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'Transmission dynamics of trichomoniasis in bisexuals' without the 'E'

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Abstract. A deterministic model for transmission dynamics of Trichomonas vaginalis in a population with bisexuals is formulated and analysed. The disease free equilibrium point has been shown to be globally asymptotically stable when the reproduction number is less than a unity. Furthermore, the endemic equilibrium point has been shown to be locally asymptotically stable and globally asymptotically stable for when the reproduction number is greater than a unity, using the Centre manifold theory and Liapunovs functional approach, respectively. Analysis of the reproduction number has shown that an increase in the number of infected bisexuals result in an increase in the number of infectives among heterosexuals and vise-versa. This suggests that straight women are turning into bisexuals already infected and that bisexuals are linked to straight females indirectly by males. Simulations results has shown that treatment is the major parameter in controlling the spread of the infection.

Keywords: reproduction number, bisexuality, treatment.

1 Introduction

Trichomoniasis is abbreviated as TV, it is a sexually transmitted Infection (STI) or a sexually transmitted disease (STD)[10]. A single celled protozoan (microscopic parasite) called *trichomonas vaginalis* is the causative agent, usually this parasite is found in the vagina and urethral tissues[4, 5, 10, 14]. *Trichomoniasis* is diagnosed by visually observing the *trichomonands* via a microscope, this is so, because, *trichomonands* are too small to be seen by a naked eye[6, 10, 17]. The *trichomonands* are pear shaped and have several flagella (whip-like tails) at one end. In women, TV is detected through inserting a speculum into the vagina, followed by collecting a sample of vaginal discharge using a cotton-tipped applicator. The collected sample will then be placed onto a microscopic slide and sent to a laboratory to be analyzed. Results on TV tests may also reveal small red ulcerations on the vaginal wall or cervix[9]. It is a far more sexually transmitted infection than either *Clamydia trachomatis* or *Neisseria gonorrhoea*[2, 19].

Some researchers have shown that *trichomoniasis* is more prevalent in females (67-100 percent of female sexual partners of an infected male get infected) than males (14-60 percent of male sexual partners of an infected female get infected)[2, 10, 11, 19]. The reason why females are more infected is poorly understood, although some researchers have the idea that the pro-static fluid contains zinc and other substances that are harmful to the pathogen[2, 21, 22]. The WHO has estimated that 160 million cases of infection are acquired annually worldwide[3]. The estimates for North America alone are between five and eight million new infections each year, with estimated rate of asymptomatic cases as high as fifty percent[3–5, 23]. Some researchers have also shown that *trichomonas vaginalis* is more prevalent in industrialised countries[2, 6, 12].

In females, the parasite usually affects the vagina, urethra, cervix, bladder and glands in genital areas[15, 17, 18]. In males, the parasite infects the urethra or under the foreskin of the penis if it is not circumcised. Females usually reveal symptoms, while infection in males are usually asymptomatic. Signs and

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symptoms in females includes: soreness, vaginal discharge, inflammation and vaginal itching, instability during sexual intercourse, strange unpleasant smell and pain when passing urine[7, 9, 11]. Although, males do not usually reveal symptoms, when they appear (usually after a week of infection), they are associated with discharge from the penis that may be thin and whitish, pain or burning sensation when passing urine, swelling of the scrotum and inflammation of the foreskin (though not common)[2, 8, 12].

Trichomoniasis infection is treated and cured with *metronidazole* or *tinidazole* except in the first *trimester* of pregnancy, when *clotrimazole* is used topically. Usually the treatment is given as a single-dose therapy and should be prescribed to any sexual partner(s) as well because they may be asymptomatic carriers[2, 6, 9, 13]. The medicine is usually taken by mouth as pills, tablets or capsules. Medicine given in the vagina will not cure the infection[2]. It is important not to drink alcohol while taking *metronidazole* or *tinidazole*, since the combination can lead to *abdominal* pain and vomiting[2, 6, 14].

Left untreated *trichomoniasis* may leads to complications especially in women and these includes:pelvic inflammatory of the disease, pre-term delivery, premature rapture of membranes, low birth weight infants and predisposing to HIV infection and cervical cancer[8, 9, 19]. *Trichomoniasis* can also leads to infertility in men[2]. People are encouraged to prevent infections than to cure. Since TV is a sexually transmitted infection, abstinence is the most preferable measure to avoid infection. Further, people are encouraged to practise safe sex and hygiene, for instance, use of condoms. Condoms are effective at reducing, but not wholly preventing transmission[6]. Individuals are encouraged to wash before and after sex, not to share swimsuits and towels, since, the *trichomonands* can survive for up to forty-five minutes outside the body. Individuals should also shower immediately after swimming in a public pool[2, 6].

This work extends the work done by Bhunu and Mushayabasa[1], to incorporate the aspect of bisexuality, a common phenemenon in African homosexuals.

2 Model description

The model subdivides the total population into the following sub-population: susceptible males (non homosexuals) $S_m(t)$, *Trichomanas vaginalis* infected males $I_m(t)$, susceptible straight females (non homosexuals) $S_{f_s}(t)$, infected straight females $I_{f_s}(t)$, susceptible bisexual females $S_{f_b}(t)$, infected bisexual female $I_{f_b}(t)$.

The total population $N(t)$ is given by: $N(t) = N_m(t) + N_f(t)$, $N_m = S_m + I_m(t)$, $N_f(t) = N_{f_s}(t) + N_{f_b}(t)$, $N_{f_s}(t) = S_{f_s}(t) + I_{f_s}(t)$, $N_{f_b}(t) = S_{f_b}(t) + I_{f_b}(t)$, where $N_m(t)$, $N_f(t)$, $N_{f_s}(t)$ and $N_{f_b}(t)$ are the total number of males, females, straight females and bisexual females respectively. In this model we assumed that there are no male homosexuals (gays), females are either straight or bisexuals and that homosexuality in females is a result of environmental factors[20]. Further, no person is born being a bisexual, some straight females becomes bisexuals due to environmental factors at a rate γ. Susceptible humans enter the population through birth at a rate Λ, a proportion ρ being males and a complementary proportion $1 - \rho$ being straight females. Susceptible males acquire *trichomonas vaginalis* infection following sexual contact with an infected female (straight or bisexual) at a rate λ_f which is given by:

$$\lambda_f = \frac{\beta_f(I_{f_s} + I_{f_b})}{N_{f_s} + N_{f_b}}, \tag{1}$$

with β_f being the effective contact rate for *trichomonas vaginalis* transmission from female to male. Susceptible straight females acquire *trichomonas vaginalis* infection following contact with an infected straight male at a rate λ_m which is given by:

$$\lambda_m = \frac{\beta_m I_m}{N_m}, \tag{2}$$

with β_m being the effective contact rate for *trichomonas vaginalis* transmission from male to female. Susceptible bisexual females acquire *trichomonas vaginalis* infection following sexual contact with an infected straight male or a bisexual female at a rate λ_b which is given by :

$$\lambda_b = \frac{\beta_{f_l} I_{f_b}}{N_{f_b}} + \frac{\beta_m I_m}{N_m}, \tag{3}$$

with β_{f_l} being the effective contact rate for *trichomonas vaginalis* transmission from female to female. Infected individuals are treated at a rate α and move back into their corresponding susceptible classes. Individuals experience natural death at a rate μ. Furthermore, since *trichomoniasis* infection does not kill, there is no disease induced death rate.

The structure of the model is given in Fig. 1.

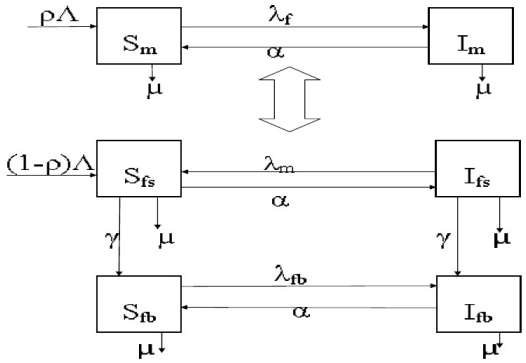


Fig. 1. Structure of model

Based on the given assumptions the following system of differential equations describes the model:

$$\begin{aligned}
S'_m(t) &= \rho\Lambda - \lambda_f S_m - \mu S_m + \alpha I_m,\\
I'_m(t) &= \lambda_f S_m - (\alpha+\mu) I_m,\\
S'_{f_s}(t) &= (1-\rho)\Lambda - \lambda_m S_{f_s} - \gamma S_{f_s} - \mu S_{f_s} + \alpha I_{f_s},\\
I'_{f_s}(t) &= \lambda_m S_{f_s} - (\mu+\alpha+\gamma) I_{f_s},\\
S'_{f_b}(t) &= \gamma S_{f_s} - \lambda_b S_{f_b} - \mu S_{f_b} + \alpha I_{f_b},\\
I'_{f_b}(t) &= \lambda_b S_{f_b} + \gamma I_{f_S} - (\mu+\alpha) I_{f_b}.
\end{aligned} \tag{4}$$

3 Invariant region

In this section, we study some basic results of the model system (4) which will be useful in the proofs of stability and persistence results. We start by showing that the solutions of the model system (4) are positive and defined on $(0,\infty)$.

Theorem 1. *For all* $S^0_m, I^0_m, S^0_{f_s}, I^0_{f_s}, S^0_{f_b}, I^0_{f_b} > 0$ *there exist* $S_m, I_m, S_{f_s}, I_{f_s}, S_{f_b}, I_{f_b}$ *such that:* $(0,\infty) \to (0,\infty)$ *which solve the model system (4) with initial conditions*
$S_m = S^0_m, I_m = I^0_m, S_{f_s} = S^0_{f_s}, I_{f_s} = I^0_{f_s}, S_{f_b} = S^0_{f_b}, I_{f_b} = I^0_{f_b}$.

Proof. Let $F_1(x) = S'_m(t)$, $F_2(x) = I'_m(t)$, $F_3(x) = S'_{f_s}(t)$, $F_4(x) = I'_{f_s}(t)$, $F_5(x) = S'_{f_b}(t)$, and $F_6(x) = I'_{f_b}(t)$ where $x = (x_1, x_2, x_3, x_4, x_5, x_6)$ with $x_1 = S_m$, $x_2 = I_m$, $x_3 = S_{f_s}$, $x_4 = I_{f_s}$, $x_5 = S_{f_b}$, and $x_6 = I_{f_b}$.
In this case: $N_m = x_1 + x_2$, $N_{f_s} = x_3 + x_4$, $N_{f_b} = x_5 + x_6$, $N_f = x_3 + x_4 + x_5 + x_6$ and the forces of infection becomes:

$$\lambda_f = \frac{(x_4+x_6)\beta_f}{x_3+x_4+x_5+x_6},\ \lambda_m = \frac{x_2\beta_m}{x_1+x_2},\ \lambda_b = \frac{x_2\beta_m}{x_1+x_2} + \frac{x_6\beta_{f_l}}{x_5+x_6}.$$

It follows that:

$$F_1(x) = \rho\Lambda - \frac{(x_4+x_6)x_1\beta_f}{x_3+x_4+x_5+x_6} - \mu x_1 + \alpha x_2,\ F_2(x) = \frac{(x_4+x_6)x_1\beta_f}{x_3+x_4+x_5+x_6} - k_1 x_2, \tag{5}$$

$$F_3(x) = (1-\rho)\Lambda - \frac{x_2 x_3 \beta_m}{x_1+x_2} - k_4 x_3 + \alpha x_4,\ F_4(x) = \frac{x_2 x_3 \beta_m}{x_1+x_2} - k_2 x_4, \tag{6}$$

$$F_5(x) = \gamma x_3 - \frac{x_2 x_5 \beta_m}{x_1+x_2} - \frac{x_5 x_6 \beta_{f_l}}{x_5+x_6} - \mu x_5 - \alpha x_6,\ F_6(x) = \frac{x_2 x_5 \beta_m}{x_1+x_2} + \frac{x_5 x_6 \beta_{f_l}}{x_5+x_6} + \gamma x_4 - k_1 x_6. \tag{7}$$

Using the properties of differentiable functions, $F_i(x)$ is differentiable for each $i = 1, 2, 3, 4, 5, 6$ and hence $F_i(x)$ are continuous functions. It follows that:

$$\frac{\partial F_1}{\partial x_1} = \frac{(x_4 + x_6)\beta_f}{N_f^2},\ N_f \neq 0;\ \frac{\partial F_1}{\partial x_2} = \alpha,\ \frac{\partial F_1}{\partial x_3} = \frac{(x_4 + x_6)x_1\beta_f}{N_f^2},\ N_f \neq 0;$$

$$\frac{\partial F_1}{\partial x_4} = \frac{(-N_f + x_4 + x_6)x_1\beta_f}{N_f^2}\ N_f \neq 0;\ \frac{\partial F_1}{\partial x_5} = \frac{\partial F_1}{\partial x_3} \text{ (by symmetry), and}$$

$$\frac{\partial F_1}{\partial x_6} = \frac{\partial F_1}{\partial x_4} \text{ (by symmetry).}$$

We see that the partial derivatives of the function $F_1(x)$ exists and are continuous.
In a similar manner, it can be shown that the partial derivatives $\frac{\partial F_j}{\partial x_i},\ i = 1, 2, 3, 4, 5, 6$ and $j = 2, 3, 4, 5, 6$ exist and are continuous, hence
$F(x) = (F_1(x), F_2(x), F_3(x), F_4(x), F_5(x), F_6(x))$ is a locally Lipschitz continuous function.
Let $x_1 = 0$ with $x_2 > 0,\ x_3 > 0,\ x_4 > 0,\ x_5 > 0,$ and $x_6 > 0$, then

$$F_1(x) = \rho\Lambda + \alpha x_2 > 0. \tag{8}$$

Let $x_2 = 0$ with $x_1 > 0,\ x_3 > 0,\ x_4 > 0,\ x_5 > 0,$ and $x_6 > 0$, then

$$F_2(x) = \frac{(x_4 + x_6)x_1\beta_f}{N_f} > 0. \tag{9}$$

Let $x_3 = 0$ with $x_1 > 0,\ x_2 > 0,\ x_4 > 0,\ x_5 > 0,$ and $x_6 > 0$, then

$$F_3(x) = (1 - \rho)\Lambda + \alpha x_4 > 0. \tag{10}$$

Let $x_4 = 0$ with $x_1 > 0,\ x_2 > 0,\ x_3 > 0,\ x_5 > 0,$ and $x_6 > 0$, then

$$F_4(x) = \frac{x_2 x_3 \beta_m}{N_m} > 0. \tag{11}$$

Let $x_5 = 0$ with $x_1 > 0,\ x_2 > 0,\ x_3 > 0,\ x_4 > 0,$ and $x_6 > 0$, then

$$F_5(x) = \gamma x_3 + \alpha x_6 > 0. \tag{12}$$

Let $x_6 = 0$ with $x_1 > 0,\ x_2 > 0,\ x_3 > 0,\ x_4 > 0,$ and $x_5 > 0$, then

$$F_6(x) = \gamma x_4 + \frac{x_2 x_5 \beta_m}{N_m} > 0. \tag{13}$$

It follows by theorem 2 that, for every $x_0 = (S_m^0, I_m^0, S_{f_s}^0, I_{f_s}^0, S_{f_b}^0, I_{f_b}^0) \in \mathbb{R}_+^6$, there exists a unique solution of $x' = F(x),\ x(0) = x_0$ with values in $\mathbb{R}_+^6$ which is defined in some interval $(0, b]$ with $b \in (0, \infty)$ or $b = \infty$. If $b < \infty$, then

$$\sup_{0 \leq t \leq b} N(t) = \infty. \tag{14}$$

Using a theorem on differential inequality[30], it follows that:

$$N' = \Lambda - \mu N. \tag{15}$$

It follows that, as t tends to infinity, that is $t \to \infty$,

$$0 < N(t) < \frac{\Lambda}{\mu}, \tag{16}$$

so $N(t)$ is bounded which is a contradiction, hence, $b = \infty$. Thus the solutions of the model system (4) are positive and are defined on $(0, \infty)$ and this completes the proof.

Theorem 2. *The system (4) is dissipative that is all the solutions are uniformly bounded on* $\Omega_3 \subseteq \mathbb{R}^6_+$.

Proof. Let $(S_m, I_m, S_{f_s}, I_{f_s}, S_{f_b}, I_{f_b}) \in \mathbb{R}^6_+$ be any solutions with non negative initial conditions as shown in the above theorem. Using a theorem on differential inequality[30], it follows that

$$\lim_{t\to\infty}(S_m + S_{f_s} + S_{f_b}) \leq \frac{\Lambda}{\mu}. \tag{17}$$

Taking the derivative of N along a solution path of the system (4) gives

$$N' = S'_m + I'_m + S'_{f_s} + I'_{f_s} + S'_{f_b} + I'_{f_b}, \tag{18}$$

that is

$$N' = \Lambda - \mu N. \tag{19}$$

All parameters and state variables of the system (4) are assumed to be non-negative for $t \geq 0$ since it monitors human population, hence $0 < N(t)$. Now by solving the above linear first order differential Eq. (19), we obtain the following result: $0 < N(t) < \frac{\Lambda}{\mu}\ as\ t \to \infty$, hence, all feasible solutions of the system (4) are bounded in $\Omega_3 \subseteq \mathbb{R}^6_+$, where

$$\Omega_3 = \left\{ \left(S_m, I_m, S_{f_s}, I_{f_s}, S_{f_b} I_{f_b}\right) : N(t) \leq \frac{\Lambda}{\mu} \right\}. \tag{20}$$

The argument above shows that Ω_3 is positively invariant and it is sufficient to consider solutions in Ω_3. Existence, uniqueness and continuation holds in Ω_3.

4 Equilibria points and stability analysis

In this section we look at the disease free equilibrium point, reproduction number of the system and its analysis and the endemic equilibrium point. To calculate the disease free equilibrium point, we equate the system (4) to the zero vector and solve for the unknown variables $S_m, I_m, S_{f_s}, I_{f_s}, S_{f_b}, I_{f_b}$.
Let

$$Q_0 = \left(S^0_m, I^0_m, S^0_{f_s}, I^0_{f_s}, S^0_{f_b}, I^0_{f_b}\right). \tag{21}$$

be the disease free equilibrium point.
At the disease free equilibrium point, we have:

$$I_m = I_{f_s} = I_{f_b} = 0. \tag{22}$$

Now by solving the system (4) as mentioned above we have:

$$Q_0 = \left(\frac{\rho\Lambda}{\mu}, 0, \frac{(1-\rho)\Lambda}{\mu+\gamma}, 0, \frac{\gamma(1-\rho)\Lambda}{\mu(\mu+\gamma)}, 0\right). \tag{23}$$

The disease free equilibrium point Q_0 attracts the region:

$$\Omega_0 = \left\{ \left(S^0_m, I^0_m, S^0_{f_s}, I^0_{f_s}, S^0_{f_b}, I^0_{f_b}\right) \in \Omega_3 : I^0_m = I^0_{f_s} = I^0_{f_b} = 0 \right\}. \tag{24}$$

The disease free equilibrium point assist us in calculating the effective reproduction number which we shall call R_T, which is the average number of secondary new cases of *trichomoniasis* produced by each infected individual in a totally susceptible population of heterosexuals and homosexuals in the presence of treatment.Following van den Driessche and Watmough[24], the reproduction number is given by:

$$R_T = \frac{\beta_{f_l}}{3k_1} + \frac{2^{\frac{1}{3}}k_2k_4h_1}{3k_1(h_2 + \sqrt{h_2^2 - 4k_2^6k_4^6h_1^3})^{\frac{1}{3}}} + \frac{2^{\frac{2}{3}}(h_2 + \sqrt{h_2^2 - 4k_2^6k_4^6h_1^3})^{\frac{1}{3}}}{6k_1k_2k_4}. \tag{25}$$

where

$$h_1 = \beta_{f_l}^2 + 3\beta_f\beta_m,\ h_2 = k_2^2k_4^2[(-18\mu k_1 + 9\mu\gamma + 9\gamma k_2)\beta_f\beta_{f_l}\beta_m + 2k_2k_4\beta_{f_l}^3],\ k_1 = \alpha + \mu > 0,$$
$$k_2 = \alpha + \mu + \gamma,\ k_3 = \frac{\rho}{1-\rho},\ \text{and } k_4 = \mu + \gamma,$$

throughout the manuscript.
Theorem 3 follows from van den Driessche and Watmough[24].

Theorem 3. *The disease free equilibrium point Q_0 of the system (4) is locally asymptotically stable for $R_T < 1$ and unstable otherwise.*

Theorem 4. *The disease free equilibrium point Q_0 of the system (4) is globally asymptotically stable provided $R_T < 1$.*

Proof. Following Castillo-Chavez et al.[25], we write the system (4) in the form

$$X'(t) = F(X,Y), Y'(t) = G(X,Y), G(X,0) = 0, \tag{26}$$

where the components of $X \in \mathbb{R}_+^3$ denotes the number of uninfected individuals and the components of $Y \in \mathbb{R}_+^3$ denotes the number of infected individuals. In this case the disease free equilibrium point is denoted by $Q_0 = (\overline{X_0}, \mathbf{0})$, where

$$\overline{X_0} = \left(\frac{\rho\Lambda}{\mu}, \frac{(1-\rho)\Lambda}{\mu+\gamma}, \frac{\gamma(1-\rho)\Lambda}{\mu(\mu+\gamma)}\right). \tag{27}$$

It suffices to prove the two conditions that: For $X'(t) = F(X,0)$, $\overline{X_0}$ is globally asymptotically stable. Secondly

$$G(X,Y) = UY - G^*(X,Y), G^*(X,Y) \geq 0\ X,Y \in \Omega. \tag{28}$$

Now consider

$$F(X,0) = \begin{bmatrix} \rho\Lambda - \mu S_m \\ (1-\rho)\Lambda - (\mu+\gamma)S_{f_s} \\ \gamma S_{f_s} - \mu S_{f_b} \end{bmatrix} \quad \text{and} \quad U = \begin{bmatrix} -k_1 & k_3\beta_f & k_3\beta_f \\ \frac{\mu\beta_m}{k_3k_4} & -k_2 & 0 \\ \frac{\gamma\beta_m}{k_3k_4} & \gamma & \beta_{f_l} - k_1 \end{bmatrix},$$

which implies that

$$G^*(X,Y) = \begin{bmatrix} G_1^*(X,Y) \\ G_2^*(X,Y) \\ G_3^*(X,Y) \end{bmatrix},$$

where $G_1^*(X,Y) = \beta_f(I_{f_s} + I_{f_b})(k_3 - \frac{S_m}{N_f})$, $G_2^*(X,Y) = \beta_m I_m\left[\frac{\mu}{k_3k_4} - \frac{S_{f_s}}{N_m}\right]$ and $G_3^*(X,Y) = \beta_m I_m(\frac{\gamma}{k_3k_4} - \frac{S_{f_b}}{N_m}) + \beta_{f_l}I_{f_b}(1 - \frac{S_{f_b}}{N_{f_b}})$.

It suffices to show that $G_1^*(X,Y)$, $G_2^*(X,Y)$ and $G_3^*(X,Y)$ are all greater than or equal to zero.
Now, since $S_{f_s} \leq S_{f_s}^0$ and $N_m \longrightarrow \frac{\rho\Lambda}{\mu}$ as $t \longrightarrow \infty$, we have:

$$G_2^*(X,Y) = \beta_m I_m\left[\frac{\mu}{k_3k_4} - \frac{S_{f_s}}{N_m}\right] \geq \beta_m I_m\left[\frac{\mu}{k_3k_4} - \frac{(1-\rho)\Lambda}{k_4N_m}\right] \longrightarrow 0\ as\ t \longrightarrow \infty,$$

hence $G_2^*(X,Y) \geq 0$. Also, since $S_m \leq S_m^0$ and $N_{f_s} + N_{f_b} \longrightarrow N_{f_s}^0 + N_{f_b}^0$ as $t \longrightarrow \infty$, we have:

$$G_1^*(X,Y) = \beta_f(I_{f_s} + I_{f_b})\left(k_3 - \frac{S_m}{N_f}\right) \geq \beta_f(I_{f_s} + I_{f_b})\left(k_3 - \frac{\rho\Lambda}{\mu N_f}\right) \longrightarrow 0 \; as \; t \longrightarrow \infty,$$

hence $G_1^*(X,Y) \geq 0$. Similarly, since $S_{f_b} \leq S_{f_b}^0$, $1 - \dfrac{S_{f_b}}{N_{f_b}} \geq 0$ and $N_m \longrightarrow \dfrac{\rho\Lambda}{\mu}$ as $t \longrightarrow \infty$, we have:

$$G_3^*(X,Y) = \beta_m I_m\left(\frac{\gamma}{k_3k_4} - \frac{S_{f_b}}{N_m}\right) + \beta_{f_l} I_{f_b}\left(1 - \frac{S_{f_b}}{N_{f_b}}\right) \geq \beta_m I_m\left[\frac{\gamma}{k_3k_4} - \frac{\gamma(1-\rho)\Lambda}{\mu k_4 N_m}\right] \longrightarrow 0 \; as \; t \longrightarrow \infty,$$

hence $G_3^*(X,Y) \geq 0$. Therefore $G^*(X,Y) \geq 0$ and the result follow.

4.1 Analysis of reproduction number

In this section, we look into the effect an increase in *trichonomiasis* infections among heterosexuals has on bisexuals and vice-versa. Also, we look into the effect an increase in *trichonomiasis* infections among heterosexuals has on the total population, as well as the effect an increase in *trichonomiasis* infections among bisexuals has on the whole population. Furthermore, we analyze the effects that the parameters that defines the reproduction number has on the average number of secondary new cases of *trichonomiasis* produced by an average infected individual in a totally susceptible population. Now for us to analyze the effect that treatment has on the average number of secondary new cases of *trichonomiasis* produced by an average infected individual in a totally susceptible population, we consider first the limit of the reproduction number R_T as the parameter α tends to infinity. It follows by properties of limits that $\lim\limits_{\alpha\to\infty} R_T$ is given by:

$$\lim_{\alpha\to\infty}\left(\frac{\beta_{f_l}}{3k_1}\right) + \lim_{\alpha\to\infty}\left(\frac{2^{\frac{1}{3}}k_2k_4h_1}{3k_1(h_2 + \sqrt{h_2^2 - 4k_2^6k_4^6h_1^3})^{\frac{1}{3}}}\right) + \lim_{\alpha\to\infty}\left(\frac{2^{\frac{2}{3}}(h_2 + \sqrt{h_2^2 - 4k_2^6k_4^6h_1^3})^{\frac{1}{3}}}{6k_1k_2k_4}\right) = 0. \quad (29)$$

This suggest that treatment has the potential to reduce infection. In other words, provision of enough treatment, especially in the form of *metronidazole* or *tinidazole* reduces the average number of secondary new cases of *trichonomiasis* produced by an average infected individual in a totally susceptible population.

We now continue to do the analysis of the reproduction number R_T by considering the case where all straight females turn to be strictly lesbians, that is the case where $\beta_m = \beta_f = 0$. In this case the reproduction number becomes $R_T = \dfrac{\beta_{f_l}}{k_1}$. In this manuscript, we shall call this reproduction number R_{f_l}, thus $R_{f_l} = \dfrac{\beta_{f_l}}{k_1}$, where R_{f_l} represents the average number of secondary *trichomoniasis* infections in females caused by one infected female (non straight female) in a fully susceptible population of non straight females. It is clear that for case where all females turn to be lesbians, the reproduction number depends on the parameters α, μ and β_{f_l}. In this case, the analysis of the reproduction number R_{f_l} is the same as that which has been done by Bhunu and Mushayabasa[1].

Similarly, as shown by Bhunu and Mushayabasa[1] the reproduction number that governs the relationship between males and females is:

$$R_{fm} = \sqrt{\frac{\beta_m\beta_f}{(\alpha+\mu)^2}}, \quad (30)$$

where R_{fm} denotes the average number of secondary *trichomoniasis* infections caused by one infected straight male or non lesbian female in a fully susceptible population of heterosexuals in the presence of treatment. Hence, in the case where there is no contribution of bisexuality in *trichonomiasis* transmission dynamics, the analysis of the reproduction number has been dealt with, by Bhunu and Mushayabasa[1]. Now, for us to complete, the analysis of the reproduction number R_T, it suffices to express the reproduction number R_T in terms of both R_{fm} and R_{f_l}, so that we can analyze the effect that the change in either R_{fm} or R_{f_l} has on the reproduction number R_T and vice-versa. In other words, this helps us to analyze the effect that the

change either in the number of *trichonomiasis* infected individuals amongst heterosexuals or in the number of *trichonomiasis* infected individuals amongst non- heterosexuals has on the total population. It follows that

$$R_T = \frac{R_{f_l}}{3} + H\left(G_1, G_2\right), \tag{31}$$

where

$$\begin{aligned} G_1 &= R_{f_l}^2 + 3R_{fm}^2, \\ G_2 &= k_2^2 k_4^2[(-18\mu k_1 + 9\mu\gamma + 9\gamma k_2)R_{f_l}R_{fm}^2 + 2k_2k_4R_{f_l}^3], \\ H\left(G_1, G_2\right) &= \frac{2^{\frac{1}{3}}k_2k_4G_1}{3(G_2 + \sqrt{G_2^2 - 4k_2^6k_4^6G_1^3})^{\frac{1}{3}}} + \frac{2^{\frac{2}{3}}(G_2 + \sqrt{G_2^2 - 4k_2^6k_4^6G_1^3})^{\frac{1}{3}}}{6k_2k_4}. \end{aligned}$$

It is clear that $G_1 > 0$, $G_2 > 0$ since, $h_1 > 0$, $h_2 > 0$ and $k_1 > 0$. Using the chain rule, we have:

$$\begin{aligned} \frac{\partial R_T}{\partial R_{f_l}} &= \frac{1}{3} + \frac{\partial H\left(G_1, G_2\right)}{\partial G_1}\frac{\partial G_1}{\partial R_{f_l}} + \frac{\partial H\left(G_1, G_2\right)}{\partial G_2}\frac{\partial G_2}{\partial R_{f_l}}, \\ \frac{\partial R_T}{\partial R_{fm}} &= \frac{\partial H\left(G_1, G_2\right)}{\partial G_1}\frac{\partial G_1}{\partial R_{fm}} + \frac{\partial H\left(G_1, G_2\right)}{\partial G_2}\frac{\partial G_2}{\partial R_{fm}}. \end{aligned}$$

It follows that

$$\frac{\partial H\left(G_1, G_2\right)}{\partial G_1} = \frac{2^{\frac{1}{3}}(h_3^{\frac{1}{3}}) + 16^{\frac{1}{3}}(h_3^{\frac{-1}{3}})h_4k_2^6k_4^6G_1^3}{3h_3^{\frac{2}{3}}} - \frac{32^{\frac{1}{3}}k_2^6k_4^6G_1^2h_3^{\frac{-1}{3}})h_4}{6k_2k_4} > 0. \tag{32}$$

$$\frac{\partial H\left(G_1, G_2\right)}{\partial G_2} = \frac{16^{\frac{1}{3}}}{12k_2^5k_4^5G_1^2}\left(h_3^{\frac{-4}{3}}(1 + G_2h_4)(4k_2^6k_4^6G_1^3) + h_3^{\frac{2}{3}}(1 - G_2h_4)\right) + h_5 > 0, \tag{33}$$

where

$$h_3 = G_2 + \sqrt{G_2^2 - 4k_2^6k_4^6G_1^3},\ h_4 = (G_2^2 - 4k_2^6k_4^6G_1^3)^{\frac{-1}{2}} \text{ and } h_5 = \frac{2^{\frac{2}{3}}(1 + G_2h_4)h_3^{\frac{-2}{3}}}{3}.$$

Also,

$$\frac{\partial G_2}{\partial R_{f_l}} = k_2^2k_4^2[(-18\mu k_1 + 9\mu\gamma + 9\gamma k_2)R_{fm}^2 + 6k_2k_4R_{f_l}^2] > 0$$

and

$$\frac{\partial G_2}{\partial R_{fm}} = 2k_2^2k_4^2(-18\mu k_1 + 9\mu\gamma + 9\gamma k_2)R_{f_l}R_{fm} > 0.$$

Moreover,

$$\frac{\partial G_1}{\partial R_{f_l}} = 2R_{f_l} > 0 \text{ and } \frac{\partial G_1}{\partial R_{fm}} = 2R_{fm} > 0.$$

Hence

$$\frac{\partial R_T}{\partial R_{f_l}} > 0 \text{ and } \frac{\partial R_T}{\partial R_{fm}} > 0.$$

The above mathematical arguments indicate that an increase in *trichonomiasis* infection either amongst heterosexual or non-heterosexuals result in an increase in the cases amongst the individuals in the population.

4.2 Existence and uniqueness of the endemic equilibrium

The are a number of theoretically feasible endemic equilibrium states:

- the first case is where there are no homosexuals and this has been dealt with by Bhunu and Mushayabasa[1] and the endemic equilibrium point has been shown to exist for $R_{fm} > 1$ as well as being both locally and globally asymptotically stable for the case where it exists.
- The second case is where all females becomes strictly homosexuals. We can do the analysis for this case by simply setting $\beta_m = \beta_f = 0$.
- the last case is where both heterosexuality and homosexuality exist. For this case we shall call the endemic equilibrium point the interior equilibrium point.

Now, for the case where the contribution of bisexuality in *trichonomiasis* transmission dynamics is involved, it is a daunting task to solve for the forces of infection in terms of the reproduction number R_T in order for us to show the existence of the endemic equilibrium point, but, the global stability of the disease free equilibrium point for $R_T < 1$ guarantees the existence of the endemic equilibrium point for $R_T > 1$.
Alternatively, to establish the existence of the endemic equilibrium point for $R_T > 1$, we can employ the mean value theorem to do this job. Firstly, by equating the system (4) to the zero vector in $\mathbb{R}^6$ and solve for the unknown variables $S_m^*, I_m^*, S_{f_s}^*, I_{f_s}^*, S_{f_b}^*$ and $I_{f_b}^*$ we have:

$$S_m^* = \frac{k_1\rho\Lambda}{\mu(k_1+\lambda_f^*)},\ I_m^* = \frac{\rho\Lambda\lambda_f^*}{\mu(k_1+\lambda_f^*)},$$

$$S_{f_s}^* = \frac{k_2(1-\rho)\Lambda}{k_4(k_2+\lambda_m^*)},\ I_{f_s}^* = \frac{(1-\rho)\Lambda\lambda_m^*}{k_4(k_2+\lambda_m^*)},$$

$$S_{f_b}^* = \frac{(1-\rho)\Lambda\gamma(k_1k_2+\alpha\lambda_m^*)}{\mu k_4(k_2+\lambda_m^*)(k_1+\lambda_b^*)},\ \text{and}\ I_{f_b}^* = \frac{(1-\rho)\Lambda\gamma[(k_1k_2+\alpha\lambda_m^*)\lambda_b^* + \mu\lambda_m^*(k_1+\lambda_b^*)]}{\mu k_1k_4(k_2+\lambda_m^*)(k_1+\lambda_b^*)}. \tag{34}$$

Thus, the interior endemic equilibrium point is given by:

$$Q^* = \left(S_m^*, I_m^*, S_{f_s}^*, I_{f_s}^*, S_{f_b}^*, I_{f_b}^*\right), \tag{35}$$

where $S_m^*, I_m^*, S_{f_s}^*, I_{f_s}^*, S_{f_b}^*$ and $I_{f_b}^*$ are as given in the system (34). Using the above Eq. (34), we can now solve for N_m^*, N_f^* and $N_{f_b}^*$ to get:

$$N_m^* = \frac{\rho\Lambda}{\mu},\ N_f^* = \frac{(1-\rho)\Lambda}{k_4}\ \text{and}\ N_{f_b}^* = \frac{(1-\rho)\Lambda\gamma}{\mu k_4}. \tag{36}$$

Now, for us to solve for the forces of infections, we have to consider the following equations:

$$\lambda_f^* = \frac{\beta_f(I_{f_s}^* + I_{f_b}^*)}{N_{f_s}^* + N_{f_b}^*},\ \lambda_m^* = \frac{\beta_m I_m^*}{N_m^*}\ \text{and}\ \lambda_b^* = \frac{\beta_{f_l} I_{f_b}^*}{N_{f_b}^{**}} + \frac{\beta_m I_m^*}{N_m^*}. \tag{37}$$

By substituting the systems (34) and (36) for $I_m^*, I_{f_b}^*, N_m^*$ and $N_{f_b}^*$ in (37) we obtain:

$$\lambda_b^* = \beta_{f_l}\frac{k_2\lambda_b^* + \mu\lambda_m^* + \lambda_b^*\lambda_m^*}{(k_2+\lambda_m^*)(k_1+\lambda_b^*)} + \frac{\beta_m I_m^*}{N_m^*}. \tag{38}$$

Secondly, the endemic equilibrium point exist for all values of β_m, β_f and β_{f_l}. Without loss of generality, we now use the fact that the endemic equilibrium point exist for all values of β_m, in particular, it exist for $\beta_m = 0$, that is the case where there is no heterosexuality, which is case (2) of the equilibrium states. By, substituting $\beta_m = 0$ in Eq. (38), we get:

$$\lambda_b^* = \beta_{f_l} \frac{\lambda_b^*}{k_1 + \lambda_b^*}. \tag{39}$$

Upon solving the previous Eq. (39), we obtain: $\lambda_b^* = 0$ or $\lambda_b^* = \beta_{f_l} - k_1$.

In this case $\lambda_b^* = 0$ corresponds to the disease free equilibrium point and $\lambda_b^* = \beta_{f_l} - k_1$ corresponds to the endemic equilibrium point. The endemic equilibrium point exist for $\lambda_b^* > 0$, that is for:

$$\frac{\beta_{f_l}}{k_1} > 1, \tag{40}$$

hence, indeed the endemic equilibrium point exist for $R_{f_l} > 1$. Now to show existence for the case where $R_T > 1$, that is the existence of the interior equilibrium point, it suffices to take the reproduction R_T as function of β_m, that is, $R_T = R_T(\beta_m)$.

Consider

$$R_T(\beta_m) = \frac{\beta_{f_l}}{3k_1} + \frac{2^{\frac{1}{3}} k_2 k_4 h_1}{3k_1 (h_2 + \sqrt{h_2^2 - 4k_2^6 k_4^6 h_1^3})^{\frac{1}{3}}} + \frac{2^{\frac{2}{3}} (h_2 + \sqrt{h_2^2 - 4k_2^6 k_4^6 h_1^3})^{\frac{1}{3}}}{6k_1 k_2 k_4}, \tag{41}$$

it follows by properties of differentiable functions that, $R_T(\beta_m)$ is a differentiable function, hence, it is also a continuous function. Also, since β_m is the probability of infection, it is a non-negative number such that: $0 \leq \beta_m \leq 1$. Moreover, the rate of change of the function $R_T = R_T(\beta_m)$ with respect to β_m is non-negative, hence by the mean value theorem,it follows that the the function $R_T = R_T(\beta_m)$ is a monotonic increasing function on the domain $0 \leq \beta_m \leq 1$. Moreover, since $R_T(\beta_m)$ is a continuous function on a closed and bounded interval $[0, 1]$, it implies that the function itself is bounded and attains its minimum and maximum values.

Now by definition of infimum:

$$R_T \geq \inf_{0 \leq \beta_m \leq 1} R_T. \tag{42}$$

It follows that, since $R_T = R_T(\beta_m)$ is a monotonic increasing function on the domain $0 \leq \beta_m \leq 1$, the infimum of R_T on the interval $0 \leq \beta_m \leq 1$ is the value of R_T at the point where $\beta_m = 0$, that is:

$$\inf_{0 \leq \beta_m \leq 1} R_T = R_T(0), \tag{43}$$

this implies

$$\inf_{0 \leq \beta_m \leq 1} R_T = \frac{\beta_{f_l}}{3k_1} + \frac{\beta_{f_l}}{3k_1} + \frac{\beta_{f_l}}{3k_1}, \tag{44}$$

hence,

$$\inf_{0 \leq \beta_m \leq 1} R_T = \frac{\beta_{f_l}}{k_1} = R_{f_l}, \tag{45}$$

it follows that: $R_T \geq R_{f_l}$.

Since at the endemic equilibrium point, $R_{f_l} > 1$, it follows by the previous result that the endemic equilibrium point exist for $R_T > 1$ and this lead to the following theorem:

Theorem 5. *The interior endemic equilibrium point Q^* of the system (4) exist for $R_T > 1$.*

Theorem 6. *The interior endemic equilibrium point Q^* of the system (4) guaranteed by theorem (5) is locally asymptotically stable for $R_T > 1$.*

Proof. We now employ the Centre-Manifold theory (Carr, 1981) to determine the local stability of the interior endemic equilibrium point Q^*. Here, we use the following substitution or change of variable in order to apply the Centre-Manifold theory:

Let $F_1(x) = S'_m(t),\ F_2(x) = I'_m(t),\ F_3(x) = S'_{f_s}(t),\ F_4(x) = I'_{f_s}(t),\ F_5(x) = S'_{f_b}(t),$ and $F_6(x) = I'_{f_b}(t)$ where $x = (x_1, x_2, x_3, x_4, x_5, x_6)$ with $x_1 = S_m,\ x_2 = I_m,\ x_3 = S_{f_s},$ $x_4 = I_{f_s},\ x_5 = S_{f_b}$ and $x_6 = I_{f_b}$.

In this case: $N_m = x_1 + x_2, N_{f_s} = x_3 + x_4, N_{f_b} = x_5 + x_6, N_f = x_3 + x_4 + x_5 + x_6$ so that $N(t) = x_1 + x_2 + x_3 + x_4 + x_5 + x_6$ and the forces of infection becomes:

$$\lambda_f = \frac{(x_4+x_6)\beta_f}{x_3+x_4+x_5+x_6},\ \lambda_m = \frac{x_2\beta_m}{x_1+x_2} \text{ and } \lambda_b = \frac{x_2\beta_m}{x_1+x_2} + \frac{x_6\beta_{f_l}}{x_5+x_6}.$$

Using the vector notation, let $X = (x_1, x_2, x_3, x_4, x_5, x_6)^T$, then the model system (4) can be written in the form:

$$\frac{dX}{dt} = F(X), \tag{46}$$

where $F(X) = (F_1(x), F_2(x), F_3(x), F_4(x), F_5(x), F_6(x))^T$ and $F_i(x),\ i = 1, 2, 3, 4, 5, 6$ are as given in (5).

Now, since *trichomoniasis* affects females more than males, we have $\beta_m > \beta_f$, hence we can write $\beta_m = k_5\beta_f,\ k_5 > 1$. Similarly, $\beta_f = k_6\beta_{f_l},\ k_6 > 1$. Thus$\beta_m = k_7\beta_{f_l},\ k_7 = k_5k_6 > 1$ and $\beta_m\beta_f = k_6k_7\beta_{f_l}^2,\ k_6k_7 > 1$. We can now express h_1 and h_2 in terms of β_{f_l}, that is: $h_1 = k_8\beta_{f_l}^2$ and $h_2 = k_9\beta_{f_l}^3$ where $k_8 = 1 + 3k_6k_7$ and $k_9 = k_2^2k_4^2[k_6k_7(-18\mu k_1 + 9\mu\gamma + 9\gamma k_2) + 2k_2k_4]$.

Having expressed h_1 and h_2 in terms of β_{f_l}, we now express the effective reproduction number R_T in terms of β_{f_l} also, that is

$$R_T = \frac{\beta_{f_l}}{3k_1} + \frac{2^{\frac{1}{3}}k_2k_4k_8\beta_{f_l}}{3k_1(k_9 + \sqrt{k_9^2 - 4k_2^6k_4^6k_8^3})^{\frac{1}{3}}} + \frac{2^{\frac{2}{3}}(k_9 + \sqrt{k_9^2 - 4k_2^6k_4^6k_8^3})^{\frac{1}{3}}\beta_{f_l}}{6k_1k_2k_4}. \tag{47}$$

If β_{f_l} is taken as a bifurcation point and consider $R_T = 1$ and solve for β_{f_l} to obtain:

$$\beta_{f_l}^* = \frac{1}{\sigma}, \tag{48}$$

where

$$\sigma = \frac{1}{3k_1} + \frac{2^{\frac{1}{3}}k_2k_4k_8}{3k_1(k_9 + \sqrt{k_9^2 - 4k_2^6k_4^6k_8^3})^{\frac{1}{3}}} + \frac{2^{\frac{2}{3}}(k_9 + \sqrt{k_9^2 - 4k_2^6k_4^6k_8^3})^{\frac{1}{3}}}{6k_1k_2k_4}. \tag{49}$$

Let $J(Q_0)$ be the Jacobian matrix of the system (4) evaluated at the disease free equilibrium point Q_0. It follows that:

$$J(Q_0) = \begin{bmatrix} -\mu & \alpha & 0 & \frac{-\beta_f S_m^0}{N_{f_s}^0 + N_{f_b}^0} & 0 & \frac{-\beta_f S_m^0}{N_{f_s}^0 + N_{f_b}^0} \\ 0 & -(\alpha+\mu) & 0 & \frac{\beta_f S_m^0}{N_{f_s}^0 + N_{f_b}^0} & 0 & \frac{\beta_f S_m^0}{N_{f_s}^0 + N_{f_b}^0} \\ 0 & \frac{-\beta_m S_{f_s}^0}{N_m^0} & -(\mu+\gamma) & \alpha & 0 & 0 \\ 0 & \frac{\beta_m S_{f_s}^0}{N_m^0} & 0 & -(\alpha+\mu+\gamma) & 0 & 0 \\ 0 & \frac{-\beta_m S_{f_b}^0}{N_m^0} & \gamma & 0 & -\mu & \alpha - \frac{\beta_{f_l} S_{f_b}^0}{N_{f_b}^0} \\ 0 & \frac{\beta_m S_{f_b}^0}{N_m^0} & 0 & \gamma & 0 & -(\alpha+\mu) + \frac{\beta_{f_l} S_{f_b}^0}{N_{f_b}^0} \end{bmatrix}. \tag{50}$$

The linearised system of (4) with $\beta_{f_l} = \beta_{f_l}^*$ has a simple zero eigenvalue, hence the Centre-Manifold theory can be used to analyse the dynamics of the system (4) near the point $\beta_{f_l} = \beta_{f_l}^*$. The Jacobian matrix (50) of the system (4) has a right eigenvector associated with the zero eigenvalue which is given by: $\mathbf{u} = (u_1, u_2, u_3, u_4, u_5, u_6)^T$, where

$$u_1 = -\left(\frac{2\beta_m\beta_f}{k_2k_4} + 1\right)u_2 < 0,\ u_2 = u_2 > 0,\ u_3 = \frac{\mu\beta_m}{k_3k_4^2}\left(\frac{\alpha}{k_2} - 1\right)u_2 < 0,$$

$$u_4 = \frac{\mu\beta_m u_2}{k_2k_3k_4} > 0,\ u_5 = \frac{(\alpha-\beta^*_{f_l})u_6}{\mu} + \frac{\gamma u_3}{\mu} - \frac{\gamma\beta_m u_2}{\mu k_3 k_4} < 0,\ u_6 = \frac{k_1u_2}{k_3\beta_f} + \frac{\mu\beta_m}{k_3k_4^2}\left(\frac{1-\alpha}{k_2}\right)u_2 > 0.. \quad (51)$$

Similarly, by using the transpose of the Jacobian matrix (50), the left eigenvector of the Jacobian matrix (50) is given by $\mathbf{v} = (v_1, v_2, v_3, v_4, v_5, v_6)^T$ where $v_1 = 0,\ v_2 = v_2 > 0,\ v_3 = 0.$

$$v_4 = (\frac{\gamma k_3\beta_f}{k_2(k_1-\beta^*_{f_l})} + \frac{k_3\beta_f}{k_2})v_2 > 0,\ for\ k_1 - \beta^*_{f_l} > 0,\ v_5 = 0,\ v_6 = \frac{\gamma k_3\beta_f v_2}{k_1-\beta^*_{f_l}} > 0. \quad (52)$$

Having found the right and the left eigenvectors associated with the zero eigenvalue, we can now use Centre-Manifold theorem as used by Castillo-Chavez et al.[25] to do the analysis of the stability of the interior endemic equilibrium point. It follows that:

$$\begin{aligned} a &= \sum v_k u_i u_j \frac{\partial^2 F_k(0,0)}{\partial x_i \partial x_j},\ i,j,k = 1,2,3,4,5,6, \\ b &= \sum v_k u_i \frac{\partial^2 F_k(0,0)}{\partial x_i \partial \beta^*_{f_l}},\ i,k = 1,2,3,4,5,6. \end{aligned} \quad (53)$$

Since, $v_1 = 0, v_3 = 0$ and $v_5 = 0$ the above definitions for a and b (53) reduces to:

$$\begin{aligned} a &= \sum v_2 u_i u_j \frac{\partial^2 F_2(0,0)}{\partial x_i \partial x_j} + v_4 u_i u_j \frac{\partial^2 F_4(0,0)}{\partial x_i \partial x_j} + v_6 u_i u_j \frac{\partial^2 F_6(0,0)}{\partial x_i \partial x_j},\ i,j = 1,2,3,4,5,6 \\ b &= \sum v_2 u_i \frac{\partial^2 F_2(0,0)}{\partial x_i \partial \beta^*_{f_l}} + v_4 u_i \frac{\partial^2 F_4(0,0)}{\partial x_i \partial \beta^*_{f_l}} + v_6 u_i \frac{\partial^2 F_6(0,0)}{\partial x_i \partial \beta^*_{f_l}},\ i = 1,2,3,4,5,6. \end{aligned} \quad (54)$$

Since the functions $F_k\ k = 1,2,3,4,5,6$ are continuous, it implies that:

$$\frac{\partial^2 F_k}{\partial x_i \partial x_j} = \frac{\partial^2 F_k}{\partial x_j \partial x_i}. \quad (55)$$

It follows that the non-zero second derivatives of the function F_2 are:

$$\begin{aligned} &\frac{\partial^2 F_2(0,0)}{\partial x_j \partial x_1} = \frac{\partial^2 F_2(0,0)}{\partial x_1 \partial x_j} = \frac{\mu\beta_f}{(1-\rho)\Lambda},\ j = 4,6, \\ &\frac{\partial^2 F_2(0,0)}{\partial x_4 \partial x_3} = \frac{\partial^2 F_2(0,0)}{\partial x_3 \partial x_4} = -\frac{\mu k_3\beta_f}{(1-\rho)\Lambda}, \\ &\frac{\partial^2 F_2(0,0)}{\partial x_6 \partial x_3} = \frac{\partial^2 F_2(0,0)}{\partial x_3 \partial x_6} = -\frac{\mu k_3\beta_f}{(1-\rho)\Lambda},\ \frac{\partial^2 F_2(0,0)}{\partial x_4^2} = -2\frac{\mu k_3\beta_f}{(1-\rho)\Lambda}, \\ &\frac{\partial^2 F_2(0,0)}{\partial x_5 \partial x_4} = \frac{\partial^2 F_2(0,0)}{\partial x_4 \partial x_5} = -\frac{\mu k_3\beta_f}{(1-\rho)\Lambda},\ \frac{\partial^2 F_2(0,0)}{\partial x_6 \partial x_4} = \frac{\partial^2 F_2(0,0)}{\partial x_4 \partial x_6} = -2\frac{\mu k_3\beta_f}{(1-\rho)\Lambda}, \\ &\frac{\partial^2 F_2(0,0)}{\partial x_6 \partial x_5} = \frac{\partial^2 F_2(0,0)}{\partial x_5 \partial x_6} = -\frac{\mu k_3\beta_f}{(1-\rho)\Lambda},\ \frac{\partial^2 F_2(0,0)}{\partial x_6^2} = -2\frac{\mu k_3\beta_f}{(1-\rho)\Lambda}, \\ &\frac{\partial^2 F_4(0,0)}{\partial x_2 \partial x_1} = \frac{\partial^2 F_4(0,0)}{\partial x_1 \partial x_2} = -\frac{\mu^2\beta_m}{k_3k_4\rho\Lambda},\ \frac{\partial^2 F_4(0,0)}{\partial x_2^2} = -2\frac{\mu^2\beta_m}{k_3k_4\rho\Lambda}, \\ &\frac{\partial^2 F_4(0,0)}{\partial x_3 \partial x_2} = \frac{\partial^2 F_4(0,0)}{\partial x_2 \partial x_3} = \frac{\mu\beta_m}{\rho\Lambda},\ \frac{\partial^2 F_6(0,0)}{\partial x_2 \partial x_1} = \frac{\partial^2 F_6(0,0)}{\partial x_1 \partial x_2} = -\frac{\mu\gamma\beta_m}{k_3k_4\rho\Lambda}, \\ &\frac{\partial^2 F_6(0,0)}{\partial x_2^2} = -2\frac{\mu\gamma\beta_m}{k_3k_4\rho\Lambda},\ \frac{\partial^2 F_6(0,0)}{\partial x_5 \partial x_2} = \frac{\partial^2 F_6(0,0)}{\partial x_2 \partial x_5} = \frac{\mu\beta_m}{\rho\Lambda}. \end{aligned}$$

Following the above arguments together with the system (54), the value of the scalar a is given by:

$$a = k_{10}(u_4+u_6)\frac{2\mu\beta_f v_2}{(1-\rho)\Lambda} + \frac{k_{11}\mu + k_3k_4u_3}{k_3k_4}\frac{2\mu\beta_m u_2 v_4}{\rho\Lambda} + \frac{k_{11}\gamma + k_3k_4u_5}{k_3k_4}\frac{2\mu\beta_m u_2 v_6}{\rho\Lambda}, \tag{56}$$

where $k_{10} = u_1 + k_3u_3 - k_3u_4 + k_3u_5 - k_3u_6 < 0$ and $k_{11} = u_1 - u_2 < 0$. We can see that $a < 0$, since u_1, u_3 and u_5 are all negative. To compute the value of the scalar b, it requires us to express the functions $F_j,\ j = 1,2,3,4,5,6$ in (5) in terms β_{f_l} by using the relationships: $\beta_f = k_6\beta_{f_l},\ k_6 > 1$ and $\beta_m = k_7\beta_{f_l},\ k_7 = k_5k_6 > 1$, that is

$$\begin{aligned}
F_1(x) &= \rho\Lambda - \frac{(x_4+x_6)x_1k_6\beta_{f_l}}{x_3+x_4+x_5+x_6} - \mu x_1 + \alpha x_2,\\
F_2(x) &= \frac{(x_4+x_6)x_1k_6\beta_{f_l}}{x_3+x_4+x_5+x_6} - k_1x_2,\\
F_3(x) &= (1-\rho)\Lambda - \frac{x_2x_3k_7\beta_{f_l}}{x_1+x_2} - k_4x_3 + \alpha x_4,\\
F_4(x) &= \frac{x_2x_3k_7\beta_{f_l}}{x_1+x_2} - k_2x_4,\\
F_5(x) &= \gamma x_3 - \frac{x_2x_5k_7\beta_{f_l}}{x_1+x_2} - \frac{x_5x_6\beta_{f_l}}{x_5+x_6} - \mu x_5 - \alpha x_6,\\
F_5(x) &= \frac{x_2x_5k_7\beta_{f_l}}{x_1+x_2} + \frac{x_5x_6\beta_{f_l}}{x_5+x_6} + \gamma x_4 - k_1x_6.
\end{aligned} \tag{57}$$

It follows from the above system of (57) that the non-zero derivatives are: $\frac{\partial^2F_2(0,0)}{\partial x_4\partial\beta^*_{f_l}} = k_3k_6,\ \frac{\partial^2F_2(0,0)}{\partial x_6\partial\beta^*_{f_l}} = k_3k_6,\ \frac{\partial^2F_4(0,0)}{\partial x_2\partial\beta^*_{f_l}} = \frac{\mu k_7}{k_3k_4},\ \frac{\partial^2F_6(0,0)}{\partial x_2\partial\beta^*_{f_l}} = \frac{\gamma k_7}{k_3k_4}$ and $\frac{\partial^2F_6(0,0)}{\partial x_6\partial\beta^*_{f_l}} = 1$. Now, it follows by the definition of b in (54) that:

$$b = (u_4+u_6)k_3k_6v_2 + u_6v_6 + \frac{\mu k_7u_2v_4}{k_3k_4} + \frac{\gamma k_7u_2v_6}{k_3k_4}. \tag{58}$$

Clearly $b > 0$ since $u_2, u_4, u_6, v_2, v_4, v_6, k_3, k_4$ and k_7 are all strictly positive. Therefore, by using the Centre-Manifold theory, the interior endemic equilibrium point is locally asymptotically stable for $R_T > 1$, since $a < 0$ and $b > 0$. This completes the proof.

Theorem 7. *The interior endemic equilibrium point Q^* of the system (4) guaranteed by theorem (5) is globally asymptotically stable for $R_T > 1$.*

Proof. To prove this theorem,we make use of the Lyapunov functional theorem, to which we show that our interior endemic equilibrium point is the only stationary point of the Lyapunov function. To do that, we change the variable such that: $S_m = x_1,\ I_m = x_2,\ S_{f_s} = x_3,\ I_{f_s} = x_4,\ S_{f_b} = x_5$ and $I_{f_b} = x_6$. Define a function $V(x_1,x_2,x_3,x_4,x_5,x_6)$ by:

$$V = x_1 - x_1^*Inx_1 + x_2 - x_2^*Inx_2 + x_3 - x_3^*Inx_3 + x_4 - x_4^*Inx_4 + x_5 - x_5^*Inx_5 + x_6 - x_6^*Inx_6, \tag{59}$$

this implies that:

$$\frac{\partial V}{\partial x_i} = 1 - \frac{x_i^*}{x_i},\ i = 1,2,3,4,5,6. \tag{60}$$

By equating the partial derivatives to zero and solve for x_i we get: $x_i = x_i^*, i = 1,2,3,4,5,6$.

It is clear that the interior endemic equilibrium point is the only stationary point of the function $V(x_1,x_2,x_3,x_4,x_5,x_6)$.

$$\frac{\partial^2 V}{\partial x_i^2} = \frac{x_i^*}{x_i},\ i = 1,2,3,4,5,6, \tag{61}$$

it is clear that the second partial derivative of the function $V(x_1,x_2,x_3,x_4,x_5,x_6)$ with respect to x_i for all i, is positive, hence the interior endemic equilibrium point is the global minimum point of the function $V(x_1,x_2,x_3,x_4,x_5,x_6)$, for all $v \in \mathbb{R}^6_+$ and $V' = 0$ only at the interior endemic equilibrium point.
Further, the function $V(x_1,x_2,x_3,x_4,x_5,x_6)$ is continuous everywhere by properties of continuous functions and has continuous first order partial derivatives. Therefore to conclude that the the function

$V(x_1, x_2, x_3, x_4, x_5, x_6)$ is a Lyapunov function, it requires us to prove that, the rate of change of the function $V(x_1, x_2, x_3, x_4, x_5, x_6)$ with respect to time is less than or equal to zero as t approaches infinity. Now

$$V'(t) = \sum (x_i - x_i^*)\frac{x_i'(t)}{x_i(t)},\ i = 1, 2, 3, 4, 5, 6. \tag{62}$$

Since the set of solutions for the system of (4) is bounded, there exist a positive real number k such that: $k = \min x_i,\ i = 1, 2, 3, 4, 5, 6$, hence

$$\sum (x_i - x_i^*)\frac{x_i'(t)}{x_i(t)} \leq \frac{1}{k}\sum (x_i - x_i^*)x_i'(t),\ i = 1, 2, 3, 4, 5, 6. \tag{63}$$

Also, since

$$N'(t) = \Lambda - \mu N, \tag{64}$$

where $0 < N \leq \frac{\Lambda}{\mu}, \Longrightarrow 0 \leq N'(t) < \Lambda$ hence, the derivatives are also bounded, that is $x_i'(t) \in L^\infty$. Thus

$$\frac{1}{k}\sum (x_i - x_i^*)x_i'(t) < \frac{\Lambda}{k}\sum (x_i - x_i^*),\ i = 1, 2, 3, 4, 5, 6. \tag{65}$$

Therefore

$$V'(t) < \frac{\Lambda}{k}\sum (x_i - x_i^*),\ i = 1, 2, 3, 4, 5, 6. \tag{66}$$

Using the result derived from Barbalat lemma (Barbalat 1959),

$$\sum (x_i - x_i^*) \longrightarrow 0,\ i = 1, 2, 3, 4, 5, 6 \text{ as } t \longrightarrow \infty, \tag{67}$$

hence $V'(t) < 0$, this implies that the function $V(x_1, x_2, x_3, x_4, x_5, x_6)$ is a strict Lyapunov function with the endemic equilibrium point only as its stationary point, which is a global minimum point. Thus the endemic equilibrium point Q^* is globally asymptotically stable for $R_T > 1$.

5 Numerical simulations

In this chapter, we carry out detailed numerical simulations using Matlab programming language to assess the effect that certain parameters have on the population size. Using the data in Tab. 1 above we asses the effect

Table 1. Model parameters

Model parameter	Value (Range)	Source
Λ	$0.029\text{yr}^{-1}\times 200000$	Bhunu et al.[1]
μ	0.02yr^{-1}	Bhunu et al.[1]
β_m	0.850yr^{-1}	Catterall,[26]
β_f	0.450yr^{-1}	Weston and Nicol,[29]
β_{f_l}	0.00001yr^{-1}	Assumed
γ	0.000125yr^{-1}	Assumed
α	0.90yr^{-1}	Gulmezoglu and Garner,[27]
ρ	0.505yr^{-1}	NationMaster,[28]

of parameters in the absence of treatment subject to the following initial conditions: $S_m(0) = 100000.0$, $I_m(0) = 1000.0$, $S_{f_s}(0) = 110000.0$, $I_{f_s}(0) = 1300$, $S_{f_b} = 1.0$ and $I_{f_b}(0) = 0.0$. Further we will also assess the effect that *trichomoniasis* have in the community in the presence of treatment. Finally we will do the sensitivity analysis of the reproduction numbers to certain parameters using Monte Carlo Simulations.

Fig. 2 shows the effect of varying the female to male contact rate on infected males, infected straight females and infected bisexual females. The Figure has shown that as we increased the value of β_f the number

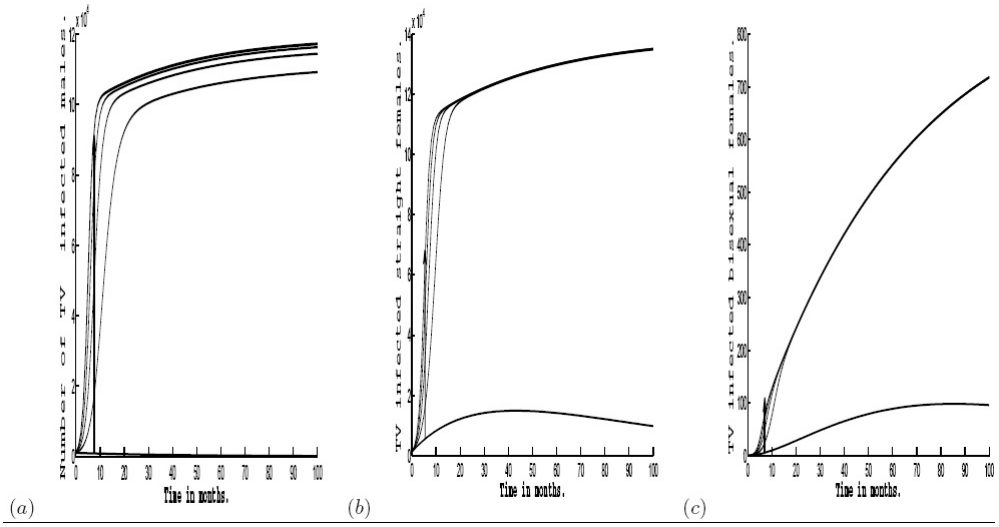


Fig. 2. Effects of varying the female to male contact rate starting from $\beta_f = 0$ to $\beta_f = 1$ with a step size of 0.25 . Parameter values used are in Tab. 1

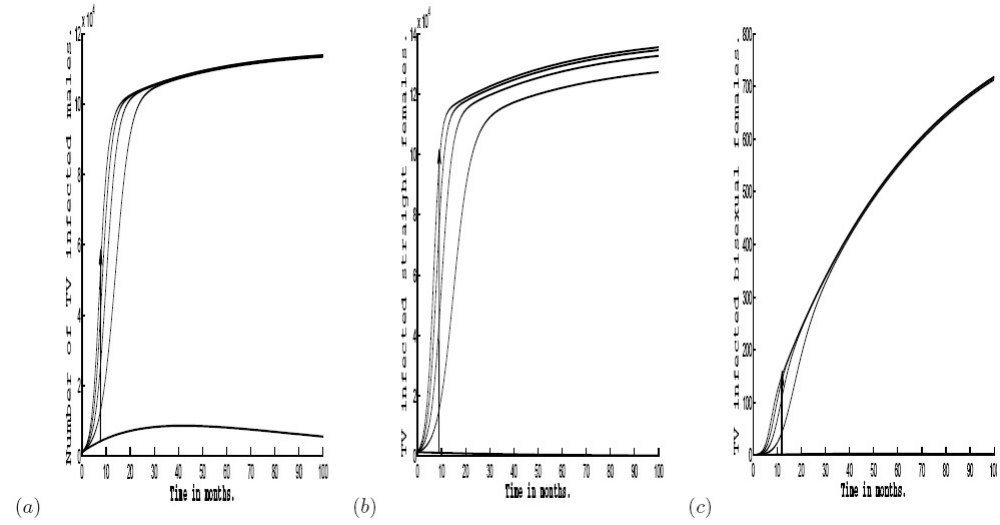
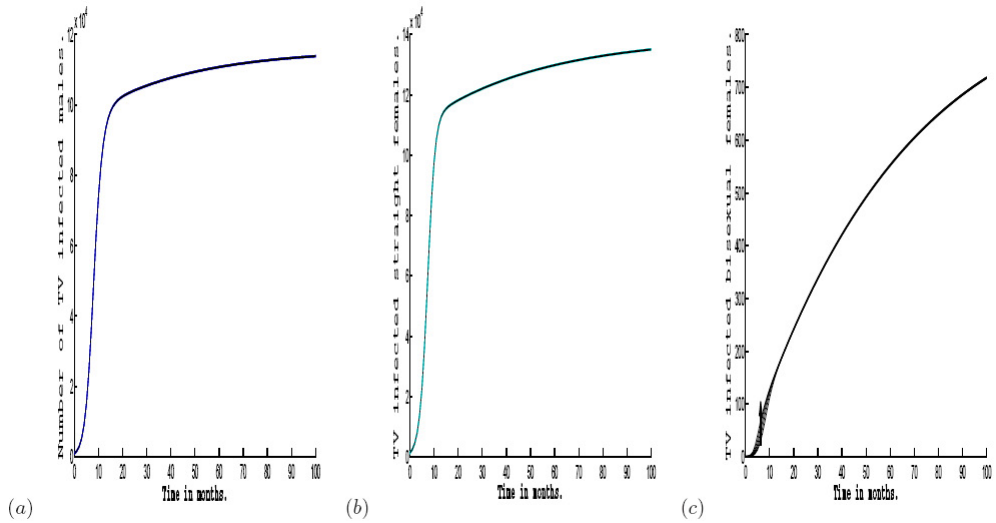


Fig. 3. Effects of varying the male to female contact rate starting from $\beta_m = 0$ to $\beta_m = 1$ with a step size of 0.25 . Parameter values used are in Tab. 1

of infected individuals increases as shown by the arrows pointing upwards on Fig. 2 (a), (b) and (c). However, by comparing Fig. 2 (a), (b) and (c), we can see that straight females are more affected by the female to male contact rate whereas bisexual females are less affected by the same contact rate. This might be the reason that bisexuals has no direct sexual relationship with straight females but they are linked indirectly by males.

Fig. 3 shows the effect of varying the female to male contact rate on infected males, infected straight females and infected bisexual females. The Figure has shown that as we increased the value of β_f the number of infected individuals increases as shown by the arrows pointing upwards on Fig. 3 (a), (b) and (c). However, by comparing Fig. 3 (a), (b) and (c), we can see that straight females are more affected by the female to male contact rate whereas bisexual females are less affected by the same contact rate. This might be the reason that bisexuals has no direct sexual relationship with straight females but they are linked indirectly by males.

Fig. 4 has shown that as the value of β_m increases, the number of infected individuals increases as shown by the arrows pointing upwards on Fig. 4 (a), (b) and (c). As shown in Fig. 4 (a), (b) and (c), the increase in the number of infected straight females leads to the increase in the number of infected males more than bisexual infected females. This might be the fact that straight females have a direct sexual relationship with males.



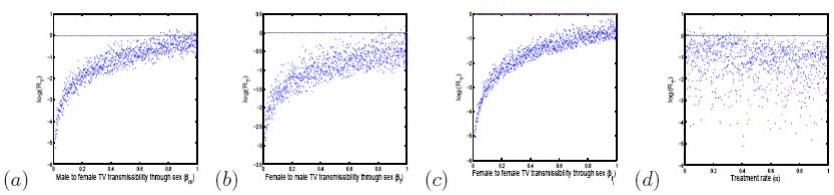
(a) (b) (c)

Fig. 4. Effects of varying the female to female contact rate starting from $\beta_{f_l} = 0$ to $\beta_{f_l} = 1$ with a step size of 0.25 . Parameter values used are in Tab. 1

The parameter β_{f_l} has a negligible effect on males and straight females whereas bisexual females are more affected as shown on Fig. 4 (a), (b) and (c). Also as the female to female contact rate increases the number of infected individuals increases as shown on Fig. 4 (a), (b) and (c).

5.1 Sensitivity analysis

In this section, we look at the sensitivity of the reproduction number to certain parameters. We start by representing graphically the relationships between the parameters that defines the reproduction number and the reproduction number R_T using the Monte Carlo Simulations as shown in Fig. 5.



(a) (b) (c) (d)

Fig. 5. Latin Hypercube Sampling graphs

Fig. 5 (a) shows the correlation between the variable β_m and R_T.It reveals a strong positive relationship between male to female probability of infection and the average number of secondary infected individuals. That is, as the male to female contact rate increases the reproduction also increases.
Fig. 5 (b) is a scatter graph that illustrate the correlation between the female to male contact rate β_f and the reproduction number R_T. It shows that the is a positive correlation between these variables. That is, increase in the value of the female to male contact rate leads to an increase in the average number of *trichomoniasis* infected individuals in a totally susceptible population. However, Fig. 5 (a) and (b) reveals that the reproduction number is more sensitive to the male to female contact rate as compared to the female to male contact rate. This reflects that females are more prone to *trichomoniasis* infection as compared to males.
Similarly, just like as shown on Fig. 5 (a) and (b) , the Scatter graph Fig. 5 (c) also reveals a positive relationship between the female to female contact rate β_{f_l} and the reproduction number R_T. However, the rate at which β_{f_l} increases the value of the reproduction number is negligible as compared to β_m and β_f as shown on the graph Fig. 5. Thus in general an increase in the in the probability of being infected results in an increase in the number of people infected in a totally susceptible population. This might be the case that individuals are

falling into sexual contact with asymptomatic carriers who are not seeking treatment.
The relationship between the treatment rate α and the reproduction number R_T is shown on Fig. 5 (d). The Figure indicates that an increase in the treatment rate decrease the value of the reproduction number thereby reducing the number of TV infected individuals. Fig. 5 (d) clearly indicates that at least 77 percent of treatment is needed to reduce the value of the reproduction number to less than a unity. This indicates that the TV infection can die out if treatment is increased to at least 77 percent thereby controlling TV prevalence. Hence it is advisable for individuals to visit a nearest hospital or medical clinic to seek for their status before they fall into sexual contact. Thus treatment plays a vital role in controlling the rate at which the infection spreads.

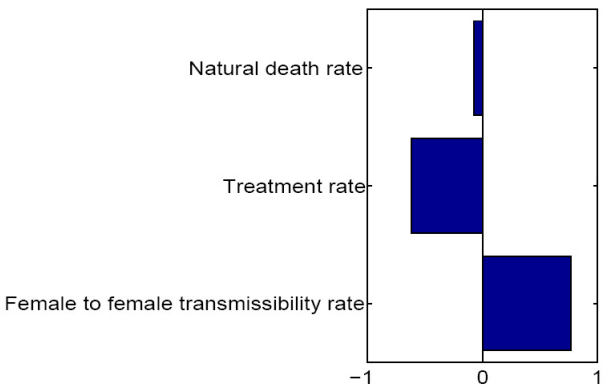


Fig. 6. Sensitivity of R_{f_l} to all parameters using partial rank correlation coefficients

In Fig. 6, partial rank correlation coefficients $(PRCC)$ were calculated to estimate the correlation between the values of female to female reproduction number R_{f_l} and the three parameters α, μ and β_{f_l}. In this Fig. 6 a large $PRCC$ is an indicative of high sensitivity to parameter estimate ($PRCC > 0$ will increase R_{f_l} when the parameters are increased). In contrast, small $PRCC$ reflects low sensitivity ($PRCC < 0$ will decrease the value of R_{f_l} when the parameters are increased). Also, Fig. 6 and shows that the reproduction number R_{f_l} is more sensitive to treatment rate, in this case the $PRCC$ is negative, clearly suggesting that infected individuals should be encouraged to seek treatment so as to reduce TV prevalence. In contrast, the female to female transmissibility rate has a great effect in increasing the rate at which the female to female reproduction numbers increases.

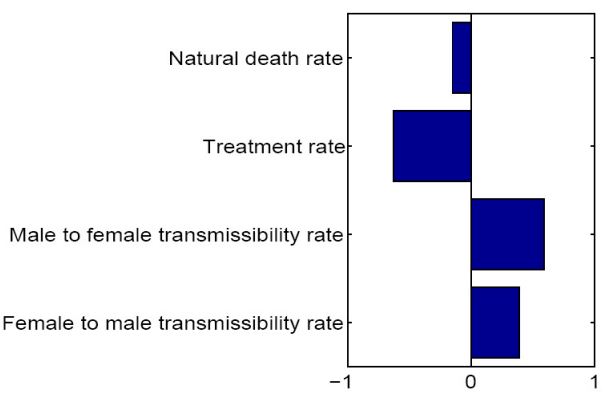


Fig. 7. Sensitivity of R_{f_l} to all parameters using partial rank correlation coefficients

Fig. 7 shows the correlation between the values of R_fm and the four parameters α, μ, β_m and β_f. On Fig. 7 a large $PRCC$ is an indicative of high sensitivity to parameter estimate ($PRCC > 0$ will increase R_{fm} when the parameters are increased). In contrast, small $PRCC$ reflects low sensitivity ($PRCC < 0$ will decrease the value of R_{fm} when the parameters are increased). Also,Fig. 7 shows that the reproduction

number R_{fm} is more sensitive to the treatment rate, this is seen by clearly observing that the $PRCC$ is negative. This shows that the treatment rate has a vital role in reducing the value of the reproduction R_fm when it is increased. This also suggest that infected individuals should be encouraged to seek treatment so as to reduce TV prevalence. Since the reproduction number R_T depends on both R_{f_l} and R_{fm}, it follows directly from the above argument that the reproduction number R_T is also more sensitive to the treatment rate.

6 Conclusion

A mathematical model for the transmission dynamics of *trichomoniasis* in the presence of bisexuality has been presented and its analysis has been dealt with using both numerical and analytical methods. It has been shown that increase in the infection among bisexuals can lead to an increase number of infected individuals amongst heterosexuals. With the help of the Centre-Manifold theorem and the Lyapunov theorem, we managed to show both the local and global stability of the endemic equilibrium point when the reproduction number is greater than a unity. The disease free equilibrium point is shown to be globally asymptotically stable when the reproduction number is less than a unity. Thus we managed to show that the infection dies out when the reproduction number is less than a unity. In contrast the infection will persist if the reproduction number is greater than a unity.

The analysis of the reproduction number together with numerical simulations revealed that the reproduction number is more sensitive to the treatment rate and contact rates as compared to other parameters. The contacts rates have been shown to have negative effect on the population since they lead to an increase the number of infected individuals in a totally susceptible population if they increases. However, we do not have control over this transmissibilities, so it is better to seek treatment to avoid the spread of the infection.

Treatment rate has been shown to have a positive effect on the total population if it is increased especially if it is at least 77 percent. This so since the sensitivity analysis reveals that the treatment rate can reduce the value of the reproduction number if it is increased. Hence it is best to encourage individuals to seek treatment so as to reduce the transmission rate of *trichomoniasis* infection. So the community is encouraged to provide enough treatment especially in the form of *tinidazole* and *metronidazole* so as to reduce the average number of infected individuals in a totally susceptible population.

The mathematical arguments have also indicated that females are more prevalent to *trichomoniasis* infection than males. However, it does not necessarily mean that males can not be infected, but the may be asymptomatic carriers. Hence males should also seek for treatment in order to prevent the spread of *trichomoniasis* infection.

However just like an other model, we can not say the model is complete, it can be extended to include such factors as the use of condoms, intervention of educational services and the fact that individuals can be born naturally as homosexuals.

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