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A TWO-SEX MODEL FOR THE INFLUENCE OF HEAVY ALCOHOL CONSUMPTION ON THE SPREAD OF HIV/AIDS

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Abstract. The HIV/AIDS epidemic, one of the leading public health problems to have affected sub-Sahara Africa, is a multifaceted problem with social, behavioral and biological aspects. In the absence of a cure, behavioral change has been advocated as an intervention strategy for reversing the epidemic. Empirical studies have found heavy alcohol consumption to be a fueling factor for HIV/AIDS infection and progression. Previously [20], we formulated and analyzed a one-sex deterministic model to capture the dynamics of this deadly interaction. But, since alcohol drinking habits, consequent risky sexual practices, alcohol-induced immune suppression, etc., can be different for men and women, the primary objective of our present paper is to construct a two-sex model aimed at shedding light on how both sexes, with varying heavy alcohol consumption trends, contribute differently to the HIV/AIDS spread. Based on numerical simulations, supported by the UNAIDS epidemiological software SPECTRUM and using the available data, our study identifies heavy drinking among men and women to be a major driving force for HIV/AIDS in Botswana and sub-Sahara Africa and quantifies its hazardous outcomes in terms of increased number of active TB cases and economic burden caused by increased need for AntiRetroviral Therapy (ART). Our simulations point to the heavy-drinking habits of men as a major reason for the continuing disproportionate impact of HIV/AIDS on women in sub-Sahara Africa. Our analysis has revealed the possibility of the phenomenon of backward bifurcation. In contrast to the result in some HIV vaccination models [52], backward bifurcation in our model is not removed by replacing the corresponding standard incidence function with a mass action incidence, but is removed by merging the two susceptible classes of the same sex into one, i.e., by ignoring acquisition of, and ongoing recovery from, heavy-drinking habits among the susceptible population.

1. **Introduction.** Since the early 80's, most countries worldwide have been battling to control the spread of HIV and AIDS. An estimated $33.4m$ ($31.1m$ - $35.8m$) individuals are infected with HIV which, in 2008 alone, claimed the lives of $2m$ ($1.7m$ - $2.4m$) infectives [75]. Although HIV is a global health issue, the disease has mostly affected sub-Sahara Africa. One in 20 adults in this region is estimated to be living with HIV. Globally, 67% of people living with HIV and 90% of HIV infected children under the age of 15 reside in sub-Sahara Africa [75]. Southern Africa is the worst affected region of sub-Sahara Africa with more than 35% of

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HIV infections and 38% of AIDS deaths occurring in the region in 2007 [74]. Heterosexual intercourse remains the primary mode of transmission of the disease in sub-Sahara Africa. Although globally, women account for half of all people living with HIV/AIDS, in sub-Sahara Africa the proportion of women infected with HIV/AIDS is 60% [75], representing a significant skewness in the number of HIV infected towards females. In Botswana, a country with a population of about 1.8 m, of which 48% are males and 52% females [9], it is estimated that the national HIV prevalence rate for 2008 was 17.6% [8], with the gender-wise prevalence significantly higher among females (20.4%), compared to males (14.2%). The average incidence rate for 2008 also follows a similar pattern with a national estimate of 2.9%, resulting in a gender distribution among females and males of 3.5% and 2.3% respectively.

Another alarming trend seen worldwide and especially in the African region is the increasing alcohol consumption [81]. Worldwide, it is estimated that about 2 billion people consume alcoholic beverages to varying degrees [81]. WHO estimates [81] the proportions of the drinking population among males and females in Africa E region[1], to be 55% and 30% respectively, and about 10.3% of the drinking population in these countries aged 15 and above is classified as heavy-drinking. Sub-Sahara Africa is also known to have the highest estimated total alcohol consumption (in liters of absolute alcohol) per adult drinker, which is 16.6 liters of alcohol [81]. Studies [10][44] have shown that globally, there are gender differences in drinking behavior between males and females with males consuming more alcohol, on average, than females, although in recent years the gap has been growing smaller [48][55][79], particularly in low-income countries and noticeably among younger women [26][34][57][82].

From the time HIV/AIDS was detected as a disease, it was recognized that the disease was as much a social and behavioral problem as it was a biological one [11]. Hence, behavioral change has always been advocated as a strategy for reversing the epidemic. There are many behavioral factors that have been fueling the HIV/AIDS epidemic, heavy alcohol consumption being one of them [24]. Although, there is strong evidence linking heavy alcohol consumption to high risky sexual behavior in most cultures [71], it is only recently that attention is being given to studying the impact of this link [46] and various studies have since explored this relationship [1][6][47]. Numerous studies conducted in sub-Sahara Africa have found that people who drink alcohol irresponsibly are at a higher risk of contracting HIV than moderate and responsible drinking individuals, and quantify the risk to be at least 50% greater for heavy alcohol users [2][23][27][60][62]. Moreover, a cross-cultural study in eight countries [80] on correlational association between alcohol use indicators (frequency, quantity, problems) and risky sex indicators (regretted intercourse, number of partners) and supported by several studies and reviews [5][6][21][36][39][40] [49][53][59][61][63]-[66][68][78] found positive correlation between these indicators. In Southern Africa, the deviant behavior [33][51][72] of having multiple and concurrent partners is found to be common among both males and females. The study by D. de Walque et.al. [14] found that in about 30% - 40% of the infected couples, only the woman was found to be infected indicating that extra marital sexual activities among women in union is a substantial source of vulnerability to HIV. In Botswana the latest AIDS Impact survey shows that 38.6% of youngsters in the age group 15-24 have had sex with a non-marital, non-cohabiting sexual partner in the last

[1]WHO Africa E region consists of those African countries with high child and very high adult mortality rates and include, among others, countries like Botswana, South Africa, and Zimbabwe.

12 months whereas the corresponding percentage for women and men aged 15-49 who had sexual intercourse with more than one partner during the same period was 11.2% [8]. An intensive, longitudinal structured daily phone interview study held among 82 HIV- positive individuals for 42 days in South Africa, found that of all the factors mentioned, the amount of alcohol consumed before sex was the determinant of the proportion and the number of unprotected sex events that occurred [67]. Since more frequent episodes of alcohol intoxication can be associated in a dose-dependent manner with an increased risk of having more sexual partners [28] and since concurrent partnerships can exponentially increase the number of infected individuals and the growth rate of the epidemic during the initial phase [50][58][66], the role of heavy alcohol consumption in the spread of the epidemic has become critical.

Apart from fueling risky sex behaviors, alcohol can form a deadly pair with HIV/AIDS [15][18], and make heavy consumers of alcohol more susceptible to HIV [19][35], affect treatment adherence [4][13][15][17][29][32][43] and hasten the progression of the infection [22][25]. Heavy alcohol consumption is associated with a lower CD4 cell count among HIV infected persons not receiving Anti Retroviral Treatment (ART) [30], making heavy drinkers to have a higher than normal incidence of pathogenic and opportunistic infections [70] and Protein Energy Malnutrition [12], and studies [16] also suggest differential effects of alcohol on males' and females' immune responses causing gender differences in alcohol-induced immune suppression. Thus, the impact of alcohol consumption on the infection and progression of HIV/AIDS is a critical and topical subject, one which involves crucial gender difference. In [20] we constructed a one-sex model to quantify the impact of heavy alcohol consumption on infection and progression of HIV/AIDS. But in order to quantify the spread of the disease, and the severity caused thereby, among men and women separately, against differential patterns of heavy alcohol consumption and consequent risky sexual behaviors, immune responses as well as treatment adherence, we design a two-sex deterministic model in this paper.

With regard to the severity caused by the disease, we also look at the increased incidence of tuberculosis (TB) activated by HIV using the software SPECTRUM [73] which uses the prevalence determined from our model and the Botswana demographic data for age groups 15-49 years. Although there are several HIV/AIDS opportunistic infections, it is known that TB is the leading infectious killer of people living with HIV, and accounts for an estimated 13% of AIDS deaths worldwide. Since HIV and TB epidemics reinforce one another, they are often referred to as co-epidemics or dual epidemics [76]. Also, as the number of HIV infected increases, the need for ART increases as well, putting additional burden on many national economies. Based on the prevalence levels and the demographic data for Botswana, we have determined the need for ART for various scenarios. Simulation results based on our model are helpful to quantify the burden caused by HIV/AIDS.

The organization of the paper is as follows: Section 2 contains the description and analysis of the model; Section 3 deals with a special case of the model with mass action incidence; Section 4 is another special case of the model with one susceptible class for each sex; Numerical simulation results are included in Section 5 and Discussion of results in Section 6. An Appendix follows with details of computations as well as tables with description of parameter values.

2. **Description of the model.** In this model, we extend the one-sex model by Thomas and Lungu [20] which investigated the impact of behavioral change on the spread of HIV/AIDS in a population in which an adult individual was classified as either a moderate or a heavy-drinker and belonging to either a class of susceptible, or infected or AIDS individuals. In this paper, by 'heavy-drinking' males, we will refer to males who habitually drink more than 14 drinks per week or more than 5 drinks per occasion; for women the respective thresholds will be more than 7 drinks per week or more than 4 drinks per occasion. Males or females taking alcohol below the above respective thresholds will be referred to as 'moderate' drinkers [7][42][77]. We include non-drinkers also in the class of moderate drinkers. By the term 'ongoing recovery', we will refer to ongoing recovery of heavy-drinkers from heavy-drinking habits to moderate drinking. In this study, our primary purpose is to develop a two-sex model, with four sub-classes susceptible to HIV infection (who will be generally referred to as 'susceptibles'), two each for both sexes, aimed at understanding how both sexes contribute to the spread of HIV/AIDS, taking into consideration the differential aspects of alcohol drinking habits, consequent risky sexual practices, alcohol-induced immune suppression, etc., for men and women, one for which parameters can be plausibly estimated based on survey and clinical data.

We compartmentalize the adult population (aged 15+) as follows: The male population is subdivided into moderate susceptible males, S_{N_m}, heavy-drinking susceptible males, S_{D_m}, moderate males infected with HIV, I_{N_m}, (who will be referred to as 'moderate infective males'), heavy-drinking males infected with HIV, I_{D_m}, (who will be referred to as 'heavy-drinking infective males'), moderate males living with AIDS, A_{N_m}, (who will be referred to as 'moderate MLWA'), and heavy-drinking males living with AIDS, A_{D_m}, (who will be referred to as 'heavy-drinking MLWA'). Similarly, the female population is divided into moderate susceptible females, S_{N_f}, heavy-drinking susceptible females, S_{D_f}, moderate females infected with HIV, I_{N_f}, (who will be referred to as 'moderate infective females'), heavy-drinking females infected with HIV, I_{D_f}, (who will be referred to as 'heavy-drinking infective females'), moderate females living with AIDS, A_{N_f}, (who will be referred to as 'moderate FLWA'), and heavy-drinking females living with AIDS, A_{D_f} (who will be referred to as 'heavy-drinking FLWA').

Let Π_m and Π_f denote constant recruitment rates into the classes of susceptible males and females respectively and let p_m $(0 \leq p_m \leq 1)$ $[p_f$ $(0 \leq p_f \leq 1)]$ denote the proportion of susceptible males [females] who are moderate drinkers and $(1 - p_m)$ $[(1 - p_f)]$, the proportion of susceptible males [females] who are heavy-drinkers. Denote by μ_m and μ_f the constant per capita death rates for male and female sub-populations, respectively, in the absence of HIV/AIDS. We consider only hetero-sexual transmission of HIV which is the primary mode of transmission in sub-Sahara Africa. In our model, the susceptible male population is infected following effective contacts with any of the four infected female classes (moderate and heavy-drinking infective females and FLWA) and similarly the susceptible female population is infected following effective contacts with any of the four infected male classes (moderate and heavy-drinking infective males and MLWA). Suppose that the moderate susceptible males can acquire HIV infection through contact with moderate infective females at a transmission rate $\beta_{fm} = c_f \gamma_f$ where c_f is the average number of moderate susceptible male sexual partners for a moderate female infective per unit time and γ_f is the disease transmission probability per

unit time per partnership, with moderate susceptible males, for moderate infective females which, in turn, is given by $\gamma_f = [1-(1-\eta_{fm})^{n_f}]$, where η_{fm} represents the probability of HIV transmission per coital act from female to male and n_f is the average number of coital acts per partnership, with moderate susceptible males, for a moderate infective female per unit time. Similarly, suppose that the moderate susceptible females can acquire HIV infection through contact with moderate infective males at a transmission rate $\beta_{mf} = c_m\gamma_m$ where c_m is the average number of moderate susceptible female sexual partners for a moderate male infective per unit time and γ_m is the disease transmission probability per unit time per partnership, with moderate susceptible females, for moderate infective males which, in turn, is given by $\gamma_m = [1-(1-\eta_{mf})^{n_m}]$, where η_{mf} represents the probability of HIV transmission per coital act from male to female and n_m is the average number of coital acts per partnership, with moderate susceptible females, for a moderate male infective per unit time. We have assumed that the heavy-drinking infective males (females) have a higher chance, relative to moderate infective males (females), of generating new infections among moderate susceptible females (males) by a factor $\psi_{1_m} \geq 1$ ($\psi_{1_f} \geq 1$). Similarly, the modification parameter for the moderate MLWA (FLWA) for generating new infections among moderate susceptible females (males) is taken to be $\psi_{2_m} > 0$ ($\psi_{2_f} > 0$). Likewise, we have assumed that the heavy-drinking MLWA (FLWA) have a higher chance, relative to moderate male (female) infectives, of generating new infections among moderate susceptible females (males) by a factor $\psi_{3_m} > 0$ ($\psi_{3_f} > 0$). We also assume that heavy-drinking susceptible males (females), S_{D_m} (S_{D_f}), have a higher chance, relative to moderate susceptible males (females), S_{N_m} (S_{N_f}), of acquiring new infections following contacts with the respective infected class by a factor $\omega_m \geq 1$ ($\omega_f \geq 1$).

Let ψ_{N_m} (ψ_{N_f}) denote the rate at which moderate susceptible males (females) become heavy-drinking susceptible males (females) and ψ_{D_m} (ψ_{D_f}) denote the rate at which heavy-drinking susceptible males (females) become moderate susceptible males (females). Moderate infective males and females can, for various reasons, become heavy-drinkers at a constant transfer rate α_{N_m} and α_{N_f} respectively and heavy-drinking infective males and females can, through counseling, or due to health reasons, etc., change their drinking habits and become moderate male and female infectives at constant ongoing recovery rates α_{D_m} and α_{D_f}, respectively. Let ε_{N_m} and ε_{N_f} denote the progression rates to the AIDS class for moderate male and female infectives respectively and ε_{D_m} and ε_{D_f}, the corresponding progression rates for heavy-drinking male and female infectives respectively. Similarly, let δ_{N_m} and δ_{D_m} denote mortality rates due to AIDS for moderate and heavy-drinking MLWA respectively and δ_{N_f} and δ_{D_f} be the corresponding mortality rates for moderate and heavy-drinking FLWA respectively. It is also possible for moderate MLWA and FLWA to become heavy-drinkers at constant rates ω_{N_m} and ω_{N_f} respectively and heavy-drinking MLWA and FLWA can change their heavy drinking behavior due to many reasons and become moderate drinkers at constant ongoing recovery rates ω_{D_m} and ω_{D_f} respectively.

The dynamics of the model is presented in the following system of ODEs and its flow chart is given in Figure 1:

$$
\begin{aligned}
\dot{S}_{N_m} &= p_m\Pi_m + \psi_{D_m}S_{D_m} - (\mu_m + \lambda_f + \psi_{N_m})S_{N_m} \\
\dot{S}_{D_m} &= (1-p_m)\Pi_m + \psi_{N_m}S_{N_m} - (\mu_m + \omega_m\lambda_f + \psi_{D_m})S_{D_m} \\
\dot{I}_{N_m} &= \lambda_f S_{N_m} + \alpha_{D_m}I_{D_m} - Q_{1_m}I_{N_m} \\
\dot{I}_{D_m} &= \omega_m\lambda_f S_{D_m} + \alpha_{N_m}I_{N_m} - Q_{2_m}I_{D_m} \\
\dot{A}_{N_m} &= \varepsilon_{N_m}I_{N_m} + \omega_{D_m}A_{D_m} - Q_{3_m}A_{N_m} \\
\dot{A}_{D_m} &= \varepsilon_{D_m}I_{D_m} + \omega_{N_m}A_{N_m} - Q_{4_m}A_{D_m} \\
\dot{S}_{N_f} &= p_f\Pi_f + \psi_{D_f}S_{D_f} - (\mu_f + \lambda_m + \psi_{N_f})S_{N_f} \\
\dot{S}_{D_f} &= (1-p_f)\Pi_f + \psi_{N_f}S_{N_f} - (\mu_f + \omega_f\lambda_m + \psi_{D_f})S_{D_f} \\
\dot{I}_{N_f} &= \lambda_m S_{N_f} + \alpha_{D_f}I_{D_f} - Q_{1_f}I_{N_f} \\
\dot{I}_{D_f} &= \omega_f\lambda_m S_{D_f} + \alpha_{N_f}I_{N_f} - Q_{2_f}I_{D_f} \\
\dot{A}_{N_f} &= \varepsilon_{N_f}I_{N_f} + \omega_{D_f}A_{D_f} - Q_{3_f}A_{N_f} \\
\dot{A}_{D_f} &= \varepsilon_{D_f}I_{D_f} + \omega_{N_f}A_{N_f} - Q_{4_f}A_{D_f}
\end{aligned}
\tag{1}
$$

where

$$
\begin{aligned}
\lambda_f &= \frac{\beta_{fm}(I_{N_f} + \psi_{1_f}I_{D_f} + \psi_{2_f}A_{N_f} + \psi_{3_f}A_{D_f})}{N_f} \\
\lambda_m &= \frac{\beta_{mf}(I_{N_m} + \psi_{1_m}I_{D_m} + \psi_{2_m}A_{N_m} + \psi_{3_m}A_{D_m})}{N_m} \\
N_j &= S_{N_j} + S_{D_j} + I_{N_j} + I_{D_j} + A_{N_j} + A_{D_j} \\
Q_{1_j} &= \alpha_{N_j} + \mu_j + \varepsilon_{N_j}, \qquad Q_{2_j} = \alpha_{D_j} + \mu_j + \varepsilon_{D_j} \\
Q_{3_j} &= \omega_{N_j} + \mu_j + \delta_{N_j}, \qquad Q_{4_j} = \omega_{D_j} + \mu_j + \delta_{D_j}, \qquad j = m, f.
\end{aligned}
$$

Lemma 2.1. *The closed set*

$$
\begin{aligned}
\mathcal{M}_1 = \{&(S_{N_m}, S_{D_m}, I_{N_m}, I_{D_m}, A_{N_m}, A_{D_m}, S_{N_f}, S_{D_f}, I_{N_f}, I_{D_f}, A_{N_f}, A_{D_f}) \in R_+^{12} \\
&: S_{N_m} + S_{D_m} + I_{N_m} + I_{D_m} + A_{N_m} + A_{D_m} + S_{N_f} + S_{D_f} + I_{N_f} + I_{D_f} \\
&+ A_{N_f} + A_{D_f} \leq \frac{\Pi}{\mu}\}
\end{aligned}
$$

where $\Pi = \Pi_m + \Pi_f$ *and* $\mu = min\{\mu_m, \mu_f\}$ *is positively invariant and attracting with respect to the model (1).*

Proof. Since model (1) deals with humans, all state variables and parameter values are assumed to be non-negative for all $t \geq 0$ (See Tables 1 and 2). From the equation for the total population, we have

$$
\begin{aligned}
\dot{N} &= \dot{N}_m + \dot{N}_f \\
&= \Pi_m + \Pi_f - \mu_m N_m - \mu_f N_f - \delta_{N_m}A_{N_m} - \delta_{N_f}A_{N_f} - \delta_{D_m}A_{D_m} - \delta_{D_f}A_{D_f} \\
&\leq \Pi - \mu_m N_m - \mu_f N_f \\
&\leq \Pi - \mu N
\end{aligned}
\tag{2}
$$

where $\Pi = \Pi_m + \Pi_f$, $N = N_m + N_f$ and $\mu = min\{\mu_m, \mu_f\}$.

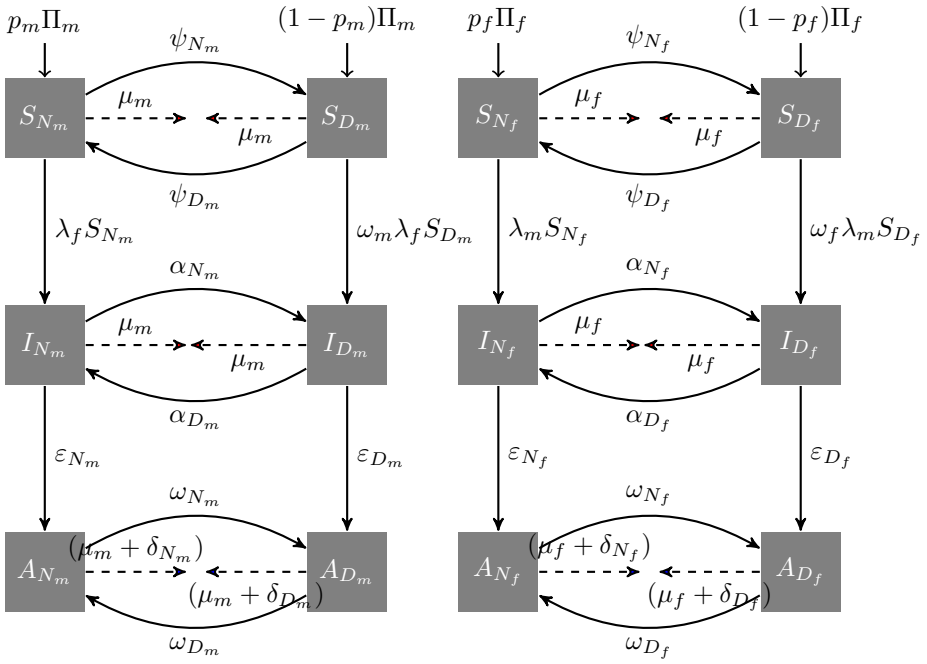

FIGURE 1. Flow diagram for the model (1)

If $N(t) > \frac{\Pi}{\mu}$ then clearly $\dot{N} < 0$, and the solution enters $\mathcal{M}_1$ in finite time or $N(t)$ approaches $\frac{\Pi}{\mu}$ and the infected variables $I_{N_m}, I_{D_m}, A_{N_m}, A_{D_m}, I_{N_f}, I_{D_f}, A_{N_f}, A_{D_f}$ approach 0. For $N(t) \leq \frac{\Pi}{\mu}$, we can write the solution of (2) as

$$\begin{aligned} N(t) &= N_m(t) + N_f(t) \\ &\leq N_m(0)\exp(-\mu_m(t)) + \frac{\Pi_m}{\mu_m}[1-\exp(-\mu_m(t)] \\ &\quad + N_f(0)\exp(-\mu_f(t)) + \frac{\Pi_f}{\mu_f}[1-\exp(-\mu_f(t)] \\ &\leq \frac{\Pi}{\mu} \quad \text{if } N(0) \leq \frac{\Pi}{\mu} \end{aligned}$$

Hence, $N(0) \leq \frac{\Pi}{\mu} \Longrightarrow N(t) \leq \frac{\Pi}{\mu}$.

Thus, the closed set $\mathcal{M}_1$ is positively invariant and attracting. □

The model (1) is mathematically and epidemiologically meaningful and we can proceed to its analysis and simulation.

2.1. **Equilibria and stability analysis.** The model (1) has a disease free equilibrium (DFE) point, E_{0_1}, obtained by taking $I_{N_m} = I_{D_m} = A_{N_m} = A_{D_m} = I_{N_f} = I_{D_f} = A_{N_f} = A_{D_f} = 0$, given by

$$E_{0_1} = (\frac{\Pi_m\theta_m}{\mu_m}, \frac{\Pi_m(1-\theta_m)}{\mu_m}, 0, 0, 0, 0, \frac{\Pi_f\theta_f}{\mu_f}, \frac{\Pi_f(1-\theta_f)}{\mu_f}, 0, 0, 0, 0)$$

where

$$\theta_i = \frac{p_i\mu_i + \psi_{D_i}}{\mu_i + \psi_{N_i} + \psi_{D_i}} \quad i = m, f$$

The endemic equilibria of the model (1), E_{1_1}, where at least one of the infected components is non zero, are given by

$$E_{1_1} = (S^*_{N_m}, S^*_{D_m}, I^*_{N_m}, I^*_{D_m}, A^*_{N_m}, A^*_{D_m}, S^*_{N_f}, S^*_{D_f}, I^*_{N_f}, I^*_{D_f}, A^*_{N_f}, A^*_{D_f})$$

where

$$\begin{aligned}
S^*_{N_m} &= \frac{\Pi_m[J_{3m} + p_m\omega_m\lambda^*_f]}{\Gamma_m}, & S^*_{N_f} &= \frac{\Pi_f[J_{3f} + p_f\omega_f\lambda^*_m]}{\Gamma_f} \\
S^*_{D_m} &= \frac{\Pi_m[J_{4m} + (1-p_m)\lambda^*_f]}{\Gamma_m}, & S^*_{D_f} &= \frac{\Pi_f[J_{4f} + (1-p_f)\lambda^*_m]}{\Gamma_f} \\
I^*_{N_m} &= \frac{\Pi_m[J_{5m}\lambda^{*^2}_f + J_{6m}\lambda^*_f]}{\Omega_{3m}\Gamma_m}, & I^*_{N_f} &= \frac{\Pi_f[J_{5f}\lambda^{*^2}_m + J_{6f}\lambda^*_m]}{\Omega_{3f}\Gamma_f} \\
I^*_{D_m} &= \frac{\Pi_m[J_{7m}\lambda^{*^2}_f + J_{8m}\lambda^*_f]}{\Omega_{3m}\Gamma_m}, & I^*_{D_f} &= \frac{\Pi_f[J_{7f}\lambda^{*^2}_m + J_{8f}\lambda^*_m]}{\Omega_{3f}\Gamma_f} \\
A^*_{N_m} &= \frac{\Pi_m[J_{9m}\lambda^{*^2}_f + J_{10m}\lambda^*_f]}{\Omega_{3m}\Omega_{4m}\Gamma_m}, & A^*_{N_f} &= \frac{\Pi_f[J_{9f}\lambda^{*^2}_m + J_{10f}\lambda^*_m]}{\Omega_{3f}\Omega_{4f}\Gamma_f} \\
A^*_{D_m} &= \frac{\Pi_m[J_{11m}\lambda^{*^2}_f + J_{12m}\lambda^*_f]}{\Omega_{3m}\Omega_{4m}\Gamma_m}, & A^*_{D_f} &= \frac{\Pi_f[J_{11f}\lambda^{*^2}_m + J_{12f}\lambda^*_m]}{\Omega_{3f}\Omega_{4f}\Gamma_f}
\end{aligned}$$

and $\Gamma_m = [\omega_m\lambda^{*^2}_f + J_{1m}\lambda^*_f + J_{2m}]$, $\Gamma_f = [\omega_f\lambda^{*^2}_m + J_{1f}\lambda^*_m + J_{2f}]$. Using the technique by Van den Driesche and Watmough [54], the basic reproduction number, $\mathcal{R}_{0_1}$, is obtained to be $\mathcal{R}_{0_1} = \rho(\mathbf{F^{(1)}V^{-1}}) = \sqrt{R_{0_{1m}}R_{0_{1f}}}$, where

$$\begin{aligned}
R_{0_{1m}} &= \frac{\beta_{mf}(\theta_m M_{1_m} + \omega_m(1-\theta_m)M_{2_m})}{\Omega_{3_m}\Omega_{4_m}}, \\
R_{0_{1f}} &= \frac{\beta_{fm}(\theta_f M_{1_f} + \omega_f(1-\theta_f)M_{2_f})}{\Omega_{3_f}\Omega_{4_f}},
\end{aligned}$$

the matrices for new infections and other transitions, denoted by $F^{(1)}$ and V respectively, are given in Appendix A, and $\Omega_{3_j}, \Omega_{4_j}$, $J_{1j}, ..., J_{17j} (j = m, f)$ and M_{1j}, M_{2j} are given in Appendix B. The forces of infection λ^*_m and λ^*_f are given in terms of R_{0_1} as follows:

$$\lambda^*_f = \frac{\beta_{fm}(J_{16f}\lambda^{*^2}_m + J_{17f}\lambda^*_m)}{J_{13f}\lambda^{*^2}_m + J_{14f}\lambda^*_m + J_{15f}}$$

and λ^*_m is the positive real root of the equation

$$A\lambda^{*^4}_m + B\lambda^{*^3}_m + C\lambda^{*^2}_m + D\lambda^*_m + E = 0 \tag{3}$$

where

$$\begin{aligned}
A &= \beta_{fm}^2 J_{16f}^2 J_{13m} + \beta_{fm} J_{16f} J_{13f} J_{14m} + J_{15m} J_{13f}^2,\\
B &= \beta_{fm}^2 2J_{16f} J_{17f} J_{13m} + \beta_{fm} J_{16f} J_{14f} J_{14m} + \beta_{fm} J_{17f} J_{13f} J_{14m}\\
&\quad +2J_{15m} J_{13f} J_{14f} - \beta_{fm}^2 \beta_{mf} J_{16f}^2 J_{16m} - \beta_{fm}\beta_{mf} J_{17m} J_{16f} J_{13f},\\
C &= \beta_{fm}^2 J_{17f}^2 J_{13m} + \beta_{fm} J_{16f} J_{15f} J_{14m} + \beta_{fm} J_{17f} J_{14f} J_{14m} + J_{15m} J_{14f}^2\\
&\quad +2J_{13f} J_{15m} J_{15f}(1 - \frac{R_{0_1}^2}{2}) - 2\beta_{fm}^2 \beta_{mf} J_{16f} J_{16m} J_{17f}\\
&\quad -\beta_{fm}\beta_{mf} J_{17m} J_{16f} J_{14f},\\
D &= \beta_{fm} J_{17f} J_{15f} J_{14m} + 2J_{14f} J_{15m} J_{15f}(1 - \frac{R_{0_1}^2}{2})\\
&\quad -\beta_{fm}^2 \beta_{mf} J_{17f}^2 J_{16m} - \beta_{fm}\beta_{mf} J_{17m} J_{16f} J_{15f},\\
E &= J_{15m} J_{15f}^2 (1 - R_{0_1}^2),
\end{aligned}$$

and $R_{0_1}^2$ is given by

$$R_{0_1}^2 = \frac{\beta_{mf}\beta_{fm} J_{17f} J_{17m}}{J_{15m} J_{15f}}.$$

Theorem 2.2. *The DFE point, E_{0_1}, of the model (1) is*

1. *locally asymptotically stable if $R_{0_{1m}} < 1$ and $R_{0_{1f}} < 1$ or $R_{0_{1i}} > 1$ and $R_{0_{1j}} < \frac{1}{R_{0_{1i}}}$, $i, j = m, f, (i \neq j)$*
2. *unstable if $R_{0_{1m}} > 1$ and $R_{0_{1f}} > 1$ or $R_{0_{1i}} < 1$ and $R_{0_{1j}} > \frac{1}{R_{0_{1i}}}$, $i, j = m, f (i \neq j)$.*

Proof. We use the Next Generation Operator Method to prove the LAS of the DFE point, E_{0_1}. It is clear that $\mathcal{R}_{0_1} > 0$, hence by theorem [54], the DFE point, E_{0_1}, of the model (1) is LAS if $\mathcal{R}_{0_1} < 1$ and unstable if $\mathcal{R}_{0_1} > 1$. Since $\mathcal{R}_{0_1} = \sqrt{R_{0_{1m}} R_{0_{1f}}}$, the proof follows. □

Now we investigate the possibility of backward bifurcation, that is, the possibility of the DFE and the endemic equilibrium co-existing even for $\mathcal{R}_{0_1} < 1$. From the coefficients of equation (3), we have the following:

1. Clearly $A > 0$. Also $E \geq (\leq) 0 \iff R_{0_1} \leq (\geq) 1$.
2. Consider the case $R_{0_1} < 1 \iff E > 0$. Then, the product of the roots $\frac{E}{A} > 0$ and thus there can be two or four positive roots λ_m^{**} of (3); four positive roots if $B, D < 0$ and $C > 0$ and two positive roots if $B, C > 0$ and $D < 0$ or $B, C < 0$ and $D > 0$ or $C, D > 0$ and $B < 0$. Hence the possibility of backward bifurcation exists in this case.
3. Consider the case $R_{0_1} > 1 \iff E < 0$. Then the product of the roots $\frac{E}{A} < 0$ and thus there can be one or three positive roots λ_m^{**} of (3); one positive root occurs if $B, C, D > 0$ and three positive roots occur if $B, C < 0$ and $D > 0$ or $B, D < 0$ and $C > 0$ or $C, D > 0$ and $B < 0$. In this case there is a possibility of multiple endemic equilibria.

Thus, we have the following theorem:

Theorem 2.3. *The model (1) exhibits backward bifurcation for $R_{0_1} < 1$.*

Since our model (1) exhibits backward bifurcation, the DFE is only LAS and an endemic equilibrium co-exists for $R_{0_1} < 1$. This tells us that it is necessary,

but not sufficient, to bring R_{0_1} below unity to eliminate the disease. Hence, we investigate ways of removing the backward bifurcation from the model. First, we investigate, as a special case, whether the incidence function of the model (1) has any decisive role in causing the bifurcation behavior. This is motivated by the fact that in various HIV models [52], backward bifurcation has been observed to occur only with standard incidence and not with mass action incidence and the presence or absence of standard incidence was crucial for the presence or absence of backward bifurcation. We, therefore, consider the following special case of the model (1).

3. **Special case I: Model with mass action incidence.** In this special case of the model (1), we replace the standard incidence function with mass action incidence. For simplicity, we consider the model with constant male and female populations, ie. $N_m(t) = N_m =$ constant and $N_f(t) = N_f =$ constant. The forces of infection in this case, therefore, reduce to

$$\lambda_{f_m} = \beta_{fm}(I_{N_f} + \psi_{1_f} I_{D_f} + \psi_{2_f} A_{N_f} + \psi_{3_f} A_{D_f}) \tag{4}$$

$$\lambda_{m_m} = \beta_{mf}(I_{N_m} + \psi_{1_m} I_{D_m} + \psi_{2_m} A_{N_m} + \psi_{3_m} A_{D_m}) \tag{5}$$

where β_{fm} and β_{mf} are scaled down by the constants N_f and N_m respectively. The feasible region of this special case is $\mathcal{M}_1$ as in the model (1).

3.1. **Equilibria and stability analysis.** The disease free equilibrium point of the mass action incidence model is the same as E_{0_1}. The endemic equilibria of the this special case, E_{1_2}, are given by

$$E_{1_2} = (S^{**}_{N_m}, S^{**}_{D_m}, I^{**}_{N_m}, I^{**}_{D_m}, A^{**}_{N_m}, A^{**}_{D_m}, S^{**}_{N_f}, S^{**}_{D_f}, I^{**}_{N_f}, I^{**}_{D_f}, A^{**}_{N_f}, A^{**}_{D_f})$$

where

$$S^{**}_{N_m} = \frac{\Pi_m[J_{3m} + p_m\omega_m\lambda^{**}_{f_m}]}{\Gamma_{m_m}}, \qquad S^{**}_{N_f} = \frac{\Pi_f[J_{3f} + p_f\omega_f\lambda^{**}_{m_m}]}{\Gamma_{f_m}}$$

$$S^{**}_{D_m} = \frac{\Pi_m[J_{4m} + (1-p_m)\lambda^{**}_{f_m}]}{\Gamma_{m_m}}, \qquad S^{**}_{D_f} = \frac{\Pi_f[J_{4f} + (1-p_f)\lambda^{**}_{m_m}]}{\Gamma_{f_m}}$$

$$I^{**}_{N_m} = \frac{\Pi_m[J_{5m}\lambda^{**^2}_{f_m} + J_{6m}\lambda^{**}_{f_m}]}{\Omega_{3m}\Gamma_{m_m}}, \qquad I^{**}_{N_f} = \frac{\Pi_f[J_{5f}\lambda^{**^2}_{m_m} + J_{6f}\lambda^{**}_{m_m}]}{\Omega_{3f}\Gamma_{f_m}}$$

$$I^{**}_{D_m} = \frac{\Pi_m[J_{7m}\lambda^{**^2}_{f_m} + J_{8m}\lambda^{**}_{f_m}]}{\Omega_{3m}\Gamma_{m_m}}, \qquad I^{**}_{D_f} = \frac{\Pi_f[J_{7f}\lambda^{**^2}_{m_m} + J_{8f}\lambda^{**}_{m_m}]}{\Omega_{3f}\Gamma_{f_m}}$$

$$A^{**}_{N_m} = \frac{\Pi_m[J_{9m}\lambda^{**^2}_{f_m} + J_{10m}\lambda^{**}_{f_m}]}{\Omega_{3m}\Omega_{4m}\Gamma_{m_m}}, \qquad A^{**}_{N_f} = \frac{\Pi_f[J_{9f}\lambda^{**^2}_{m_m} + J_{10f}\lambda^{**}_{m_m}]}{\Omega_{3f}\Omega_{4f}\Gamma_{f_m}}$$

$$A^{**}_{D_m} = \frac{\Pi_m[J_{11m}\lambda^{**^2}_{f_m} + J_{12m}\lambda^{**}_{f_m}]}{\Omega_{3m}\Omega_{4m}\Gamma_{m_m}}, \qquad A^{**}_{D_f} = \frac{\Pi_f[J_{11f}\lambda^{**^2}_{m_m} + J_{12f}\lambda^{**}_{m_m}]}{\Omega_{3f}\Omega_{4f}\Gamma_{f_m}}$$

and $\Gamma_{m_m} = [\omega_m\lambda^{**^2}_{f_m} + J_{1m}\lambda^{**}_{f_m} + J_{2m}]$, $\Gamma_{f_m} = [\omega_f\lambda^{**^2}_{m_m} + J_{1f}\lambda^{**}_{m_m} + J_{2f}]$. The basic reproduction number, $\mathcal{R}_{0_2}$, is calculated, using [54], to be $\mathcal{R}_{0_2} = \rho(\mathbf{F^{(2)}V^{-1}}) = \sqrt{R_{0_{2m}}R_{0_{2f}}}$, where

$$R_{0_{2m}} = \frac{\Pi_m}{\mu_m}\frac{\beta_{mf}(\theta_m M_{1_m} + \omega_m(1-\theta_m)M_{2_m})}{\Omega_{3_m}\Omega_{4_m}},$$

$$R_{0_{2f}} = \frac{\Pi_f}{\mu_f}\frac{\beta_{fm}(\theta_f M_{1_f} + \omega_f(1-\theta_f)M_{2_f})}{\Omega_{3_f}\Omega_{4_f}},$$

and the matrices $F^{(2)}$ and V are given in Appendix A. This leads to the following theorem:

Theorem 3.1. *The DFE point, E_{0_2}, of (4)and (5) in the model (1) is*

1. *locally asymptotically stable if $R_{0_{2m}} < 1$ and $R_{0_{2f}} < 1$ or $R_{0_{2i}} > 1$ and $R_{0_{2j}} < \frac{1}{R_{0_{2i}}}$, $i, j = m, f, (i \neq j)$*
2. *unstable if $R_{0_{2m}} > 1$ and $R_{0_{2f}} > 1$ or $R_{0_{2i}} < 1$ and $R_{0_{2j}} > \frac{1}{R_{0_{2i}}}$, $i, j = m, f (i \neq j)$.*

Proof. Proof is similar to that for Theorem (2.2). □

The forces of infection $\lambda^{**}_{m_m}$ and $\lambda^{**}_{f_m}$ are given in terms of R_{0_2} as follows:

$$\lambda^{**}_{f_m} = \frac{\beta_{fm}\Pi_f(J_{16f}\lambda^{**^2}_{m_m} + J_{17f}\lambda^{**}_{m_m})}{\Omega_{3f}\Omega_{4f}(\omega_f\lambda^{**^2}_{m_m} + J_{1f}\lambda^{**}_{m_m} + J_{2f})}$$

and $\lambda^{**}_{m_m}$ is the positive real root of the equation

$$A_m\lambda^{**^4}_{m_m} + B_m\lambda^{**^3}_{m_m} + C_m\lambda^{**^2}_{m_m} + D_m\lambda^{**}_{m_m} + E_m = 0 \tag{6}$$

where

$$\begin{aligned}
A_m &= \Omega_{3m}\Omega_{4m}(\Pi_f^2\beta_{fm}^2J_{16f}^2\omega_m + \Pi_f\Omega_{3f}\Omega_{4f}\beta_{fm}J_{16f}\omega_fJ_{1m} + \Omega_{3f}^2\Omega_{4f}^2J_{2m}\omega_f^2)\\
B_m &= \Omega_{3m}\Omega_{4m}[2\Pi_f^2\beta_{fm}^2J_{16f}J_{17f}\omega_m + \Pi_f\Omega_{3f}\Omega_{4f}\beta_{fm}(J_{16f}J_{1f}J_{1m}\\
&\quad + J_{17f}\omega_fJ_{1m}) + 2\Omega_{3f}\Omega_{4f}^2J_{2m}\omega_fJ_{1f}]\\
&\quad -\Pi_f\beta_{fm}\beta_{mf}J_{16f}(\Pi_f\beta_{fm}J_{16f}J_{16m} + \Pi_m\Omega_{3f}\Omega_{4f}J_{17m}\omega_f)\\
C_m &= \Omega_{3m}\Omega_{4m}[\Pi_f^2\beta_{fm}^2J_{17f}^2\omega_m + \Pi_f\Omega_{3f}\Omega_{4f}\beta_{fm}J_{16f}J_{2f}J_{1m}\\
&\quad + \Pi_f\Omega_{3f}\Omega_{4f}\beta_{fm}J_{17f}J_{1f}J_{1m} + \Omega_{3f}^2\Omega_{4f}^2J_{2m}J_{1f}^2]\\
&\quad + 2\Omega_{3m}\Omega_{4m}\Omega_{3f}^2\Omega_{4f}^2\omega_fJ_{2m}J_{2f}(1 - \frac{R_{0_2}^2}{2})\\
&\quad -\Pi_m\Pi_f\beta_{mf}\beta_{fm}(2\beta_{fm}J_{16f}J_{16m}J_{17f} + \Omega_{3f}\Omega_{4f}J_{17m}J_{16f}J_{1f})\\
D_m &= \Omega_{3m}\Omega_{3f}\Omega_{4m}\Omega_{4f}[\Pi_f\beta_{fm}J_{17f}J_{2f}J_{1m} + 2\Omega_{3f}\Omega_{4f}J_{1f}J_{2m}J_{2f}(1 - \frac{R_{0_2}^2}{2})]\\
&\quad -\Pi_m\Pi_f\beta_{mf}\beta_{fm}(\beta_{fm}\Pi_fJ_{17f}^2J_{16m} + \Omega_{3f}\Omega_{4f}J_{17m}J_{16f}J_{2f})\\
E_m &= \Omega_{3m}\Omega_{4m}\Omega_{3f}^2\Omega_{4f}^2J_{2m}J_{2f}^2(1 - R_{0_2}^2)
\end{aligned}$$

since

$$R_{0_2}^2 = \frac{\Pi_m\Pi_f\beta_{mf}\beta_{fm}J_{17f}J_{17m}}{\Omega_{3m}\Omega_{3f}\Omega_{4m}\Omega_{4f}J_{15m}J_{15f}}.$$

Thus we have the following theorem:

Theorem 3.2. *The mass action incidence version of the model (1), described in Special case I, exhibits backward bifurcation for $R_{0_2} < 1$.*

Proof. Since $A_m > 0$, and $E \geq (\leq)0 \iff R_{0_2} \leq (\geq)1$, proof follows from analyzing the positive roots of the equation (6) and is similar to that given for Theorem (2.3). □

4. **Special case II: One susceptible class for each sex.** We have shown that replacing the standard incidence function with mass action incidence in (1) did not remove the possibility of backward bifurcation from the model. We now consider the effect of merging the two susceptible male classes to form one susceptible class, S_m, i.e., $S_{N_m} + S_{D_m} = S_m$. Similarly the two susceptible female classes are merged into one susceptible class, S_f, i.e., $S_{N_f} + S_{D_f} = S_f$. Obviously, $\psi_N = \psi_D = 0$ and $\omega_m = \omega_f = 1$. This case is important because the behavior of susceptible individuals is not monitored clinically and the data with regard to acquisition of, or ongoing recovery from, heavy-drinking habits among susceptible individuals is not easy to measure. The transfer dynamics patterning to this special case are as follows:

$$\begin{aligned}
\dot{S}_m &= \Pi_m - (\mu_m + \lambda_f)S_m \\
\dot{I}_{N_m} &= p_m\lambda_f S_m + \alpha_{D_m} I_{D_m} - Q_{1_m} I_{N_m} \\
\dot{I}_{D_m} &= (1-p_m)\lambda_f S_m + \alpha_{N_m} I_{N_m} - Q_{2_m} I_{D_m} \\
\dot{A}_{N_m} &= \varepsilon_{N_m} I_{N_m} + \omega_{D_m} A_{D_m} - Q_{3_m} A_{N_m} \\
\dot{A}_{D_m} &= \varepsilon_{D_m} I_{D_m} + \omega_{N_m} A_{N_m} - Q_{4_m} A_{D_m} \\
\dot{S}_f &= \Pi_f - (\mu_f + \lambda_m)S_f \\
\dot{I}_{N_f} &= p_f\lambda_m S_f + \alpha_{D_f} I_{D_f} - Q_{1_f} I_{N_f} \\
\dot{I}_{D_f} &= (1-p_f)\lambda_m S_f + \alpha_{N_f} I_{N_f} - Q_{2_f} I_{D_f} \\
\dot{A}_{N_f} &= \varepsilon_{N_f} I_{N_f} + \omega_{D_f} A_{D_f} - Q_{3_f} A_{N_f} \\
\dot{A}_{D_f} &= \varepsilon_{D_f} I_{D_f} + \omega_{N_f} A_{N_f} - Q_{4_f} A_{D_f}
\end{aligned} \tag{7}$$

where

$$N_j = S_j + I_{N_j} + I_{D_j} + A_{N_j} + A_{D_j}, \quad j = m, f.$$

Lemma 4.1. *The closed set*

$$\begin{aligned}
\mathcal{M}_2 = \{&(S_m, I_{N_m}, I_{D_m}, A_{N_m}, A_{D_m}, S_f, I_{N_f}, I_{D_f}, A_{N_f}, A_{D_f}) \in R_+^{10} \\
&: S_m + I_{N_m} + I_{D_m} + A_{N_m} + A_{D_m} + S_f + I_{N_f} + I_{D_f} + A_{N_f} + A_{D_f} \leq \frac{\Pi}{\mu}\}
\end{aligned}$$

where $\Pi = \Pi_m + \Pi_f$ *and* $\mu = min\{\mu_m, \mu_f\}$ *is positively invariant and attracting with respect to the model (7).*

Proof. Proof is similar to that for Lemma (2.1). □

4.1. **Equilibria and stability analysis.** The model (7) has a disease free equilibrium point, E_{0_3}, given by

$$E_{0_3} = (\frac{\Pi_m}{\mu_m}, 0, 0, 0, 0, \frac{\Pi_f}{\mu_f}, 0, 0, 0, 0)$$

and a unique endemic equilibrium point E_{1_3}, given by

$$E_{1_3} = (S_m^{***}, I_{N_m}^{***}, I_{D_m}^{***}, A_{N_m}^{***}, A_{D_m}^{***}, S_f^{***}, I_{N_f}^{***}, I_{D_f}^{***}, A_{N_f}^{***}, A_{D_f}^{***})$$

where $(S_m^{***}, I_{N_m}^{***}, I_{D_m}^{***}, A_{N_m}^{***}, A_{D_m}^{***}, S_f^{***}, I_{N_f}^{***}, I_{D_f}^{***}, A_{N_f}^{***}, A_{D_f}^{***})$ are given in Appendix C. Computations, using the Next Generation Operator method [54], give the basic reproduction number, $\mathcal{R}_{0_3}$, for the model (7) as $\mathcal{R}_{0_3} = \rho(\mathbf{F^{(3)}V^{-1}}) = \sqrt{R_{0_{3m}} R_{0_{3f}}}$ where

$$R_{0_{3m}} = \frac{\beta_{mf}(p_m M_{1_m} + (1-p_m)M_{2_m})}{\Omega_{3_m}\Omega_{4_m}}, \quad R_{0_{3f}} = \frac{\beta_{fm}(p_f M_{1_f} + (1-p_f)M_{2_f})}{\Omega_{3_f}\Omega_{4_f}}$$

and the matrix $F^{(3)}$ of new infections is given in Appendix A along with the matrix of other infections V. In terms of the reproduction number, $\mathcal{R}_{0_3}$, the forces of infection can be written as:

$$\lambda_f^{***} = \frac{\Omega_{3_m}\Omega_{4_m}\Omega_{3_f}\Omega_{4_f}(\mathcal{R}_{0_3}^2-1)}{\Omega_{3_f}\Omega_{4_f}\Omega_{7_m}+\beta_{mf}\Omega_{7_f}\Omega_{8_m}}, \quad \lambda_m^{***} = \frac{\Omega_{3_m}\Omega_{4_m}\Omega_{3_f}\Omega_{4_f}(\mathcal{R}_{0_3}^2-1)}{\Omega_{3_m}\Omega_{4_m}\Omega_{7_f}+\beta_{fm}\Omega_{7_m}\Omega_{8_f}}$$

where, $\Omega_{1_j}, ..., \Omega_{8_j}$ $(j = m, f)$ are defined in Appendix B. In this special case also, we can cite the following theorem as in the case of the original model.

Theorem 4.2. *The DFE point, E_{0_3}, of the model (7) is*

1. *locally asymptotically stable if $R_{0_{3m}} < 1$ and $R_{0_{3f}} < 1$ or $R_{0_{3i}} > 1$ and $R_{0_{3j}} < \frac{1}{R_{0_{3i}}}$, $i, j = m, f, (i \neq j)$*
2. *unstable if $R_{0_{3m}} > 1$ and $R_{0_{3f}} > 1$ or $R_{0_{3i}} < 1$ and $R_{0_{3j}} > \frac{1}{R_{0_{3i}}}$, $i, j = m, f (i \neq j)$.*

Proof. Proof is similar to that for Theorem (2.2). □

Now we see from the equations for λ_f^{***} and λ_m^{***}, that the positive endemic equilibrium E_{1_3} exists if only if $\mathcal{R}_{0_3} > 1$. Hence, in this case there is no possibility of backward bifurcation. Thus, we have the following theorem:

Theorem 4.3. *The Special case II of the model (1), given in (7), does not undergo backward bifurcation for $\mathcal{R}_{0_3} < 1$.*

5. **Numerical simulation.** The numerical simulation of the model (1) poses practical difficulties as it includes some parameters whose values have not been estimated clinically or experimentally. As in [20], we restrict the simulation to Botswana, for which some of our model parameters are known. The unit of time assumed in the simulations is one year.

5.1. **Parameter estimates.** A population based study [65] conducted in Botswana in 2004, among 654 females and 614 males, on alcohol and high-risk sexual behaviors, found 39% of male and 25% of female respondents to have satisfied our criteria for heavy-drinking [2]. But the Botswana AIDS Impact Survey III (BAIS III) [8], which is the third national sexual behavioral population level survey, conducted in 2008, found the distribution of males and females who have ever taken alcoholic drink to be 48.9% and 27.7% respectively, but the levels of their drinking are not specified. Based on the above studies, we assume a range of $0.51 - 0.61$ for p_m and $0.72 - 0.75$ for p_f. The Botswana Demographic Survey 2006 [9] estimates the life expectancy at birth for men and women to be 48.8 and 60 years respectively. Considering 15 - 49 to be the reproductive age, the average duration of sexual activity for men and women are hence taken as 34.8 and 35 years respectively, which yield baseline values $\mu_m = \mu_f = 0.029$. But we will use the range $\mu_m = \mu_f = [0.025, 0.029]$ in sensitivity analysis.

The mean progression time from HIV infection to AIDS gives a variety of estimates, among which the difference between developed and developing countries is noteworthy. Based on UNAIDS estimate, we assume a baseline value of 7.5 years for moderate male infectives and 8.5 years for moderate female infectives as mean progression times, but we will investigate the sensitivity of our model with regard

[2]The criteria for levels of drinking adopted in [65] is slightly different from what we have assumed. But we have calculated from it the proportion of moderate and heavy-drinkers as per our criteria

to progression times, as we vary the progression time in the range 7.5 - 8.5 for moderate male infectives and 8.5 to 9.5 for moderate female infectives. The corresponding mean progression times for heavy drinking infectives are faster than for moderate infectives, since alcohol is known to hasten the progression of the infection [22][25] and can negatively affect treatment seeking behavior [17] of the infected, although no conclusive clinical data is available to this effect. Hence, we assume the UNAIDS estimates for faster progression to be applicable to heavy drinking infectives and accordingly take a baseline value of 6.1 years for males and 6.9 years for females, but we will investigate the sensitivity of our model in the range [6.1,7.1] years for heavy-drinking male infectives and [6.9,7.9] years for heavy-drinking female infectives. Hence, the range of values for the progression rates per year are $\varepsilon_{N_m} = [0.118, 0.133]$, $\varepsilon_{N_f} = [0.105, 0.118]$, $\varepsilon_{D_m} = [0.141, 0.164]$, and $\varepsilon_{D_f} = [0.127, 0.145]$.

The probability of HIV transmission per heterosexual contact has been studied extensively [41][56]. Based on the systematic review and meta-analysis by Boily et.al. [41], we take the baseline values of η_{mf} and η_{fm} to be 0.00143 and 0.00164 respectively, but our sensitivity analysis for these parameters will be conducted in the range $\eta_{mf} = [0.00124, 0.00143]$ and $\eta_{fm} = [0.00143, 0.00164]$.

Todd et.al. [33] compared data from four longitudinal studies conducted in three sub-Sahara African countries, namely, Uganda, Zimbabwe and South Africa, about reported number of sexual partners and found that the mean number of reported sexual partners in the past 12 months for non-virgin men, across the different sites and between the different rounds, ranged from 1.19 to 1.90 and for non-virgin women, the overall average number of reported partners was lower than the same for males and varied between 0.82 and 1.09. Based on the cited study [33], we take $c_m = [1.19, 1.9]$ and $c_f = [0.82, 1.09]$. The average number of sexual contacts per partner per year, in many contexts, is assumed to be a decreasing function of the number of partners per year. Sub-Saharan studies [56] have estimated a mean of 8.3 coital acts and 9.7 coital acts per month for HIV positive men and women respectively in heterosexual monogamous HIV-discordant sexual contacts. Hence, as in [31], a simple function of the partner acquisition rate per year that gives the sexual contact rates in [56] and decreases to 1 contact per partner as the number of partners increases is taken to be $n = n(c) = 100c^{-\rho} + 1$ for males and $n = n(c) = 116c^{-\rho} + 1$ for females, where c is the number of partners per year and n is the number of sexual contacts per partner and ρ is a parameter which controls how fast the function decreases. We will look for sensitivity of the model for the parameters n_m and n_f in the range $n_m = [62, 99]$ and $n_f = [108, 143]$.

Studies have found heavy alcohol consumption of males and females to be associated with risky sex outcomes including multiple partners, increased number of sexual contacts, unprotected sex, exchanging sex for money or commodities, increased vulnerability to STIs, etc., which in turn will increase chances of HIV infection. Studies among HIV positive individuals in sub-Sahara Africa have shown that drinking alcohol before sex by the female partner or male partner, or by both partners, increased the proportion and number of subsequent unprotected sex events. Kiene et.al. [67], in their daily phone interview study in South Africa for 42 days, found that 24 HIV positive male participants reported 1299 sex events and 58 HIV positive females reported 3628 sex events during the period of study, and more than half of the events in both cases were with partners who were HIV negative or whose HIV status was unknown. Our present simulation, in calculating the modification

parameters for the rate of transmission for heavy-drinking infectives and PLWA, namely ψ_{i_j}, $(i = 1, 3\ j = m, f)$, considers only the factors of increased number of partners and number of sexual events per partner for the heavy-drinking infected classes. We assume an average three-times increase in the number of sexual partners per year for heavy drinking male infectives, compared to the average baseline values in the range $c_m = [1.19, 1.9]$ for moderate infectives. With regard to the number of contacts per partner, we consider the following two cases: The first case is one in which the number of contacts per partner reduces as per the formula $n = n(c) = 100c^{-\rho} + 1$ (assuming $\rho = 1$). The second situation is one in which the number of contacts per partner does not decrease (that is, $\rho = 0$). Based on computation of the rate of transmission in both cases, we investigate the sensitivity of the model to values of ψ_{1m} in the range $[1.06, 3]$. Similarly we assume an average two-times increase in the number of sexual partners per year for heavy-drinking female infectives and, in line with a similar argument given above, we assume ψ_{1f} in the range $[1.04, 2]$. The number of partners per year for moderate PLWA, both male and female, is assumed to be the same as that in the moderate infective class, but we compare the possibility of the number of contacts per partner per year reducing from those of the moderate infectives to half with the number of contacts not reducing, and assume $\psi_{2m} = [0.51, 1]$ and $\psi_{2f} = [0.52, 1]$. For heavy-drinking PLWA, we assume the number of partners per year to be equal to that for heavy-drinking infectives, but in this case we assume a decrease in the number of contacts per partner, varying ρ, thus assuming $\psi_{3m} = [0.91, 1.08]$ and $\psi_{3f} = [0.97, 1.06]$.

The heavy drinking susceptibles have a higher chance of acquiring partners. Assuming an average of 2 (1.5) fold increase in the number of partners for moderate male (female) infectives per year among heavy-drinking susceptible females (males) than what they have among moderate susceptibles, and contrasting the cases where sexual contacts are decreasing and where they are not decreasing, we assume $\omega_m = [1.04, 2]$ and $\omega_f = [1.03, 1.5]$.

We assume the average duration of moderate male and female PLWA in the AIDS class to be 2-3 years. Hence, we take $\delta_{Nm} = \delta_{Nf} = [0.33, 0.5]$. Studies [32], among HIV infected men and women with identified alcohol problems, have shown that on days in which the participants consumed alcohol, there was almost nine times higher odds of medication non-adherence, with each drink increasing the odds by 20%. Due to possible immune-suppression and treatment non-adherence, we assume a faster progression for heavy-drinking males and females in the range 1.5-2.5 years, taking $\delta_{Dm} = \delta_{Df} = [0.4, 0.67]$.

Since parameter values for transfer rate from moderate classes to heavy drinking classes are not available, we assume a 0 - 0.2 and 0 - 0.1 one year transition probabilities for susceptible males and females respectively which yield, using the formula $Pr(event) = 1 - exp(-rate \times time)$, $\psi_{N_m} = [0, 0.22]$ and $\psi_{N_f} = [0, 0.11]$. Corresponding transition probabilities per year for moderate male and female infectives are assumed to be 0 - 0.3 and 0 - 0.2 respectively, which give $\alpha_{N_m} = [0, 0.36]$ and $\alpha_{N_f} = [0, 0.22]$. Similarly, we take transition probabilities per year for moderate male and female PLWA to be 0 - 0.1 and 0 - 0.05 respectively, which give $\omega_{N_m} = [0, 0.11]$ and $\omega_{N_f} = [0, 0.05]$. Regarding the ongoing recovery of heavy-drinking males and females to corresponding moderate classes, we make the following assumptions: BAIS III [8] estimates the percentage of males and females who reported that they do not take alcoholic drink anymore as 6.8% and 6.1% respectively. Based on this, we make a rough estimate for transition probabilities per

year of heavy-drinking susceptible males and females to be 0.068 - 0.1 and 0.061 - 0.15 respectively, to get $\psi_{D_m} = [0.07, 0.11]$ and $\psi_{D_f} = [0.06, 0.16]$. Study by Kip and Kubanji [17] among a sample of 158 HIV infected alcohol drinking individuals, most of whom were on ART, in Botswana found 43% of the respondents saying they were extremely confident of reducing their alcohol use. Based on this we take a 0 - 0.43 probability of male and female PLWA reducing their alcohol use, thus getting $\omega_{D_m} = \omega_{D_f} = [0, 0.56]$. Since most of the heavy-drinking infective males and females are not on ART, we take a reduced transition probability of 0 - 0.3 in both cases, thus getting $\alpha_{D_m} = \alpha_{D_f} = [0, 0.36]$. We assume all our parameter values to be uniformly distributed and all the assumed parameter ranges along with mean and variance are given in Tables 1 and 2.

5.2. **Sensitivity analysis.** Using the range of parameter values given in Tables 1 and 2, we have investigated the uncertainties in the model parameters and their sensitivity using Latin Hypercube sampling techniques, with 1000 samples. Figure 2 shows the tornado chart of Partial Rank Correlation Coefficient (PRCC) for various parameters relative to one of our output variables, R_{0_1}. We notice that, for the assumed range of values, the parameters $\psi_{1_m}, c_m, n_m, \omega_m, c_f, \psi_{N_m}, n_f, \psi_{1_f}$, $\eta_{fm}, \alpha_{N_m}, \omega_f, \eta_{mf}, \psi_{N_f}$, and α_{N_f} are highly positively correlated to R_{0_1}. Hence extreme care should be taken in the estimation of the values of these parameters as a slight change in any of these parameters can bring major variations in the estimated values of R_{0_1}. We specially note that among the parameters capturing acquisition of or recovery from heavy-drinking habits and modification parameters, ψ_{1_m}, ψ_{1_f}, α_{N_m} and α_{N_f} are highly positively and α_{D_m} and α_{D_f} are highly negatively correlated to R_{0_1}. This shows that the male and female heavy-drinking infective classes have the potential to contribute decisively to the spread or reduction of the epidemic. This is in agreement with observations [67] as these individuals are found to be more promiscuous and more prone to sexual-risk taking than the heavy-drinking PLWA, since the latter are more likely to quit drinking [17]. Hence to reduce the number of infections, our model suggests that intervention strategies aimed at reducing the level of heavy-drinking among men and women infectives would yield better results than those programmes which target PLWA. However an intervention programme which promotes ongoing recovery from heavy-drinking, both among PLWA and HIV infectives, would yield even bigger reduction in the number of infections.

5.3. **Results.** With regard to the simulations shown in Figures 3 - 11, the varying parameters are indicated in each figure and the other parameter values, which are kept fixed, are given in Table 3. The initial values assumed for all our simulations are as follows: $S_{N_m}(0) = 1,53,080$; $S_{D_m}(0) = 97,870$; $I_{N_m}(0) = 115$; $I_{D_m}(0) = 74$; $A_{N_m}(0) = 8$; $A_{D_m}(0) = 5$; $S_{N_f}(0) = 2,07,986$; $S_{D_f}(0) = 69,329$; $I_{N_f}(0) = 131$; $I_{D_f}(0) = 44$; $A_{N_f}(0) = 4$; $A_{D_f}(0) = 2$.

Figures 3 and 4 give examples of contour plots for R_{0_1} relative to various parameters. Figure 3 shows a decline in the basic reproduction number for increasing ongoing recovery parameters for HIV infected males and females, α_{D_m} and α_{D_f}. Reduction in R_{0_1} for increasing ongoing recovery parameters for the heavy-drinking MLWA and FLWA, ω_{D_m} and ω_{D_f}, can be similarly observed. Thus, ongoing recovery of HIV/AIDS infected males and females from heavy-drinking would reduce the HIV prevalence.

Figure 4 captures the increase in R_{01} for increasing values of the susceptibility modification parameters for heavy-drinking susceptibles, ω_m and ω_f. Similar increase in R_{01} is observed for increasing c_m and c_f, n_m and n_f, ω_{N_m} and α_{N_m}, ω_{N_f} and α_{N_f}, etc.

Figures 5 - 11 are examples showing the severity of the impact of heavy alcohol consumption of both males and females on the HIV epidemic. Using the epidemiological software SPECTRUM [73], we have been able to assess the impact of heavy alcohol consumption on the epidemic and to project the results to the year 2012. Figures 5 - 8 give simulation results for the cases when $c_m = 1.19$ and $c_f = 0.82$ and when $c_m = 1.9$ and $c_f = 1.09$, that is, on average, moderate infective males have 1.19 versus 1.9 female partners among moderate susceptibles and moderate infective females have 1.09 versus 0.82 male partners among moderate susceptibles. The value of R_{0_1} for $c_m = 1.19$ and $c_f = 0.82$ is 1.7991 ($R_{0_{1m}} = 2.1937, R_{0_{1f}} = 1.4755$) whereas for $c_m = 1.9$ and $c_f = 1.09$, it is 2.6210 ($R_{0_{1m}} = 3.5025, R_{0_{1f}} = 1.9613$). Figure 5 gives results for the total population of HIV infected individuals as time increases. We see that when $c_m = 1.19$ and $c_f = 0.82$ the male and female HIV infected populations are almost the same reaching a peak of 12,774 infectives for females and 12,214 infectives for males, whereas when the parameter c_m is increased to 1.9 and c_f is increased to 1.09, the female HIV population increases to a peak of 156,771 infectives before declining while the male population reaches a peak of 140,638 infectives. Figures 6 - 8 show that if infectives were not promiscuous with regard to sexual partners, ie. $c_m = 1.19$ and $c_f = 0.82$, then the annual number of AIDS deaths, the number of infected individuals in need of ART and the number of TB cases would have been much lower, than, which most probably is the current scenario, with $c_m = 1.9$ and $c_f = 1.09$. The decline in the number of AIDS deaths for both men and women for $c_m = 1.19$ and $c_f = 0.82$ would have been approximately one twelfth of the current level (Figure 6). The number of women in need of ART, if the parameters c_m and c_f were $c_m = 1.19$ and $c_f = 0.82$, would have been one eleventh of the current level, and for men, those in need of ART would have been one tenth of the current level (Figure 7). Similarly, the number of TB cases would have been significantly lower if each infective had maintained less sexual partners only (Figure 8).

Figures 9 and 10 give simulation results with the number of sexual partners fixed at the current level, ie. $c_m = 1.9$ and $c_f = 1.09$, but varying the ongoing recovery parameters for males and females at the infective stage, α_{D_m} and α_{D_f}, with all the other parameters fixed as in the Figures 5 - 8. Figure 9 shows that the number of infected individuals for each sex is higher when the ongoing recovery rates are zero than when the same rates are non-zero. Comparing the cases $\alpha_{D_m} = \alpha_{D_f} = 0$ and $\alpha_{D_m} = \alpha_{D_f} = 0.36$, we see that the decline in the total number of infected among the female population is approximately 35%. Similarly among the male population also the decline is more than 30%. The value of R_{0_1} for $\alpha_{D_m} = \alpha_{D_f} = 0.36$ is 2.5059 ($R_{0_{1m}} = 3.2869, R_{0_{1f}} = 1.9104$) whereas for $\alpha_{D_m} = \alpha_{D_f} = 0$, it is 2.8505 ($R_{0_{1m}} = 3.9161, R_{0_{1f}} = 2.0749$). Figure 10 shows that there would be significant benefits if infected individuals began recovery from heavy alcohol consumption, specifically, there would be fewer new HIV infections. Figure 11 gives simulation results for varying the differential susceptibility parameters of the susceptible heavy-drinking males and females, ω_m and ω_f, with all the other parameters fixed as given in Table 3. If $\omega_m = 1.04$ and $\omega_f = 1.03$, that is if the heavy-drinkers are almost similar in susceptibility to moderate drinkers, the HIV male population is reduced

to one fifth and female population is reduced to one fourth, approximately, than it would have been if $\omega_m = 2$ and $\omega_f = 1.5$. The value of R_{0_1} for $\omega_m = 1.04$ and $\omega_f = 1.03$ is 2.2379 ($R_{0_{1m}} = 2.7606, R_{0_{1f}} = 1.8142$) whereas for $\omega_m = 2$ and $\omega_f = 1.5$, it is 2.9871 ($R_{0_{1m}} = 4.2443, R_{0_{1f}} = 2.1023$).

6. **Discussion.** We have formulated a deterministic two-sex model that considers the impact of heavy alcohol consumption among males and females on the spread of HIV/AIDS. This model is particularly relevant to sub-Sahara Africa where heavy alcohol consumption among young people is on the increase, age of onset of alcohol use is on the decrease and HIV/AIDS is highly prevalent posing a serious threat to public health [3][34][37][38][81]. The relevance of this study lies in its focus on gender differences that exist both in alcohol drinking patterns and the resulting high risk sexual behaviors and also HIV incidence and prevalence.

Our model explores the benefits of ongoing recovery among infectives (Figure 3) and the corresponding decline in the number of HIV population, new HIV infections, etc. (Figures 9, 10). This underlines the need for education and counseling interventions to initiate ongoing recovery among heavy-drinkers and also to prevent moderate infectives from acquiring heavy-drinking habits. Sensitivity analysis for our model parameters, for the assumed range of values given in Tables 1 and 2, shows that the parameters $\psi_{1_m}, c_m, n_m, \omega_m, c_f, \psi_{N_m}, n_f, \psi_{1_f}, \eta_{fm}, \alpha_{N_m}, \omega_f, \eta_{mf}, \psi_{N_f}$, and α_{N_f} are highly positively correlated and sensitive to R_{0_1} (Figure 2). Hence we recommend sample surveys and clinical investigations for the accurate statistical estimation of these parameters, as small changes in these parameters can cause an over estimation or under estimation of the parameter R_{0_1}. Our model also quantifies (Figures 5 - 8) the severity of the epidemic in terms of multiple and concurrent partners, behavioral patters common among both males and females in Southern Africa [33][72], where alcohol serving venues are known to serve as meeting places for potential sexual partners [62]. This highlights the need for disseminating strong messages of responsible drinking and faithfulness to a single partner along with the messages of condom use, circumcision and other intervention strategies.

Our model also points to the economic burden the epidemic has brought to governments and the public. Botswana is the first African country to implement a full-scale national antiretroviral (ARV) program and by mid-2008 over 100,000 people have been put on life-saving ARV therapy across the nation. The program is named 'MASA', the local word for 'new dawn' [45]. As per the assumed parameter values given in Table 3, our simulation shows that in 2005 alone an approximate 33000 male ART cases and 37000 female ART cases could have been averted in Botswana if the infected had chosen to avoid promiscuity and remained faithful to single partners. As per the cost analysis [69] of 'MASA' programme in 2005, the total cost of treating 1000 patients per year was US $ 1m, half of which was spent on drugs and the remaining half spent on human resources, infrastructure, education campaigns, etc. Hence, according to our model, education campaigns could have saved about US $ 70m in 2005 alone. Also ongoing recovery of male and female heavy-drinking infectives by 30% would have decreased the number of female infected in need of ART by 16000 and the male infected in need of ART by 10000, resulting in a reduction in expenses amounting to over US $ 26m. Educational campaigns and interventions for ongoing recovery among the infected and among the general public would, therefore, reduce the economic burden at the macro-level.

Comparing the impact of the HIV related heavy-drinking risks on the genders, all our simulations show that the female population is more at risk than the males. The female population has more HIV cases, higher HIV prevalence, more individuals in need of ART and more active TB cases. Taken in a hetero-sexual scenario, this is because it is the males who are engaging more in heavy-drinking and consequent risky sex behaviors than the females. This is clear from the observation that the male component in R_{0_1}, namely $R_{0_{1_m}}$, is higher than the female component, $R_{0_{1_f}}$, in all simulations. Thus the female population is forced to suffer the HIV related burden of the heavy-drinking risks engaged by males. It is known that women and girls continue to be affected disproportionately by HIV in sub-Saharan Africa [75]. Our simulations point to the heavy-drinking pattern prevalent among the male population to be a critical factor for this scenario. This is also substantiated by the sensitivity analysis in which we observe that the male parameters ψ_{1_m}, c_m, n_m and ω_m are the most sensitive to R_{0_1}, and more sensitive than any of the female parameters. Infection of women has added risks in countries where Prevention of Mother To Child Transmission (PMTCT) is not yet a success, since pregnant women remain a major vulnerable group with higher incidence rate, and ultimately more infections among women will cause more infections among the new-born babies as well. This underlines the urgent need of ongoing recovery programmes to target the male population.

We have observed the phenomenon of backward bifurcation, where the stable DFE coexists with the stable endemic equilibrium, in our model (1). This tells us that bringing $R_{0_1} < 1$ is not sufficient to eliminate the disease. We have also seen that replacing the standard incidence function with mass action incidence could not remove the backward bifurcation in this model, but the phenomenon is removed by merging the two susceptible classes for each sex to a single susceptible class. This suggests that in similar multi-group models choice of the incidence function is not decisive in the backward bifurcation phenomenon, in contrast to what was observed in many HIV models [52] exhibiting backward bifurcation. However, further studies are needed in this area to establish and characterize this observation.

The drawback for our study is the unavailability of accurate parameter values necessary for the calibration of the model. Hence our findings should be understood in the background of this limitation. But we have made maximum effort to make use of the available data and also to assume realistic values guided by a sensitivity analysis for the parameters where accurate data is not available. With regard to the impact of heavy-drinking on HIV transmission, we did not consider the factors of unprotected sex or inconsistent use of condoms and STD prevalence which may be higher among the heavy-drinking infectives and PLWA, than among the moderate ones, and which in turn increase the HIV transmission probability.

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Appendix A. The matrix for new infections of model (1), $F^{(1)}$, is given by:

$$\mathbf{F^{(1)}} = \begin{bmatrix} \mathbf{0} & \mathbf{F_{12}^{(1)}} \\ \mathbf{F_{21}^{(1)}} & \mathbf{0} \end{bmatrix}$$

where

$$\mathbf{F_{12}^{(1)}} = \begin{bmatrix} \rho_1\beta_{fm}\theta_m & \rho_1\beta_{fm}\psi_{1_f}\theta_m & \rho_1\beta_{fm}\psi_{2_f}\theta_m & \rho_1\beta_{fm}\psi_{3_f}\theta_m \\ \rho_1\beta_{fm}\omega_m\theta'_m & \rho_1\beta_{fm}\omega_m\psi_{1_f}\theta'_m & \rho_1\beta_{fm}\omega_m\psi_{2_f}\theta'_m & \rho_1\beta_{fm}\omega_m\psi_{3_f}\theta'_m \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}$$

$$\mathbf{F_{21}^{(1)}} = \begin{bmatrix} \rho_2\theta_f\beta_{mf} & \rho_2\theta_f\beta_{mf}\psi_{1_m} & \rho_2\theta_f\beta_{mf}\psi_{2_m} & \rho_2\theta_f\beta_{mf}\psi_{3_m} \\ \omega_f\rho_2\theta'_f\beta_{mf} & \omega_f\rho_2\theta'_f\beta_{mf}\psi_{1_m} & \omega_f\rho_2\theta'_f\beta_{mf}\psi_{2_m} & \omega_f\rho_2\theta'_f\beta_{mf}\psi_{3_m} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}$$

The matrix for new infections of the special case I (given in 4 and 5) of the model (1), $F^{(2)}$, is given by:

$$\mathbf{F^{(2)}} = \begin{bmatrix} \mathbf{0} & \mathbf{F_{12}^{(2)}} \\ \mathbf{F_{21}^{(2)}} & \mathbf{0} \end{bmatrix}$$

where

$$\mathbf{F_{12}^{(2)}} = \begin{bmatrix} \rho_3\beta_{fm}\theta_m & \rho_3\beta_{fm}\psi_{1_f}\theta_m & \rho_3\beta_{fm}\psi_{2_f}\theta_m & \rho_3\beta_{fm}\psi_{3_f}\theta_m \\ \rho_3\beta_{fm}\omega_m\theta'_m & \rho_3\beta_{fm}\omega_m\psi_{1_f}\theta'_m & \rho_3\beta_{fm}\omega_m\psi_{2_f}\theta'_m & \rho_3\beta_{fm}\omega_m\psi_{3_f}\theta'_m \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}$$

$$\mathbf{F_{21}^{(2)}} = \begin{bmatrix} \rho_4\theta_f\beta_{mf} & \rho_4\theta_f\beta_{mf}\psi_{1_m} & \rho_4\theta_f\beta_{mf}\psi_{2_m} & \rho_4\theta_f\beta_{mf}\psi_{3_m} \\ \omega_f\rho_4\theta'_f\beta_{mf} & \omega_f\rho_4\theta'_f\beta_{mf}\psi_{1_m} & \omega_f\rho_4\theta'_f\beta_{mf}\psi_{2_m} & \omega_f\rho_4\theta'_f\beta_{mf}\psi_{3_m} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}$$

The matrix for new infections of the special case II (model 7) of the model (1), $\mathbf{F^{(3)}}$, is given by

$$\mathbf{F^{(3)}} = \begin{bmatrix} \mathbf{0} & \mathbf{F_{12}^{(3)}} \\ \mathbf{F_{21}^{(3)}} & \mathbf{0} \end{bmatrix}$$

where

$$\mathbf{F_{12}^{(3)}} = \begin{bmatrix} \rho_1 p_m\beta_{fm} & \rho_1 p_m\beta_{fm}\psi_{1_f} & \rho_1 p_m\beta_{fm}\psi_{2_f} & \rho_1 p_m\beta_{fm}\psi_{3_f} \\ \rho_1 p'_m\beta_{fm} & \rho_1 p'_m\beta_{fm}\psi_{1_f} & \rho_1 p'_m\beta_{fm}\psi_{2_f} & \rho_1 p'_m\beta_{fm}\psi_{3_f} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}$$

$$\mathbf{F_{21}^{(3)}} = \begin{bmatrix} \rho_2 p_f\beta_{mf} & \rho_2 p_f\beta_{mf}\psi_{1_m} & \rho_2 p_f\beta_{mf}\psi_{2_m} & \rho_2 p_f\beta_{mf}\psi_{3_m} \\ \rho_2 p'_f\beta_{mf} & \rho_2 p'_f\beta_{mf}\psi_{1_m} & \rho_2 p'_f\beta_{mf}\psi_{2_m} & \rho_2 p'_f\beta_{mf}\psi_{3_m} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}$$

The matrix for other infections, V, for the models (1), (7) and (4, 5) is given by

$$\mathbf{V} = \begin{bmatrix} \mathbf{V_{11}} & \mathbf{0} \\ \mathbf{0} & \mathbf{V_{22}} \end{bmatrix}$$

where

$$\mathbf{V_{11}} = \begin{bmatrix} Q_{1_m} & -\alpha_{D_m} & 0 & 0 \\ -\alpha_{N_m} & Q_{2_m} & 0 & 0 \\ -\varepsilon_{N_m} & 0 & Q_{3_m} & -\omega_{D_m} \\ 0 & -\varepsilon_{D_m} & -\omega_{N_m} & Q_{4_m} \end{bmatrix}$$

$$\mathbf{V_{22}} = \begin{bmatrix} Q_{1_f} & -\alpha_{D_f} & 0 & 0 \\ -\alpha_{N_f} & Q_{2_f} & 0 & 0 \\ -\varepsilon_{N_f} & 0 & Q_{3_f} & -\omega_{D_f} \\ 0 & -\varepsilon_{D_f} & -\omega_{N_f} & Q_{4_f} \end{bmatrix}$$

and

$$\begin{array}{rclllll} \mathbf{0} & = & [0]_{4\times 4}, & \rho_1 = \dfrac{\Pi_m \mu_f}{\Pi_f \mu_m}, & \rho_2 = \dfrac{\Pi_f \mu_m}{\Pi_m \mu_f}, & \rho_3 = \dfrac{\Pi_m}{\mu_m}, & \rho_4 = \dfrac{\Pi_f}{\mu_f} \\ \theta'_m & = & 1-\theta_m, & \theta'_f = 1-\theta_f, & p'_m = 1-p_m, & p'_f = 1-p_f & \end{array}$$

Appendix B. For $j = m, f$

$$\begin{aligned} M_{11_j} &= Q_{2_j}\Omega_{4_j} \\ M_{12_j} &= \alpha_{N_j}\Omega_{4_j} \\ M_{13_j} &= Q_{2_j}Q_{4_j}\varepsilon_{N_j} + \varepsilon_{D_j}\omega_{D_j}\alpha_{N_j} \\ M_{14_j} &= Q_{2_j}\varepsilon_{N_j}\omega_{N_j} + Q_{3_j}\varepsilon_{D_j}\alpha_{N_j} \\ M_{21_j} &= \alpha_{D_j}\Omega_{4_j} \\ M_{22_j} &= Q_{1_j}\Omega_{4_j} \\ M_{23_j} &= Q_{4_j}\varepsilon_{N_j}\alpha_{D_j} + Q_{1_j}\varepsilon_{D_j}\omega_{D_j} \\ M_{24_j} &= \alpha_{D_j}\varepsilon_{N_j}\omega_{N_j} + Q_{1_j}Q_{3_j}\varepsilon_{D_j} \\ \Omega_{1_j} &= p_j\alpha_{N_j} + (1-p_j)Q_{1_j} \\ \Omega_{2_j} &= p_jQ_{2_j} + (1-p_j)\alpha_{D_j} \\ \Omega_{3_j} &= Q_{1_j}Q_{2_j} - \alpha_{N_j}\alpha_{D_j} \\ \Omega_{4_j} &= Q_{3_j}Q_{4_j} - \omega_{N_j}\omega_{D_j} \\ \Omega_{5_j} &= p_jM_{13_j} + (1-p_j)M_{23_j} \\ \Omega_{6_j} &= p_jM_{14_j} + (1-p_j)M_{24_j} \\ \Omega_{7_j} &= \Omega_{2_j}\Omega_{4_j} + \Omega_{1_j}\Omega_{4_j} + \Omega_{5_j} + \Omega_{6_j} \\ \Omega_{8_j} &= \Omega_{2_j}\Omega_{4_j} + \psi_{1_j}\Omega_{1_j}\Omega_{4_j} + \psi_{2_j}\Omega_{5_j} + \psi_{3_j}\Omega_{6_j} \\ M_{1_j} &= M_{11_j} + \psi_{1_j}M_{12_j} + \psi_{2_j}M_{13_j} + \psi_{3_j}M_{14_j} \\ M_{2_j} &= M_{21_j} + \psi_{1_j}M_{22_j} + \psi_{2_j}M_{23_j} + \psi_{3_j}M_{24_j} \\ J_{1j} &= \omega_j(\mu_j + \psi_{N_j}) + \mu_j + \psi_{D_j} \\ J_{2j} &= \mu_j(\psi_{N_j} + \mu_j + \psi_{D_j} \\ J_{3j} &= p_j\mu_j + \psi_{D_j} \\ J_{4j} &= (1-p_j)\mu_j + \psi_{N_j} \\ J_{5j} &= \omega_j[Q_{2_j}p_j + (1-p_j)\alpha_{D_j} \\ J_{6j} &= Q_{2_j}J_{3j} + \omega_jJ_{4j}\alpha_{D_j} \\ J_{7j} &= \omega_j[Q_{1_j}(1-p_j) + p_j\alpha_{N_j}] \\ J_{8j} &= \alpha_{N_j}J_{3j} + \omega_jJ_{4j}Q_{1_j} \end{aligned}$$

$$
\begin{aligned}
J_{9j} &= \omega_j[M_{13j}p_j + (1-p_j)M_{23j}] \\
J_{10j} &= M_{13j}J_{3j} + \omega_j J_{4j}M_{23j} \\
J_{11j} &= \omega_j[M_{14j}p_j + (1-p_j)M_{24j}] \\
J_{12j} &= M_{14j}J_{3j} + \omega_j J_{4j}M_{24j} \\
J_{13j} &= J_{5j}\Omega_{4j} + J_{7j}\Omega_{4j} + J_{9j} + J_{11j} \\
J_{14j} &= \Omega_{3j}\Omega_{4j}[p_j\omega_j + (1-p_j)]J_{6j}\Omega_{4j} + J_{8j}\Omega_{4j} + J_{10j} + J_{12j} \\
J_{15j} &= \Omega_{3j}\Omega_{4j}(J_{3j} + J_{4j}) \\
J_{16j} &= J_{5j}\Omega_{4j} + J_{7j}\psi_{1j}\Omega_{4j} + \psi_{2j}J_{9j} + \psi_{3j}J_{11j} \\
J_{17j} &= J_{6j}\Omega_{4j} + J_{8j}\psi_{1j}\Omega_{4j} + \psi_{2j}J_{10j} + \psi_{3j}J_{12j}
\end{aligned}
$$

Appendix C.

$$
\begin{aligned}
S_m^{***} &= \frac{\Pi_m}{\mu_m + \lambda_f^{***}}, & S_f^{*} &= \frac{\Pi_f}{\mu_f + \lambda_m^{***}} \\
I_{N_m}^{***} &= \frac{\Pi_m\Omega_{2_m}\lambda_f^{***}}{\Omega_{3_m}(\mu_m + \lambda_f^{***})}, & I_{N_f}^{***} &= \frac{\Pi_f\Omega_{2_f}\lambda_m^{***}}{\Omega_{3_f}(\mu_f + \lambda_m^{***})} \\
I_{D_m}^{***} &= \frac{\Pi_m\Omega_{1_m}\lambda_f^{***}}{\Omega_{3_m}(\mu_m + \lambda_f^{***})}, & I_{D_f}^{***} &= \frac{\Pi_f\Omega_{1_f}\lambda_m^{***}}{\Omega_{3_f}(\mu_f + \lambda_m^{***})} \\
A_{N_m}^{***} &= \frac{\Pi_m\Omega_{5_m}\lambda_f^{***}}{\Omega_{3_m}\Omega_{4_m}(\mu_m + \lambda_f^{***})}, & A_{N_f}^{***} &= \frac{\Pi_f\Omega_{5_f}\lambda_m^{***}}{\Omega_{3_f}\Omega_{4_f}(\mu_f + \lambda_m^{***})} \\
A_{D_m}^{***} &= \frac{\Pi_m\Omega_{6_m}\lambda_f^{***}}{\Omega_{3_m}\Omega_{4_m}(\mu_m + \lambda_f^{***})}, & A_{D_f}^{***} &= \frac{\Pi_f\Omega_{6_f}\lambda_m^{***}}{\Omega_{3_f}\Omega_{4_f}(\mu_f + \lambda_m^{***})}.
\end{aligned}
$$

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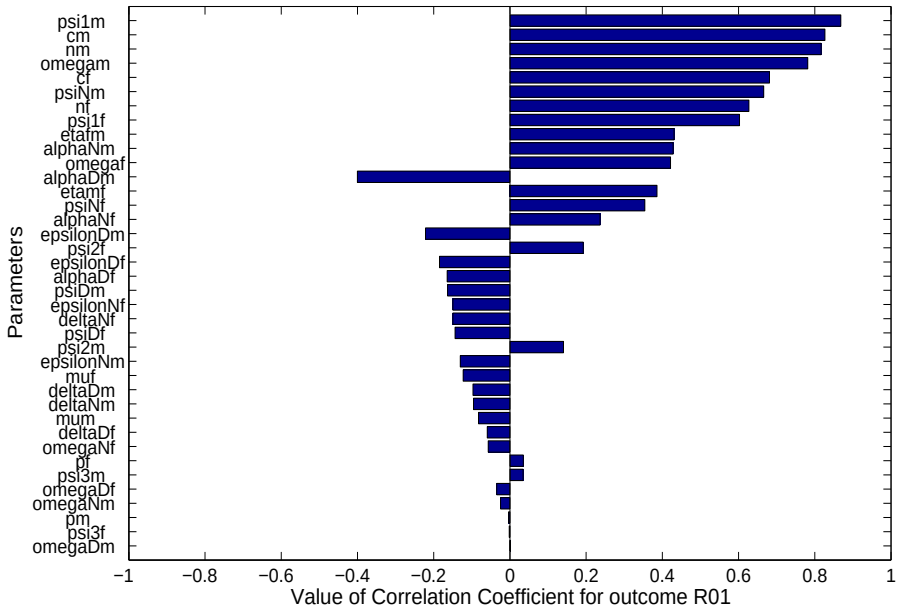

FIGURE 2. PRCC plot for parameters

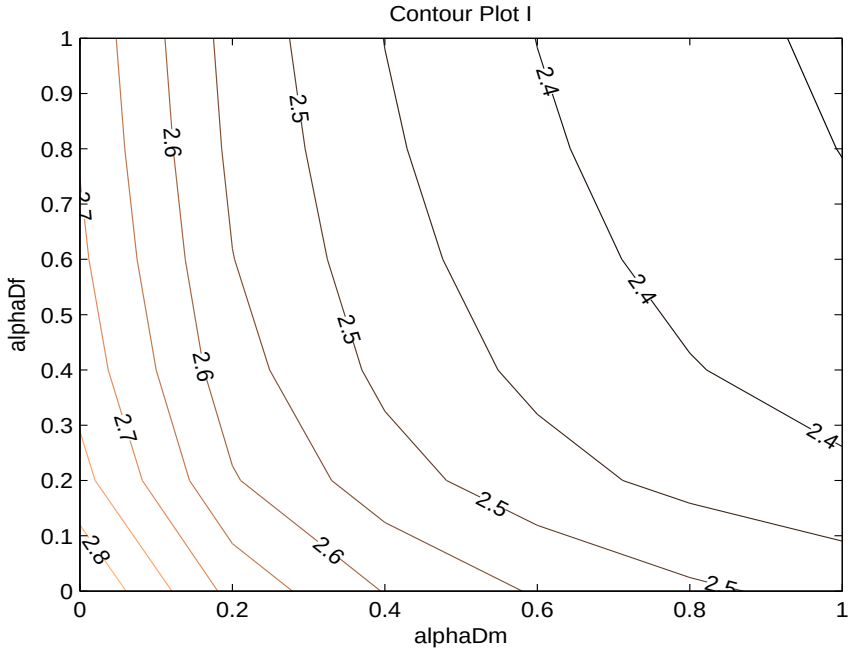

FIGURE 3. Contour plot for α_{D_m} and α_{D_f} for R_{0_1}

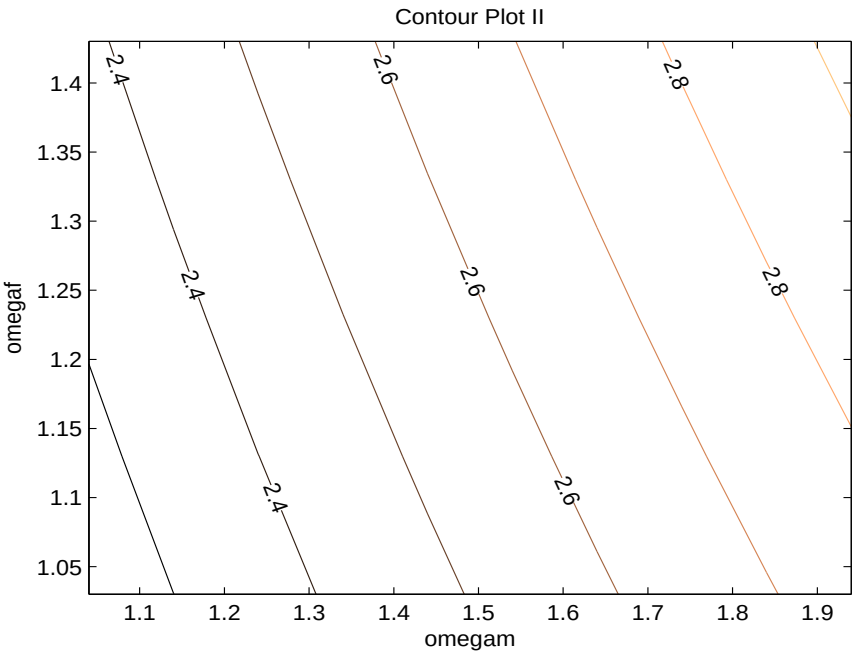

FIGURE 4. Contour plot for ω_m and ω_f for R_{0_1}

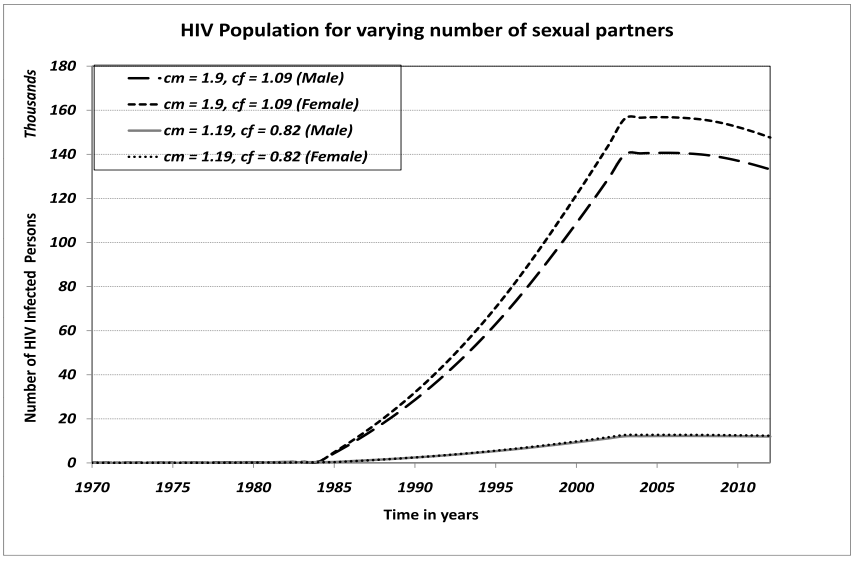

FIGURE 5. Comparison of HIV Population for varying c_m and c_f

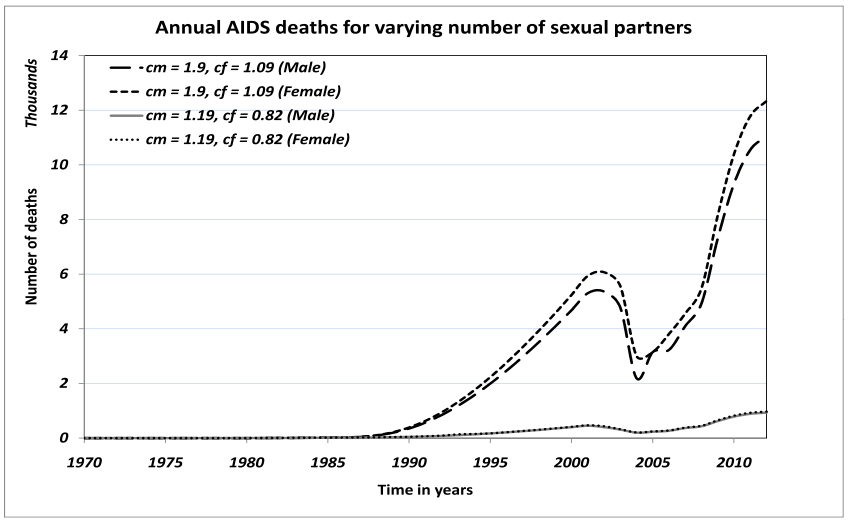

FIGURE 6. Comparison of annual AIDS deaths for varying c_m and c_f

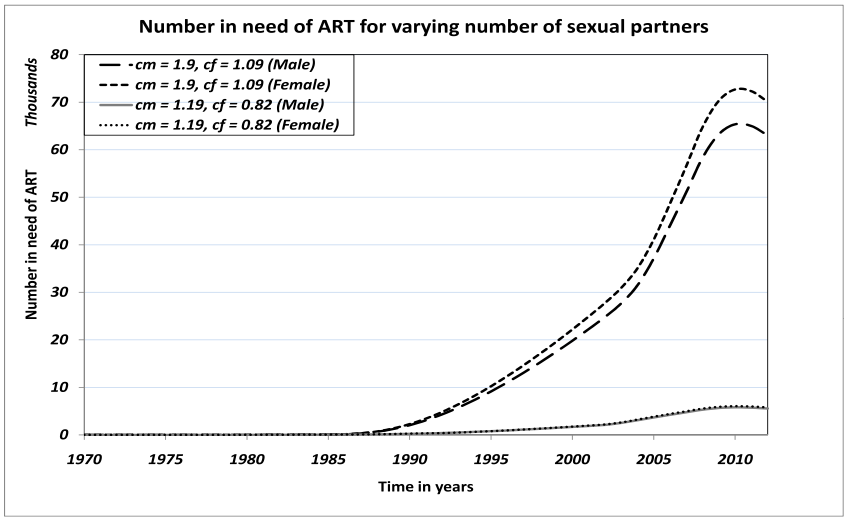

FIGURE 7. Comparison of need for ART for varying c_m and c_f

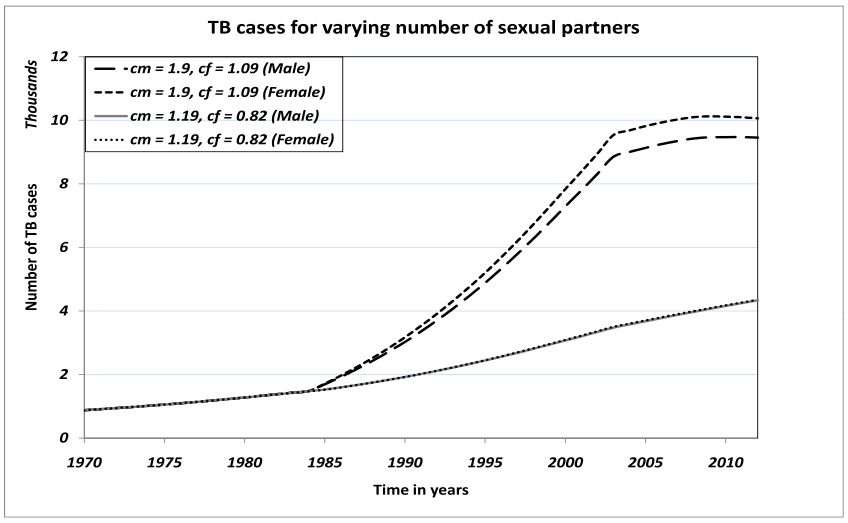

FIGURE 8. Comparison of TB cases for varying c_m and c_f

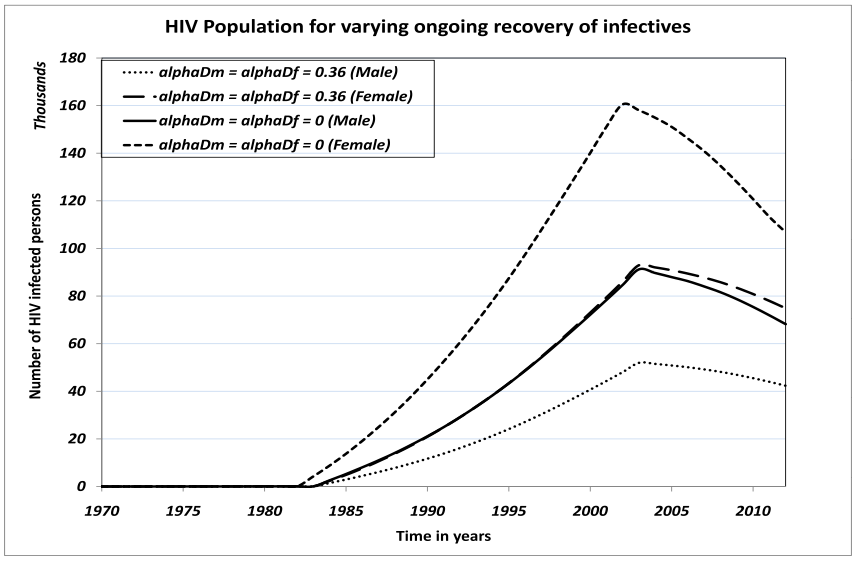

FIGURE 9. Comparison of HIV population for varying α_{D_m} and α_{D_f}

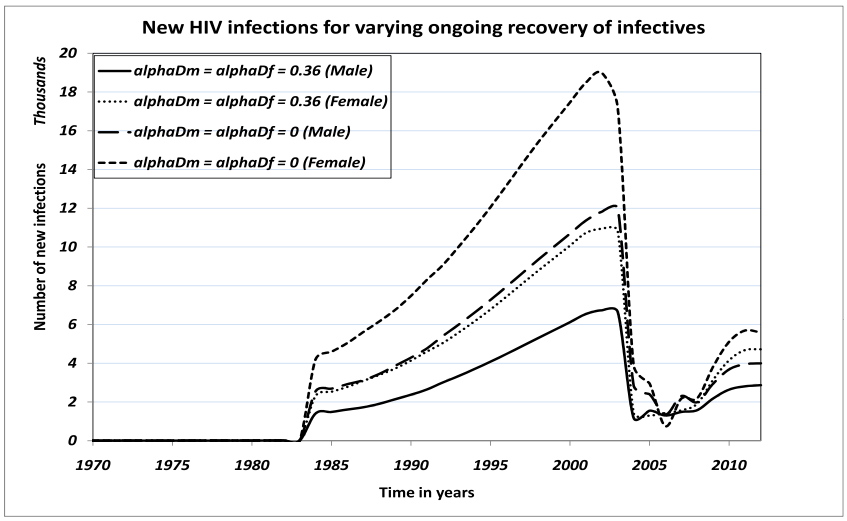

FIGURE 10. Comparison of new HIV infections for varying α_{D_m} and α_{D_f}

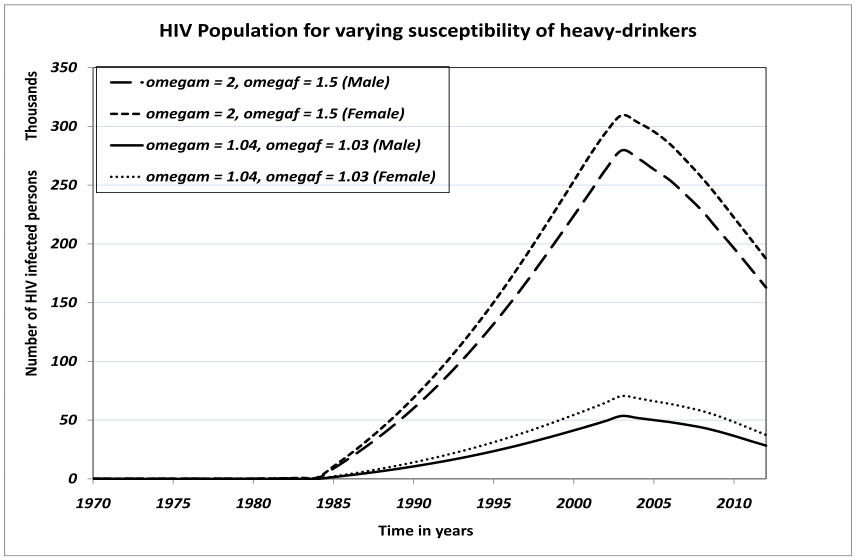

FIGURE 11. Comparison of HIV population for varying ω_m and ω_f

TABLE 1. Description of parameters and range of values

Par.	Description	Range	Mean	Var.
Π_m	Recruitment rate of male susceptibles			
Π_f	Recruitment rate of female susceptibles			
p_m	Proportion of male susceptibles who are moderate drinkers	0.51-0.61	0.56	0.0008
p_f	Proportion of female susceptibles who are moderate drinkers	0.72-0.75	0.735	0.00007
μ_m	Annual death rate of males in the absence of HIV/AIDS	0.025-0.029	0.027	1.3×10^{-6}
μ_f	Annual death rate of females in the absence of HIV/AIDS	0.025-0.029	0.027	1.3×10^{-6}
c_m	Average number of sexual partners for moderate infective males among moderate susceptible females per year	1.19-1.9	1.55	0.04
c_f	Average number of sexual partners for moderate infective females among moderate susceptible males per year	0.82-1.09	0.96	0.006
η_{fm}	Probability of HIV transmission/coital act - female to male	0.00143-0.00164	0.00154	3.7×10^{-9}
η_{mf}	Probability of HIV transmission/coital act - male to female	0.00124-0.00143	0.00134	3×10^{-9}
n_f	Av. no. of coital acts/partner/year for moderate infective females	108-143	125	102
n_m	Av. no. of coital acts/partner/year for moderate infective males	62-99	80	114
ψ_{1_m}	Modification parameter for heavy drinking infective males for causing new infections among moderate susceptible females	1.06-3	2.03	0.31
ψ_{1_f}	Modification parameter for heavy drinking infective females for causing new infections among moderate susceptible males	1.04-2	1.52	0.08
ψ_{2_m}	Modification parameter for moderate MLWA for causing new infections among moderate susceptible females	0.51-1	0.76	0.02
ψ_{2_f}	Modification parameter for moderate FLWA for causing new infections among moderate susceptible males	0.52-1	0.76	0.02
ψ_{3_m}	Mod. parameter for heavy-drinking MLWA for causing new infections among moderate susceptible females	0.91-1.08	1	0.002
ψ_{3_f}	Modification parameter for heavy-drinking FLWA for causing new infections among moderate susceptible males	0.97-1.06	.99	0.0006
ψ_{N_m}	Rate at which moderate male susceptibles become heavy-drinkers	0-0.22	0.11	0.004

TABLE 2. Description of parameters and range of values

Par.	Description	Range	Mean	Var.
ψ_{N_f}	Rate at which moderate female susceptibles become heavy-drinkers	0 - 0.11	0.055	0.001
ψ_{D_m}	Rate at which heavy-drinking male susceptibles become moderate drinkers	0.07 - 0.11	0.09	0.0001
ψ_{D_f}	Rate at which heavy-drinking female susceptibles become moderate drinkers	0.06 - 0.16	0.11	0.0008
ω_m	Modification parameter for heavy-drinking susceptible males for acquiring new infections relative to moderate susceptible males	1.04 - 2	1.52	0.08
ω_f	Modification parameter for heavy-drinking susceptible females for acquiring new infections relative to moderate susceptible females	1.03 - 1.5	1.27	0.02
α_{N_m}	Rate at which moderate male infectives become heavy drinkers	0-0.36	0.18	0.01
α_{N_f}	Rate at which moderate infective females become heavy drinkers	0-0.22	0.11	0.004
α_{D_m}	Rate at which heavy drinking male infectives become moderate infectives	0-0.36	0.18	0.01
α_{D_f}	Rate at which heavy drinking female infectives become moderate infectives	0-0.36	0.18	0.01
ω_{N_m}	Rate at which moderate MLWA become heavy drinkers	0-0.11	0.055	0.001
ω_{N_f}	Rate at which moderate FLWA become heavy drinkers	0-0.05	0.025	0.0002
ω_{D_m}	Rate at which heavy drinking MLWA become moderate	0-0.56	0.28	0.03
ω_{D_f}	Rate at which heavy drinking FLWA become moderate	0-0.56	0.28	0.03
ε_{N_m}	Progression rate to AIDS for moderate male infectives	0.118-0.133	0.126	0.00002
ε_{N_f}	Progression rate to AIDS for moderate female infectives	0.105-0.118	0.112	0.00001
ε_{D_m}	Progression rate to AIDS for heavy-drinking male infectives	0.141-0.164	0.153	0.00004
ε_{D_f}	Progression rate to AIDS for heavy-drinking female infectives	0.127-0.145	0.136	0.00003
δ_{N_m}	Death rate due to AIDS for moderate MLWA	0.33-0.5	0.415	0.0024
δ_{N_f}	Death rate due to AIDS for moderate FLWA	0.33-0.5	0.415	0.0024
δ_{D_m}	Death rate due to AIDS for heavy-drinking MLWA	0.4-0.67	0.535	0.006
δ_{D_f}	Death rate due to AIDS for heavy-drinking FLWA	0.4-0.67	0.535	0.006

TABLE 3. Parameter values used in simulations (Figures 3 - 11)

Par.	p_m	p_f	μ_m	μ_f	c_m	c_f	η_{fm}	η_{mf}
Value	0.56	0.735	0.027	0.027	1.9	1.09	0.00164	0.00143
Par.	n_m	ψ_{1_m}	ψ_{1_f}	ψ_{2_m}	ψ_{2_f}	ψ_{3_m}	ψ_{3_f}	ψ_{N_m}
Value	101	2.03	1.52	0.76	0.76	1	0.99	0.11
Par.	ψ_{D_m}	ψ_{D_f}	ω_m	ω_f	α_{N_m}	α_{N_f}	α_{D_m}	α_{D_f}
Value	0.09	0.11	1.52	1.27	0.18	0.11	0.18	0.18
Par.	ω_{N_f}	ω_{D_m}	ω_{D_f}	ε_{N_m}	ε_{N_f}	ε_{D_m}	ε_{D_f}	$\delta_{N_m} = \delta_{N_f}$
Value	0.025	0.28	0.28	0.126	0.112	0.153	0.136	0.415
Par.	n_f	ψ_{N_f}	ω_{N_m}	$\delta_{D_m} = \delta_{D_f}$				
Value	117	0.055	0.055	0.535				