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Review of SIR Epidemic Model with Application To Transmission Dynamics of HIV/AIDS in a Proportional Mixing Population

Umar Mallam Abdulkarim
Department of Mathematical Sciences, Nasarawa State University Keffi, Nigeria

Abstract: In this study, we examined, the Susceptibles-Infectives-Removed/Recovered, (SIR) epidemic model and applied it to horizontal transmission of HIV/AIDS in a homogeneous mixing population. with additional assumption that AIDS virus does not kill instead; AIDS-infectives are removed from circulation until death by non disease induced. Also the stability of the equilibrium points are examined via the basic reproductive number of the infection and trace-determinant condition of the Jacobian matrix at the equilibrium point, for a system of non-linear differential equation. The threshold conditions on the model parameters, which allows stability of the disease-free equilibrium and the endemic equilibrium points are derived and their biological interpretations given.

Key words: Basic reproductive number, infectivity kernel, HIV, AIDS, transmission rate, stability, vital dynamics, transmission dynamics, disease free-equilibrium point, endemic equilibrium point, transmissibility of the virus, epidemics

INTRODUCTION

The compartments of Susceptibles, Infectives and Removed classes, simply called SIR model is often applied to contagious illness in a closed population, The simplest of this type of model without vital dynamics was proposed by Ronald Ross in 1911 (Bailey, 1975) as,

$$S = -\beta IS$$
$$I = -\beta IS$$

Where β, is the transmission rate, while S(t) and I(t) are the population (density) of the susceptibles and infectives. However, Kermack and Mckendrick extended Ronald Ross epidemic model with a general time infectivity kernel, $\bar{A}(\tau)$, (Diekmann *et al*., 1993) and the following assumptions:

- A single infection triggers an autonomous process within the host.
- The disease results in either complete immunity or death.
- Contact are according to the law of mass-action.
- All individuals are equally susceptible.
- The population is closed.
- The population size is large to sustain a deterministic description.

These assumptions leads to the integral equation, (Diekmann *et al*., 1993)

$$S(t) = S(t)\int_0^\infty \bar{A}(\tau)S(t-\tau)d\tau \quad (1)$$

Where S(t) is the (spatial) density of susceptibles, (number of individual per unit of area) and $\bar{A}(\tau)$ is time infectivity kernel, described as the infectivity of an individual that became infected τ units of time ago, (Diekmann *et al*., 1993) -S(t) is the incidence at time t and-S(t-τ) is the number of individual arising per unit of time who at time have been infected for τ time units, (Diekmann *et al*., 1993). However, defining,

$$I(t) = -\frac{1}{\beta}\int_0^\infty \bar{A}(\tau)S(t-\tau)d\tau = -\frac{1}{\beta}\int_{-\infty}^t \bar{A}(t-\tau)S(t)d\tau \quad (2)$$

and differentiating, using time-infectivity kernel, $\bar{A}(\tau) = \beta e^{-\gamma\tau}$, Diekmann *et al*. (1993) obtained the following system of differential equations,

$$\begin{aligned} S(t) &= -\beta S(t)\, I(t) \\ I(t) &= \beta S(t)\, I(t) - \gamma\, I(t) \end{aligned} \quad (3)$$

Where β is the probability of transmission of the infection from an infectious person to a susceptible person and γ is the rate of removal/recovery of the infectives. Some studies simply refers to this model as Kermack and Mckendrick epidemic model, as seen in the research of Murray (1989) and Leah (2005) while others like Diekmann *et al*. (1993) disagree, noting that the Kermack and Mckendrick model deals with a general

time-infectivity kernel and that model (3) satisfies only the infectivity kernel $\bar{A}(\tau) = \beta e^{-\gamma\tau}$. However, the conventional Susceptible-Infective-Removed/recovered model (SIR) without vital dynamics has the form, (Murray, 1989)

$$\begin{aligned} S(t) &= -\beta S(t)\, I(t) \\ I(t) &= \beta S(t)\, I(t) - \alpha I(t) \\ R(t) &= \alpha I(t) \end{aligned} \tag{4}$$

Where α is the rate at which infectives are removed/recovered from the infection, β is the rate of transmission from a susceptible to an infectious person. While S(t), I(t) and R(t), are the population of susceptibles, infectives and removed/recovered classes, respectively.

In this research, we intends to use the approach in Eq. 1-4 to examine the transmission dynamic of HIV/AIDS, with vital dynamics.

THE MATHEMATICAL MODEL

Let the following parameters be represented as follows:

β : Probability of transmission of the virus from an infectious person to a susceptible.
b : The birth rate, assumed the same as the death rate.
α : The rate at which HIV-infected developed AIDS.
C : The average number of partners per unit time
S: Population of susceptibles, (HIV-negative).
I: Population of HIV-infectives, (HIV-positive).
A : Population of AIDS-infectives.

We have assumed in this compartmental model that death rate is the same as the birth rate and death in the AIDS class is not disease induced. Introducing AIDS class of infectives, denoted A in the SIR epidemic model, reduces it to a simple SIA model, where the removed/recovered class forms the class of AIDS infectives, without disease induced death. Since they are assumed, non-sexually active and are quarantined. They don't contribute to transmission dynamics of the infection.

However, this is not realistic but would be employed for the purpose of this study. Also suppose the following assumptions hold:

- Infectives remain so for an exponential distributed period of time with parameter β,
- All individuals are exposed to a death rate b;
- Susceptibles produce offspring at a per capital rate b;
- Infectives do not produce offspring (since they are sexually inactive).
- Latency of HIV is negligible.
- HIV-infectives progress to AIDS at the rate of α
- The average number of partners per unit time, by a susceptible with an HIV-infective, of disease transmitting type is C.

These assumptions lead to the following system of non-linear ordinary differentiation equations,

$$\begin{aligned} S &= bN - \beta C\frac{IS}{N} - bS \\ I &= \beta C\frac{IS}{N} - \alpha I - bI \\ A &= \alpha I - bA \\ &\text{Where } S + I + A = N \end{aligned} \tag{5}$$

At the equilibrium points, we have, the following equations satisfied,

$$\frac{dS}{dt} = \frac{dI}{dt} = \frac{dA}{dt} = 0 \tag{6}$$

The disease-free equilibrium point are determined as, E_0 = (N,0,0) and the Jacobian matrix at the equilibrium point is obtained as,

$$J_{E_0} = \begin{pmatrix} -b & -\beta C & 0 \\ 0 & \beta C - (\alpha + b) & 0 \\ 0 & \alpha & -b \end{pmatrix}$$

If λ_j (j = 1, 2, 3) are the eigenvalues of J_{E_0}, then

$$(\lambda + b)\det\begin{pmatrix} \beta C - (\alpha + b) - \lambda & 0 \\ \alpha & -b - \lambda \end{pmatrix} = 0$$

Let

$$B = \begin{pmatrix} \beta C - (\alpha + b) & 0 \\ \alpha & -b \end{pmatrix}$$

then we would have det(B) = $(\alpha+b)b - b\beta C$, which is greater than zero whenever $\beta C < \alpha + b$. Also the $B = \beta C - (\alpha + 2b) < 0$, since $(\alpha + 2b) > \beta C$. Where βC is the net transmission of the virus and $1/\alpha+b$ is the average length of infection for AIDS. Thus the trace of B is negative and the disease-free equilibrium point E_0 is stable provided $(\beta C/\alpha+b) < 1$. To have an AIDS free stable population, the product of the net transmission of HIV-infection and the average length of infection for AIDS should be less than unity.

POSSIBILITY OF EXISTENCE OF ENDEMIC EQUILIBRIUM POINT

Let

$$E_1 = (S^*,\ I^*\ A^*) = \left(N\frac{(\alpha+b)}{\beta C}\quad \frac{\beta C}{\alpha+b}-1\quad \frac{\beta C}{\alpha+b}-1 \right), \quad (7)$$

where $S^* = N\frac{(\alpha+b)}{\beta C},\ I^* = \frac{\beta C}{\alpha+b}-1,\ A^* = \frac{\beta C}{\alpha+b}-1$

(S^*, I^*, A^*), is a non-zero endemic equilibrium point. Thus, the population consists of some proportion of each type, provided, $E_1 = (S^*, I^*, A^*)$ are all positive quantities. And for I* and A* to be positive we must have,

$$\frac{\beta C}{\alpha+b} > 1 \quad (8)$$

The population of infectives I* and A* AIDS, exist if (8) is satisfied. The Jacobian matrix at the endemic equilibrium point is given by,

$$J_{E_1} = \begin{pmatrix} \beta C\left(\frac{\beta Cb}{\alpha+b}+b\right)-b & -(\alpha+b) & 0 \\ \beta Cb(\beta C-1) & 0 & 0 \\ 0 & \alpha & -b \end{pmatrix}$$

The eigenvalues of matrix J_{E_1} satisfies,

$$(\lambda+b)\det\begin{pmatrix} \beta C\left(\frac{\beta C}{\alpha+b}+b\right) & -b-\lambda & -(\alpha+b) \\ \beta Cb(\beta C-1) & & -\lambda \end{pmatrix} = 0$$

Let matrix

$$B = \begin{pmatrix} \beta C\left(\frac{\beta C}{\alpha+b}+b\right)-b & -(\alpha+b) \\ \beta Cb(\beta C-1) & 0 \end{pmatrix}$$

Then the determinant of matrix $B = \beta Cb\,(\alpha+b)\,(\beta C-1) > 0$, if $\beta C>1$. However, evaluating the trace of matrix B we get, trace =

$$\beta C\left(\frac{\beta C}{\alpha+b}+b\right)-b = \frac{(\beta C)^2}{\alpha+b}+\beta Cb-b. > 0$$

which contradicts the requirment of stability for an equilibrium point. Thus the endemic equilibrium point, E_1. is not stable. However, Mugisha *et al.* (2005) and Heffernan *et al.* (2005) gave insights into the use of the basic reproductive number R_0 in analyzing stability of endemic equilibrium point.

They observed that if $R_0>1$, then the system has a unique endemic equilibrium point that is globally asymptotically stable and the disease-free equilibrium is unstable. However, if $R_0<1$, then the disease-free equilibrium is locally asymptotically stable and the endemic equilibrium is unstable.

Adopting this approach using the next generation operator by Diekmann (Herffernan *et al.*, 2005) we categorized the population into two classes, as X = (S), Z = (I) and the disease-free equilibrium is $E_0 = (N, 0, 0)$. We would then get the following equations from our model, in line with Herffernan *et al.* (2005) as,

$$\begin{aligned} \dot{X} &= bN - \beta C\frac{ZX}{N} - bX = f(X,Y,Z \\ \dot{Z} &= \beta C\frac{ZX}{N} - (\alpha+b)Z = h(X,Y,Z) \end{aligned} \quad (9)$$

Let the matrix $H = \partial h/\partial z$, then $H = \beta C-(\alpha+b)$

Letting H = M-D with M>0 and D>0 a diagonal matrix, $M = \beta C$ and $D = (\alpha+b)$

The basic reproductive number is defined as spectral radius (dominant eigenvalue) of the matrix $M\ D^{-1}$ (Mugisha *et al.*, 2005; Herffernan *et al.*, 2005) and obtained as,

$$R_0 = \rho(MD^{-1}) = \frac{\beta C}{(\alpha+b)}$$

Where βC is the net transmission rate and $1/\alpha+b$ is the average duration of infectiousness, or the incubation period, for AIDS, in this study. For a stable disease-free equilibrium point, we must have

$$\frac{\beta C}{(\alpha+b)} < 1 \ \text{ or } \ \beta C<(\alpha+b)$$

This requirement is also obtained using determinant/trace method of investigating stability of equilibrium point for two system of non-linear differential equations above.

SUB-MODEL WITHOUT PROGRESSION TO AIDS ($\alpha = 0$)

Consider a sub-model without AIDS and using the assumption that the population is constant, we reduce our system in Eq. 5 to a two equations model in Susceptibles

and HIV-infectives, consistent with our earlier assumption that AIDS is not death induced. Thus, the class of AIDS is regarded as removed/recovered class equivalent to a simple SIR model, as in Murray *et al.* (1989) and Bailey (1975). Thus we would have,

$$\begin{aligned} \dot{S} &= bN - \beta C\frac{IS}{N} - bS \\ \dot{I} &= \beta C\frac{IS}{N} - bI \\ \text{Where } A &= N - I - S \end{aligned} \tag{10}$$

The equilibrium points are (N, 0) and ((Nb/βC), $\frac{b(N-S)}{b}$). The Jacobian at the Disease-Fee Equilibrium points, (DFE) is,

$$J_{E_0} = \begin{pmatrix} -b & -\beta C \\ 0 & \beta C - b \end{pmatrix}$$

stability of DFE implies that the trace <0 and the determinant >0, (Kimbir *et al.*, 2003). Using matrix J_{E_1} the equilibrium point is stable if the trace <0 and the determinant >0. But trace = βC-2b<0. This is negative when (βC/2b)<1 and determinant = b-βC>0, only when b>βC or (βC/b)<1. Thus stability implies (βC/b)<1. This is consistent with the value of the basic reproductive number of HIV-infection, obtained using next generation operator as, R_0 = βC/b, with α = 0. The disease-free equilibrium is locally asymptotically stable if R_0= (βC/b)<1, which is consistent with the model for HIV/AIDS, with α≠0. Consider, the endemic equilibrium point,

$$E_1 = \left(\frac{Nb}{\beta C}, N-S\right)$$

where Nb/βC is the fraction of the population of the susceptibles and N-S is the fraction of HIV-infectious population. The associated Jacobian matrix,

$$J_{E_1} = \begin{pmatrix} \frac{-\beta C(N-S)}{N} - b & -b \\ \frac{\beta C(N-S)}{N} & 0 \end{pmatrix}$$

The trace

$$J_{E_1} = \frac{-\beta C}{N}(N-S) - b < 0 \Rightarrow -\frac{\beta C}{N}(N-S) - b < 0.$$

and the determinant

$$= \frac{\beta Cb}{N}(N-S) > 0$$

both satisfy the required conditions for stability of equilibrium point. Thus the endemic equilibrium point E_1 is stable.

CONCLUSION

Our intention in this research has been to study the SIR epidemic model and apply it to transmission dynamics of HIV/AID in a proportional mixing population, with vital dynamics and also examined the concept of non AIDS induced death as alternative to the conventional assumption of AIDS induced death and derive the threshold conditions for the two equilibrium points, DFE and EE points. Two types of models are examined in this respect, these includes a model for HIV and AIDS transmission dynamics and a submodel for HIV transmission dynamics without AIDS. The disease-free equilibrium and the endemic equilibrium points are determined and also their stability examined, using the basic reproduction number of the infection and trace and determinant stability condition of equilibrium points, for two systems of non-linear differential equations. For the model with progression from HIV infection to AIDS. (α≠0), we observed that the DFE is globally asymptotically stable, whenever it exists, provided that (βC/α+b)<1 and the endemic equilibrium point is not stable. For the submodel without disease progression, to AIDS, (α = 0). we observed that there exists a unique DFE which is locally asymptotically stable, provided, (βC/b)<1. Otherwise there exists an endemic equilibrium point which is locally asymptotically stable. In both cases the birth rate should be greater than the net transmission of the HIV-infection, as obtained via R_0 and the trace-determinant stability condition for system of non-linear differential equations.

In biological terms we can deduce the result of the study as:

- The condition βC<α+b, in the HIV/AIDS transmission model, means that the net Transmission rate in the model of HIV/AIDS infection is less than the sum of the rate of progression from HIV to AIDS and the birth rate. This threshold condition is observed in the computation of R_0 and the other method. Thus to minimized the transmissibility of HIV-infection and AIDS, we would need to lower the net transmission below unity and decrease the AIDS progression rate so that we would have longer incubation period for AIDS. HIV-infectives who develop AIDS after long incubation period are then removed, waiting to die by natural cause and not disease induced death.

- The condition ($\beta C/b$)<1, in the submodel without AIDS, means that net transmission of HIV-infection is less than the birth/death rate. Thus for the virus to be eradicated the birth rate should be greater than net transmission rate.
- In general, increasing the birth rate, decreasing AIDS progression rate and Minimizing net Transmission for both cases may eradicate HIV/AIDS, but would give long incubation period for AIDS.

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