexual or heterosexual men. We supposed that eventually homosexual men had sexual contact with women, and that heterosexual men had sexual relation with homosexual men. Consequently, we consider bisexual behavior among these groups because the transmission from homosexuals to heterosexual men or women goes through the bisexuals. Female homosexual transmission is not considered in the dynamic of infection. For more information see references [3,12].

The total population $N(t)$ is divided into 8 classes; $S_h(t)$ represents susceptible homosexual men, $I_h(t)$ untreated infected homosexual men, $S_w(t)$ susceptible women, $I_w(t)$ untreated infected women, $S_m(t)$ susceptible heterosexual men, $I_m(t)$ untreated infected heterosexual men, $T(t)$ treated individuals on ART and $A(t)$ individuals living with AIDS.

Figure 1 represents the transmission dynamics between the three studied sexual preferences. Each vertex of the triangle represents one population, and the sides of triangle denote the different forms of transmission between the populations involved. To begin, the exclusive transmission among homosexual men is illustrated by the upper circular dotted arrow labeled as λ_h . Then, the transmission between homosexual and heterosexual men, and the transmission between homosexual men and women are represented by dashed

Figure 1. Triangle Transmission in Sexual Preferences: Homosexual Men, Heterosexual Men and Women. Adapted of [8].



lines identified as λ_{hm} and λ_{hw} , respectively. Finally, heterosexual transmission between men and women is continuous line represented by $\lambda_{m,w}$. The direction of the arrows represents the sense of the analyzed contagion; nonetheless, contagions can biologically occur in all directions. Consequently, it is assumed two following hypotheses: the only form of contagion among homosexuals is among themselves, and heterosexual people become infected due to the contact with homosexual men or heterosexuals of the opposite sex. Thus, dashed lines have only one direction, while the continuous line between heterosexual men and women has two. The following assumed hypotheses in the model were evaluated by HIV/AIDS specialist Dr. Alexandre Naime Barbosa in Stadual University of Sao Paulo, UNESP, Botocatu, Brazil.

Assumed Hypotheses in Model III

- H1** Constant recruitment in all susceptible classes is assumed.
- H2** Sexual transmission in discordant couples is considered.
- H3** Homosexual individuals get infected among themselves. The HIV transmission in female susceptible population happens through sexual relations with infected heterosexual men or with infected homosexual men. Susceptible heterosexual men can get infected by infected women or infected homosexual men.
- H4** There is no gender differentiation in either sexual preference in treated individuals or individuals living with AIDS.
- H5** Individuals living with AIDS could be treated or untreated, noting that an individual that developed AIDS during a hospital treatment will be diagnosed and enrolled in ART.
- H6** It is considered both natural mortality in all classes and induced mortality in individuals living with AIDS.

Parameters in the model

The constant recruitment in all susceptible classes is denoted by Ψ . Men proportion is labeled by θ , $0 \leq \theta \leq 1$. Heterosexual proportion is represented by γ , $0 \leq \gamma \leq 1$. Proportion of initially treated individuals is p , $0 \leq p \leq 1$, consequently $(1 - p)$ denotes the proportion of untreated. Natural mortality rate is symbolized by μ . Induced mortality rate in individuals living with AIDS is d . AIDS development rate in treated individuals is δ . Departure rate of infected individuals is α . Subscripts s, h, hw, hm mean sexual contact between heterosexual men and women, among homosexual men, between homosexual men and women and finally between homosexual men and heterosexual men, respectively; thus $\beta_{s,h,hw,hm}$ represents probability of transmission and $c_{s,h,hw,hm}$ mean sexual partners rate in the aforementioned contacts. $B_h = c_h \beta_h$, $B_s = c_s \beta_s$, $B_{hm} = c_{hm} \beta_{hm}$, $B_{hw} = c_{hw} \beta_{hw}$ rates will be considered for parameter simplification. All parameters are non negatives and are listed in table 1.

Parameter	Description
Ψ	Constant Recruitment
θ	Men Proportion
γ	Heterosexual Proportion
p	Proportion of Initially Treated Individuals
μ	Natural Mortality Rate
d	Induced Disease Mortality Rate
δ	AIDS Development Rate in Treated Individuals
α	Departure Rate of infected individuals
$\beta_{s,h,hw,hm}$	Sexual Transmission Probability
$c_{s,h,hw,hm}$	Sexual Partners Rate.

Table 1. Description of Parameters. Adapted of [8].

Initially treated individuals and individuals living with AIDS receiving ART are disregarded from the transmission because their viral load is negligible. In figure 1, we assume that susceptible homosexual men only get infected by infected homosexual men, and susceptible women (or men) get infected by infected men (or women) or infected homosexual men. This means that susceptible homosexual men selects their partner randomly from the infected homosexual population, while women or men selects her/his partner randomly from the infected heterosexual or infected homosexual population. Adapted of [8].

The force of infection or disease incidence function measures the susceptible person’s risk to becoming infected. In some epidemic models, this function is assumed to be bilinear in both the infected individuals and the susceptible individuals. Besides, a bilinear force of infection or mass action law incidence may not yield appropriate results for several reasons. In particular, this force of infection does not permit to consider the difference among infected individuals. Thus, we decided that since this function represents the contact between an infected person and a susceptible one, the denominator would have to be only formed by susceptible individuals, and those who can transmit the disease. Excluding both treated individuals and people living with AIDS because under ART, their viral charge is negligible; besides, people living whit AIDS are too sick, and their sexual life can be considered as almost null. Therefore, the following infection forces by sexual contact are:

$$\lambda_h = B_h \frac{I_h}{S_h + I_h}$$

Exclusive Homosexual Contact,

$$\lambda_{hw} = B_{hw} \frac{I_h}{S_m + S_h + I_m + I_h}$$

Contact between Homosexual Men and Women,

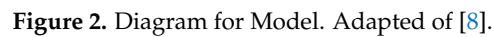
$$\lambda_{hm} = B_{hm} \frac{I_h}{S_m + S_h + I_m + I_h}$$

Contact between Homosexual Men and Heterosexual Men,

$$\lambda_{m,w} = B_s \frac{I_{m,w}}{S_{m,w} + I_{m,w}}$$

Heterosexual Contact.

It is important to perceive that in exclusive homosexual contact, the fraction denotes untreated infected homosexual men among susceptible and untreated infected homosexual men. Whereas, in the contact between homosexual men and women, the fraction denotes untreated infected homosexual men among susceptible heterosexual men and untreated homosexual men because in this contact, is considered bisexual behavior. The same reasoning should be made for the contact between homosexual men and heterosexual men. For heterosexual contact, the fraction denotes untreated infected heterosexual men (women) among susceptible heterosexual men (women) and untreated infected heterosexual men (women). The compartmental model is presented in figure 2. The dynamic is governed by the system of nonlinear ordinary differential equations (1)–(8), where a dot represents differentiation with respect to t .



$$\dot{A} = \alpha(1-p)(I_h + I_w + I_m) + \delta T - (d + \mu)A. \quad (8)$$

160

161

162

163

111

4.3.3

166

acquire the virus with rate λ_{hw} and λ_{hm} . Finally, they can die from natural causes with rate μ .

Infected individuals such as homosexual men, women, and heterosexual men $I_h(t)$, $I_w(t)$ and $I_m(t)$, grows with the infection in rates λ_h , λ_m , and λ_w . However, women and heterosexual men grow with rates λ_{hw} and λ_{hm} , respectively. This infected population reduces because its individuals become treated or people living with AIDS in rates α and $\alpha(1 - p)$, respectively. Finally, they die from natural causes with rate μ .

Treated individuals, $T(t)$, grow because of the entrance of infected ones enrolled in ART and they develop AIDS with rate δ or die from natural causes with rate μ .

Individuals living with AIDS, $A(t)$ grows due of the entrance of infected ones without treatment or treated whom develop AIDS; they die from natural causes with rate μ and from induced disease death with rate d .

The correspondent mathematical analysis of this ordinary differential equations system is developed as follows.

2.1. Positiveness and Boundedness

Theorem 1. Let be the initial conditions $S_h(0) > 0$, $I_h(0) \geq 0$, $S_w(0) \geq 0$, $I_w(0) \geq 0$, $S_m(0) \geq 0$, $I_m(0) \geq 0$, $T(0) \geq 0$, $A(0) \geq 0$. Then the solutions $S_h(t)$, $I_h(t)$, $S_w(t)$, $I_w(t)$, $S_m(t)$, $I_m(t)$, $T(t)$, $A(t)$ of the system (1) to (8) will be positive for all time $t > 0$.

Proof. Let $t_1 = \sup\{t > 0 : S_h(t) > 0, I_h(t) > 0, S_w(t) > 0, I_w(t) > 0, S_m(t) > 0, I_m(t) > 0, T(t) > 0, A(t) > 0\}$. From the first equation (1), we have

$$\frac{dS_h}{dt}(t) = \Psi\theta(1 - \gamma) - B_h \frac{I_h(t)}{S_h(t) + I_h(t)} S_h(t) - \mu S_h(t) = \Psi\theta(1 - \gamma) - (\lambda_h(t) + \mu) S_h(t).$$

Which can be re-written as

$$\begin{aligned} \frac{d}{dt} \left(S_h(t) \exp \left[\mu t + \int_0^t \lambda_h(\tau) d\tau \right] \right) &= \Psi\theta(1 - \gamma) \exp \left[\mu t + \int_0^t \lambda_h(\tau) d\tau \right] \\ S_h(t_1) \exp \left[\mu t_1 + \int_0^{t_1} \lambda_h(\tau) d\tau \right] - S_h(0) &= \Psi\theta(1 - \gamma) \int_0^{t_1} \exp \left[\mu y + \int_0^y \lambda_h(\tau) d\tau \right] dy \\ S_h(t_1) &= S_h(0) \exp \left[-\mu t_1 - \int_0^{t_1} \lambda_h(\tau) d\tau \right] \\ &\quad + \exp \left[-\mu t_1 - \int_0^{t_1} \lambda_h(\tau) d\tau \right] \Psi\theta(1 - \gamma) \int_0^{t_1} \exp \left[\mu y + \int_0^y \lambda_h(\tau) d\tau \right] dy \geq 0. \end{aligned}$$

Similarly, it can be shown that $I_h(t)$, $S_w(t)$, $I_w(t)$, $S_m(t)$, $I_m(t)$, $T(t)$, $A(t)$ are non-negatives for all time $t > 0$. In this way all solutions of the system remain positive for all non-negative initial conditions. \square

Theorem 2. All the solutions of the system (1) to (8) are uniformly bounded. It means any trajectory stars in \mathbb{R}_8^+ remains in \mathbb{R}_8^+ for all time $t \geq 0$.

Proof. Adding all eight equations from (1) to (8), gives

$$\begin{aligned}\frac{dN}{dt} &= \Psi - \mu N - dA \\ &\leq \Psi - \mu N.\end{aligned}$$

Solving the differential in-equation, we have

$$N(t) \leq \left(N(0) - \frac{\Psi}{\mu}\right) \exp(-\mu t) + \frac{\Psi}{\mu}. \quad (9)$$

Therefore all solutions of the system will enter into the region

$$\Omega_{III} = \left\{ \left(S_h(t), I_h(t), S_w(t), I_w(t), S_m(t), I_m(t), T(t), A(t) \right) \in \mathbb{R}_8^+ : N(t) \leq \frac{\Psi}{\mu} \right\}. \quad (10)$$

In equation (9) if $N(0) \leq \frac{\Psi}{\mu}$ then $N(t) \leq \frac{\Psi}{\mu}$, if $N(0) \geq \frac{\Psi}{\mu}$ then either the solution enters in Ω_{III} in finite time or $N(t)$ approaches $\frac{\Psi}{\mu}$ asymptotically. Therefore Ω_{III} attracts all solutions in \mathbb{R}_8^+ .

□

The previous theorems allow us to conclude that the region Ω_{III} is a positively invariant set.

2.2. Basic Reproduction Number

The basic reproduction number, R_0 , determines the ability of the virus to develop and persist in the population. It is the average number of individuals that a single infected individual can infect during his life time when introduced into a wholly susceptible population. If $R_0 < 1$ then on average a few infected individuals brought into a fully susceptible population will not be able to replace themselves and the disease will not spread. If $R_0 > 1$ then the number of infected individuals will increase with each generation and the disease will spread.

In this research we use the next generation matrix method as presented in [7]. This method is as follows:

Let $x = (x_1, x_2, \dots, x_n)^T$ be the number of individuals in each compartment, where the first $m < n$ compartments contain infected individuals. Consider these equations written in the form

$$\dot{x}_i = f_i(x) = \mathcal{F}_i(x) - \mathcal{V}_i(x), \quad \text{for } i = 1, \dots, m. \quad (11)$$

In this splitting, $\mathcal{F}_i(x)$ is the rate of appearance of new infections in compartment i and $\mathcal{V}_i(x) = \mathcal{V}_i^-(x) - \mathcal{V}_i^+(x)$, where $\mathcal{V}_i^+(x)$ is the rate of transfer of individuals into compartment i by all other and $\mathcal{V}_i^-(x)$ is the rate of transfer of individuals out of the i compartment.

Note that $\mathcal{F}_i(x)$ include only infections that are newly arising, but does not include terms which describe the transfer of infectious individuals from one compartment to another. Let $X_s = \{x \geq 0 \mid x_i = 0, i = 1, \dots, m\}$ be the DFE. Assume that \mathcal{F}_i and \mathcal{V}_i satisfy the following axioms outlined by [7].

- (A₁) If $x \geq 0$ then $\mathcal{F}_i, \mathcal{V}_i^+, \mathcal{V}_i^- \geq 0$ for $i = 1, \dots, m$.
- (A₂) If $x_i = 0$, then $\mathcal{V}_i^- = 0$. In particular, if $x \in X_s$ then $\mathcal{V}_i^- = 0$ for $i = 1, \dots, m$.
- (A₃) $\mathcal{F}_i = 0$ if $i > m$.
- (A₄) If $x \in X_s$, then $\mathcal{F}_i(x) = 0$ and $\mathcal{V}_i^+ = 0$ for $i = 1, \dots, m$.
- (A₅) All eigenvalues of $Df(x_0)$ have negative real parts, where $Df(x_0)$ is the Jacobian matrix evaluated at the disease free equilibrium x_0 .

Theorem 3. Exposed in [7]

If x_0 is the disease free equilibrium (DFE) and $f_i(x)$ satisfies $(A_1) - (A_5)$, then the derivatives $D\mathcal{F}(x_0)$ and $D\mathcal{V}(x_0)$ are partitioned as

$$D\mathcal{F}(x_0) = \begin{pmatrix} F & 0 \\ 0 & 0 \end{pmatrix}, \quad D\mathcal{V}(x_0) = \begin{pmatrix} V & 0 \\ J_3 & J_4 \end{pmatrix}.$$

Where F and V are the $m \times m$ matrices defined by

$$F = \left[\frac{\partial \mathcal{F}_i}{\partial x_j}(x_0) \right], \quad V = \left[\frac{\partial \mathcal{V}_i}{\partial x_j}(x_0) \right] \quad \text{with } 1 \leq i, j \leq m.$$

Further, F is non-negative, V is a non-singular M -matrix and all eigenvalues of J_4 have positive real part.

According to [6], FV^{-1} is called the next generation matrix for model (11), and the spectral radius (dominant eigenvalue) is the basic reproduction number.

$$R_0 = \rho(FV^{-1}). \quad (12)$$

Theorem 4. Exposed in [7]

Consider the disease transmission model given by (11) with $f(x)$ satisfying conditions $(A_1) - (A_5)$. If x_0 is a DFE of the model, then x_0 is locally asymptotically stable if $R_0 < 1$, but unstable if $R_0 > 1$, where R_0 is defined by equation (12).

The basic reproduction number is defined as the spectral radius of the matrix FV^{-1} , and denoted by

$$R_0 = \max \left\{ \frac{B_s}{\alpha + \mu}, \frac{B_h}{\alpha + \mu} \right\} = \max \{ R_0^{het}, R_0^{hom} \}. \quad (13)$$

Details are exposed in Appendix A.

2.3. Stationary Points

For calculate stationary points we solve the associated homogeneous system (14) – (21), state variables with a star (*) superscript will be assumed to be equilibrium value.

$$0 = \Lambda_h - B_h \frac{I_h^*}{S_h^* + I_h^*} S_h^* - \mu S_h^*, \quad (14)$$

$$0 = B_h \frac{I_h^*}{S_h^* + I_h^*} S_h^* - (\alpha + \mu) I_h^*, \quad (15)$$

$$0 = \Lambda_w - B_s \frac{I_m^*}{S_m^* + I_m^*} S_w^* - B_{hw} \frac{I_h^*}{S_m^* + S_h^* + I_m^* + I_h^*} S_w^* - \mu S_w^*, \quad (16)$$

$$0 = B_s \frac{I_m^*}{S_m^* + I_m^*} S_w^* + B_{hw} \frac{I_h^*}{S_m^* + S_h^* + I_m^* + I_h^*} S_w^* - (\alpha + \mu) I_w^*, \quad (17)$$

$$0 = \Lambda_m - B_s \frac{I_w^*}{S_w^* + I_w^*} S_m^* - B_{hm} \frac{I_h^*}{S_m^* + S_h^* + I_m^* + I_h^*} S_m^* - \mu S_m^*, \quad (18)$$

$$0 = B_s \frac{I_w^*}{S_w^* + I_w^*} S_m^* + B_{hm} \frac{I_h^*}{S_m^* + S_h^* + I_m^* + I_h^*} S_m^* - (\alpha + \mu) I_m^*, \quad (19)$$

$$0 = \alpha p (I_h^* + I_w^* + I_m^*) - (\delta + \mu) T^*, \quad (20)$$

$$0 = \alpha (1 - p) (I_h^* + I_w^* + I_m^*) + \delta T^* - (d + \mu) A^*. \quad (21)$$

Where

233
234
235

236

237
238

239
240

241
242
243
244

245

246

247
248

249

$$\Lambda_h = \Psi\theta(1 - \gamma), \quad \Lambda_w = \Psi(1 - \theta), \quad \Lambda_m = \Psi\theta\gamma, \quad B_h = c_h\beta_h, \quad B_s = c_s\beta_s, \\ B_{hm} = c_{hm}\beta_{hm}, \quad B_{hw} = c_{hw}\beta_{hw}.$$

Thus, stationary points are

- Disease Free Equilibrium

This happens when $I_h^* = I_w^* = I_m^* = 0$ and represents absence of infection. It is

$$E_0 = \left(\frac{\Lambda_h}{\mu}, 0, \frac{\Lambda_w}{\mu}, 0, \frac{\Lambda_m}{\mu}, 0, 0, 0 \right). \quad (22)$$

- Boundary Equilibrium

This occurs when $I_h^* = 0$, the men homosexual population is null, and I_w^*, I_m^* are non zero, The subscript (*) means boundary equilibrium coordinate, it is

$$E_1 = \left(\frac{\Lambda_h}{\mu}, 0, \overline{S_w^*}, \overline{I_w^*}, \overline{S_m^*}, \overline{I_m^*}, \overline{T^*}, \overline{A^*} \right), \quad \text{where}$$

$$\overline{S_w^*} = \frac{\Lambda_w}{B_s - \alpha}, \quad \overline{S_m^*} = \frac{\Lambda_m}{B_s - \alpha}, \\ \overline{I_w^*} = \frac{\Lambda_w}{B_s - \alpha} [R_0^{het} - 1], \quad \overline{I_m^*} = \frac{\Lambda_m}{B_s - \alpha} [R_0^{het} - 1], \\ \overline{T^*} = \frac{\Psi p \alpha [1 - \theta(1 - \gamma)]}{(B_s - \alpha)(\delta + \mu)} [R_0^{het} - 1], \quad \overline{A^*} = \frac{\Psi \alpha [1 - \theta(1 - \gamma)] [\delta + \mu(1 - p)]}{(B_s - \alpha)(\delta + \mu)(d + \mu)} [R_0^{het} - 1]. \quad (23)$$

Note that the boundary equilibrium only exists when $R_0^{het} > 1$ (implying $B_s > \alpha$).

- Endemic Equilibrium

This represents persistence of the infection, it is

$$E_2 = \left(S_h^*, I_h^*, S_w^*, I_w^*, S_m^*, I_m^*, T^*, A^* \right), \quad \text{where} \quad (24)$$

$$S_h^* = \frac{\Lambda_h}{B_h - \alpha}, \quad I_h^* = \frac{\Lambda_h}{B_h - \alpha} [R_0^{hom} - 1], \\ S_w^* = \frac{\Lambda_w - (\alpha + \mu)I_w^*}{\mu}, \quad S_m^* = \frac{\Lambda_m - (\alpha + \mu)I_m^*}{\mu}, \\ T^* = \frac{\alpha p [\Lambda_h(R_0^{hom} - 1) + (B_h - \alpha)(I_w^* + I_m^*)]}{(B_h - \alpha)(\delta + \mu)}, \quad (25) \\ A^* = \frac{\alpha (\delta + \mu(1 - p)) [\Lambda_h(R_0^{hom} - 1) + (B_h - \alpha)(I_w^* + I_m^*)]}{(B_h - \alpha)(\delta + \mu)(d + \mu)}.$$

$$I_w^* = \frac{\Lambda_w (\alpha I_m^* - \Lambda_m) \left[B_s I_m^* (B_h - \alpha) + B_{hw} \Lambda_h (R_0^{hom} - 1) \right] - B_s \Lambda_h \Lambda_w \mu R_0^{hom} I_m^*}{(\alpha + \mu) \left\{ (\alpha I_m^* - \Lambda_m) \left[(B_h - \alpha) \left(I_m^* (B_s - \alpha) + \Lambda_m \right) + B_{hw} \Lambda_h (R_0^{hom} - 1) \right] - \Lambda_h \mu R_0^{hom} \left(I_m^* (B_s - \alpha) + \Lambda_m \right) \right\}}. \quad (26)$$

I_m^* is given by the roots of fourth degree polynomial

$$p(I_m^*) = a_4 (I_m^*)^4 + a_3 (I_m^*)^3 + a_2 (I_m^*)^2 + a_1 (I_m^*) + a_0. \quad (27)$$

Coefficients a_0, a_1, a_2, a_3 and a_4 are shown in Appendix B.

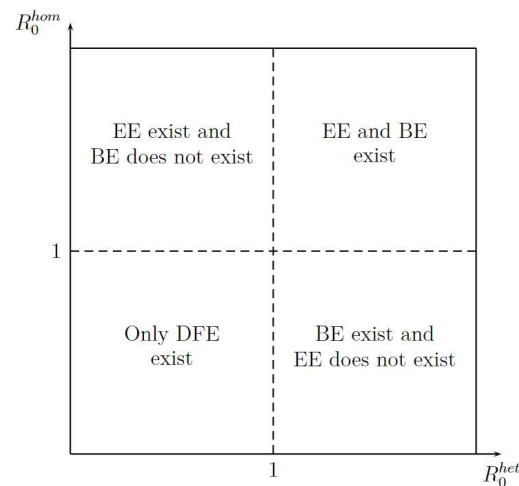


Figure 3. Stationary Points Existence

EE exists when $R_0^{hom} > 1$. For infected males and females the following inequalities (28) must be satisfied. Otherwise the populations of susceptible male S_m^* and female S_w^* will be negatives.

$$0 < I_m^* < \frac{\Lambda_m}{\alpha + \mu} \quad \text{and} \quad 0 < I_w^* < \frac{\Lambda_w}{\alpha + \mu}. \quad (28)$$

Figure 3 shows the existence of equilibrium points, such as Disease Free Equilibrium (DFE), Boundary Equilibrium (BE) and Endemic equilibrium (EE) in function of R_0^{het} and R_0^{hom} . It shows two important aspects; First, the DFE is the only one stationary point that exist when R_0^{hom} or R_0^{het} are less than one, it gives an idea of how stability can be. Second, for existence of EE the R_0^{hom} is more important that R_0^{het} because when R_0^{hom} is greater than 1 the EE exit, whereas when R_0^{het} is greater than 1 is necessary that R_0^{hom} will be greater than 1.

2.4. Local stability of Disease Free Equilibrium

Theorem 5. The DFE $E_0 = \left(\frac{\Lambda_h}{\mu}, 0, \frac{\Lambda_w}{\mu}, 0, \frac{\Lambda_m}{\mu}, 0, 0, 0 \right)$ is LAS if $R_0^{hom} < 1$ and $R_0^{het} < 1$, unstable when $R_0^{hom} > 1$.

Proof. LAS will be demonstrate with the eigenvalues of jacobian matrix related to the system (1) to (8) evaluated in E_0 , it is

$$J(E_0) = \begin{pmatrix} -\mu & -B_h & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & B_h - (\alpha + \mu) & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -B_{hw} \frac{1-\theta}{\theta} & -\mu & 0 & 0 & -B_s \frac{1-\theta}{\theta} & 0 & 0 \\ 0 & B_{hw} \frac{1-\theta}{\theta} & 0 & -(\alpha + \mu) & 0 & B_s \frac{1-\theta}{\theta} & 0 & 0 \\ 0 & -B_{hm} \gamma & 0 & -B_s \frac{\theta \gamma}{1-\theta} & -\mu & 0 & 0 & 0 \\ 0 & B_{hm} \gamma & 0 & B_s \frac{\theta \gamma}{1-\theta} & 0 & -(\alpha + \mu) & 0 & 0 \\ 0 & \alpha p & 0 & \alpha p & 0 & \alpha p & -(\delta + \mu) & 0 \\ 0 & \alpha(1-p) & 0 & \alpha(1-p) & 0 & \alpha(1-p) & \delta & -(d + \mu) \end{pmatrix} \quad (29)$$

The characteristic polynomial is

$$p(\lambda) = (\lambda + \mu)^3 (\lambda + d + \mu) (\lambda + \delta + \mu) (\lambda + \alpha + \mu - B_h) \left[(\lambda + \lambda + \mu)^2 - B_s^2 \right].$$

Eigenvalues are

261
262
263

264
265
266
267
268
269
270

271
272
273

274
275

276

Parameter	Baseline	Range	PDF	Source
γ	0.9	$[1e^{-1}, 1]$	Uniforme	Assumed
p	0.85	$[1e^{-2}, 1]$	Uniforme	Assumed
μ	0.0140	$[0.01, 0.02]$	Uniforme	[20]
α	0.3333	$[1e^{-2}, 1]$	Uniforme	[1]
B_h	2.64	$[0.05, 3.95]$	Uniforme	Assumed
B_s	0.04	$[1e^{-3}, 0.5]$	Uniforme	[10]
B_{hw}	0.04	$[1e^{-3}, 0.5]$	Uniforme	Assumed
B_{hm}	0.3	$[1e^{-3}, 0.5]$	Uniforme	Assumed

Table 2. Parameters used in Sensitivity Analysis through Latin Hipercube Sampling and Partial Rank Correlation Coefficients (LHS/PRCC).

$$\begin{aligned} \lambda_1 &= -\mu & \lambda_2 &= -\mu & \lambda_3 &= -\mu, \\ \lambda_4 &= -(d + \mu), & \lambda_5 &= -(\delta + \mu), & \lambda_6 &= -(\alpha + \mu + B_s), \\ \lambda_7 &= -(\alpha + \mu) \left[1 - R_0^{het} \right], & \lambda_8 &= -(\alpha + \mu) \left[1 - R_0^{hom} \right]. \end{aligned} \tag{30}$$

$R_0 = \max\{R_0^{hom}, R_0^{het}\} < 1$ imply $R_0^{hom} < 1$ and $R_0^{het} < 1$, thus, all eigenvalues are negatives it follows that E_0 is LAS. By other hand, if $R_0 > 1$, then $R_0^{hom} > 1$ or $R_0^{het} > 1$ implying that λ_7 or λ_8 will be positive respectively and in this case E_0 is unstable. \square

2.5. Sensitivity Analysis

A sensitivity analysis will help us better understand which of the parameters in the model we should focus on estimating most precisely, answering the questions: Which ones contribute most to output variability? and Which ones require additional research or are insignificant? These questions can be answered performing an analysis with Latin Hyerpcube Sampling (LHS) and Partial Rank Coefficient (PRCC). We use Matlab to solve the system or ordinary differential equations and to implement most of the SA functions described throughout the manuscript available in <http://malthus.micro.med.umich.edu/lab/usanalysis.html>.

The model contains 12 parameters, however to perform sensitivity analysis only parameters related to HIV infection and related to a basic reproduction number are considered, they are: $\gamma, p, \mu, \alpha, B_h, B_s, B_{hw}, B_{hm}$. According to [15] an uniform distribution was chosen over the Gaussian (normal) because we have no evidence that the ends of the ranges, and carrying out multiple runs (NR = 300), parameter, baseline, range and probability density function are listed in table 2. Partial Rank Correlation Coefficient was created for each infected population, in addition scatterplots for each parameter aforementioned are presented in figures 4, 5 and 6.

3. Discussion

This analysis focuses on identifying the main parameters that play a dominant role in three different response outputs such as, I_h , untreated infected homosexual men, I_w , untreated infected women and, I_m , untreated infected heterosexual men. These more sensitive parameters are: Departure Rate of infected individuals, α , Infection rates in homosexuals and heterosexuals, B_h and B_s , respectively. Scatterplots show the variation in the infected populations size with changes in parameters when examined, thus providing specific qualitative information on the relationship between an infected population and a parameter. Parameters with positive PRCCs will increase $I_{h,w,m}$ when their value is increased, whereas parameters with negative PRCCs will decrease $I_{h,w,m}$ when their value is increased. PRCC values are represented in figures 4, 5 and 6.

Figure 4. Prcc Diagram and Scatterplot for each Parameter in table 2 respect to Untreated Infected Homosexual Men, I_h .

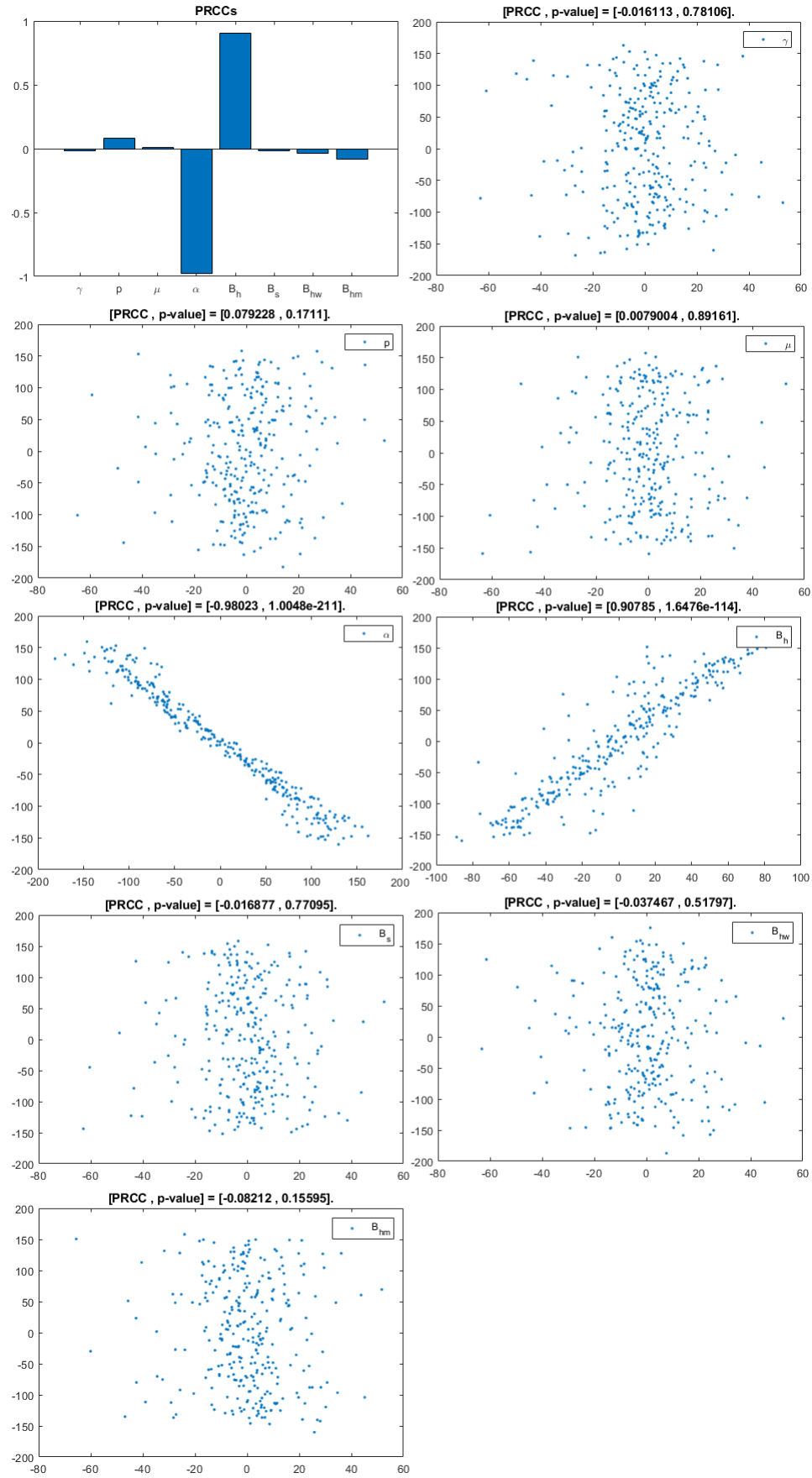


Figure 5. Prcc Diagram and Scatterplot for each Parameter in table 2 respect to Untreated Infected Women, I_w .

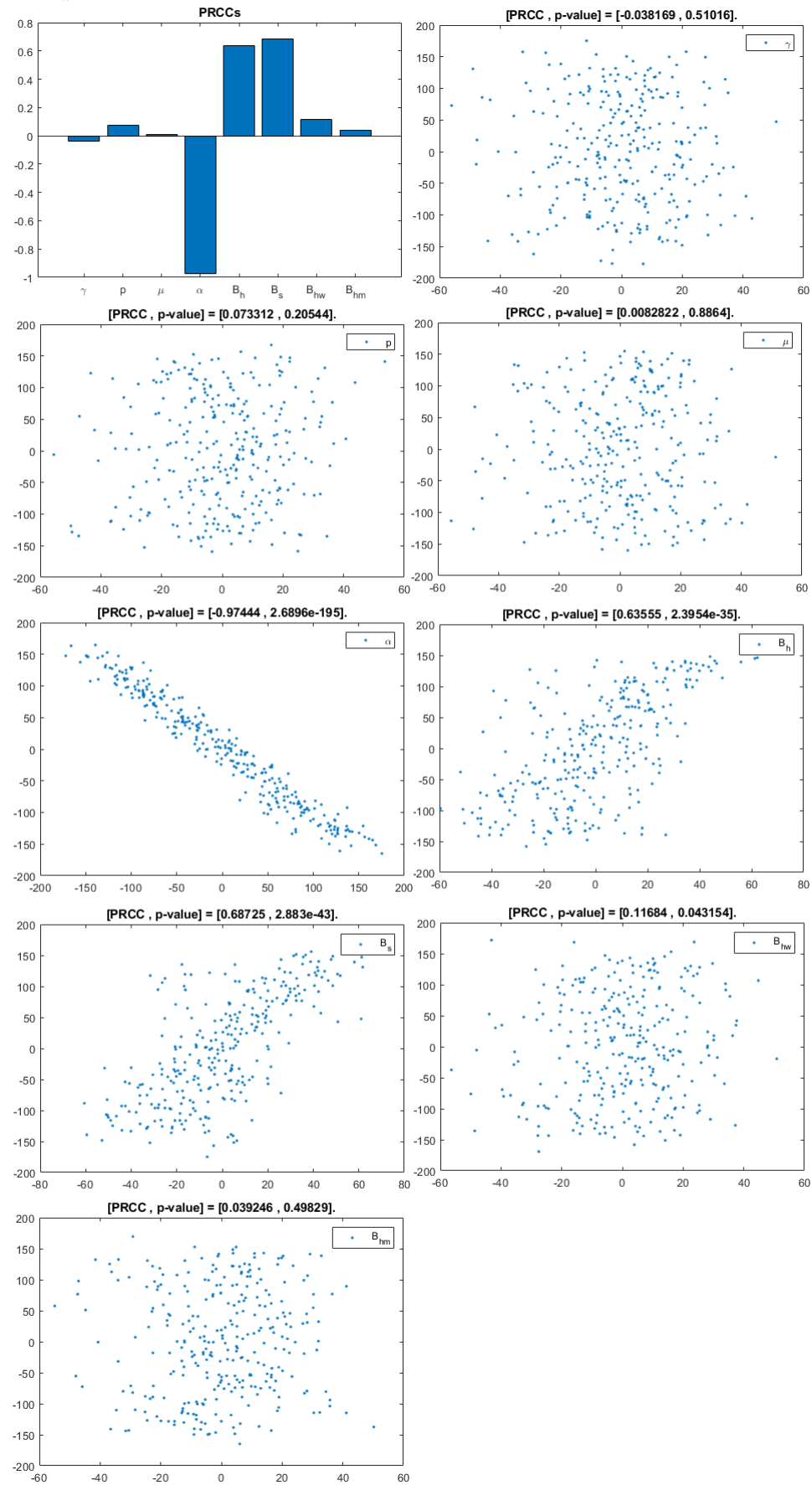
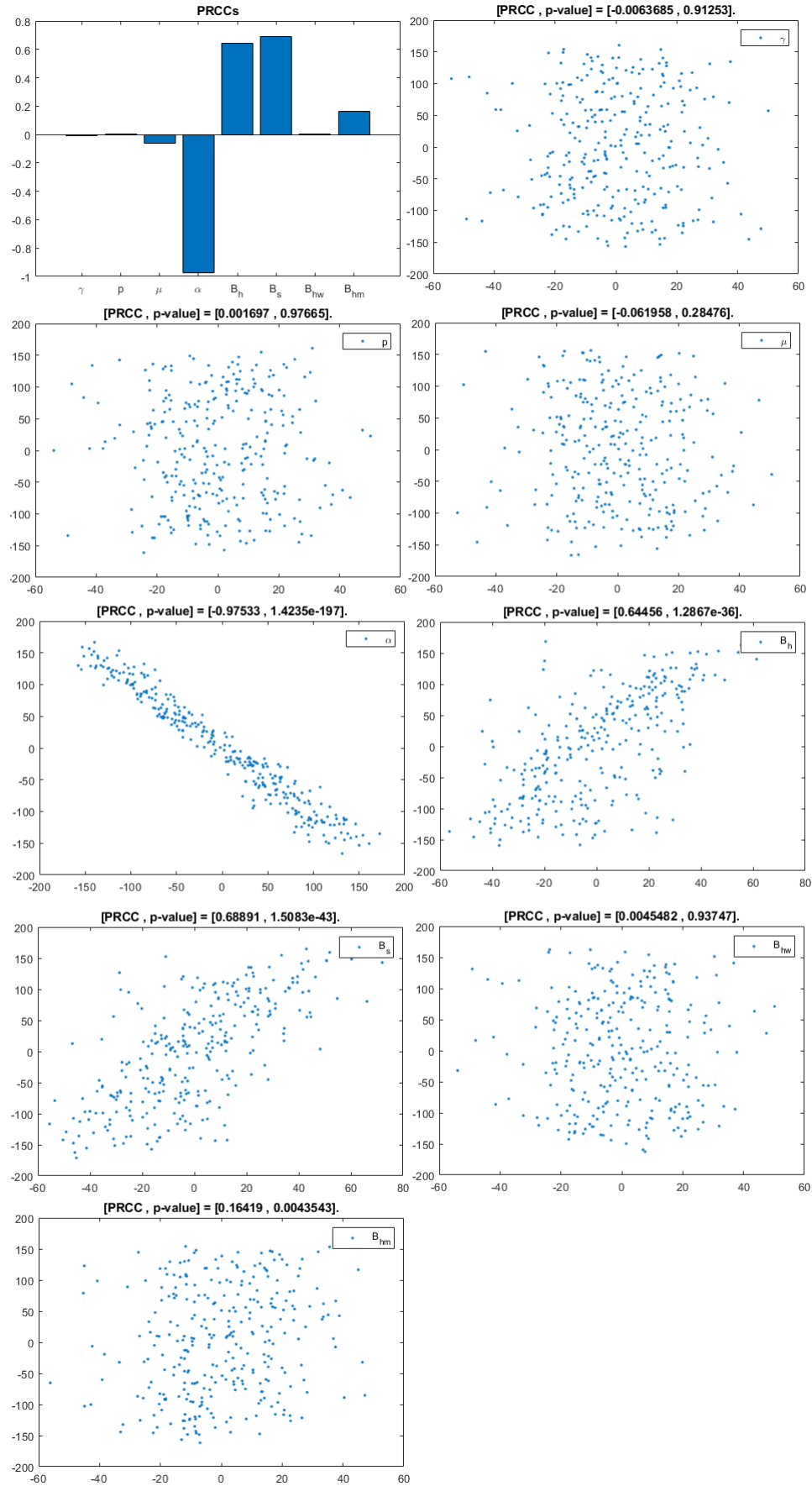


Figure 6. Prcc Diagram and Scatterplot for each Parameter in table 2 respect to Untreated Infected Heterosexual Men, I_m .



It follows from figure 4 that untreated infected homosexuals, I_h , has a negative correlation with α parameter, in fact $PRCC = -0.98023$, allow us to conclude that an increase in α parameter means a decrease in untreated infected men homosexual untreated, in figure 5 untreated infected women untreated, I_w , has a positive correlation with rate of infection in heterosexuals B_s , in fact $PRCC=0.68725$, thus an increase of heterosexual contact implies an increase of women infected, analogously, this population has a negative correlation with α parameter. Figure 6 allow us to conclude that γ parameter does not influence untreated infected heterosexual men, I_m , in fact $PRCC=-0.0063685$, showing that an increase in this parameter has little influence on the number of infected heterosexual men. In addition infection rates such as B_s and B_h have similar behavior in infected heterosexual men, I_m , as above for infected women population, I_w .

4. Conclusions

Models will be a tool for understanding the disease dynamics and for predicting possible trends. Obviously, more accurate predictions require more complex models with more classes and compartments; although such models are relatively easy to formulate, their mathematical analysis is difficult and obtaining the necessary social and sexual behavior data is more complicated. Several key features could be included to create more realistic HIV / AIDS models in human population, such as infectious class or injectable drug users through needle sharing transmission.

Sensitivity analysis for the eight parameters related to infection population allow to conclude that most influential parameter in the HIV dynamic is the departure rate for infected individuals, α , because it presents the highest PRCC coefficient. This behavior can be explained because α parameter is present in the basic reproduction number and governs how those infected people are emerging from untreated status to obtain treatment or to develop AIDS.

Bisexual parameters such as those of the probability of infection by sexual contact between homosexual men with heterosexual men, β_{hm} , and between homosexual men and women, β_{hw} , allow to conclude that higher values of β_{hm} and β_{hw} parameters imply a high infection in untreated infected women and heterosexual men. For decision-makers in establishing health policies.

Mathematics can provide for the decision maker, would it be the case to promote awareness campaigns aimed at this specific population. It research allow to conclude that the best way to reduce contagion and consequently to reach a DFE is thought to be the reduction of homosexual partners rate as they are the most affected population by the virus, and are therefore the most likely to become infected and to spread the infection. Increasing the departure rate of infected individuals, leads to a decrease in untreated infected heterosexual men and untreated infected women. However, it is not the only to prevent and curb the rate of contagion in San Juan de Pasto. Consequently, it is also necessary increasing antiretroviral treatment.

With the population parameters of San Juan de Pasto, several numerical simulations were performed by modifying parameters that turn the basic reproduction number greater than or less than one. This seems to suggest that when $R_0^{het} < 1$ and $R_0^{hom} > 1$, there is a general decline in the HIV infection over in the next few years, but the infection persists. As a result, we can conclude that the most important observation from our findings is that in the population, there is a short-term rise in HIV infection in which there exists a significant increase in new HIV infections followed by a decline in the generation of new infections.

The dynamic of HIV / AIDS epidemic, to a large extent, depends on changes of basic reproduction number for homosexuals, R_0^{hom} , which was also evidenced by modifying several parameters in the scenarios above. In the background section, it was mentioned that probability of HIV infection in homosexuals is bigger than in heterosexuals, thus, the basic reproduction number in heterosexuals, R_0^{het} , is less influential. In addition, investigations such as [4] and [5] permits to conclude that sexual partners rate in homosexuals is greater than sexual partners rate in heterosexuals, thus, $R_0^{hom} > R_0^{het}$. This suggests that HIV

infection can be controlled or eliminated from the community if control programs are directed towards reducing R_0^{hom} to values less than one. The model shows the persistence of the disease when $R_0^{hom} > 1$.

The dynamics of HIV/AIDS are, in general, too complex to allow for intuitive predictions and require the support of mathematical modeling for qualitatively and quantitatively assessing and understanding the functioning system. Furthermore, one of the most difficult tasks of mathematical modeling is obtaining parameters for a chosen model. Moreover, by using real parameter values to study and analyze the behavior in San Juan de Pasto, the proposed HIV/AIDS model tries to be as approximate as possible to the current situation of this infection. The emphasis was not on the accuracy of the scenarios, but on the actions that can be taken as a result of comprehending the state of the epidemic in the future. For example, scenario 5 shows that when the number of sexual partners is high, the basic reproduction number is greater than 1 and the infection spreads more easily, implying that more and more people are being treated with higher public health costs and, therefore, it is better and a lot more economical to invest in educational campaigns. These actions can involve, among other things, the prevention of new infections, provision and delivery of antiretroviral therapy and educational campaigns such as those that aim to reduce the number of sexual partners or the use of condom for self-protection.

This application in San Juan de Pasto show the effects of modifying the parameters related to infected populations. These variations imply in huge social and economic expenses which can be !and should be avoided through government actions such as educational campaigns. In this way, this research aims to be a useful tool in the design of establishing strategies for implementing valid public health policies and introducing efficient public health campaigns.

Funding: This research received no external funding

Conflicts of Interest: The authors declare no conflict of interest.

Abbreviations

The following abbreviations are used in this manuscript:

HIV	Human Immunodeficiency virus
AIDS	Acquired Immunodeficiency Syndrome
MSM	Men who have sex with men
IDU	Injectable Drug Users
LHS	Latin Hipercube Sampling
PRCC	Partial Rank Correlation Coefficients

Appendix A

Calculus of Basic Reproduction number

According to Lemma 3 to find the basic reproduction number is necessary to know the disease free equilibrium (DFE) of the model, it is

$$E_0 = \left(\frac{\Psi\theta(1-\gamma)}{\mu}, 0, \frac{\Psi(1-\theta)}{\mu}, 0, \frac{\Psi\theta\gamma}{\mu}, 0, 0, 0 \right).$$

(A. 1)

Let be $x = (I_h, I_w, I_m, T, A)$ the vector of infected population, and $\mathcal{F}(x)$ the vector with new infections, $\mathcal{V}^+(x)$ rate of transfer of individuals into compartment, $\mathcal{V}^-(x)$ rate of transfer of individuals out of compartment and $\mathcal{V}(x) = \mathcal{V}^-(x) - \mathcal{V}^+(x)$ the transfer vector and regarding terms. Thus if \dot{x} denotes the derivative of vector x , then $\dot{x} = f_i(x) = \mathcal{F}(x) - \mathcal{V}(x)$, where

$$\mathcal{F}(x) = \begin{pmatrix} B_h \frac{I_h}{S_h + I_h} S_h \\ B_s \frac{I_m}{S_m + I_m} S_w + B_{hw} \frac{I_h}{S_m + S_h + I_m + I_h} S_w \\ B_s \frac{I_w}{S_w + I_w} S_m + B_{hm} \frac{I_h}{S_m + S_h + I_m + I_h} S_m \\ 0 \\ 0 \end{pmatrix}, \quad \mathcal{V}^-(x) = \begin{pmatrix} (\alpha + \mu) I_h \\ (\alpha + \mu) I_w \\ (\alpha + \mu) I_m \\ (\delta + \mu) T \\ (d + \mu) A \end{pmatrix} \quad (\text{A. 2})$$

$$\mathcal{V}^+(x) = \begin{pmatrix} 0 \\ 0 \\ 0 \\ \alpha p(I_h + I_w + I_m) \\ \delta T + \alpha(1-p)(I_h + I_w + I_m) \end{pmatrix}, \quad \mathcal{V}(x) = \begin{pmatrix} (\alpha + \mu) I_h \\ (\alpha + \mu) I_w \\ (\alpha + \mu) I_m \\ (\delta + \mu) T - \alpha p(I_h + I_w + I_m) \\ (d + \mu) A - \delta T - \alpha(1-p)(I_h + I_w + I_m) \end{pmatrix} \quad (\text{A. 3})$$

Jacobian matrices for $\mathcal{F}(x)$ and $\mathcal{V}(x)$ evaluated in disease free equilibrium, $x_0 = E_0$, are given by

$$\mathcal{DF}(x_0) = F(E_0) = \begin{pmatrix} B_h & 0 & 0 & 0 & 0 \\ B_{hw} \frac{1-\theta}{\theta} & 0 & B_s \frac{1-\theta}{\theta\gamma} & 0 & 0 \\ c_{hm} \beta_{hm} \gamma & B_s \frac{\theta\gamma}{1-\theta} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}, \quad (\text{A. 4})$$

$$\mathcal{DV}(x_0) = V(E_0) = \begin{pmatrix} \alpha + \mu & 0 & 0 & 0 & 0 \\ 0 & \alpha + \mu & 0 & 0 & 0 \\ 0 & 0 & \alpha + \mu & 0 & 0 \\ -\alpha p & -\alpha p & -\alpha p & \delta + \mu & 0 \\ -\alpha(1-p) & -\alpha(1-p) & -\alpha(1-p) & -\delta & d + \mu \end{pmatrix} \quad (\text{A. 5})$$

The following is the verification of the fulfillment of the assumptions $(A_1) - (A_5)$: From previous basic properties we can conclude that $\mathcal{F}(x)$, $\mathcal{V}^-(x)$, $\mathcal{V}^+(x)$ and $\mathcal{V}(x)$ are positives, it means (A_1) is true. Trivially if $x = 0$ then $V^- = 0$ follows (A_2) . Notice that $\mathcal{F}_i = 0$ if $i > 3$, thus (A_3) is true. If $x \in X_s$ then $\mathcal{F}_i(x) = 0$ and $\mathcal{V}_i^+(x) = 0$ if $i = 1, 2, 3$. then (A_4) is true. Finally eigenvalues of $Df_i(x_0)$ are calculated as:

If $x = (I_h, I_w, I_m, T, A)$ then

$$Df(x_0) = Df(E_0) = \begin{pmatrix} B_h - (\alpha + \mu) & 0 & 0 & 0 & 0 \\ B_{hw} \frac{1-\theta}{\theta} & -(\alpha + \mu) & B_s \frac{1-\theta}{\theta\gamma} & 0 & 0 \\ B_{hm} \gamma & B_s \frac{\theta\gamma}{1-\theta} & -(\alpha + \mu) & 0 & 0 \\ \alpha p & \alpha p & \alpha p & -(\delta + \mu) & 0 \\ \alpha(1-p) & \alpha(1-p) & \alpha(1-p) & \delta & -(d + \mu) \end{pmatrix} \quad (\text{A. 6})$$

Its characteristic polynomial is:

$$p(\lambda) = (d + \lambda + \mu)(\delta + \lambda + \mu)(-\alpha + B_h - \lambda - \mu) \left[(\alpha + \lambda + \mu)^2 - B_s^2 \right].$$

Its eigenvalues are:

$$\lambda_1 = B_h - (\alpha + \mu), \quad \lambda_2 = B_s - (\alpha + \mu), \quad \lambda_3 = -(d + \mu), \quad \lambda_4 = -B_s - (\alpha + \mu), \quad \lambda_5 = -(\delta + \mu). \quad (\text{A. 7})$$

Since $R_0^{hom} = \frac{B_h}{\alpha + \mu} < 1$ and $R_0^{het} \frac{B_s}{\alpha + \mu} < 1$ eigenvalues above has negative real part. 413

These eigenvalues have negative real part if $R_0 < 1$, being fulfilled (A_5) . Thus, 414
 $(A_1) - (A_5)$ are satisfied, according [7], Therefore, we have 415
416

$$FV^{-1} = \begin{pmatrix} \frac{B_{hw}}{\alpha + \mu} & 0 & 0 & 0 & 0 \\ \frac{B_{hw}(1-\theta)}{\theta(\alpha + \mu)} & 0 & \frac{B_s(1-\theta)}{\theta\gamma(\alpha + \mu)} & 0 & 0 \\ \frac{c_{hm}\beta_{hm}\gamma}{\alpha + \mu} & \frac{B_s\gamma\theta}{(1-\theta)(\alpha + \mu)} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix} \quad (A. 8)$$

The characteristic polynomial of FV^{-1} is 417

$$p(\lambda) = \lambda^5 - \frac{B_h}{\alpha + \mu}\lambda^4 - \frac{B_s^2}{(\alpha + \mu)^2}\lambda^3 + \frac{B_h B_s^2}{(\alpha + \mu)^3}\lambda^2. \quad (A. 9)$$

The eigenvalues are 418

$$\lambda_1 = 0, \quad \lambda_2 = 0, \quad \lambda_3 = \frac{B_h}{\alpha + \mu}, \quad \lambda_4 = \frac{B_s}{\alpha + \mu}, \quad \lambda_5 = -\frac{B_s}{\alpha + \mu}. \quad (A. 10)$$

The basic reproduction number is defined as the spectral radius of the matrix FV^{-1} and denoted by

$$R_0 = \max \left\{ \frac{B_s}{\alpha + \mu}, \frac{B_h}{\alpha + \mu} \right\} = \max \{ R_0^{het}, R_0^{hom} \}. \quad (A. 11)$$

Appendix B 419

Endemic Equilibrium 420

I_m^* is given by the roots of fourth degree polynomial 421

$$p(I_m^*) = a_4(I_m^*)^4 + a_3(I_m^*)^3 + a_2(I_m^*)^2 + a_1(I_m^*) + a_0. \quad (B. 1)$$

$$\begin{aligned} a_4 &= \alpha^2(\alpha + \mu)(B_h - \alpha)^2(B_s - \alpha)(\alpha + B_s + \mu), \\ a_3 &= \alpha(B_h - \alpha) \left\{ (\alpha + B_s + \mu) \left[3\alpha\Lambda_m(B_h - \alpha)(\alpha - B_s) + \mu \left(\Lambda_m(\alpha - B_h)(2B_s - 3\alpha) + 2\alpha\Lambda_h R_0^{hom}(\alpha - B_s) \right) \right] \right. \\ &\quad \left. + 2\Lambda_h\mu^2 R_0^{hom}(\alpha - B_s) \right] + B_{hm}\Lambda_h(R_0^{hom} - 1)(\alpha + \mu) \left(\alpha(\alpha + \mu) - B_s\mu \right) + \alpha B_{hw}\Lambda_h(R_0^{hom} - 1)(\alpha + \mu)(B_s + \mu) \right\}, \end{aligned}$$

422

$$\begin{aligned}
a_2 = & 3\alpha\Lambda_m(B_h - \alpha) \left[\Lambda_m(\alpha - B_h)(\alpha^2 - B_s^2) - \alpha\Lambda_h(R_0^{hom} - 1)(\alpha B_{hm} + B_{hw}B_s) \right] \\
& + \mu^2 \left\{ B_{hm}\Lambda_h(R_0^{hom} - 1) \left[(B_s - 2\alpha) \left(\Lambda_m(B_h - \alpha) + \alpha\Lambda_h R_0^{hom} \right) - \alpha B_{hw}\Lambda_h(R_0^{hom} - 1) \right] \right. \\
& + 2\Lambda_h\Lambda_m(B_h - \alpha) \left[\alpha B_{hw} + R_0^{hom} \left(-\alpha(4\alpha + B_{hw}) + B_s^2 + \alpha B_s \right) \right] + \Lambda_m^2(B_h - \alpha)^2(B_s - 3\alpha) \\
& + \alpha\Lambda_h^2 R_0^{hom}(\alpha + B_s) \left(B_{hw}(1 - R_0^{hom}) + R_0^{hom}(B_s - \alpha) \right) \left. \right\} + \Lambda_h\mu^3 R_0^{hom} \left[2\Lambda_m(B_h - \alpha)(B_s - 2\alpha) \right. \\
& + \Lambda_h \left(B_{hm}(R_0^{hom} - 1)(B_s - \alpha) + \alpha B_{hw} + R_0^{hom}[-\alpha(2\alpha + B_{hw}) + B_s^2 + \alpha B_s] \right) \left. \right] \\
& + \mu \left\{ \alpha\Lambda_h\Lambda_m(B_h - \alpha) \left[B_{hm}(R_0^{hom} - 1)(2B_s - 5\alpha) + 2(\alpha + B_s) \left(B_{hw}(1 - R_0^{hom}) + 2R_0^{hom}(B_s - \alpha) \right) \right] \right. \\
& + \Lambda_m^2(B_h - \alpha)^2(3\alpha + B_s)(B_s - 2\alpha) - \alpha^2\Lambda_h^2(R_0^{hom} - 1) \left(B_{hm}B_{hw}(R_0^{hom} - 1) + \alpha B_{hm}R_0^{hom} + B_{hw}B_sR_0^{hom} \right) \left. \right\} \\
& + \Lambda_h^2\mu^4(R_0^{hom})^2(B_s - \alpha),
\end{aligned}$$

423

$$\begin{aligned}
a_1 = & \Lambda_m \left\{ \Lambda_m(B_h - \alpha) \left[\Lambda_m(B_h - \alpha)(\alpha^2 - B_s^2) + 3\alpha\Lambda_h(R_0^{hom} - 1)(\alpha B_{hm} + B_{hw}B_s) \right] + \mu^2 \left[\Lambda_m^2(B_h - \alpha)^2 \right. \right. \\
& + \Lambda_h\Lambda_m(B_h - \alpha) \left((R_0^{hom} - 1)(B_{hm} + B_{hw}) + 4\alpha R_0^{hom} \right) + \Lambda_h^2 \left([B_{hm}(R_0^{hom} - 1) + B_s R_0^{hom}](B_{hw}(R_0^{hom} - 1) \right. \\
& - B_s R_0^{hom}) + \alpha(R_0^{hom} - 1)R_0^{hom}(3B_{hm} + B_{hw}) + \alpha^2(R_0^{hom})^2 \left. \right) \left. \right] + \mu \left[2\alpha\Lambda_m^2(B_h - \alpha)^2 \right. \\
& + \Lambda_h\Lambda_m(B_h - \alpha) \left((\alpha + B_s)[B_{hw}(R_0^{hom} - 1) + 2R_0^{hom}(\alpha - B_s)] - B_{hm}(R_0^{hom} - 1)(B_s - 4\alpha) \right) \\
& + 2\alpha\Lambda_h^2(R_0^{hom} - 1) \left(B_{hm}B_{hw}(R_0^{hom} - 1) + \alpha B_{hm}R_0^{hom} + B_{hw}B_sR_0^{hom} \right) \left. \right] + \Lambda_h\mu^3 R_0^{hom} \left[2\Lambda_m(B_h - \alpha) \right. \\
& + \Lambda_h \left((R_0^{hom} - 1)(B_{hm} + B_{hw}) + 2\alpha R_0^{hom} \right) \left. \right] + \Lambda_h^2\mu^4(R_0^{hom})^2 \left. \right\}, \\
a_0 = & -\Lambda_h\Lambda_m^2(R_0^{hom} - 1) \left\{ \Lambda_m(B_h - \alpha) \left(B_{hm}(\alpha + \mu) + B_{hw}B_s \right) + B_{hw}\Lambda_h\mu \left(B_{hm}(R_0^{hom} - 1) + R_0^{hom}B_s \right) \right. \\
& + B_{hm}\Lambda_h\mu B_h \left. \right\}
\end{aligned}$$

424

Using Descartes' rule, we have the number of untreated infected heterosexual men in equilibrium EE is given by positive roots of polynomial $p(I_m^*)$ in equation (27). This equilibrium point exists when $R_0^{hom} > 1$, note that a_0 coefficient is negative when $R_0^{hom} > 1$ and a_4 coefficient depends on term $(B_s - \alpha)$. The number of possible positive roots in polynomial $p(I_m^*)$ depends on the signs of the coefficients a_{1-3} . Our purpose is to guarantee when the polynomial $p(I_m^*)$ has positive roots. Table A1 shows the number of possible positive roots according to coefficients signs. Note that if $R_0^{hom} > 1$ infection rate in homosexual men is greater than rate out of infected, it means $B_h > \alpha$. Analogously if $R_0^{het} > 1$ infection rate in heterosexual men is greater than rate out of infected, $B_s > \alpha$.

425

426

427

428

429

430

431

432

433

434

Note that when $R_0^{het} > 1$ the existence of EE is always guaranteed.

435

436

Table of number of possible positive roots of $p(I_m^*)$ allows to conclude the following theorem

437

438

Theorem A1. If $R_0^{hom} > 1$ the triangle transmission model in equations (1) – (8)

439

(i) Has a unique EE if $R_0^{het} > 1$ and whenever cases a1, a4, a6 and a7 of table A1 holds,

440

(ii) Could have more than one EE if $R_0^{het} > 1$ and whenever cases a2, a3, a5 and a8 of table A1 holds,

441

442

Case	a_0	a_1	a_2	a_3	a_4	Number of sign changes	Possible positive roots
a1	-	+	+	+	+	1	1
a2	-	+	+	-	+	3	1, 3
a3	-	+	-	+	+	3	1, 3
a4	-	-	+	+	$+ B_s > \alpha$	1	1
a5	-	+	-	-	$+ (R_0^{het} > 1)$	3	1, 3
a6	-	-	-	+	+	1	1
a7	-	-	-	-	+	1	1
a8	-	-	+	-	+	3	1, 3
b1	-	+	+	+	-	2	0, 2
b2	-	+	+	-	-	2	0, 2
b3	-	-	-	-	-	0	0
b4	-	+	-	-	$- B_s < \alpha$	2	0, 2
b5	-	+	-	+	-	4	0, 2, 4
b6	-	-	+	+	-	2	0, 2
b7	-	-	-	+	-	2	0, 2
b8	-	-	+	-	-	2	0, 2

Table A1. Number of possible positive roots of $p(I_m^*)$.

- (iii) Could have two or none EE if $B_s < \alpha$ and whenever cases b1, b2, b4, b6, b7 and b8 of table A1 holds,443
- (iv) Could have forth EE if $R_0^{het} < 1$ and whenever case b5 of table A1 holds,445
- (v) There is not EE if $R_0^{het} < 1$ and whenever case b3 of table A1 holds.446

References

1. Akudibillah, Gordon and Pandey, Abhishek and Medlock, Jan. Optimal control for HIV treatment. Mathematical biosciences and engineering: MBE. 1 (2018) 373–396. <http://doi.org/10.3934/mbe.2019018>.448

2. Beloqui, Jorge A. 2008. Risco relativo para AIDS entre homo/bissexuais em relaao aos heterossexuais [Relative risk for AIDS between homo/bisexual and heterosexual men]. Revista de saude publica. 42(3)1–6. <http://doi.org/10.1590/S0034-89102008005000014>.449

3. Chan. Likely female to female sexual transmission of HIV in Texas, 2012. Available online: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5779339/pdf/209-212.pdf> (accessed on 20th october, 2018).450

4. Cohen, Myron S and Chen, Ying Q and McCauley, Marybeth and Gamble, Theresa and Hosseinipour, Mina C and Kumarasamy, Nagalingeswaran and Hakim, James G and Kumwenda, Johnstone and Grinsztejn, Beatriz and Pilotto, Jose HS and others. 2011. Prevention of HIV-1 infection with early antiretroviral therapy. New England journal of medicine. 365(6) 493–505. <https://doi.org/10.1056/NEJMoa1105243>.451

5. Del Romero, Jorge and Baza, Mara Begona and Rıo, Isabel and Jeronimo, Adrian and Vera, Mar and Hernando, Victoria and Rodrıguez, Carmen and Castilla, Jesus. 2016. Natural conception in HIV-serodiscordant couples with the infected partner in suppressive antiretroviral therapy: A prospective cohort study. Medicine. 95(30) 30. <http://doi.org/10.1097/MD.0000000000004398>.452

6. Diekmann, Odo and Heesterbeek, Johan Andre Peter and Metz, Johan AJ. 1990. On the definition and the computation of the basic reproduction ratio R_0 in models for infectious diseases in heterogeneous populations. Journal of Mathematical Biology. 28 (4) 365–385. <http://doi.org/10.1111/j.1467-9574.1996.tb01482>.453

7. Van den Driessche, Pauline and Watmough, James. 2002. Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. Mathematical biosciences. 180 (1-2) 29–48. [http://doi.org/10.1016/S0025-5564\(02\)00108-6](http://doi.org/10.1016/S0025-5564(02)00108-6).454

8. Espitia C, Botina M, Solarte M, Hernandez I, Riascos R and Meyer. J. 2022. Mathematical Model of HIV/AIDS Considering Sexual Preferences Under Antiretroviral Therapy, a Case Study in San Juan de Pasto, Colombia. Journal of Computational Biology. 29 (5) 483-493. <http://doi.org/10.1089/cmb.2021.0323>.455

9. Glick, Sara Nelson and Morris, Martina and Foxman, Betsy and Aral, Sevgi O and Manhart, Lisa E and Holmes, King K and Golden, Matthew R. 2012. A comparison of sexual behavior patterns among men who have sex with men and heterosexual men and women. Journal of acquired immune deficiency syndromes (1999). 60(1)83. <http://doi.org/10.1097/QAI.0b013e318247925e>.456

10. Huo, Hai-Feng and Chen, Rui and Wang, Xun-Yang. Modelling and stability of HIV/AIDS epidemic model with treatment. Applied Mathematical Modelling. 40(2016)6550–6559. <http://doi.org/10.1016/j.apm.2016.01.054>.457

11. Keeling, Matt J and Rohani, Pejman.2008. Modeling infectious diseases in humans and animals. Princeton University Press.458

12. Kwakwa, Helena A and Ghobrial, Michel W. 2003. Female-to-female transmission of human immunodeficiency virus. *Journal Clinical Infectious Diseases*. 36 (3) e40–e41. <http://doi.org/10.1086/345462>. 477

13. Lloyd, Alun L and May, Robert M. 1996. Spatial heterogeneity in epidemic models. *Journal of theoretical biology*. 179(1)1–1. <http://doi.org/10.1006/jtbi.1996.0042>. 478

14. Mastro, Timothy D and De Vincenzi, Isabelle. Probabilities of sexual HIV-1 transmission. *Aids* 10(1996) S75–S82. <http://doi.org/10.1097/00002030-199601001-00011>. 479

15. Moghadas, Seyed M and Gumel, Abba B and McLeod, Robert G and Gordon, Richard. Could condoms stop the AIDS epidemic? *Computational and Mathematical Methods in Medicine* 3-4(2003) 171–181. <http://doi.org/10.1080/10273660412331315147>. 480

16. Mukandavire, Zindoga and Chiyaka, Christinah and Magombedze, Gesham and Musuka, Godfrey and Malunguza, Noble J. 2009. Assessing the effects of homosexuals and bisexuals on the intrinsic dynamics of HIV / AIDS in heterosexual settings. *Journal Mathematical and computer modelling*. 49 (9-10) 1869–1882. <http://doi.org/10.1016/j.mcm.2008.12.012>. 481

17. Powers, Kimberly A and Poole, Charles and Pettifor, Audrey E and Cohen, Myron. 2008. Rethinking the heterosexual infectivity of HIV-1: a systematic review and meta-analysis. *The Lancet infectious diseases*. 8(9) 553–563. [http://doi.org/10.1016/S1473-3099\(08\)70156-7](http://doi.org/10.1016/S1473-3099(08)70156-7). 482

18. Rosario, Margaret and Schrimshaw, Eric W and Hunter, Joyce and Braun, Lisa. 2006. Sexual identity development among lesbian, gay, and bisexual youths: Consistency and change over time. *Journal of sex research*. 43 (1) 46 – 58. <http://doi.org/10.1080/00224490609552298>. 483

19. Sani, A and Kroese, DP and Pollett, PK. 2007. Stochastic models for the spread of HIV in a mobile heterosexual population. *Journal Mathematical Biosciences*. 208 (1) 98–124. <http://doi.org/10.1016/j.mbs.2006.09.024>. 484

20. Sun, Xiaodan and Xiao, Yanni and Peng, Zhihang and Wang, Ning. Modelling HIV / AIDS epidemic among men who have sex with men in China. *BioMed research international*. 2013. <http://doi.org/10.1155/2013/413260>. 485

21. Thompson, Elisabeth Morgan and Morgan, Elizabeth M. 2008. Mostly straight young women: Variations in sexual behavior and identity development. *Journal Developmental Psychology*. 44 (1) 15. <https://doi.org/10.1037/0012-1649.44.1.15>. 486

22. Tuladhar, Anup. Mathematical Model for HIV Epidemic in far western Nepal due to Seasonal male labor migration to India. PhD dissertation. Kathmandu University 2018. <https://n9.cl/2qn51>. 487

23. UNAIDS. Global HIV & AIDS statistics — 2020 fact sheet (2020). Switzerland: Centers for Disease Control and Prevention. <https://n9.cl/tx8a>. Consulted 30th October of 2020 (accessed 4 February 2020). 488

489

490

491

492

493

494

495

496

497

498

499

500

501

502

503