ISSN 1 746-7233, England, UK
World Journal of Modelling and Simulation
Vol. 7 (2011) No. 4, pp. 243-257

Socio-economic status and HIV/AIDS dynamics: a modeling approach*

S. Mushayabasa[1†], C. P. Bhunu[2], E. J. Schwartz[3], G. Magombedze[4], J. M. Tchuenche[5]

[1] Department of Applied Mathematics, Modelling Biomedical Systems Research Group, P. O. Box 939 Ascot, Bulawayo, Zimbabwe
[2] National University of Science and Technology, Bulawayo P. O. Box 939 Ascot, Zimbabwe
[3] Department of Mathematics and School of Biological Sciences, Washington State University, PO Box 641227, Pullman, WA, USA
[4] Department of Mathematics and Statistics, University of Guelph, Guelph ON N1G 2W1, Canada
[5] Institute of Infectious Diseases and Molecular medicine, Computational Biology Group, Bioinformatics, University of Cape Town, South Africa

(*Received May 14 2010, Accepted March 1 2011*)

Abstract. HIV/AIDS is one of the most challenging public health burdens worldwide, with more than 34 million people living with the disease at the end of 2010. The epidemic continues to erase decades of health, economic and social progress, reducing life expectancy and deepening poverty in the hardest hit communities. We formulate a deterministic model to investigate the effects of socioeconomic status on the transmission dynamics of HIV/AIDS. The qualitative features of its equilibria are analyzed and conditions under which they are stable are provided. Sensitivity analysis of the reproductive number is carried out to determine the relative importance of model parameters to initial disease transmission. Results suggest that if individuals referred to as poor in this study indulge in risky sexual behavior for any reason whatsoever, then HIV/AIDS will persist in the community as long as the transients/infectives abound.

Keywords: HIV/AIDS model, poverty, reproductive number, risky sexual behavior, stability

1 Introduction

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 region also accounted for 72% of the world AIDS-related deaths in 2008. The epidemic continues to have an enormous impact on households, communities, businesses, public services and national economies in the region[38]. Poverty is associated with weak endowments of human and financial resources, such as low levels of education with associated low levels of literacy and few marketable skills, generally poor health status and low labour productivity as a result[13]. Poor households typically have few if any financial or other assets and are often socially marginalised. These conditions of social exclusion increase the problems of reaching these populations through programmes aimed at changing sexual and other behaviors. Hence, it is not at surprising in these circumstances that the poor adopt behaviors which expose them to HIV infection[13, 16], particularly among women who may engage in transactional sex to procure food for themselves and their children[18]. Moreover, poor people are more likely to be food insecure and malnourished. Malnutrition is known to weaken the immune system, which in turn may lead to a greater risk of HIV transmission in any unprotected sexual encounter[5, 7, 18, 27, 30, 32].

A significant number of statistical studies have measured the association between socioeconomic status and HIV transmission[2, 7, 9, 13, 18, 22, 23, 31, 33, 41]. However, these studies have mixed results: only a few have

* The authors are grateful to the anonymous referee and the handling editor for their valuable comments and suggestions.
† Corresponding author. *E-mail address*: steadymushaya@gmail.com, smushayabasa@nust.ac.zw.

found a negative or no association, whereas most have found a positive association[2, 7, 13, 22, 23, 28, 31]. The aim is to gain insights into the association between socioeconomic status and HIV infection in a population sub-divided into two categories, namely the poor and the rich. A recent study considered such dynamics with additional transmission from clinical settings[34], and this study therefore complements it. The poor class constitutes individuals with low levels of financial resources, maybe due to low levels of education with usually low incomes or low wages as a result of the economics associated with the area where these individuals live. For instance, the respondents of an ethnographic study in the southern province of Zambia[10] identified frequent droughts and limited wage labor opportunities, after the post-economic liberalization closure of companies, as the factors behind the increasing resort of women to transactional sex. Due to low financial resources these individuals (in the poor class) are associated with generally poor health status. Moreover, it is not simply that information, education and communication activities are unlikely to reach the poor (which may be the case in some instances) but that such messages are often irrelevant and inoperable to a fraction of these individuals given the reality of their lives. For example, in a qualitative study in Malawi[8] certain social groups were found to continue to engage in high-risk behaviors despite knowing the risks. They did so, the authors contend, to affirm their social identity and to deny that anything they do makes a difference to what they perceive as a life of powerlessness and despair? Thus, in this study we shall consider the poor to access antiretroviral therapy through government or non-governmental organization assistance. The rich class constitutes individuals with average or better financial resources, generally associated with better health than the poor and can afford the cost of antiretroviral therapy. There is ample evidence that wealthier populations do better on most measures of health status, including nutrition, morbidity and mortality, and of healthcare utilization[1, 26, 29]. Recent studies have shown that safer sexual practices are now considered important in all socio-economic classes, although a high proportion of these individuals are from the rich class[14, 17, 19, 21, 22, 24, 37–39].

Drawing insight from the background described above, a mathematical model to investigate the association between socioeconomic status and HIV transmission is formulated and analyzed. Particularly, we wish to quantify the role of risky sexual behavior, malnutrition and antiretroviral therapy. The paper is structured as follows: Section 2 is the model framework. Stability analysis of the equilibria is carried out in Section 3. Section 4 contains the numerical illustrations and the last Section concludes the paper.

2 Model formulation

The total population is sub-divided into eight classes according to disease status, namely: rich susceptibles S_r; rich HIV infectives not on antiretroviral therapy I_r; rich HIV infectives who are on antiretroviral therapy I_{r_1}; AIDS cases in rich class A_r; poor susceptibles S_p; poor HIV infectives not on antiretroviral therapy I_p; poor HIV infectives receiving antiretroviral therapy I_{p_1}; AIDS cases in poor class A_p. Thus, the total population denoted by N is given by $N = S_r + S_p + I_r + I_{r_1} + I_p + I_{p_1} + A_r + A_p$. Let Λ denote a constant recruitment rate into the susceptible population through birth. Quantifying the proportion of children born from each class would require an extensive sensitivity analysis with parameter values estimated from real demographic data. However, studies focusing on family planning programmes have shown that couples from poor families have the highest fertility, the lowest contraceptive use and the highest unmet need for contraception[20, 36]. Thus, it is assumed that a proportion π_0 $(0 < \pi_0 < 1)$ of these children is from the rich class while the complementary proportion $(1 - \pi_0) = \pi_1$ is from the poor class, with $\pi_0 < \pi_1$. The susceptible sub-populations can be infected following effective contacts with any of the four infected classes $(I_r, I_{r_1}, I_p, I_{p_1})$, since the model assumes that individuals in the AIDS class are no longer sexual active, because of their ill health. Due to risky sexual behavior, individuals in S_p have a higher chance, relative to S_r, of acquiring new infections following contacts with the respective infected class by a factor $\sigma \geq 1$. The forces of infection denoted by λ_r and λ_p for S_r and S_p, respectively, are given by

$$\lambda_r = \frac{\beta[I_r + I_p + \alpha(\kappa_r I_{r_1} + \kappa_p I_{p_1})]}{H}, \lambda_p = \sigma\lambda_r = \frac{\sigma\beta[I_r + I_p + \alpha(\kappa_r I_{r_1} + \kappa_p I_{p_1})]}{H}. \tag{1}$$

The parameter β is the product of the effective contact rate for HIV infection and the transmission probability of HIV per contact. The relative infectiousness of individuals who are on antiretroviral therapy and are sexually

active is measured by α $(0 < \alpha < 1)$, since it has been estimated by an analysis of longitudinal cohort data that antiretroviral therapy reduces per-partnership infection by as much as 60% (so that $\alpha \approx 0.4$)[6, 40]. A fraction κ_i for *i= r,p* of infectives who are on antiretroviral therapy are still sexually active (with $\kappa_r < \kappa_p$, denoting rich and poor respectively), while the complementary proportion $(1-\kappa_i)$, for *i= r,p* have abstained from risky sexual behavior, so that the total sexually active population is given by $H = N-[(A_r+A_p)+(1-\kappa_r)I_{r_1}+(1-\kappa_p)I_{p_1}]$. Infectives are treated at a rates ϕ_r and ϕ_p for rich and poor respectively, with $\phi_p < \phi_r$, due to the fact that rich infectives can afford the antiretroviral cost, while poor infectives usually depend upon government support or non-governmental organisations. Infectives on treatment and those not on treatment progress to AIDS at rates $\omega, \eta\omega$,(for rich and poor respectively) $\gamma, \eta\gamma$ (for rich and poor respectively), respectively. It is known that malnutrition weakens the immune system, which in turn may lead to a greater risk of HIV transmission in any unprotected sexual encounter[7, 18]. Thus, the modification parameter $\eta \geq 1$, captures the relative malnutritional effect of the individuals from the poor class. The natural mortality rate μ is assumed to be constant in all classes, with individuals in the AIDS class suffering additional mortality due to the disease at rates $\nu, \eta\nu$. The model flow diagram is depicted in Fig. 1 below. From the descriptions and assumptions on

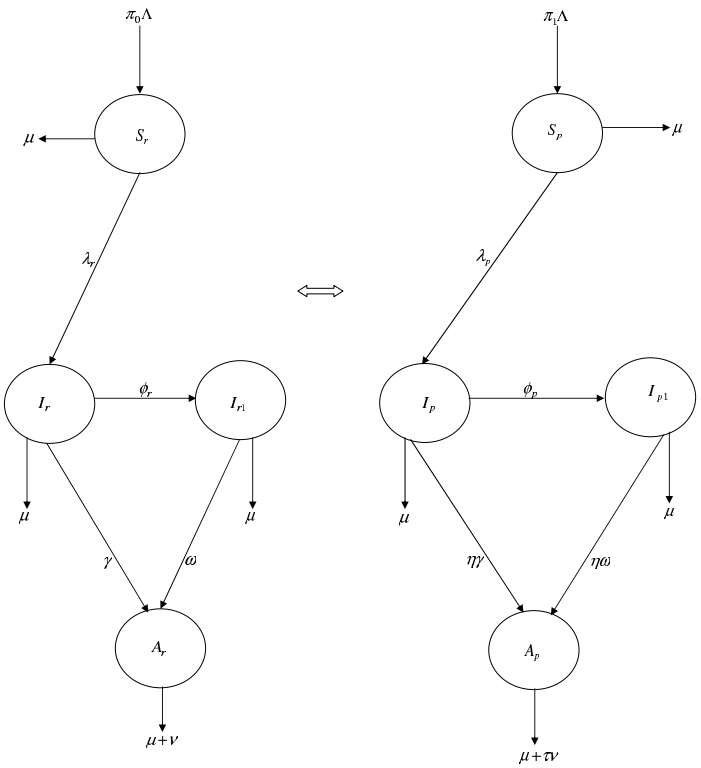

Fig. 1. Small structure of the model

the dynamics of the epidemic made above, the following are the model equations.

$$\text{Resource-rich} \begin{cases} S_r' = \pi_0\Lambda - (\lambda_r + \mu)S_r, \\ I_r' = \lambda_r S_r - (\gamma + \phi_r + \mu)I_r, \\ I_{r_1}' = \phi_r I_r - (\omega + \mu)I_{r_1}, \\ A_r' = \gamma I_r + \omega I_{r_1} - (\mu + \nu)A_r \end{cases} \tag{2}$$

$$\text{Resource-constrained (poor)} \begin{cases} S_p' = \pi_1\Lambda - (\lambda_p + \mu)S_p, \\ I_p' = \lambda_p S_p - (\eta\gamma + \phi_p + \mu)I_p, \\ I_{p_1}' = \phi_r I_p - (\eta\omega + \mu)I_{p_1}, \\ A_r' = \eta(\gamma I_p + \omega I_{p_1}) - (\mu + \eta\nu)A_p. \end{cases}$$

2.1 Model basic properties

The model Eq. (2) basic properties which are essential in the proofs of stability are provided below.

Lemma 1. *The equations preserve positivity of solutions.*

Proof. The vector field given by the right hand side of Eq. (2) points inward on the boundary of $\mathbb{R}^8_+ \setminus \{0\}$. For example, if $I_{r_1} = 0$ then $I'_{r_1} = \phi I_r \geq 0$. All the other components are similar.

Lemma 2. *Each non-negative solution is bounded in L^1-norm by* $\max\{N(0), \Lambda/\mu\}$.

Proof. The L^1 norm of each non-negative solution is N and it satisfies the inequality $N' \leq \Lambda - \mu N$. Solutions to the arbitrary equation $M' = \Lambda - \mu M$ are monotone increasing and bounded by Λ/μ if $M(0) < \Lambda/\mu$. They are monotone decreasing and bounded above if $M(0) \geq \Lambda/\mu$. Since $N' \leq M'$ the claim follows.

Corollary 1. *The region*

$$\Phi = \left\{ (S_r, S_p, I_r, I_{r_1}, I_p, I_{p_1}, A_r, A_p) \in \mathbb{R}^8_+ : N \leq \frac{\Lambda}{\mu} \right\}, \tag{3}$$

is invariant and attracting for system Eq. (2).

Theorem 1. *For every non-zero, non-negative initial value, solutions of model system Eq. (2) exist* $\forall t \geq 0$.

Proof. Local existence of solutions follows from standard arguments since the right-hand side of Eq. (2) is locally Lipschitz. Global existence follows from the (a priori) bounds.

The model has a number of invariant sets that correspond to epidemiologically limiting cases of the problem. The effect of poverty on the spread of HIV/AIDS is modeled through the risky sexual behavior and malnutrition, with corresponding parameters σ and η, and the recruitment of individuals into the two classes is determined by π_0, π_1. If we set $\sigma = 1$, $\eta = 1, \phi_r = \phi_p, \kappa_r = \kappa_p$, the we can simply add the respective compartments $S = S_r + S_p$ and so on, and we obtain a standard SIR-model. If, on the other hand, we allow only one group to enter the system, then we obtain invariant sets.

Lemma 3. *If $\pi_0 = 0$ then the set $\{S_r = I_r = I_{r_1} = A_r = 0\}$ is invariant and attracting for system Eq. (2). If $\pi_1 = 0$ then the set $\{S_p = I_p = I_{p_1} = A_p = 0\}$ is invariant and attracting.*

3 Equilibrium states, reproductive number and stability

The disease-free equilibrium (DFE) and endemic equilibrium (EE) of model system Eq. (2) are derived and their stability investigated via the reproductive number. Model system Eq. (2) has an evident DFE given by

$$\boldsymbol{E}^0 = \left(S_r^0, S_p^0, I_r^0, I_p^0, I_{r_1}^0, I_{p_1}^0, A_r^0, A_p^0\right) = \left(\frac{\pi_0\Lambda}{\mu}, \frac{\pi_1\Lambda}{\mu}, 0, 0, 0, 0, 0, 0\right).$$

The linear stability of E^0 is governed by the basic reproduction number R_0 which is defined as the spectral radius of the next generation matrix[34, 35]. The basic reproduction number can often be interpreted as the expected number of secondary infections produced by a single infectious individual during his/ her entire infectious period. However, in our model an infectious individual can be in one of the four classes $I_r, I_{r_1}, I_p, I_{p_1}$ and the expected number of secondary infections depends on the class. We consider the different possibilities in detail. Following van den Driessche and Watmough[15], we calculate the next generation matrix as

$$\boldsymbol{K} = \beta \begin{bmatrix} \frac{\pi_0}{\mu+\gamma+\phi_r}\left(1+\frac{\alpha\phi_r\kappa_r}{\mu+\omega}\right) & \frac{\pi_0\alpha\phi_r\kappa_r}{\mu+\omega} & \frac{\pi_0}{\mu+\phi_p+\eta\gamma}\left(1+\frac{\alpha\phi_p\kappa_p}{\mu+\eta\omega}\right) & \frac{\pi_0\alpha\kappa_r}{\mu+\omega} \\ 0 & 0 & 0 & 0 \\ \frac{\sigma\pi_1}{\mu+\gamma+\phi_r}\left(1+\frac{\alpha\phi_r\kappa_r}{\mu+\omega}\right) & \frac{\pi_1\alpha\phi_r\kappa_r}{\mu+\omega} & \frac{\sigma\pi_1}{\mu+\phi_p+\eta\gamma}\left(1+\frac{\alpha\phi_p\kappa_p}{\mu+\eta\omega}\right) & \frac{\pi_1\sigma\alpha\kappa_r}{\mu+\omega} \\ 0 & 0 & 0 & 0 \end{bmatrix}. \tag{4}$$

The rows and columns refer to $I_r, I_{r_1}, I_p, I_{p_1}$, in that order. Since individuals in class A_r and A_p do not contribute to new infections, we can ignore them for K. The (i, j)-entry of this matrix is the expected number of secondary infections in class i resulting from a single primary infective in class j. We consider two special cases first.

Case 1. The entire population in the community consists of rich individuals only.

We set $\pi_1 = 0$. Then the matrix K has rank one and the spectral radius is given by

$$R_r = \frac{\beta}{\mu + \gamma + \phi_r}\Big(1 + \frac{\alpha\phi_r\kappa_r}{\mu + \omega}\Big), \tag{5}$$

This reproductive number is sometimes referred to as *the back of the napkin*[3, 35]. It is defined as the number of secondary HIV cases produced by a single infected individual during his/her entire infectious period in a totally naive (susceptible) population in the presence of antiretroviral therapy. An associated epidemiological threshold, R_{0_r}, obtained using the same technique of the next generation operator[34], by considering model system Eq. (2) in the absence of HIV intervention strategies, is given by

$$R_{0_r} = \frac{\beta}{\mu + \gamma}. \tag{6}$$

The basic reproductive number R_{0_r} is simply the ratio of the per capita rate of infection and the average lifetime of an individual in class I_r.

Case 2. The entire population in the community consists of poor individuals only.

In this case, we have $\pi_0 = 0$. Again, the matrix K has rank one, and the spectral radius in the absence and presence of treatment is given by, respectively,

$$R_{0_p} = \frac{\beta\sigma}{\mu + \eta\gamma},\ R_p = \frac{\beta\sigma}{\mu + \phi_p + \eta\gamma}\Big(1 + \frac{\alpha\phi_p\kappa_p}{\mu + \eta\omega}\Big). \tag{7}$$

Case 3. The general case.

When $\pi_0, \pi_1 > 0$, the matrix K has rank two and its spectral radius in the absence and presence of treatment, respectively, can be calculated explicitly as

$$\begin{aligned} R_{0_{rp}} &= \frac{\beta[\pi_0(\mu + \eta\gamma) + \sigma\pi_1(\mu + \gamma)]}{(\mu + \gamma)(\mu + \eta\gamma)} = R_{0_r} + R_{0_p}. \\ R_{rp} &= \beta\Big[\frac{\pi_0}{\mu + \gamma + \phi_r}\Big(1 + \frac{\alpha\phi_r\kappa_r}{\mu + \omega}\Big) + \frac{\sigma\pi_1}{\mu + \phi_p + \eta\gamma}\Big(1 + \frac{\alpha\phi_p\kappa_p}{\mu + \eta\omega}\Big)\Big] = R_r + R_p. \end{aligned} \tag{8}$$

The next result follows from Theorem 2 in [34].

Theorem 2. *The disease-free equilibrium E^0 of model Eq. (2) is locally asymptotically stable if $R_{rp} < 1$, and unstable otherwise.*

Using a theorem from Castillo-Chavez et al.[12], we can even show global stability of the DFE in the case that the reproduction number is less than unity.

Theorem 3. *The DFE E^0 of our model system Eq. (2) is globally asymptotically stable provided $R_{rp} < 1$.*

Proof. Following Castillo-Chavez et al.[11], we write system Eq. (2) in the form

$$X'(t) = F(X, Y), Y'(t) = G(X, Y),\ G(X, \mathbf{0}) = 0, \tag{9}$$

where $X = (S_r, S_p)$ and $Y = (I_r, I_{r_1}, A_r, I_p, I_{p_1}, A_p)$. Here $X \in \mathbb{R}^2_+$ denotes (its components) the number of uninfected individuals and $Y \in \mathbb{R}^6_+$ denoting (its components) the number of infected individuals. The

disease-free equilibrium is now denoted by $E^0 = (X_0, \mathbf{0})$ where $X_0 = \left(\frac{\pi_0 \Lambda}{\mu}, \frac{\pi_1 \Lambda}{\mu}\right)$. We have to prove that the two conditions

$$\begin{array}{l} (H1)\ \text{For } X'(t) = F(X,0),\ X \text{ is a globally asymptotically stable,} \\ (H2)\ G(X,Y) = UY - \widehat{G}(X,Y),\ \widehat{G}(X,Y) \geq 0 \text{ for } (X,Y) \in \Phi_1, \end{array} \tag{10}$$

are satisfied where Φ_1 is a positively invariant attracting domain.

Consider

$$\boldsymbol{F}(X,0) = \begin{bmatrix} \pi_0 \Lambda - \mu S_r \\ \pi_1 \Lambda - \mu S_p \end{bmatrix},$$

$$\widehat{\boldsymbol{G}}(X,Y) = \begin{bmatrix} \widehat{G}_1(X,Y) \\ \widehat{G}_2(X,Y) \\ \widehat{G}_3(X,Y) \\ \widehat{G}_4(X,Y) \\ \widehat{G}_5(X,Y) \\ \widehat{G}_6(X,Y) \end{bmatrix} = \begin{bmatrix} \beta[I_r + I_p + \alpha(\kappa_r I_{r_1} + \kappa_p I_{p_1})]\left(\pi_0 - \frac{S_r}{H}\right) \\ 0 \\ 0 \\ \beta\sigma[I_r + I_p + \alpha(\kappa_r I_{r_1} + \kappa_p I_{p_1})]\left(\pi_1 - \frac{S_p}{H}\right) \\ 0 \\ 0 \end{bmatrix}.$$

Therefore, $\widehat{G}(X,Y) \geq 0$ whenever $\pi_0 \geq \dfrac{S_r}{H}$ and $\pi_1 \geq \dfrac{S_p}{H}$, implying that E^0 is globally asymptotically stable for $R_{rp} < 1$ in

$$\Phi_1 = \left\{(S_r, S_p) \in \mathbb{R}^2_+ : S_r \leq \frac{\pi_0 \Lambda}{\mu}, S_p \leq \frac{\pi_1 \Lambda}{\mu}\right\} \subset \Phi,$$

which is also positively invariant and attracting.

Theorem 3 has obvious public health importance since it tells us that the disease can be eradicated completely from the community in the long run, whenever $R_{rp} < 1$.

3.1 Endemic equilibria and stability analysis

Model system Eq. (2) has three possible endemic equilibria: the poverty free endemic equilibrium with a population of rich individuals only, the endemic equilibrium when the whole population is made up of poor individuals and the equilibrium where rich and poor co-exist, herein referred to as the interior equilibrium point.

3.1.1 Poverty-free endemic equilibrium

We set $\pi_1 = 0$, so that the whole population in the community consists of the rich class only. Since the subspace $\{S_p = I_p = I_{p_1} = A_p = 0\}$ is attracting, we only consider the four dimensional system of S_r, I_r, I_{r_1}, A_r, and its endemic equilibrium (E^*_r) is given by

$$E^*_r \begin{cases} S^*_r = \frac{\Lambda}{\mu + \lambda^*_r}, \\ I^*_r = \frac{\Lambda \lambda^*_r}{(\mu+\lambda^*_r)(\mu+\gamma+\phi_r)}, \\ I^*_{r_1} = \frac{\Lambda \phi_r \lambda^*_r}{(\mu+\omega)(\mu+\lambda^*_r)(\mu+\gamma+\phi_r)}, \\ A^*_r = \frac{\Lambda \phi_r \lambda^*_r}{(\mu+\nu)(\mu+\omega)(\mu+\lambda^*_r)(\mu+\gamma+\phi_r)}. \end{cases} \tag{11}$$

In order to investigate the global stability of the endemic equilibrium, we adopt the approach by Korobeinikov[25]. Assume that $R_r > 1$, then E^*_r exists for all $S_r, I_r, I_{r_1} > \epsilon$, for some $\epsilon > 0$. Let $\lambda S_r := g(S_r, I_r, I_{r_1})$ be a positive and monotonic function, and define the following continuous function in $\mathbb{R}^3_+$ (for more details, see Korobeinikov [25]). A function

$$V(S_r, I_r, I_{r_1}) = S_r - \int_\epsilon^{S_r} \frac{g(S_r^*, I_r^*, I_{r_1}^*)}{g(\tau, I_r^*, I_{r_1}^*)} \mathrm{d}\tau + I_r - \int_\epsilon^{I_r} \frac{g(S_r^*, I_r^*, I_{r_1}^*)}{g(S_r^*, \tau, I_{r_1}^*)} \mathrm{d}\tau + I_{r_1} - \int_\epsilon^{I_{r_1}} \frac{g(S_r^*, I_r^*, I_{r_1}^*)}{g(S_r^*, I_r^*, \tau)} \mathrm{d}\tau. \quad (12)$$

If $g(S_r, I_r, I_{r_1})$ is monotonic with respect to its variables, then the endemic state E_r^* is the only extremum and the global minimum of this function. Indeed since

$$\frac{\partial V}{\partial S_r} = 1 - \frac{g(S_r^*, I_r^*, I_{r_1}^*)}{g(S_r, I_r^*, I_{r_1}^*)}, \ \frac{\partial V}{\partial I_r} = 1 - \frac{g(S_r^*, I_r^*, I_{r_1}^*)}{g(S_r^*, I_r, I_{r_1}^*)}, \ \frac{\partial V}{\partial I_{r_1}} = 1 - \frac{g(S_r^*, I_r^*, I_{r_1}^*)}{g(S_r^*, I_r^*, I_{r_1})}, \quad (13)$$

grow monotonically, then the function $g(S_r, I_r, I_{r_1})$ has only one stationary point. Furthermore, since

$$\frac{\partial^2 V}{\partial S_r^2} = \frac{g(S_r^*, I_r^*, I_{r_1}^*)}{[g(S_r, I_r^*, I_{r_1}^*]^2} \cdot \frac{\partial g(S_r, I_r^*, I_{r_1}^*)}{\partial S_r}, \quad (14)$$

$$\frac{\partial^2 V}{\partial I_r^2} = \frac{g(S_r^*, I_r^*, I_{r_1}^*)}{[g(S_r^*, I_r, I_{r_1}^*)]^2} \cdot \frac{\partial g(S_r^*, I_r, I_{r_1}^*)}{\partial I_r}, \quad (15)$$

$$\frac{\partial^2 V}{\partial I_{r_1}^2} = \frac{g(S_r^*, I_r^*, I_{r_1}^*)}{[g(S_r^*, I_r^*, I_{r_1})]^2} \cdot \frac{\partial g(S_r^*, I_r^*, I_{r_1})}{\partial I_{r_1}}, \quad (16)$$

are non-negative, then the point E_r^* is a minimum. That is, $V(S_r, I_r, I_{r_1}) \geq V(S_r^*, I_r^*, I_{r_1}^*)$ and hence, V is a Lyapunov function.

The Lyapunov function Eq. (12) satisfies

$$\begin{aligned}
&\frac{\mathrm{d}V}{\mathrm{d}t} S_r' - S_r' \left(\frac{g(S_r^*, I_r^*, Ir_1{}^*)}{g(S_r, I_r^*, I_{r_1}^*)} \right) + I_r' - I_r' \left(\frac{g(S_r^*, I_r^*, I_{r_1}^*)}{g(S_r^*, I_r, I_{r_1}^*)} \right) + I_{r_1}' - I_{r_1}' \left(\frac{g(S_r^*, I_r^*, I_{r_1}^*)}{g(S_r^*, I_r^*, I_{r_1})} \right) \quad (17) \\
&= \Lambda - g(S_r, I_r, I_{r_1}) - \mu S_r - \Lambda \frac{g(S_r^*, I_r^*, I_{r_1}^*)}{g(S_r, I_r^*, I_{r_1}^*)} + g(S_r, I_r, I_{r_1}) \frac{g(S_r^*, I_r^*, I_{r_1}^*)}{g(S_r, I_r^*, I_{r_1}^*)} \\
&\quad + \mu S_r \frac{g(S_r^*, I_r^*, I_{r_1}^*)}{g(S_r, I_r^*, I_{r_1}^*)} + g(S_r, I_r, I_{r_1}) - (\mu + \gamma + \phi_r) I_r - g(S_r, I_r, I_{r_1}) \frac{g(S_r^*, I_r^*, I_{r_1}^*)}{g(S_r^*, I_r, I_{r_1}^*)} \\
&\quad + (\mu + \gamma + \phi_r) I_r \frac{g(S_r^*, I_r^*, I_{r_1}^*)}{g(S_r^*, I_r, I_{r_1}^*)} + \phi_r I_r - (\mu + \omega) I_{r_1} - \phi_r I_r \frac{g(S_r^*, I_r^*, I_{r_1}^*)}{g(S_r^*, I_r^*, I_{r_1})} + (\mu + \omega) I_{r_1} \frac{g(S_r^*, I_r^*, I_{r_1}^*)}{g(S_r^*, I_r^*, I_{r_1})}.
\end{aligned}$$

Recall that

$$\Lambda = g(S_r^*, I_r^*, I_{r_1}^*) + \mu S_r^*, \ \ (\mu + \gamma + \phi_r) I_r^* = g(S_r^*, I_r^*, I_{r_1}^*), \ \ \phi_r I_r^* = (\mu + \omega) I_{r_1}^*. \quad (18)$$

Thus,

$$\begin{aligned}
\frac{\mathrm{d}V}{\mathrm{d}t} &= \mu S_r^* \left(1 - \frac{S_r}{S_r^*}\right) \left(1 - \frac{g(S_r^*, I_r^*, I_{r_1}^*)}{g(S_r, I_r^*, I_{r_1}^*)}\right) + g(S_r^*, I_r^*, I_{r_1}^*) \left(1 - \frac{g(S_r^*, I_r^*, I_{r_1}^*)}{g(S_r, I_r^*, I_{r_1}^*)} - \frac{g(S_r, I_r, I_{r_1})}{g(S_r, I_r^*, I_{r_1}^*)}\right) \quad (19) \\
&\quad + g(S_r^*, I_r^*, I_{r_1}^*) \left(-\frac{I_r}{I_r^*} + \frac{I_r}{I_r^*} \frac{g(S_r^*, I_r^*, I_{r_1}^*)}{g(S_r^*, I_r, I_{r_1}^*)} - \frac{g(S_r, I_r, I_{r_1})}{g(S_r^*, I_r, I_{r_1}^*)}\right) + (\mu + \omega) I_{r_1}^* \left(\frac{I_r}{I_r^*} - \frac{I_{r_1}}{I_{r_1}^*}\right) \left(1 - \frac{g(S_r^*, I_r^*, I_{r_1}^*)}{g(S_r^*, I_r^*, I_{r_1}}\right) \\
&= \mu S_r^* \left(1 - \frac{S_r}{S_r^*}\right) \left(1 - \frac{g(S_r^*, I_r^*, I_{r_1})}{g(S_r, I_r^*, I_{r_1}^*)}\right) + g(S_r^*, I_r^*, I_{r_1}^*) \left(1 - \frac{g(S_r^*, I_r^*, I_{r_1}^*)}{g(S_r, I_r^*, I_{r_1}^*)}\right) \left(1 - \frac{g(S_r, I_r, I_{r_1})}{g(S_r^*, I_r, I_{r_1}^*)}\right) \\
&\quad + g(S_r^*, I_r^*, I_{r_1}^*) \left(\frac{I_r}{I_r^*} - \frac{g(S_r, I_r, I_{r_1})}{g(S_r, I_r^*, I_{r_1}^*)}\right) \left(\frac{g(S_r^*, I_r^*, I_{r_1}^*)}{g(S_r^*, I_r, I_{r_1}^*)} - 1\right) + (\mu + \omega) I_{r_1}^* \left(\frac{I_r}{I_r^*} - \frac{I_{r_1}}{I_{r_1}^*}\right) \left(1 - \frac{g(S_r^*, I_r^*, I_{r_1}^*)}{g(S_r^*, I_r^*, I_{r_1})}\right).
\end{aligned}$$

Since $E_r^* > 0$, the function $g(S_r, I_r, I_{r_1})$ is concave with respect to I_r, I_{r_1}, and

$$\frac{\partial^2 g(S_r, I_r, I_{r_1})}{\partial I_r^2} \leq 0, \cdots, \frac{\partial^2 g(S_r, I_r, I_{r_1})}{\partial I_{r_1}^2} \leq 0,$$

then $\dfrac{\mathrm{d}V}{\mathrm{d}t} \leq 0$ for all $S_r, I_r, I_{r_1} > 0$. Also, the monotonicity of $g(S_r, I_r, I_{r_1})$ with respect to S_r ensures that

$$\left(1-\frac{S_r}{S_r^*}\right)\left(1-\frac{g(S_r^*,I_r^*,I_{r_1}^*)}{g(S_r,I_r^*,I_{r_1}^*)}\right)\leq 0, \tag{20}$$

$$\left(1-\frac{g(S_r^*,I_r^*,I_{r_1}^*)}{g(S_r,I_r^*,I_{r_1}^*)}\right)\left(1-\frac{g(S_r,I_r,I_{r_1}}{g(S_r^*,I_r,I_{r_1}^*)}\right)\leq 0, \tag{21}$$

holds for all $S_r, I_r, I_{r_1} > 0$. Since $g(S_r, I_r, I_{r_1})$ is a monotonic function $g(S_r^*, I_r, I_{r_1}^*) \geq g(S_r^*, I_r^*, I_{r_1}^*)$ it implies that $I_r \geq I_r^*$ and vice-versa then

$$\left(\frac{I_r}{I_r^*}-\frac{g(S_r,I_r,I_{r_1})}{g(S_r,I_r^*,I_{r_1}^*)}\right)\left(\frac{g(S_r^*,I_r^*,I_{r_1}^*)}{g(S_r^*,I_r,I_{r_1}^*)}-1\right)\leq 0, \tag{22}$$

if

$$\begin{aligned}&\frac{g(S_r,I_r,I_{r_1})}{g(S_r,I_r^*,I_{r_1}^*)}\geq\frac{I_r}{I_r^*} \text{ when } g(S_r^*,I_r,I_{r_1}^*)\leq g(S_r^*,I_r^*,I_{r_1}^*), \quad (\forall I_r\leq I_r^*) \text{ and}\\ &\frac{g(S_r,I_r,I_{r_1})}{g(S_r,I_r^*,I_{r_1}^*)}\leq\frac{I_r}{I_r^*} \text{ when } g(S^*,I_r,I_{r_1}^*)\geq g(S_r^*,I_r^*,I_{r_1}^*) \quad (\forall I_r\geq I_r^*).\end{aligned} \tag{23}$$

Also,

$$\left(\frac{I_r}{I_r^*}-\frac{I_{r_1}}{I_{r_1}^*}\right)\left(1-\frac{g(S_r^*,I_r^*,I_{r_1}^*)}{g(S_r^*,I_r^*,I_{r_1})}\right)\leq 0 \tag{24}$$

if

$$\begin{aligned}&\frac{I_{r_1}}{I_{r_1}^*}\geq\frac{I_r}{I_r^*} \text{ when } g(S_r^*,I_r^*,I_{r_1})\geq g(S_r^*,I_r^*,I_{r_1}^*) \text{ and}\\ &\frac{I_{r_1}}{I_{r_1}^*}\leq\frac{I_r}{I_r^*} \text{ when } g(S_r^*,I_r^*,I_{r_1})\leq g(S_r^*,I_r^*,I_{r_1}^*).\end{aligned} \tag{25}$$

holds for all $S_r, I_r, I_{r_1} > 0$. Since $g(S_r, I_r, I_{r_1})$ is a monotonic function $g(S_r^*, I_r^*, I_{r_1}) \geq g(S_r^*, I_r^*, I_{r_1}^*)$, it implies that $I_{r_1} \geq I_{r_1}^*$ and vice versa. In Eqs. (23) and (25) will hold for any concave function and are sufficient to ensure that $\frac{dV}{dt} \leq 0$. Thus, we have established the following result:

Theorem 4. *The endemic equilibrium E_r^* is globally asymptotically stable whenever conditions Eqs. (20) ∼ (25) are satisfied.*

3.1.2 Poverty-free only endemic equilibrium

This occurs when we set $\pi_0 = 0$. The endemic equilibrium (E_p^*) is given by

$$E_p^*\begin{cases} S_p^*=\frac{\Lambda}{\mu+\lambda_p^*},\\ I_p^*=\frac{\Lambda\lambda_p^*}{(\mu+\lambda_p^*)(\mu+\gamma+\phi_p)},\\ I_{p_1}^*=\frac{\Lambda\phi_p\lambda_p^*}{(\mu+\omega)(\mu+\lambda_p^*)(\mu+\gamma+\phi_p)},\\ A_p^*=\frac{\Lambda\phi_p\lambda_p^*}{(\mu+\eta\nu)(\mu+\eta\omega)(\mu+\lambda_p^*)(\mu+\gamma+\phi_p)}.\end{cases} \tag{26}$$

Based on the results of the previous discussion on Section 3.1.1, we claim the following result.

Theorem 5. *The endemic equilibrium E_p^* is globally asymptotically stable.*

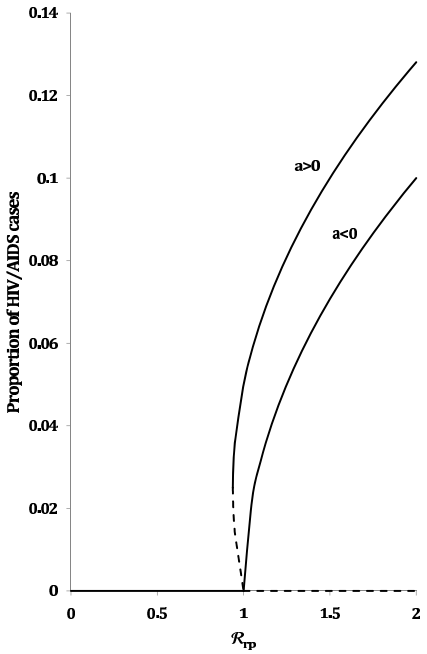

Fig. 2. Bifurcation diagram

3.2 Interior equilibrium point

We now present the endemic equilibrium when both the rich and the poor co-exist in the community. This state is denoted by E^*_{rp}

$$E^*_{rp}\begin{cases} S^*_r = \frac{\pi_0\Lambda}{\mu+\lambda^{**}_r}, \\ S^*_p = \frac{\pi_1\Lambda}{\mu+\lambda^{**}_p}, \quad I^*_r = \frac{\pi_0\Lambda\lambda^{**}_r}{(\mu+\lambda^{**}_r)(\mu+\gamma+\phi_r)}, \quad I^*_p = \frac{\pi_1\Lambda\lambda^{**}_p}{(\mu+\lambda^{**}_p)(\mu+\gamma+\phi_p)}, \\ I^*_{r_1} = \frac{\pi_0\Lambda\phi_r\lambda^{**}_r}{(\mu+\omega)(\mu+\lambda^{**}_r)(\mu+\gamma+\phi_r)}, \quad I^*_{p_1} = \frac{\pi_1\Lambda\phi_p\lambda^{**}_p}{(\mu+\omega)(\mu+\lambda^{**}_p)(\mu+\gamma+\phi_p)}, \\ A^*_r = \frac{\pi_0\Lambda\phi_r\lambda^{**}_r}{(\mu+\nu)(\mu+\omega)(\mu+\lambda^{**}_r)(\mu+\gamma+\phi_r)}, \quad A^*_p = \frac{\pi_1\Lambda\phi_p\lambda^{**}_p}{(\mu+\eta\nu)(\mu+\eta\omega)(\mu+\lambda^{**}_p)(\mu+\gamma+\phi_p)}. \end{cases} \tag{27}$$

Before starting our main results, we give the following lemma which will be useful in the subsequent section.

Theorem 6. *(See [11, 12]) Consider the following general system of ordinary differential equations with a parameter ϕ,*

$$\frac{dx}{dt} = f(x,\phi), f : \mathbb{R}^n \times \mathbb{R} \to \mathbb{R}^n and \;\; f \in \mathbb{C}^2(\mathbb{R}^n \times \mathbb{R}). \tag{28}$$

Without loss of generality, it is assumed that 0 is an equilibrium for System Eq. (28) for all values of the parameter ϕ, that is $f(0,\phi) = 0$ for all ϕ and assume

A1: $A = D_x f(0,0) = (\frac{\partial f_i}{\partial x_j}(0,0))$ is the linearisation of system Eq. (28) around the equilibrium 0 with ϕ evaluated at 0. Zero is a simple eigenvalue of A and other eigenvalues of A have negative real parts;

A2: Matrix A has a right eigenvector w and a left eigenvector v corresponding to the zero eigeinvalue.

Let f_k be the K^{th} component of f and

$$a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0),$$
$$b = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \phi}(0,0). \tag{29}$$

The local dynamics of Eq. (28) around 0 are totally governed by a and b.

i. $a > 0, b > 0$. *When* $\phi < 0$ *with* $|\phi| << 1$, 0 *is locally asymptotically stable, and there exists a positive unstable equilibrium; when* $0 < \phi << 1$, 0 *is unstable and there exists a negative and locally asymptotically stable equilibrium;*

ii. $a < 0, b < 0$. *When* $\phi < 0$ *with* $|\phi| << 1$, 0 *is unstable; when* $0 < \phi << 1$, 0 *is asymptotically stable, and there exists a positive unstable equilibrium;*

iii. $a > 0, b < 0$. *When* $\phi < 0$ *with* $|\phi| << 1$, 0 *is unstable, and there exists a locally asymptotically stable negative equilibrium; when* $0 < \phi << 1$, 0 *is stable, and a positive unstable equilibrium appears;*

iv. $a < 0, b > 0$. *When* ϕ *changes from negative to positive,* 0 *changes its stability from stable to unstable. Correspondingly, a negative equilibrium becomes positive and locally asymptotically stable.*

In order to apply the Center Manifold Theory, we make the following change of variables. Set $S_r = x_1$, $I_r = x_2$, $I_{r_1} = x_3$, $A_r = x_4$, $S_p = x_5$, $I_p = x_6$, $I_{p_1} = x_7$, and $A_p = x_8$, so that

$$\lambda_r = \frac{\beta[x_2 + x_6 + \alpha(\kappa_r x_3 + \kappa_p x_7)]}{x_1 + x_2 + (1-\kappa_r)x_3 + x_5 + x_6 + (1-\kappa_p)x_7}.$$

and $\lambda_p = \sigma\lambda_r$. Further, by using vector notation $\boldsymbol{x} = (x_1, x_2, x_3, x_4, x_5, x_6, x_7, x_8)^T$, model system Eq. (2) can be written in the form $\frac{\mathrm{d}\boldsymbol{x}}{\mathrm{d}t} = F(x)$, with $\boldsymbol{F} = (f_1, f_2, f_3, f_4, f_5, f_6, f_7, f_8)^T$, that is:

$$\begin{aligned}
&x_1' = f_1 = \pi_0\Lambda - (\lambda_r + \mu)x_1, \quad x_2' = f_2 = \lambda_r x_1 - (\gamma + \phi_r + \mu)x_2, \\
&x_3' = f_3 = \phi_r x_2 - (\omega + \mu)x_3, \quad x_4' = f_4 = \gamma_r x_2 + \omega x_3 - (\mu + \nu)x_4, \\
&x_5' = f_5 = \pi_1\Lambda - (\lambda_p + \mu)x_5, \quad x_6' = f_6 = \lambda_p, \quad x_5 - (\eta\gamma + \phi_p + \mu)x_6, \\
&x_7' = f_7 = \phi_p, \quad x_6 - (\eta\omega + \mu)x_7, \quad x_8' = f_8 = \eta(\gamma x_6 + \omega x_7) - (\mu + \tau\nu)x_8.
\end{aligned} \tag{30}$$

The method entails evaluating the Jacobian of the system Eq. (30) at the disease-free equilibrium (E^0) denoted by $J(E^0)$, with

$$J(E^0) = \begin{bmatrix}
-\mu & -\beta\pi_0 & -\alpha\beta\kappa_r\pi_0 & 0 & 0 & -\beta\pi_0 & -\beta\pi_0\kappa_p & 0 \\
0 & \beta\pi_0 - k_1 & \alpha\beta\kappa_r\pi_0 & 0 & 0 & \beta\pi_0 & \beta\pi_0\kappa_p & 0 \\
0 & \phi_r & -(\mu+\omega) & 0 & 0 & 0 & 0 & 0 \\
0 & \gamma & \omega & -(\mu+\nu) & 0 & 0 & 0 & 0 \\
0 & -\sigma\beta\pi_1 & -\alpha\sigma\beta\pi_1\kappa_r & 0 & -\mu & -\sigma\beta\pi_1 & -\sigma\beta\pi_1\kappa_p & 0 \\
0 & \sigma\beta\pi_1 & \alpha\sigma\beta\pi_1\kappa_r & 0 & 0 & \sigma\beta\pi_1 - k_2 & \sigma\beta\pi_1\kappa_p & 0 \\
0 & 0 & 0 & 0 & 0 & \phi_p & -(\eta\omega+\mu) & 0 \\
0 & 0 & 0 & 0 & 0 & \eta\gamma & \omega & -(\mu+\tau\nu)
\end{bmatrix} \tag{31}$$

where $k_1 = \gamma + \mu + \phi_r$, $k_2 = \eta\gamma + \phi_p + \mu$. From Eq. (31), it can be shown that

$$R_{rp} = \beta\Big[\frac{\pi_0}{\mu+\gamma+\phi_r}\Big(1 + \frac{\alpha\phi_r\kappa_r}{\mu+\omega}\Big) + \frac{\sigma\pi_1}{\mu+\phi_p+\eta\gamma}\Big(1 + \frac{\alpha\phi_p\kappa_p}{\mu+\eta\omega}\Big)\Big]. \tag{32}$$

Suppose β, is chosen as a bifurcation parameter. Solving Eq. (32) for $R_{rp} = 1$, one gets

$$\beta = \beta^* = \frac{1}{\Big[\frac{\pi_0}{\mu+\gamma+\phi_r}\Big(1 + \frac{\alpha\phi_r\kappa_r}{\mu+\omega}\Big) + \frac{\sigma\pi_1}{\mu+\phi_p+\eta\gamma}\Big(1 + \frac{\alpha\phi_p\kappa_p}{\mu+\eta\omega}\Big)\Big]}. \tag{33}$$

Eigenvectors of $J(E^0)$ It can be shown that the Jacobian $J(E^0)$ of system Eq. (31) at $\beta = \beta^*$ has a right eigenvector (corresponding to the zero eigenvalue) given by $\mathbf{w} = (w_1, w_2, w_3, w_4, w_5, w_6, w_7, w_8)^T$, where

$$\mathbf{w} = \begin{cases}
w_1 = -\frac{\beta^*\pi_0}{\mu}\Big[w_2\Big(1 + \frac{\alpha\phi_r\kappa_r}{\mu+\omega}\Big) + w_6\Big(1 + \frac{\alpha\phi_p\kappa_p}{\mu+\eta\omega}\Big)\Big], \quad w_2 > 0, \quad w_3 = \frac{\phi_r w_2}{\mu+\omega}, \\
w_4 = \frac{[\mu\gamma + (\gamma+\phi_r)]w_2}{(\mu+\nu)(\mu+\omega)}, \quad w_5 = -\frac{\sigma\beta^*\pi_1}{\mu}\Big[w_2\Big(1 + \frac{\alpha\phi_r\kappa_r}{\mu+\omega}\Big) + w_6\Big(1 + \frac{\alpha\phi_p\kappa_p}{\mu+\eta\omega}\Big)\Big], \\
w_6 > 0, \quad w_7 = \frac{\phi_p w_6}{(\mu+\eta\omega)}, \quad w_8 = \frac{\eta[\gamma\mu + \omega(\phi_p+\eta\gamma)]w_6}{(\mu+\eta\omega)(\mu+\tau\nu)}.
\end{cases} \tag{34}$$

Further, the Jacobian $J(E^0)$ has a left eigenvector (associated with the zero eigenvalue) given by $\mathbf{v} = (v_1, v_2, v_3, v_4, v_5, v_6, v_7, v_8)^T$, where

$$\mathbf{v} = \begin{cases} v_1 = v_4 = v_5 = v_8 = 0, \quad v_2 > 0, \quad v_6 > 0, \\ v_3 = \frac{\alpha\beta^*\kappa_r(\pi_0 v_2 + \sigma\pi_1 v_6)}{\mu+\omega}, \quad v_7 = \frac{\alpha\beta^*\kappa_p(\pi_0 v_2 + \sigma\pi_1 v_6)}{\mu+\eta\omega}. \end{cases} \tag{35}$$

Computations of a and b. It can be shown, after some algebraic manipulations (involving the associated non-zero partial derivatives of F (at the DFE) to be used in the expression for a and b in Theorem 6), that

$a = \xi_1 + \xi_2 + \xi_3$, where

$$\xi_1 = -\frac{2\beta^* w_6^2(\pi_0 v_2 + \sigma\pi_1 v_6)}{\Lambda}\left(1 + \frac{\phi_p(1+\kappa_p(\alpha-1)) + \alpha\kappa_p(1-\kappa_p))}{\mu+\eta\omega},\right)$$

$$\xi_2 = -\frac{2\beta^{*2}\pi_0\pi_1(v_2+\sigma^2 v_6)}{\Lambda}\left[w_2\left(1+\frac{\alpha\phi_r\kappa_r}{\mu+\omega}\right) + w_6\left(1+\frac{\alpha\phi_p\kappa_p}{\mu+\eta\omega}\right)\right],$$

$$\xi_3 = -\frac{2\beta^*\mu\phi_r(\pi_0 v_2 + \sigma\pi_1 v_6)}{\Lambda(\mu+\omega)}\left(\frac{\kappa_r(1-\kappa_r)\phi_r}{\mu+\omega}w_2 + \frac{\phi_p[(1+\kappa_r(\alpha-1)) + (\kappa_r(1-\kappa_p) + \kappa_p(1-\kappa_r))]}{\mu+\eta\omega}w_6\right),$$

$$\text{and, } b = (\pi_0 v_2 + \pi_2 v_6)\left[w_2\left(1+\frac{\alpha\phi_r\kappa_r}{\mu+\omega}\right) + w_6\left(1+\frac{\alpha\phi_p\kappa_p}{\mu+\omega}\right)\right] > 0. \tag{36}$$

Since $a < 0, b < 0$, it follows from Theorem 6 item (iv), that

Theorem 7. *The endemic equilibrium E^*_{rp} is locally asymptotically stable for $R_{rp} > 1$ but close to one.*

Table 1. Model parameters and their interpretation.

Parameter	Symbol	Value	Source
Recruitment rate for humans	Λ	0.029yr^{-1}	[4, 11]
Natural mortality rate for humans	μ	0.02yr^{-1}	[4, 11]
Disease-induced mortality rate	ν	0.33yr^{-1}	[4, 11]
Natural rate of progression to AIDS	γ, ω	(0.125,0.1)yr^{-1}	[4, 11]
Modification parameter	α	0.4	[6, 40]
Modification parameter	η, σ	1.25,1.35	Assumed
Effective contact rate	β	0.35(0.011-0.95)	[4, 11]
Proportion of children born from rich families	π_0	0.35	Assumed
Treatment rate	ϕ_r, ϕ_p	variable	Assumed
A fraction of individuals who are on antiretroviral therapy and still indulging in risky sexual behavior	κ_r, κ_p	0.1,0.15	Assumed.

The results for Theorem 7 are illustrated in Fig. 2.

3.3 Effects of risky sexual behavior

The reproductive number R_{rp} is analyzed to determine whether there is an association between HIV/AIDS and socioeconomic status. We shall investigate the relative importance of the different factors responsible for initial disease transmission, which is directly related to the magnitude of R_{rp}. The effect of risky sexual behavior on the spread of HIV/AIDS is investigated by partially differentiating R_{rp} with respect to κ_p, κ_r, and σ

$$(a)\ \frac{\partial R_{rp}}{\partial \kappa_p} = \frac{\beta\epsilon\sigma\pi_1\phi_p}{(\mu+\eta\omega)(\mu+\eta\gamma+\phi_p)} > 0,$$

$$(b)\ \frac{\partial R_{rp}}{\partial \kappa_r} = \frac{\beta\epsilon\pi_0\phi_r}{(\mu+\omega)(\mu+\gamma+\phi_r)} > 0,$$

$$(c)\ \frac{\partial R_{rp}}{\partial \sigma} = \frac{\beta\pi_1(\mu+\eta\omega+\epsilon\kappa_p\phi_p)}{(\mu+\eta\omega)(\mu+\eta\gamma+\phi_p)} > 0. \tag{37}$$

The first and second expressions in Eq. (37) measure the impact of risky sexual behavior associated with individuals on antiretroviral therapy from the poor and rich classes, respectively. These expressions (a) and (b) suggest that if individuals who are on antiretroviral therapy do not change their sexual behavior either due to low financial resources, to affirm their social identity, or to deny that 'anything they do makes a difference to what they perceive as a life of powerlessness and despair'[8, 10], then HIV/AIDS will continue to spread into the community, irregardless of their socioeconomic status. However, the literature above has shown that $\kappa_r < \kappa_p$, implying that a high proportion of these individuals are from the poor class. Then socioeconomic status will have an impact on the transmission dynamics, since the poor will be fueling the epidemic. It is also worth noting that, from the expression of R_{rp} ($R_{rp} = R_r + R_p$), an increase in either the *rich-induced reproductive ratio* R_r, or the *poverty-induced reproductive ratio* R_p, may lead to an increase in HIV/AIDS in the community. Thus, if individuals from the poor class continue to live risky sexual lives due to poor living standards[8, 10], then HIV/AIDS will continue to spread in the community affecting all. Furthermore, malnutrition is known to weaken the immune system, which in turn may lead to a greater risk of HIV transmission in any unprotected sexual encounter[7, 18], which is a factor usually associated with the poor. From the expression of R_{rp}, an increase of R_p will lead to an increase in R_{rp}, which may result in an increase in HIV/AIDS in the community.

4 Numerical simulations

To illustrate the results of the foregoing analysis, numerical simulations of the model system Eq. (2) are carried out, using parameter values given in Tab. 1.

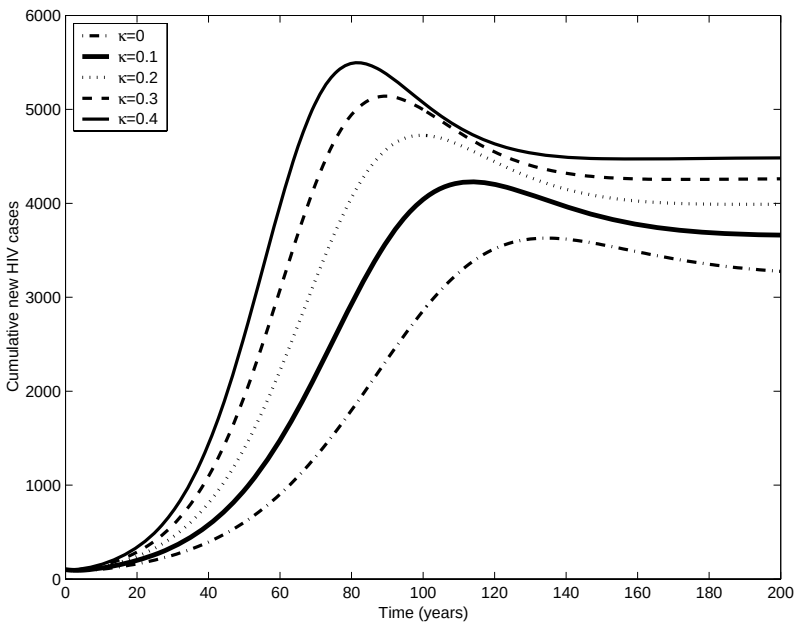

Fig. 3. Simulations of model system (2), showing the effects of risky sexual behavior associated with individuals on antiretroviral therapy, are demonstrated over time. In this case we assume $\kappa = (\kappa_r = \kappa_p)$ and an initial incidence of HIV to be constant at 100 cases per 100,000 per year. The rest of the parameter values are fixed on their baseline values from Tab. 1. When $\kappa = 0.0, 0.1, 0.2, 0.3, 0.4$ the reproductive number R_{rp} is $1.43, 1.49, 1.54, 1.56, 1.65$

Fig. 3 is a graphical representation showing the effects of varying the contribution $(\kappa = (\kappa_r = \kappa_p))$ of HIV infectives who are on antiretroviral therapy. It suggests that an increase of risky sexual behavior associated with individuals on antiretroviral therapy irregardless of their socioeconomic status will increase HIV prevalence.

We evaluate the effect of varying the contribution (σ) of risky sexual behavior associated with individuals from the poor class as defined in our study. The results shown in Fig. 4 follow a similar trend to the results

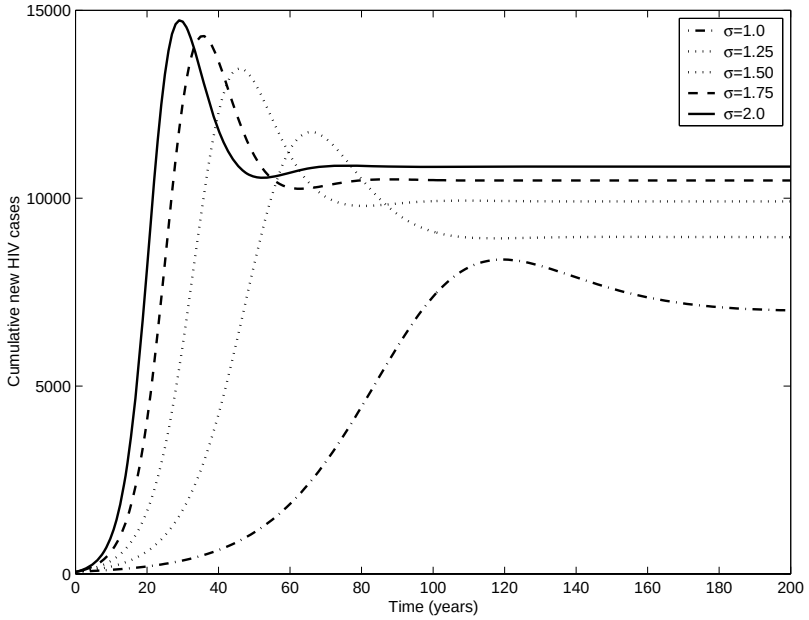

Fig. 4. Simulations of model system Eq. (2) showing the effects of risky sexual behavior associated with individuals from the poor class is demonstrated over time. We vary the modification parameter σ from 1.0 to 2.0 in the steps of 0.25, other parameter values are fixed on their baseline values from Tab. 1. When σ is $1.0, 1.25, 1.5, 1.75, 2.0$ the reproductive number R_{rp} is $1.22, 1.42, 1.62, 1.82, 2.02$

in Fig. 3 and they also support the analytical results illustrated in Eq. (37)(c), that risky sexual behavior increases HIV prevalence. The simulations (Fig. 4) also suggest that an increase in risky sexual behavior by individuals from the poor class due to poor living standards, or any other reason aforementioned in this study, will increase the epidemic prevalence. In addition, results from Fig. 4 further suggest that in the absence of effective programmes aimed at changing sexual and other behaviors for the poor, then HIV/AIDS will continue to be a health burden, since the poor may persist with behaviors which expose them to HIV infection.

5 Conclusion

A mathematical model for the transmission dynamics of HIV/AIDS in the context of socioeconomic status (with two sub-populations: the rich and the poor) is formulated and its mathematical properties are investigated in order to assess the effect of socioeconomic status on the transmission dynamics of HIV/AIDS. The disease-free equilibrium is shown to be globally stable when the corresponding reproductive number is less than unity. The poor class is defined herein as those individuals with few if any financial or other assets and are often socially marginalised. Due to low financial resources, we assume that these individuals are involved in risky sexual behavior, as to procure food for themselves and their children. Results from the study suggest that risky sexual behavior of individuals who are on antiretroviral therapy will increase HIV prevalence in the community. If poor individuals indulge in risky sexual behavior due to social marginalisation or to procure food for themselves and their children, then HIV/AIDS will remain a public health burden.

References

[1] N. Adler, K. Newman. Socioeconomic disparities in health: pathways and policies. *Health Aff (Millwood)*, 2002, **21**: 60–76.

[2] M. Ainsworth, I. Semali. *Who is most likely to die of aids? Socioeconomic correlates of adult deaths in Kagera region, Tanzania.* Confronting AIDS: evidence for developing world, 1998. Edited by Ainsworth M, Fransen L, Over M. Brussels: European Commission.

[3] J. Arino, F. Brauer, et al. A model for influenza with vaccination and antiviral treatment. *Journal of Theory Biology*, 2008, **253**: 118–130.

[4] C. Bhunu, S. Mushayabasa, et al. Mathematical analysis of an HIV/AIDS model: Impact of educational programs and abstinence in Sub-Saharan Africa. *Journal of Mathematical Modelling and Algorithms*, 2011, **10**: 31–55.
[5] C. Bhunu, J. Tchuenche, et al. Modelling the effects of schistosomiasis on the transmission dynamics of HIV/AIDS. *Journal of Biological Systems*, 2010, **18**(2): 277–297. DOI No.: 10.1142/S0218339010003196.
[6] S. Blower, E. Bodine, et al. The antiretriovial roll out and drug resistant HIV in Africa: insights from empirical data and theoretical models. *AIDS*, 2005, **19**(1): 1–14.
[7] F. Booysen. Hiv/aids and poverty: Evidence from a household impact study conducted in the free state province, South Africa. *DPRU Conference*, 2002. Johannesburg.
[8] D. Bryceson, J. Fonseca. An enduring or dying peasantry: interactive impact of famine and HIV/AIDS in rural.
[9] A. Buve, E. Lagarde, et al. Study group on heterogeneity of hiv epidemics in African cities. interpreting sexual behavior data: validity issues in the multicentre study on factors determining the differential spread of hiv in four African cities. *AIDS*, 2001, **15**(4): 117–126.
[10] E. Byron, S. Gillespie, P. Hamazakaza. Local perceptions of risk and HIV prevention in Aouthern Zambia. *RENEWAL working paper*, 2007. www.ifpri.org/renewal.
[11] C. Castillo-Chavez, Z. Feng, W. Huang. On the computation of r0 and its role on global stability. 2002. Math.la.asu.edu/chavez/2002/JB276.pdf.
[12] C. Castillo-Chavez, B. Song. Dynamical models of tuberculosis and their applications. *Mathematical Biosciences and Engineering*, 2004, **1**(2): 361–404.
[13] D. Cohen. Poverty and HIV/AIDS in sub-Saharan Africa, undp HIV and development programme. 2009, **27**. Http : //www:undp:org=hiv=publications=issues=english=issue27e:html, retrieved August 04.
[14] I. Cremin, C. Naymukapa, et al. Patterns of self-reported behaviour change associated with receiving voluntary counselling and testing in a longtudinal study from manicaland, Zimbabwe. *AIDS Behav*, 2010, **14**(708-715).
[15] V. Driessche, J. Watmough. Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Math. Biosci.*, 2002, 29–48.
[16] L. Fenton. Preventing HIV/AIDS through poverty reduction: the only sustainable solution? *Lancet*, 2004, **364**: 1186–1187.
[17] J. Fotso, B. Kuate-Defo. Socioeconomic inequalities in early childhood malnutrition and morbidity: modification of the household-level effects by the community ses. *Health Place*, 2005, **11**: 205–225.
[18] S. Gillespie, S. Kadiyala, R. Greener. Is poverty or wealth driving HIV transmission? *AIDS*, 2007, **21**. 21 (suppl 7): S5S16.
[19] J. Glynn, M. Carael, et al. Does increased general schooling protect against HIV infection? a study in four African cities. *Tropical Medicine & International Health*, 2004, **9**: 4–14.
[20] J. Glynn, M. Carael, et al. Does increased general schooling protect against HIV infection? a study in four African cities. *Tropical Medicine & International Health*, 2004, **9**: 4–14.
[21] S. Gregreson, E. Gonese, et al. Hiv decline in Zimbabwe due to reductions in risky sex? Evidence from a comprehensive epidemiological review. *International Journal of Epidemiology*, 2010.
[22] S. Gregson, G. Garnett, et al. HIV decline associated with behaviour change in Eastern Zimbabwe. *Science*, 2006, **311**: 664–666.
[23] S. Gregson, H. Waddell, S. Chandiwana. School education and HIV control in sub-Saharan Africa: from discord to harmony? *Journal of International Development*, 2001, **13**: 467–485.
[24] J. Hargreaves, J. Glynn. Educational attainment and hiv-1 infection in developing countries: a systematic review. *Tropical Medicine & International Health*, 2002, **7**: 489–498.
[25] A. Korobeinikov. Lyapunov functions and global stability for sir and sirs epidemiological models with non-linear transmission. *Bulletin of Mathematical Biology*, 2006, **30**: 615–626.
[26] B. Kuate-Defo. Effects of socioeconomic disadvantage and women status on women health in Cameroon. *Social Science & Medicine*, 1997.
[27] J. Lasalle. *The stability of dynamical systems SIAM*, Philadelphia, PA. 1976.
[28] B. Lopman, J. Lewis, et al. Hiv incidence and poverty in manicaland, Zimbabwe: is HIV becoming a disease of the poor? *AIDS*, 2007, **21**(7): 57–66.
[29] Malawi. Aids, poverty and hunger: challenges and responses. 2006, **44**: 1023–1042. Edited by Gillespie SR. Washington DC: IFPRI.
[30] C. Mccluskey. Lyapunov functions for tuberculosis models with fast and slow progression. *MBE*, 2006, **3**: 603–614.
[31] A. Mishra, V. Bignami-Van, et al. HIV infection does not disproportionately affect the poorer in sub-Saharan Africa. *AIDS*, 2007, **21**(7): 17–28.
[32] S. Mushayabasa, J. Tchuenche, et al. Modeling gonorrhea and HIV co-interaction. *BioSystems*, 2011, **103**(1): 27–37.
[33] M. Nyindo. Complementary factors contributing to the rapid spread of HIV-1 in sub-Saharan Africa: a review. *East African Medical Journal*, 2005, **82**: 40–46.

[34] S. Pedro, J. Tchuenche. HIV/AIDS dynamics: Impact of economic classes with transmission from poor clinical settings. *Journal of Theory Biology*, 2010, **267**(4): 471–485.

[35] T. Porco, J. Martin, et al. Decline in hiv infectivity following the introduction of highly active antiretroviral therapy. *AIDS*, 2004, **18**: 81–88.

[36] N. Prata. Making family planning accessible in resource-poor settings. *Philosophical Transactions of the Royal Society B*, 2009, **364**: 3093–3099.

[37] UNAIDS. Sub-Saharan Africa AIDS epidemic update regional summary. 2008. Jointed United NAtions Programme on HIV/AIDS (UNAIDS) and World Health Organisation,(WHO), Geneva, Switerland.

[38] UNAIDS. Sub-Saharan Africa AIDS epidemic update regional summary. 2009. Jointed United NAtions Programme on HIV/AIDS (UNAIDS) and World Health Organisation,(WHO), Geneva, Switer-land.

[39] D. Walque, J. Nakiyingi-Miiro, et al. Changing association between schooling levels and HIV-1 infection over 11 years in a rural population cohort in Southwest Uganda. *Tropical Medicine & International Health*, 2005, **10**: 993–1001.

[40] D. Walque, J. Nakiyingi-Miiro, et al. Changing association between schooling levels and HIV-1 infection over 11 years in a rural population cohort in Southwest Uganda. *Tropical Medicine & International Health*, 2005, **10**: 993–1001.

[41] J. Wojciki. Socioeconomic status as a risk factor for HIV infection in women in east, central and Southern Africa: a systematic review. *Journal of Biosoc Science*, 2005, **37**: 1–36.