International Journal of Applied Mathematics
Volume 25 No. 4 2012, 525-545

A SIMPLY HIV/AIDS MODELS WITH DENSITY-DEPENDENT DEMOGRAPHICS

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Abstract: The models investigated for the spread of HIV/AIDS epidemic in a host population are of SIR type with a standard incidence expression. The change in the population size is governed by the modification of the logistic differential equation which incorporates a term for disease related deaths. The reproduction numbers, equilibria and stability of the system of differential equations for each model were determined. The persistence of HIV/AIDS epidemic and disease related deaths might lead to a new equilibrium population size below the carrying capacity, that is backward bifurcation, and hence this might also lead to the extinction of the host population.

AMS Subject Classification: 92C60, 92D25
Key Words: density-dependent demographics, demographics epidemiology, equilibrium, logistic equation, HIV/AIDS

1. Introduction

It is never possible that a population grows exponentially as several factors regulate population size. However, when species are introduced into a new and favorable habitat, their numbers may increase very rapidly. The rapid population growth rate can be approximated by the exponential growth curve. In reality, changes in population size and structure are caused by changes in the birth rate, the death rate and the net migration rates. Understanding a society's demography is an essential tool in determining current and future public health needs, Roberts and Heesterbeek [4].

Received: June 22, 2012

The asymptotic behaviour of solutions of an infectious model depends not only on the epidemiological formulation, but also on the demographic processes as incorporated into the model by Gao and Hethcote [2]. Hsieh and Sheu [5] proposed a model for heterosexual transmission of HIV/AIDS in a population of varying size with intervention program in which treatment and/or behaviour change of infecteds occur as increasing functions of density of the infected population. They made use of the conservation law of total sexual contacts which enabled them to reduce the two-sex model to a simpler one-sex formulation. Further, they derived reproduction numbers from which they deduced conditions for the reduction or persistence of the HIV epidemic. Our model is a modification of their model, incorporated AIDS patients in the disease transmission. The model is suitable for resource poor settings where due to already constrained health systems, provision of high scale highly active anti-retroviral therapy (HAART) can not be realized easily. Kgosimore and Lungu [7] developed a model that incorporates treatment of both juveniles who were infected with HIV/AIDS infected adults. They derived conditions under which the burden of HIV/AIDS can be reduced in the population both in the absence of and in the presence of vertical transmission. Further, they determined the critical threshold parameter which represents the demographic replacement of infectives through vertical transmission, below which treated infected juveniles can reach adulthood without causing an epidemic. Moreover, they found the reproduction number to be a decreasing function of treatment but an increasing function of the critical threshold parameter which represents the demographic replacement of infectives through vertical transmission.

An ordinary differential equation (ODE) compartmental model for disease transmission with density-dependent demographics was formulated by Gao and Hethcote [2]. We also formulated a three compartmental HIV/AIDS model with density-dependent demographics with the aim of investigating the role played by population demographics in the dynamics of HIV transmission. This is also a modification of the model in Gao and Hethcote [2] in that (i) our model is for HIV/AIDS, a disease without recovery in the absence of treatment (ii) our model has two infective classes while the model in, Gao and Hethcote [2] has one infective class. Mathematical models have been used extensively in research into the epidemiology of HIV/AIDS in order to help improve our understanding of the major contributing factors in the epidemic Naresh *et. al* [12]. The purpose of this paper is to improve our understanding in the spread of HIV/AIDS pandemic by the inclusion of density-dependent demographics in the presence of vertical transmission.

This paper is organized as follows. In Section 2, we introduce HIV/AIDS

model with density-dependent demographics. In Section 3 and Section 4 we calculate the reproduction number of the model and analyze the disease-free equilibrium point only. In Section 5, we extend the first model by including vertical transmission, then from Section 7 to Section 8 we compute the reproduction ratio of the model and analyze the disease-free equilibrium point only. Section 9 depicts the results for our analysis using a few chosen numerical simulations. Section 10 discusses the relevance of the results presented in this paper and make some recommendations for future work.

2. Model Formulation

General mathematical models for the spread of infectious diseases have been described previously by Brauer, Gao and Hethcote, Mena-Lorca and Hethcote, Kgosimore and Lungu, Nyabadza and Roberts and Heesterbeek, see [1, 2, 4, 7, 10, 13], respectively. We study a host population divided into three compartments, namely the susceptibles $X(t)$, chronic infectives $Y(t)$ and full blown AIDS $Z(t)$ populations. The study assumes the AIDS individuals in $Z(t)$ are active and have a role in the disease transmission dynamics. The total sexually active population is given by

$$N(t) = X(t) + Y(t) + Z(t).$$

The model diagram is as shown below. The model with density-dependent

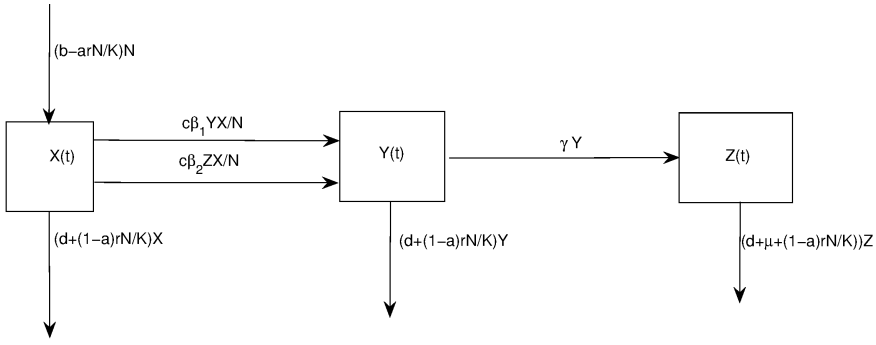

Figure 1: Model Diagram

demographics of the sexually active host population is governed by the following

system of equations derived from Figure 1:

$$\begin{aligned}
\frac{dX(t)}{dt} &= \left(b - ar\frac{N}{K}\right)N - \frac{(c\beta_1 Y + c\beta_2 Z)X}{N} - \left(d + (1-a)r\frac{N}{K}\right)X, \\
\frac{dY(t)}{dt} &= \frac{(c\beta_1 Y + c\beta_2 Z)X}{N} - \left(\gamma + d + (1-a)r\frac{N}{K}\right)Y, \\
\frac{dZ(t)}{dt} &= \gamma Y - \left(\mu + d + (1-a)r\frac{N}{K}\right)Z.
\end{aligned} \tag{1}$$

Summing the three equations in system (1), we obtain

$$\frac{dN}{dt} = r(1 - \frac{N}{K})N - \mu Z. \tag{2}$$

In the absence of $AIDS$ class the second term of the equation (2) disappears, exhibiting the logistic growth phenomenon for the population which in actual fact concurs with [2].

Following [2], consider the space of XYZ to be positively invariant subset of $X + Y + Z \leq K$ in the first octant where the space is given by

$$D = \{X, Y, Z \in \Re^3_+ : X \geq 0, Y \geq 0, Z \geq 0, 0 \leq N \leq K\}. \tag{3}$$

The continuity of the system (1) and equation (2) and its derivatives implies that unique solutions occur on maximal time intervals. Taking $r > 0$ solution paths move towards, enter or remain in D, these paths are bounded and continuous so they exist for every positive time, see Hale [3]. Therefore, the initial value problem for the system (1) and equation (2) is mathematically well-posed and biologically-feasible defined region.

Individuals enter the sexual active population at the rate bN and we assume that the susceptible population grows due to natural births, where b is the natural birth rate constant. Since the population can not just grow exponentially without any constrains, therefore we have the carrying capacity of the environment denoted by K. The convex combination constant is denoted by a and its main purpose is to reduce the rate. The net growth rate will also be denoted by r and the total population is denoted by N. Thus, the rate of entry of new susceptibles into the sexually active population is $(b - \frac{arN}{K})N$. The progression rate of individuals infected with HIV to AIDS class is denoted by γ. Individuals leave the sexual active population at per capita rate $d + (1-a)\frac{rN}{K}$, where d is the natural death rate and $(1-a)$ is the fraction allocated to increase in the death rate. The fraction a of the density-dependence is allocated to reduce the birth rate in the host population. Individuals acquire new sexual partners at a rate c, and the fraction of the sexual active population who are infected is

given by $c\beta_1\frac{Y}{N} + c\beta_2\frac{Z}{N}$ and the incidence of the force of infection with HIV is then the product of the rate of infection and the susceptible population, given by $(c\beta_1\frac{Y}{N} + c\beta_2\frac{Z}{N})X$. β_1 is the probability of individuals transmitting infection from chronic infective partners to susceptible partners. β_2 is the probability of transmitting infection from the AIDS partners to the susceptible partners. The AIDS patients are assumed to leave the class due to death induced rate which is denoted by μ. The model parameters are as follows:

Parameters	definitions
a	convex combination constant,
b	natural birth rate constant,
d	natural death rate constant,
r	$b - d$ = growth rate constant,
K	carrying capacity of the environment,
c	daily contact rate,
$\beta 1$	the probability of transmitting infection from the chronic infective partner to the susceptible partner,
β_2	the probability of transmitting infection from the AIDS infective partner to the susceptible partner,
γ	progression rate to AIDS and
μ	disease-related death rate constant.

3. Analysis of Equilibria and Reproduction Number

3.1. Steady-State Solutions

The equilibrium of this model is obtained by setting the right hand side of the system (1) to zero. It follows that system (1) has a disease-free equilibrium point $E_0 = (K, 0, 0)$ and $E_0 = (0, 0, 0)$, where the last disease-free equilibrium point is a trivial solution. The endemic equilibrium point was analyzed numerically because it raises some challenges in mathematical calculations. Therefore, qualitative analysis was restricted on the disease-free equilibrium point

$E_0 = (K, 0, 0)$.

3.2. Reproduction Number

Following Van de Driessche and Watmough's technique (see [15]) and notations, we have

$$F = \begin{pmatrix} c\beta_1 & c\beta_2 \\ 0 & 0 \end{pmatrix},$$

$$V = \begin{pmatrix} \gamma + d + (1-a)r & 0 \\ -\gamma & \mu + d + (1-a)r \end{pmatrix}.$$

The dominant eigenvalue of FV^{-1} is given by

$$\frac{c\beta_1}{\gamma + d + (1-a)r} + \frac{c\beta_2\gamma}{(\gamma + d + (1-a)r)(\mu + d + (1-a)r)}.$$

Thus, the reproduction number for the model system (1) is the spectral radius of $FV^{-1} = \rho(FV^{-1})$, where

$$\rho(FV^{-1}) = \Re_0^d$$

with

$$\Re_0^d = \frac{d + (1-a)r}{\gamma + d + (1-a)r} R_{01}^d + \frac{\gamma}{(\gamma + d + (1-a)r} R_{02}^d,$$

where

$$R_{01}^d = \frac{c\beta_1}{d + (1-a)r}, \quad R_{02}^d = \frac{c\beta_2}{\mu + d + (1-a)r}.$$

The superscript d simply means that demographics are taken into account.

Remarks. We note that as $a \to 1$, the results of R_{01}^d and R_{02}^d collapse to the results of the model studied in [8], that is, $R_{01} = \dfrac{c\beta_1}{d}$, and $R_{02} = \dfrac{c\beta_2}{\mu + d}$. Note that in the case $a = 1$, the dynamical system will be more influenced by the death rates. The case $a = 0$ reduces to a system where the births have much influence on the the dynamics so that,

$$R_{01}^d = \frac{c\beta_1}{b}, \text{ and } R_{02}^d = \frac{c\beta_2}{\mu + b}.$$

In the case $0 < a < 1$, we have $R_{01} > R_{01}^d$ and $R_{02}^d > R_{02}^d$, showing that the chronic and AIDS infectives in [8] generate more secondary cases in the absence

of density-dependent demographics. We can prove this as follows:

$$R_{01} - R_{01}^d = \frac{r(1-a)c\beta_1}{d[d+(1-a)r]} > 0,$$

since $0 < a < 1$, implying that $1 - a > 0$, therefore, we have $R_{01} > R_{01}^d$. Similarly, we prove that $R_0 > \Re_0^d$.

Proof.

$$R_0 - \Re_0^d = \frac{c\beta_1(1-a)r}{(\gamma+d)(\gamma+d+(1-a)r)} + \frac{r(1-a)[\gamma+2d+\mu+(1-a)r]c\gamma\beta_2}{(\gamma+d)(\mu+d)(\gamma+d+(1-a)r)(\mu+d+(1-a)r)} > 0,$$

since $0 < a < 1$. Hence, we have

$$R_0 > \Re_0^d.$$

In a similar manner we can easily prove that $R_{02}^d > R_{02}^d$. This result shows that omission of population demographics has the effect of underestimating the generation of secondary infections and hence the magnitude of the epidemic. Setting $\gamma = 0$ implies that there are no chronic infectives progressing to AIDS and this allows us to see the significance of infectives in the chronic stage X. On the other hand setting $\gamma \to \infty$ implies that all infectives develop AIDS and this measures the role of AIDS individuals in the spread of disease. Knowing estimates of R_{01}^d, and R_{02}^d gives an indication of the contribution from each group, and provides guidelines as to which group to target for intervention. Clearly, any intervention program leading to $R_{01}^d = R_{02}^d = 1$, subsequently leads to $\Re_0^d$, which clears the disease from the host population.

4. Stability Analysis

4.1. Analysis of Disease-Free Equilibrium

The general Jacobian matrix evaluated at disease-free equilibrium point $(D.F.E)$ $E_0^d = (K, 0, 0)$ is given by

$$J_0 = \begin{pmatrix} -r & d-ar & d-ar \\ 0 & ar-b & 0 \\ 0 & 0 & -d-\mu \end{pmatrix}.$$

From the above Jacobian matrix J_0, there are three eigenvalues, that is,

$$\lambda_1 = -r, \;\; \lambda_2 = -(d+\mu) \;\text{ and }\; \lambda_3 = -(b-ar).$$

Since $0 < a < 1$ and $r = b - d$ it follows that

$$b - ar = b(1-a) + ad > 0,$$

clearly, showing that $\lambda_3 < 0$ and hence all the roots are negative. Thus the disease-free equilibrium point is locally asymptotically stable. Basically, this implies that the infection vanishes out in the host population if the initial conditions are sufficiently close to the $(D.F.E)$ point. We shall summarize this result in the following theorem.

Theorem 1. *The disease-free equilibrium point E_0^d of system (1) is asymptotically stable for $\Re_0^d < 1$ and unstable if $\Re_0^d > 1$.*

Biologically speaking, this implies that if the average number of secondary infectives generated by a single infective in this host population is less than unity, then the disease will clears out or will be reduced to a controllable state in the host population but otherwise it grows.

Theorem 2. *The disease-free equilibrium point E_0^d of system (1) is globally asymptotically stable for $\Re_0^d \leq 1$.*

Proof. The global stability of the disease-free equilibrium point E_0^d shall be proved by $V = Y + Z$ as our Lyapunov function. Apparently, $V = 0$ is at the $D.F.E$ point only. Therefore, we have,

$$\frac{dV}{dt} = P_1 \left[B_0 \left(\frac{c\beta_1}{B_0} - 1 \right) Y^* + C_0 \left(\frac{c\beta_2}{C_0} - 1 \right) Z^* \right]$$

By setting $N^* = X^* = K$ at the $D.F.E$, then we have

$$\frac{dV}{dt} = P_1 [B_1 (R_{01}^d - 1) Y^* + C_1 (R_{02}^d - 1) Z^*] < 0,$$

where

$$P_1 = \frac{X^*}{N^*}, \;\; B_0 = d + (1-a) r \frac{N^*}{K}, \;\; C_0 = \mu + d + (1-a) r \frac{N^*}{K},$$

and

$$B_1 = d + (1-a) r, \;\; C_1 = \mu + d + (1-a) r.$$

Since R_{01}^d and R_{01}^d are both less than $\Re_0^d$ which is less than unity at the $D.F.E$ therefore, it follows that there are also less than unity, which implies that $\frac{dV}{dt} < 0$ since $R_{01}^d < 1$ and $R_{02}^d < 1$. Therefore, we shall conclude that E_0^d is globally asymptotically stable whenever $\Re_0^d \leq 1$. From the biological point of view, it implies that HIV epidemic is wiped out or is reduced to a manageable state irrespective of the initial conditions if the average number of infectives generated by a single infective is less than unity one when put in a completely susceptible population.

5. Modification of SIR Model to Include Vertical Transmission

Generally, diseases can be transmitted in different ways, and we normally classify transmission as either horizontal or vertical. In the case of HIV/AIDS, horizontal transmission can result from direct physical contact between an infected individual and a susceptible individual, see Kgosimore and Lungu [7]. Vertical transmission is a term used to describe the direct transfer of a disease from infected mother to an unborn or newly born baby and that is, before, during or just after birth. It is commonly referred to as mother-to-child transmission, Mugisha and Luboobi [11]. In Section 1 we focused on the interactive effect of horizontal HIV/AIDS transmission with density-dependent demographics and its analysis. We now focus on the interactive effect of horizontal transmission, vertical transmission and density-dependent demographics and its analysis.

According to the UNAIDS/WHO [14], at the end of 2004 an estimated 39.4 million people globally were living with HIV/AIDS. From this numerical figure, 17.6 million were children under the age of 15 years. The estimated numerical number of death due to the disease was at 2.6 million for adults and 0.51 million for children, while the new infections were estimated at 4.94 million, consisting of 4.3 million adults between $15 - 49$ years and 0.64 million children under 15 years. Sub-Saharan Africa was the hardest hit region in the world, with 25.4 million adults and children living with HIV/AIDS. According to Kgosimore and Lungu [7], there were 3.1 million new infections of adults and children, 2.3 million deaths, and the infection rate estimated at 7.4%. These statistics, particularly on the number of children affected by HIV/AIDS confirm the importance of incorporating vertical transmission in the study of HIV/AIDS models.

6. Model Formulation

The total population size $N(t)$ is divided into three distinct epidemiological sub-classes which are susceptible, infectious and AIDS patients, with sizes denoted by $X(t), Y(t)$ and $Z(t)$, respectively. The total population is given by $N(t) = X(t) + Y(t) + Z(t)$. Let α be the fraction of newborns who are not infected through vertical transmission so that $1-\alpha$ is the fraction of those who are infected by vertical transmission. The infected children who survive the maturation age $\tau = 15$ years progress to the chronic infective class. The probability of surviving the juvenile stage $0-15$ years is denoted by $e^{-\mu_0 \tau}$, where μ_0 is the natural death rate of the juveniles. It is assumed that the offsprings progress to full-blown AIDS and never live long to become adult infectives, Kgosimore and Lungu [7]. Here, d is the intrinsic natural per capita death rate, $r = b - d$ is the positive intrinsic per capita net growth rate, a is the convex combination constant, with $0 < a < 1$ and K is the environment carrying capacity.

Figure 2 is the transfer diagram for this vertical transmission model.

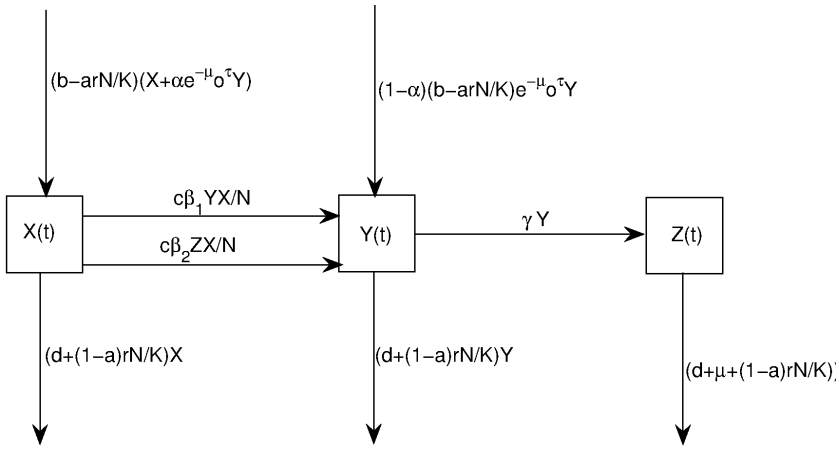

Figure 2: Model Diagram

The autonomous differential equations corresponding to the transfer dia-

gram, Figure 2, are as follows:

$$\frac{dX}{dt} = \left(b - ar\frac{N}{K}\right)(X + \alpha e^{-\mu_0\tau}Y) - \frac{(c\beta_1 Y + c\beta_2 Z)X}{N} \tag{4}$$
$$- \left(d + (1-a)r\frac{N}{K}\right)X,$$
$$\frac{dY}{dt} = \frac{(c\beta_1 Y + c\beta_2 Z)X}{N} - \gamma Y - \left(d + (1-a)r\frac{N}{K}\right)Y \tag{5}$$
$$+ (1-\alpha)\left(b - ar\frac{N}{K}\right)e^{-\mu_0\tau}Y,$$
$$\frac{dZ}{dt} = \gamma Y - \left(d + \mu + (1-a)r\frac{N}{K}\right)Z. \tag{6}$$

Summing up equations (4)-(6), we have

$$\frac{dN}{dt} = r\left(1 - \frac{N}{K}\right)N - \mu Z. \tag{7}$$

The system is satisfied in the positively invariant octant in

$$D = \{(X, Y, Z) | Z \geq 0, \quad Y \geq 0, \quad 0 \leq X + Y + Z \leq K, \quad 0 \leq X \leq K\}.$$

System (4) is well-posed in a similar manner like in system (1).

7. Equilibria and the Reproduction Number

7.1. Steady-State Solutions

The equilibria of this model are obtained by setting the right of the system (4)-(6) to zero. It follows that system (4)-(6) has a DFE point $E_0^v = (K, 0, 0)$ and $E_0^v = (0, 0, 0)$. The endemic equilibrium point is difficult to find and we therefore restrict our analysis to the DFE point $E_0^d = (K, 0, 0)$.

7.2. Reproduction Number

For the computation of the reproduction number of the model, we employ the technique introduced by Van de Driessche and Watmough [15], whose main principle is to differentiate between new infections and other states. Using the notation in Van de Driessche and Watmough [15], we have

$$\mathcal{F} = \begin{pmatrix} \frac{c\beta_1 X^* Y^*}{N^*} + \frac{c\beta_2 Z^* X^*}{N^*} \\ 0 \\ 0 \end{pmatrix},$$

$$\mathcal{V} = \begin{pmatrix} (\gamma + d)Y^* + \frac{(1-a)r(N^*)Y^*}{K} - (1-\alpha)(b - ar\frac{N^*}{K})e^{-\mu_0\tau} \\ (d+\mu)Y^* + \frac{Z^*}{(}1 - a)r(N^*)K - \gamma Y^* \\ -(b - ar\frac{N^*}{K})(X^* + \alpha e^{-\mu_0\tau}Y^*) + \left(d + (1-a)r\frac{N^*}{K}\right)X^*. \end{pmatrix}.$$

In this model the infected classes are Y and Z, giving $m = 2$. The dominant eigenvalue of FV^{-1} is given by

$$\Re_0^v = \frac{(b-ar)(1-(1-\alpha))e^{-\mu_0\tau}}{\gamma + (b-ar)(1-(1-\alpha))e^{-\mu_0\tau}} R_{01}^v + \frac{\gamma}{\gamma + (b-ar)(1-(1-\alpha))e^{-\mu_0\tau}} R_{02}^v, \quad (8)$$

where v symbolizes vertical transmission,

$$R_{01}^v = \frac{c\beta_1}{(b-ar)(1-(1-\alpha))e^{-\mu_0\tau}}, \quad R_{02}^v = \frac{c\beta_2}{(\mu + d + (1-a)r)}.$$

Thus, the reproduction number for model system (1) is the spectral radius of $FV^{-1} = \rho(FV^{-1})$, where

$$\rho(FV^{-1}) = \Re_0^v.$$

Note that setting $\alpha = 1$ and $a = 1$ yields

$$R_0^v = R_0 = \frac{c\beta_1}{(\gamma + d)} + \frac{c\beta_2\gamma}{(\gamma + d)(\mu + d)}$$

which concurs with R_0 found in [8]. Where $\alpha = 1$, means there are no infectives through vertical, $\alpha = 0$ means the fraction of new born babies who are infected through vertical transmission, and $\tau = 0$ means that there are no survival of infected children. Moreover, note that in equation (8), if α increases that is, if the proportion of children getting infected through vertical transmission increases, then R_0^v also increases.

Remarks. In the case $0 < a < 1$, we have $R_{01}^d > R_{01}^v$. The proof is as follows,

$$R_{01}^d - R_{01}^v = \frac{1}{(d + (1-a)r)(1-\zeta)} > 0,$$

where $\zeta = \frac{1}{(1-\alpha)e^{-\mu_0\tau}}$, since $0 < \alpha < 1$ and $0 < a < 1$, hence $R_{01}^d > R_{01}^v$.

Similarly, it can be shown that

$$R_0^d > R_0^v.$$

This result is important in the dynamics of HIV/AIDS. It shows that the inclusion of population demographics and vertical transmission affects the generation of secondary infections and hence the magnitude of the epidemic will not be underestimated or overestimated.

8. Stability of Disease-Free Equilibrium

8.1. Analysis of Disease-Free

The Jacobian matrix evaluated at disease-free equilibrium point E_0^v is given by

$$J_0 = \begin{pmatrix} -r & -r + (b-ar)\alpha e^{-\mu_0\tau} & -r \\ 0 & -[(b-ar)(1-(\alpha-1))e^{-\mu_0\tau}] & 0 \\ 0 & 0 & -(d+\mu) - (1-a)r \end{pmatrix}.$$

The corresponding eigenvalues of the Jacobian matrix are

$$\begin{aligned} \lambda_1 &= -r \\ \lambda_2 &= -[(b-ar)(1-(\alpha-1))e^{-\mu_0\tau}] \\ \lambda_3 &= -[(d+\mu) + (1-a)r]. \end{aligned}$$

Since $0 \leq a \leq 1, \quad 0 \leq \alpha \leq 1$ and $r = b - d$, it follows that

$$(b-ar)(1-(\alpha-1))e^{-\mu_0\tau} > 0 \quad \text{and} \quad (d+\mu) + (1-a)r > 0,$$

clearly showing that all roots are negative. Thus, the disease free equilibrium point is locally asymptotically stable. Biologically speaking, the infection clears out in the host population provided the initial conditions are close to the $D.F.E$ point. The result of the stability analysis can be summarized as follows.

Theorem 3. *The disease-free equilibrium point E_0^v of system (4) is asymptotically stable for $\Re_0^v < 1$ and unstable if $\Re_0^v > 1$.*

Biologically speaking, this implies that if the average number of secondary infectives generated by a single infective in this host population is less than unity then the disease will clears out or will be reduced to a controllable state in the host population but otherwise it grows.

Theorem 4. *The disease-free equilibrium point E_0^v of system (4) is globally asymptotically stable for $\Re_0^v \leq 1$.*

Proof. The global stability of the disease-free equilibrium point E_0^v can be proved by taking $V = Y + Z$ as our Lyapunov function. Clearly, $V = 0$ is at the $D.F.E$ point only. Therefore, we have

$$\frac{dV}{dt} = P_1 \left[B_2 \left(\frac{c\beta_1}{B_2} - 1 \right) Y^* + C_2 \left(\frac{c\beta_2}{C_2} - 1 \right) Z^* \right].$$

Let $N^* = X^* = K$ at the $D.F.E$ so that

$$\frac{dV}{dt} = P_1[B_3(R_{01}^v - 1)Y^* + C_3(R_{02}^v - 1)Z^*] < 0,$$

where P_1 as defined before,

$$B_2 = d + (1-a)r\frac{N^*}{K}, \;\; C_2 = \mu + d + (1-a)r\frac{N^*}{K},$$

and

$$B_3 = (b - ar)(1 - (\alpha - 1))e^{-\mu_0 \tau}, \;\; C_3 = \mu + d + (1-a)r.$$

Similarly, it follows that R_{01}^v and R_{02}^v are both less than unity by the fact that there are both less than $\Re_0^v$ which is less unity at the disease-free equilibrium point. Therefore, $\dfrac{dV}{dt} < 0$, hence we shall conclude that E_0^v is globally asymptotically stable whenever $\Re_0^v \leq 1$.

9. Numerical Simulations

We now carry out numerical simulations of the system (1) and system (4)-(6), respectively. The purpose of this section is to numerically illustrate some of the analytical results which we derived. We shall use the following parameter

values in the table below:

Parameters	Parameters values	citation
c	3	Estimated
b	0.09	Estimated
a	[0,1]	[2]
β_1	0.125	Estimated
β_2	0.03	Estimated
d	0.02	[9]
μ	0.05	Estimated
γ	0.01149	[6]
K	2000000	Estimated
e	2.7182183	

(9)

We shall estimate parameter α by taking it from the interval $[0, 1]$, the natural death rate of the juveniles $\mu_0 = 0.02$ and the survival maturation age τ, all of the new parameters are from Kgosimore and Lungu [7].

From Figure 3 it is clear that as we move from death to birth density-dependent demographics, the figures shows an increase in secondary infectives. For instance, part A in Figure 3 has death density dependence which implies that there is sustenance of susceptible population, but depletion of the chronic and AIDS infectives. In part D on the same figure we have birth density dependence implying that there is a depletion of susceptible population, and the endemic increases in infectives. Figure 3 shows that the total population size in a logistic demographic model approaches the carrying capacity K, but the persistence of the disease will lower the host population size. Further, it shows that the population settle to endemic values showing that the disease will remain in the host population for a long time. This is particularly a worry for the case of HIV/AIDS since we do not have a particular treatment at this present moment, therefore, we would be happier that it settles at a very low endemic levels or at the disease-free equilibrium state. Hence, there is a need for the interventions in order to eradicate the epidemic persistence in the host population.

It is apparent that in Figure 4 as we increase the value of α, the susceptible population also increases. The reason is when the value of α is low it signifies

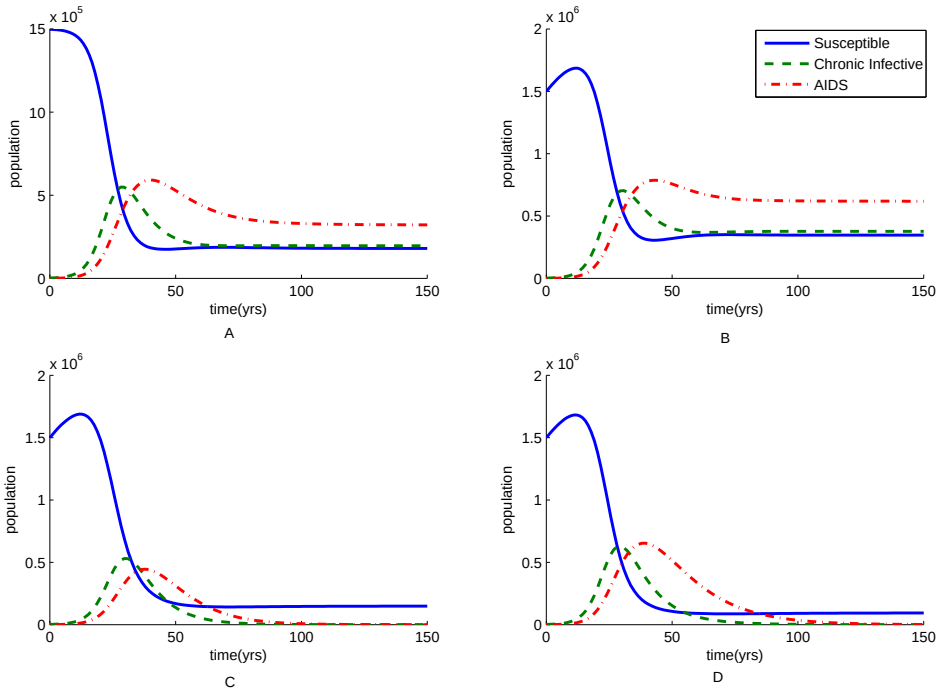

Figure 3: Sensitivity of density-dependent demographics to the convex combination parameter, $0 \leq a \leq 1$. Other parameters are fixed at:$c = 3$, $\beta_1 = 0.125$, $\beta_2 = 0.03$ $\mu = 0.05$, $d = 0.02$, $b = 0.09$, $\gamma = 0.1149$, $r = b - d, K = 2000000$, and $r = b - d$; and $c = 3$. The reproduction number are as follows $\Re_0^d = 2.1907$, $\Re_0^d = 2.7868$, and $\Re_0^d = 3.8749$ respectively.

that there is high mortality if children hence the depletion of the susceptible population. Figure 4 further shows that as we increase the values of α the reproduction number also increases implying that the epidemic persist in the host population due to more recruits in the host population. With the introduction of vertical transmission two things can happen, that is, the disease clears out at a delayed time or the epidemic re-establishes itself. Moreover, the increase in the rate of infection causes the susceptible population to decrease rapidly in both figures A, B, C and D. Since the infected population will continue to fall the non infected which is the susceptible population will eventually grow. As for the vertical transmission model in part C, the susceptible population remains above the chronic infectives and the AIDS infectives. This is due to

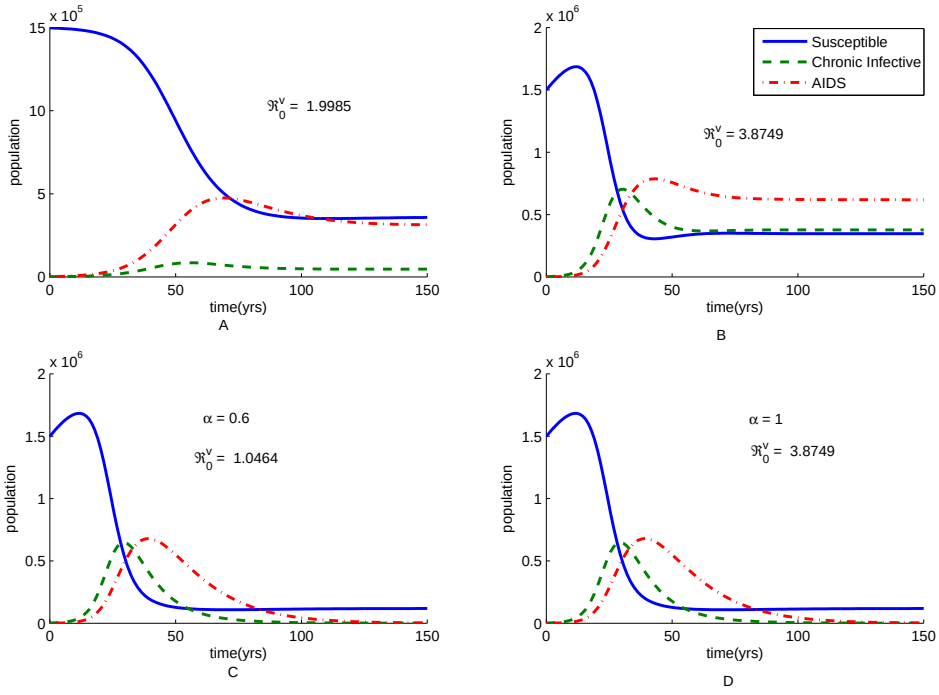

Figure 4: The figure shows the effect of adding vertical transmission to the previous models. Density-dependent demographics A and B: Vertical transmission model C and D: $\mu = 0.05,\ d = 0.02,\ \beta_1 = 0.125,\ \beta_2 = 0.03\ \gamma = 0.1149,\ \alpha = 1,\ K = 2000000,\ \tau = 15,\ \mu_0 = 0.02,\quad e = 2.7182183; r = b - d;\ 0 \leq a \leq 1;$ and $c = 3$.

the fact that there are survivals of infected children and the birth rate is density independent whereas death rate is density dependent.

10. Discussion

One of the greatest challenges that needs to be confronted now and the years to come is to uphold common goals of good public health, scientific and policy urgency concerning an HIV/AIDS epidemic that is already established and common but continues to take too many lives unnecessarily, see Nyabadza [13]. Regardless of the economic challenges in Southern Africa, the fight against

HIV/AIDS has become a central pivot, since it is life threatening. The most important tool used in combating HIV/AIDS pandemic is to educate the nation at large and to provide prevention programmes like voluntary counseling and testing (VCT), condom use and health education that targets the high risk groups.

In this paper both the birth rate and death rate depend on the number of individuals in the population. The rate of change of the total population N is governed by the logistic equation.

From the model diagram, Figure 1, the rate of entry into the sexually active population,

$$\left(b - ar\frac{N}{K}\right)N,$$

has a density-dependent per capita birth rate coefficient $b - ar\dfrac{N}{K}$. The rate at which individuals leave each of the compartments, has a density-dependent per capita death rate coefficient $d + (1-a)r\dfrac{N}{K}$. For $0 < a < 1$ the birth rate decreases and the death rate increases to its carrying capacity K; these are consistent with the limited resources associated with density-dependence. The birth rate is density independent when $a = 0$ and the death rate is density independent when $a = 1$, Gao and Hethcote [2] and Yoshida and Hara [16]. In all the models we observed that if the basic reproduction number is less or equal to unity, the disease dies out but if it is greater than unity, the disease persists in the host population. This result is significant in the sense that it shows that the HIV epidemic can be controlled by targeting these parameters, like the number of sexual partners, with the aim of lowering the average numbers of new infections from a single infective to less than unity. In [8], we observed that the disease free equilibrium point and the endemic equilibrium point coincide when the reproduction number is equal to unity, and there is an exchange of stability at that point. We can regard this as a bifurcation point. Furthermore, some interesting results can be obtained when we compare $\Re_0^d$, which is the effective reproduction number for the model with density dependent demographics and $\Re_0^v$ which is the effective reproduction number for the model with density dependent demographics and vertical transmission. We note that it may be better to study models which include density-dependent demographics and vertical transmission because these give a better estimate of the epidemic. Intervention efforts must pay particular attention to the control of vertical transmission since this impacts strongly on the level of the epidemic. This can be done by the implementing prevention of mother to child transmission programmes.

The models developed in this paper have their own shortcomings, as is the

case with many mathematical models. In reality no model is all inclusive, so are the ones we developed. In this study we have assumed that as soon as an individual progresses to AIDS, that individual remains sexually active. In reality the introduction of ARVs has made this perception true. One can remain sexually active because ARVs reduce the viral load and infectivity, and in the process, restoring normality in an individual. It would therefore be necessary to include treatment and behavioral change parameters in the models. Many authors have however included treatment in HIV/ADIS models, Kgosimore and Lungu, Nyabadza and Hsieh and Sheu [5, 7, 13] respectively, to mention a few. This work presents the basic models of HIV/AIDS in the presence of density-dependence demographics and vertical transmission. They can be used as a stepping stone for further studies. The model can be extended by finding the endemic equilibrium points.

Finally, we note that Gao and Hethcote [2] established that under certain threshold conditions which depend on the convex combination parameter, the disease can either clear out or persist. We have also found a similar threshold conditions which concur with them.

Acknowledgement

We thank Dr. M. Kgosimore for his valuable comments on the writing of this manuscript. We would also like to thank the referees very much for their comments and suggestions.

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