

On a Two-Sex Model for Gonorrhea Transmission Dynamics Incorporating Treatment and Condom Use

Patrick Noah Okolo^{1*} Onoja Abu²

1. Department of Mathematics and Statistics, Nasarawa State Polytechnic, Lafia, Nigeria
2. Department of Mathematics/Statistics, Federal Polytechnic, Idah, Nigeria

*Email of the corresponding author: patricknoahokolo@yahoo.com; Tel No: +2347039681282

Abstract

In this study we developed a two sex model for gonorrhea transmission dynamics incorporating treatment and condom use as control measures using a system of ordinary differential equations. We further derive an epidemic threshold as the effective reproduction number, R , using the next generation method. We established both the disease-free equilibrium and endemic equilibrium states using the linearization method and the manifold theory respectively. From the analysis of the model and results, it was shown that, the disease free equilibrium state is locally and asymptotically stable if $R < 1$. The implication is that gonorrhea can be eliminated from the population if the effective reproduction number, R , is less than unity. The endemic equilibrium state is locally and asymptotically stable for $R > 1$ and this implies that gonorrhea disease can spread and there could be disease persistence.

Key words: Gonorrhea disease, effective reproduction number, equilibrium state, centre manifold theory

1. Introduction

Gonorrhea is a sexually transmitted disease (STD) caused by a bacterial species called *Neisseria gonorrhoeae*. It can infect both men and women; and cause infections in the genitals, rectum, and throat. It is a very common infection, especially among young people ages 15-24 years. Gonorrhea can be contracted by having unprotected anal, vaginal, or oral sex with someone who has gonorrhea. The preventive cautions are avoiding sex with persons who have tested positive and using latex condoms and dental dams the right way every time you have sex (CDC, 2013; CDC, 2014).

The incubation period for gonorrhea is 2-14 days. Many men and most women that have gonorrhea are asymptomatic. If present, women may experience a white, yellow, or green discharge from the vagina, pain during intercourse, and lower abdominal pain. For men symptoms include discharge, inflammation of the urethra. Itching and dysuria may also be experienced. In some cases, gonorrhea may cause skin lesions and infection of the joints. If left untreated, women can acquire pelvic inflammatory disease (PID), which may cause serious complications with pregnancy. Untreated gonorrhea can also increase a person's risk of acquiring HIV (CDC, 2015).

Globally, the gonorrhea disease burden is high. About 87.7 and 106.1 cases of gonorrhea were reported in 2005 and 2008 respectively [WHO, 2008; WHO, 2012]. Likelihood of transmission by various routes differs. The chance of male to female through semen is approximately 50%-70% per episode of vaginal intercourse. The likelihood of female vagina to male urethra is approximately 20% per episode of vaginal intercourse and increases to approximately 60%-80% after 4 or more exposures. Rectal intercourse transmission rates have not been quantified, but rectal intercourse appears to be an efficient mode of transmission. Pharyngeal gonorrhea is readily acquired by fellatio but less efficiently acquired by cunnilingus. Perinatal transmission (mother to infant) can occur during vaginal delivery CDC, (2013).

The plan of this article is as follows. Section 2 is devoted to model formulation. Section 3 is devoted to analysis and results. Section 4 is devoted to discussion of results. Concluding remarks are made in section 5.

2. Model Formulation

Mathematical models for gonorrhea transmission and control abound. We do not claim to give an exhaustive list in this article. Lajmanovich and Yorke (1976) formulated a deterministic model for gonorrhea in a non-homogeneous Population, where the population is divided into n homogeneous groups. Hethcote and Yorke (1984) did a review of model articles on gonorrhea transmission dynamics and control. Lima and Torres (1995)

proposed models for the transmission dynamics of gonorrhoea in a homosexually-active population to investigate among other things the effect of a partially effective vaccine on gonorrhoea dynamics, the role of an antibiotic-driven mutation with respect to the survival and spread of resistant gonorrhoea strains and the impact of multiple strains of gonorrhoea. A mathematical model to explore the co-interaction of gonorrhoea and HIV in the presence of antiretroviral therapy and gonorrhoea treatment was designed by Mushayabasa (2010). Henry (2012) developed a population based model of gonorrhoea and interventions against increased antibiotic resistance. Recently, Adesanya et al (2016) formulated a mathematical model for gonorrhoea and performed mathematical and sensitivity analysis of efficacy of condom on the transmission of gonorrhoea disease. Our interest in this paper is a two-sex version of the model by Adesanya et al (2016).

2.1 The Variable and Parameter of the Model

The variables and parameters are defined as follows.

x_1 = the number of susceptible females,

x_2 = the number of susceptible males,

x_3 = the number of infected females,

x_4 = the number of infected males,

x_5 = the number of recovered females,

x_6 = the number of recovered males,

ξ_1, ξ_2 = recruitment rates into female and males,

η_1, η_2 = efficacy rates of condoms for females and males respectively,

θ_1, θ_2 = condom compliance rates for females and males respectively,

β_1, β_2 = transmission coefficients for females and males respectively,

γ_1, γ_2 = immunity loss rates for females and males respectively,

τ_1, τ_2 = natural recovery rates,

μ = natural mortality rate,

σ_1, σ_2 = treatment rates for females and males respectively,

c_1, c_2 = average numbers of sex partners in female and male populations .

2.2: Model Equations

We propose our model as follows.

$$\frac{dx_1}{dt} = \xi_1 - \eta_1 \theta_1 c_1 \beta_1 x_1 x_4 - \mu x_1 + \gamma_1 x_5 \quad (1)$$

$$\frac{dx_2}{dt} = \xi_2 - \eta_2 \theta_2 c_2 \beta_2 x_2 x_3 - \mu x_2 + \gamma_2 x_6 \quad (2)$$

$$\frac{dx_3}{dt} = \eta_1 \theta_1 c_1 \beta_1 x_1 x_4 - (\mu + \sigma_1 + \tau_1) x_3 \quad (3)$$

$$\frac{dx_4}{dt} = \eta_2 \theta_2 c_2 \beta_2 x_2 x_3 - (\mu + \sigma_2 + \tau_2) x_4 \quad (4)$$

$$\frac{dx_5}{dt} = \sigma_1 x_3 - (\mu + \gamma_1) x_5 \quad (5)$$

$$\frac{dx_6}{dt} = \sigma_2 x_4 - (\mu + \gamma_2) x_6 \quad (6)$$

3. Results

The analysis and the ensuing results are as follows.

3.1: The Disease-Free Equilibrium State

The disease-free equilibrium state E_0 for the model (1) – (6) is given by

$$E_0 = (\bar{x}_1, \bar{x}_2, \bar{x}_3, \bar{x}_4, \bar{x}_5, \bar{x}_6) = \left(\frac{\xi_1}{\mu}, \frac{\xi_1}{\mu}, 0, 0, 0, 0 \right).$$

3.2 Effective Reproduction Number

To compute the effective reproduction number, we use the recipe by Van den Driessche and Watmough (2002), Heffernan (2005) and Ameh (2009). The appearance of new infections is given by the vector

$$F(x) = \begin{pmatrix} \eta_2 \theta_2 c_2 \beta_2 x_2 x_3 \\ \eta_1 \theta_1 c_1 \beta_1 x_1 x_4 \end{pmatrix}$$

and the vector of other transfer terms is given by

$$V(x) = \begin{pmatrix} (\mu + \sigma_2 + \tau_2)x_4 \\ (\mu + \sigma_1 + \tau_1)x_3 \end{pmatrix}.$$

The effective reproduction number for our model is given by

$$R = \sqrt{\left(\frac{\eta_1 \theta_1 c_1 \beta_1 \xi_1}{(\mu + \sigma_1 + \tau_1)} \right) \left(\frac{\eta_2 \theta_2 c_2 \beta_2 \xi_2}{(\mu + \sigma_2 + \tau_2)} \right)}$$

3.3 Local Stability of the Disease-Free Equilibrium (DFE)

The Jacobian matrix for our model at the disease-free equilibrium is given by

$$J_{E_0} = \begin{pmatrix} -\mu & 0 & 0 & -\eta_1 \theta_1 c_1 \beta_1 \bar{x}_1 & \gamma_1 & 0 \\ 0 & -\mu & -\eta_2 \theta_2 c_2 \beta_2 \bar{x}_2 & 0 & 0 & \gamma_2 \\ 0 & 0 & -(\mu + \sigma_1 + \tau_1) & \eta_1 \theta_1 c_1 \beta_1 \bar{x}_1 & 0 & 0 \\ 0 & 0 & \eta_2 \theta_2 c_2 \beta_2 \bar{x}_2 & -(\mu + \sigma_2 + \tau_2) & 0 & 0 \\ 0 & 0 & \sigma_1 & 0 & -(\mu + \gamma_1) & 0 \\ 0 & 0 & 0 & \sigma_2 & 0 & -(\mu + \gamma_2) \end{pmatrix}.$$

Theorem 1 If $R < 1$, then the disease-free equilibrium state E_0 is locally and asymptotically stable.

Proof: To prove theorem 1, it suffices to show that all the eigenvalues of the characteristic equation for the Jacobian matrix J_{E_0} above have negative real parts.

The corresponding characteristic equation to J_{E_0} in λ is given by

$$\begin{vmatrix} -(\mu + \lambda) & 0 & 0 & -\eta_1 \theta_1 c_1 \beta_1 \bar{x}_1 & \gamma_1 & 0 \\ 0 & -(\mu + \lambda) & -\eta_2 \theta_2 c_2 \beta_2 \bar{x}_2 & 0 & 0 & \gamma_2 \\ 0 & 0 & -(\mu + \sigma_1 + \tau_1 + \lambda) & \eta_1 \theta_1 c_1 \beta_1 \bar{x}_1 & 0 & 0 \\ 0 & 0 & \eta_2 \theta_2 c_2 \beta_2 \bar{x}_2 & -(\mu + \sigma_2 + \lambda) & 0 & 0 \\ 0 & 0 & \sigma_1 & 0 & -(\mu + \gamma_1 + \tau_2 + \lambda) & 0 \\ 0 & 0 & 0 & \sigma_2 & 0 & -(\mu + \gamma_2 + \lambda) \end{vmatrix} = 0.$$

$$\text{Therefore, } (\mu + \lambda)(\mu + \lambda)(\mu + \gamma_1 + \lambda)(\mu + \gamma_2 + \lambda) \begin{vmatrix} -(\mu + \sigma_1 + \tau_1 + \lambda) & \eta_1 \theta_1 c_1 \beta_1 \bar{x}_1 \\ \eta_2 \theta_2 c_2 \beta_2 \bar{x}_2 & -(\mu + \sigma_2 + \lambda) \end{vmatrix} = 0.$$

Now, $\lambda_1 = -\mu$, $\lambda_2 = -\mu$, $\lambda_3 = -(\mu + \gamma_1)$ and $\lambda_4 = -(\mu + \gamma_2)$ are four of the eigenvalues.

$$\text{Let } A = \begin{pmatrix} -(\mu + \sigma_1 + \tau_1) & \eta_1 \theta_1 c_1 \beta_1 \bar{x}_1 \\ \eta_2 \theta_2 c_2 \beta_2 \bar{x}_2 & -(\mu + \sigma_2 + \tau_2) \end{pmatrix}, \text{ then}$$

$$\text{Trace}(A) = -(2\mu + \sigma_1 + \tau_1 + \sigma_2 + \tau_2)$$

and

$$\text{Det}(A) = (\mu + \sigma_1 + \tau_1)(\mu + \sigma_2 + \tau_2) - \eta_1 \theta_1 c_1 \beta_1 \bar{x}_1 \eta_2 \theta_2 c_2 \beta_2 \bar{x}_2 = (\mu + \sigma_1 + \tau_1)(\mu + \sigma_2 + \tau_2) \left[1 - \frac{\eta_1 \theta_1 c_1 \beta_1 \bar{x}_1 \eta_2 \theta_2 c_2 \beta_2 \bar{x}_2}{(\mu + \sigma_1 + \tau_1)(\mu + \sigma_2 + \tau_2)} \right]$$

$= (\mu + \sigma_1)(\mu + \sigma_2)[1 - R^2] > 0$ if $R < 1$, since $R < 1 \Rightarrow R^2 < 1$. Thus, all the characteristic roots are negative, provided $R < 1$. Therefore, the disease-free equilibrium state is locally asymptotically stable for $R < 1$.

3.4: Existence and Stability of Endemic Equilibrium (EE)

In this section, we investigate the bifurcation behavior of a general epidemic model around the critical value $R_0 = 1$ in a neighbourhood of a disease-free equilibrium E_0 . Let $\varphi = R_0 - 1$ and rewrite our general epidemic model in the following way:

$$x' = f(x, \varphi)$$

with the assumption that f is continuously differentiable at least twice. We have the following result. Let $D_x f(E_0, 0)$ be the matrix of partial derivatives of f at the disease-free equilibrium. Also let u and v be the right and the left eigenvectors of $D_x f(E_0, 0)$ respectively.

Theorem 2: (Mukandavire *et al* 2010). Consider the disease transmission model defined by (1.1 – 1.6) with the function $f(x, \varphi)$, φ is the parameter as described from the foregoing. Assume that the zero eigenvalue of $D_x f(E_0, 0)$ is simple. Let

$$a = \sum_{k,i,j=1}^n v_k u_i v_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(E_0, 0), \quad b = \sum_{k,i=1}^n v_k u_i \frac{\partial^2 f_k}{\partial x_i \partial \varphi}(E_0, 0). \quad \text{Assume that } b \neq 0. \text{ Then, there exists } \delta > 0 \text{ such that}$$

- (i) If $a > 0, b > 0$, when $\varphi < 0$ with $|\varphi| < 1$, E_0 is locally asymptotically stable, and there exists a positive unstable equilibrium; when $0 < \varphi < 1$, E_0 is unstable and there exists a negative asymptotically stable equilibrium;
- (ii) If $a < 0, b < 0$, when $\varphi < 0$ with $|\varphi| < 1$, E_0 is unstable; when $0 < \varphi < 1$, E_0 is asymptotically stable, and there exists a positive unstable equilibrium;
- (iii) If $a > 0, b < 0$, when $\varphi < 0$ with $|\varphi| < 1$, E_0 is unstable, and there exists a locally asymptotically stable negative equilibrium; when $0 < \varphi < 1$, E_0 is stable and a positive unstable equilibrium appears
- (iv) If $a < 0, b > 0$, when φ changes from negative to positive, E_0 changes its stability from stable to unstable. Corresponding negative equilibrium becomes positive and locally asymptotically stable.

Theorem 2 is applied in the sequel. The left eigenvector for J_{E_0} is given by $v = (v_1, v_2, v_3, v_4, v_5, v_6)^T$, where

$$v_1 = v_2 = v_5 = v_6 = 0; \quad v_3 = 1, \quad v_4 = \frac{(\mu + \sigma_1)}{\eta_2 \theta_2 c_2 \beta_2 \bar{x}_2}. \quad \text{Also, the right eigenvector for } J_{E_0} \text{ is given by } u = (u_1, u_2, u_3, u_4, u_5, u_6)^T, \text{ where}$$

$$u_1 = < 0; \quad u_2 = < 0; \quad u_3 = 1, \quad u_4 = \frac{(\mu + \sigma_2)}{\eta_2 \theta_2 c_2 \beta_2 \bar{x}_2}, \quad u_5 = \frac{\sigma_1}{(\mu + \sigma_1 + \tau_1)}, \quad u_6 > 0.$$

It is clear from (1.1 – 1.6) that

$$\frac{\partial^2 f_3}{\partial x_1 \partial x_4} = \eta_1 \theta_1 c_1 \beta_1; \quad \frac{\partial^2 f_4}{\partial x_2 \partial x_3} = \eta_2 \theta_2 c_2 \beta_2, \quad \frac{\partial^2 f_3}{\partial x_4 \partial \beta_1} = \eta_1 \theta_1 c_1 \bar{x}_1 \text{ and } \frac{\partial^2 f_4}{\partial x_3 \partial \beta_2} = \eta_2 \theta_2 c_2 \bar{x}_2$$

Therefore, $a = v_3 u_1 v_4 \eta_1 \theta_1 c_1 \beta_1 + v_3 u_2 v_4 \eta_2 \theta_2 c_2 \beta_2 < 0$ and $b = v_3 u_4 \eta_1 \theta_1 c_1 \bar{x}_1 + v_4 u_3 \eta_2 \theta_2 c_2 \bar{x}_2 > 0$.

Therefore, by item (iv) of theorem 2, the disease-free equilibrium becomes unstable and the endemic equilibrium becomes locally asymptotically stable as R_0 changes values from less than one to values slightly greater than one.

4. Discussion of Results

Gonorrhoea is a bacterial disease and has grave consequences on the health of adults. In this article a mathematical model has been developed and analyzed. The main results are in Theorems 1 and 2. Fundamental in our results is the effective reproductive number R . The effective reproductive number is a threshold parameter that determines whether an epidemic can take off or not. Based on this threshold, we have proved theorem 1 and established that the disease-free equilibrium state is locally asymptotically stable for values of R less than one. This shows that the disease can be eliminated from the population. For the stability of the endemic equilibrium state, the centre manifold Theorem 2 is applied. The results of equilibrium analysis, using centre manifold theory showed that the endemic equilibrium state is locally and asymptotically stable for $R_0 > 1$. This implies that gonorrhoea disease can spread and there could be disease persistence.

5. Conclusion

In this article, a mathematical model for gonorrhoea transmission dynamics considering condom and treatment parameters as controls is formulated and analyzed. The model can be seen in section 2.1. The effective reproductive number was computed by the next generation method. This can be seen in section 2.3. The local stability analysis of the equilibrium states was carried out. The main results can be found in theorems 1 and 2. The results show that the disease-free state (respectively endemic equilibrium state) is locally and asymptotically stable for $R < 1$ (respectively $R > 1$).

Reference

- Ameh, E.J. (2009). The basic reproduction number: Bifurcation and Stability, PGD project at AIMS.
- Adesanya, A.O, Olopade, I.A, Akanni J.O, Oladapo, A.O and Omoloye, M.A. (2016). Mathematical and Sensitivity Analysis of Efficacy of Condom on the Transmission of Gonorrhoea Disease. *IJIR*, 2(11):367-375
- CDC (2013). Gonorrhoea factsheet
- CDC (2014). Gonorrhoea factsheet
- CDC (2015). "Gonorrhoea - CDC Fact Sheet." *Centers for Disease Control and Prevention*. Centers for Disease Control and Prevention, 16 Dec. 2014. Web. 30 Apr. 2015.
<<http://www.cdc.gov/std/Gonorrhoea/STDFactgonorrhoea.htm>>.
- Heffernan, J.M. (2005). Perspectives on the basic reproductive ratio. *J. R. Soc. Interface*, 2, 281-293.
- Henry, C.N. (2012). Population based model of gonorrhoea and interventions against increased antibiotic resistance. M.Sc. Dissertation, Virginia Commonwealth University
- Hethcote, H.W and Yorke, J. A. (1984). *Gonorrhoea transmission dynamics and control*. Springer-Verlag
- Lajmanovich, A. and Yorke, J.A. (1976). A deterministic model for gonorrhoea in a non-homogeneous population. *Mathematical Biosciences*. 28: 221-236
- Lima, S.K. and Torres, M. (1995). *Models for the Transmission Dynamics of Gonorrhoea in a Homosexually-Active Population*
- Mushayabasaa, S., Tchuenche, J.M., Bhunua, C.P. . Ngarakana-Gwasira, E. (2010). Modeling gonorrhoea and HIV co-interaction. *BioSystems* 103: 27-37
- Mukandavire, Z., Das, P., Chiyaka, C. and Nyabadza, F. (2010). Global analysis of an HIV/AIDS epidemic model, *WJMS*, 6(3):231-240, UK.
- Vanden Driessche, P., Watmough, J., (2002). Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Math. Bio Sci.* 180, 29-48.
- WHO (2008). *Global incidence and prevalence of selected curable sexually transmitted infections*. © World Health Organization 2012
- WHO (2012). *Baseline report on global sexually transmitted infection surveillance 2012* © World Health Organization 2013