
**MATHEMATICAL MODEL OF HIV/AIDS AT TECHIMAN MUNICIPALITY,
GHANA**

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ABSTRACT: *The study examined the prevalence of HIV/AIDS in the Techiman Municipality of Ghana. We used SI model with standard incidence to analyze, model and predict the prevalence of HIV/AIDS in the Techiman Municipality. The model has two equilibrium states: the disease-free equilibrium and the endemic equilibrium states respectively. The stability condition of each equilibrium point is discussed. The basic reproductive number (R_0) of HIV/AIDS infection is estimated to be 0.4641 in the Techiman Municipality. Our work shows that the reproductive number of HIV/AIDS infection in the Techiman Municipality is less than 1 ($R_0 < 1$) and therefore concluded that the disease is not epidemic in the municipality as described by the GAC in their study in 2005. We recommend that education on HIV/AIDS in the municipality should be intensified so as to decrease the rate of transmission of HIV in the municipality.*

KEYWORDS: SI model, Stability analysis, Equilibrium points, Mathematical model, Sensitivity Analysis

INTRODUCTION

HIV/AIDS is a global pandemic [1]. It is transmitted through the exchange of bodily fluids, predominantly through unprotected sexual intercourse, but also through sharing of unsterilized needles or transfusion with infected blood supplies [2]. As of 2012, approximately 35.3 million people were living with HIV globally [3]. Of these, approximately 17.2 million were men, 16.8 million were women and 3.4 million were less than 15 years old. There were about 1.8 million deaths from AIDS in 2010, down from 2.2 million in 2005 [4].

Sub-Saharan Africa is the region most affected. In 2010, an estimated 68% (22.9 million) of all HIV cases and 66% of all deaths (1.2 million) occurred in this region. This means that about 5% of the adult populations is infected. Here in contrast to other regions women compose nearly 60% of cases. South Africa has the largest population of people with HIV of any country in the world at 5.9 million [4]. South and South East Asia (a region with about 2 billion people as of 2010, over 30% of the global population) has an estimated 4 million cases (12% of all people living with HIV), with about 250,000 deaths in 2010. Approximately 2.5 million of these cases are in India, where however the prevalence is only about 0.3% (somewhat higher than that found in Western and Central Europe or Canada). Prevalence is lowest in East Asia at 0.1% [4].

In 2008, approximately 1.2 million people in the United States had HIV; 20% did not realize that they were infected [5]. It resulted in about 17,500 deaths [5]. In the United Kingdom, as of 2009, there were approximately 86,500 cases and 516 deaths [6]. In Australia, as of 2009, there were about 21,171 cases and around 23 deaths. [6] In Canada, as of 2008 there were about 65,000 cases and 53 deaths [8]. Since AIDS was first recognized in 1981 and by 2009 has led to nearly 30 million deaths [9].

Ghana is one of the five countries in sub-Saharan Africa whose HIV prevalence declined by more than 52 per cent between 2001 and 2010 among young people aged between 15 and 24, according to a UNAIDS 2011 report [10]. The national HIV prevalence declined from 3.6 per cent in 2003 to 1.3 per cent in 2013 [11]. Prevalence among sex workers had reduced from 35 per cent in 2006 to 11 per cent in 2011 [12]. HIV infection estimates showed that in 2012, 235,982 persons were living with HIV, with 27,734 being children. An estimated 7,138 new infections were recorded as well as 11,655 AIDS deaths and 852 new child infections in the same year [12].

The strategic location of Techiman as a commercial centre and transit point attract a large number of migrants in and out of the Municipality. There is therefore a high rate of commercial sex activity and high risk behaviours. These among others have resulted in the high prevalence rate of HIV/AIDS of 4.2% as compared to the regional rate of 4.7% and national rate of 2.7% as at the year 2005 [13].

Techiman has been identified as a high HIV prevalence area. A study conducted by the Ghana AIDS Commission (GAC) in 2005, revealed that out of 1,180 people interviewed 54% of men and 52% women reported having had two sexual partners in the previous four weeks. 50.5% of the people socializing at the hotspots reported never used condoms [13].

High risk behaviour is therefore common, facilitating the spread of HIV. HIV/AIDS featured for the first time as one of the top ten causes of admission and death in 2005 [13]. This indicates that the disease is spreading at a faster rate in the Municipality. We therefore want to use *SI* disease model to study the current state of HIV/AIDS at Techiman Municipality.

METHODOLOGY

The population used for the study is the people of Techiman Municipality. A primary data was obtained from the Holy Family Hospital of the municipality. The *SI* model with standard incidence was used to analyze, model and predict the prevalence of HIV/AIDS in the municipality. Ordinary differential equations were used to formulate the model equations. Stability and sensitivity analysis were performed in the model and ode45 Matlab software was used for the graph simulation.

MATHEMATICAL MODEL

In the *SI* model with standard incidence, the population is divided into two compartments. These are the susceptible (*S*): the number (or proportion) of individuals who are susceptible to

the disease but are not yet infected and infectives (I): the number (or proportion) of individuals who are infected with the disease, and are infectious. The proportions of individuals in the compartments S and I at time t is denoted by $S(t)$ and $I(t)$ respectively.. The diagram in Figure 1, is the schematic diagram of the SI model with standard incidence.

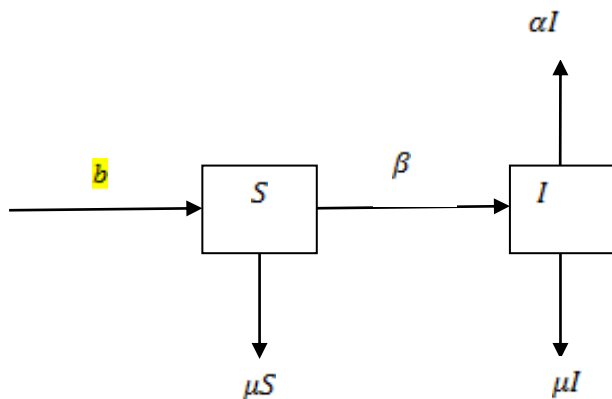


Figure 1: Schematic diagram of SI model with standard incidence, where β is the transmission rate coefficient, b is the birth rate, α is the disease induced death rate and μ is the natural death rate coefficient

The Model Assumptions

We made the following assumptions in the model:

1. The population size is constant. That is $S(t) + I(t) = N$. Where $S(t)$ and $I(t)$ are the susceptible and infective fractions of the population respectively.
2. Individuals in the infectious compartment do not recover from the infection.
3. The members of the population mix homogenously (have the same interaction with each other to the same degree).
4. The infectives continue to spread the disease till the end of the epidemic.

The model equations are:

$$\begin{aligned} \frac{dS}{dt} &= b - \beta SI - \mu S \\ \frac{dI}{dt} &= \beta SI - \mu I - \alpha I \end{aligned} \tag{3.0}$$

The system of equations 3.0 is written as:

$$\begin{aligned} \frac{dS}{dt} &= b - \beta SI - \mu S \\ \frac{dI}{dt} &= \beta SI - (\mu + \alpha)I \end{aligned}$$

Parameter	Description	Value
β	Transmission rate	0.8654
μ	Natural death rate	1.6142
α	Disease-induced death rate	0.2504
b	birth rate	12.1560

Table 1: Model Parameters**MODEL ANALYSIS****The Basic Reproductive Number**

The basic reproductive number (R_0) is the average number of infectives produced when one infective individual is introduced into a completely susceptible population [14, 15].

We determine the reproductive number of the model as follows:

$$\frac{ds}{dt} = b - \beta SI - \mu S \quad 4.0$$

$$\frac{di}{dt} = \beta SI - (\mu + \alpha)I$$

From the infective compartment of the system of equations 3.0,

$$F = \frac{\delta F}{\delta I} = \beta \quad V = \frac{\delta V}{\delta I} = \mu + \alpha$$

$$V^{-1} = \frac{1}{\mu + \alpha} \quad FV^{-1} = \frac{\beta}{\mu + \alpha}$$

but $FV^{-1} = R_0$, implies that

$$R_0 = \frac{\beta}{\mu + \alpha} \quad 4.1$$

We consider $S = N(0)$ and therefore $\beta \times S/N(0) = \beta$. When $R_0 > 1$, the solution is unstable and epidemic occurs. When $R_0 < 1$, there is no epidemic and the disease dies out. When $R_0 = 1$, the disease becomes endemic meaning, the disease remains in the population at a constant rate, individuals transmits the disease to one susceptible [16].

The Disease-Free Equilibrium and its analysis

The disease-free equilibrium is the situation where there is no infection. That is where $I = 0$. Setting only $\frac{dS}{dt} = 0$, the disease-free equilibrium is $(S^*, I^*) = (N, 0)$. We analyze the stability of the disease-free equilibrium by considering the linearized system of the system of equations 3.1 about the equilibrium point by taking the Jacobian matrix.

$$J(S^*, I^*) = \begin{bmatrix} -\beta I - \mu & -\beta S \\ \beta I & \beta S - \mu - \alpha \end{bmatrix} \quad 4.2$$

At the critical point $(S^*, I^*) = (S, 0)$ the Jacobian matrix is given by

$$J(S, 0) = \begin{bmatrix} -\mu & -\beta S \\ 0 & \beta S - \mu - \alpha \end{bmatrix}$$

$$\text{Det}(J - \lambda) = \begin{vmatrix} -\mu - \lambda & -\beta S \\ 0 & \beta S - \mu - \alpha - \lambda \end{vmatrix} = 0 \quad 4.3$$

$$\lambda_1 = -\mu \text{ and } \lambda_2 = \beta S - \mu - \alpha \quad 4.4$$

For $\text{Det}(J - \lambda)$ to be asymptotically stable, both eigenvalues must be negative. From $\text{Det}(J - \lambda) = 0$, it is obvious that $\lambda_1 = -\mu$ is negative and therefore if

$\lambda_2 = \beta S - \mu - \alpha < 0$, then both eigenvalues are negative and $R_0 < 1$. Hence the disease-free equilibrium is asymptotically stable if $R_0 < 1$. On the other hand, if $\lambda_2 = \beta S - \mu - \alpha > 0$, then, $\text{Det}(J - \lambda)$ is unstable.

The Endemic Equilibrium and its analysis

We analyze the local stability of the endemic equilibrium by considering the condition under which all the two compartmental states can co-exist in the equilibrium. That is when $S^* = \frac{\mu + \alpha}{\beta}$ and $I^* = \frac{b\beta - \mu(\mu + \alpha)}{\beta(\mu + \alpha)}$

Thus the endemic equilibrium is

$$(S^*, I^*) = \left(\frac{\mu + \alpha}{\beta}, \frac{b\beta - \mu(\mu + \alpha)}{\beta(\mu + \alpha)} \right)$$

To prove that (S^*, I^*) is asymptotically stable, we substitute $S = S^*$ and $I = I^*$ into the Jacobian matrix 4.2 to find the eigenvalues λ .

$$(J - \lambda I) = \begin{bmatrix} -\beta \left(\frac{b\beta - \mu(\mu + \alpha)}{\beta(\mu + \alpha)} \right) - \mu - \lambda & -\beta \left(\frac{\mu + \alpha}{\beta} \right) \\ \beta \left(\frac{b\beta - \mu(\mu + \alpha)}{\beta(\mu + \alpha)} \right) & \beta \left(\frac{\mu + \alpha}{\beta} \right) - (\mu + \alpha) - \lambda \end{bmatrix}$$

$$\text{Det}(J - \lambda I) = \begin{vmatrix} -\frac{b\beta}{(\mu+\alpha)} - \lambda & -(\mu + \alpha) \\ \frac{b\beta - \mu(\mu+\alpha)}{(\mu+\alpha)} & -\lambda \end{vmatrix} = 0$$

The characteristics equation of $(J - \lambda I)$ is given by

$$\Rightarrow \lambda^2 + \left(\frac{b\beta}{(\mu+\alpha)}\right)\lambda - b\beta + \mu(\mu + \alpha) = 0 \quad 4.7$$

If the trace $\lambda_1 + \lambda_2 < 0$ and its determinant $-b\beta + \mu(\mu + \alpha) > 0$ is positive, the endemic equilibrium is asymptotically stable. On the other hand, it is unstable.

We now compare equation 4.7 to the quadratic equation $\lambda^2 - p\lambda + q = 0$

$$\Rightarrow p = -\frac{b\beta}{\mu+\alpha} \quad \text{and} \quad q = -b\beta + \mu(\mu + \alpha)$$

If λ_1 and λ_2 are the eigenvalues of J , then we have

$$p(\lambda) = (\lambda - \lambda_1)(\lambda - \lambda_2) = \lambda^2 - (\lambda_1 + \lambda_2)\lambda + \lambda_1\lambda_2 \quad 4.8$$

Thus we have the identities

$$T = \lambda_1 + \lambda_2 = \text{tr}(J) \quad \text{and} \quad \Delta = \lambda_1\lambda_2 = \det(J)$$

If $p = \text{tr}(J)$ and $q = \det(J)$ then

$$\lambda_1\lambda_2 = \frac{p \pm \sqrt{p^2 - 4q}}{2}$$

Hence

$$\lambda_1, \lambda_2 = \frac{-\left(\frac{b\beta}{(\mu+\alpha)}\right) \pm \sqrt{\left(\frac{b\beta}{(\mu+\alpha)}\right)^2 - 4(-b\beta + \mu(\mu + \alpha))}}{2}$$

The nature of the roots is determined by the discriminant

$$D = \left(\frac{b\beta}{(\mu+\alpha)}\right)^2 - 4(-b\beta + \mu(\mu + \alpha))$$

and the parameters p, q and Δ allow us to determine the stability of the fixed points (origin) of the endemic equilibrium [16,17,18].

Sensitivity analysis

If the value of the transmission rate (β) is increased to 2 and the values of α and μ maintained the same, $R_0 > 1$, the disease-free equilibrium is asymptotically stable and the endemic equilibrium maintain is unstable in the model but if the value of β is reduced to any figure less than 0.9654, $R_0 < 1$.

NUMERICAL SIMULATIONS

In this section, we seek to study the behaviour of the disease near the equilibrium points by generating the respective Phase Portrait of the nonlinear *SI* differential equation model from the parameter values obtained from the Techiman Municipality. Further analysis of the model were made to better understand the nature of the disease in the municipality by plotting the *SI* model graph with time. (See Figure 2 and Figure 3 below).

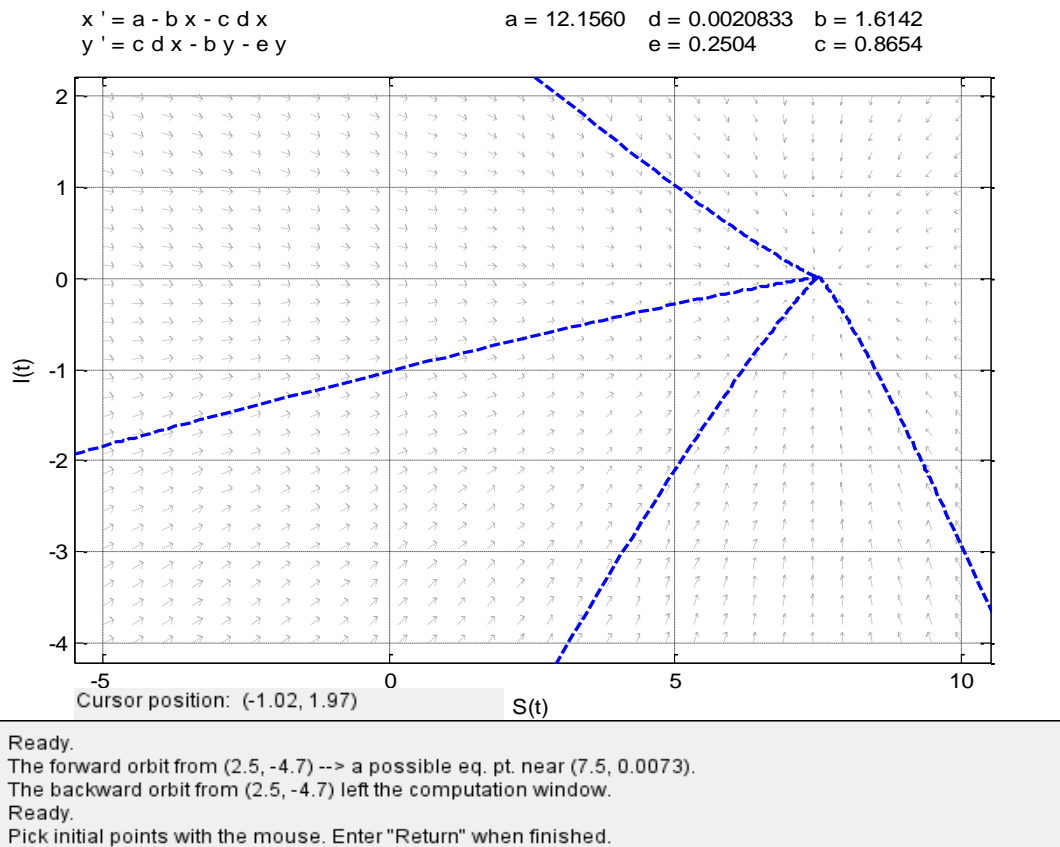


Figure 2: A phase portrait of the *SI* model with $\beta = 0.8654, \mu = 1.6142,$
 $\alpha = 0.2504$ and $b = 12.1560$.

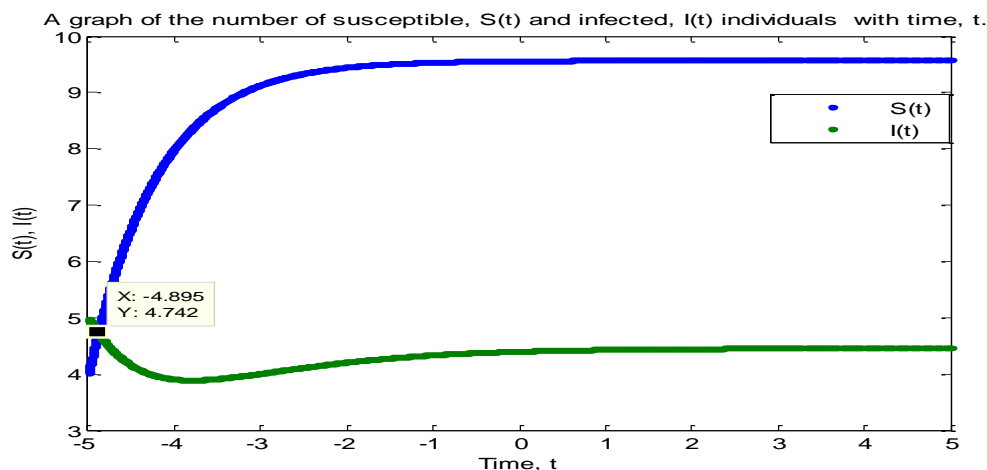


Figure 3: A numerical simulation of the SI model with $S(0) = 4800$ and $I(0) = 10$

DISCUSSION OF RESULTS

We used *SI* disease model with standard incidence to study the state of HIV/AIDS at Techiman Municipality. We discussed the existence and stability of the disease-free and endemic equilibria of the model and performed sensitivity analysis of the model parameters in the research.

We estimated the basic reproductive number of HIV at Techiman Municipality to be $R_0 = 0.4641$. This indicates that the disease is not endemic in the municipality.

In the stability analysis of the equilibrium points, the disease-free equilibrium is asymptotically stable. This is so because the eigenvalues at the disease-free equilibrium point of the model are $\lambda_1 = -1.6140$ and $\lambda_2 = -1.8132$, which is a nodal sink. From the phase portrait in Fig 2, the trajectories move toward from infinite-distant out and eventually converge to the critical point which confirms the asymptotic stability of the origin. These therefore agrees with the reproductive number of the model that the HIV/AIDS is not in its endemic state in the municipality. The eigenvalues obtained at the endemic equilibrium state are $\lambda_1 = 1.1120$ and $\lambda_2 = -6.7539$. This is a saddle and therefore unstable. This indicates the presence of the disease in the municipality. In the sensitivity analysis of the basic reproductive number, whenever the value of the transmission rate coefficient is increased and the natural death rate and disease induced death rate maintained the same reproductive number is greater than 1 ($R_0 > 1$). This means that if the disease is not combated in the municipality epidemic may occur.

CONCLUSION

Our work shows that the reproductive number of HIV/AIDS infection in the Techiman Municipality is less than 1 ($R_0 < 1$) and therefore the belief that the prevalence HIV/AIDS at the municipality is high as posited by the GAC in their study in 2005 at the municipality cannot be justified. We recommend that education on HIV/AIDS in the municipality should

be intensified so as to decrease the rate of transmission of HIV in the municipality in order not to affect the development of human resource in Ghana.

REFERENCES

- [1] Cohen, M. S., Hellmann, N., Levy, J. A., DeCock, K., and Lange, J.(2008) The spread, treatment, and prevention of HIV-1: evolution of a global pandemic". The Journal of Clinical Investigation , **118** (4): 1244–54. doi:10.1172/JCI34706. PMC 2276790. PMID 18382737. Retrieved 17 September 2012.
- [2] Blower, S. M. Gershengorn, H. B., and Grant, R. M. (2000) A tale of two futures: HIV and antiretroviral therapy in San Francisco, Science;287(5453):650–654.
- [3] "Fact Sheet". *UNAIDS.org*. 2013. (Assessed 25 July, 2014)
Available: http://en.wikipedia.org/wiki/Epidemiology_of_HIV/AIDS
- [4] "Global Report Fact Sheet". UNAIDS 2011 pg. 1-50. (Assessed 25 September, 2014)
Available: <http://www.malariagen.net/community/partner-studies/cp1-png>
- [5] Centers for Disease Control and Prevention, (CDC). "HIV surveillance--United States, 1981-2008.". *MMWR. Morbidity and mortality weekly report* 2011; **60** (21): 689–93. PMID 21637182.
- [6] Health Protection Agency (2010). HIV in the United Kingdom: 2010 Report. Assesed September 29, 2014
Available: <http://www.bmj.com/rapid-response/2011/11/03/hiv-united-kingdom-2010-report-health-protection-agency>
- [7] Benson and Kate(2010) HIV rate rising but other infections less common". The Sydney Morning Herald. Retrieved 19 October 2010.
Available: http://en.wikipedia.org/wiki/Epidemiology_of_HIV/AIDS
- [8] Surveillance; riches, Risk Assessment Division = Le VIH et le sida au Canada: rapport de surveillance en date du 31 décembre 2009 / Division de la surveillance et de l'évaluation des (2010). HIV and AIDS in Canada : surveillance report to December 31, 2009. Ottawa: Public Health Agency of Canada, Centre for Communicable Diseases and Infection Control, Surveillance and Risk Assessment Division. ISBN 978-1-100-52141-1.
- [9] "Global Report Fact Sheet". *UNAIDS*. 2010. (Assessed 25 July, 2014)
Available: www.unaids.org/documents/20101123_FS_Global_em_en.pdf
- [10] UNAIDS World Aids Day Report 2011. Page 17
- [11] Ghana's HIV Prevalence Rate Is 1.3 Percent - Politics – Nairaland] (Assessed 25 July, 2014). Available:
<http://www.nairaland.com/1998913/ghanas-hiv-prevalence-rate-1.3>
<http://www.ghanaweb.com/GhanaHomePage/health/artikel.php?ID=335047>
- [12] Statistics on Ghana's AIDS Prevalence, September 12, 2013, (Accessed on July 25, 2014)
Available: <http://omgghana.com/statistics-ghanas-aids-prevalence>
- [13] Techiman Municipal Asembly (Assessed 25 July, 2014)
Available: http://techiman.ghanadistricts.gov.gh/?arrow=atd&_=42&sa=1702
- [14] Anderson, R. M. and May, R. M. (1979) *Population Biology of infectious disease*: part i. Nature , 280; 361 – 367.
- [15] Gross K. (2004) Lecture notes on epidemiological modeling. Epi-AIMS. pages 1-3

- [16] Trottier, H. & Philippe, P.(2001) Deterministic modeling of infectious diseases: theory and methods. "The internet Journal of infectious diseases 2001.
Available: en.wikipedia.org/wiki/Epidemic_model
- [17] Sanchez, D. A. (1968) Ordinary Differential Equations and Stability Theory: An Introduction, W.H. Freeman and Company, pages 11-12.
- [18] Adu, I. K., Aidoo, A. Y., Darko, I. O. and Frimpong, E. O. (2014) Mathematical Model of Hepatitis B in the Bosomtwe District of Ashanti Region, Ghana. *Applied Mathematical Sciences*, Vol8, No 67, pages 3343-3358.