Modeling HIV Transmission Dynamics among Male prisoners in Sub-Saharan Africa

S. Mushayabasa, and C. P. Bhunu

Abstract— HIV epidemic has struck prisons and other places of detention around the world with particular severity. A deterministic HIV/AIDS model which incorporates male prisoners and homosexuality is considered. An epidemic threshold value, the reproductive number (R_0) is proposed, and qualitatively used to examine the stability of the system. Depending on the value of the threshold value this state can be either infection-free $(R_0 \leq 1)$ or endemic $(R_0 \geq 1)$. Comprehensive analyses on these two steady states (infection-free and endemic) have shown that they are both globally stable. Sensitivity analysis is performed on R_0 . A key result arising from this model is that, efforts to control the HIV epidemic in Africa that ignore the prison situation are probably doomed to failure.

Index Terms—HIV/AIDS, male prisoners, homosexuality, reproductive number, stability

I. INTRODUCTION

ABOUT 668, 000 men and women are incarcerated in sub-Saharan Africa. South Africa has the highest prison population with 157,402 people behind bars in the region and 335 prisoners per 100,000 of the national population; it has the ninth largest prison population in the world [22]. Sub-Saharan Africa remains the region most heavily affected by HIV. In 2008, sub-Saharan Africa accounted for 67% of HIV infections worldwide, 68% of new HIV infections among adults and 91% of new HIV infections among children. The HIV epidemic has struck prisons and other places of detention around the world with particular severity [11], [12]. International data show that HIV prevalence among prisoners is between six to fifty times higher than that of the general adult population. For example, in the USA the ratio is 6:1; in France it is 10:1; in Switzerland 27:1 and in Mauritius 50:1 [12]. On a global scale, the prison population is growing rapidly, with high

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incarceration rates leading to overcrowding, which largely stems from national law and criminal justice policies. In most countries, overcrowding and poor physical conditions prevail [14]. This phenomenon poses significant health concerns with regard to control of infectious diseases-and HIV prevention and care most of all [15]. Prison populations are predominantly male and most prisons are male-only institutions, including the prison staff. In such a gender exclusive environment, male-to-male sexual activity (prisoner-to-prisoner and guard-to-prisoner) is frequent [13]. The actual number of instances is likely to be much higher than what is reported mainly due to continual denial, fear of being exposed or the criminalization of sodomy and homosexuality. Homosexual activity is illegal in every southern African country with the exception of South Africa. According to UNAIDS report released in 1995, 8.4 percent of men in the Zambian prison of Kamfinsa reported anal sex, with the true figure likely to be higher [11]. A 1999 Penal Reform International study of Zomba prison in Malawi reported respondents as estimating that between 10 to 60 percent of prisoners had participated in, homosexual activity at least once. While much of the sex among men in prisons is consensual, rape and sexual abuse are often used to exercise dominance in the culture of violence that is typical of prison life [11]. Inmate rape, including male rape, is considered one of the most ignored crimes. Sexual and physical abuse in custody remains a tremendous human rights problem [1]. Among prisoners, the rate of sexual abuse is as high as 27 per cent, including rape by prison guards [22]. Available data indicate that rape is used as a disciplinary tactic and a control mechanism by prison authorities who not only ignore or do not prevent inmate rape, but encourage it as a punishment tool [19]. Prisoners form alliances, hierarchies and enmities that thrive on creating an atmosphere of fear and control where trade of sexual favors and sexual enslavement is widespread. However, those involved in sexual abuse do not consider themselves to be bisexual or homosexual, thus often hindering the dissemination of HIV prevention messages.

Three aspects of man-to-man sexual activity in prison make it a high risk for HIV transmission: anal intercourse, rape and the presence of sexually transmitted infections (STIs). Related problems in prisons across Southern Africa include overcrowding, shortages, corruption, and the presence of juveniles alongside adult prisoners. The potential for the

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spread of HIV is also increased by a lack of information and education, and a lack of proper medical care. STIs, if left untreated, can greatly increase a person's vulnerability to HIV through sexual contact, UNAIDS noted [23].

Mathematical models have become invaluable management tools for epidemiologists, both shedding light on the mechanisms underlying the observed dynamics as well as making quantitative predictions on the effectiveness of different control measures. The literature and development of mathematical epidemiology is well documented and can be found in [2], [4], [5]. Modeling the spread of HIV is an important and interesting topic for a lot of researchers. Many mathematical models have been proposed by different researchers; see for instance Daley and Gani [6], [7], [9], [10]. Ching et al, [6] proposed a fast numerical algorithm for solving the equilibrium values of the spread of HIV in a network of prisons. This paper seeks to use mathematical models to gain insights on transmission of HIV among male prisoners in the context of homosexuality.

II. MODEL FORMULATION

We divide the host population into the following epidemiological classes or subgroups:

- S_{nh} : susceptible non-homosexual,
- S_h : susceptible homosexual,
- I_{nh} : non-homosexual HIV infectives,
- I_h :homosexual HIV infectives,
- A : AIDS cases.

Thus, the total population is given by $N = S_{nh} + I_{nh} + S_h + I_h + A$. We assume that all individual within the terminal form of AIDS are too sick to engage in homosexual and therefore do not contribute to the HIV/AIDS transmission dynamics, thus the total sexually active population is given by $N_h = S_h + I_h$. Individuals are recruited into the prisons through committing different crimes at a constant rate Λ . Quantifying the proportion individuals recruited into each of the aforementioned epidemiological classes will require an extensive sensitivity analysis with parameter values estimated from real demographic data. However, we assume that a fraction π_0, π_1, π_2 , and π_3 are recruited into S_{nh} , I_{nh} , S_h , and I_h respectively. Since individuals in the AIDS class do not influence the dynamics of the epidemic we have neglected the proportion of recruitment into this class. Non-homosexuals susceptible individuals and nonhomosexuals HIV infectives are assumed to become homosexuals at a constant rate lpha . Susceptible homosexuals S_{h} are infected with the disease following unprotected sexual contact with a homosexual HIV infective at rate $\lambda = \frac{\beta I_h}{N_h}$. The parameter β is the product of effective

contact rate for HIV infection and the transmission probability of HIV per contact. Non-homosexual HIV

infectives who would not join homosexuality behavior progress to AIDS stage at a constant rate γ , while homosexual HIV infectives progress to AIDS stage at a constant rate σ . Natural mortality rate μ is assumed to be constant in all classes, with individuals in AIDS class suffering an additional mortality due to the disease at a rate ν . Furthermore, individuals are released from the prison at rate ω , which is assumed to be constant in all classes. The model flow diagram is depicted in Figure 1 below.

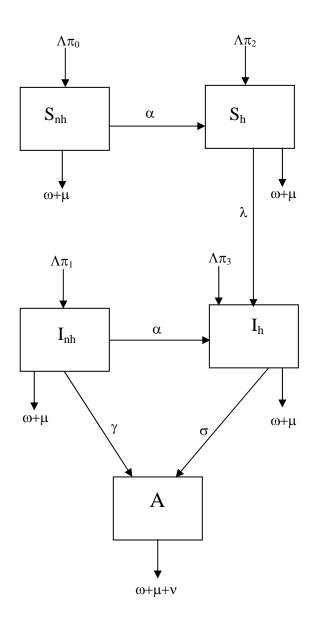


Figure 1: Model flow diagram

From the descriptions and assumptions on the dynamics of the epidemic made above, the following are the model equations.

$$\frac{dS_{nh}}{dt} = \Lambda \pi_0 - (\mu + \omega + \alpha)S_{nh},$$

$$\frac{dI_{nh}}{dt} = \Lambda \pi_1 - (\mu + \alpha + \omega + \gamma)I_{nh},$$

$$\frac{dS_h}{dt} = \Lambda \pi_2 + \alpha S_{nh} - (\lambda + \omega + \mu)S_h,$$

$$\frac{dI_h}{dt} = \Lambda \pi_0 + \alpha I_{nh} + \lambda S_h - (\sigma + \omega + \mu)I_h,$$

$$\frac{dA}{dt} = \gamma I_{nh} + \sigma I_h - (\omega + \mu + \nu)A.$$
(1)

From system (1) S_{nh} and I_{nh} can be presented as

$$S_{nh}^{*} = \frac{\Lambda \pi_{0}}{\mu + \alpha + \omega},$$

$$I_{nh}^{*} = \frac{\Lambda \pi_{1}}{\mu + \alpha + \gamma + \omega}.$$
(2)

So that system (1) reduces to

$$\frac{dS_{h}}{dt} = \Lambda \pi_{2} + \alpha S_{nh}^{*} - (\lambda + \omega + \mu)S_{h},$$

$$\frac{dI_{h}}{dt} = \Lambda \pi_{0} + \alpha I_{nh}^{*} + \lambda S_{h} - (\sigma + \omega + \mu)I_{h},$$

$$\begin{cases} (3) \\ \frac{dA}{dt} = \gamma I_{nh}^{*} + \sigma I_{h} - (\omega + \mu + \nu)A. \end{cases}$$

System (3) is epidemiologically and mathematically wellposed in the domain

$$\Phi = \left\{ (S_h, I_h, A) \in \mathfrak{R}^3_+ : N \leq \frac{\Lambda}{\mu} \right\}$$

The domain Φ is valid epidemiologically since all parameters and state variables for model system (3) are assumed to be non-negative for $t \ge 0$.

III. EQUILIBRIUM STATES, REPRODUCTIVE NUMBER AND STABILITY

System (3) has an evident disease-free (DFE) given by

$$\Omega = (S_h^0, I_h^0, A^0) = \left\{ \frac{\Lambda([\alpha \pi_0 + (\mu + \alpha + \omega)\pi_2])}{\mu(\mu + \alpha + \omega)}, 0, 0 \right\}$$

A. Reproductive number

The basic reproductive number: a threshold value representing how many secondary infections result from the introduction of one infected individual into a population of susceptible [8]. Following the next generation approach in [8] the reproductive number for model system (3) is given by

$$R_0 = \frac{\beta}{\sigma + \omega + \mu}$$

The reproductive number (R_0) measures the average number of secondary HIV cases produced by a single HIV homosexual infective during his/her entire sexual period in prison in the absence of HIV intervention strategies. The value that (R_0) takes can indicate the circumstances in which an epidemic is possible. The interpretation of (R_0) is very straightforward: when the product of effective contact rate for HIV infection and the transmission probability of HIV per contact β the numerator of R_0 is greater than the sum of the natural death rate, prison release rate and rate of progression to AIDS stage for homosexual infectives $(\mu + \omega + \sigma)$ then, epidemic prevalence will rise.

B. Sensitivity analysis of the reproductive number

We now investigate the sensitivity of R_0 to each of the parameters, following Arriola and Hyman [3]. The normalized forward sensitivity index with respect to each of the parameters is presented below:

$$\frac{\beta}{R_{0}} \frac{\partial R_{0}}{\partial \beta} = 1,$$

$$\frac{\mu}{R_{0}} \frac{\partial R_{0}}{\partial \mu} = \left| \frac{-\mu}{\mu + \sigma + \omega} \right| < 1,$$

$$\frac{\sigma}{R_{0}} \frac{\partial R_{0}}{\partial \sigma} = \left| \frac{-\sigma}{\mu + \sigma + \omega} \right| < 1,$$

$$\frac{\omega}{R_{0}} \frac{\partial R_{0}}{\partial \omega} = \left| \frac{-\omega}{\mu + \sigma + \omega} \right| < 1.$$
(4)

From the above calculations (4) we observe that R_0 is most sensitive to changes in β . An increase in β will bring about an increase of the same proportion in R_0 and a decrease in β will result in a decrease in R_0 with about an equivalent magnitude. Sensitivity analysis of μ and σ have an inverse proportional relationship with R_0 implying that any increase in either natural mortality rate or rate of progression to AIDS stage will reduce the reproductive

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number, but however this is not practical. Although, sensitivity analysis of ω have an inverse proportional relationship with R_0 , this will only reduce HIV prevalence in prison but increase the epidemic burden in the community since some individuals released from the prison are HIV infectives, furthermore some would have adopted the homosexuality behavior which they were not doing before going to prison. Results from the sensitivity analysis of R_0 suggest that more effort should be done on reducing β , and this can be attained through the provision of HIV preventative measures such as condoms and so on.

C. Stability analysis of disease-free

We now investigate the local stability of the trivial equilibrium point Ω . The Jacobean ($J(\Omega)$) of system (3) at disease-free is given by

$$J(\Omega) = \begin{bmatrix} -\mu - \omega & -\beta & 0\\ 0 & \beta - (\mu + \omega + \sigma) & 0\\ 0 & \sigma & -\mu - \omega - v \end{bmatrix}$$

The eigenvalues of $J(\Omega)$ are:

$$\kappa_1 = -\omega - \mu,$$

$$\kappa_2 = \beta - (\mu + \sigma + \omega),$$

$$\kappa_3 = -\omega - \mu - \nu.$$

The two eigenvalues κ_1 and κ_3 , are clearly real and negative, while the third eigenvalues κ_2 is real and negative if and only if $\beta < (\sigma + \omega + \mu)$. Thus, κ_2 satisfies the necessary conditions for local stability of Ω whenever $R_0 < 1$. We summarize the result in Theorem 1 below.

Theorem 1: The disease-free equilibrium point Ω is locally asymptotically stable if $R_0 < 1$, and unstable if $R_0 > 1$.

We now examine the global stability of the Ω using the Comparison Theorem as in [17], [18]. Note that the equations of infected components in system (3) can be written as

$$\begin{bmatrix} \frac{dI_h}{dt} \\ \frac{dA}{dt} \end{bmatrix} = \begin{bmatrix} F - V \end{bmatrix} \begin{bmatrix} I_h \\ A \end{bmatrix} - \beta \begin{bmatrix} 1 - \frac{S_h}{N_h} \end{bmatrix} \begin{bmatrix} 1 & 0 \\ 0 & 0 \end{bmatrix} \begin{bmatrix} I_h \\ A \end{bmatrix},$$

Where F and V are given by; $F = \begin{bmatrix} \beta & 0 \\ 0 & 0 \end{bmatrix}$

$$V = \begin{bmatrix} \mu + \sigma + \omega & 0 \\ -\sigma & \omega + \mu + \nu \end{bmatrix}$$

Since, $S_h \leq N_h$ (for all $t \geq 0$) in Φ it follows that

$$\begin{bmatrix} \frac{dI_{h}}{dt} \\ \frac{dA}{dt} \end{bmatrix} \leq \begin{bmatrix} F - V \end{bmatrix} \begin{bmatrix} I_{h} \\ A \end{bmatrix}$$
(5)

Using the fact that the eigenvalues of the matrix F - V all have negative real parts, it follows that the linearized differential inequality system (5) is stable whenever $R_0 < 1$. Consequently, $(I_h, A) \rightarrow (0,0)$, as $t \rightarrow \infty$. Thus, by Comparison Theorem [17], $(I_h, A) \rightarrow (0,0)$, as $t \rightarrow \infty$. Evaluating system (3) at $I_h = A = 0$, one gets $S_h = S_h^0$ for $R_0 \le 1$. Hence, the disease-free is globally asymptotically-stable for $R_0 \le 1$. We summarize the result in Theorem 2 below.

Theorem 2: The disease-free equilibrium point Ω is globally asymptotically stable if $R_0 < 1$, and unstable if $R_0 > 1$.

D. Endemic equilibrium

Endemic equilibrium points are steady-state solutions where the disease persists in the population (all state variables are positive). By assuming that $\beta > (\sigma + \omega + \mu)$, we are looking for positive equilibrium point for system (3). In this case the endemic equilibrium is denoted by $\Theta = (S_h^*, I_h^*, N_h^*)$. Individuals in the AIDS class do not influence the dynamics of the disease hence we shall omit the last equation of system (3).

Definition 1: The disease is said to persist if there exists some $\mathcal{E} > 0$ such that

$$\limsup_{t \to \infty} \frac{I_h(t)}{N_h(t)} \ge \varepsilon \text{ , for all nonnegative}$$

solutions of (3) with, $I_h(0) > 0$ and $S_h(0) >> 0$.

We now prove the above definition using Lyapunov's direct stability method. Thus;

$$\frac{dI_{h}}{dt} > \frac{\beta I_{h} S_{h}}{N_{h}} - (\mu + \omega + \sigma)$$
$$\Leftrightarrow \frac{dI_{h}}{dt} > (\sigma + \omega + \mu) \left[\frac{\beta}{\sigma + \omega + \mu} - 1 \right] I_{h}$$
$$\Rightarrow \frac{dI_{h}}{dt} > (\sigma + \omega + \mu) [R_{0} - 1]$$

The linearized differential inequality system (10) holds for $R_0 > 1$. We summarize the result in Theorem 3, below.

Theorem 3: If $R_0 > 1$, the endemic equilibrium point Θ exists.

We now investigate the global stability of Θ . In order to investigate the global stability of the endemic equilibrium, we adopt the approach in [16]. Assume $\beta > (\sigma + \omega + \mu)$, then Θ exists for all $S_h, I_h > \varepsilon$, for some $\varepsilon > 0$. Let $\lambda = g(S_h, I_h)$ be a positive and monotonic function, and define the following continuous function in \Re^2_+ (for more details, see Korobeinikov, 2006 [16]. A function

$$V(S_{h}, I_{h}) = S_{h} - \int_{\varepsilon}^{S_{h}} \frac{g(S_{h}^{*}, I_{h}^{*})}{g(\tau, I_{h}^{*})} d\tau + I_{h} - \int_{\varepsilon}^{I_{h}} \frac{g(S_{h}^{*}, I_{h}^{*})}{g(S_{h}^{*}, \tau)} d\tau$$
(6)

If $g(S_h, I_h)$ is monotonic with respect to its variables, then the endemic state Θ is the only extremum and the global minimum of this function. Indeed

$$\frac{\partial V}{\partial S_{h}} = 1 - \frac{g(S_{h}^{*}, I_{h}^{*})}{g(S_{h}, I_{h}^{*})}, \left\{ \begin{array}{c} \end{array} \right\}$$

$$\frac{\partial V}{\partial I_{h}} = 1 - \frac{g(S_{h}^{*}, I_{h}^{*})}{g(S_{h}^{*}, I_{h})} \right\}$$
(7)

Grow, monotonically, and then the function $g(S_h, I_h)$ has only one stationary point. Further, since

$$\frac{\partial^{2} V}{\partial S_{h}^{2}} = \frac{g(S_{h}^{*}, I_{h}^{*})}{\left[g(S_{h}, I_{h}^{*})\right]^{2}} \times \frac{\partial g(S_{h}, I_{h}^{*})}{\partial S_{h}} \bigg|_{\left(\frac{\partial^{2} V}{\partial I_{h}^{2}}\right)^{2}} = \frac{g(S_{h}^{*}, I_{h}^{*})}{\left[g(S_{h}^{*}, I_{h})\right]^{2}} \times \frac{\partial g(S_{h}^{*}, I_{h})}{\partial I_{h}} \bigg|_{\left(\frac{\partial^{2} V}{\partial I_{h}^{*}}\right)^{2}} = \frac{g(S_{h}^{*}, I_{h}^{*})}{\left[g(S_{h}^{*}, I_{h})\right]^{2}} \times \frac{\partial g(S_{h}^{*}, I_{h})}{\partial I_{h}} \bigg|_{\left(\frac{\partial^{2} V}{\partial I_{h}^{*}}\right)^{2}}$$

Equations in system (8) are non negative, then the point Θ is a minimum, that is, $V(S_h, I_h) \ge V(S_h^*, I_h^*)$, hence V is a Lyapunov function, and its time derivative is given by

$$\begin{aligned} \frac{dV}{dt} &= S_{h}^{'} - S_{h}^{'} \left[\frac{g(S_{h}^{*}, I_{h}^{*})}{g(S_{h}, I_{h}^{*})} \right] + I_{h}^{'} - I_{h}^{'} \left[\frac{g(S_{h}^{*}, I_{h}^{*})}{g(S_{h}^{*}, I_{h})} \right] \\ &= (\mu + \omega) S_{h}^{*} \left[1 - \frac{S_{h}}{S_{h}^{*}} \right] \left[1 - \frac{g(S_{h}^{*}, I_{h}^{*})}{g(S_{h}, I_{h}^{*})} \right] \\ &+ (\mu + \omega + \sigma) I_{h}^{*} \left[1 - \frac{I_{h}}{I_{h}^{*}} \right] \left[1 - \frac{g(S_{h}^{*}, I_{h}^{*})}{g(S_{h}^{*}, I_{h})} \right] \\ &+ g(S_{h}^{*}, I_{h}^{*}) \left[1 - \frac{g(S_{h}^{*}, I_{h}^{*})}{g(S_{h}, I_{h}^{*})} \right] \left[\frac{g(S_{h}^{*}, I_{h}^{*})}{g(S_{h}^{*}, I_{h})} - 1 \right] \\ &+ g(S_{h}^{*}, I_{h}) \left[1 - \frac{g(S_{h}^{*}, I_{h}^{*})}{g(S_{h}^{*}, I_{h})} \right] \left[\frac{g(S_{h}^{*}, I_{h}^{*})}{g(S_{h}^{*}, I_{h})} - 1 \right] \end{aligned}$$

Since $\Theta > 0$, the function $g(S_h, I_h)$ is concave with respect to I_h , and $\frac{\partial^2 g(S_h, I_h)}{\partial I_h^2} \le 0$, then $\frac{dV}{dt} \le 0$, for

all $S_h, I_h > 0$. Also, monotonicity of $g(S_h, I_h)$ with respect to S_h and I_h ensures that

$$\begin{bmatrix} 1 - \frac{S_h}{S_h^*} \end{bmatrix} \begin{bmatrix} 1 - \frac{g(S_h^*, I_h^*)}{g(S_h, I_h^*)} \end{bmatrix} \le 0, \\ \begin{bmatrix} 1 - \frac{I_h}{I_h^*} \end{bmatrix} \begin{bmatrix} 1 - \frac{g(S_h^*, I_h^*)}{g(S_h^*, I_h)} \end{bmatrix} \le 0, \\ \begin{bmatrix} 1 - \frac{g(S_h^*, I_h^*)}{g(S_h, I_h^*)} \end{bmatrix} \begin{bmatrix} \frac{g(S_h^*, I_h^*)}{g(S_h^*, I_h)} - 1 \end{bmatrix} \le 0, \\ \begin{bmatrix} 1 - \frac{g(S_h^*, I_h^*)}{g(S_h^*, I_h)} \end{bmatrix} \begin{bmatrix} \frac{g(S_h^*, I_h^*)}{g(S_h, I_h^*)} - 1 \end{bmatrix} \le 0. \end{bmatrix}$$
(9)

Equation in (9) holds for S_h , $I_h > 0$. Thus, we establish the following result.

Theorem 4: The endemic equilibrium Θ is globally asymptotically stable whenever conditions outlined in (9) are satisfied.

IV. CONCLUSION

HIV/AIDS situation in prisons in Africa has been a highly neglected area. In this paper, a deterministic HIV/AIDS model in the context of male prisoners and homosexuality is formulated. The qualitative features of its equilibrium are analyzed. It is found that both the disease-free equilibrium and the endemic equilibrium exists whenever the reproduction number R_0 is less than a unity and greater than unity respectively, and are globally asymptotically stable. Sensitivity analysis on the reproduction number identifies β , the product of effective contact rate for HIV infection and the transmission probability of HIV per contact, as the most useful parameter to target for the

reduction of R_0 . For practical purposes, this corresponds to the need to address HIV prevention in prisons in the context of homosexuality. Although, sensitivity analysis have shown that an increase in ω , (the rate at which individual are released from the prison after completing serving their sentence or due to amnesty) will reduce the reproductive, we however note that this will only HIV in the prison but will continue to increase HIV prevalence in the community, since some of these individuals released into the community are infected, and some would have adopted the homosexuality behavior. Furthermore, we have noted that it's important for collection of data relevant to the key parameters by epidemiologists and treatment providers at a global level as this would be of significant use from an implementation and policy perspective.

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REFERENCES

- [1] Amnesty International (2001). Abuse of Women in Custody: Sexual Misconduct and Shackling of Pregnant Women.
- [2] Anderson, R.M. May, Infectious Diseases of Humans, Dynamics and Control, Oxford University Press, 1991.
- [3] Arriola L, Hyman J, Lecture notes, forward and adjoint sensitivity analysis: with applications in Dynamical Systems, Linear Algebra and Optimisation Mathematical and Theoretical Biology Institute, Summer, 2005.
- [4] Bailey N, The Mathematical Theory of Infectious Diseases, Charles Griffin, 1975.
- [5] Brauer F, C. Castillo-Chavez, Mathematical Models in Population Biology and Epidemiology, Springer, 2000.
- [6] Ching W, T. Ng, and S. Chung, On Modeling SARS in Hong Kong, International Journal of Applied Mathematics, 13(2003), pp. 1–7.
- [7] Daley Dand J. Gani, Epidemic modelling: An Introduction, Cambridge University Press, Cambridge, 1999.
- [8] Diekmann 0, J.A.P. Heesterbeek, Mathematical Epidemiology of Infectious Diseases, John Wiley 171 & Son, Ltd, 2000.
- [9] Greenhalgh D and F. Lewis, The General Mixing of Addicts and Needles in a Variable infectivity Needle-sharing Environment, Journal of Mathematical Biology, 44(2002), pp.561-598.
- [10] Huang X and M. Villasana, an Extension of the Kermack-Mckendrick Model for AIDS Epidemic, Journal of the Franklin Institute, 342(2005), pp. 341-351.
- [11] Human Rights Watch (1991). No Escape: Male Rape in USA Prisons.
- [12] Human Rights Watch (2006). HIV/AIDS in Prisons.
- [13] Human Rights Watch (2002). World Report; Human Rights Watch (1999). World Report. Special Programs and Campaigns—Prisons.
- [14] International Centre for Prison Studies (2006). The World Female Imprisonment List. King's College, London, UK.
- [15] International Centre for Prison Studies (2007). The World Prison Population List. King's College, London, UK.
- [16] Korobeinikov A (2006) Lyapunov functions and global stability for SIR and SIRS epidemiological models with non-linear transmission. Bulletin of Mathematical Biology 30: 615-626.
- [17] Lakshmikantham V, Leela S. and Martynyuk A.A. Stability analysis of nonlinear systems, Marcel Dekker, New York, 1989. ISBN 0-8247-8067-1. Pure and Applied Mathematics: A Series of Monographs and Textbooks, Vol. 125.
- [18] Mushayabasa S, Tchuenche J.M, Bhunu C.P., Ngarakana-Gwasira E (2011), Modeling gonorrhea and HIV co-interaction. Journal of Biological and Information Processing Sciences 103: 27-37.
- [19] Parenti J (1999). Rape as a Disciplinary Tactic; Gordon N (2001). Rape Used as a Control in U.S. Prisons; Berger V (2002). Sentenced to Rape.

- [20] The World Prison Population List Seventh Edition- Jan 2007, International Centre for Prison Studies, King's College, London.
- [21] UNAIDS, Sub-Saharan Africa AIDS epidemic update regional summary, Jointed United Nations Programme on HIV/AIDS (UNAIDS) and World Health Organisation,(WHO), Geneva, Switerland, 2009.
- [22] US Centers for Disease Control and Prevention (2002). Prison Rape Spreading Deadly Diseases; Lehner E (2001). Hell Behind Bars: The Crime That Dare Not Speak Its Name.
- [23] UNAIDS (2006). AIDS Epidemic Update. Geneva, UNAIDS.