The Role of Treatment on Controlling Chancroid Prevalence

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Abstract. Chancroid is a highly infectious and curable sexually transmitted disease caused by the bacterium Haemophilus Ducreyl (also known as H. Ducreyl). A deterministic mathematical model for investi- gating the role of treatment on controlling chancroid epidemic is formulated and rigorously analyzed. A threshold quantity known as the reproductive number, which measures the number of secondary infections produced by a single chancroid infective when introduced in a population of susceptibles in the presence of treatment has been derived. Equilibria for the model are determined and their stability are examined. Latin hypercube sampling has been used to perform the sensitivity analysis of the reproductive number.

Keywords: Chancroid, treatment, reproductive number, stability, sensitivity analysis.

1. Introduction

Despite an estimated 7 million chancroid cases occurring each year, it remains one of the important neglected sexually transmitted diseases [36, 26]. It is common in many parts of the world's poorest regions with the weakest health infrastructure as those in Africa, Asia and the Caribbean [26]. These regions also have the highest rates of HIV in the world and is common in all 18 countries where adult HIV prevalence surpasses 8% [26]. This confluence of high rates of chancroid, genital and HIV points to a cofactor that may account for a large proportion of new HIV infections acquired heterosexually in most severely affected countries of the world [26]. Chancroid incidence and prevalence varies greatly by country and region, however for

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the reasons that poorly understood [36]. The global epidemiology of chancroid is so poorly documented that it is not in World Health Organisation estimates of the global incidence of curable sexually transmitted diseases [3]. Chancroid is an acute bacterial infection caused by Haemophilus ducreyi, a Gram-negative coccobacillus and is usually located in the genital area [9]. Three to ten days following exposure to Haemophilus ducreyi a painful vescular papule begins, that quickly becomes an ulcer with bright red areola and shelving mar- gins [35]. There may be single or multiple lessions that are round or irregular with undetermined borders ranging from 3-20mm in size [35]. Its biology and pathogenesis have been well described [27, 20]. Clinical differentiation of chancroid from other types of genital ulcers is not reliable [10, 18] and Haemophilus ducreyi is also hard to culture [33]. This makes it difficult to control using etiological identification mechanisms. Chancroid symptoms are much more noticiable in males but it can be asymptomatic in both sexes or limited pain during urination, defecation or sexual activity [1] and in some cases there may be rectal bleeding or vaginal discharge [1]. In 30-50% of chancroid cases painful enlargement of inguinal lymph nodes occur after 7-14 days and most often rupture and discharge pus [35]. Secondary infections at times occur and result in the destruction of the affected tissue [5]. Chancroid can be treated with macrolides, quinolones, and some third-generation cephalosporins [22, 25, 30]. Single doses of certain antibiotics, such as ciprofloxacin and azithromycin, are highly effective [22, 2] although longer treatments may be more effective in uncircumcised males and patients with HIV infection [14, 31]. Antibiotics may also provide some protection from reinfection: one study estimated that the prophylactic effect of a single dose of azithromycin against new H. ducreyi infection lasted as long as two months after treatment [28].

Malaria, Tuberculosis, Hepatitis B & C, HIV/AIDS, Trichomanas vaginalis and other sexually transmitted infections have all being mathematically accounted for (see[29, 16, 4, 5, 6, 19, 34, 24] to mention just a few of them). Despite chancroid being an old infection which is still affecting mankind no mathematical account of it have been carried out to the best knowledge of the authors. Here we attempt to give possibly a first mathematical account of chanroid infection dynamics taking into account one's sexual orientation (heterosexual or homosexual). In the next section the model is formulated and some of its properties illustrated. In Section 3 some analysis of the model is carried out and some numerical simulations are carried out in Section 4. Finally, a discussion of our findings is given at the end.

2.   Model Description

The model sub-divides the total population into the following sub-populations: susceptible straight (non-homosexuals) males $S_{m1}(t)$, susceptible homosexual males $S_{m2}(t)$, chancroid infected straight males $I_{m1}(t)$, chancroid infected homosexual males $I_{m2}(t)$, treated straight males $T_{m1}(t)$, treated homosexual males $T_{m2}(t)$, susceptible straight females $S_{f1}(t)$, susceptible lesbian $S_{f2}(t)$, chancroid infected straight females $I_{f1}(t)$, chancroid infected lesbian $I_{f2}(t)$, treated straight females $T_{f1}(t)$, treated lesbians $T_{f2}(t)$. The total population is given by $N(t) = N_m(t) + N_f(t)$ with

$$N_m(t) = N_{m1}(t) + N_{m2}(t), \ N_{m1}(t) = S_{m1}(t) + I_{m1}(t) + T_{m1}(t), \tag{1}$$

$$N_{m2}(t) = S_{m2}(t) + I_{m2}(t) + T_{m2}(t), \ N_f(t) = N_{f1}(t) + N_{f2}(t),$$

$$N_{f1}(t) = S_{f1}(t) + I_{f1}(t) + T_{f1}(t), \ N_{f2}(t) = S_{f2}(t) + I_{f2}(t) + T_{f2}(t).$$

with $N_m(t)$, $N_f(t)$, N_{m1}, N_{f1}, N_{m2}, and N_{f2} being the total number of males, females, straight males, straight females, homosexual males and lesbians, respectively. We assume that once an individual becomes a homosexual she (he) only egages in sexual encounters with women (men) only. Susceptible humans enter the population through birth at a rate Λ, a proportion ρ being straight males and the complementary proportion $(1 - \rho)$ entering the straight females population. Here, the authors assume that no individual is born being a homosexual (this is the authors' assumption). Susceptible straight males acquire chancroid infection following sexual contact with an infected straight female at a rate $\lambda_{f1}(t) = \frac{\beta_{f1}I_{f1}(t)}{N_f(t)}$ with β_{f1} being the effective contact rate for chancroid transmission from female to male. Susceptible homosexual males acquire chancroid infection at a rate $\lambda_{m2}(t) = \frac{\beta_{m2}I_{m2}(t)}{N_{m2}(t)}$ with β_{m2} being the effective contact rate for chancroid transmission from male to male during sexual intercourse. Susceptible straight females acquire chancroid infection following sexual intercourse with an infected straight male at a rate $\lambda_{m1}(t) = \frac{\beta_{m1}I_{m1}(t)}{N_m(t)}$ with β_{m1} being the effective contact rate for chancroid transmission from male to female. Susceptible lesbians acquire chancroid infection following sexual contact with an infected lesbian at a rate $\lambda_{f2}(t) = \frac{\beta_{f2}I_{f2}(t)}{N_{f2}(t)}$ with λ_{f2} being the effective contact rate for chancroid transmission from lesbian to lesbian during sexual intercourse. Upon getting infected the different susceptible groups move into the corresponding infected classes, respectively. It is worth mentioning that some straight men and straight women become homosexuals and lesbians at rates γ_m and γ_f, respectively, due to factors like: (i) a broken and stressful love relationship with a member of the opposite sex, (ii) peer pressure especially among teenagers among a host of other social factors. It is further assumed that once an individual becomes a homosexual he/she will only have sexual relations with members of the same sex as him/her (this is the authors' assumption). Infected individuals are treated at a rate α and move into the corresponding treated classes. Individuals who have been treated have some degree of immunity against re-infection with new H. ducreyi [28], so individuals in T_{m1}, T_{m2}, T_{f1}, and T_{f2} are infected by H. ducreyi at rates $\sigma_{f1}\lambda_{f1}$, $\sigma_{m2}\lambda_{m2}$, $\sigma_{m1}\lambda_{m1}$, and $\sigma_{f2}\lambda_{f2}$, respectively. Here, σ_{f1}, σ_{m1}, σ_{f2}, $\sigma_{m2} \in (0,1)$ accounting for the reduced rate of acquiring a new infection for those previously infected and successfully treated. Individuals experience natural death at a rate μ. Chancroid alone does not kill those suffering from it, so there is no disease induced death in this model. The model flow diagram is shown in Figure 1. Based on these assumptions the following system of differential equations describe the model.

$$S'_{m1}(t) = \rho\Lambda - \lambda_{f1}S_{m1} - (\mu + \gamma_m)S_{m1},$$

$$S'_{m2}(t) = \gamma_m S_{m1} - \lambda_{m2}S_{m2} - \mu S_{m2},$$

$$I'_{m1}(t) = \lambda_{f1}S_{m1} + \sigma_{f1}\lambda_{f1}T_{m1} - (\gamma_m + \alpha + \mu)I_{m1},$$

$$I'_{m2}(t) = \lambda_{m2}S_{m2} + \sigma_{m2}\lambda_{m2}T_{m2} + \gamma_m I_{m1} - (\alpha + \mu)I_{m2},$$

$$T'_{m1}(t) = \alpha I_{m1} - \sigma_{f1}\lambda_{f1}T_{m1} - (\mu + \gamma_m)T_{m1},$$

$$T'_{m2}(t) = \alpha I_{m2} + \gamma_m T_{m1} - \sigma_{m2}\lambda_{m2}T_{m2} - \mu T_{m2},$$

$$S'_{f1}(t) = (1 - \rho)\Gamma - \lambda_{m1}S_{f1} - (\mu + \gamma_f)S_{f1}, \tag{2}$$

$$S'_{f2}(t) = \gamma_f S_{f1} - \lambda_{f2}S_{f2} - \mu S_{f2},$$

$$I'_{f1}(t) = \lambda_{m1}S_{f1} + \sigma_{m1}\lambda_{m1}T_{f1} - (\gamma_f + \alpha + \mu)I_{f1},$$

$$I'_{f2}(t) = \lambda_{f2}S_{f2} + \sigma_{f2}\lambda_{f2}T_{f2} + \gamma_f I_{f1} - (\alpha + \mu)I_{f2},$$

$$T'_{f1}(t) = \alpha I_{f1} - \sigma_{m1}\lambda_{m1}T_{f1} - (\mu + \gamma_f)T_{f1},$$

$$T'_{f2}(t) = \alpha I_{f2} + \gamma_f T_{f1} - \sigma_{f2}\lambda_{f2}T_{f2} - \mu T_{f2}$$

Invariant Region The model system (2) will be analysed in a suitable region as follows. We first show that system (2) is dissipative that is all solutions are uniformly bounded in a proper subset $\Omega \subseteq \mathbb{R}^{12}_+$. Let,

$$(S_{m1}, S_{m2}, I_{m1}, I_{m2}, T_{m1}, T_{m2}, S_{f1}, S_{f2}, I_{f1}, I_{f2}, T_{f1}, T_{f2}) \in \mathbb{R}^{12}_+, \tag{3}$$

be any solution with non-negative initial conditions. Using a theorem on differential inequality [3] it follows that

$$\limsup_{t \to \infty}(S_{m1} + S_{m2} + S_{f1} + S_{f2}) \leqslant \frac{\Lambda}{\mu}. \tag{4}$$

Taking the derivative of N along a solution path of the system gives,

$$N'(t) = \Lambda - \mu N \tag{5}$$

Model system (2) has a varying population size $(N' \neq 0)$ only when the natural death rate is not the same as the natural birth rate. It follows (using Birkhoff and Rota [3]) that

$$0 \leqslant N(t) = \frac{\Lambda}{\mu} + \left(N(0) - \frac{\Lambda}{\mu}\right)e^{-\mu t} \leqslant \frac{\Lambda}{\mu} + N(0)e^{-\mu t} \tag{6}$$

where $N(0)$ represents the value of (2) evaluated at the initial values of the respective variables. Thus as $t \to \infty$

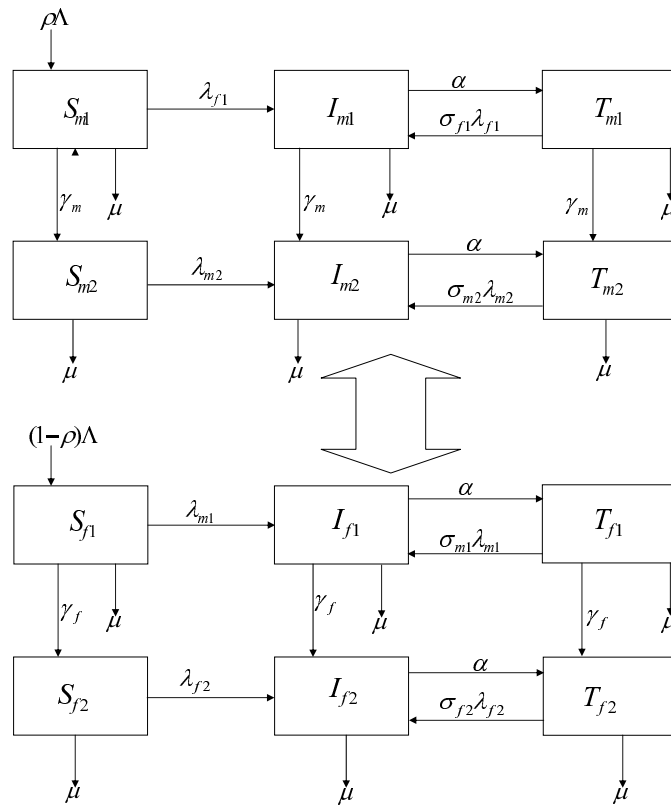$$0 \leqslant N \leqslant \frac{\Lambda}{\mu}. \tag{7}$$

Figure 1. Model structure.

Therefore, all feasible solutions of system (2) enter the region

$$\Omega = \left\{ (S_{m1}, S_{m2}, I_{m1}, I_{m2}, T_{m1}, T_{m2}, S_{f1}, S_{f2}, If1, I_{f2}, T_{f1}, T_{f2}) \in \mathbb{R}^{12}_{+} : N \leqslant \frac{\Lambda}{\mu} \right\}. \quad (8)$$

Thus, Ω is positively invariant and it is sufficient to consider solutions in Ω. Existence, uniqueness and continuation results for system (2) hold in this region. It can be shown that all solutions of model system (2) in Ω remain in Ω for all $t \geqslant 0$. All parameters and state variables for model system (2) are assumed to be non-negative for $t \geqslant 0$ since it monitors human population.

3. Analytical results

3.1 *Disease-Free Equilibrium (DFE)*

System (2) has an evident disease-free equilibrium (DFE) is given by

$$\mathcal{E}_0 = \left(S^0_{m_1}, S^0_{m_2}, I^0_{m_1}, I^0_{m_2}, T^0_{m_1}, T^0_{m_2}, S^0_{f_1}, S^0_{f_2}, I^0_{f_1}, I^0_{f_2}, T^0_{f_1}, T^0_{f_2} \right)$$

$$= \left(\frac{\rho\Lambda}{\mu + \gamma_m}, \frac{\gamma_m \rho\Lambda}{\mu(\mu + \gamma_m)}, 0, 0, 0, 0, \frac{(1-\rho)\Lambda}{\mu + \gamma_f}, \frac{\gamma_f(1-\rho)\Lambda}{\mu(\mu + \gamma_f)}, 0, 0, 0, 0 \right). \quad (9)$$

It can be shown that $\mathcal{E}_0$ attracts the region

$$\Omega_0 = \{(S_{m_1}, S_{m_2}, I_{m_1}, I_{m_2}, S_{f_1}, S_{f_2}, I_{f_1}, I_{f_2}) \in \Omega : \tag{10}$$
$$I_{m_1} = I_{m_2} = T_{m_1} = T_{m_2} = I_{f_1} = I_{f_2} = T_{f_1} = T_{f_2} = 0\}.$$

3.2 Reproductive Number

Following van den Driessche and Watmough [32], the reproduction number of the model system (2) is found as

$$\mathcal{R}_c = \max\{\mathcal{R}_{m_2}, \mathcal{R}_{f_m}, \mathcal{R}_{f_2}\} \tag{11}$$
$$= \left\{\frac{\beta_{m_2}}{\mu + \alpha}, \sqrt{\frac{\mu^2 \beta_{m_1} \beta_{f_1}}{(\gamma_m + \mu + \alpha)(\gamma_f + \mu + \alpha)(\gamma_m + \mu)(\gamma_f + \mu)}}, \frac{\beta_{f_2}}{\mu + \alpha}\right\}$$

with $\mathcal{R}_{m_2}$ being the number of secondary chancroid infections caused by one infected male homosexual in fully susceptible population of male homosexuals in the presence of treatment, $\mathcal{R}_{f_m}$ being the number of secondary chancroid infections caused by one infected male (non-homosexual) or female (non-lesbian) in a fully susceptible heterosexual population in the presence of treatment, $\mathcal{R}_{f_2}$ being the number of secondary chancroid infections caused by one infected lesbian in a fully susceptible population of lesbians. Theorem 3.1 follows from van den Driessche and Watmough [32] (Theorem 2).

THEOREM 3.1 *The disease-free equilibrium point $\mathcal{E}_0$ is locally asymptotically stable for $\mathcal{R}_c < 1$ and unstable otherwise.*

Using a theorem from Castillo-Chavez *et al.* [8], we show global stability when the reproduction number is less than unity.

THEOREM 3.2 *The disease-free equilibrium of system (2) is globally asymptotically stable provided $\mathcal{R}_c < 1$.*

Proof Following Castillo-Chavez *et al.* [8], we write system (2) in the form

$$X'(t) = F(X, Y) \tag{12}$$
$$Y'(t) = G(X, Y), \ G(X, 0) = 0$$

where $X = (S_{m_1}, S_{m_2}, S_{f_1}, S_{f_2})$ and $Y = (I_{m_1}, I_{m_2}, I_{f_1}, I_{f_2})$. Here, $X \in \mathbb{R}_+^4$ denotes (its components) the number of uninfected individuals and $Y \in \mathbb{R}_+^4$ denotes (its components) the number of infected individuals. The disease-free equilibrium is now denoted by $\mathcal{E}_0 = (\mathbf{X}_0, 0)$ where

$$\mathbf{X}_0 = \left(\frac{\rho\Lambda}{\mu + \gamma_m}, \frac{\gamma_m \rho\Lambda}{\mu(\mu + \gamma_m)}, \frac{(1-\rho)\Lambda}{\mu + \gamma_f}, \frac{\gamma_f(1-\rho)\Lambda}{\mu(\mu + \gamma_f)}\right).$$

Here, we have to prove that the two conditions

$(H1)$ For $X'(t) = F(X,0)$, X_0 is globally asymptotically stable

$(H2)$ $G(X,Y) = UY - \widehat{G}(X,Y)$, $\widehat{G}(X,Y) \geqslant 0$, for $(X,Y) \in \Omega$.

(13)

Consider $F(X,0) = \begin{bmatrix} \rho\Lambda - (\mu + \gamma_m)S_{m_1} \\ \gamma_m S_{m_1} - \mu S_{m_2} \\ (1-\rho)\Lambda - (\mu + \gamma_f)S_{f_1} \\ \gamma_f S_{f_1} - \mu S_{f_2} \end{bmatrix}$,

$$U = \begin{bmatrix} -(\mu + \gamma_m + \alpha) & 0 & \dfrac{\beta_{f_1}\rho\mu}{(\mu + \gamma_m)(1-\rho)} & 0 \\[2ex] \gamma_m & \beta_{m_2} - (\mu + \alpha) & 0 & 0 \\[2ex] \dfrac{\beta_{m_1}\mu(1-\rho)}{\rho(\mu + \gamma_f)} & 0 & -(\mu + \gamma_f + \alpha) & 0 \\[2ex] 0 & 0 & \gamma_f & \beta_{f_2} - (\mu + \alpha) \end{bmatrix}$$

and $\widehat{G}(X,Y) = \begin{bmatrix} \widehat{G_1}(X,Y) \\ \widehat{G_2}(X,Y) \\ \widehat{G_3}(X,Y) \\ \widehat{G_4}(X,Y) \end{bmatrix}$

$$= \begin{bmatrix} \beta_{f_1}I_{f_1}\left(\dfrac{\rho\mu}{(\mu + \gamma_m)(1-\rho)} - \dfrac{S_{m_1}}{N_f}\right) \\[3ex] \beta_{m_2}I_{m_2}\left(1 - \dfrac{S_{m_2}}{N_{m_2}}\right) \\[3ex] \beta_{m_1}I_{m_1}\left(\dfrac{(1-\rho)\mu}{(\mu + \gamma_f)\rho} - \dfrac{S_{f_1}}{N_m}\right) \\[3ex] \beta_{f_2}I_{f_2}\left(1 - \dfrac{S_{f_2}}{N_{f_2}}\right) \end{bmatrix}.$$

(14)

Clearly, $\widehat{G_2}(X,Y)$ and $\widehat{G_4}(X,Y)$ are positive. Now we have to show that $\widehat{G_1}(X,Y)$ and $\widehat{G_3}(X,Y)$ are both positive. Showing that $\widehat{G_1}(X,Y)$ and $\widehat{G_3}(X,Y)$ are both positive starting with $\widehat{G_1}(X,Y)$.

$$\widehat{G_1}(X,Y) = \beta_{f_1}I_{f_1}\left(\frac{\rho\mu}{(\mu + \gamma_m)(1-\rho)} - \frac{S_{m_1}}{N_f}\right) \geqslant \frac{\beta_{f_1}I_{f_1}\rho}{\mu + \gamma_m}\left(\frac{\mu}{1-\rho} - \frac{\Lambda}{N_f}\right)$$

(15)

$\left(\text{since } S_{m_1} \leqslant S_{m_1}^0 = \dfrac{\rho\Lambda}{\mu + \gamma_m}\right).$

Since $N_f \approx \dfrac{(1-\rho)\Lambda}{\mu}$ as $t \to \infty$, then from equation (15) we have

$$\frac{\beta_{f_1} I_{f_1} \rho}{\mu + \gamma_m} \left(\frac{\mu}{1-\rho} - \frac{\Lambda}{N_f} \right) \approx 0. \tag{16}$$

Hence, $\widehat{G_1}(X,Y) \geqslant 0$. Now we have to show that $\widehat{G_3}(X,Y)$ is positive too.

$$\widehat{G_3}(X,Y) = \beta_{m_1} I_{m_1} \left(\frac{(1-\rho)\mu}{(\mu+\gamma_f)\rho} - \frac{S_{f_1}}{N_m} \right) \geqslant \frac{\beta_{m_1} I_{m_1}(1-\rho)}{\mu+\gamma_f} \left(\frac{\mu}{\rho} - \frac{\Lambda}{N_m} \right) \tag{17}$$

$$\left(\text{since } S_{f_1} \leqslant S_{f_1}^0 = \frac{(1-\rho)\Lambda}{\mu+\gamma_f} \right).$$

Since $N_m \approx \dfrac{\rho\Lambda}{\mu}$ as $t \to \infty$, then from equation (17) we have

$$\frac{\beta_{m_1} I_{m_1}(1-\rho)}{\mu+\gamma_f} \left(\frac{\mu}{\rho} - \frac{\Lambda}{N_m} \right) \approx 0. \tag{18}$$

Hence, $\widehat{G_3}(X,Y) \geqslant 0$. Thus, $\widehat{G}(X,Y) \geqslant 0$. Therefore the disease-free equilibrium $\mathcal{E}_0$ is globally asymptotically stable. ∎

3.3   *Endemic Equilibrium*

System (2) has an endemic equilibrium given by (not presented here, due to its complex nature)

$$\mathcal{E}_* = \left(S_{m_1}^*, S_{m_2}^*, I_{m_1}^*, I_{m_2}^*, T_{m_1}^*, T_{m_2}^*, S_{f_1}^*, S_{f_2}^*, I_{f_1}^*, I_{f_2}^*, T_{f_1}^*, T_{f_2}^* \right). \tag{19}$$

We claim the following result

THEOREM 3.3 *The endemic equilibrium $\mathcal{E}_*$ is globally asymptotically stable for $\mathcal{R}_c > 1$ and unstable otherwise.*

Proof To prove Theorem 3.3, we assume that the entire population consists of homosexual only i.e S_{m_2}, I_{m_2}, and T_{m_2} (for simplicity), hence we have the following system

$$S'_{m_2} = -\frac{\beta_{m_2} I_{m_2} S_{m_2}}{N_{m_2}} - \mu S_{m_2},$$

$$I'_{m_2} = \frac{\beta_{m_2} I_{m_2}(S_{m_2} + \sigma_{m_2} T_{m_2})}{N_{m_2}} - (\alpha + \mu)I_{m_2}, \tag{20}$$

$$T'_{m_2} = \alpha I_{m_2} - \frac{\sigma_{m_2} \beta_{m_2} I_{m_2} T_{m_2}}{N_{m_2}} - \mu T_{m_2}.$$

Dividing system (20), by N_{m_2} we have

$$s'_{m_2} = -\beta_{m_2} i_{m_2} s_{m_2} - \mu s_{m_2},$$

$$i'_{m_2} = \beta_{m_2} i_{m_2}(s_{m_2} + \sigma_{m_2} t_{m_2}) - (\alpha + \mu)i_{m_2}, \tag{21}$$

$$t'_{m_2} = \alpha i_{m_2} - \sigma_{m_2} \beta_{m_2} i_{m_2} t_{m_2} - \mu t_{m_2},$$

so that $s_{m_2} + i_{m_2} + t_{m_2} \leqslant 1$. Furthermore; $s_{m_2} = 1 - i_{m_2} - t_{m_2}$. Thus, System (21) reduces to

$$i'_{m_2} = \beta_{m_2} i_{m_2}[1 - i_{m_2} - (1 - \sigma_{m_2})t_{m_2}] - (\alpha + \mu)i_{m_2},$$

$$t'_{m_2} = \alpha i_{m_2} - \sigma_{m_2}\beta_{m_2} i_{m_2} t_{m_2} - \mu t_{m_2}. \tag{22}$$

Denote the right-hand side of (22) by (f, g) and choose a Dulac *function* as $D(i_{m_2}, t_{m_2}) = 1/i_{m_2}t_{m_2}$. Then we have

$$\frac{\partial(Df)}{\partial i_{m_2}} + \frac{\partial(Dg)}{\partial t_{m_2}} = -\frac{(\alpha + \beta t_{m_2})}{t_{m_2}^2} < 0, \tag{23}$$

Hence, no limit cycles exist and the non-trivial steady state is globally stable for $\mathcal{R}_c > 1$. ∎

3.4 *Analysis of the Reproduction Number*

In many epidemiological models, the magnitude of the reproductive number is associated with the level of infection. The same is true in model (2). Here, we investigate the impact of chancroid infections among heterosexuals and homosexuals. To effectively do that requires us to express $\beta_{m_2} = A_m\beta_{f_1}$ with $A_m = 1$ signifying that rate of males is the same for homosexuals and heterosexuals; $A_m \in (0, 1)$ signifying a reduced rate of acquiring chancroid for male homosexuals when compared to their heterosexual counterparts; $A_m > 1$ signifies an increased rate of acquiring chancroid for male homosexuals when compared to their counterparts (heterosexuals). Similarly assume $\beta_{f_2} = A_f\beta_{m_1}$ with $A_f = 1$, $A_f \in (0, 1)$ or $A_f > 1$, taking the explanations similar to A_m as defined earlier. With that in mind we can now express $\frac{\beta_{f_1}}{\gamma_m + \alpha + \mu} = B_1\mathcal{R}_{m_2}$, $B_1 = \frac{\mu + \alpha}{A_m(\mu + \alpha + \gamma_m)}$ and $\frac{\beta_{m_1}}{\gamma_f + \alpha + \mu} = B_2\mathcal{R}_{f_2}$, $B_2 = \frac{\mu + \alpha}{A_f(\mu + \alpha + \gamma_f)}$ so that

$$\mathcal{R}_{f_m} = \sqrt{\left(\frac{(\mu + \alpha)\mathcal{R}_{m_2}}{(\mu + \alpha + \gamma_m)A_m}\right)\left(\frac{(\mu + \alpha)\mathcal{R}_{f_2}}{(\mu + \alpha + \gamma_f)A_f}\right)\left(\frac{\mu}{\mu + \gamma_m}\right)\left(\frac{\mu}{\mu + \gamma_f}\right)}. \tag{24}$$

Partially differentiating $\mathcal{R}_{m_2}$ and $\mathcal{R}_{f_2}$ with respect to $\mathcal{R}_{f_m}$ we have:

$$\frac{\partial\mathcal{R}_{m_2}}{\partial\mathcal{R}_{f_m}} = \frac{2\mathcal{R}_{m_2}}{\mathcal{R}_{f_m}} > 0 \text{ and } \frac{\partial\mathcal{R}_{f_2}}{\partial\mathcal{R}_{f_m}} = \frac{2\mathcal{R}_{f_2}}{\mathcal{R}_{f_m}} > 0, \tag{25}$$

respectively. The fact that both equations in (25) are positive suggests that increasing in chancroid infections among heterosexuals result in an increase of the cases amongst homosexuals. This could be a result of people becoming homosexuals already being chancroid infected. This result suggests targeting heterosexuals in chancroid infection control may have a positive impact among homosexuals. Now, we use Latin Hypercube Sampling (LHS) technique to perform sensitivity analysis of the reproductive number. Sensitivity analysis assesses the amount and type of change inherent in the model as captured by the terms that define the reproductive number ($\mathcal{R}_c$). If $\mathcal{R}_c$ is very sensitive to a particular parameter, then a perturbation of the conditions that connect the dynamics to such a parameter may prove useful in identifying policies or intervention strategies that reduce epidemic prevalence.
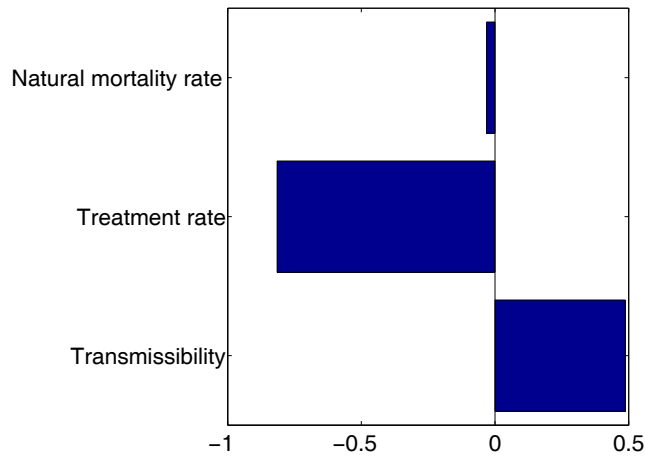
Figure 2. Sensitivity of $\mathcal{R}_c$ to all parameters using partial rank correlation coefficients.

Partial rank correlation coefficients (PRCC) were calculated to estimate the of correlation between values of $\mathcal{R}_c$ and the three model parameters (which define $\mathcal{R}_{m_2}$ and $\mathcal{R}_{f_2}$) across 10^3 random draws from the empirical distribution of $\mathcal{R}_c$ and its associated parameters. A large PRCC is indicative of high sensitivity to parameter estimates (PRCCs > 0 will increase $\mathcal{R}_c$ when they are increased), while a small PRCC is reflects low sensitivity (PRCCs < 0 will decrease $\mathcal{R}_c$ when they are increased) [33-36]. Figure 2 illustrates that $\mathcal{R}_c$ is most sensitive to treatment rate, clearly suggesting that individuals infected with chancroid should be encourage to seek treatment so that chancroid prevalence can be reduced. Since the treatment parameter has a significant effect on $\mathcal{R}_c = (\mathcal{R}_{m_2}, \mathcal{R}_{f_2})$, we examined the dependence of $\mathcal{R}_c$ in this parameter in more detail. We used Latin Hypercube Sampling and Monte Carlo simulations to run 1000 simulations, where all parameters were simultaneously drawn from across their ranges.
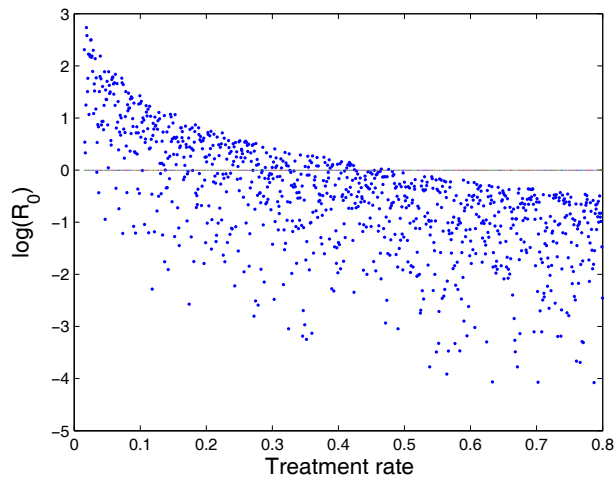

Figure 3. Latin Hypercube Sampling for varying treatment rates. If treatment rate is greater than 40%, then the disease can be controlled.

Figure 3 illustrates the effect of treatment on controlling chancroid prevalence. The results demonstrates that an increase in α results in a decrease on the reproductive ratio. The results demonstrates that maintaining high level of chancroid infectives

who seek treatment may be an important intervention strategy for controlling chancroid in the community. Furthermore, Figure 3 demonstrates that if treatment rate is more than 40% then the reproductive number is less than unity, then the disease will be controlled. Similar analysis can be done if $\mathcal{R}_c = \mathcal{R}_{fm}$, using LHS.

4. Discussion

Chancroid is a sexually transmitted genital ulcer disease (GUD) caused by the gram-negative bacillus Haemophilus ducreyi. Chancroid is characterized by the presence of painful ulcers and inflammatory inguinal adenopathy. Successful treatment for chancroid cures the infection, resolves the clinical symptoms, and prevents transmission to others. A deterministic mathematical model for assessing the role of treatment on controlling chancroid transmission dynamics is formulated and comprehensively analyzed. A dimensionless expression known as the reproductive number has been derived and qualitatively used to investigate the stability of equilibrium states, and chancroid prevalence in the presence of treatment. Analytical results have shown that the model has a globally-stable disease-free equilibrium and a globally-stable endemic equilibrium whenever the reproductive number is less and greater than unity, respectively. Furthermore, results from the study confirm that successful chancroid treatment is crucial for reducing epidemic prevalence. However, it is worth noting that the study has highlighted that for total control of chancroid, the rate of treatment should be above 40%, hence chancroid infectives should be encouraged to seek treatment, since chancroid is a known cofactor in HIV transmission.

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