

A MECHANISM BY WHICH MASS INCARCERATION CONTRIBUTES TO HIV DISPARITIES IN THE UNITED STATES

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ABSTRACT. In this work, we develop a mathematical model of HIV epidemiology to explore a possible mechanism by which mass incarceration can lead to increased HIV incidence. The results are particularly relevant for the African American community in the United States that represents only 12% of the total population but accounts for 45% of HIV diagnoses and 40% of the incarcerated population. While most explanations of the link between mass incarceration (or anything else that leads to a population with a low ratio of males to females) and higher HIV burden are based on the complicated idea of sexual concurrency, we propose a much simpler mechanism based on the idea of sexual activity compensation.

The primary assumption behind this mechanism is that females determine the overall level of sexual activity in a population. Consequently, sexual activity will remain relatively stable even when male-to-female ratios are low.

For this to be possible, the pool of men will increase their sexual activity to meet the demands of the female population. Through mathematical analysis and numerical simulation, we demonstrate that these assumptions produce a situation in which mass incarceration (and low male-to-female ratios, in general) lead to higher HIV incidence.

1. INTRODUCTION

While the prevalence of HIV in the general population of the United States is relatively low, disparity remains a defining characteristic of the American epidemic that is present across many dimensions. For example, HIV disproportionately affects: men who have sex with men as opposed to heterosexuals; the black and Hispanic populations as opposed to the white population; groups of lower socioeconomic status; and individuals residing in a particular geographical settings [21]. Consequently, the design of effective HIV interventions in the United States requires not only detailed data to assess the HIV epidemic in a particular setting but also an understanding of the drivers of HIV in each specific population and setting.

In this work, we focus our attention on HIV amongst heterosexual African Americans in the United States. Understanding the HIV epidemic in this population is crucial given that African Americans represent only 12% of the US population but

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accounted for 45% of all HIV diagnoses [19] in the US in 2015. Buot et al. [4] examined the relationship between numerous social indicators and HIV incidence across eighty large US cities. Interestingly, they found gender imbalance to be a “uniquely strong predictor of HIV incidence amongst black individuals.” By gender imbalance, we refer to the situation where the ratio of the numbers of males to females in the population (which we will refer to as simply the “male-female ratio”) is significantly different than 1. Settings where particular industries disproportionately attract men (e.g. military bases, oil and gas sector, etc.) can often have male-female ratios larger than 1. Male-female ratios less than 1 are often observed in settings with high rates of violent crime and incarceration which both act to remove men from the population at higher rates than women. The latter is of particular concern in many African American settings given disparities in imprisonment rates amongst races in the US with 40% of imprisoned individuals being from the 12% of population that is African American [20]. Given our focus on HIV in African Americans, understanding the mechanisms by which low male-female ratios lead to increased HIV incidence is vital.

The predominant explanation of how low male-female ratios increase HIV incidence revolves around the idea of sexual concurrency which has been correlated to low male-female ratios in multiple studies [2, 4, 13, 14, 17]. Concurrent sexual partnership describes the situation where an individual has ongoing sexual relationships with multiple partners over some period of time. In other words, the time courses of an individual’s sexual relationships overlap. Sexual concurrency is then essentially the opposite of serial monogamy where an individual has at most one sexual partner over a given period of time. With serial monogamy, an individual’s current sexual relationship ends before their next sexual relationship begins.

Concurrent sexual relationships have the potential to significantly increase the size of HIV epidemics [11]. When an individual who is engaged in concurrent sexual relationships becomes infected, a subsequent transmission is likely to occur more rapidly [10, 12]. This is due to the fact that the individual can infect another one of their current sexual partners right away. In serial monogamy, the newly infected partner only has a sexual relationship with the partner that infected them. Therefore, future infection can not occur until this initial sexual relationship has ended and a new relationship has begun.

The theory of incarceration and concurrency is then that as mass incarceration drives male-female ratios down, the females left behind by their incarcerated partners acquire additional, often concurrent, relationships with the smaller pool of males in the general population [9]. Moreover, the low male-female ratio shifts the power dynamics of these relationships to the male partner limiting the female partner’s ability to negotiate condom use and/or monogamy [9].

While it is well-established that sexual concurrency plays a role in the sexual transmission of HIV, quantifying the relative size of its contribution has remained difficult and is a source of debate in the HIV community [9]. This is mostly because modeling sexual concurrency requires network-based models (rather than simpler differential equation models) and detailed data on human sexual behavior that is incredibly difficult to obtain.

In this work, we demonstrate a related but simpler mechanism by which a low male-female ratio can lead to increased HIV incidence in a population. The primary assumption behind this mechanism is that females on average determine the level of

sexual activity in a population. Therefore, the level of sexual activity of females in a population will remain relatively stable, even in a low male-female ratio scenario. For this to be possible, the pool of men in the low male-female ratio population will increase their sexual activity to meet the demands of the female population. We emphasize that we do not propose this mechanism as an alternative to the concurrency explanation, but as an additional avenue by which low male-female ratios can lead to higher HIV incidence in a population.

In Section 2, we develop a mathematical model, and present model parameters. In Section 3, we present mathematical analyzes and calculate basic reproductive numbers. Section 4 is devoted to analyzing the model via numerical simulations. Finally in Section 5, we discuss our findings.

2. MATHEMATICAL MODEL

We develop an SI -type model for HIV dynamics that includes the effect of incarceration. The model consists of the following six classes of individuals: susceptible females in the general population (S_f), infected females in the general population (I_f), susceptible males in the general population (S_m), infected males in the general population (I_m), susceptible incarcerated men (S_j), and infected incarcerated men (I_j). We let $N_f = S_f + I_f$ denote the total number of females in the general population and $N_m = S_m + I_m$ the total number of males in the general population (not incarcerated).

In this model, we assume that individuals enter the sexually-active population with a maturation rate of Λ . All individuals enter the population as susceptible; half are female and half are male. Individuals leave that population at a background mortality rate denoted by μ . We use a standard incidence formulation (i.e. one in which the rate of transmission depends on the proportion of the population that is susceptible rather than the number of individuals that are susceptible) of HIV transmission with coefficient σ_1 for male-to-female transmission and σ_2 for female-to-male transmission. We assume women and men have sexual contacts at rates c_1 and c_2 , respectively. Infected individuals leave the population at an HIV-induced mortality rate of μ_A . We assume that males in the general population are incarcerated at a rate γ_1 and return to the general population at a rate of γ_2 . The resulting system of ordinary differential equations is given by

$$\begin{aligned}
 S'_f &= \frac{\Lambda}{2} - \frac{c_1\sigma_1 S_f I_m}{N_m} - \mu S_f, \\
 I'_f &= \frac{c_1\sigma_1 S_f I_m}{N_m} - (\mu + \mu_A) I_f, \\
 S'_m &= \frac{\Lambda}{2} - \frac{c_2\sigma_2 S_m I_f}{N_f} - \mu S_m - \gamma_1 S_m + \gamma_2 S_j, \\
 I'_m &= \frac{c_2\sigma_2 S_m I_f}{N_f} - (\mu + \mu_A) I_m - \gamma_1 I_m + \gamma_2 I_j, \\
 S'_j &= -\mu S_j + \gamma_1 S_m - \gamma_2 S_j, \\
 I'_j &= -(\mu + \mu_A) I_j + \gamma_1 I_m - \gamma_2 I_j.
 \end{aligned} \tag{2.1}$$

Parameter definitions and values are summarized in Table 1.

The flow diagram of our model is as in Figure 1.

TABLE 1. Model parameters: definitions and values

	Symbol	Value	Reference
Maturation rate	Λ	1/30	[16]
Sexual contact rate for females	c_1	0.75	[6, 16]
Sexual contact rate for males	c_2	1.25	[6, 16]
Transmission coefficient for HIV from male-to-female	σ_1	1/30	[1, 6, 7, 15]
Transmission coefficient for HIV from female-to-male	σ_2	1/15	[1, 6, 7, 15]
Background mortality rate	μ	1/30	[6]
Disease-induced mortality rate	μ_A	1/15	[16]
Incarceration rate for males	γ_1	Range	assump.
Rate of reintroduction (i.e. release from Prison) for males	γ_2	Range	assump.

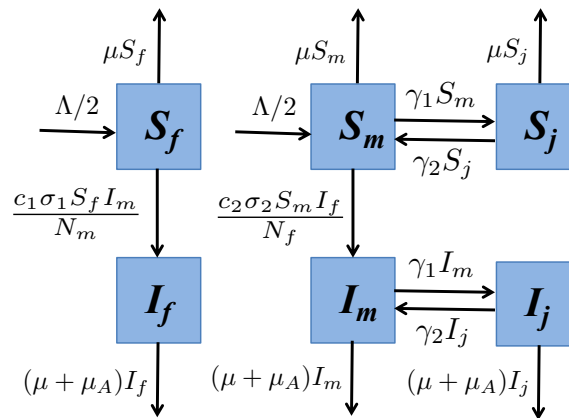


FIGURE 1. Flow diagram with birth and death rates

The goal of our work is to examine the effect of a low male-female ratio has on HIV disease burden. To do so, we will analyze two versions of the model:

- Constant male sexual activity: male sexual activity in the general population is independent of the male-female ratio (i.e. c_2 a fixed constant)
- Male sexual activity compensation: male sexual activity in the general population dynamically fluctuates in relation to the gender ratio (i.e. $c_2 = c_1 \frac{N_f}{N_m}$).

Again, the underlying assumption in the male sexual activity compensation version is that the female sexual contact rate, c_1 , remains fixed regardless of the gender ratio and consequently the remaining men in the low male-female ratio population will increase their sexual activity to meet the demands of the female population. To justify our mathematical formulation of sexual activity compensation (i.e. $c_2 = c_1 \frac{N_f}{N_m}$), we note that the total number of sexual contacts for the entire male population and the entire female should be equal as we are modeling heterosexual populations. To achieve this, we rescale the male contact rate by the ratio of females to males in the population so that $c_1 N_m = (c_2 \frac{N_f}{N_m}) N_m = c_2 N_f$.

At this point, a brief discussion of certain modeling assumptions is warranted. In our model formulation, we describe the mechanism that removes males from the general population as “incarceration.” We have done so because this is the dominant

cause discussed in the literature regarding low male-female ratios. Importantly, our modeling results hold regardless of what the actual mechanism is that leads to low male-female ratios whether it be death, migration, etc. Also, our formulation also assumes that HIV transmission does not occur in the incarcerated population which is quite likely not the case. We have done this intentionally so the modeling work isolates the focal mechanism of this study (i.e. male sexual activity compensation).

3. MATHEMATICAL ANALYSIS

To establish the structure of our population when the disease is not present, we begin by identifying the disease-free equilibrium (DFE). Without infection, the model given in System ((2.1) reduces to

$$\begin{aligned} S'_f &= \Lambda/2 - \mu S_f, \\ S'_m &= \Lambda/2 - \mu S_m - \gamma_1 S_m + \gamma_2 S_j, \\ S'_j &= -S_j \mu - \gamma_2 S_j + \gamma_1 S_m. \end{aligned}$$

To find the DFE, we set $S'_f = S'_m = S'_j = 0$ and solve to get

$$(S_f^*, I_f^*, S_m^*, I_m^*, S_j^*, I_m^*) = \left(\frac{\Lambda}{2\mu}, 0, \frac{(\mu + \gamma_2)\Lambda}{2\mu(\mu + \gamma_1 + \gamma_2)}, 0, \frac{\gamma_1 \Lambda}{2\mu(\mu + \gamma_1 + \gamma_2)} \right).$$

Importantly, the DFE explicitly tells us how the gender ratio of the general population will be determined by the rates at which males move in and out of the isolated class (i.e. γ_1, γ_2). Specifically, the ratio of males to females in the general population at DFE is given by

$$\frac{N_m^*}{N_f^*} = \frac{S_m^* + I_m^*}{S_f^* + I_f^*} = \frac{\mu + \gamma_2}{\mu + \gamma_1 + \gamma_2}.$$

Therefore, it is clear to see that the male-female ratio is a decreasing function of the incarceration rate γ_1 . Lastly, we note that the DFE is the same for both versions of the model because the formulation of male sexual activity level (i.e. c_2 fixed versus $c_2 = c_1 \frac{N_f}{N_m}$) has no effect when the disease is not present in the population (i.e. $I_f = I_m = I_j = 0$).

3.1. Basic reproductive number. The basic reproduction number (\mathcal{R}_0) measures the average number of new infections generated by a single infectious individual in a completely susceptible population [3, 5, 8, 18]. If $\mathcal{R}_0 < 1$, an average infectious individual is unable to replace itself and the disease will die out. If $\mathcal{R}_0 > 1$, the number of infected individuals rises and an epidemic results.

3.1.1. \mathcal{R}_0 , assuming constant male sexual activity. Using the next generation approach of [5, 18], we can formulate \mathcal{R}_0 as the spectral radius of the next-generation matrix, $\rho(FV^{-1})$, where F is the matrix of rates of new infection and V is the matrix of rates of transfer for infectious compartments. For the constant male sexual activity version of the model given by system ((2.1), matrices F and V are given by

$$F = \begin{bmatrix} 0 & \frac{c_1 \sigma_1 S_f}{S_m + I_m} & 0 \\ \frac{c_2 \sigma_2 S_m}{S_f + I_f} & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \quad \text{and} \quad V = \begin{bmatrix} \mu + \mu_A & 0 & 0 \\ 0 & \mu + \mu_A + \gamma_1 & -\gamma_2 \\ 0 & -\gamma_1 & \mu + \mu_A + \gamma_2 \end{bmatrix}.$$

Direct calculation then yields

$$\begin{aligned} \rho(FV^{-1}) &= \frac{\sqrt{(S_f + I_f)(S_m + I_m) (\mu + \mu_A + \gamma_1 + \gamma_2) c_1 \sigma_1 S_f c_2 \sigma_2 S_m (\mu + \mu_A + \gamma_2)}}{(S_f + I_f)(S_m + I_m) (\mu + \mu_A + \gamma_1 + \gamma_2) (\mu + \mu_A)}, \end{aligned}$$

which we then linearize around the DFE by evaluating at $(S_f^*, I_f^*, S_m^*, I_m^*, S_j^*, I_m^*) = \left(\frac{\Lambda}{2\mu}, 0, \frac{(\mu + \gamma_2)\Lambda}{2\mu(\mu + \gamma_1 + \gamma_2)}, 0, \frac{\gamma_1\Lambda}{2\mu(\mu + \gamma_1 + \gamma_2)}\right)$. The resulting expression for the basic reproductive number of constant male sexual activity version of our model is given by

$$\mathcal{R}_0 = \sqrt{\frac{c_1 c_2 \sigma_1 \sigma_2 (\mu + \mu_A + \gamma_2)}{(\mu_A + \mu + \gamma_1 + \gamma_2) (\mu + \mu_A)^2}}. \tag{3.1}$$

3.1.2. \mathcal{R}_0 , assuming male sexual activity compensation. Next we calculate the basic reproduction number \mathcal{R}_0 assuming male sexual activity dynamically fluctuates in relation to the gender ratio (i.e. $c_2 = c_1 \frac{N_f}{N_m} = c_1 \frac{S_f + I_f}{S_m + I_m}$). For this version of the model, we have that the matrices of rates of new infection and rates of transfer are given by

$$F = \begin{bmatrix} 0 & \frac{c_1 \sigma_1 S_f}{S_m + I_m} & 0 \\ \frac{c_1 \sigma_2 S_m}{S_m + I_m} & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \quad \text{and} \quad V = \begin{bmatrix} \mu + \mu_A & 0 & 0 \\ 0 & \mu + \mu_A + \gamma_1 & -\gamma_2 \\ 0 & -\gamma_1 & \mu + \mu_A + \gamma_2 \end{bmatrix}.$$

The spectral radius of the next-generation matrix is then calculated to be

$$\rho(FV^{-1}) = \frac{c_1 \sqrt{(\mu + \mu_A + \gamma_1 + \gamma_2) \sigma_1 S_f \sigma_2 S_m (\mu + \mu_A + \gamma_2)}}{(\mu + \mu_A + \gamma_1 + \gamma_2) (S_m + I_m) (\mu + \mu_A)}.$$

With

$$(S_f^*, I_f^*, S_m^*, I_m^*, S_j^*, I_m^*) = \left(\frac{\Lambda}{2\mu}, 0, \frac{(\mu + \gamma_2)\Lambda}{2\mu(\mu + \gamma_1 + \gamma_2)}, 0, \frac{\gamma_1\Lambda}{2\mu(\mu + \gamma_1 + \gamma_2)}\right),$$

we have that the basic reproductive number of the sexual activity compensation version of our model is

$$\mathcal{R}_0 = \frac{c_1 \sqrt{\mu + \gamma_2 \sigma_1 \sigma_2 (\mu + \mu_A + \gamma_2)}}{(\mu + \mu_A) \sqrt{\mu_A + \mu + \gamma_1 + \gamma_2} \sqrt{\mu + \gamma_1 + \gamma_2}}. \tag{3.2}$$

3.1.3. Effect of incarceration on \mathcal{R}_0 . Having established analytic expressions for both versions of the model, we can examine the relationship between the rate of incarceration and invasion potential of HIV at the DFE under the different assumptions regarding male sexual activity. Doing so under the assumption of constant male sexual activity (i.e. \mathcal{R}_0 as in ((3.1)), we find that

$$\frac{\partial \mathcal{R}_0}{\partial \gamma_1} = -\frac{\sqrt{c_2 \sigma_2 c_1 \sigma_1 (\mu + \mu_A + \gamma_2)}}{2 (\mu_A + \mu + \gamma_1 + \gamma_2)^{3/2} (\mu + \mu_A)} < 0. \tag{3.3}$$

Assuming that male sexual activity dynamically fluctuates in relation to the gender ratio (i.e. \mathcal{R}_0 as in ((3.2)), we have that

$$\frac{\partial \mathcal{R}_0}{\partial \gamma_1} = -\frac{\sqrt{\mu + \gamma_2} \sqrt{\sigma_1} \sqrt{\sigma_2} \sqrt{\mu + \mu_A + \gamma_2} c_1 (\mu_A + 2\mu + 2\gamma_1 + 2\gamma_2)}{2 (\mu_A + \mu + \gamma_1 + \gamma_2)^{3/2} (\mu + \gamma_1 + \gamma_2)^{3/2} (\mu + \mu_A)} < 0. \tag{3.4}$$

We see that changing the incarceration rate has the same qualitative effect on both versions of the model. Specifically, higher rates of incarceration will produce lower values for the basic reproductive number. This means that HIV is less likely to become established when introduced into a population with higher rates of incarceration and that mass incarceration could actually increase the chance of eradication in a population with sufficiently low HIV prevalence. However, we will see in the next section that this positive effect of mass incarceration will not hold in populations in which HIV is endemic.

4. SIMULATION RESULTS

Our mathematical analyzes in the previous section have shown that increasing incarceration rates among the male population leads to a reduction in the basic reproductive number (i.e. $\frac{\partial \mathcal{R}_0}{\partial \gamma_1} < 0$) in both the version of the model with constant male sexual activity (i.e. c_2 fixed) and the version with male sexual activity compensating for gender ratio (i.e. $c_2 = c_1 \frac{N_f}{N_m}$).

Unfortunately, calculations of the endemic equilibria for both versions of the model have proven to be too cumbersome to provide any useful insight. To examine the relationship between incarceration rates and disease incidence for the model with male sexual activity compensation, we turn to numerical simulation. Figure 2 shows the dynamics of HIV prevalence that result from simulating the model with the parameter values from Table 1, $\gamma_2 = 0.10$ and varying the incarnation rate, γ_1 , from 0 to 0.04 to 0.20. In both the female and male populations (Figures 2a and 2b, respectively), we see that increased incarceration rates produce an earlier peaking epidemic and higher HIV prevalence at the endemic equilibrium for the sexual activity compensation model.

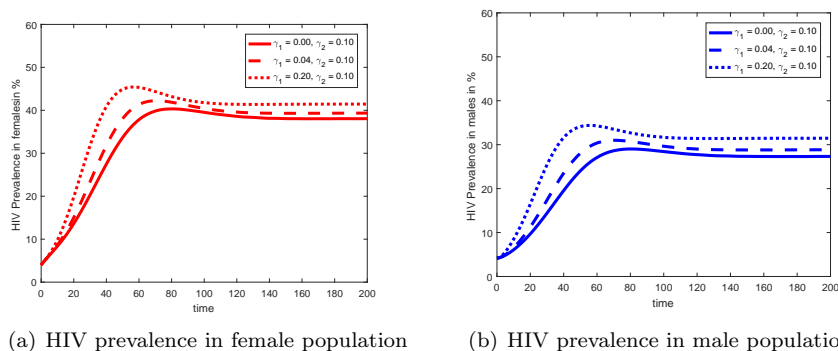


FIGURE 2. Dynamics of HIV prevalence in the female and male populations at various incarceration rates.

While, our mathematical analyzes showed that increased incarceration has the same qualitative effect on the basic reproductive number for both versions of the model (i.e. $\frac{\partial \mathcal{R}_0}{\partial \gamma_1} < 0$), it is important to obtain a *quantitative* understand of the relationship as well. To do so, we calculate the \mathcal{R}_0 using parameter values from Table 1 and allowing incarceration rates to vary through wide ranges ($0 < \gamma_1 < 0.01$ and $0 < \gamma_2 < 0.05$, specifically). Considering the system without incarceration as

a base case (i.e. $\gamma_1 = \gamma_2 = 0$), we note that the baseline parameters values in Table 1 yield reproductive numbers of $\mathcal{R}_0 = 1.84$ and $\mathcal{R}_0 = 1.59$ for the constant activity and the activity compensation models, respectively. The results for the full ranges of incarceration and reintroductions rates are presented in Figure 3. We immediately see that \mathcal{R}_0 is significantly larger for the model with fixed male sexual activity (Figure 3a) than with sexual activity compensation for all combinations of incarceration rates (Figure 3b). We also see that while the quantitative effect of increased incarceration rates is the same for both models, the quantitative relationship is quite different as the reproductive number of the model with male sexual activity compensation is much more sensitive to incarceration rates (i.e. \mathcal{R}_0 decreases by larger amount).

Lastly, we look at the relationship between HIV prevalence and incarceration by simulating the model to endemic equilibrium for ranges of incarceration rates. The results are shown in Figure 4 and demonstrate that incarceration has opposite effects in the model with fixed male sexual activity (see Figures 4a and c) and in the model with male sexual activity compensation (see Figures 4b and d). When the number of incarcerated individuals is highest (i.e. γ_1 large, γ_2 small), we see HIV prevalence in the female population of $\approx 30\%$ in the constant male sexual activity model (see Figure 4a) versus an $\approx 46\%$ prevalence in the model with sexual activity compensation (see Figure 4b). In the male population, we have an HIV prevalence of $\approx 19\%$ in the constant male sexual activity model (see Figure 4c) versus an $\approx 39\%$ prevalence in the model with sexual activity compensation (see Figure 4d) when the number of incarcerated individuals is highest.

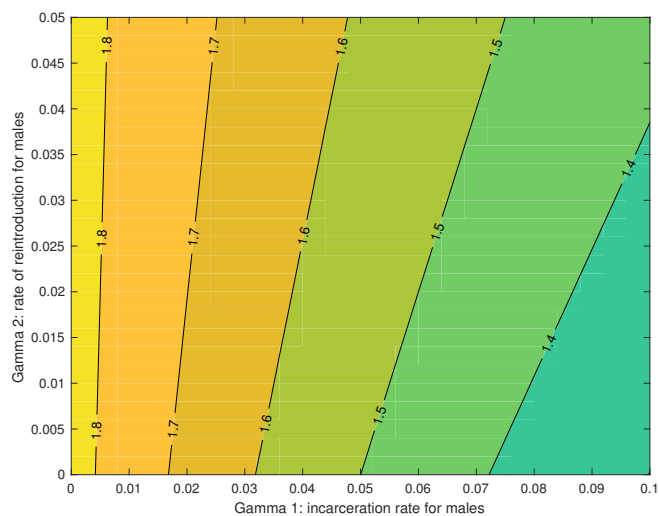
5. DISCUSSION

In this work, we have used a basic mathematical model for the epidemiology of HIV in heterosexual populations to investigate the relationship between mass incarceration and HIV burden. More specifically, we examined the effect of assuming that females on average determine the level of sexual activity in a population and that men in populations with a low male-female ratio will increase their sexual activity to meet the demands of the females.

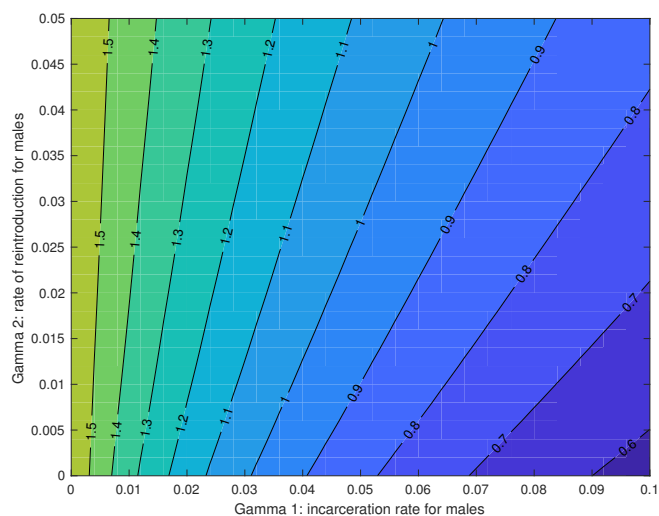
Our modeling work shows that this male sexual activity compensation can indeed significantly increase the HIV burden in a population with low male-female ratios. Surprisingly, we found that this assumption increases disease burden at the endemic equilibrium while also reducing the basic reproductive number. Such behavior indicates the possible existence of backward bifurcation.

As the goal of this research is to demonstrate that the mechanism of sexual activity compensation will lead to a higher HIV burden in the general population, our work does not aim to quantify the amount by which this effect has contributed to increased HIV incidence in particular settings. Such a study will require a more detailed model that adequately describes the unique dynamics of HIV infection and includes HIV transmission among incarcerated population. In addition to adjusting the model, the study will also require detailed data on sexual behavior and incarceration rates.

To conclude, we again emphasize that we do not propose this mechanism as an alternative to the concurrency explanation but as an additional avenue by which low male-female ratios can lead to increased HIV incidence.



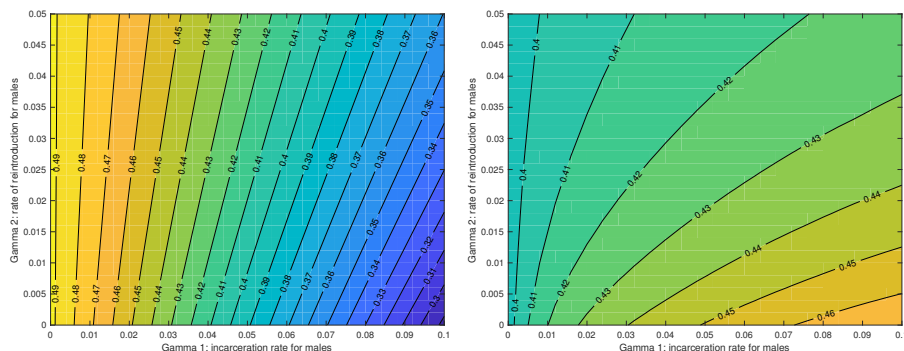
(a) \mathcal{R}_0 assuming constant male sexual activity level (i.e. c_2 fixed)



(b) \mathcal{R}_0 assuming male sexual activity compensation for gender ratio (i.e. $c_2 = c_1 \frac{N_f}{N+m}$)

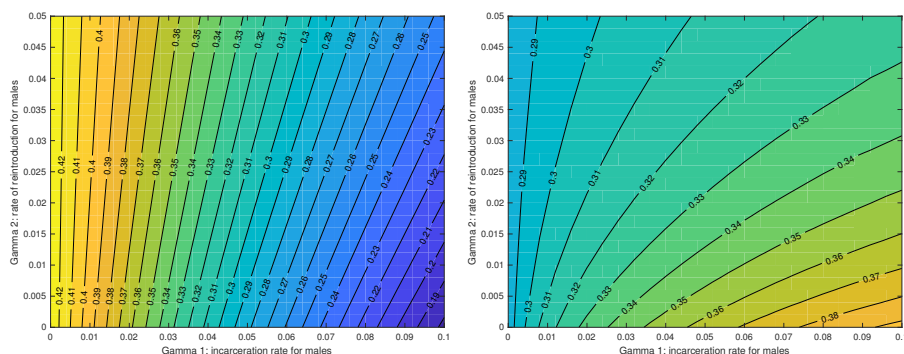
FIGURE 3. Comparison of basic reproductive number, \mathcal{R}_0 , as a function of incarceration rates for model versions with constant male sexual activity level (i.e. c_2 fixed) and with male sexual activity compensating for gender ratio (i.e. $c_2 = c_1 \frac{N_f}{N+m}$).

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(a) Endemic HIV prevalence in females, assuming constant male sexual activity level

(b) Endemic HIV prevalence in females when male sexual activity compensates for gender ratio



(c) Endemic HIV prevalence males, assuming constant male sexual activity level

(d) Endemic HIV prevalence in males when male sexual activity compensates for gender ratio

FIGURE 4. Comparison of HIV prevalence at endemic equilibrium as a function of incarceration rates for model versions with constant male sexual activity level (i.e. c_2 fixed) and with male sexual activity compensating for gender ratio (i.e. $c_2 = c_1 \frac{N_f}{N_m}$).

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