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A Mathematical Model for HIV/AIDS Under Pre-Exposure and Post-Exposure Prophylaxis

Erick Delgado Moya[1], Diego Samuel Rodrigues[2], Alain Pietrus[3], Aymee Marrero Severo[4]

[1]Institute of Collective Health, Federal University of Bahia, Salvador, Brazil
erickdelgadomoya@gmail.com 0000-0001-5937-5374

[2]School of Technology, University of Campinas (UNICAMP), Limeira, Sao Paulo, Brazil
diego.rodrigues@ft.unicamp.br 0000-0002-0016-1715

[3]Department of Mathematics and Computer Sciences,
University of Antilles, Pointre-a-Pitre, Guadeloupe, France
alain.pietrus@univ-antilles.fr

[4]Department of Applied Mathematics, Faculty of Mathematics and Computer Science,
University of Havana, Havana, Cuba
aymee@matcom.uh.cu 0000-0003-4823-0078

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Abstract: **HIV/AIDS has a strong impact on society, the economy, and health. Early diagnosis of cases, adherence to treatment, and prevention are important factors in controlling the epidemic in the population. In this paper, we present a new mathematical model for the study of HIV/AIDS transmission. Our model is stratified in men and women, to account for the main forms of sexual transmission homosexual and heterosexual relationships, and infectiousness in the HIV and AIDS stages. In addition, in the construction of the model, we take into account the influence of Pre-Exposure Prophylaxis (PrEP) and Post-Exposure Prophylaxis (PEP) to study the impact of these implementations, diagnosis, and effectiveness of treatment based on viral load undetectability. We study the basic reproduction number by subpopulation (men and women) and general. Working by subpopulations allows us to study men who have sex with men who have a strong impact on virus transmission. Also, we study the infection-free equilibrium points due to their relationship with the basic reproduction number and demonstrate the global stability by subpopulation and general. To explore our model, we performed computational simulations on a scenario designed with data from the literature and assumed, studying the influence of the parameters associated with the use of PrEP, PEP, and undetectability on the basic reproduction number by varying them individually and jointly. We concluded that in women the basic reproduction number is always lower than unity and that in men the parameter associated with the undetectability of the viral load in HIV men has a strong influence on the dynamics. We also address the impact of PrEP, PEP, and undetectability in HIV and AIDS on the compartments, considering different scenarios varying the parameters jointly and independently and by sex which show difficulty in reducing women with AIDS. The scenario that showed the best results in the reduction of the number of HIV and AIDS cases was when the parameters associated with undetectability in HIV and AIDS men and women take the 90-90-90 that is proposed in the World Health Organization (WHO) strategy.**

Keywords: **HIV/AIDS, Undetectable, Pre-Exposure Prophylaxis, Post-Exposure Prophylaxis, Model**

MSC2020: **92B05, 92D30, 39A12, 00A71**

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I. Introduction

HIV (human immunodeficiency virus) is a virus that attacks the body's immune system. If HIV is not treated, it can lead to AIDS (acquired immunodeficiency syndrome). HIV has three stages: (1) acute HIV infection is the initial stage of HIV infection, which usually begins 2-4 weeks after the body acquires the virus, (2) chronic HIV infection (also called asymptomatic HIV infection or clinical latency); at this stage, HIV continues to replicate in the body but at very low levels, and the person may be asymptomatic and (3) AIDS, the final and most severe stage of HIV infection when the virus has severely damaged the immune system and the body is unable to fight off opportunistic infections. HIV-positive people are diagnosed with AIDS if they have a CD4 count below 200 cells/mm^3 or if they acquired certain opportunistic infections [1]. HIV is transmitted primarily through sexual contact with an infected person. Other less common ways of transmission are through sharing needles and/or syringes with someone infected, through transfusions of infected blood, or drug use. Infected pregnant women can infect their children before or after delivery or through breastfeeding after birth [2].

HIV treatment (antiretroviral therapy, ART) has evolved from strenuous regimens with high pill burden, cumbersome dosing, treatment-limiting toxicities, food-drug interactions, incomplete viral suppression, and the emergence of drug resistance. We now have once- or twice-daily pill regimens that can be initiated early in HIV disease and control viral replication for much of an individual's life. The life expectancy of individuals who have achieved immune reconstitution and remain virologically suppressed should be near normal [3, 4]. Viral load (VL) testing is essential for monitoring adherence to antiretroviral therapy (ART) [5]. A suppressed/undetectable VL in HIV-infected individuals will mean that the individual does not infect his or her sexual partner. This factor is relevant in controlling HIV/AIDS transmission in a population.

People with HIV should undergo antiretroviral therapy as soon as possible. If taken as prescribed, ART reduces the amount of HIV in the body (viral load) to a low level, which keeps the immune system working and prevents illness. This is called viral suppression – defined as having less than 200 copies of HIV per milliliter of blood. HIV treatment by ART can make the viral load so low that a test cannot detect it. This is called an undetectable viral load [6]. Achieving and maintaining an undetectable viral load (or staying virally suppressed) is the best way to stay healthy and protect others. Treatment helps prevent transmission to others. If one has an undetectable viral load, he/she is not at risk of transmitting HIV to an HIV-negative partner through sex.

Pre-exposure prophylaxis (or PrEP) is a medication taken to prevent getting HIV. PrEP is very effective in preventing HIV when taken continuously as recommended by a specialist. The use of PrEP reduces the risk of infection through sexual contact by 99% and through injection drug use by 74% [7,8]. Post-Exposure Prophylaxis (PEP) is the use of antiretroviral drugs after a single high-risk event to stop HIV seroconversion. PEP should be initiated as soon as possible to be effective, and always within 72 hours of a potential exposure [8]. The use of PEP involves social factors and it is necessary to make the population aware of the use of this therapy as soon as possible. In cases of sexual abuse, the use of PEP is crucial because it was an exposure that can have consequences [9,10]. If one uses PEP, one will need to take HIV medicines every day for 28 days, following up with a doctor. After finishing taking PEP, it is necessary to get tested for HIV and other tests. In our work, we affirm the effectiveness or not of PEP after one year and with a final negative test.

HIV remains a global health crisis and the world must reckon with the 1.5 million [1.0 million – 2.0 million] new HIV infections and 680 000 [480 000 – 1 000 000] deaths from AIDS-related causes that occurred in 2020. There were 37.7 million [30.2 million – 45.1 million] people living with HIV in 2020, including 10 million [9.8 million – 10.2 million] who were not on HIV treatment. Among those not on treatment, about 4.1 million did not know their HIV-positive status and 6.1 million knew their HIV status but could not access treatment [11]. The 90-90-90 targets are: 90% of people living with HIV know their HIV status, 90% of people who know their HIV-positive status are accessing treatment, and 90% of people on treatment have suppressed viral loads. The 90-90-90 targets were not achieved by 2020, 84% of people living with HIV knew their HIV status, 87% of people living with HIV who knew their HIV status were accessing antiretroviral therapy, and 90% of people on treatment were virally suppressed [11].

In recent years, there has been an increase in the number of studies on HIV/AIDS transmission [12–22]. For example, Moya and Marrero [12] presented a stochastic model using Markov chains to study the transmission of HIV/AIDS and control chain elements proposed control strategies. Moya and Marrero pro-

posed mathematical models for the transmission and treatment efficiency of HIV/AIDS using ordinary differential equations and to study the variation of the parameters transform system in differential inclusions.

Attaullah and M. Sohaib [14] implemented two numerical schemes for solving the model that has the behavior of CD4+ T-cells, infected CD4+ T-cells, and free HIV particles after HIV infection and highlight the accuracy and efficiency of the proposed schemes with other traditional schemes. Omondi et al. [15] presented a sex-stratified mathematical model that takes into account the sexual orientation of individuals and incorporates the use of PrEP. Bozkurt and Peker [16] formulated a mathematical model stratified into three subclasses, HIV negative, HIV positive who do not know their serostatus, and HIV positive who know they are infected and validated the model for India.

Akinwumi et al. [17] formulated and studied a mathematical model for the transmission of HIV/AIDS with early treatment. Ogunlaran and Noutchie [18] proposed a mathematical model with two control variables, where the uninfected CD4+T cells follow the logistic growth function and the incidence term is saturated with free virus and apply optimal control approach to maximize the concentration of uninfected CD4+T cells in the body by using minimum drug therapies. Lu et al. [19] developed a compartmentalized model for annual reporting of HIV in men who have sex with men (MSM) from 2007 to 2019 in the Zhejiang region and proved that the 90-90-90 target alone may not eliminate the HIV epidemic in Chinese men who have sex with men.

Sultanoğlu et al. [20] proposed a mathematical model to assess the dynamics of HIV infection in Cyprus. Arenas et al. [21] proposed a mathematical model to describe intracellular infection accounting for the time delay between HIV entry and the production of new virus using differential delay equations. Li et al. [22] formulated a mathematical model to evaluate the impact of PrEP, biomedical interventions, and their combinations and studied it over 20 years.

In the references presented above and in others in the literature, PrEP, PEP, diagnosis, and viral load undetectability due to treatment adherence are studied separately. The main contribution of our work is presenting a mathematical model that takes into account all these elements in the same population. In our approach, stratification by sex allows us to study the impact of these elements in the subpopulations of men and women separately and also in the general population. With this model, we study how these implementations impact on the transmission of HIV/AIDS in the subpopulations as well as in the basic number of reproduction and help to make decisions.

This paper is organized as follows: in Section 2, we present the model and we study its mathematical and epidemiological properties. Section 3 is devoted to illustrative situations using computational simulations, and Section 4 presents the final remarks and the conclusions of the paper.

II. Construction of the Mathematical Model

The model has 14 compartments and the population is stratified by biological gender. The compartments of the model differentiated by sex (H-men, M-Women) are susceptible ($H_S,\ M_S$), exposed ($H_E,\ M_E$), HIV positive ($H_V,\ M_V$), AIDS ($H_A,\ M_A$), undiagnosed ($H_N,\ M_N$) and undetectable ($H_I,\ M_I$). The compartments that include both genders are people who use PrEP (P_E) and those who use PEP (P_O). The equations that model the behavior of the susceptibles using nonlinear ordinary differential equations are:

$$\begin{aligned}\frac{dH_S}{dt} &= M_1 + e_H H_E + \alpha_{PF} P_E + \alpha_{P1} P_O \\ &\quad - (\mu_H + \alpha_P + \lambda_H) H_S, \qquad (1)\\ \frac{dM_S}{dt} &= M_2 + e_M M_E + \beta_{PF} P_E + \beta_{P1} P_O \\ &\quad - (\mu_M + \beta_P + \lambda_M) M_S. \qquad (2)\end{aligned}$$

The M_1 and M_2 are recruitment rates for men and women respectively. The HIV-infection rates for men and women is defined as

$$\begin{aligned}\lambda_H &= \alpha^* \frac{\varepsilon_{H1}(H_N + \varepsilon_H^* H_V + \varepsilon_H^{**} H_A)}{N} \\ &\quad + \alpha^* \frac{\varepsilon_{H2}(M_N + \varepsilon_H^* M_V + \varepsilon_H^{**} M_A)}{N}, \qquad (3)\\ \lambda_M &= \beta^* \frac{\varepsilon_{D1}(H_N + \varepsilon_M^* H_V + \varepsilon_M^{**} H_A)}{N} \\ &\quad + \beta^* \frac{\varepsilon_{D2}(M_N + \varepsilon_M^* M_V + \varepsilon_M^{**} M_A)}{N}, \qquad (4)\end{aligned}$$

where α^* and β^* are the effective contact rates and N is the total population $N = N_H + N_M + P_E + P_O$, where $N_H = H_S + H_E + H_V + H_A + H_N + H_I$, and $N_M = M_S + M_E + M_V + M_A + M_N + M_I$. HIV infection rates for men and women are different because we must take into account the different types of sexual relations, the exposure of the man or woman, and the HIV status of the infected person, for which we use modification parameters. The ε_{H1} is associated with homosexual contact between men and ε_{D2} with homosexual contact between women. The ε_{H2} and ε_{D1}

are associated with heterosexual relations starting from a man and a woman respectively. The serological status (HIV or AIDS) of the susceptible contact is also taken into account, by means of modification parameters. Thus, ε_H^* and ε_M^* are associated with contact with an HIV case, and ε_H^{**} and ε_M^{**} with contact with an AIDS case. The μ_H and μ_M are the natural cause death rates for men and women, respectively. Parameters e_H and e_M are associated with cases that were infected but did not acquire the virus during exposure. The differential equations for the behavior of those exposed to HIV are:

$$\begin{aligned}\frac{dH_E}{dt} &= \lambda_H H_S \\ &\quad - (e_H + \alpha_{PO} + \mu_H + \alpha_N + \alpha_V + \alpha_A)H_E, \end{aligned} \tag{5}$$

$$\begin{aligned}\frac{dM_E}{dt} &= \lambda_M M_S \\ &\quad - (e_M + \beta_{PO} + \mu_M + \beta_N + \beta_V + \beta_A)M_E. \end{aligned} \tag{6}$$

Parameters α_{PO} and β_{PO} represent the rates of people who were exposed to HIV and at the correct time make use of PEP, for men and women respectively. The α_V and β_V represent the rates for HIV cases that were diagnosed but did not use PEP and α_A and β_A for AIDS for women and men, respectively. In particular, parameters α_A and β_A represent cases that, when exposed and diagnosed their immune system is compromised by consequences different from HIV/AIDS, for example, cancer, active tuberculosis, or autoimmune disease. Parameters α_N and β_N represent exposed cases to the virus and are undiagnosed and infected that can infect susceptible individuals in the community. We define d_H and d_M as the rate of death in HIV as a consequence of the virus for males and females, respectively. The modification parameters τ_H and τ_M adapt the virus-related death in the AIDS state. We assume that death from the virus in the undiagnosed would be in the AIDS state since the person does not use treatment and the immune system would be debilitated. The parameters α_{N1} and β_{N1} represent cases that were undiagnosed (undiagnosed compartment) and are diagnosed with HIV and α_{N2} and β_{N2} for those diagnosed with AIDS for men and women respectively. Then, the undiagnosed compartments are represented as:

$$\begin{aligned}\frac{dH_N}{dt} &= \alpha_N H_E \\ &\quad - (\mu_H + \tau_H d_H + \alpha_{N1} + \alpha_{N2})H_N, \end{aligned} \tag{7}$$

$$\begin{aligned}\frac{dM_N}{dt} &= \beta_N M_E \\ &\quad - (\mu_M + \tau_M d_M + \beta_{N1} + \beta_{N2})M_N. \end{aligned} \tag{8}$$

We assume that patients in the HIV, AIDS, and undetectable compartments are using antiretroviral therapy, our model did not account for therapy drop-out cases as a compartment because those cases may return over time to the dynamics. We define α_{V1} and β_{V1} as the rates of HIV cases that are on treatment and progress to AIDS cases and α_{V2} and β_{V2} those that progress from AIDS to HIV status for women and men, respectively. The parameters α_{VI} and α_{AI} represent the cases that, with adherence to antiretroviral treatment, progress from HIV and AIDS status to undetectable virus status for men and β_{VI} and β_{AI} for women. The parameters α_{NH}, β_{NH} and α_{NA}, β_{NA} are the cases that lose the undetectable virus status and move to HIV and AIDS status. We assume that the cases that die in the undetectable state do so at the d_H and d_m rates. The HIV, AIDS, and undetectable compartments are modeled with the following equations:

$$\begin{aligned}
\frac{dH_V}{dt} &= \alpha_{N1}H_N + \alpha_V H_E + \alpha_{V2}H_A + \alpha_{NH}H_I \\
&\quad + \alpha_{P2}P_O - (\mu_H + d_H + \alpha_{V1} + \alpha_{VI})H_V, \\
\frac{dM_V}{dt} &= \beta_{N1}M_N + \beta_V M_E + \beta_{V2}M_A + \beta_{NH}M_I \\
&\quad + \beta_{P2}P_O - (\mu_M + d_M + \beta_{V1} + \beta_{VI})M_V, \\
\frac{dH_A}{dt} &= \alpha_{N2}H_N + \alpha_A H_E + \alpha_{V1}H_V + \alpha_{NA}H_I \\
&\quad - (\mu_H + \tau_H d_H + \alpha_{V2} + \alpha_{AI})H_A, \\
\frac{dM_A}{dt} &= \beta_{N2}M_N + \beta_A M_E + \beta_{V1}M_V + \beta_{NA}M_I \\
&\quad - (\mu_M + \tau_M d_M + \beta_{V2} + \beta_{AI})M_A, \\
\frac{dH_I}{dt} &= \alpha_{VI}H_V + \alpha_{AI}H_A \\
&\quad - (\mu_H + d_H + \alpha_{NA} + \alpha_{NH})H_I, \\
\frac{dM_I}{dt} &= \beta_{VI}M_V + \beta_{AI}M_A \\
&\quad - (\mu_M + d_M + \beta_{NA} + \beta_{NH})M_I.
\end{aligned} \tag{9}$$

In the P_E and P_O compartments, we do not differentiate by sex within the compartments, but the entry and exit are structured by sex. We assume that people will be in the PEP (P_O) compartment until it is not confirmed that the therapy was effective or not, thus we have a stage of medication use and periods of time where HIV tests are repeated.

In the PrEP compartment, we assume that patients have immunity to HIV/AIDS as long as they correctly use the precautionary therapy, also providing them with the information that they have protection from HIV/AIDS, but not from other sexually transmitted diseases, such that the use of male and female condoms

and other means is recommended [23–25]. Parameters α_P and β_P are associated with the PrEP use rate of susceptible men and women, respectively. Parameters α_{PF} and β_{PF} represent the failure of the therapy either by lack of health system, non-continuity of use or other situation and we assume that in that case, the person is again susceptible to HIV/AIDS. Parameters α_{P1} and β_{P1} represent cases that were exposed and used PEP and were effective and return to susceptible status and α_{P2} and β_{P2} represent cases that were not effective and enter the HIV diagnosed compartment. We assume that the person was not previously infected, as he/she would be HIV positive in the tests. The equations showing the behavior of patients using PrEP and PEP are:

$$\begin{aligned}\frac{dP_E}{dt} &= \alpha_P H_S + \beta_P M_S \\ &\quad - (\mu_H + \mu_M + \alpha_{PF} + \beta_{PF})P_E, \end{aligned} \tag{10}$$

$$\begin{aligned}\frac{dP_O}{dt} &= \alpha_{PO} H_E + \beta_{PO} M_E \\ &\quad - (\mu_H + \mu_M + \alpha_{P1} + \alpha_{P2} + \beta_{P1} + \beta_{P2})P_O. \end{aligned} \tag{11}$$

The Table I shows the definition of the parameters used in the model (12). Figure 1 shows the dynamics of the model (12). In summarizing, HIV transmission with the presence of PrEP, PEP, and with undiagnosed and undetectable statuses is modeled with the following system of differential equations:

$$\begin{aligned}\frac{dH_S}{dt} &= M_1 + e_H H_E + \alpha_{PF} P_E + \alpha_{P1} P_O \\ &\quad - (\mu_H + \alpha_P + \lambda_H)H_S, \\ \frac{dM_S}{dt} &= M_2 + e_M M_E + \beta_{PF} P_E + \beta_{P1} P_O \\ &\quad - (\mu_M + \beta_P + \lambda_M)M_S, \\ \frac{dH_E}{dt} &= \lambda_H H_S \\ &\quad - (e_H + \alpha_{PO} + \mu_H + \alpha_N + \alpha_V + \alpha_A)H_E, \\ \frac{dM_E}{dt} &= \lambda_M M_S \\ &\quad - (e_M + \beta_{PO} + \mu_M + \beta_N + \beta_V + \beta_A)M_E, \\ \frac{dH_N}{dt} &= \alpha_N H_E - (\mu_H + \tau_H d_H + \alpha_{N1} + \alpha_{N2})H_N, \\ \frac{dM_N}{dt} &= \beta_N M_E - (\mu_M + \tau_M d_M + \beta_{N1} + \beta_{N2})M_N, \\ \frac{dH_V}{dt} &= \alpha_{N1} H_N + \alpha_V H_E + \alpha_{V2} H_A + \alpha_{NH} H_I \\ &\quad + \alpha_{P2} P_O - (\mu_H + d_H + \alpha_{V1} + \alpha_{VI})H_V, \\ \frac{dM_V}{dt} &= \beta_{N1} M_N + \beta_V M_E + \beta_{V2} M_A + \beta_{NH} M_I \\ &\quad + \beta_{P2} P_O - (\mu_M + d_M + \beta_{V1} + \beta_{VI})M_V, \\ \frac{dH_A}{dt} &= \alpha_{N2} H_N + \alpha_A H_E + \alpha_{V1} H_V + \alpha_{NA} H_I \\ &\quad - (\mu_H + \tau_H d_H + \alpha_{V2} + \alpha_{AI})H_A, \\ \frac{dM_A}{dt} &= \beta_{N2} M_N + \beta_A M_E + \beta_{V1} M_V + \beta_{NA} M_I \\ &\quad - (\mu_M + \tau_M d_M + \beta_{V2} + \beta_{AI})M_A, \\ \frac{dH_I}{dt} &= \alpha_{VI} H_V + \alpha_{AI} H_A \\ &\quad - (\mu_H + d_H + \alpha_{NA} + \alpha_{NH})H_I, \\ \frac{dM_I}{dt} &= \beta_{VI} M_V + \beta_{AI} M_A \\ &\quad - (\mu_M + d_M + \beta_{NA} + \beta_{NH})M_I, \\ \frac{dP_E}{dt} &= \alpha_P H_S + \beta_P M_S \\ &\quad - (\mu_H + \mu_M + \alpha_{PF} + \beta_{PF})P_E, \\ \frac{dP_O}{dt} &= \alpha_{PO} H_E + \beta_{PO} M_E \\ &\quad - (\mu_M + \mu_H + \alpha_{P1} + \alpha_{P2} + \beta_{P1} + \beta_{P2})P_O, \end{aligned} \tag{12}$$

with the following initial condition:
$H_S(0) > 0$, $M_S(0) > 0$, $H_E(0) > 0$, $M_E(0) > 0$, $H_N(0) > 0$, $M_N(0) > 0$, $H_V(0) > 0$, $M_V(0) > 0$, $H_A(0) > 0$, $M_A(0) > 0$, $H_I(0) > 0$, $M_I(0) > 0$, $P_E(0) > 0$, and $P_O(0) > 0$.

A. Basic Properties

In this section, we establish the positivity of the solution and find the biologically feasible region. These results allow us to justify that the model is well posed in the biologically feasible region.

Theorem II.1. *Let the initial data for the model* (12) *be* $H_S(0) > 0, M_S(0) > 0, H_E(0) > 0, M_E(0) > 0, H_N(0) > 0, M_N(0) > 0, H_V(0) > 0, M_V(0) > 0, H_A(0) > 0, M_A(0) > 0, H_I(0) > 0, M_I(0) > 0, P_E(0) > 0$, *and* $P_O(0) > 0$. *Then, the solutions* $(H_S(t), M_S(t), H_E(t), M_E(t), H_N(t), M_N(t), H_V(t), M_V(t), H_A(t), M_A(t), H_I(t), M_I(t), P_E(t), P_O(t))$ *of the model* (12)*, with positive initial data, will remain positive for all time* $t > 0$.

Proof: The first equation of the model (12) corresponding to the behavior of susceptible men is:

$$\begin{aligned}\frac{dH_S}{dt} &= M_1 + e_H H_E + \alpha_{PF} P_E + \alpha_{P1} P_O \\ &\quad - (\mu_H + \alpha_P + \lambda_H)H_S. \end{aligned}$$

Then,

$$\frac{dH_S}{dt} \geq -(\mu_H + \alpha_P + \lambda_H)H_S. \tag{13}$$

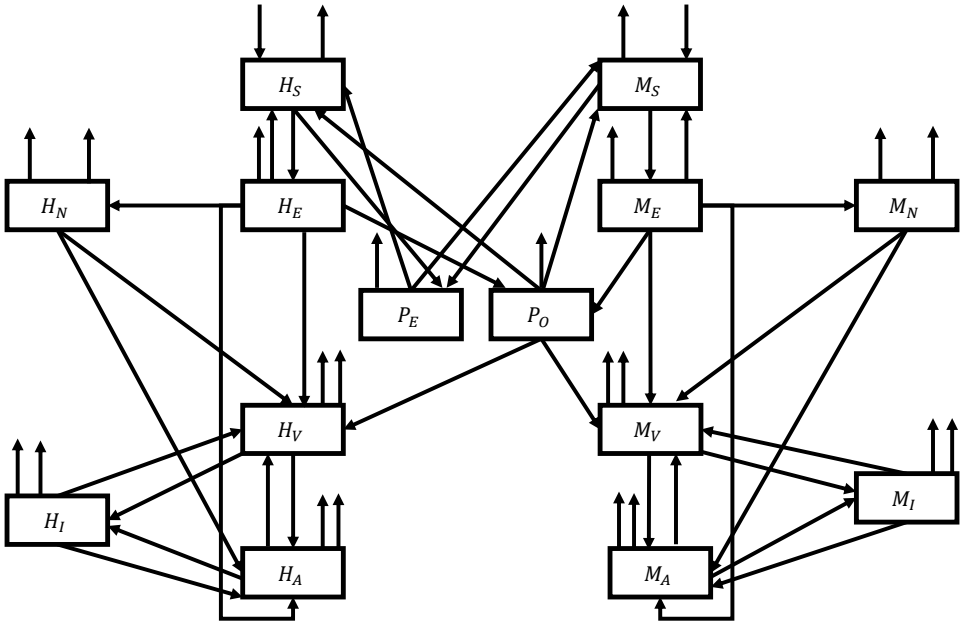


Fig. 1: Diagram of model (12).

Table I: Description and numerical values of parameters of the model (12).

Parameter	Description	Values	Reference
M_1, M_2	Recruitment rates	$0.000686N$, $0.000714N$	[15]
α^*, β^*	Effective contact rates	2, 2	[15]
d_H, d_M	HIV death Rate	0.00915, 0.0005	[15], Assumed
μ_H, μ_M	Natural death rate	0.00139, 0.00139	[15]
α_P, β_P	PrEP use rates	0.08, 0.02	Assumed
α_{PF}, β_{PF}	Failure rate use of PrEP	0.08, 0.08	[33]
α_{PO}, β_{PO}	Progression rates of exposed to PEP use	0.0001, 0.001	Assumed
α_{P1}, β_{P1}	Effectiveness rate use of PEP	0.96, 0.96	Assumed
α_{P2}, β_{P2}	Failure rate use of PEP	0.04, 0.04	[34]
α_V, β_V	Progression rates of exposed to HIV	0.8, 0.75	Assumed
e_H, e_M	Rate of cases exposed to HIV that became infected without the use of medication	0.1, 0.05	Assumed
α_A, β_A	Progression rates of exposed to AIDS	0.05, 0.01	Assumed
α_N, β_N	Progression rates of exposed to undiagnosed	0.5, 0.5	[32,35]
α_{N1}, β_{N1}	Progression rates of undiagnosed to HIV	1.3042, 1.2941	[32], Assumed
α_{N2}, β_{N2}	Progression rates of undiagnosed to AIDS	0.1751, 0.1542	[32], Assumed
α_{V1}, β_{V1}	Progression rates of HIV to AIDS	0.096, 0.096	[31,36]
α_{V2}, β_{V2}	Progression rates of AIDS to HIV	0.112, 0.112	[31,36]
α_{VI}, β_{VI}	Progression rates of HIV to undetectable	0.45, 0.65	Assumed
α_{AI}, β_{AI}	Progression rates of AIDS to undetectable	0.60, 0.60	Assumed
α_{NA}, β_{NA}	Progression rates of undetectable to AIDS	0.15, 0.05	Assumed
α_{NH}, β_{NH}	Progression rates of undetectable to HIV	0.45, 0.42	Assumed
τ_H, τ_M	Modification parameters	1.27, 1.27	[15]
$\varepsilon_H^*, \varepsilon_M^*$	Modification parameters	0.003, 0.0062	[15]
$\varepsilon_H^{**}, \varepsilon_M^{**}$	Modification parameters	0.0014, 0.149	[15]
$\varepsilon_H, \varepsilon_M$	Modification parameters	0.05, 0.01	Assumed
$\varepsilon_{H1}, \varepsilon_{D2}$	Modification parameters	0.8, 0.05	Assumed
$\varepsilon_{H2}, \varepsilon_{D1}$	Modification parameters	0.08, 0.2	Assumed

Integrating (13), by separation of variable. We have

$$\int \frac{dH_S}{H_S} \geq -\int (\mu_H + \alpha_P + \lambda_H)dt, \tag{14}$$

$$\ln H_S \geq -(\mu_H + \alpha_P + \lambda_H)t. \tag{15}$$

Applying the exponential function on both sides, we have:

$$\begin{aligned} H_S &\geq \exp\{-(\mu_H + \alpha_P + \lambda_H)t\}, \\ &\implies H_S(t) \geq H_S(0)\exp\{-(\mu_H + \alpha_P + \lambda_H)t\}, \\ &\implies H_S(t) \geq 0. \end{aligned} \tag{16}$$

Analogously, a similar result can be shown for $M_S(t)$, $H_E(t)$, $M_E(t)$, $H_N(t)$, $M_N(t)$, $H_V(t)$, $M_V(t)$, $H_A(t)$, $M_A(t)$, $H_I(t)$, $M_I(t)$, $P_E(t)$, and $P_O(t)$ for $t > 0$. Thus, all solutions of the model (12) remain positive for non-negative initial conditions. ■

Lemma II.2. *The closed set* $\Omega = \Omega_H \times \Omega_M$ *where*

$$\Omega_H = \left\{ (H_S, H_E, H_N, H_V, H_A, H_I) \in \mathbb{R}^6_+ : N_H \leq \frac{M_1}{\mu_H} \right\},$$

$$\Omega_M = \left\{ (M_S, M_E, M_N, M_V, M_A, M_I) \in \mathbb{R}^6_+ : N_M \leq \frac{M_2}{\mu_M} \right\}$$

and including P_E *and* P_0 *is positively-invariant and attracts all positive solutions of the model* (12)*.*

Proof: Given that $N_H = H_S + H_E + H_V + H_A + H_N + H_I$, and $N_M = M_S + M_E + M_V + M_A + M_N + M_I$, we have the following expressions:

$$\frac{dN_H}{dt} \leq M_1 - \mu_H N_H,$$

$$\frac{dN_M}{dt} \leq M_2 - \mu_M N_M.$$

Then,

$$\frac{dN_H}{dt} \leq 0, \text{ if } N_H(t) \geq \frac{M_1}{\mu_H}$$

and

$$\frac{dN_M}{dt} \leq 0, \text{ if } N_M(t) \geq \frac{M_2}{\mu_M}.$$

Hence, a standard comparison Theorem [26] can be used to show that

$$N_H(t) \leq N_H(0)\exp\{-\mu_H t\} + \frac{M_1}{\mu_H}\big(1 - \exp\{-\mu_H t\}\big)$$

and

$$N_M(t) \leq N_M(0)\exp\{-\mu_M t\} + \frac{M_2}{\mu_M}\big(1 - \exp\{-\mu_M t\}\big).$$

In particular, if $N_H(0) \leq \frac{M_1}{\mu_H}$ and $N_M(0) \leq \frac{M_2}{\mu_M}$, then $N_H(t) \leq \frac{M_1}{\mu_H}$ and $N_M(t) \leq \frac{M_2}{\mu_M}$ for all $t > 0$. Hence, the domain Ω is positively invariant. Furthermore, if $N_H(0) > \frac{M_1}{\mu_H}$ an $N_M(0) > \frac{M_2}{\mu_M}$ then either the solution enters the domain Ω in finite time or $N_H(t)$ approaches $\frac{M_1}{\mu_H}$ and $N_M(t)$ approaches $\frac{M_2}{\mu_M}$ asymptotically as $t \to \infty$. Hence, the domain Ω attracts all solutions in $\mathbb{R}^{14}_+$. ■

B. Basic Reproduction Number

Homosexual sex between men is known to have a strong impact on the transmission of HIV/AIDS. We will study the basic reproduction number in the male and female subpopulation and the influence of PrEP, PEP, viral load undetectability, and diagnosis on this important parameter. To find the basic reproduction numbers for the subpopulations ($\Re_0^H$, $\Re_0^M$) and the general model ($\Re_0$), we use the new generation matrix method presented in [27–29].

To study the men's subpopulation, all the women's compartments and the parameters associated with women ($\beta_P, \mu_M, \beta_{PF}, \beta_{P1}, \beta_{P2}$) in equations (10) and (11) are zero.

The infection-free equilibrium point for the men subpopulation is defined as:

$$\varepsilon_0^H = \big(S_H, 0, 0, 0, 0, 0, 0, 0\big),$$

where

$$S_H = \frac{M_1}{\mu_H + \alpha_P},$$

and the biologically feasible region is

$$\Omega_H^1 = \left\{ (H_S, H_E, H_N, H_V, H_A, H_I, P_E, P_O) \in \mathbb{R}^8_+ : N_H \leq \frac{M_1}{\mu_H + \alpha_P} \right\}.$$

Using the next-generation matrix methodology [27–29], we define the basic reproduction number for the men subpopulation as:

$$\Re_0^H = \frac{\Re_{0,1}^H}{\Re_{0,2}^H}, \tag{17}$$

where

$$\begin{aligned}\Re_{0,1}^H = M_1\alpha^*\varepsilon_{H1}\Bigg[&\varepsilon_H^*k_5\Big(\alpha_{P2}\alpha_{PO}k_2k_4\\&+k_7\Big(\alpha_{V2}(\alpha_N\alpha_{N2}+\alpha_Ak_2)\\&\quad+k_4(\alpha_N\alpha_{N1}+\alpha_Vk_2)\Big)\Big)\\&+\varepsilon_H^{**}\Big(\alpha_{P2}\alpha_{PO}\alpha_{V1}k_2k_5\\&+k_7\Big(k_5(\alpha_V\alpha_{V1}k_2+\alpha_N\alpha_{N2}k_3+\alpha_N\alpha_{N1}\alpha_{V1})\\&\quad-\alpha_{NH}\alpha_{VI}(\alpha_Ak_2+\alpha_N\alpha_{N2})\Big)\Big)\\&+\alpha_N\Big(k_7(k_3k_4k_5-\alpha_{NH}\alpha_{VI}k_4-\alpha_{V1}\alpha_{V2}k_5)\\&\quad-k_3k_4k_5\Big)\\&+\alpha_{NA}\alpha_{VI}\Big(\alpha_{P2}\alpha_{PO}\varepsilon_H^{**}k_2\\&\quad+(\alpha_V\varepsilon_H^{**}k_2+\alpha_N\alpha_{N1}\varepsilon_H^{**}-\alpha_N\alpha_{V2})k_7\Big)\\&-\alpha_{AI}\Big(\alpha_N(\alpha_{V1}-\alpha_{N2}\varepsilon_H^*)-\alpha_A\varepsilon_H^*k_2\Big)k_7\\&+\alpha_{NA}\Big(\alpha_{P2}\alpha_{PO}\varepsilon_H^*k_2+\alpha_V\varepsilon_H^*k_2k_7\\&\quad+\alpha_N(\alpha_{N1}\varepsilon_H^*+k_3)k_7\Big)\Bigg],\end{aligned}$$

$$\begin{aligned}\Re_{0,2}^H &= N_Hk_1k_2k_7\Big(\alpha_{AI}(\alpha_{NH}\alpha_{V1}+\alpha_{NA}k_3)\\&\quad+\alpha_{VI}(\alpha_{NH}k_4+\alpha_{NA}\alpha_{V2})-k_3k_4k_5\Big),\\k_1 &= \mu_H+\alpha_{PO}+e_H+\alpha_N+\alpha_V+\alpha_A,\\k_2 &= \mu_H+\tau_Hd_H+\alpha_{N1}+\alpha_{N2},\\k_3 &= \mu_H+d_H+\alpha_{VI}+\alpha_{V1},\\k_4 &= \mu_H+\tau_Hd_H+\alpha_{V2}+\alpha_{AI},\\k_5 &= \mu_H+d_H+\alpha_{NA}+\alpha_{NH},\\k_6 &= \mu_H+\alpha_{PF},\\k_7 &= \mu_H+\alpha_{P1}+\alpha_{P2}.\end{aligned}$$

Now, we list two conditions that, if met, also guarantee the global asymptotic stability of the disease-free equilibrium point. Following [30], we rewrite the submodel associated to men subpopulation as:

$$\begin{aligned}\frac{dS}{dt} &= F(S,I),\\\frac{dI}{dt} &= G(S,I),\quad G(S,0)=0,\end{aligned}$$

where $S\in\mathbb{R}_+^1$ is the vector whose components are the number of uninfected and recovered individuals H_S and $I\in\mathbb{R}_+^7$ denotes the number of infected, exposed, undiagnosed, using PrEP and PEP.

The disease-free equilibrium is now denoted by $E_0^H=(S_H,0)$.

The conditions that must be fulfilled to guarantee the global asymptotic stability of E_0^H are:

$$\begin{aligned}(H_1):\ &\text{For } \frac{dS}{dt}=F(S,0),\\&\quad S_H \text{ is globally asymptotically stable,}\\(H_2):\ &G(S,I)=AI-G^*(S,I),\\&\quad G^*(S,I)\geq 0,\ \text{for } (S,I)\in\Omega_H^1,\end{aligned}$$

where $A=D_IG(S_H,0)$ $\big($the Jacobian of G at $(S_H,0)$ is $D_IG(S_H,0)\big)$ is an M-matrix (the off-diagonal elements of A are non-negative) and Ω_H^1 is the region where the submodel makes biological sense (biologically feasible region).

If the submodel of men subpopulation satisfies the conditions (H_1) and (H_2), then the following result holds.

Lemma II.3. *The fixed point E_0^H is a globally asymptotically stable equilibrium of submodel provided that $\Re_0^H<1$ and that the conditions (H_1) and (H_2) are satisfied.*

Proof: Let $F(S,0)=\big(M_1-(\mu_H+\alpha_P)H_S\big)$. As $F(S,0)$ is a linear equation, we have that S_H is globally stable, hence H_1 is satisfied.

Then,

$$A=D_IG(S_H,0)=\begin{pmatrix}-k_1 & \alpha^*\varepsilon_H & \alpha^*\varepsilon_H\varepsilon_H^* & \alpha^*\varepsilon_H\varepsilon_H^{**} & 0 & 0 & 0\\ \alpha_N & -k_2 & 0 & 0 & 0 & 0 & 0\\ \alpha_V & \alpha_{N1} & -k_3 & \alpha_{V2} & \alpha_{NH} & 0 & \alpha_{P2}\\ \alpha_A & \alpha_{N2} & \alpha_{V1} & -k_4 & \alpha_{NA} & 0 & 0\\ 0 & 0 & \alpha_{VI} & \alpha_{AI} & -k_5 & 0 & 0\\ 0 & 0 & 0 & 0 & 0 & -k_6 & 0\\ \alpha_{PO} & 0 & 0 & 0 & 0 & 0 & -k_7\end{pmatrix}$$

$$I=(H_E,H_N,H_V,H_A,H_I,P_E,P_O),$$

$$G^*(S,I)=AI^T-G(S,I),$$

$$G^*(S,I)=\begin{pmatrix}G_1^*(S,I)\\G_2^*(S,I)\\G_3^*(S,I)\\G_4^*(S,I)\\G_5^*(S,I)\\G_6^*(S,I)\\G_7^*(S,I)\end{pmatrix}$$

$$= \begin{pmatrix} \alpha^* \varepsilon_{H1}(H_N + \varepsilon_H^* H_V + \varepsilon_H^{**} H_A)\left(1 - \dfrac{H_S}{N_H}\right) \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}.$$

Since H_S is always less than or equal to N_H, $\dfrac{H_S}{N_H} \leq 1$. Thus, $G^*(S,I) \geq 0$ for all $(S,I) \in \Omega_H^1$ and E_0^H is globally asymptotically stable. ■

To study the women subpopulation all the men compartments and the parameters associated with men $(\alpha_P, \mu_H, \alpha_{PF}, \alpha_{P1}, \alpha_{P2})$ in equations (10) and (11) are zero.

The infection-free equilibrium point for the women subpopulation is:

$$\varepsilon_0^M = \big(S_M, 0, 0, 0, 0, 0, 0, 0\big),$$

where

$$S_M = \frac{M_2}{\mu_M + \beta_P},$$

and the biologically feasible region is

$$\Omega_M^1 = \left\{ (M_S, M_E, M_N, M_V, M_A, M_I, P_E, P_O) \in \mathbb{R}_+^8 : N_M \leq \frac{M_2}{\mu_M + \beta_P} \right\}.$$

Using the next-generation matrix methodology [27–29], we define the basic reproduction number for the women subpopulation as:

$$\Re_0^M = \frac{\Re_{0,1}^M}{\Re_{0,2}^M}, \tag{18}$$

where

$$\begin{aligned} \Re_{0,1}^M = M_2 \beta^* \varepsilon_{D2} \Bigg[& \varepsilon_M^* k_{15} \Big(\beta_{P2}\beta_{PO} k_{12} k_{14} \\ & + k_{17}\Big(\beta_{V2}(\beta_N \beta_{N2} + \beta_A k_{12}) \\ & \quad + k_{14}(\beta_N \beta_{N1} + \beta_V k_{12})\Big)\Big) \\ & + \varepsilon_M^{**}\Big(\beta_{P2}\beta_{PO}\beta_{V1} k_{12} k_{15} \\ & + k_{17}\Big(k_{15}(\beta_V \beta_{V1} k_{12} + \beta_N \beta_{N2} k_{13} + \beta_N \beta_{N1} \beta_{V1}) \\ & \quad - \beta_{NH}\beta_{VI}(\beta_A k_{12} + \beta_N \beta_{N2})\Big)\Big) \\ & + \beta_N \Big(k_{17}(k_{13} k_{14} k_{15} - \beta_{NH}\beta_{VI} k_{14} - \beta_{V1}\beta_{V2} k_{15}) \\ & \quad - k_{13} k_{14} k_{15}\Big) \\ & + \beta_{NA}\beta_{VI}\Big(\beta_{P2}\beta_{PO}\varepsilon_M^{**} k_{12} \\ & \quad + (\beta_V \varepsilon_M^{**} k_{12} + \beta_N \beta_{N1} \varepsilon_M^{**} - \beta_N \beta_{V2}) k_{17}\Big) \\ & - \beta_{AI}\Big(\beta_N(\beta_{V1} - \beta_{N2}\varepsilon_M^*) - \beta_A \varepsilon_M^* k_{12}\Big) k_{17} \\ & + \beta_{NA}\Big(\beta_{P2}\beta_{PO}\varepsilon_M^* k_{12} + \beta_V \varepsilon_M^* k_{12} k_{17} \\ & \quad + \beta_N(\beta_{N1}\varepsilon_M^* + k_{13}) k_{17}\Big)\Bigg], \end{aligned}$$

$$\begin{aligned} \Re_{0,2}^M = N_M k_{11} k_{12} k_{17} \Big(& \beta_{AI}(\beta_{NH}\beta_{V1} + \beta_{NA} k_{13}) \\ & + \beta_{VI}(\beta_{NH} k_{14} + \beta_{NA}\beta_{V2}) - k_{13} k_{14} k_{15}\Big), \end{aligned}$$

$$\begin{aligned} k_{11} &= \mu_M + \beta_{PO} + e_M + \beta_N + \beta_V + \beta_A, \\ k_{12} &= \mu_M + \tau_M d_M + \beta_{N1} + \beta_{N2}, \\ k_{13} &= \mu_M + d_M + \beta_{VI} + \beta_{V1}, \\ k_{14} &= \mu_M + \tau_M d_M + \beta_{V2} + \beta_{AI}, \\ k_{15} &= \mu_M + d_M + \beta_{NA} + \beta_{NH}, \\ k_{16} &= \mu_M + \beta_{PF}, \\ k_{17} &= \mu_M + \beta_{P1} + \beta_{P2}. \end{aligned}$$

Using an analogous methodology applied to the men's submodel for the case of the women's submodel, we obtain the global stability of the infection-free equilibrium point. The model (12) has a disease-free equilibrium, given by

$$\varepsilon_0^G = (S_H, S_M, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0).$$

We computed the basic reproduction number as in the previous submodels by next-generation matrix method. The dominant eigenvalues of the next-generation matrix are $\Re_0^H$ and $\Re_0^M$. Therefore, the basic reproduction number of the model (12) is

$$\Re_0 = \max\{\Re_0^H, \Re_0^M\}.$$

C. Global Stability

Now, we derive the conditions that ensure global asymptotic stability of the disease-free equilibrium point. Following [30], we can rewrite the model (12) as

$$\begin{aligned} \frac{dS}{dt} &= F(S,I), \\ \frac{dI}{dt} &= G(S,I), \quad G(S,0) = 0, \end{aligned}$$

where $S \in \mathbb{R}^2_+$ is the vector whose components are the number of uninfected and recovered and $I \in \mathbb{R}^{12}_+$ denotes the number of infected, exposed, undiagnosed, using PrEP and PEP.

The disease-free equilibrium is now denoted by $E_0^G = (S_0^*, 0)$, where

$$S_0^* = (S_H, S_M),$$

$$S_H = \frac{M_1}{\mu_H + \alpha_P},$$

$$S_M = \frac{M_2}{\mu_M + \beta_P}.$$

The conditions (H_1) and (H_2) below must be satisfied to guarantee the global asymptotic stability of E_0^G:

$$(H_1): \text{For } \frac{dS}{dt} = F(S,0),$$
$$S_0^* \text{ is globally asymptotically stable,}$$

$$(H_2): G(S,I) = AI - G^*(S,I),$$
$$G^*(S,I) \geq 0, \text{ for } (S,I) \in \Omega,$$

where $A = D_I G(S_0^*, 0)$ $\big($the Jacobian of G at $(S_0^*, 0)$ is $D_I G(S_0^*, 0)\big)$ is an M-matrix (the off-diagonal elements of A are non-negative) and Ω is the biologically feasible region.

We have the following result:

Theorem II.4. *The fixed point E_0^G is a globally asymptotically stable equilibrium of model* (12) *provided that $\Re_0 < 1$ and that the conditions (H_1) and (H_2) are satisfied.*

Proof: Let

$$F(S,0) = \begin{pmatrix} M_1 - (\mu_H + \alpha_P)H_S \\ M_2 - (\mu_M + \beta_P)M_S \end{pmatrix}.$$

As $F(S,0)$ is a linear equation, we obtain that S_0^* is globally asymptotic stable, thus H_1 is satisfied.

Then, $\mathbf{A} = [\mathbf{A_1} \mid \mathbf{A_2}]$, where

$\mathbf{A_1} =$

$$\begin{pmatrix}
-k_1 & 0 & \alpha^*\varepsilon_{H1} & \alpha^*\varepsilon_{H2} & \alpha^*\varepsilon_{H1}\varepsilon_H^* & \alpha^*\varepsilon_{H2}\varepsilon_H^* \\
0 & -k_{11} & \alpha^*\varepsilon_{D1} & \alpha^*\varepsilon_{D2} & \alpha^*\varepsilon_{D1}\varepsilon_M^* & \alpha^*\varepsilon_{D2}\varepsilon_M^* \\
\alpha_N & 0 & -k_2 & 0 & 0 & 0 \\
0 & \beta_N & 0 & -k_{12} & 0 & 0 \\
\alpha_V & 0 & \alpha_{N1} & 0 & -k_3 & 0 \\
0 & \beta_V & 0 & \beta_{N1} & 0 & -k_{13} \\
\alpha_A & 0 & \alpha_{N2} & 0 & \alpha_{V1} & 0 \\
0 & \beta_A & 0 & \beta_{N2} & 0 & \beta_{V1} \\
0 & 0 & 0 & 0 & \alpha_{VI} & 0 \\
0 & 0 & 0 & 0 & 0 & \beta_{VI} \\
0 & 0 & 0 & 0 & 0 & 0 \\
\alpha_{PO} & \beta_{PO} & 0 & 0 & 0 & 0
\end{pmatrix}$$

$\mathbf{A_2} =$

$$\begin{pmatrix}
\alpha^*\varepsilon_{H1}\varepsilon_H^{**} & \alpha^*\varepsilon_{H2}\varepsilon_H^{**} & 0 & 0 & 0 & 0 \\
\alpha^*\varepsilon_{D1}\varepsilon_M^{**} & \alpha^*\varepsilon_{D2}\varepsilon_M^{**} & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
\alpha_{V2} & 0 & \alpha_{NH} & 0 & 0 & \alpha_{P2} \\
0 & \beta_{V2} & 0 & \beta_{NH} & 0 & \beta_{P2} \\
-k_4 & 0 & \alpha_{NA} & 0 & 0 & 0 \\
0 & -k_{14} & 0 & \beta_{NA} & 0 & 0 \\
\alpha_{AI} & 0 & -k_5 & 0 & 0 & 0 \\
0 & \beta_{AI} & 0 & -k_{15} & 0 & 0 \\
0 & 0 & 0 & 0 & -k_6 - k_{16} & 0 \\
0 & 0 & 0 & 0 & 0 & -k_7 - k_{17}
\end{pmatrix}$$

$$\mathbf{I} = (H_E, M_E, H_N, M_N, H_V, M_V, H_A, M_A, H_I, M_I, P_E, P_O),$$

$$G^*(S,I) = AI^T - G(S,I),$$

$$G^*(S,I) = \begin{pmatrix}
G_1^*(S,I) \\ G_2^*(S,I) \\ G_3^*(S,I) \\ G_4^*(S,I) \\ G_5^*(S,I) \\ G_6^*(S,I) \\ G_7^*(S,I) \\ G_8^*(S,I) \\ G_9^*(S,I) \\ G_{10}^*(S,I) \\ G_{11}^*(S,I) \\ G_{12}^*(S,I)
\end{pmatrix}$$

$$= \begin{pmatrix}
\alpha^*\big(\varepsilon_{H1}(H_N + \varepsilon_H^* H_V + \varepsilon_H^{**} H_A) \\
+\varepsilon_{H2}(M_N + \varepsilon_H^* M_V + \varepsilon_H^{**} M_A)\big)\left(1 - \dfrac{H_S}{N}\right) \\
\beta^*\big(\varepsilon_{D1}(H_N + \varepsilon_M^* H_V + \varepsilon_M^{**} H_A) \\
+\varepsilon_{D2}(M_N + \varepsilon_M^* M_V + \varepsilon_M^{**} M_A)\big)\left(1 - \dfrac{M_S}{N}\right) \\
0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0
\end{pmatrix}.$$

Since H_S, and M_S are always less than or equal to N, $\dfrac{H_S}{N} \leq 1$, and $\dfrac{M_S}{N} \leq 1$. Thus, $G^*(S,I) \geq 0$ for all $(S,I) \in \Omega$, the E_0^G is globally asymptotically stable. ■

III. Numerical Simulations

For the numerical simulations, we use a set of parameters extracted from [6, 15, 31–36] for illustrative purposes and to support the analytical results, see Tables I-II. We use the fourth-order Runge–Kutta numerical scheme coded in MATLAB2021b programming language. The initial conditions do not represent a specific demographic area, but fall within the range of actual achievable data, see Table II. The parameter values and initial conditions assumed were discussed and validated by specialists. In particular, for the assumed parameters, the behavior of the disease, treatment, and transmission for men and women were taken into account.

Table II: Numerical values for the initial conditions of model (12).

Variable	Value	Variable	Value
$H_S(0)$	12000000	$M_S(0)$	11700000
$H_E(0)$	25000	$M_E(0)$	12000
$H_N(0)$	55000	$M_N(0)$	35000
$H_V(0)$	60000	$M_V(0)$	45000
$H_A(0)$	9000	$M_A(0)$	3000
$H_I(0)$	5000	$M_I(0)$	3500
$P_E(0)$	10000	$P_O(0)$	3000

In the susceptible compartments, women after the year of study outnumber men until the end. Therefore, women manage to maintain a higher number of susceptible cases. Susceptible males decrease during the whole study, which implies that males enter in greater numbers in the dynamics concerning females, see Figure 2a. In the exposed compartments, there is a higher number of exposed cases compared to women, but at the end of the period, the opposite situation occurs. In both subpopulations, the number of exposed cases increases at the beginning of the study, but decreases approximately one year later until the end of the study, see Figure 2b. In the undiagnosed, a decrease was reported throughout the study period. This is a favorable factor for the dynamics because it is evidence that we were able to diagnose the cases and apply treatment, see Figure 2c.

In the HIV compartments, we see that at the beginning of the study we have an increase in the number of cases. Approximately one year into the study, both subpopulations have a decrease until the end of the study, this is significant because the cases either become undetectable or reach the AIDS stage because HIV/AIDS still has no cure. Throughout the study, more men were reported to be HIV positive than women, but at the end of the study period the opposite was the case, see Figure 2d. In the AIDS compartments, it occurs analogously to HIV. In the beginning, a growth that reaches the maximum value at approximately two years of age. AIDS males outnumber females throughout the study. Regarding the number of cases, the decrease is more significant in men than in women, see Figure 2e. In the case of AIDS women, the minimum value of cases reported is the initial value, which implies that throughout the study women exceeded the initial value. In the case of men with AIDS, the value reached was at the end of the period with ≈ 8300 cases reported when the initial value was 9000 cases. A greater number of HIV cases than AIDS cases were reported for both subpopulations. Diagnosed infected males outnumbered females and the reduction in HIV and AIDS during the study concerning the number of cases was more significant in males than in females.

As we report a reduction in the number of HIV and AIDS cases, there is an increase in the number of undetectable cases. This means that treatment is having a positive impact and that we have adherence to treatment concerning undetectability. At the beginning of the study we reported a higher number of undetectable cases in men than in women, but approximately 8 years into the study the opposite is true. In men, approximately 4 years into the study, there was a significant decrease in the number of undetectable cases, see Figure 2f. In scenarios with characteristics similar to those of our study, we recommend a more rigorous control of the correct use of the treatment through more continuous consultations and resistance tests to avoid a decrease in the number of undetectable cases in men and women, mainly men due to the impact they have on transmission. Achieving viral load undetectability helps to control the epidemic because a person with an undetectable viral load does not transmit the virus in the main form of transmission, sexual transmission.

Throughout the study, there is a growth in the number of people using PrEP therapy and it is an important tool to prevent the transmission of the virus in society, see Figure 3a. In the case of PEP use, the number of cases decreases significantly from the beginning, see Figure 3b. The use of PEP needs awareness of the people who were exposed because the difficulty is to apply the therapy quickly after the risk contact, otherwise there is no effectiveness. Better results are achieved if carrying out campaigns to promote the use of PrEP and PEP and raise awareness among doctors and police authorities about the use of PEP and have it as the first action in cases of rape. In the specific case of PrEP, we can reduce the impact of HIV/AIDS if we increase the number of sexually active people using

this therapy. We know that it has a high cost but it is effective and even more so if we stimulate the use of male and female condoms and adherence to treatment of seropositive cases.

A. Basic Reproduction Number Study

The objective of this section is to study the behavior of basic reproduction numbers when we vary the rates associated with PrEP, PEP, undetectability in HIV and AIDS. If $0 < \Re_0 < 1$ the infection will die out in the long run and if $\Re_0 > 1$ the infection will be able to spread in a population [27]. The higher the $\Re_0$ the more difficult it is to control the epidemic. The $\Re_0$ can be affected by several factors, such as the duration of infectivity of the affected patients, the infectivity of the organism, and the degree of contact between the susceptible and infected populations.

For $\alpha_P \in [0.08, 0.7]$ then $\Re_0^H \in [0, 1570, 1.3570]$, it takes values greater and less than unity. For $\alpha_P \approx 0.109$, $\Re_0^H = 1.000$ and as the behavior is decreasing, we have that for $\alpha_P > 0.107$, $\Re_0^H$ is less than unity, see Figure 4a. This means that if we get the rate of men using PrEP to be greater than the unity we manage to reduce infectiousness in the subpopulation. When $\beta_P \in [0.02, 0.4]$ the $\Re_0^M \in [0.0189, 0.3544]$, and its behavior is decreasing, and for any variation of β_P increasing $\Re_0^M$ is reduced, see Figure 4b. In this case, as the basic reproduction number of the general model is the maximum per subpopulation, we have that if we manage to reduce $\Re_0^H$, we reduce the infectivity in the population. This is evidence of the high degree of infectivity in men who have sex with men that affects the general population.

For the parameters associated with the use of PEP, we have the following results. For $\alpha_{PO} \in [0.0001, 0.5]$, then $\Re_0^H \in [1.0982, 1.3528]$, i.e. for any variation of α_{PO}, $\Re_0^H$ takes values greater than unity but has a decreasing behavior, see Figure 4c. In the case of $\beta_{PO} \in [0.001, 0.6]$, then $\Re_0^M \in [0.2449, 0.3544]$ and has a decreasing behavior, see Figure 4d. In this case, we conclude that the use of PEP independently does not reduce the infectivity of HIV/AIDS in the population because it will be greater than unity.

To study the influence on the basic reproduction number of adherence to treatment concerning undetectability, we start with HIV cases. For $\alpha_{VI}, \beta_{VI} \in [0.40, 0.90]$, the $\Re_0^H \in [1.1211, 1.5818]$, $\Re_0^M \in [0.3106, 0.3982]$ and are increasing but in males it is greater than unity for the variation of α_{VI}, see Figures 5a-5b. In AIDS we have that for $\alpha_{AI}, \beta_{AI} \in [0.40, 0.90]$, then $\Re_0^H \in [1.0067, 1.3528]$, $\Re_0^M \in [0.3544, 0.4391]$ but here the male population decreases but is greater than unity and the female population decreases and is less than unity, see Figures 5c-5d. For the case of men as it is always greater than unity, we must study the relationship of the parameters α_{VI}, α_{AI} with others to create a strategy to reduce this value and to maintain in women a constant study to avoid that this growth does not lead to values greater than unity.

In summary, in the case of any independent variation of the parameters associated with the use of PrEP, PEP, and undetectability of viral load in HIV and AIDS, values greater than unity are found, which means that men who have sex with men continue to contribute to HIV/AIDS infectiousness in the population despite the strategies applied.

We are now going to report the joint relationships of these parameters focusing on the parameter α_{VI} because of its increasing behavior and greater than unity.

For $\alpha_{AI}, \alpha_{VI} \in [0.4, 0.9]$, we have that when α_{AI} takes smaller values and α_{VI} its larger values $\Re_0^H$ is less than unity, see Figures 6a-6b. This evidences the influence that HIV has a greater subpopulation of men concerning AIDS and that antiretroviral therapy achieves effectiveness in less time concerning AIDS.

For $\alpha_{PO} \in [0.0001, 0.5]$ and $\alpha_{VI} \in [0.4, 0.9]$ here $\Re_0^H$ is always greater than unity. In this case, with antiretroviral therapy in HIV and the use of PEP alone, it is not possible to achieve a $\Re_0^H$ less than unity and thus reduce the transmission of HIV/AIDS, see Figures 6c-6d.

When we study $\alpha_P \in [0.08, 0.7]$ and $\alpha_{VI} \in [0.4, 0.9]$, $\Re_0^H$ reaches values greater and less than unity, see Figures 6e-6f. This shows that by achieving greater adherence to treatment with respect to undetectability and by increasing the rate of PrEP use in men, we can achieve a $\Re_0^H$ lower than unity and reduce HIV/AIDS transmission in the population.

We remind the reader that there is currently no cure for HIV/AIDS. This way, if the value of the basic reproduction number is lower than unity this does not mean the disappearance of the disease. In addition, since $\Re_0 = \max\{\Re_0^H, \Re_0^M\}$, if $\Re_0^H > 1$ then this affects the whole dynamics. With this subpopulation study of the basic reproduction number, we were able to verify the influence that men who have sex with men have on the transmission and control of the epidemic.

Carrying out an analogous study for the women subpopulation, the behavior of the joint variations is analogous to that of the men subpopulation (the maximum values of the basic reproduction number are

reached when β_{AI}, β_{PO} and β_P achieve the maximum value under study) but the basic reproduction number is always less than unity, see Figure 7.

B. PrEP Effect

The aim of this section is to study the impact of the rate of PrEP use on the dynamics of HIV/AIDS. We will use the fixed values of Table I and vary the parameters α_P and β_P using logical values and discussed with epidemiologists. For α_P, we study the values $0.08, 0.2, 0.4, 0.5, 0.7$ and for β_P, the values $0.02, 0.1, 0.25, 0.3, 0.4$.

Table III shows the value of the basic reproduction number for the studied parameter values. In this case of the variation of α_P we have $\Re_0 = \Re_0^H$ but it affects all the dynamics. We can observe that in men the $\Re_0$ is greater than unity for $\alpha_P = 0.08$ and for the other values less than unity and this implies that the behavior of reduction of $\Re_0$ is combined with the reduction of HIV and AIDS cases (see Figure 8a-8d) but in the case of $\alpha_P = 0.08$ we should study this behavior because using the $\Re_0$ as a parameter that measures the infectivity in the population for that case despite the decrease of male HIV and AIDS cases the epidemic is not extinguished. In the case of the variation of β_P, in the female subpopulation the $\Re_0^M$ is less than unity but as the $\Re_0 = \max\{\Re_0^H, \Re_0^M\}$ the epidemic in the population is not extinguished for this variation of $\Re_0^M$.

Interpreting the results obtained in Table V, we can observe that α_P significantly influences the reduction in HIV/AIDS in men and β_P in HIV/AIDS in women independently. In the case of α_P there was a reduction in the intervals of the reported HIV and AIDS cases in men (minima and maxima) and for women too, but not significantly. For the variation of β_P in men the opposite situation occurs, for higher β_P the number of male cases increases (minima and maxima), and the number of female cases decreases (minima and maxima). This factor is related to the fact that we set $\alpha_P = 0.08$ and provides evidence that it is necessary to increase the rate of PrEP use in men to achieve better results. The graphical behavior for the variation of α_P and β_P is shown in Figures 8-9. In the case of the exposed men, the most significant result is that when we increase α_P the number of exposed men decreases but the variation of α_P in women is not significant and for the β_P variations the opposite is the case, see Figures 8e, 8f, 9e and 9f. Another important result is that the initial condition of women with AIDS (3000 cases) for any variation of α_P and β_P is not reduced, so the variation of these parameters (independent form) in this situation was not effective.

Table III: Values of the basic reproduction numbers for the parameter values α_P and β_P.

α_P	$\Re_0^H$	$\Re_0$	β_P	$\Re_0^M$	$\Re_0$
0.08	1.3570	1.3570	0.02	0.3544	1.3570
0.1	1.0880	1.0880	0.1	0.0784	1.3570
0.4	0.2747	0.2747	0.25	0.0288	1.3570
0.5	0.2195	0.2195	0.3	0.0245	1.3570
0.7	0.1570	0.1570	0.4	0.0189	1.3570

Table IV: Values of the basic reproduction numbers for the parameter α_{PO} and β_{PO}.

α_{PO}	$\Re_0^H$	$\Re_0$	β_{PO}	$\Re_0^M$	$\Re_0$
0.001	1.3528	1.3528	0.001	0.3544	1.3528
0.01	1.3552	1.3552	0.01	0.3408	1.3528
0.09	1.3001	1.3001	0.2	0.3017	1.3528
0.2	1.2412	1.2412	0.3	0.2849	1.3528
0.5	1.0982	1.0982	0.4	0.2449	1.3528

For the study of the joint variation of α_P and β_P, we created three possible scenarios discussed with the specialists:

- Scenario I: $\alpha_P = 0.08$, $\beta_P = 0.02$ (fixed values from Table I) with $\Re_0 = 1.3570$.
- Scenario II: $\alpha_P = 0.2$, $\beta_P = 0.25$ ($\alpha_P < \beta_P$) with $\Re_0 = 0.5485$.
- Scenario III: $\alpha_P = 0.70$, $\beta_P = 0.40$ ($\alpha_P > \beta_P$) with $\Re_0 = 0.1570$.

The value of $\Re_0$ in these scenarios shows that scenarios II and III have $\Re_0$ lower than unity, which implies that this variation can lead to the extinction of the epidemic, which is the opposite of what happens in the scenario I.

The study of the three scenarios showed that scenarios II and III reduce the number of cases of HIV and AIDS in men and women concerning scenario I, which is the one with the values under study. The best results in the reduction of cases were obtained in scenario III, which shows that by achieving better results in the use of PrEP in men, we can reduce the impact of the epidemic, see Figure 10. In the studies of the HIV/AIDS epidemic, it was shown that men have a greater impact on transmission compared to women, so when we increase the number of men who use PrEP, we avoid having more infected, and that exposure to the virus does not lead consequently to infection due to the effectiveness of PrEP therapy. For our study and others like it, we recommend increasing the use of PrEP in sexually active people, with an emphasis on men.

Table V: Minimum and maximum values for α_P and β_P for the different HIV/AIDS compartments.

	HIV Men		HIV Women		AIDS Men		AIDS Women	
α_P	Min	Max	Min	Max	Min	Max	Min	Max
0.08	36765	81553	38088	53161	8338	21173	3000	10518
0.2	35583	81211	38031	53161	8058	21050	3000	10517
0.4	34301	80719	38805	53161	7757	20877	3000	10515
0.5	33836	80507	37980	53162	7649	20802	3000	10514
0.7	33107	80123	37926	53163	7480	20670	3000	10512
β_P	Min	Max	Min	Max	Min	Max	Min	Max
0.02	36765	81553	38008	53161	8338	21173	3000	10518
0.1	36654	81560	36723	53085	8356	21175	3000	10459
0.25	36954	81569	35122	52959	8383	21179	3000	1364
0.3	36983	81571	34733	52921	8390	21180	3000	10337
0.4	37034	81574	34092	52849	8402	21183	3000	10286

Table VI: Minimum and maximum values for α_{PO} and β_{PO} for the different HIV/AIDS compartments.

	HIV Men		HIV Women		AIDS Men		AIDS Women	
α_{PO}	Min	Max	Min	Max	Min	Max	Min	Max
0.0001	36765	81553	38088	53161	8338	21173	3000	10518
0.001	36754	81547	38006	53161	8333	21171	3000	10517
0.09	35763	80928	37829	53172	8103	20981	3000	10514
0.2	34727	80250	37642	53186	7862	20771	3000	10496
0.5	32637	78749	37255	53221	7377	20304	3000	10518
β_{PO}	Min	Max	Min	Max	Min	Max	Min	Max
0.001	36765	81553	38088	53161	8338	21173	3000	10518
0.01	36765	81556	37916	53137	8337	21173	3000	10507
0.2	36753	81576	36237	52710	8334	21173	3000	10298
0.3	336749	81587	35520	52518	8324	21174	3000	10207
0.6	36737	81612	33838	52037	8329	21174	3000	9984

C. PEP Effect

In this section, we study the influence of the PEP usage rate on HIV/AIDS dynamics. We study the variation of para α_{PO} and β_{PO} independently. We simulate for $\alpha_{PO} = 0.0001, 0.001, 0.09, 0.2, 0.5$ and $\beta_{PO} = 0.001, 0.01, 0.2, 0.3, 0.6$ independently, which were discussed with specialists.

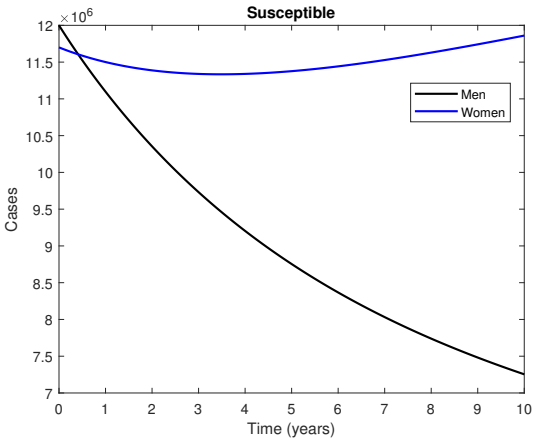
Table IV shows the value of the basic reproduction number for the studied parameter values. In this case of the variation of α_{PO}, we have $\Re_0 = \Re_0^H$ is always greater than unity which implies that with this variation of the parameter the epidemic is not going to disappear using the basic reproduction number as a base. In the case of the variation of β_{PO}, in the subpopulation of women the $\Re_0^M$ is always less than unity but the $\Re_0$ is greater than unity and the epidemic in the population is not extinguished even though the number of HIV and AIDS cases are reducing at the end of the study period, see Figures 11a-11d.

For the variation of α_{PO}, we have that in men and women for higher a, fewer cases are reported. Better results were achieved for males with respect to females. For the cases where $\alpha_{PO} = 0.0001$ and $\alpha_{PO} = 0.001$ the difference in the results was not significant so we need better results in the use of PEP, see Table VI and Figures 11a-11b.

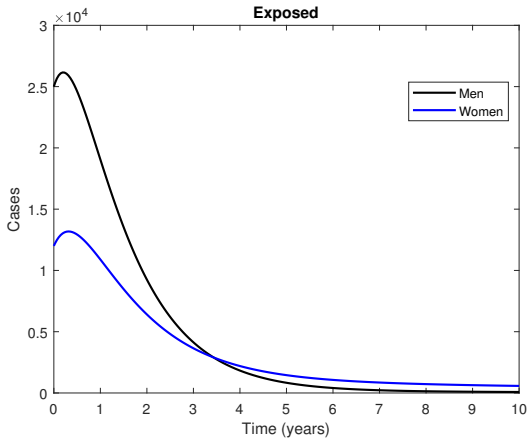
In AIDS cases, the same is true for HIV in men and women. In the case of women, for no value of α_{PO}, the minimum value, which is the initial condition, was able to be reduced, see Table VI and Figures 11c-11d.

For the variations of β_{PO}, we have that for male HIV there is no significant reduction in the number of cases. In this case, we recommend implementing other strategies to reduce male HIV cases. For female HIV cases, a reduction in the number of cases is achieved for higher values of β_{PO}, except for $\beta_{PO} = 0.001$ and $\beta_{PO} = 0.01$ where there is no significant difference in the number of cases. Better results would be obtained if β_{PO} were greater than 0.01, see Figures 12a-12b. For AIDS cases, a similar situation occurs as for HIV. We also fail to reduce the minimum number of women, which is the initial condition (3000 cases), see Table VI and Figures 12c-12d.

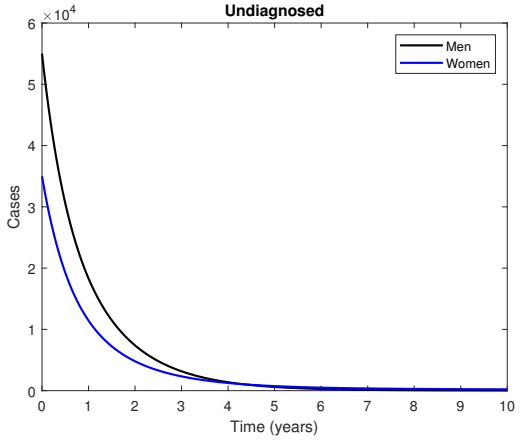
For neither of the variations were we able to reduce the minimum value of female AIDS, which is the initial condition.



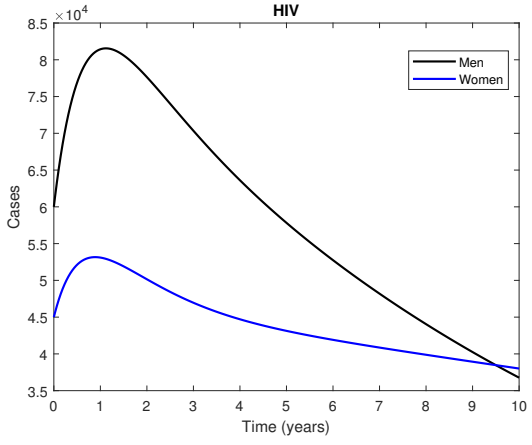
(a) Susceptible behavior.



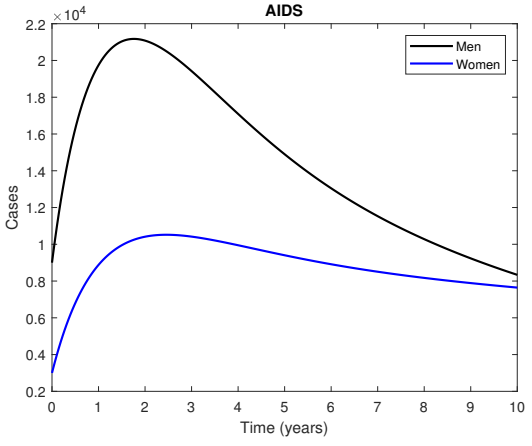
(b) Exposed behavior.



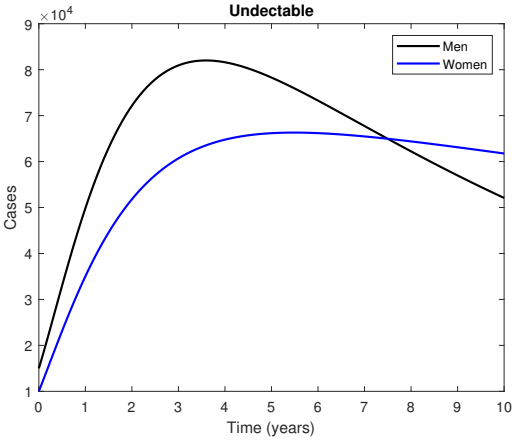
(c) Behavior of undiagnosed with the virus.



(d) HIV cases behavior.

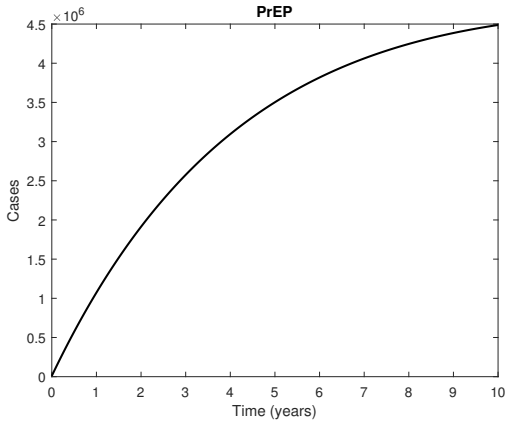


(e) AIDS case behavior.

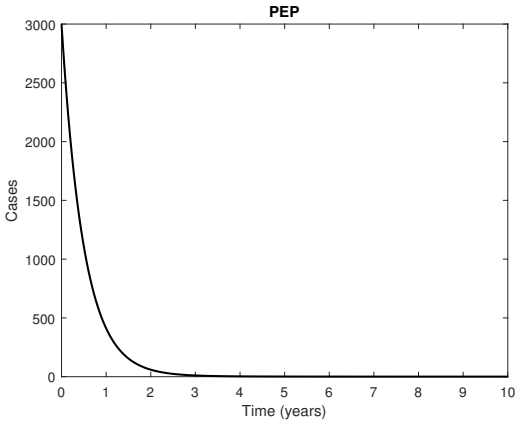


(f) Behavior of cases with undetectable viral load.

Fig. 2: Behavior of men and women compartments for the values in Tables I-II, for a 10-year study.

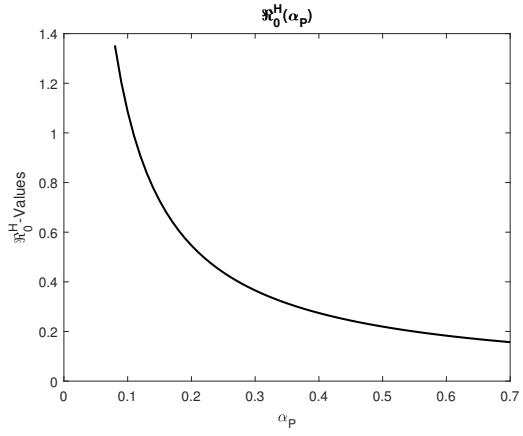


(a) Behavior of PrEP use in the population.

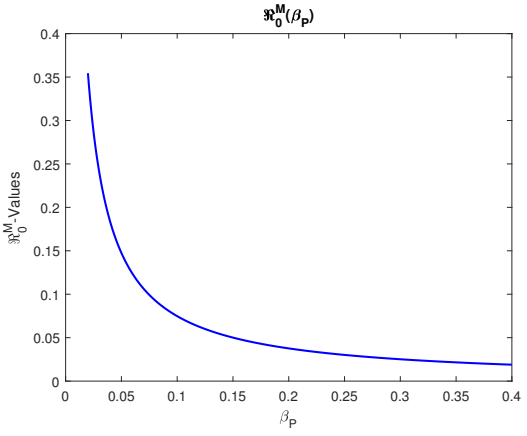


(b) Behavior of the use of PEP in cases that had a risky situation.

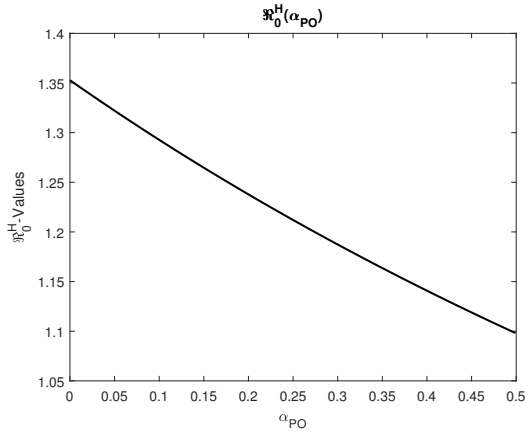
Fig. 3: PrEP and PEP use in the population for the values presented in Tables I-II, for 10 years of study.



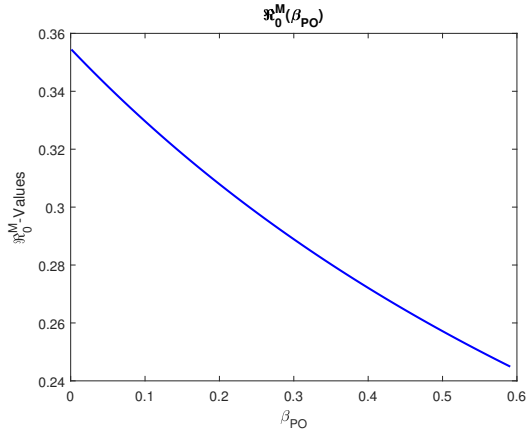
(a) Behavior of $\Re_0^H$ for $\alpha_P \in [0.08, 0.7]$.



(b) Behavior of $\Re_0^M$ for $\beta_P \in [0.02, 0.4]$.

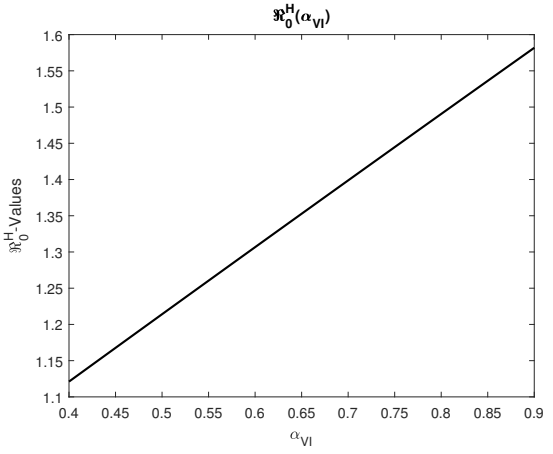


(c) Behavior of $\Re_0^H$ for $\alpha_{PO} \in [0.0001, 0.5]$.

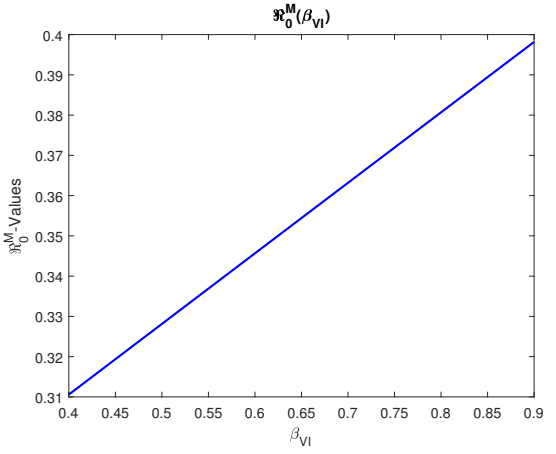


(d) Behavior of $\Re_0^M$ for $\beta_{PO} \in [0.001, 0.6]$.

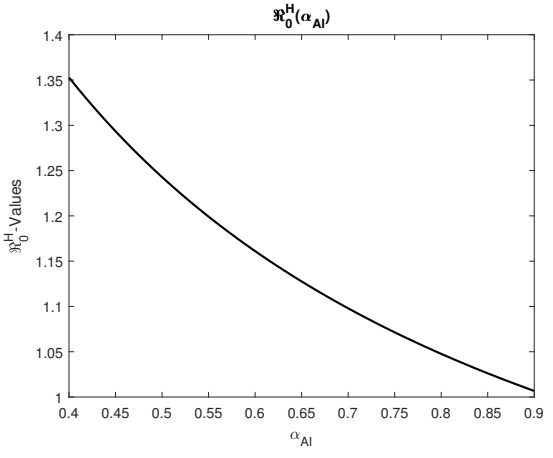
Fig. 4: Behavior of the basic reproduction number with respect to the parameters associated with the use of PrEP and PEP.



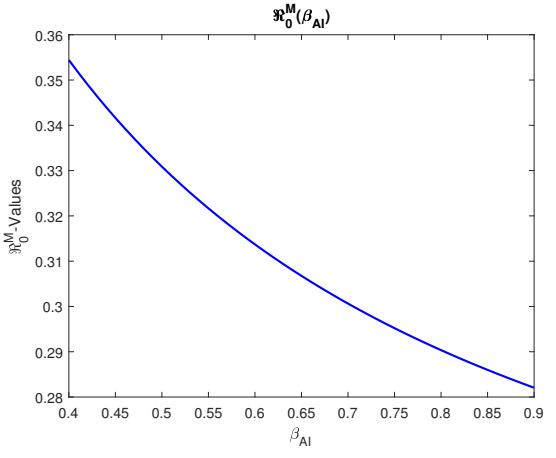
(a) Behavior of $\Re_0^H$ for $\alpha_{VI} \in [0.4, 0.9]$.



(b) Behavior of $\Re_0^M$ for $\beta_{VI} \in [0.4, 0.9]$.



(c) Behavior of $\Re_0^H$ for $\alpha_{AI} \in [0.4, 0.9]$.



(d) Behavior of $\Re_0^M$ for $\beta_{AI} \in [0.4, 0.9]$.

Fig. 5: Behavior of basic reproduction number with respect to parameters associated with undetectability in HIV and AIDS.

D. Undetectable Viral Load Effect

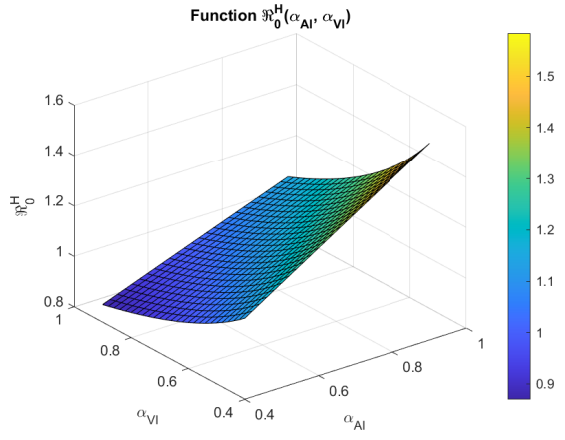
An undetectable HIV viral load is an important achievement in adherence to antiretroviral therapy. In this section, we study the rate of adherence to treatment with respect to viral load from HIV and AIDS status for men and women together. For the variation of α_{VI} and β_{VI}, we study the following scenarios:

- Scenario I: $\alpha_{VI} = 0.65$ and $\beta_{VI} = 0.65$ (fixed values of Table I) with $\Re_0 = 1.3570$.
- Scenario II: $\alpha_{VI} = 0.65$ and $\beta_{VI} = 0.20$ ($\alpha_{VI} > \beta_{VI}$) with $\Re_0 = 1.3570$.
- Scenario III: $\alpha_{VI} = 0.25$ and $\beta_{VI} = 0.45$ ($\alpha_{VI} < \beta_{VI}$) with $\Re_0 = 1.3570$.
- Scenario IV: $\alpha_{VI} = 0.90$ and $\beta_{VI} = 0.90$ (objective of the health systems and WHO) with $\Re_0 = 1.5818$.

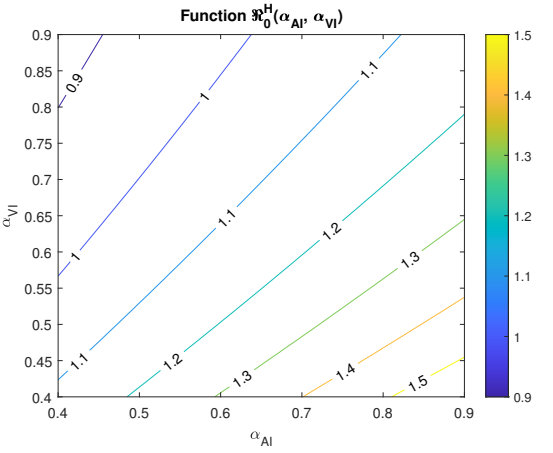
Using the value of $\Re_0$ as a reference, and according to the model, these studied scenarios do not make the epidemic disappear.

In the case of HIV men, scenarios I and II did not have a significant difference in the reduction of the number of HIV cases, the worst results were achieved for scenario III and the best and most significant result regarding the reduction of HIV cases was scenario IV, see Figure 13a. In HIV-positive women, better results are obtained for higher β_{PO} and the most significant reduction in cases was scenario IV, see Figure 13b.

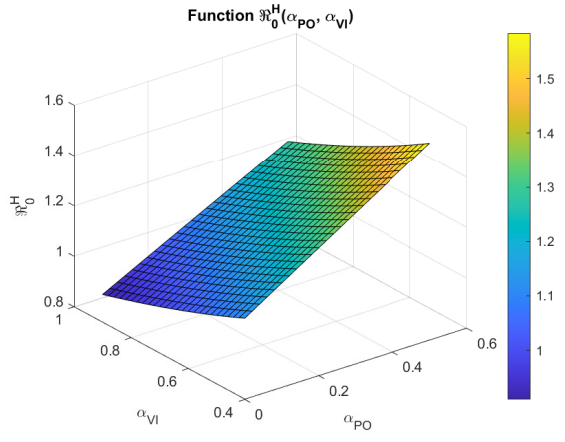
In the case of AIDS for women and men, the best results for both sexes were achieved by scenario IV, see Figures 13c-13d.



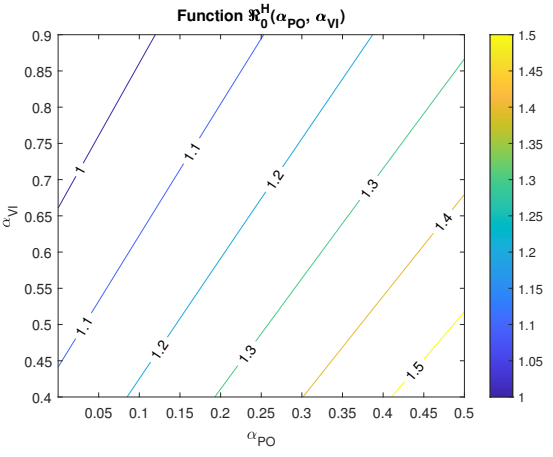
(a) Behavior of $\Re_0^H$ for $\alpha_{AI}, \alpha_{VI} \in [0.4, 0.9]$.



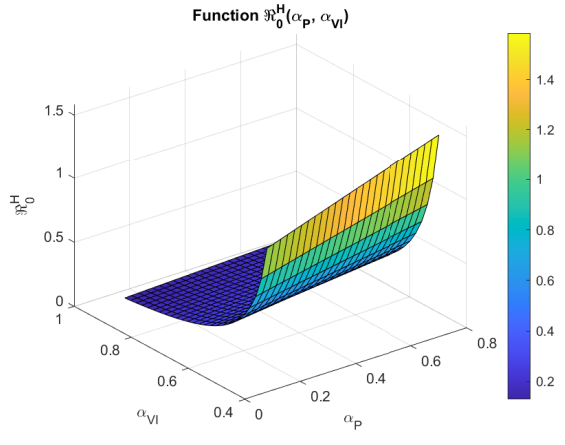
(b) Contour lines $\Re_0^H(\alpha_{AI}, \alpha_{VI})$.



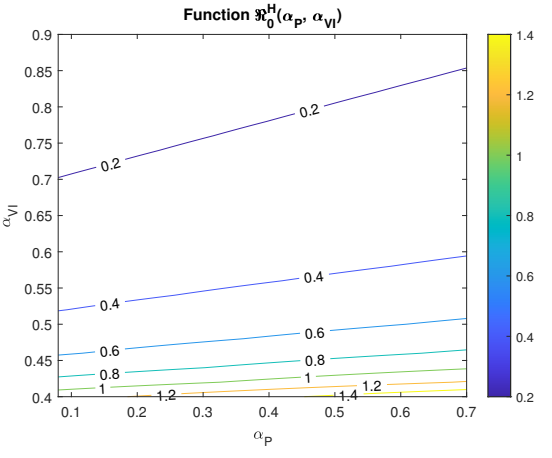
(c) Behavior of $\Re_0^H$ for $\alpha_{PO} \in [0.0001, 0.5]$ and $\alpha_{VI} \in [0.4, 0.9]$.



(d) Contour lines $\Re_0^H(\alpha_{PO}, \alpha_{VI})$.

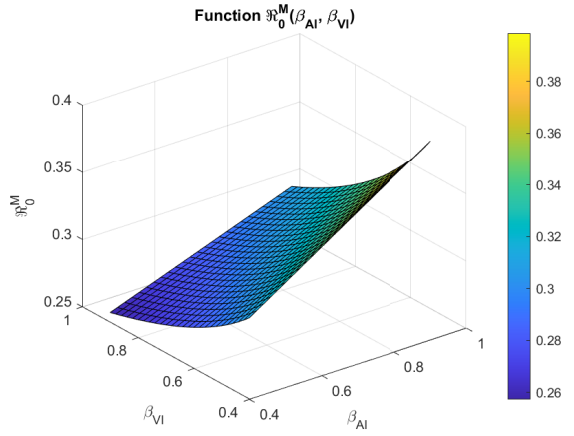


(e) Behavior of $\Re_0^H$ for $\alpha_P \in [0.08, 0.7]$ and $\alpha_{VI} \in [0.4, 0.9]$.

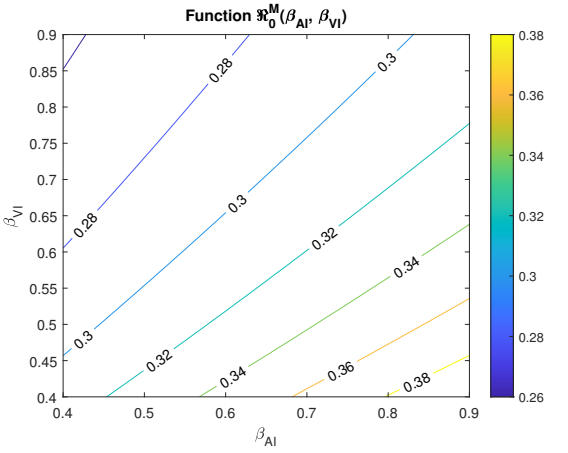


(f) Contour lines $\Re_0^H(\alpha_P, \alpha_{VI})$.

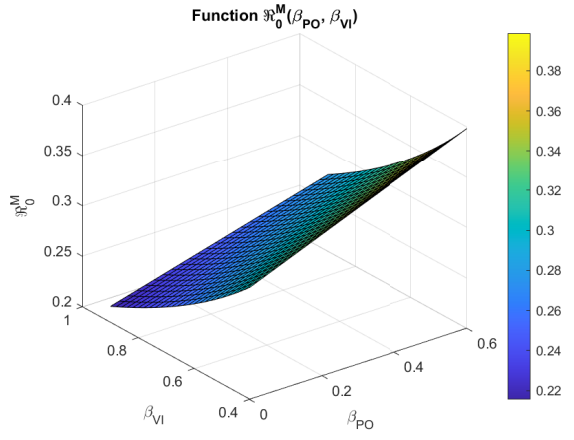
Fig. 6: Graphical behavior for the variation of parameters associated with the use of PrEP, PEP, and undetectability in viral load for HIV and AIDS in the $\Re_0^H$.



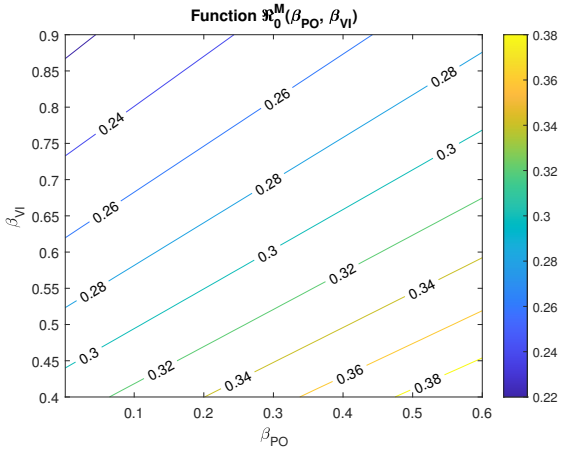
(a) Behavior of $\Re_0^M$ for $\beta_{AI}, \beta_{VI} \in [0.4, 0.9]$.



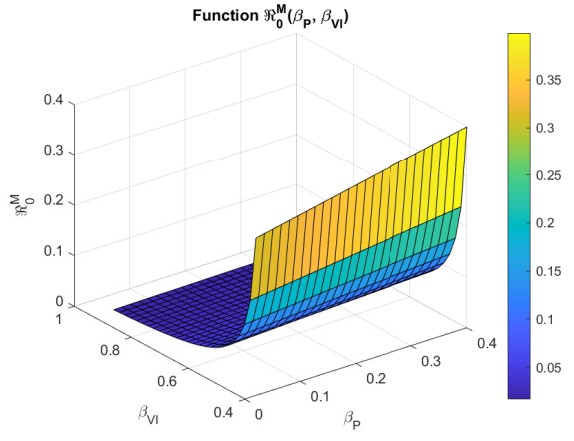
(b) Contour lines $\Re_0^M(\beta_{AI}, \beta_{VI})$.



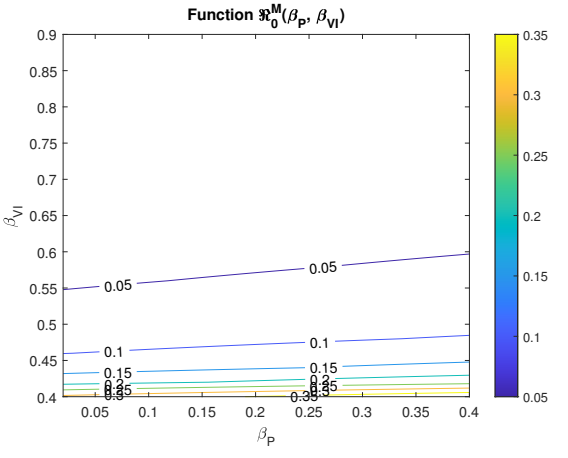
(c) Behavior of $\Re_0^M$ for $\beta_{PO} \in [0.001, 0.6]$ and $\beta_{VI} \in [0.4, 0.9]$.



(d) Contour lines $\Re_0^M(\beta_{PO}, \beta_{VI})$.

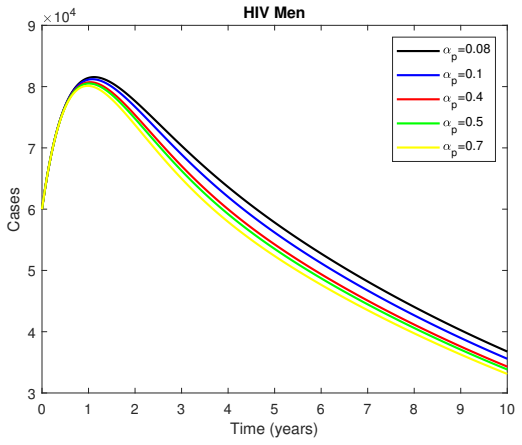


(e) Behavior of $\Re_0^M$ for $\beta_P \in [0.02, 0.4]$ and $\beta_{VI} \in [0.4, 0.9]$.

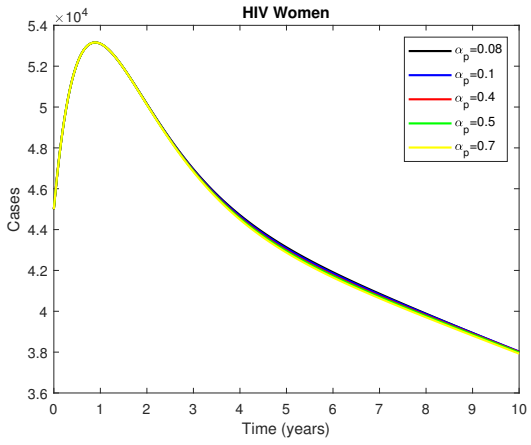


(f) Contour lines $\Re_0^M(\beta_P, \beta_{VI})$.

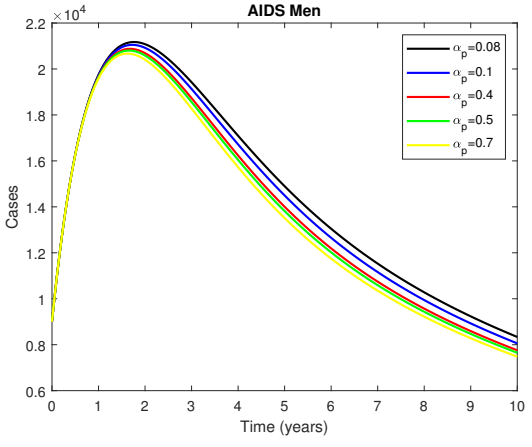
Fig. 7: Graphical behavior for the variation of parameters associated with the use of PrEP, PEP, and undetectability in viral load for HIV and AIDS in the $\Re_0^M$.



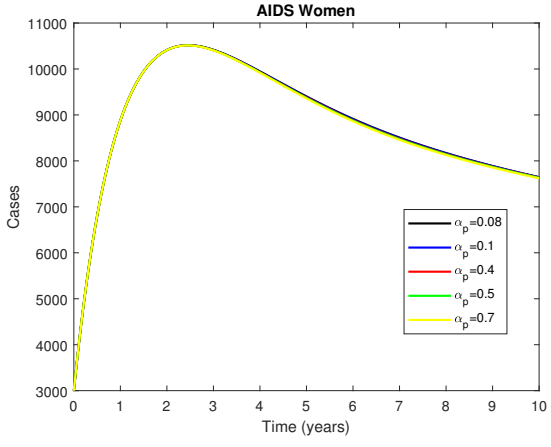
(a) Behavior of HIV-positive men for different α_P.



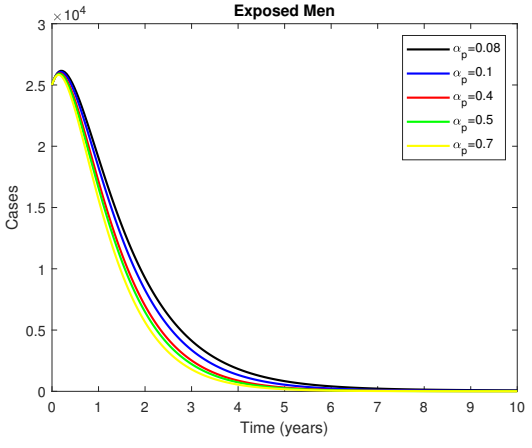
(b) Behavior of HIV-positive women for different α_P.



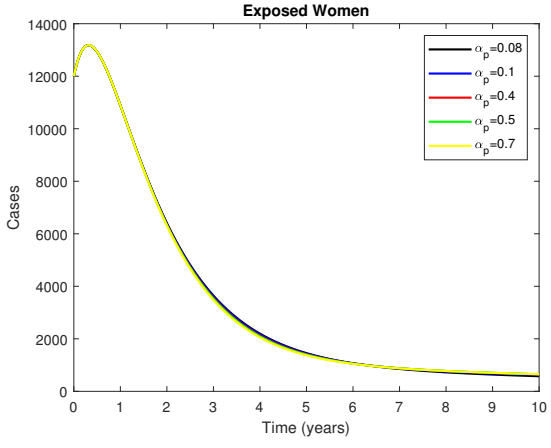
(c) Behavior of AIDS men for different α_P.



(d) Behavior of AIDS women for different α_P.

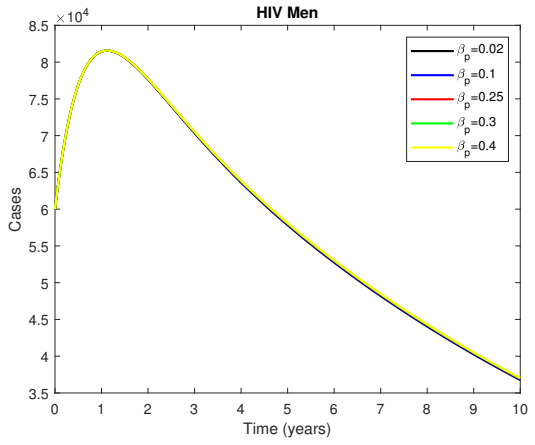


(e) Behavior of exposed men for different α_P.

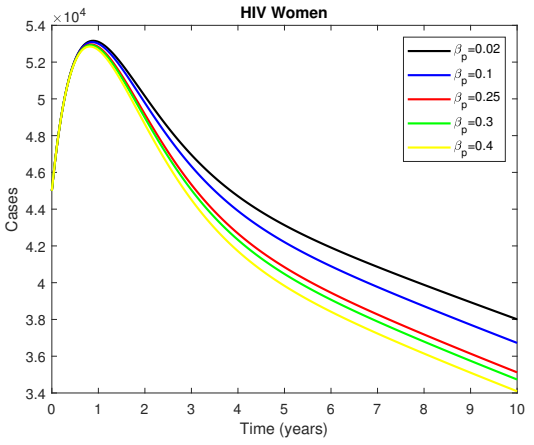


(f) Behavior of exposed women for different α_P.

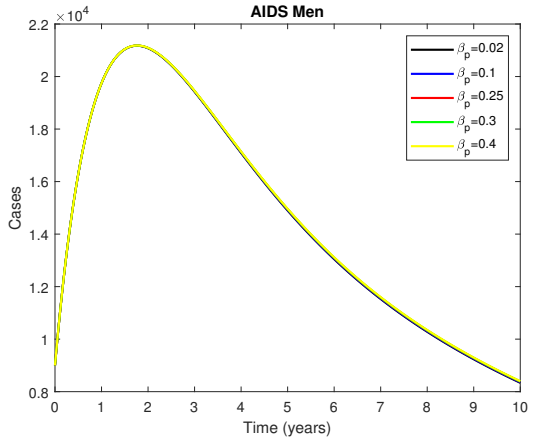
Fig. 8: Behavior of HIV, AIDS and exposed for men and women varying α_P, 10-year study.



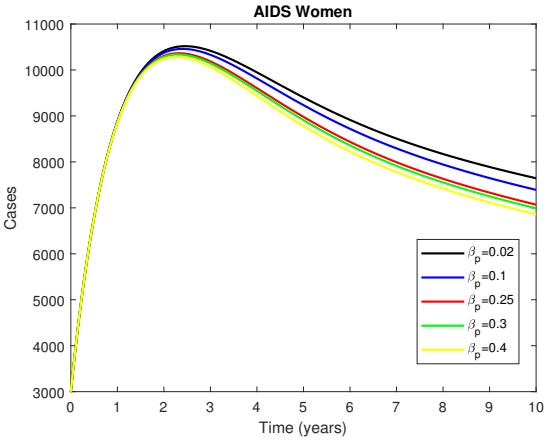
(a) Behavior of HIV-positive men for different β_P.



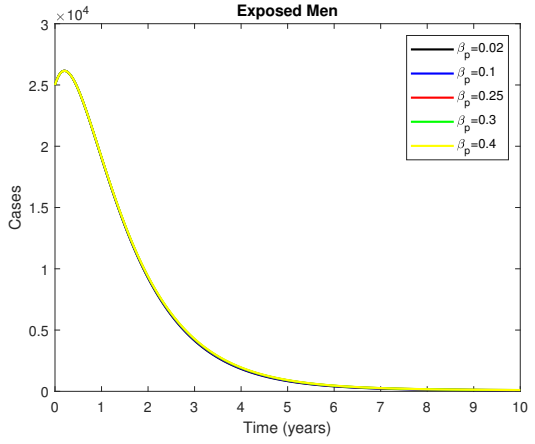
(b) Behavior of HIV-positive women for different β_P.



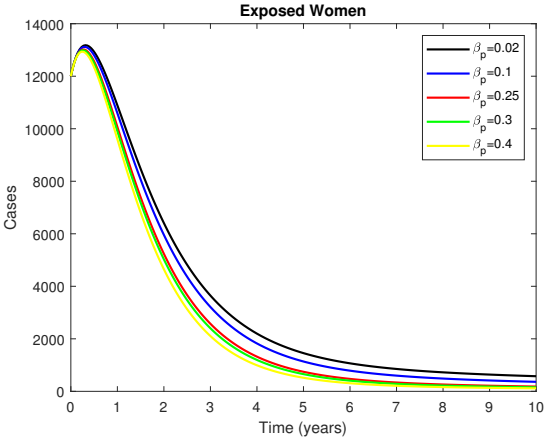
(c) Behavior of AIDS men for different β_P.



(d) Behavior of AIDS women for different β_P.

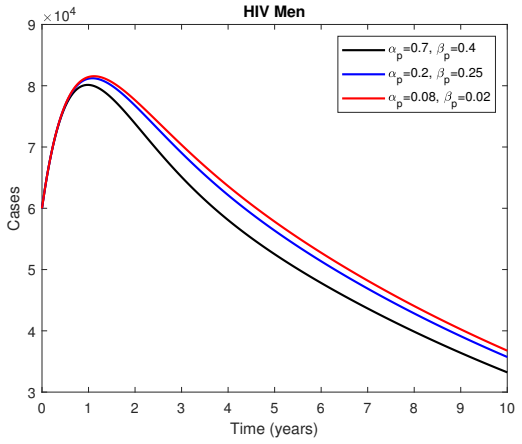


(e) Behavior of exposed men for different β_P.

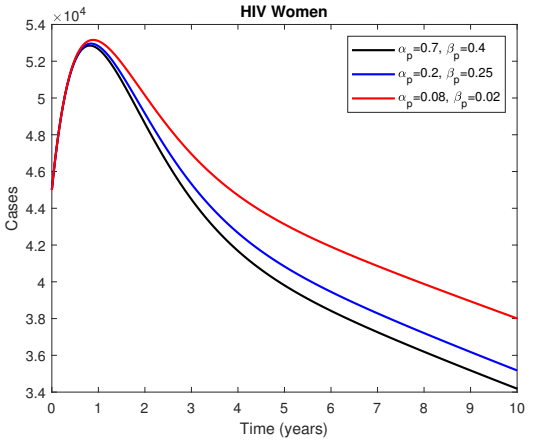


(f) Behavior of exposed women for different β_P.

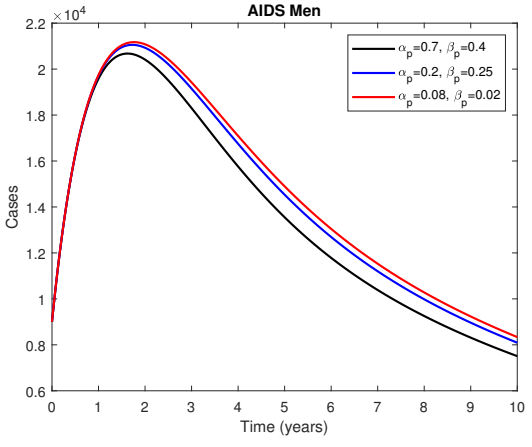
Fig. 9: Behavior of HIV, AIDS and exposed for men and women varying β_P, 10-year study.



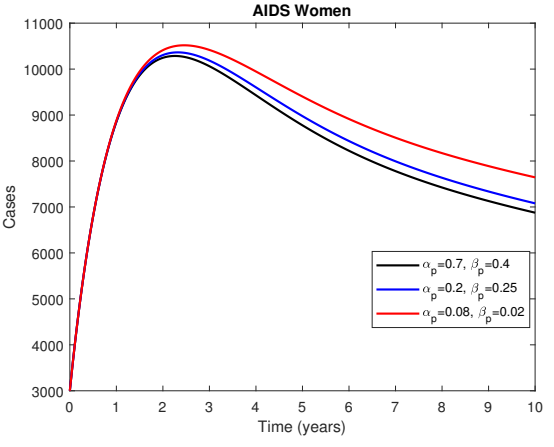
(a) HIV-positive men for different α_P and β_P.



(b) HIV-positive women for different α_P and β_P.



(c) AIDS men for different α_P and β_P.



(d) AIDS women for different α_P and β_P.

Fig. 10: Study of the joint variations of the HIV and AIDS compartments for men and women.

Now, let's study the scenarios for virus undetectability in AIDS:

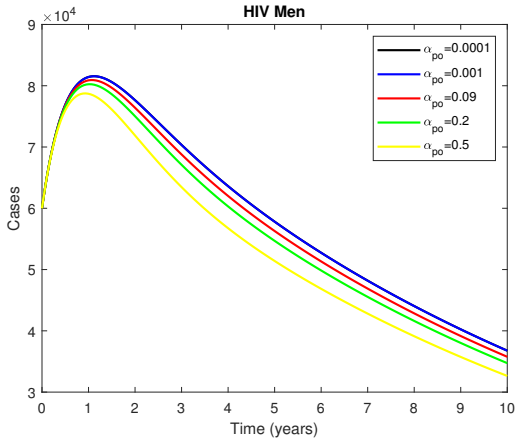
- Scenario I: $\alpha_{AI} = 0.40$ and $\beta_{AI} = 0.40$ (fixed values of Table I)) with $\Re_0 = 1.3528$.
- Scenario II: $\alpha_{AI} = 0.70$ and $\beta_{AI} = 0.20$ ($\alpha_{VI} > \beta_{VI}$) with $\Re_0 = 1.0979$.
- Scenario III: $\alpha_{AI} = 0.30$ and $\beta_{AI} = 0.50$ ($\alpha_{VI} < \beta_{VI}$) with $\Re_0 = 1.5085$.
- Scenario IV: $\alpha_{AI} = 0.90$ and $\beta_{AI} = 0.90$ (objective of the health systems and WHO) with $\Re_0 = 1.0067$.

Using the value of $\Re_0$ as a reference, and according to the model, these studied scenarios do not make the epidemic disappear.

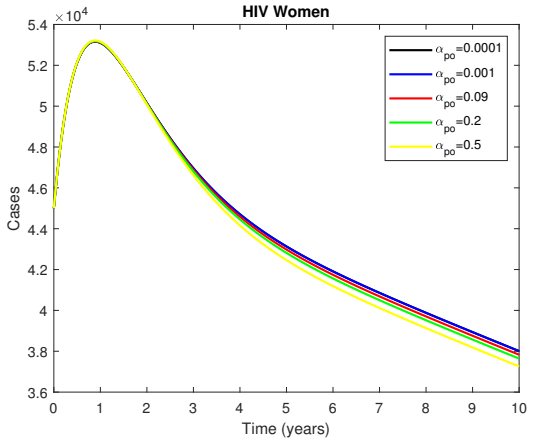
In the case of HIV cases, scenario IV has a variable behavior but does not give the best results in the reduction of HIV cases. In the case of women, the results achieved are better than those for men, see Figure 14a-14b. In women, the best results are achieved in scenario II. In the AIDS cases, for both women and men, the best results are achieved for scenario IV. In the case of men, the results show that for higher α_{AI}, the number of AIDS cases is reduced the most. In the case of women, the same is true for the β_{AI} values, see Figures 14c-14d. These results of independent α_{VI} and β_{VI} variations show that we should think about other joint implementations to achieve better results.

Now, let us study all the joint implementations in the following scenarios:

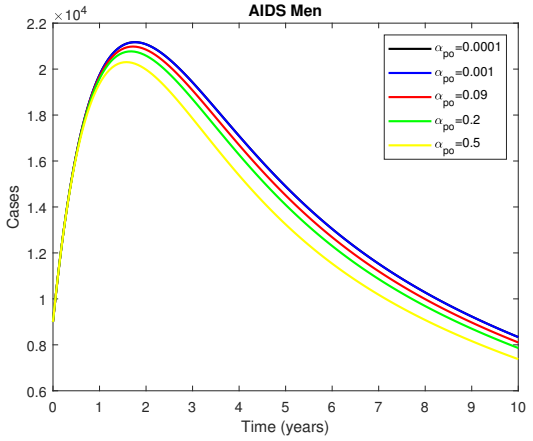
- Scenario I: $\alpha_{VI} = 0.65$, $\beta_{VI} = 0.65$ $\alpha_{AI} = 0.40$ and $\beta_{AI} = 0.40$ (fixed values of Table I) with $\Re_0 = 1.3066$.



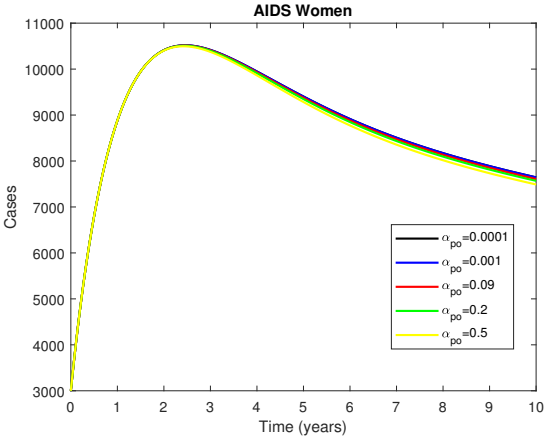
(a) Behavior of HIV-positive men for different α_{PO}.



(b) Behavior of HIV-positive women for different α_{PO}.



(c) Behavior of AIDS men for different α_{PO}.



(d) Behavior of AIDS women for different α_{PO}.

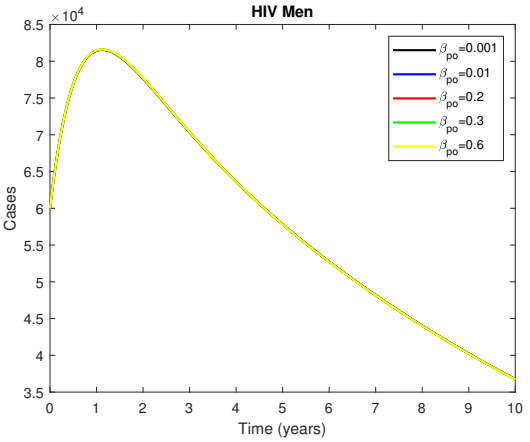
Fig. 11: Study of the compartments of HIV and AIDS for different values of α_{PO}.

- Scenario II: $\alpha_{VI} = 0.65$, $\beta_{VI} = 0.20$ $\alpha_{AI} = 0.70$ and $\beta_{AI} = 0.20$ with $\Re_0 = 1.0979$.
- Scenario III: $\alpha_{VI} = 0.90$, $\beta_{VI} = 0.90$ $\alpha_{AI} = 0.90$ and $\beta_{AI} = 0.90$ with $\Re_0 = 1.1421$.

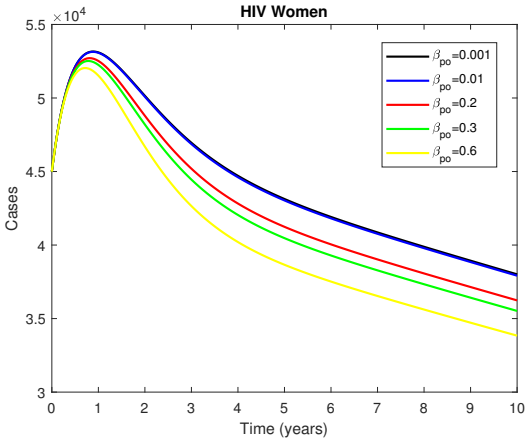
Scenario III uses the parameters with the value 90-90-90 proposed by the WHO, focused on the effectiveness of treatment in HIV and AIDS. According to the model, and using the $\Re_0$ threshold, we see that it is greater than unity in all scenarios, such that with this alone the virus does not disappear from the population.

The best results in the reduction of the number of HIV and AIDS are achieved for scenario III, i.e. the parameters at 90-90-90. This means that by having a treatment adherence rate of 0.90 for HIV and AIDS, it is possible to reduce all compartments of diagnosed infected people, which is an objective proposed by the health system, see Figure 15. Particularly in AIDS men, the second-best scenario is when $\alpha_{VI} > \beta_{VI}$ and $\alpha_{AI} > \beta_{AI}$, see Figure 15c. In AIDS women, when the rates for male and female HIV and male and female AIDS are equal, see 15d. In HIV men, the other two scenarios do not have a significant difference and in HIV women, it is the same as in AIDS females, see Figures 15a-15b.

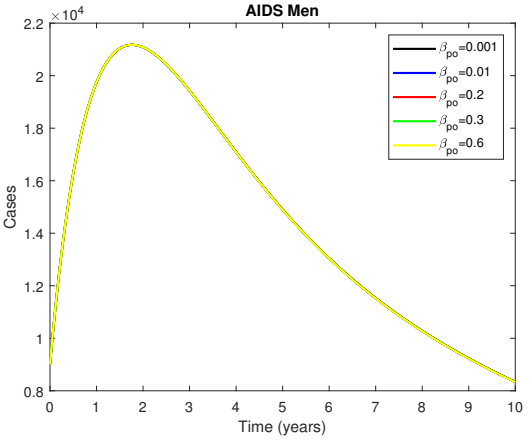
In summary, all the implementations studied (PrEP, PEP, and viral load undetectability due to treatment adherence) and all the proposed scenarios succeed in reducing the impact of HIV/AIDS in the population implementing simultaneously in men and women. Particularly in women with AIDS, we have not managed to reduce the minimum value reached in the initial condition, but the best results are achieved by the



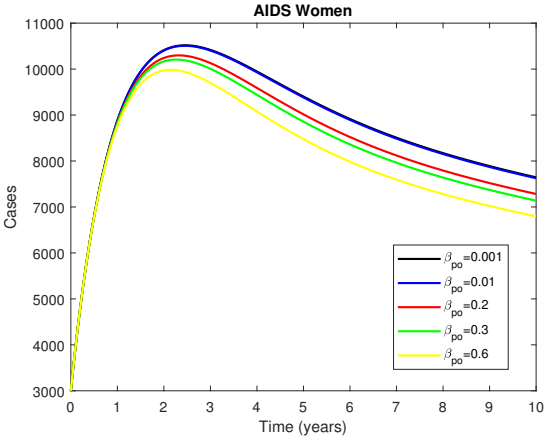
(a) Behavior of HIV-positive men for different β_{PO}.



(b) Behavior of HIV-positive women for different β_{PO}.



(c) Behavior of AIDS men for different β_{PO}.



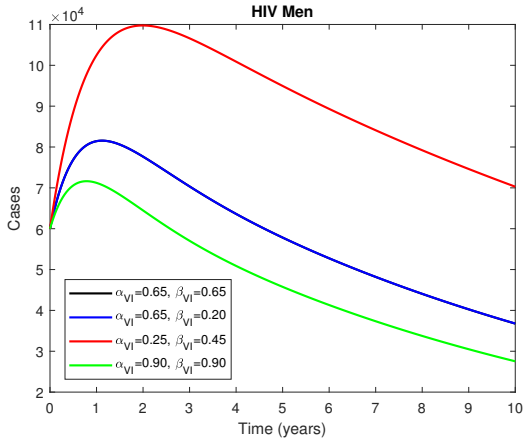
(d) Behavior of AIDS women for different β_{PO}.

Fig. 12: Study of the compartments of HIV and AIDS for different values of β_{PO}.

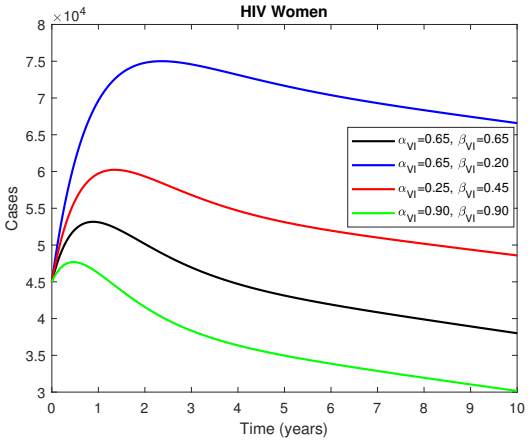
simultaneous HIV and AIDS undetectability. Undetectability rates at 0.90 show the best results and are a goal to be achieved worldwide. We propose to apply simultaneously all the strategies with the objective that with undetectability the infected patients do not transmit the virus, with PrEP we reduce the risk of exposure and the risk of transmission of the virus.

IV. Conclusions

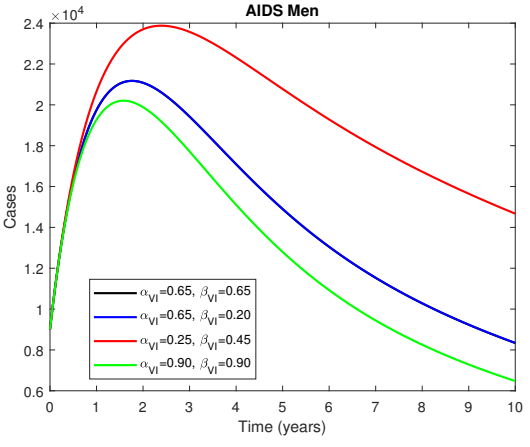
In this paper, we propose a mathematical model to jointly study the use of PrEP, PEP, diagnosis, and viral load undetectability in HIV and AIDS patients in the same dynamic. In the construction of the model, we took into account heterosexual and homosexual sexual relations and infectiousness in HIV and AIDS. We prove the existence and positivity of the solution of the model and found the biologically feasible region. We study the free-infection equilibrium point for the relationship with the basic reproduction number. We study the basic reproduction number for the male and female subpopulations and also for the complete model. We decided to study men independently because of the influence of homosexual relationships on the spread of HIV/AIDS. We demonstrated the global stability of the infection-free equilibrium points by subpopulation and general. To explore our model, we build a scenario with assumed values and data from the literature to perform computational simulations. Among the results obtained for this scenario, we have that the basic reproduction number in women when we vary the parameters associated with the use of PrEP, PEP, and undetectability in HIV and AIDS is always less than unity. In the case of men, when we study the parameter



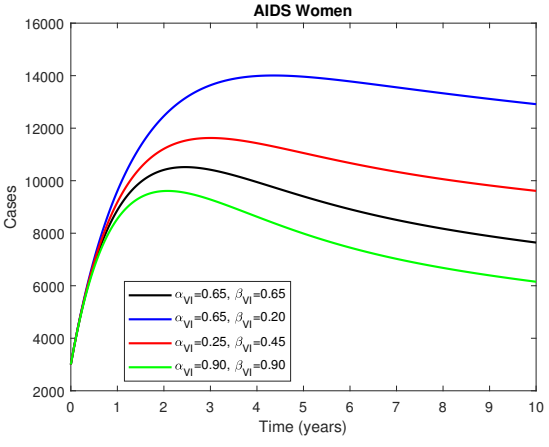
(a) HIV-positive men for different α_{VI} and β_{VI}.



(b) HIV-positive women for different α_{VI} and β_{VI}.



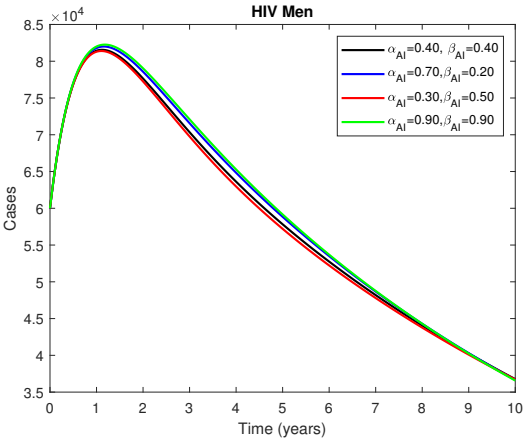
(c) AIDS men for different α_{VI} and β_{VI}.



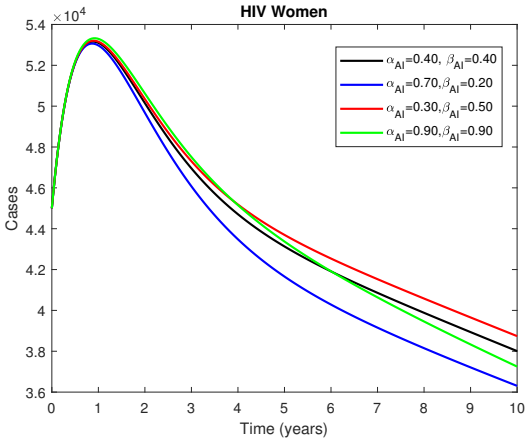
(d) AIDS women for different α_{VI} and β_{VI}.

Fig. 13: Behavior of HIV, AIDS and undetectable for the joint variations of α_{VI} and β_{VI}.

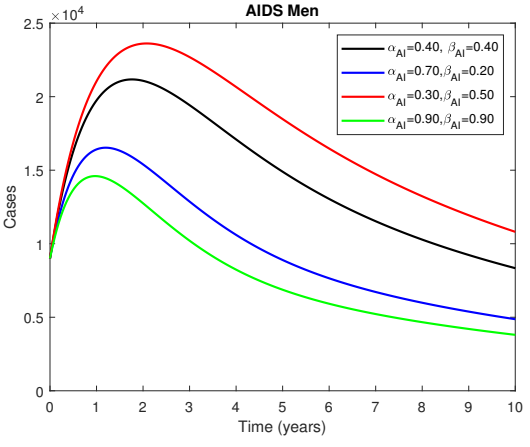
α_{VI} independently is always greater than unity. We study the joint behavior and the relationships between the undetectability between HIV and AIDS and the undetectability in HIV and the use of PrEP we can find situations to reduce the transmissibility of HIV/AIDS in the population. When we study the behavior of the compartments, the subpopulations of women maintain a higher number of susceptibles compared to men, which is a benefit because they expose fewer people to the virus. In the exposed, men at the beginning report a higher number compared to women but at the end of the study, the same situation occurs, but in both subpopulations the exposed decreases over time. In the case of those not diagnosed with the virus, there is a decrease in both subpopulations, which is a favorable factor because we were able to control a greater number of cases. In the HIV and AIDS groups in both subpopulations, there is a growth in the number of reported cases at the beginning and then they decrease. In AIDS, men report more cases than women during the whole study and in HIV the same thing happens but at the end of the period, women report more cases than men. This is evidence that we need to pay attention to the female subpopulation. The number of cases reported using PrEP is increasing throughout the study, and it is a favorable element, because in addition to having these cases controlled with constant examinations, they do not represent a risk of transmission. The number of cases reported using PEP decreases throughout the study, but we must keep in mind that PEP treatment is short and we also have the difficulty of the time to start treatment to be effective.



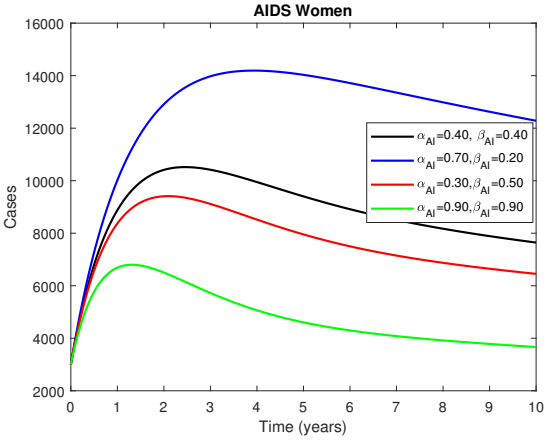
(a) HIV-positive men for different α_{AI} and β_{AI}.



(b) HIV-positive women for different α_{AI} and β_{AI}.



(c) AIDS men for different α_{AI} and β_{AI}.



(d) AIDS women for different α_{AI} and β_{AI}.

Fig. 14: Behavior of HIV, AIDS and undetectable for the joint variations of α_{AI} and β_{AI}.

We also study the direct influence of PrEP, PEP, and undetectability on HIV and AIDS in the groups defined in the model and we can conclude that in the scenarios constructed some variant manages to reduce HIV and AIDS cases but we have a problem in controlling female AIDS. In particular, the best results in the reduction of the number of HIV and AIDS are reached when we achieve an efficacy of 0.90 for viral load undetectability in HIV and AIDS in both sexes which coincides with the WHO's 90-90-90 strategy. This model can help health decision-making authorities to reduce the impact of HIV/AIDS. In future works, we intend to study the problem of optimal control to reduce the impact of HIV/AIDS, study real scenarios, estimation of parameters, and by using other mathematical approaches.

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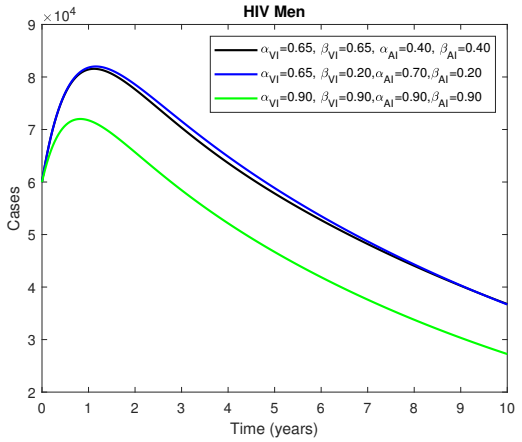
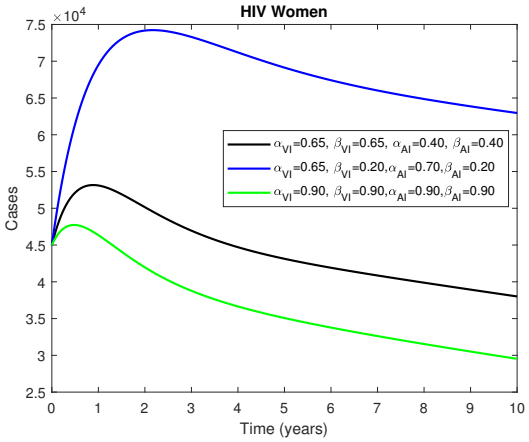
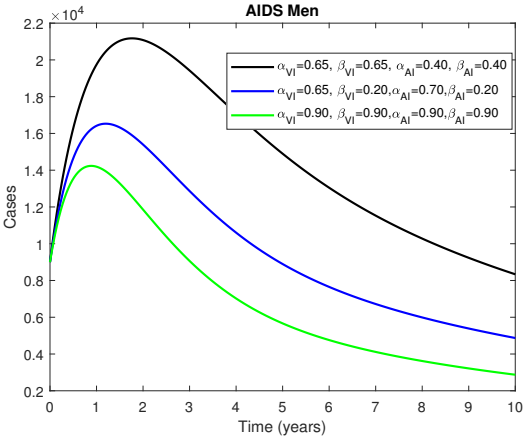


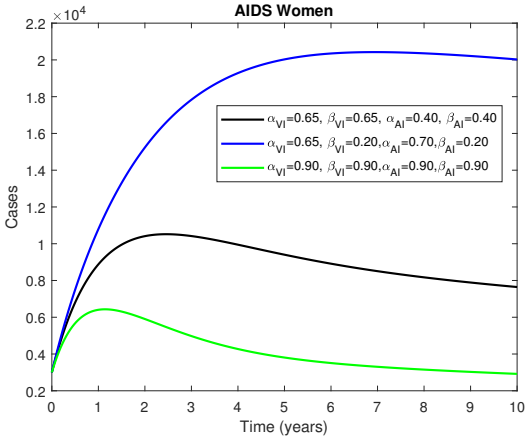
(a) HIV-positive men for different scenarios.



(b) HIV-positive women for different scenarios.



(c) AIDS men for different scenarios.



(d) AIDS women for different scenarios.

Fig. 15: Behavior of HIV and AIDS for joint variation of undetectability parameters for HIV and AIDS.

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