Transmission dynamics of *Trichomonas vaginalis*: A mathematical approach

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**Abstract**

Despite the availability of treatment that is effective, *Trichomonas vaginalis* infections are still high. A deterministic model for transmission dynamics of *Trichomonas vaginalis* is presented as a system of non-linear differential equations. Analysis of the reproduction number has shown that an increase in the number of straight women (non-lesbians) infected result in an increase in the number of lesbians infected. This suggests that straight women are turning into lesbians already infected. The disease-free equilibrium is shown to be globally asymptotically stable when the corresponding reproduction number is less than unity. Analytical results and numerical simulations both show that treatment is able to control *Trichomonas vaginalis* infections. This suggests an effective control of trichomoniasis rests in encouraging and persuading sexual partners of those displaying symptoms to seek treatment. Failure for the asymptomatic to seek treatment (mostly males given that the majority of males does not show symptoms) will continue to fuel the infection.

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1. Introduction

*Trichomonas vaginalis* also called trichomoniais is one of the commonest sexually transmitted pathogens in the world with an estimated 174 million cases occurring each year [33]. It is a far more prevalent sexually transmitted infection than either *Chlamydia trachomatis* or *Neisseria gonorrhoeae*, yet in stark contrast little attention is paid to trichomoniasis [26]. In the United States, it was recently estimated that 5 million new cases appear annually [27,33,7,10] compared with 3 million cases of chlamydia and 650 000 cases of gonorrhea annually. The epidemiology of the disease is still poorly understood and some practitioners continue to question its importance [3]. *Trichomonas vaginalis* is more prevalent in females (67%–100% female sexual partners of infected man get infected) than in males (14%–60% of male sexual partners of an infected woman get infected) [1,16,13]. The reason why females are more infected is poorly understood, although some researchers [17,9] think the prostatic fluid contains zinc and other substances harmful to the pathogen.

In females trichomoniasis is associated with vaginal discharge, vaginal itching, pain when urinating and up to one-third of women do not show symptoms [15]. Although most males do not show symptoms related to *Trichomonas vaginalis* infection, some do experience urethral discharge, pain when urinating and swelling of the scrotum [15]. Usually treatment consists of metronidazole and tinidazole. Left untreated chronic *Trichomonas vaginalis* infections may lead to complications including; pelvic inflammatory disease, premature rapture of membranes, low-birth-weight infants, pre-term delivery and abortion [29,21,5]. Cudmore and Garber [8] showed that the development of a vaccine against *Trichomonas vaginalis* could reduce the human costs (pregnancy complications, infertility), medical costs and increased susceptibility to HIV and societal
costs. It has been shown that *Trichomonas vaginalis* infections lead to an increase in the risk of transmitting HIV two-fold [18,11,21,14].

Malaria, Tuberculosis, Hepatitis B, HIV/AIDS and other sexually transmitted infections have all being mathematically accounted for (see [28,2,24,31,22,25] to mention just a few of them). Quite recently some authors [34,19] did analyse some general SIR and SEIRS models with interesting dynamics. The later [19] did analyse SEIRS model incorporating multiple infectious stages. Despite *Trichomonas vaginalis* being an old infection which is still affecting mankind no mathematical account of it has been carried out to the best knowledge of the authors. Here we attempt to give mathematical analysis of it taking into consideration effects of differential infectivity patterns between males and females as well as between lesbians and non-lesbians. To the best of the author this may be the first manuscript to look into these aspects. The rest of the manuscript is organised as follows: In the next section we describe and present the model. 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 males \( S_m(t) \), infected males \( I_m(t) \), susceptible straight women \( S_f(t) \), infected straight women \( I_f(t) \), susceptible lesbians \( S_{f2}(t) \) and infected lesbians \( I_{f2}(t) \). The total population is given by

\[
N(t) = N_m(t) + N_{f1}(t) + N_{f2}(t), \quad N_m(t) = S_m(t) + I_m(t), \quad N_{f1}(t) = S_{f1}(t) + I_{f1}(t), \quad N_{f2}(t) = S_{f2}(t) + I_{f2}(t) \tag{1}
\]

with \( N_m(t) \), \( N_{f1}(t) \) and \( N_{f2}(t) \) being the total number of males, straight females and lesbians, respectively. We assume that once a woman becomes a lesbian she only engages in sexual encounters with women only. Susceptible humans enter the population through birth at a rate \( \Lambda \), a proportion \( \rho \) being males and the complementary proportion \( (1 - \rho) \) entering the straight females population. Here we further assume no woman is born as a lesbian (this is the authors’ assumption). Susceptible males acquire *Trichomonas vaginalis* following sexual contact with infected straight female at a rate \( \lambda_{f1}(t) = \frac{\beta_{f1}I_{f1}(t)}{N_{f1}(t)} \) where \( \beta_{f1} \) is the effective contact rate for *Trichomonas vaginalis* transmission from female to male. Susceptible straight females acquire *Trichomonas vaginalis* following sexual contact with an infected male at a rate \( \lambda_{m}(t) = \frac{\beta_{m}I_{m}(t)}{N_{m}(t)} \) where \( \beta_{m} \) is the effective contact rate for *Trichomonas vaginalis* transmission from male to female. Lesbian females acquire *Trichomonas vaginalis* following sexual contact with an infected lesbian where there is vulva to vulva sexual contact [20] or through sharing unclean sexual toys at a rate \( \lambda_{f2}(t) = \frac{\beta_{f2}I_{f2}(t)}{N_{f2}(t)} \) where \( \beta_{f2} \) is the effective contact rate for *Trichomonas vaginalis* transmission from lesbian to lesbian. The case of male to male sex transmitting *Trichomonas vaginalis* is not documented meaning they are non-existent as far as the current literature is concerned. Upon getting infected the different susceptible groups move into the corresponding infected classes, respectively. It is worth mentioning that some straight women become lesbians at a rate \( \alpha \) due to a number of 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. Infected individuals are treated at a rate \( \gamma \) and move back into their corresponding susceptible classes. Individuals experience natural death at a rate \( \mu \). Trichomoniassis illness alone does not kill those suffering from it, so there is no disease induced death in this model. Based on these assumptions the following system of differential equations describes the model:

\[
\begin{align*}
S_m'(t) &= \rho \Lambda - \mu S_m - \lambda_{f1} S_m + \gamma I_m, \\
I_m'(t) &= \lambda_{f1} S_m - (\mu + \gamma) I_m, \\
S_{f1}'(t) &= (1 - \rho) \Lambda - (\mu + \alpha) S_{f1} - \lambda_{m} S_{f1} + \gamma I_{f1}, \\
I_{f1}'(t) &= \lambda_{m} S_{f1} - (\mu + \alpha + \gamma) I_{f1}, \\
S_{f2}'(t) &= \alpha S_{f1} - \mu S_{f2} - \lambda_{f2} S_{f2} + \gamma I_{f2}, \\
I_{f2}'(t) &= \lambda_{f2} S_{f2} + \alpha I_{f1} - (\mu + \gamma) I_{f2}.
\end{align*}
\tag{2}
\]

The model flow diagram is shown in Fig. 1.

2.1. Model basic properties

In this section, we study the basic results of solutions of model system (2), which are essential in the proofs of stability results.

**Lemma 1.** The equations preserve positivity of solutions.
**Proof.** The vector field given by the right-hand side of (2) points inward on the boundary of $\mathbb{R}^6_+ \setminus \{0\}$. For example, if $S_m = 0$ then $S_m' = \rho \Lambda + \gamma I_m \geq 0$. All the other components are similar. \qed

**Lemma 2.** Each non-negative solution is bounded in $L^1$-norm by $\max\{N(0), \Lambda/\mu\}$.

**Proof.** The norm $L^1$-norm of each non-negative solution is $N$ and it satisfies the inequality $N' \leq \Lambda - \mu N$. Solutions to the equation $M' = \Lambda - \mu M$ are monotone increasing and bounded by $\Lambda/\mu$ if $M(0) < \Lambda/\mu$. They are monotone decreasing and bounded above if $M(0) \geq \Lambda/\mu$. Since $N' \leq M'$ the claim follows. \qed

**Corollary 1.** The region

$$\Phi = \{(S_m, I_m, S_{f1}, I_{f1}, S_{f2}, I_{f2}) \in \mathbb{R}^6_+: N \leq \frac{\Lambda}{\mu}\}$$

is invariant and attracting for system (2).

**Theorem 1.** For every non-zero, non-negative initial value, solutions of model system (2) exist for all times.

**Proof.** Local existence of solutions follows from standard arguments since the right-hand side of (2) is locally Lipschitz. Global existence follows from the a priori bounds. \qed

### 3. Disease-free equilibrium and stability analysis

The disease-free equilibrium of model system (2), $E^0$ is given by

$$E^0 = \left(\frac{\rho \Lambda}{\mu}, 0, \frac{(1 - \rho) \Lambda}{\mu + \alpha}, 0, \frac{(1 - \rho) \Lambda \alpha}{\mu (\mu + \alpha)}, 0\right).$$

(4)

Following [30], the effective reproduction number of model system (2) is given as

$$R_T = \max\{R_L, R_N\} = \max\left\{\frac{\beta_f}{\mu + \gamma}, \sqrt{\frac{\beta_m \beta_f}{(\mu + \gamma)(\mu + \gamma + \alpha)}}, \frac{\beta_f}{\mu + \gamma}\right\}$$

(5)

with $R_L$ being the number of secondary *Trichomonas vaginalis* infections caused by one infected lesbian in fully susceptible population of lesbians in the presence of treatment and $R_N$ being the number of secondary *Trichomonas vaginalis* infections caused by one infected male or female (non-lesbian) in a fully susceptible heterosexual population in the presence of treatment. We now offer some analysis of the reproduction number. To that we have to know that the danger posed by vagina to vagina contact is not the same as the one posed by penis to vagina penetration so $\beta_f = A \beta_m$, $A \in (0, 1)$. The later poses more risky, that is, heterosexual females are at an increased risk of getting infected than the lesbians. Thus, we write
Fig. 2. Simulation results showing the relationship between the lesbian related reproduction number and the non-lesbian related reproduction number. Parameter values used are in Table 2.

\[ R_N = \sqrt{\frac{\beta_m \beta f_1}{(\mu + \gamma)(\mu + \gamma + \alpha)}} = \frac{\beta f_1 R_L}{A(\mu + \gamma + \alpha)} \Rightarrow \frac{\partial R_L}{\partial R_N} = \frac{2AR_N(\mu + \gamma + \alpha)}{\beta f_1} > 0. \] (6)

The fact that Eq. (6) is positive suggests that an increase in *Trichomonas vaginalis* cases among non-lesbiens in the long term translates to its increase among lesbians. This may be due to straight women (non-lesbians) becoming lesbians already infected. This is further illustrated in Fig. 2.

In Fig. 3 different infection and treatment rates are analysed in an effort to get the ideal treatment rate for a particular rate of infection.

Theorem 2 follows from [30].
Theorem 2. The disease-free equilibrium $E^0$ of model system (2) is locally asymptotically stable if $R_{S_D} < 1$ and unstable otherwise.

Using a theorem from [6], we show global stability when the reproduction number is less than unity.

Theorem 3. The disease-free equilibrium of system (2) is globally asymptotically stable provided $R_T < 1$.

Proof. Following Castillo-Chavez et al. [6], we write system (2) in the form

$$X'(t) = F(X, Y),$$
$$Y'(t) = G(X, Y), \quad G(X, 0) = 0$$

where $X = (S_m, f_1, f_2)$ and $Y = (I_m, f_1, f_2)$. Here, $X \in \mathbb{R}^3_+$ denotes (its components) the number of uninfected individuals and $Y \in \mathbb{R}^3_+$ denotes (its components) the number of infected individuals. The disease-free equilibrium is now denoted by $E^0 = (X_0, 0)$ where $X_0 = \left(\frac{\rho}{\mu}, \frac{\Lambda}{\mu}, \frac{\Lambda(1-\rho)}{\mu+\alpha}\right)$. Here, we have to prove the two conditions:

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

(H2) $G(X, Y) = N f - \hat{G}(X, Y), \quad \hat{G}(X, Y) \geq 0, \quad \text{for } (X, Y) \in \Phi.$

Consider

$$F(X, 0) = \begin{bmatrix}
\rho \Lambda - \mu S_m \\
\alpha S f_1 - \mu S f_2 \\
\end{bmatrix},$$

$$U = \begin{bmatrix}
-\mu + \gamma & \frac{\beta f_1 \rho(\mu+\alpha)}{\mu(1-\mu)} & 0 \\
\frac{\beta m \mu(1-\rho)}{\rho(\mu+\alpha)} & -\mu + \gamma + \alpha & 0 \\
\alpha & \beta f_2 - (\mu + \gamma) & \\
\end{bmatrix}$$

and

$$\hat{G}(X, Y) = \begin{bmatrix}
\hat{G}_1(X, Y) \\
\hat{G}_2(X, Y) \\
\hat{G}_3(X, Y)
\end{bmatrix} = \begin{bmatrix}
\beta f_1 f_1 \rho(\mu+\alpha) & -\frac{S_m}{N f_1} \\
\beta m f_1 f_2 \rho(\mu+\alpha) & -\frac{S f_1}{N f_2} \\
\beta f_2 f_2 \rho(\mu+\alpha) & -\frac{S f_2}{N f_2}
\end{bmatrix}.$$ (9)

Clearly $\hat{G}_3(X, Y)$ is positive. Now we have to show that $\hat{G}_1(X, Y)$ and $\hat{G}_2(X, Y)$ are both positive. To do this we prove by contradiction. Assume that statements in (10) are true

(i) $\frac{\rho(\mu+\alpha)}{\mu(1-\rho)} < \frac{S_m}{N f_1}$

and (ii) $\frac{\mu(1-\rho)}{\rho(\mu+\alpha)} < \frac{S f_1}{N f_2}.$

From statement (i) in (10) we have that $\frac{N f_1}{N m} < \frac{\mu(1-\rho)}{\rho(\mu+\alpha)}$. Combining this with statement (ii) in (10) we have

$$\frac{N f_1}{S_m} < \frac{\mu(1-\rho)}{\rho(\mu+\alpha)} \frac{S f_1}{N m} \quad \Rightarrow \quad \frac{N f_1}{S_m} < \frac{S f_1}{N m} \quad \Rightarrow \quad N f_1 N_m < S f_1 S_m.$$ (11)

a contradiction as statement (11) is not true. Thus,

$$N f_1 N_m \geq S f_1 S_m \quad \Rightarrow \quad \frac{\rho(\mu+\alpha)}{\mu(1-\rho)} \geq \frac{S_m}{N f_1} \quad \text{and} \quad \frac{\mu(1-\rho)}{\rho(\mu+\alpha)} \geq \frac{S f_1}{N f_2}.$$ (12)

Thus, $\hat{G}(X, Y) \geq 0$. Therefore the disease-free equilibrium $E^0$ is globally asymptotically stable. \hfill \Box

3.1. Existence and uniqueness of endemic equilibrium

The endemic equilibrium of model system (2) is given by $E^e = (S_m^*, f_1^*, f_2^*, I_m^*, f_1^*, f_2^*)$ in terms of the forces of infection $(\lambda_m^*, \lambda_{f_1}^*, \lambda_{f_2}^*)$ where

$$S_m^* = \frac{\rho \Lambda (\mu+\gamma)}{\mu(\mu+\gamma+\lambda_{f_1})}, \quad I_m^* = \frac{\rho \Lambda}{\mu(\mu+\gamma+\lambda_{f_1})}, \quad N_m^* = \frac{\rho \Lambda \mu}{\mu}, \quad N_{f_1}^* = \frac{(1-\rho) \Lambda}{\mu+\alpha},$$

$$f_1^* = \frac{\Lambda (1-\rho)(\mu+\gamma)}{\mu+\gamma+\alpha}, \quad I_{f_1}^* = \frac{\Lambda (1-\rho) \lambda_{f_1}^*}{\mu+\gamma+\alpha}.$$ (13)

$$S_{f_1}^* = \frac{f_2^* (\mu+\gamma+\alpha)(\mu+\alpha)+\lambda_{f_2}^*(\mu+\alpha)}{\mu+\gamma+\alpha}.$$ (14)
why does the world still experiencing the existence of asymptotically negligible levels signifying that treatment is able to control and female infectives increase until they reach their corresponding endemic equilibrium state. In the presence of treatment

\[ \mathcal{R}_L > 1 \] and \( \mathcal{R}_L < 1 \).

It is worth mentioning that \( \lambda \) and \( \mu \) are positive. Substituting Eq. (15) into the equation for the force of infection \( \lambda^*_m \) we obtain

\[ \lambda^*_m = \frac{-\mu^*}{\mu + \gamma + \lambda^*_m}. \] (16)

Substituting Eq. (14) into the equation of the force of infection \( \lambda^*_f_1 = \frac{\beta_{f_1} \gamma}{\mu + \alpha + \lambda^*_m} \) we obtain

\[ \lambda^*_f_1 = \frac{\beta_{f_1} \gamma}{\mu + \gamma + \lambda^*_m}. \] (17)

Substituting (16) into (17) we obtain

\[ \lambda^*_f_1 h(\lambda^*_f_1) = \lambda^*_f_1 (A \lambda^*_f_1 + 1 - \mathcal{R}_N^2) = 0, \]

\[ A = \mu + \gamma + \alpha + \lambda^*_m. \] (18)

In Eq. (18) \( \lambda^*_f_1 = 0 \) corresponds to the disease-free equilibrium and \( \lambda^*_f_1 = \frac{\mathcal{R}_N^2 - 1}{A} \) and this exists when \( \lambda^*_f_1 > 0 \Rightarrow \mathcal{R}_N^2 > 1 \Rightarrow \mathcal{R}_N > 1 \) since the reproduction number is always positive. Substituting Eq. (15) into the equation for the force of infection \( \lambda^*_f_2 = \frac{\beta_{f_2} \gamma}{\mu + \alpha + \lambda^*_m} \) we obtain

\[ g(\lambda^*_f_2) = A_1 \lambda^*_f_2^2 + B_1 \lambda^*_f_2 + C_1 = 0 \quad \text{where} \]

\[ A_1 = \frac{1}{\mu + \gamma}, \quad B_1 = 1 - \mathcal{R}_L, \quad C_1 = -\frac{\beta_{f_2} \mu \lambda^*_m}{(\mu + \gamma)(\mu + \gamma + \alpha + \lambda^*_m)}. \] (19)

It is worth mentioning that \( \lambda^*_m \) like \( \lambda^*_f_1 \) exists only when \( \mathcal{R}_N > 1 \). Solving for \( \lambda^*_f_2 \) in \( g(\lambda^*_f_2) = 0 \), the roots of \( g(\lambda^*_f_2) = 0 \) are explored using the Descartes rule of signs. The two possibilities are tabulated in Table 1.

The analysis of the results in Table 1 results in the following Theorem 4.

**Theorem 4.** The model system (2) has a unique endemic equilibrium if \( \mathcal{R}_T > 1 \), \( \mathcal{R}_N > 1 \) and \( \mathcal{R}_L < 1 \) (Case 1) and \( \mathcal{R}_T > 1 \), \( \mathcal{R}_N > 1 \) and \( \mathcal{R}_L > 1 \) (Case 2).

### 4. Numerical simulations

In this section, we carry out detailed numerical simulations using Matlab programming language to assess the effects of *Trichomonas vaginalis* transmission in a community in the presence of treatment. The parameter values used for numerical simulations are in Table 2.

Fig. 4 is a graphical representation showing what happens as we vary the infectives initial conditions when (a) there is no treatment \( \mathcal{R}_T > 1 \) and (b) there is treatment \( \mathcal{R}_T < 1 \). It is shown that in the absence treatment \( \mathcal{R}_T > 1 \) male and female infectives increase until they reach their corresponding endemic equilibrium state. In the presence of treatment as noted in Fig. 4(b), irregardless of the initial conditions, the infective populations (both male and female) decline to asymptotically negligible levels signifying that treatment is able to control *Trichomonas vaginalis* infections. If that is the case why does the world still experiencing the existence of *Trichomonas vaginalis* infections? It seems people who are infected are not presenting themselves for treatment or given that in most infected males it does not show any symptoms, so it is possible that they may resist visiting clinics and doctors while continuously infecting their female sexual partners. This suggests that more public health campaigns should be implemented to make males aware of their crucial role in the fight against *Trichomonas vaginalis* infections.
Table 2

<table>
<thead>
<tr>
<th>Model parameter</th>
<th>Value (range)</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\Lambda$</td>
<td>$0.029 \text{ yr}^{-1} \times 200,000$</td>
<td>[2]</td>
</tr>
<tr>
<td>$\mu$</td>
<td>$0.02 \text{ yr}^{-1}$</td>
<td>[2]</td>
</tr>
<tr>
<td>$\beta_{m}$</td>
<td>$0.851 \text{ yr}^{-1}$</td>
<td>[4]</td>
</tr>
<tr>
<td>$\beta_{f_1}$</td>
<td>$0.451 \text{ yr}^{-1}$</td>
<td>[32]</td>
</tr>
<tr>
<td>$\beta_{f_2}$</td>
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<td>Assumed</td>
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<tr>
<td>$\gamma$</td>
<td>$0.90 \text{ yr}^{-1}$</td>
<td>[12]</td>
</tr>
<tr>
<td>$\alpha$</td>
<td>$0.0125 \text{ yr}^{-1}$</td>
<td>Assumed</td>
</tr>
<tr>
<td>$\rho$</td>
<td>0.505</td>
<td>[23]</td>
</tr>
</tbody>
</table>

Fig. 4. Simulations of the infectives for various initial conditions when (a) $R_T > 1$ and (b) $R_T > 1$ using parameter values in Table 2.

In Fig. 5 we show the effects of varying rates of *Trichomonas vaginalis* transmissibility on the infectives population. In Figs. 5(a), (c) and (e) – effects of varying $\beta_{m}$ on males, straight females and lesbians, respectively. It is shown that an increase in $\beta_{m}$ results in a pronounced increase in the number of females getting infected. However, an increase in the number of females being infected leads to more males getting infected due to increased chances of an uninfected male coming across an infected partner. The same happens in reverse order if $\beta_{f_1}$ is increased as shown in Figs. 5(b), (d) and (f).

5. Discussion

This paper studies the dynamics of *Trichomonas vaginalis* transmission in a community taking into account the different modes of transmission that is (i) penis to vagina/vagina to penis (between a male and a female through sex), (ii) vulva to vulva among lesbian couples. The reproduction number of the model is computed and analysed in an effort to understand the infection and ways to mitigate it. The disease-free equilibrium of the model is shown to be globally asymptotically stable when the corresponding reproduction number is less than unity. Analysis of the reproduction number has shown that:

- The increase of trichomoniiasis cases among non-lesbians also results a result in an increase of trichomoniiasis cases among lesbians. This might be due non-lesbians turning into lesbians while they are already infected. What does that mean in terms of disease control in women? This simply suggests that control of *Trichomonas vaginalis* infections among non-lesbians has a positive impact on lesbians as well.
- Ideal treatment rates for different infection rates have been explored and it is shown that the treatment has the potential to control trichomoniiasis cases in any given community.

The analytical results obtained have also been supported by numerical simulations produced. It is noted from numerical simulations that an increase in male (female) to female (male) transmission rate leads to an increase in *Trichomonas vaginalis* infections among females (males). An increase of *Trichomonas vaginalis* infections among females translates to an increase...
of it among males in return and the triad cycle continues. How can this cycle be broken? It seems the problem lies with the males given females experience some discomfort upon getting infected which forces them to seek treatment. When it comes to males most of them do not experience any symptoms related to *Trichomonas vaginalis* infection and are most likely to go untreated for a long period while continuously infecting females they come into sexual contact with. With regard to *Trichomonas vaginalis* infections it may be the best to encourage all females infected to bring their sexual partners when
seeking treatment. Additionally, it may be necessary for relevant departments to step up health educational campaigns advocating for regular reproductive health check up for males. This way asymptomatic Trichomonas vaginalis cases in males may be found and treated. The study presented here is not exhaustive as it can be extended to include condom use as well as co-infection with HIV.

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References