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LOCAL STABILITY ANALYSIS OF NEISSERIA GONORRHEA DISEASE MODEL IN WEST AFRICA

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ABSTRACT: *Gonorrhea is a sexually transmitted infection caused by bacterium Neisseria gonorrhea. Today, this life threatening infectious disease is a great challenge to the society.In this article, we developed treatment epidemic model for Nesseria gonorrhea disease in order to gain insight into the transmission dynamics and understand epidemic situation and suggest control measures. We analyzed Local stability of the model and the basic reproduction number* R_0 *using next generation matrix method. The disease free equilibrium was determined to be locally asymptotically stable for* $R_0 < 1$ *and unstable for*$R_0 > 1$.

KEYWORDS: Neisseria ghonorrhea, local stability, endemic equilibrium, treatment, vaccination, basic reproduction.

INTRODUCTION

Gonorrhea is a sexual transmission infectious disease; it is caused by a bacterium known as *Neisseria gonorrhea,* which invades the genital organs and reproductive track causing inflammation of the tube which carries sperm and infertility [1]. New cases of gonorrhea diagnosed each year are estimated to be 78 million; in the united states alone there an estimated 820,000 new gonorrhoea infections each year while in 9963, WHO found Logos (Nigeria) to have the highest gonorrhea in the world [2-3]. Historically, gonorrhoea was discovered in 1792, in Edinburg where the surgeon Benjamin Bell clearly differentiated it from syphilis infectious disease [4]. It does not only invade the reproduce track, but can also attack throat, mucous membranes of the eyes, throat, mouth rectum and anus [3]. Untreated gonorrhea in female may lead to pelvic inflammatory disease (PID) which causes permanent damage to the reproductive organ leading to infertility while in male it develop a epididymitis condition while causes fever, severe pain and swelling [5]. Gonorrhea is transmitted sexually through the penis, vagina, mouth or anus of infected individuals spread of gonorrhoea does not depend on ejaculation; it does not have to occur for gonorrhea to be spread of acquired. Gonorrhea can also be transmitted prenatally from infected mother to baby during child delivery. The treated individuals may be reinfected if sexual contact occurs between them and infected person [6]. Untreated gonorrhea infections in human increase a person's risk of acquiring or transmitting HIV, which may lead to AIDs [7]. If a child contacts gonorrhea from infected mother during childbirth, it can cause blindness, joint infection in the body or a life threatening blood infection

in the child, urgent treatment of gonorrhea infection in pregnant woman as soon as it is discovered reduces the risk of above complications [8]. The major signs and symptoms of gonorrhea includes Vagina discharge, burning during urination, low abdominal pain, pus discharge from the male genital organ or bleeding, unusual sores, inflammation of the genital organ [1] [6]. can cause blindness, joint infection in the body or a life threatening blood infection in the child, urgent treatment of gonorrhea infection in pregnant woman as soon as it is discovered reduces the risk of above complications [8]. The major signs and symptoms of gonorrhea includes Vagina discharge, burning during urination, low abdominal pain, pus discharge from the male genital organ or bleeding, unusual sores, inflammation of the genital organ [1] [6].

The surest way to prevent transmission of gonorrhea or other sexually transmitted diseases is to abstain from sexual activities or to be a long term mutually monogamous relationship with a tested partner who is uninfected [6]. According to [11] Bersero is a meningococcal B vaccine approved in the united states since 2015, induces antibodies in humans that target Neisseria gonorrheae. Kate L, Seib, PhD, associated professor and research leader in the institute for glycomics at Griffith university in Southport, Australia, and colleagues the ability of Bexsero (Glaxosmith Kline) to illicit and immune response against N. gonorrhea and discovered it very protective. The vaccine was approved in the U.S for people between 10 to 25 years [11]. According to [12], developed a continuous transmission among homosexuals. They equally applied a non – standard discretization method to formulate a discrete time model and the results of their models were compared. According to [13] formulated a mathematical model of Gonorrhea infection. They concluded that the transmission of the disease entails interactions of the susceptible and the infected individuals in a population [14]. Studied a simple non – linear first order ordinary differential equations model for gonorrhea that determine the growth rates of promiscuous and infected humans in a homosexual population. They also carried out numerical simulation to discuss the effect of treatment rate and infective rate on the transmission and control of the gonorrhea disease.
The main of this research work is formulate vaccination treatment model that can be used to study the transmission dynamics of disease in order to suggest control measure to the deadly sexual transmission disease called Gonorrhea.

Model Assumption

- The population is closed (No migration and emigration)
- The population is homosexual
- Treatment is so effective that no disease induced mortality in the treated class.
- Age, social status and sex do not prevent one from be infected
- The vaccinated humans can be susceptible due to vaccine failure
- Disease induced mortality occurs only in the infected class

Model Description

The total population N(t) at time t is divided into six epidemiological groups namely Susceptible Individuals (S), Vaccinated Individual (V), Exposed Individuals (E), Infected Individuals (I), Treated Individual (T) and recovered individual (R).

A susceptible individual is recruited into the susceptible population through the rate λ susceptible individual become vaccinated through the rate β and due to vaccine failure the vaccinated individual becomes susceptible again through the rate α. ω is the rate at which a susceptible individual becomes exposed to the disease.θ i the rate at which exposed individuals becomes infected, γ and θ are the rate at which infected individual becomes treated and rate at which treated individuals recover from the disease through the rate ρ the natural death rate and disease induced death rate are μ_1 and μ_2 respectively .

Definitions of Variables and Parameters Used in the Model

Table 1: Definitions of Variables and Parameters

Variable / Parameter	Description
V	Total number of vaccinated individual at time t
S	Total number of susceptible individuals at time t
E	Total number of exposed individuals at time t
I	Total number of treated individuals at time t
R	Total number of recovered individuals at time t
N	Total number of population at time t
λ	Recruitment rate
β	Rate of vaccination
α	Rate of susceptible due to vaccine failure
ω	Rate at which susceptible individuals become exposed
θ	Rate at which exposed individuals becomes infected
γ	Treatment rate
ρ	Rate at which recovered individuals become susceptible
ϕ	Recovery rate
μ_1	Natural death rate
μ_2	Disease induced death rate
a	Rate of infection
K	Per capital contact rate

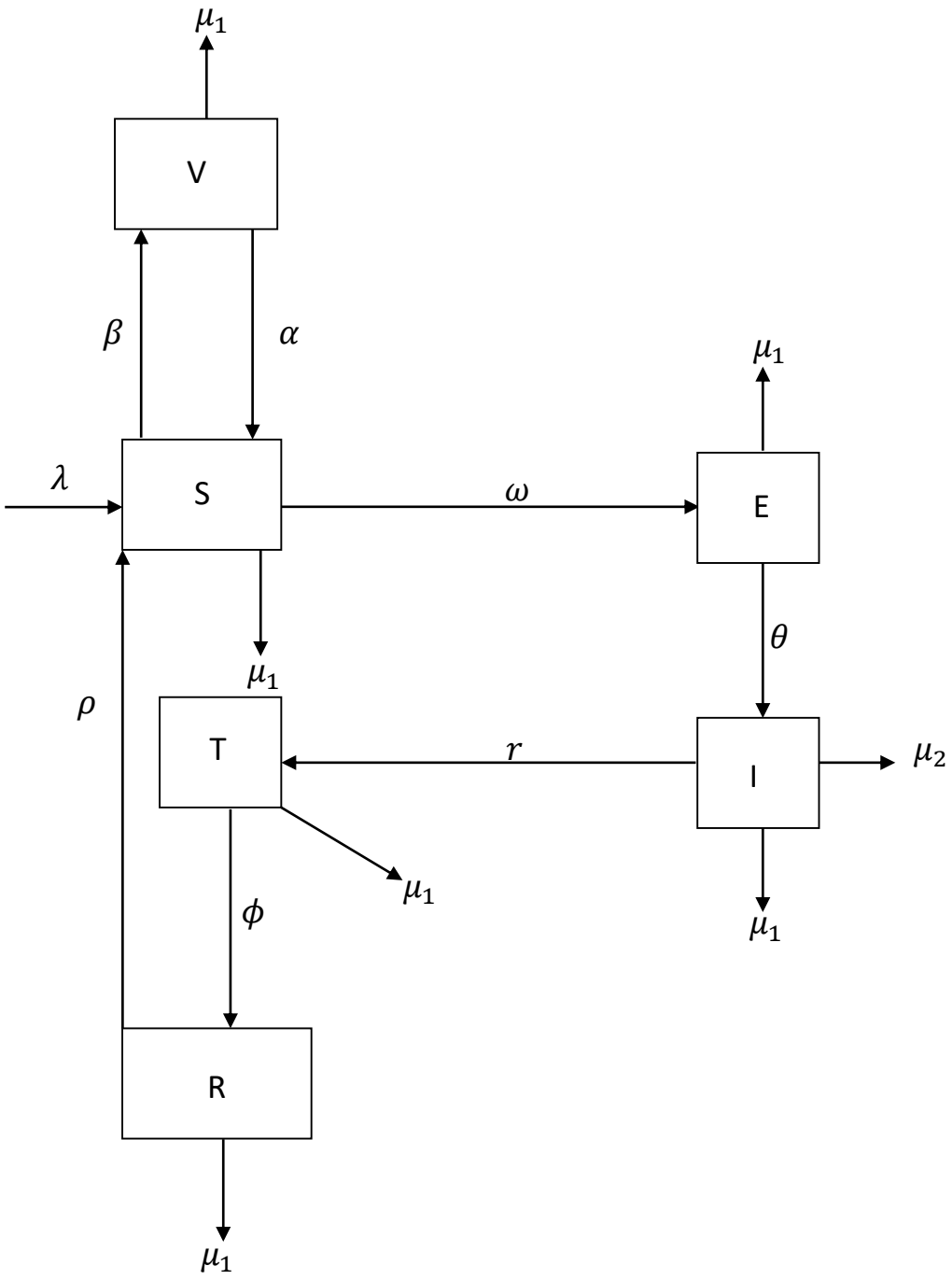

Figure 1: Schematic Diagram of the Model

$$\frac{dv}{dt} = \beta s - (\mu_1 + \alpha)v \quad 1$$

$$\frac{ds}{dt} = \lambda + v + \rho R - (\mu_1 + \beta + \omega)S \quad 2$$

$$\frac{dE}{dt} = \omega s - (\mu_1 + \theta)E \quad 3$$

$$\frac{dI}{dt} = \theta E - (\mu_1 + \mu_2 + r)I \quad 4$$

$$\frac{dT}{dt} = rI - (\mu_1 + \phi)T \quad 5$$

$$\frac{dR}{dt} = \phi T - (\mu_1 + \rho)R \quad 6$$

$$N(t) = V(t) + S(t) + E(t) + I(t) + T(t) + R(t) \quad 7$$

The initial conditions are

$$V(0) \geq 0, S(0) > 0, E(0) \geq 0, I(0) \geq 0, T(0) \geq 0, R(0) \geq 0$$

3. Results

Existence of Equilibrium State

At Equilibrium State

$\frac{dV}{dt} = \frac{dS}{dt} = \frac{dT}{dt} = 0$ $\frac{dR}{dt} = 0$ i.e

$$\frac{dV}{dt} = \beta S - (\mu_1 + \alpha)v \quad 8$$

$$\frac{dS}{dt} = \lambda + \alpha V + \rho R - (\mu_1 + \beta + \omega)S = 0 \quad 9$$

$$\frac{dE}{dt} = \omega s - ((\mu_1 + \theta)E = 0 \quad 10$$

$$\frac{dI}{dt} = \theta E - (\mu_1 + \mu_2 + r)I = 0 \quad 11$$

$$\frac{dT}{dt} = rI - (\mu_1 + \phi)T = 0 \quad 12$$

$$\frac{dR}{dt} = \phi T - (\mu_1 + \rho)R = 0 \quad 13$$

Disease free equilibrium points (DFE)

If there is no existence of disease, it implies that $I^* = 0 \quad T^* = 0 \quad R^* = 0$

Hence, $\varepsilon f = (V^*,\ S^*,\ E^*,\ T^*,\ I^*,\ R^*)$

$$= \left[\frac{-\beta\lambda}{[\alpha\beta-(\mu_1+\alpha)(\mu_1+\beta+\omega)]}, \frac{-\lambda}{(\mu_1+\beta+\omega)-\alpha\beta}, 0, 0, 0, 0\right]$$

Basic reproduction number of the model (R_0)

The basic reproduction number (R_0) is defined as the effective number of secondary infections caused by any infected individual in the whole duration of infectiousness [1] the above definition is for the models that determine spreads analysis of infectious disease in population. We then compute matrix FV^{-1} defined as the next generation matrix (Diekmann etal, 1990).

Where $fi = \begin{pmatrix} \frac{\delta\rho}{N}(E+I+T)S_O \\ 0 \end{pmatrix} \qquad Vi = \begin{pmatrix} (\mu_1+\theta)E \\ -\theta E + (\mu_1+\mu_2+\gamma)I \end{pmatrix}$

Differentiating fi and Vi w.r.t E and I we have.

$$F = \begin{bmatrix} \frac{\delta\rho}{N}S_O & \frac{\delta\rho}{N}S_O \\ 0 & 0 \end{bmatrix} \quad V = \begin{bmatrix} \mu_1 + \frac{\delta\rho}{N}S_O & 0 \\ -\theta & \mu_1+\mu_2+\gamma \end{bmatrix}$$

$$V^{-1} = \begin{bmatrix} \frac{1}{(\mu_1+\theta)} & 0 \\ 0 & \frac{1}{(\mu_1+\mu_2+\gamma)} \end{bmatrix}$$

$$FV^{-1}\begin{bmatrix} \frac{\delta\rho(\mu_1+\theta)}{N}S_O & \frac{-\theta\delta\rho}{N}S_O \\ 0 & 0 \end{bmatrix}$$

$$[FV^{-1} - \lambda I] = \begin{bmatrix} -\lambda & 0 \\ 0 & \frac{\theta}{(\mu_1+\mu_2+\gamma)} - \lambda \end{bmatrix}$$

$$\begin{bmatrix} \frac{\delta\rho(\mu_1+\theta)}{N}S_O - \lambda & \frac{-\theta\delta\rho}{N}S_O \\ 0 & -\lambda \end{bmatrix} = 0$$

$$R_{0=}\frac{\delta\rho(\mu_1+\theta)}{N}S_O$$

Endemic equilibrium point.

From equation 8 to 13 we have

$$V^* = \frac{N\beta(\mu_1+\theta)(\mu_1+\phi)(\mu_1+\mu_2+\gamma)}{ak\omega(\mu_1+\alpha)(\mu_1+\phi+\gamma)}$$

$$S^* = \frac{N\beta(\mu_1+\theta)(\mu_1+\phi)(\mu_1+\mu_2+\gamma)}{ak\omega(\mu_1+\phi+\gamma)}$$

$$E^\infty = \frac{N(\mu_1+\phi)(\mu_1+\mu_2+\gamma)}{ak(\mu_1+\phi+\gamma)}$$

$$I^* = \frac{N\theta(\mu_1+\phi)(\mu_1+\mu_2+\gamma)}{ak(\mu_1+\phi+\gamma)}$$

$$T^* = \frac{N\theta\gamma(\mu_1+\phi)}{ak(\mu_1+\phi)(\mu_1+\phi+\gamma)}$$

$$R^* = \frac{(\mu_1+\mu_2+\gamma)(\mu_1+\phi)[N(\mu_1+\beta+\omega)(\mu_1+\alpha)-N\alpha\beta(\mu_1+\theta)]-\lambda akw(\mu_1+\alpha)(\mu_1+\phi+\gamma)}{ak\omega\rho(\mu_1+\alpha)(\mu_1+\phi+\gamma)}$$

Local Stability Analysis of Disease free equilibrium

$$Let\ F = \beta S - (\mu_1 + \alpha)V \quad \text{(A)}$$

$$\frac{\partial F}{\partial V} = -(\mu_1 + \alpha)\,\frac{\partial F}{\partial S} = \beta$$

$$\frac{\partial F}{\partial E} = \frac{\partial F}{\partial I} = \frac{\partial F}{\partial T} = \frac{\partial F}{\partial R} = 0$$

$$G = \lambda + \alpha V + \rho R - (\mu_1 + \beta + \omega)S \quad \text{(B)}$$

$$\frac{\partial G}{\partial V} = \alpha\,\frac{\partial G}{\partial S} = -(\mu_1 + \beta + \omega)\,\frac{\partial G}{\partial R} = \rho$$

$$\frac{\partial G}{\partial E} = \frac{\partial G}{\partial I} = \frac{\partial G}{\partial T} = 0$$

$$H = \omega s - (\mu_1 + \theta)E \quad \text{(C)}$$

$$\frac{\partial H}{\partial V} = \frac{\partial H}{\partial I} = \frac{\partial H}{\partial T} = \frac{\partial H}{\partial R} = 0$$

$$\frac{\partial H}{\partial S} = \omega\ \frac{\partial H}{\partial E} = -(\mu_1 + \theta)$$

$$J = \theta E - (\mu_1 + \mu_2 + \gamma)I = 0 \quad \text{(D)}$$

$$\frac{\partial J}{\partial V} = \frac{\partial J}{\partial S} = \frac{\partial J}{\partial R} = \frac{\partial J}{\partial T} = 0$$

$$\frac{\partial J}{\partial E} = \theta\,\frac{\partial J}{\partial I} = -(\mu_1 + \mu_2 + \gamma)$$

$$L = \gamma I - (\mu_1 + \phi)T \quad \text{(E)}$$

$$\frac{\partial L}{\partial V} = \frac{\partial L}{\partial S} = \frac{\partial L}{\partial E} = \frac{\partial L}{\partial R}$$

$$\frac{\partial L}{\partial I}\gamma \ \frac{\partial L}{\partial T} = (\mu_1 + \phi)$$

$$N = \phi T - (\mu_1 + \rho)R \quad \text{(F)}$$

$$\frac{\partial N}{\partial V} = \frac{\partial N}{\partial S} = \frac{\partial N}{\partial E} = \frac{\partial N}{\partial I} = 0$$

$$\frac{\partial N}{\partial T} = \phi \ \frac{\partial N}{\partial R} = (\mu_1 + \rho)$$

The Jacobian matrix is given below

$$\begin{bmatrix} -(\mu_1+\alpha) & \beta & 0 & 0 & 0 & 0 \\ \alpha & -(\mu_1+\beta+\omega) & 0 & 0 & 0 & \rho \\ 0 & \omega & -(\mu_1+\theta) & 0 & 0 & 0 \\ 0 & 0 & \theta & -(\mu_1+\mu_2+\gamma) & 0 & 0 \\ 0 & 0 & 0 & \gamma & -(\mu_1+\phi) & 0 \\ 0 & 0 & 0 & 0 & \phi & -(\mu_1+\rho) \end{bmatrix}$$

$$|J(\varepsilon f) - \lambda I| =$$

$$\begin{vmatrix} -(\mu_1+\alpha) & \beta & 0 & 0 & 0 & 0 \\ \alpha & -(\mu_1+\beta+\omega) & 0 & 0 & 0 & \rho \\ 0 & \omega & -(\mu_1+\theta) & 0 & 0 & 0 \\ 0 & 0 & \theta & -(\mu_1+\mu_2+\gamma) & 0 & 0 \\ 0 & 0 & 0 & \gamma & -(\mu_1+\phi) & 0 \\ 0 & 0 & 0 & 0 & \phi & -(\mu_1+\rho) \end{vmatrix}$$

The det gives.

$$\{-(\mu_1+\alpha)-\lambda\}\{-(\mu_1+\beta+\omega)-\lambda\}\{-(\mu_1+\theta)-\lambda\}\{-(\mu_1+\mu_2+\gamma)-\lambda\}\{-(\mu_1+\phi)-\lambda\}\{-(\mu_1+\rho)-\lambda\} = 0$$

$$-(\mu_1+\alpha)-\lambda = 0 \ or \ -(\mu_1+\beta+\omega)-\lambda = 0 \ or \ -(\mu_1+\theta)-\lambda = 0 \ or \ -(\mu_1+\mu_2+\gamma)-\lambda = 0 \ or \ -(\mu_1+\phi)-\lambda = 0 \ or \ -(\mu_1+\rho)-\lambda = 0$$

$$\lambda_1 = -(\mu_1+\alpha), \lambda_2 = -(\mu_1+\beta+\omega), \lambda_3 = -(\mu_1+\theta), \lambda_4 = (\mu_1+\mu_2+\gamma), \lambda_5 = -(\mu_1+\phi), \lambda_6 = -(\mu_1+\rho)$$

Therefore $\lambda_1, \ \lambda_2, \lambda_3, \ \lambda_4, \ \lambda_5, \ \lambda_6$ are less than zero which implies stability of disease free equilibrium

Stability Analysis of Non – Zero Equilibrium State.

An important criterion by Routh – Hurwitz gives the necessary and sufficient conditions for all roots of the characteristics equation (with real coefficients) to lie in the left half of the complex

plan. In other words, all the roots of the polynomial are negative or have real roots if and only if the determinant of all Hurwitz matrices is positive.

Theorem 1 (Routh – Hurwitz conditions)

Let J = $\begin{bmatrix} f_x(x_*, y_*) & f_y(x_*, y_*) \\ g_x(x_*, y_* & g_y(x_*, y_x) \end{bmatrix}$ be the Jacobian matrix of the non – linear system

$\frac{dx}{dt} = f(x, y)$

$\frac{dx}{dt} = g(x, y)$

Evaluate at the critical point(x_*, y_*), then the critical point (x_*, y_*);

1. Is locally asymptotically stable if trace (J) < 0 and determinant > 0
2. Is stable but not asymptotically stable if trace (J) = 0 and determinant (J) = 0
3. Is unstable if either, trace (J) > 0 or determinant

Jacobian matrix of the system of equations at endemic equilibrium state is

Let $T = \begin{pmatrix} -(\mu_1 + \theta) & 0 \\ \theta & (\mu_1 + \mu_2 + \gamma) \end{pmatrix}$

$Det(T) = (\mu_1 + \theta)(\mu_1 + \mu_2 + \gamma)$

$Trace\ (T) = -(\mu_1 + \theta) - (\mu_1 + \mu_2 + \gamma)$

$= -[(\mu_1 + \theta) + (\mu_1 + \mu_2 + \gamma)]$

It can be observed that $Det(T)$ is greater than zero and $Trace\ (T)$ is less than zero which implies asymptotic stability.

4.0 **CONCLUSION**

Base on the findings of this work, the disease free equilibrium and endemic equilibrium are stable. In terms of the basic reproduction number $R_0 = \frac{\delta\rho(\mu_1+\theta)}{N} S_O$, our result shows that when $R_0 < 1$, the disease free equilibrium is locally stable and when $R_0 > 1$,the endemic equilibrium is stable. In this work we considered stability analyses, in subsequent research we shall carry out the numerical simulation to show the effects of vaccine and treatment

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