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LYAPUNOV STABILITY OF MALE CIRCUMCISION MODEL IN HIV/AIDS PREVENTIONS

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ABSTRACT

This work examines the contribution of a non-pharmaceutical control measure, male circumcision to combat the spread of the world's threatening infection, the HIV/AIDS. It establishes the condition for positivity and boundedness of the model, which enhance the existence and uniqueness of the solution of the model thereby making the model to be epidemiologically meaningful.The main mathematical technique used is the Lyapunov direct method which is applied successfully to two cases: when the population is not circumcised and when the population is fully circumcised, to study the global asymptotic stability of the model. It was established that the local stability of the model is guaranteed if the product of the probability of transmission by individuals and the average number of contact per unit time is less than the sum product of circumcision rate and that of the natural death of the individual in the population. That is if circumcision is encouraged in the population it greatly enhances the eradication of HIV/AIDS. When the population is circumcised the analysis showed that, the disease free equilibrium is globally asymptotically stable in Ω ***if*** $R_{c0} \leq 1$

Introduction

HIV/AIDS also called human immune deficiency virus (HIV)/acquired immune deficiency syndrome is a chronic immune system disease caused by HIV, spread mostly by sexual contact. HIV damages the immune system and interferes with the ability of the body to fight infection and diseases. Since there no known cure for this condition, the beast strategy to enhance the control and prevention of HIV/AIDS, is to reduce the transmissi0n coefficient. Since 1980s over thirty (30) observational studies suggest a protective effect of male circumcision on HIV acquisition in heterosexual men [1]. Male circumcision could be a traditional ritual, a religious rituals or a medical procedure.

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It consists of the cutting of all or parts of the foreskin of the male reproductive organ either traditionally, religiously or medically [2]. Since there is no established medical cure for a complex epidemic like HIV/AIDS, scientists have consistently sought to develop strategies to eradicate it from the population [2, 3, 4]. It has been discovered that HIV/AIDS may be eradicated provided that the net transmission rate of the infected individual is sufficiently reduced [2, 5].

The main reason of mathematical modelling in infection transmission is to project population level outcome from individual level inputs [1, 2, 4, 5, 6, 7]. There are many possible outcomes that can be examined with a model for example the incidence of infection, the prevalence of infection or the doubling time of the epidemic. The most basic outcome, however is the likelihood of the epidemic occurring. That is, whether there is sufficient transmission potential for a chain of epidemic to be sustained. In classic epidemic theory, this outcome is captured by a simple summary statistics; the reproduction number of infectious processR_0. In a susceptible population the R_0. Represent the expected secondary infections generated by the first infected individuals. If $R_0 > 1$ an epidemic is expected to grow, if $R_0 < 1$ the infection is expected to die out [1, 2, 3, 4, 5, 8]

Researchers have assert that the initial rate of epidemic growth depends largely on the transmission coefficient. The epidemic peaks depend on the initial fraction at risk and the stability depends on changes to recruitment to the risk population. So researchers are battling on the effort to reduce at risk population. It is assumed in this work that if a good proportion of the population is circumcised, the at risk population is drastically minimized thereby reducing the transmission coefficient [2, 4, 9]

Mathematical modelling hns made mathematics not only a source of knowledge but as a veritable tool in gaining insight into the dynamics of many real life system including the dynamics of infectious diseases [6, 7, 9, 10, 11, 12. 13. 14.]

In [6] a prey-predator fishery model in a three patch aquatic habitat with selective harvesting of predator and prey populations is considered. Attempt was made to study the qualitative behaviour of stability and co-existence steady state solution in an interaction between prey and predator populations due to variation of the harvesting effort when other model parameters are fixed using the method of numerical simulation. The innovation of this simulation technique has been used to determine the fraction of harvest and un-harvest resource biomass for prey and predator populations. Explicit expressions and values of the maximum sustainable yield (MSY) and the corresponding populations level were obtained. Some sort of control was suggested to avoid over exploitation of resource biomass. Graphical solutions of the model were provided.

The researchers in [10] presented the mathematical model of the impact of vaccination on the transmission dynamics of fowl pox in poultry. The model resulted in a system first order differential equation. Analyzing the system using methods from dynamical system theory together with Routh Harwitz theorem, it was established that the disease free equilibrium is locally stable if the effective reproductive ratio in the presence of vaccination is less than one and unstable if it is greater than one. Using the condition for control, the critical proportion that needs to be vaccinated to achieve immunity for fowl pox was established. From the research, it was discovered that fowl pox can be eradicated from the poultry through vaccination provided the critical proportion is achieved.

In [3] the Mathematical model of the effect of complacency in HIV/AIDS preventions is presented. The model was formulated under six (6) assumptions which resulted in a system of first order differential

equations. Using methods from dynamical systems theory for analysis, it was shown that the disease free state is stable, the condition for this to be possible is: 1 < (μ+ λ), that is, sum of the rate of progression to AIDS and rate of natural death is greater than 1(one). Also the endemic equilibrium state is asymptotically stable. At this point, the disease will not invade the community; otherwise the disease will invade the community. This means that there should be a bound on the rate of progression to AIDS; this is possible if the tempo of campaign against HIV/AIDS is not relaxed.

In [11] the authors formulated the mathematical model for the epidemiology of fowl pox infection transmission that incorporates discrete delay. The model results in a discrete delay system of ordinary differential equations with delay parameter $\tau \geq 0$. Analyzing the system using theorems from differential and integral calculus, we discovered that the number of infectives after a very long time from the day of inception of the epidemic is constant. $1\ 2\delta - \alpha N + \mu\ 1\ 4\delta - 1\ 2\alpha$. The disease free equilibrium and the endemic equilibrium of the system were both established. Using the computable criteria for stability of discrete delay system, unlike ordinary differential equations models, we obtained exponential polynomial equations. Analyzing the system, it was established that both the disease free equilibrium endemic equilibrium points of the system are stable in the absence of delay and unstable with increasing delay if $\beta < (\mu - \lambda)\ \alpha - \delta$, that is, the rate at which the birds are recruited into the system must be greater than the rate at which birds die and the rate at which infection transmission is taking place.

In [8], the researchers presented the mathematical model of the transmission dynamics of fowl pox infection in poultry. Approach: It described the interaction between the susceptible and the infected birds which results in a system of ordinary differential equation. Introducing the control which represents the effort in applying chemoprophylaxis control u_1 and treatment control u_2 in birds with fowl pox, the system became a system of ordinary differential equations with control. The optimal control problem involved that in which the number of birds with latent and active fowl pox infections and the cost of treatment controls $u_1(t)$ and $u_2(t)$ were minimized subject to the model differential This involves the number of birds with active and latent fowl pox respectively as well as the cost of applying chemoprophylaxis control u_1 and treatment u_2 in birds with fowl pox. Analysing the model using Pontryagin's Maximum Principle and optimality conditions, optimal effort necessary to reduce the transmission rate of fowl pox in the poultry was determined. Hence, it is possible to reduce the rate of transmission.

The researchers in [10] developed the mathematical model of bacteria-nutrient harvesting in a cultured environment. This model which assumes that the rate of harvesting of these bacteria is constant results in a system of first order differential equations. Analyzing the model, it was discovered that the product of the maximum nutrient uptake per cell and the number of cells produced per unit of nutrient uptake is constant ($\boldsymbol{VY = ln2 + h}$). It was also assumed that the rate of harvesting of these bacteria varies and a corresponding model was developed. Analyzing this model using methods from dynamical systems theory, it was seen that the system has two steady states. The first steady state is unstable while the second is globally asymptotically stable if the carrying capacity of the environment has a lower bound, which is a ratio of the harvesting coefficient of the bacteria, cost per unit effort per unit price of the bacteria.

The mathematical model for the transmission dynamics of swine flu among swine and humans with the vaccination of newborns is presented in [15]. The model assumes a vaccine with a life-long immunity. The analysis of the Disease-free Equilibrium (DFE) shows that it will be stable if there is a bound on the rate of transmission from swine to swine (βs) and the rate of transmission from human to human (βH). Endemic Equilibrium (EE) for the model shows that the disease will persist if there is a lower bound on

the rate of transmission from swine to swine (βS) and on the rate of newborn babies vaccinated (VH). The behavior of the influenza (flu) is illustrated by simulation with different parameter values.

Two models that examines the transmission dynamics of fowl pox among birds based on mode of transmission of the disease in poultry was formulated in [9]. Using methods from dynamical system theory, equilibrium analysis of the first model showed that the diseases free equilibrium is stable if $\alpha N < (d_1 + \mu + \gamma), \beta < \gamma$ The endemic equilibrium is asymptotically stable if $\beta - \gamma < \frac{\alpha(d_1+\mu+\gamma)}{k}$. That is fowl pox will not invade the poultry if the rate at which the susceptible birds β are introduced into the poultry is greater than the rate at which the susceptible birds are exposed to infection γ .It was also established that $R_0 < 1$ if $S_0 > S_c$ where $S_c = \frac{(d_1+\mu+\gamma)}{\alpha} R_0 = \frac{\gamma S_0(d_1+\mu+\gamma)}{\alpha}$. The second model is stable if the rate at which the infected birds recover and the rate at which the mosquito die are high. Also if the growth rate of mosquito is less than the death rate of mosquito

In [14], the mathematical model of the impact of vaccination on the transmission dynamics of fowl pox in poultry is presented. The model resulted in a system of ordinary differential equation. Analysing the system using methods from dynamical system theory together with Routh-Harwitz theorem, it was established that the disease free equilibrium is locally stable if the effective reproduction ratio $R_\rho = \frac{(1-\rho)\alpha\beta}{(d_1+r_1+\mu)}$ in the presence of vaccination is less than one (1). Using the condition for control, the crtical proportion that needs to be vaccinated to achieve herds immunity for fowl pox is established as $\rho_c = \frac{\alpha\beta-(d_1+\mu+\gamma)}{\alpha\beta}$ it was discovered that the fowl pox can be eradicated through vaccination provided the critical proportion ρ_c is achieved

Mathematical model of male circumcision in HIV/AIDS preventions is formulated in [5]. The male circumcision considered in the research were taken to be traditional ritual, religious rituals or medical procedure circumcision. Several assumptions were taken into consideration in formulating the model. In the analysis of the model, the steady states were established, and shown that these steady states are stable if $(\boldsymbol{\sigma}+\boldsymbol{\mu}) > -\boldsymbol{B}$ and $\boldsymbol{\sigma} > -(\boldsymbol{\mu} + \boldsymbol{\nu c})$. Also from the analysis of the model, it was observed that if the sum of the rate of death due to natural incidence ($\boldsymbol{\mu}$) and that due to infection is reduced ($\boldsymbol{\nu c}$) while the rate of circumcision is increased, this will bring the reproduction number of infectious process to less than one and the epidemic will die out of the population

Here in this article, we further analyse our previous model in [5], establishing the boundedness and positivity of the model. Construct the next generation matrix to determine the basic reproductive ratio both at when the population is not circumcised and at when the population is fully circumcised. The previous work only focused on the development of the model and establish local stability using dynamical system theory. But her we shall establish the local and global stability of the model using Lyapunov's direct method (also known as Lyapunov second method) which provides a way of analysing the stability of nonlinear systems without actually solving the differential equations.

2.0 **Model formulation**

The model as presented in [5]

2.1 **Assumptions and Parameters**

2.2 **Model Assumptions**

1. We assume that there is a proportionate recruitment rate of individuals into the heterosexual population.
2. There is proportionate rate of circumcision of both the susceptible and infected individuals.

2.3 **Model PARAMETERS**

$S_c(t)$= Number of susceptible individuals that are circumcised at time $t, t > 0$
$S_{nc}(t)$= Number of susceptible individuals that are not circumcised at time $t, t > 0$
$S(t) = S_c(t) + S_{nc}(t)$= Susceptible population at time $t, t > 0$
$I_c(t)$= Number of infected individuals that are circumcised at time $t, t > 0$
$I_{nc}(t)$= Number of infected individuals that are not circumcised at time $t, t > 0$
$I(t) = I_c(t) + I_{nc}(t)$= infected population at time $t, t > 0$
$N = S(t) + I(t) = S_c(t) + S_{nc}(t) + I_c(t) + I_{nc}(t)$= total population under the
b= Recruitment rate into the population
μ= Natural death rate of the population
V_c =Death rate of circumcised infected individuals
V_{nc} =Death rate of uncircumcised infected individuals

$$\sigma = Therateatwhichsusceptibleindidualsarebeingcircumcised$$
$$\rho = Therateatwhichinfectedindividualsarebeingcircumcised.$$
$$\beta = Theprobabilityoftransmissionbyindividuals \in classI_{nc}$$
$$\alpha = Theprobabilityoftransmissionbyindividuals \in classI_c$$
$$c = Averagenumberofcontact \vee partnersperunittime$$

$c\beta \wedge c\alpha arenettransmissionofindividuals \in classI_{nc} \wedge I_c$respectively

2.4 The Model Equations

The combination of the above assumptions and parameters result in the following model equation for male circumcision in HIV/AIDS preventions.

$$\frac{dS_c(t)}{dt} = \sigma S_{nc}(t) - B(t)S_c(t) - \mu S_c(t)$$
$$\frac{dS_{nc}(t)}{dt} = bN - B(t)S_{nc}(t) - \sigma S_{nc}(t) - \mu S_{nc}(t)$$
$$\frac{dI_c(t)}{dt} = B(t)S_c(t) - (\mu + v_c)I_c + \sigma I_{nc}(t)$$
$$\frac{dI_{nc}(t)}{dt} = B(t)S_{nc}(t) - (\mu + v_{nc})I_{nc} - \sigma I_{nc}(t)$$

Where

$$B(t) = \frac{c\beta I_{nc}(t) + c\alpha I_c(t)}{N} = incidencerateofinfection$$

3.0 ANALYSIS OF THE MODEL

3.1 *Positivity and Boundedness*

For the model (2.1) to be epidemiologically meaningful, it is important to show that all its state variables are non-negative for all time $t > 0$ and that Ω is, indeed, bounded. Following methods in [2, 3,4,5,6, 22], we claim the following:

Given the initial condition of the system

$$S_c(0) \geq 0, S_{nc}(0) \geq 0, I_c(0) \geq 0, I_{nc}(0) \geq 0 \ldots\ldots\ldots\ldots\ldots\ldots\ldots (3.1.1)$$

We can define a feasible region such that

$$\Omega = \{(S_c, S_{nc} I_c, I_{nc},) \in R_+^{\ 4} : 0 < S_c, + I_c < \frac{N(\sigma+\mu-b)}{\sigma+\mu}, 0 < S_{nc}, + I_{nc} < \frac{bN}{\sigma+\mu}$$

Then we have the following theorem

Theorem 3.1.1: Let the initial data for the model (2.1) be $S_c(0) \geq 0, S_{nc}(0) \geq 0, I_c(0) \geq 0, I_{nc}(0) \geq 0$Then the solutions$(S_c, S_{nc} I_c, I_{nc})$of the model (2.1) are positive for all time t > 0 .

Proof:

Since the right hand side of (2.1) is Lipschitz continuous, then there is existence of unique solutions

$Fori = 1 f_1(0, S_{nc} I_c, I_{nc},) = \sigma S_{nc}(t)$considering that $S_{nc} \geq 0, I_c \geq 0, I_{nc} \geq 0,\ f_1(0, S_{nc} I_c, I_{nc},) \geq 0$ and thus $S_c \geq 0 forall t$ which it exist.

$Fori = 2 f_2(S_c, 0, I_c, I_{nc},) = bN$considering that $S_c \geq 0, I_c \geq 0, I_{nc} \geq 0,\ f_2(S_c, 0 I_c, I_{nc},) \geq 0$ and thus $S_{nc} \geq 0 forall t$ which it exist.

$Fori = 3 f_3(S_c, S_{nc}, 0, I_{nc},) = B(t) S_c(t)$considering that $S_c \geq 0, S_{nc} \geq 0, I_{nc} \geq 0,\ f_3(S_c, S_{nc}, 0 I_{nc},) \geq 0$ and thus $I_c \geq 0 forall t$ which it exist.

$Fori = 4 f_4(S_c, S_{nc}, I_c, 0) = B(t) S_{nc}(t)$considering that $S_c \geq 0, S_{nc} \geq 0, I_c \geq 0,\ f_4(S_c, S_{nc}, I_c, 0) \geq 0$ and thus $I_{nc} \geq 0 forall t$ which it exist.

Therefore given the initial conditions in (3.1.1), the solutions S_c, S_{nc}, I_c, I_{nc} are positive for all t which they exist.

Likewise:

Let $t_1 = ^{\{t>0 : S_c(0) \geq 0, S_{nc}(0) \geq 0, I_c(0) \geq 0, I_{nc}(0) \geq 0 \in [0.t]\}}$. Thus, $t_1 > 0$.

We have, from the first equation of the system (2.1) that

$$\frac{dS_c(t)}{dt} + (B(t) + \mu) S_c(t) = \sigma S_{nc}(t), where B(t) = \frac{c\beta I_{nc}(t) + c\alpha I_c(t)}{N}$$

Which can be written as: $\frac{d\left(S_c(t) e^{\int_0^{t_1} (B(t)+\mu) S_c}\right)}{dt} = \sigma S_{nc}(t)$

So that $S_c(t_1) = e^{-\int_0^{t_1} (B(s)+\mu) ds} \left[\int_0^{t_1} \sigma S_{nc}(s) ds\right] > 0$

Similarly, it can be shown that $S_{nc} > 0, I_c > 0, I_{nc} > 0$

Theorem 3.1.2

The feasible region

$$\Omega = \{(S_c, S_{nc} I_c, I_{nc}) \in R_+^{\ 4} : 0 < S_c, + I_c < \frac{N(\sigma+\mu-b)}{\sigma+\mu}, 0 < S_{nc}, + I_{nc} < \frac{bN}{\sigma+\mu}$$

with the initial conditions in (3.1.1) is positively invariant and attracting.

Proof:

Recall that $N = S_c + S_{nc} + I_c + I_{nc}$,, therefore adding all the equations of (2.1) we have $\frac{dN}{dt} = \sigma S_{nc}(t) - B(t) S_c(t) - \mu S_c(t) + bN - B(t) S_{nc}(t) - \sigma S_{nc}(t) - \mu S_{nc}(t) + B(t) S_c(t) - (\mu + v_c) I_c + \sigma I_{nc}(t) + B(t) S_{nc}(t) - (\mu + v_{nc}) I_{nc} - \sigma I_{nc}(t) \ldots\ldots\ldots$

$$\frac{dN}{dt} = -\mu N + bN - \delta N, \delta = min(v_c, v_{nc}) \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (3.1.2)$$

Which can be written as

$$\frac{dN}{dt} = -\omega N$$

Where $\omega = (\mu + \delta - b)$

Thus $\int \frac{dN}{N} \leq -\int \omega dt$
So that $N(t) \leq N(0)e^{-\omega t}$
$N(t)$approaches $N(0)$ as $t \to \infty$.
Therefore the region Ω is positively invariant. Then either the solution enter Ω in finite time, or $N(t)$ approaches $N(0)$ asymptotically. Hence, the regionΩ attract all solutions in R_+ [4]

3.2 Next Generation Matrix and Basic Reproductive Ratio(Assuming $S_c^0 = 0$)

Assuming that the $S_c^0 = 0$ that is, in the absence of infection, there is no circumcision, we shall apply methods in [1, 3, 4, 9. 10. 11. 12, 15, 21] to construct the next generation matrix

$$S_{nc}^0 = \frac{bN}{\sigma+\mu} S_c^0 = 0, F = \begin{pmatrix} 0 & 0 \\ \frac{c\alpha b}{\sigma+\mu} & \frac{c\beta b}{\sigma+\mu} \end{pmatrix}$$

$$V = \begin{pmatrix} (\mu + v_c) & -\sigma \\ (\mu + v_{nc} + \sigma) & 0 \end{pmatrix}$$

$$V^{-1} = \begin{pmatrix} 0 & \frac{1}{(\mu + v_{nc} + \sigma)} \\ \frac{1}{-\sigma} & \frac{(\mu + v_c)}{\sigma(\mu + v_{nc} + \sigma)} \end{pmatrix}$$

$$V^{-1}F = \begin{pmatrix} \frac{c\alpha b}{(\mu + v_{nc} + \sigma)(\sigma + \mu)} & \frac{c\beta b}{(\mu + v_{nc} + \sigma)(\sigma + \mu)} \\ \frac{c\alpha b(\mu + v_c)}{\sigma(\mu + v_{nc} + \sigma)(\sigma + \mu)} & \frac{c\beta b(\mu + v_c)}{(\mu + v_{nc} + \sigma)(\sigma + \mu)\sigma} \end{pmatrix}$$

$$FV^{-1} = \begin{pmatrix} 0 & 0 \\ \frac{-c\beta b}{(\sigma + \mu)\sigma} & \frac{c\alpha b\sigma + c\beta b(\mu + v_c)}{\sigma(\sigma + \mu)(\mu + v_{nc} + \sigma)} \end{pmatrix}$$

$$R_0 = \rho(FV^{-1}) = \frac{c\alpha b}{(\sigma + \mu)(\mu + v_{nc} + \sigma)} + \frac{c\beta b(\mu + v_c)}{\sigma(\sigma + \mu)(\mu + v_{nc} + \sigma)} \dots\dots\dots (3.2.1)$$

3.3 Local Stability using Basic Reproductive Ratio

We shall use the basic reproductive ratio R_0 in (3.2.1) to establish the local stability of the system (2.1). The following lemmas in [1] shall be instrumental in establishing that the system (2.1) is stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Lemma 3.3.1: If a matrix K has the Z-sign pattern, then $K \geq 0$ if and only if K is a non-singular $M -$

Lemma 3.3.2: If F is nonnegative and V is a non-singular M-matrix, then
$R_0 = \rho(FV^{-1}) < 1$if and only if all eigenvalues of $K = F - V$have negative real parts.

Theorem 3.3.1: Consider the disease transmission model given by (2.1). The disease-free equilibrium of (2.1) is locally asymptotically stable if $R_0 < 1$, but unstable if $R_0 > 1$, where R_0 is as defined in (3.2.1)

Proof

Let $F = \begin{pmatrix} 0 & 0 \\ \frac{c\alpha b}{\sigma+\mu} & \frac{c\beta b}{\sigma+\mu} \end{pmatrix}$

$V = \begin{pmatrix} (\mu + v_c) & -\sigma \\ (\mu + v_{nc} + \sigma) & 0 \end{pmatrix}$, $K = F - V = \begin{pmatrix} 0 & 0 \\ \frac{c\alpha b}{\sigma+\mu} & \frac{c\beta b}{\sigma+\mu} \end{pmatrix} - \begin{pmatrix} (\mu + v_c) & -\sigma \\ (\mu + v_{nc} + \sigma) & 0 \end{pmatrix} =$

$\begin{pmatrix} -(\mu + v_c) & -\sigma \\ \frac{c\alpha b}{\sigma+\mu} - (\mu + v_{nc} + \sigma) & \frac{-c\beta b}{\sigma+\mu} \end{pmatrix}$

$K = \left[\begin{matrix} -(\mu + v_c) & -\sigma \\ \frac{c\alpha b}{\sigma+\mu} - (\mu + v_{nc} + \sigma) & \frac{-c\beta b}{\sigma+\mu} \end{matrix} \right] \frac{1}{2}\left(a + d - \sqrt{a^2 + 4bc - 2ad + d^2}\right), \frac{1}{2}\left(a + d + \sqrt{a^2 + 4bc - 2ad + d^2}\right)\}$

$$\frac{1}{2}\left(-(\mu + v_c) - \frac{c\beta b}{\sigma+\mu} + \sqrt{(\mu + v_c)^2 - 4\sigma\left(\frac{c\alpha b}{\sigma+\mu} - (\mu + v_{nc} + \sigma)\right) - 2\frac{(\mu + v_c)c\beta b}{\sigma+\mu} + \left(\frac{c\beta b}{\sigma+\mu}\right)^2} \right)$$

Where $-(\mu + v_c) - \frac{c\beta b}{\sigma+\mu}$ is the real part of the eigenvalue, which is clearly negative, that is, $-(\mu + v_c) - \frac{c\beta b}{\sigma+\mu} < 0$

3.4 LYAPONOV DIRECT METHOD OF STABILITY

Lyapunov's direct method (also known as Lyapunov second method) provides a way of analysing the stability of nonlinear systems without actually solving the differential equations. The idea behind Lyapunov's direct method is that the system is stable if there exists some Lyapunov function in the neighbourhood of the equilibrium point. Thus it can be shown that Lyapunov's direct method is a sufficient condition for the stability of nonlinear system.

We shall employ a matrix-theoretic method to construct a Lyapunov function in order to study the global stability of the disease-free equilibrium of the model equation (2.1) following methods in [2, 8,17,18,19]

The set

$$f(I_c, I_{nc}, S_c, S_{nc},) = (F - V)x - P(x, y) + Q(x, y) \dots\dots\dots\dots\dots\dots\dots\dots\dots\dots\dots (3.4.1)$$

Where $x = (I_c, I_{nc})^T, y = (S_c, S_{nc})^T$,

$P(I_c, I_{nc}, S_c, S_{nc}) = \begin{pmatrix} B(t)S_c \\ B(t)S_{nc} \end{pmatrix}$,

$Q(I_c, I_{nc}, S_c, S_{nc}) = \begin{pmatrix} (\mu + v_c)I_c - \sigma I_{nc}(t) \\ (\mu + v_{nc} + \sigma)I_{nc} \end{pmatrix}$.

$$x = (F - V)x - f(x, y)$$

Assuming that the $S_c^0 = 0$ that is, in the absence of infection, there is no circumcision, we shall apply methods in [1, 3, 4, 9. 10. 11. 12, 15, 21]to construct the next generation matrix

$$S_{nc}^0 = \frac{bN}{\sigma + \mu}$$

$$S_c^0 = 0$$

$$F = \begin{pmatrix} 0 & 0 \\ \frac{c\alpha b}{\sigma + \mu} & \frac{c\beta b}{\sigma + \mu} \end{pmatrix}$$

$$V = \begin{pmatrix} (\mu + v_c) & -\sigma \\ (\mu + v_{nc} + \sigma) & 0 \end{pmatrix}$$

$$V^{-1} = \begin{pmatrix} 0 & \frac{1}{(\mu + v_{nc} + \sigma)} \\ \frac{1}{-\sigma} & \frac{(\mu + v_c)}{\sigma(\mu + v_{nc} + \sigma)} \end{pmatrix}$$

$$V^{-1}F = \begin{pmatrix} \dfrac{c\alpha b}{(\mu+v_{nc}+\sigma)(\sigma+\mu)} & \dfrac{c\beta b}{(\mu+v_{nc}+\sigma)(\sigma+\mu)} \\ \dfrac{c\alpha b(\mu+v_c)}{\sigma(\mu+v_{nc}+\sigma)(\sigma+\mu)} & \dfrac{c\beta b(\mu+v_c)}{(\mu+v_{nc}+\sigma)(\sigma+\mu)\sigma} \end{pmatrix}$$

$$FV^{-1} = \begin{pmatrix} 0 & 0 \\ \dfrac{-c\beta b}{(\sigma+\mu)\sigma} & \dfrac{c\alpha b\sigma + c\beta b(\mu+v_c)}{\sigma(\sigma+\mu)(\mu+v_{nc}+\sigma)} \end{pmatrix}$$

$$R_{nc0} = \rho(FV^{-1}) = \frac{c\alpha b}{(\sigma+\mu)(\mu+v_{nc}+\sigma)} + \frac{c\beta b(\mu+v_c)}{\sigma(\sigma+\mu)(\mu+v_{nc}+\sigma)}$$

The left eigenvector of the nonnegative matrix, $V^{-1}F$, isobtained thus:

$$\left\{ \frac{\sigma}{\mu+v_c} - \frac{\beta\sigma}{\alpha} - \frac{\dfrac{cb\sqrt{\alpha^2\sigma^2+\beta(\mu+v_c)(\alpha+\beta)}}{\sigma(\mu+v_{nc}+\sigma)(\sigma+\mu)}}{2\dfrac{c\alpha b(\mu+v_c)}{\sigma(\mu+v_{nc}+\sigma)(\sigma+\mu)}}, 1 \right\} = \left\{ \frac{\sigma}{\mu+v_c} - \frac{\beta\sigma}{\alpha} - \frac{b\sqrt{\alpha^2\sigma^2+\beta(\mu+v_c)(\alpha+\beta)}}{\alpha(\mu+v_c)}, 1 \right\}$$

$$= \left\{ \frac{\sigma\big(\alpha-\beta(\mu+v_c)\big) - b\sqrt{\alpha^2\sigma^2+\beta(\mu+v_c)(\alpha+\beta)}}{\alpha(\mu+v_c)}, 1 \right\}$$

The left eigenvector of the nonnegative matrix, $V^{-1}F$, is$\omega^T = \left\{ \frac{\sigma(\alpha-\beta(\mu+v_c)) - b\sqrt{\alpha^2\sigma^2+\beta(\mu+v_c)(\alpha+\beta)}}{\alpha(\mu+v_c)}, 1 \right\}$

Given that $B(t) = \frac{c\beta I_{nc}(t) + c\alpha I_c(t)}{N}$ and $f(I_c, I_{nc}, S_c, S_{nc}) = \begin{bmatrix} B(t)(S_{c0} - S_c) \\ B(t)(S_{nc0} - S_{nc}) \end{bmatrix}$

Notice that, $f(I_c, I_{nc}, S_c, S_{nc}) \geq 0 \in \Omega = \{(S_c, S_{nc} I_c, I_{nc}) \in R_{+}^{4} : 0 < S_c, +I_c < \frac{N(\sigma+\mu-b)}{\sigma+\mu}, 0 < S_{nc}, +I_{nc} < \frac{bN}{\sigma+\mu}$ if $S_c \leq S_{c0} \wedge S_{nc} \leq S_{nc0}$ and $f(0,0,S_{c0},S_{nc0}) = 0$. Since $\geq 0, V^{-1} \geq 0, f(I_c, I_{nc}, S_c, S_{nc}) \geq 0$. by theorem 2.1 of [2], $\Phi = \omega^T V^{-1} \begin{pmatrix} I_c \\ I_{nc} \end{pmatrix}$ is the Lyapunuv function, where $\omega^T = \left\{ \frac{\sigma(\alpha-\beta(\mu+v_c)) - b\sqrt{\alpha^2\sigma^2+\beta(\mu+v_c)(\alpha+\beta)}}{\alpha(\mu+v_c)}, 1 \right\}$

Is the left eigenvector of the nonnegative matrix, $V^{-1}F$, by straight forward calculation,

$$\omega^T V^{-1} = \left(\frac{1}{-\sigma} \frac{\sigma\big(\alpha-\beta(\mu+v_c)\big) - b\sqrt{\alpha^2\sigma^2+\beta(\mu+v_c)(\alpha+\beta)}}{\alpha(\mu+v_c)(\mu+v_{nc}+\sigma)} + \frac{(\mu+v_c)}{\sigma(\mu+v_{nc}+\sigma)} \right)$$

$$= \left(\frac{1}{-\sigma} \frac{\sigma\big(\alpha-\beta(\mu+v_c)\big) - b\sqrt{\alpha^2\sigma^2+\beta(\mu+v_c)(\alpha+\beta)} + \sigma(\mu+v_c)^2}{\alpha(\mu+v_c)(\mu+v_{nc}+\sigma)} \right)$$

$$\Phi = \frac{I_c}{-\sigma} + \frac{\sigma\big(\alpha-\beta(\mu+v_c)\big) - b\sqrt{\alpha^2\sigma^2+\beta(\mu+v_c)(\alpha+\beta)} + \sigma(\mu+v_c)^2}{\alpha(\mu+v_c)(\mu+v_{nc}+\sigma)} I_{nc}$$

Which the Lyapunov function of the model equation (2.1). The theorem that follows give backing for the Global stability of the diseases free equilibrium of the model.

Theorem 3.4.1

The disease-free equilibrium of the model (2.1) is globally asymptotically stable in $\Omega = \{(S_c, S_{nc} I_c, I_{nc}) \in R_{+}^{4} : 0 < S_c, +I_c < \frac{N(\sigma+\mu-b)}{\sigma+\mu}, 0 < S_{nc}, +I_{nc} < \frac{bN}{\sigma+\mu}$ if$R_{nc0} \leq 1$

Proof:

Let

$$\Psi = \frac{I_c}{-\sigma} + \frac{\sigma\big(\alpha-\beta(\mu+v_c)\big) - b\sqrt{\alpha^2\sigma^2+\beta(\mu+v_c)(\alpha+\beta)} + \sigma(\mu+v_c)^2}{\alpha(\mu+v_c)(\mu+v_{nc}+\sigma)} I_{nc}$$

Be a lyapunov function of the model (2.1) on Ω with $R_0 \leq 1$ and $f(I_c, I_{nc}, S_c, S_{nc}) \geq 0$.

Differentiating Ψ along the solutions of (2.1) gives

$$\Psi^{/} = \omega^T V^{-1} x^{/}$$

Where $(I_c \quad I_{nc})^T$, $x \quad = (F - V)x - f(x, y)$

$$\Psi \quad = \omega^T V^{-1}\{(F - V)x - f(x, y)\}$$
$$\Psi \quad = \omega^T V^{-1}(F - V)x - \omega^T V^{-1} f(x, y)$$
$$\Psi \quad = (R_{nc0} - 1)\omega^T x - \omega^T V^{-1} f(x, y)$$
$$\Psi \quad = (R_0 - 1)\omega^T x - \omega^T V^{-1} f(x, y)$$
$$\Psi \quad = (R_{nc0} - 1)\left\{\frac{\sigma\big(\alpha - \beta(\mu + v_c)\big) - b\sqrt{\alpha^2\sigma^2 + \beta(\mu + v_c)(\alpha + \beta)}}{\alpha(\mu + v_c)} I_c + I_{nc}\right\} - \frac{B(t)(S_c - S_{c0})}{\sigma}$$
$$- \frac{\sigma\big(\alpha - \beta(\mu + v_c)\big) - b\sqrt{\alpha^2\sigma^2 + \beta(\mu + v_c)(\alpha + \beta)} + \sigma(\mu + v_c)^2}{\alpha(\mu + v_c)(\mu + v_{nc} + \sigma)} B(t)(S_{nc0} - S_{nc})$$

Thus it follows that $\Psi \quad \leq 0$ if $R_{nc0} \leq 1$. If $R_{nc0} = 1$ then $\Psi \quad = 0$
if and only if $I_c = I_{nc} = 0$. If $R_{nc0} = 1$ then $\Psi \quad = 0$ if and only if:
caseI: $I_c = I_{nc} = 0$, caseII: $S_c = S_{c0}$ and $S_{nc} = S_{nc0}$
Therefore every solution trajectory of equations in the model (2.1) converges to the largest compact invariant set $M = \{I_c, I_{nc}, S_{c0}, S_{nc0}\}$, and the only point in M is the disease-free equilibrium. Then by LaSalle's invariant principle [20], the disease free equilibrium is globally asymptotically stable inΩ if $R_{nc0} \leq 1$.
That is every solution trajectory of equations in the model (2.1) approaches the disease free equilibrium as $t \to \infty$

3.5 Next Generation Matrix and Basic Reproductive Ratio(Assuming $S_{nc}^0 = 0$)

Assuming that the $S_{nc}^0 = 0$ that is, in the absence of infection, all is fully circumcised, we shall apply methods in [1, 3, 4, 9. 10. 11. 12, 15, 21] to construct the next generation matrix

$$S_{nc}^0 = 0 S_c^0 = \frac{N(\sigma + \mu - b)}{\sigma + \mu}$$

$$F = \begin{pmatrix} \frac{c\alpha(\sigma + \mu - b)}{\sigma + \mu} & \frac{c\beta(\sigma + \mu - b)}{\sigma + \mu} \\ 0 & 0 \end{pmatrix} V = \begin{pmatrix} (\mu + v_c) & -\sigma \\ (\mu + v_{nc} + \sigma) & 0 \end{pmatrix}$$

$$V^{-1} = \begin{pmatrix} 0 & \frac{1}{(\mu + v_{nc} + \sigma)} \\ \frac{1}{-\sigma} & \frac{(\mu + v_c)}{\sigma(\mu + v_{nc} + \sigma)} \end{pmatrix}$$

$$V^{-1}F = \begin{pmatrix} 0 & 0 \\ \frac{c\alpha(b - \sigma - \mu)}{\sigma(\sigma + \mu)} & \frac{c\beta(b - \sigma - \mu)}{\sigma(\sigma + \mu)} \end{pmatrix}$$

$$FV^{-1} = \begin{pmatrix} \frac{c\beta(b - \sigma - \mu)}{\sigma(\sigma + \mu)} & \frac{(\sigma + \mu - b)\big(c\alpha\sigma + c\beta(\mu + v_c)\big)}{\sigma(\mu + v_{nc} + \sigma)(\sigma + \mu)} \\ 0 & 0 \end{pmatrix}$$

$$R_{c0} = \rho(FV^{-1}) = \frac{c\beta(b - \sigma - \mu)}{\sigma(\sigma + \mu)} = \frac{c\beta b}{\sigma(\sigma + \mu)} - \frac{c\beta}{(\sigma + \mu)} - \frac{c\beta\mu}{\sigma(\sigma + \mu)} \ldots\ldots\ldots\ldots (3.2)$$

3.6 Local Stability (Assuming $S_{nc}^0 = 0$)

We shall use the basic reproductive ratio R_0 in (3.1) to establish the local stability of the system (2.1) when the population is fully circumcised. **Lemma 3.1.1 and Lemma 3.1.2 above** shall be instrumental in establishing that the system (2.1) is stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Theorem 3.6.1: Consider the disease transmission model given by (2.1). Given that $S_{nc}^0 = 0$, the disease-free equilibrium of (2.1) is locally asymptotically stable if $R_0 < 1$, but unstable if $R_0 > 1$, where R_0 is as defined in (3.2)

Proof

Let $F = \begin{pmatrix} \frac{c\alpha(\sigma+\mu-b)}{\sigma+\mu} & \frac{c\beta(\sigma+\mu-b)}{\sigma+\mu} \\ 0 & 0 \end{pmatrix} V = \begin{pmatrix} (\mu+v_c) & -\sigma \\ (\mu+v_{nc}+\sigma) & 0 \end{pmatrix}$, $K = F - V =$

$$\begin{pmatrix} \frac{c\alpha(\sigma+\mu-b)-(\sigma+\mu)(\mu+v_c)}{\sigma+\mu} & \frac{c\beta(\sigma+\mu-b)}{\sigma+\mu} - \sigma \\ -(\mu+v_{nc}+\sigma) & 0 \end{pmatrix}$$

$$K = \begin{bmatrix} \frac{c\alpha(\sigma+\mu-b)-(\sigma+\mu)(\mu+v_c)}{\sigma+\mu} & \frac{c\beta(\sigma+\mu-b)}{\sigma+\mu} - \sigma \\ -(\mu+v_{nc}+\sigma) & 0 \end{bmatrix}$$

The of K is given as

$$\frac{1}{2}\left(\frac{c\alpha(\sigma+\mu-b)-(\sigma+\mu)(\mu+v_c)}{\sigma+\mu} \pm \sqrt{\left(\frac{c\alpha(\sigma+\mu-b)-(\sigma+\mu)(\mu+v_c)}{\sigma+\mu} \right)^2 - 4\left(\frac{c\beta(\sigma+\mu-b)}{\sigma+\mu} - \sigma \right)(\mu+v_{nc}+\sigma)} \right)$$

Assuming that

$$\left(\frac{c\alpha(\sigma+\mu-b)-(\sigma+\mu)(\mu+v_c)}{\sigma+\mu} \right)^2 > 4\left(\frac{c\beta(\sigma+\mu-b)}{\sigma+\mu} - \sigma \right)(\mu+v_{nc}+\sigma)$$

For the root to be real and negative, then

$$\frac{c\alpha(\sigma+\mu-b)-(\sigma+\mu)(\mu+v_c)}{\sigma+\mu} < \sqrt{\left(\frac{c\alpha(\sigma+\mu-b)-(\sigma+\mu)(\mu+v_c)}{\sigma+\mu} \right)^2 - 4\left(\frac{c\beta(\sigma+\mu-b)}{\sigma+\mu} - \sigma \right)(\mu+v_{nc}+\sigma)}$$

Square both sides, then

$$\left(\frac{c\alpha(\sigma+\mu-b)-(\sigma+\mu)(\mu+v_c)}{\sigma+\mu} \right)^2 < \left(\frac{c\alpha(\sigma+\mu-b)-(\sigma+\mu)(\mu+v_c)}{\sigma+\mu} \right)^2 - 4\left(\frac{c\beta(\sigma+\mu-b)}{\sigma+\mu} - \sigma \right)(\mu+v_{nc}+\sigma)$$

$$\frac{c\beta(\sigma+\mu-b)}{\sigma+\mu} < \sigma implies c\beta(\sigma+\mu-b) < \sigma(\sigma+\mu)$$

That is the local stability is guaranteed if the product of the probability of transmission by individuals and the average number of contact per unit time is less than the sum product of circumcision rate and that of the natural death of the individual in the population. That is if circumcision is encouraged in the population it greatly enhances the eradication of HIV/AIDS

3.7 Global Stability (Assuming $S_{nc}^0 = 0$)

We shall employ a matrix-theoretic method to construct a Lyapunov function in order to study the global stability of the disease-free equilibrium of the model equation (2.1) assuming $S_{nc}^0 = 0$ following methods from [2, 8,17,18,19]

The left eigenvector of the nonnegative matrix, $V^{-1}F = \begin{pmatrix} 0 & 0 \\ \frac{c\alpha(b-\sigma-\mu)}{\sigma(\sigma+\mu)} & \frac{c\beta(b-\sigma-\mu)}{\sigma(\sigma+\mu)} \end{pmatrix}$

is$\omega^T = \left\{\frac{\beta(\sigma+\mu-b)}{\alpha(b-\mu-\sigma)}, 1\right\}$

recall that, $B(t) = \frac{c\beta I_{nc}(t)+c\alpha I_c(t)}{N}$

$$f(I_c, I_{nc}, S_c, S_{nc}) = \begin{bmatrix} B(t)(S_{c0} - S_c) \\ B(t)(S_{nc0} - S_{nc}) \end{bmatrix}$$

Notice that, $f(I_c, I_{nc}, S_c, S_{nc}) \geq 0 \in \Omega = \{(S_c, S_{nc} I_c, I_{nc}) \in R_{+\ 4}: 0 < S_c, +I_c < \frac{N(\sigma+\mu-b)}{\sigma+\mu}, 0 < S_{nc}, +I_{nc} < \frac{bN}{\sigma+\mu}$ if $S_c \leq S_{c0} \wedge S_{nc} \leq S_{nc0}$ and $f(0,0, S_{c0}, S_{nc0}) = 0$. Since $\geq 0, V^{-1} \geq 0, f(I_c, I_{nc}, S_c, S_{nc}) \geq 0$. by theorem 2.1 of [17], $\Phi = \omega^T V^{-1} \begin{pmatrix} I_c \\ I_{nc} \end{pmatrix}$ is the Lyapunuv function, where

$$\omega^T = \left\{\frac{\beta(\sigma+\mu-b)}{\alpha(b-\mu-\sigma)}, 1\right\}$$

Is the left eigenvector of the nonnegative matrix, $V^{-1}F$, by straight forward calculation,

$$\omega^T V^{-1} = \left(\frac{1}{-\sigma} \frac{\alpha(\mu+v_c)-\sigma\beta}{\alpha\sigma(\mu+v_{nc}+\sigma)}\right)$$

$$\omega^T V^{-1} \begin{pmatrix} I_c \\ I_{nc} \end{pmatrix} = \frac{I_c}{-\sigma} + \frac{\alpha(\mu+v_c)-\sigma\beta}{\alpha\sigma(\mu+v_{nc}+\sigma)} I_{nc}$$

Which is the Lyapunov function of the model equation (2.1) for $S_{nc} = 0$. The theorem that follows give backing to the Global stability the diseases free equilibrium of the model.

Theorem 3.7.1

The disease-free equilibrium of the model (2.1) is globally asymptotically stable in $\Omega = \{(S_c, S_{nc} I_c, I_{nc}) \in R_{+\ 4}: 0 < S_c, +I_c < \frac{N(\sigma+\mu-b)}{\sigma+\mu}, 0 < S_{nc}, +I_{nc} < \frac{bN}{\sigma+\mu}$ if$R_{c0} \leq 1$

Proof:

Let

$$\Phi = \frac{I_c}{-\sigma} + \frac{\alpha(\mu+v_c)-\sigma\beta}{\alpha\sigma(\mu+v_{nc}+\sigma)} I_{nc}$$

Be a lyaponuv function of the model (2.1) on Ω with $R_{c0} \leq 1$ and $f(I_c, I_{nc}, S_c, S_{nc}) \geq 0$. Differentiating Φ along the solutions of (2.1) gives

$$\Phi^{/} = \omega^T V^{-1} x^{/}$$

Where $(I_c \quad I_{nc})^T$, $x \quad = (F - V)x - f(x, y)$

$$\Phi \quad = \omega^T V^{-1} \{(F - V)x - f(x, y)\}$$

$$\Phi \quad = \omega^T V^{-1}(F - V)x - \omega^T V^{-1} f(x, y)$$

$$\Phi = (R_{c0}-1)\omega^T x - \omega^T V^{-1} f(x,y)$$
$$\Phi = (R_{c0}-1)\omega^T x - \omega^T V^{-1} f(x,y)$$
$$\Phi = (R_{c0}-1)\left\{\frac{\beta(\sigma+\mu-b)}{\alpha(b-\mu-\sigma)} I_c + I_{nc}\right\} - \frac{B(t)(S_c - S_{c0})}{\sigma} - \frac{\alpha(\mu+v_c)-\sigma\beta}{\alpha\sigma(\mu+v_{nc}+\sigma)} B(t)(S_{nc0}-S_{nc})$$

Thus it follows that $\Phi \leq 0$ if $R_{c0} \leq 1$. If $R_{c0} = 1$ then $\Phi = 0$ if and only if $I_c = I_{nc} = 0$. If $R_{c0} = 1$ then $\Psi = 0$ if and only if case1: $I_c = I_{nc} = 0$, case2: $S_c = S_{c0}$ and $S_{nc0} = S_{nc} = 0$

Therefore every solution trajectory of equations in the model (2.1) converges to the largest compact invariant set $M = \{I_c, I_{nc}, S_{c0}, S_{nc0}\}$, and the only point in M is the disease-free equilibrium. Then by LaSalle's invariant principle [20], the disease free equilibrium is globally asymptotically stable in Ω if $R_{c0} \leq 1$.

That is every solution trajectory of equations in the model (2.1) approaches the disease free equilibrium as $t \to \infty$

4.0 SUMMARY AND CONCLUSION

This work explored the contribution of a non-pharmaceutical control measure, male circumcision to combat the spread of the world's threatening disease, the HIV/AIDS. The work considers male circumcision as a medium for narrowing the initial fraction of at risk population and the recruitment into the at risk population. This will in turn enhance the reduction of transmission coefficient in the population. It establishes the condition for positivity and boundedness of the model, which enhance the existence and uniqueness of the solution of the model thereby making the model to be epidemiologically meaningful.

The main mathematical technique used is the Lyapunov direct method which is successfully to two cases: $S_c^0 = 0, when the population is not circumcised$ **and** $S_{nc}^0 = 0, when the population is fully circumcised$**, to study the global asymptotic stability of the model. It was established that** the local stability is guaranteed if the product of the probability of transmission by individuals and the average number of contact per unit time is less than the sum product of circumcision rate and that of the natural death of the individual in the population. That is if circumcision is encouraged in the population it greatly enhances the eradication of HIV/AIDS. When $S_{nc}^0 = 0$ the analysis showed that, every solution trajectory of equations in the model (2.1) converges to the largest compact invariant set $M = \{I_c, I_{nc}, S_{c0}, S_{nc0}\}$, and the only point in M is the disease-free equilibrium. Then by LaSalle's invariantprinciple [7], the disease free equilibrium is globally asymptotically stable in Ω if $R_{c0} \leq 1$.

That is every solution trajectory of equations in the model (2.1) approaches the disease free equilibrium as $t \to \infty$

When $S_c^0 = 0$ the analysis showed that every solution trajectory of equations in the model (2.1) converges to the largest compact invariant set $M = \{I_c, I_{nc}, S_{c0}, S_{nc0}\}$, and the only point in M is the disease-free equilibrium. Then by LaSalle's invariant principle [7], the disease free equilibrium is globally asymptotically stable in Ω if $R_{nc0} \leq 1$.

That is every solution trajectory of equations in the model (2.1) approaches the disease free equilibrium as $t \to \infty$

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