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Modeling the Combined Effect of Vertical Transmission and Variable Inflow of Infective Immigrants on the Dynamics of HIV/AIDS

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Abstract: In this paper, a Non linear Mathematical model is proposed and studied the combined effect of vertical transmission (MTCT) and variable inflow of infective immigrants on the dynamics of HIV/AIDS. Vertical transmission means propagation of the disease from mother to children. 'Variable inflow of infective immigrants' includes both the aware and unaware infected immigrants. The equilibrium points of the model are found and the stability analysis of the model around these equilibrium points is conducted. The stability analysis on the model shows that the disease free equilibrium point E_0 is locally asymptotically stable when $R_0 < 1$. The positive endemic equilibrium point E^* is shown to be locally asymptotically stable when $R_0 > 1$. Further it is shown that $R_0 > R_0'$, this shows that the basic reproduction number of the present model is greater than the one which is obtained from the model modeled without vertical transmission. Through vertical transmission the disease flows from infected mother to children. That is, Vertical transmission contributes positively to the spread of the disease. Numerical simulation of the model is carried out to assess the effect of unaware HIV infective immigrants and vertical transmission (MTCT) in the spread of HIV/AIDS disease. The result showed that HIV infective immigrants and vertical transmission (MTCT) significantly affects the spread of the disease. Screening of the disease reduces the spread of HIV and also prevents mother to child transmission. It is well accepted that both vertical transmission and immigration contribute positively to the spread of the disease and these two parameters cannot be avoided in practice. Hence, the purpose of this study is to investigate the combined effect of vertical transmission, unaware and aware infected immigrants on the spread of HIV/AIDS and offers possible intervention strategies.

Keywords: HIV/AIDS, Unaware and Aware Infective Immigrant, Vertical Transmission (MTCT), Screening, Local Stability, Reproduction Number

1. Introduction

Human immunodeficiency virus (HIV) is a lent virus, a member of the retrovirus that causes Acquired Immunodeficiency Syndrome (AIDS). HIV infection in humans is considered as a pandemic disease by the world health organization (WHO). From the discovery of AIDS in the year 1981 till 2006 the records show that more than 25 million people have been killed worldwide. HIV infection is effecting about 0.6% of the world's population. In 2005, HIV infected about 90 million people in African continent and resulted with a minimum estimation of 18 million orphans. It is estimated that the unusual sexual intercourse alone accounts for about 80% of reported cases of HIV infection [1]. HIV infection transforms from an infected person to victim through blood transfusion. This transfusion can be controlled by screening of blood products. Infected blood products are required to be avoided from transfusion so as to control the spread of the disease.

The impact of migration of population on the distribution and spread of HIV/AIDS disease has to be analyzed properly and must be understood clearly. Migration and immigration of the people from one country to another country due to different reasons play a crucial role in the evolution and spread of HIV/AIDS epidemic [2-4]. Economical conditions, war situations and political unrest

are some of the reasons for migration of people. However, it shows that internal and cross border migration of male workers are at greater risk of HIV infection. These workers are more likely to spread the disease on returning back to home [5].

Vertical transmission of HIV/AIDS is also known as Mother to Child transmission (MTCT). It occurs when the virus spreads from an HIV positive woman to her baby. The transmission of the disease from mother to child may occur at different stages viz., in uterus or at the time of birth or after the birth. The risk of transmission in developing countries is around 90%. It is estimated that 220,000 children with exposed to the disease are born each year. Of these about 88,000 are infected without prevention of mother to child transmission and only 2% or 4,400 are infected with prevention of mother to child transmission [6]. It is therefore important to consider and study the effect of vertical transmission in the spread of HIV/AIDS disease.

The study of HIV transmission and the dynamics of the disease have been of a great interest to both applied mathematicians and Biologists. Mathematical modeling has proved to be an important tool in analyzing the spread and control of HIV disease [7-8]. The results of modeling and analysis help to improve understanding of the major contributing factors to the pandemic. Mathematical models have been studied and important inferences have been drawn in case of epidemics like Ebola, Breast cancer, Malaria, Tuberculosis and Influenza [9-14].

Several researchers have developed HIV/AIDS models so as to understand and explain the dynamics and the spread of the disease and succeeded to a large extent. Modeling and Analysis of the spread of AIDS epidemic with immigration of HIV infectives is studied in [1, 15]. A theoretical framework describing the transmission of HIV/AIDS with screening of unaware infective persons is presented in [16-17]. The joint effect of both medical screening and variable inflow of aware and unaware infective immigrants on the disease transmission has been studied by [5]. The spread of the disease due vertical transmission has also been studied by [18].

In this paper, we proposed an improvement of the model [5] that developed a Non-linear mathematical model and studied the effect of screening on the spread of HIV infection in a population with variable inflow of infective immigrants. The model [5] forms the motivation for the present study. Here we have investigated the combined effect of unaware infective immigrants, vertical transmission and aware infective immigrants, on the dynamics of HIV/AIDS. The results are presented graphically and discussed qualitatively in the following sections.

2. Mathematical Model

The combined effects of screening and variable inflow of infective immigrants on the spread of HIV/AIDS in a population of varying size are studied in [5]. The flow diagram of the model and the non linear deterministic model of the problem are given as follows.

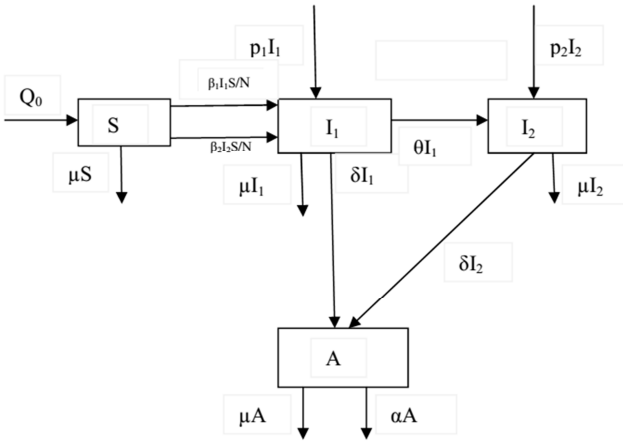

Figure 1. *Flow diagram of the model [5].*

The Non linear ordinary differential equations of model [5] are given as follows:

$$\frac{dS}{dt} = Q_0 - \left(\frac{\beta_1 I_1 + \beta_2 I_2}{N}\right) S - \mu S \quad (1)$$

$$\frac{dI_1}{dt} = p_1 I_1 + \left(\frac{\beta_1 I_1 + \beta_2 I_2}{N}\right) S - (\theta + \delta + \mu) I_1 \quad (2)$$

$$\frac{dI_2}{dt} = p_2 I_2 + \theta I_1 - (\delta + \mu) I_2 \quad (3)$$

$$\frac{dA}{dt} = \delta I_1 + \delta I_2 - (\alpha + \mu) A \quad (4)$$

2.1. Compartmentalization of the People of the Present Model

In this section we have provided compartmentalization of the people. That is, the total population is divided into compartments. We have also described the flow of the people among these compartments. Notations and the description of the model parameters are also included. Flow diagram containing the compartments and flow directions is given for better understanding of the model. A system of non linear ordinary differential equations is constructed that describes the model. Mathematical analysis of the model is conducted and the observations are included.

The mathematical modeling of the spread of HIV / AIDS disease among the population requires the whole human population to be divided in to four classes. The whole of the human population at any time t is a variable and is denoted by $N(t)$. The four classes are as follows: (i) *susceptible class* the population size of this class at any time t is denoted by $\mathrm{S}(\mathrm{t})$. The susceptible human has not yet infected by the disease but likely to get infected in future. (ii) *Unaware infective class* the population size of this class at any time t is denoted by $\mathrm{I}_1(\mathrm{t})$. The unaware infective humans have already infected by the disease but they do not know that they were already infected. (iii) *Aware infective class* the population size of this class at any time t is denoted by $\mathrm{I}_2(\mathrm{t})$. The aware infective humans have already infected by the disease and they know that they were already infected and (iv) *AIDS class* the population size of this class at any time t is denoted by $\mathrm{A}\ (\mathrm{t})$. The AIDS class people are already AIDS patients.

2.2. Flow of the People Among the Compartments

People will join the susceptible compartment S(t) by natural birth. Some of these people will vacate this compartment due to natural deaths and some others will go to $I_1(t)$ compartment after getting infected. The remaining people will stay in the S(t) compartment itself. People of S(t) compartment are likely to get infected by the people of $I_1(t)$ and $I_2(t)$ compartments only. But the people of AIDS compartment A (t), being physically too weak to participate in sexual activities, cannot transfer infection to susceptible people.

In the present study the authors considered that the transfer of HIV from infected people to susceptible people is only by sexual intercourse. Transferring HIV by any other means like sharing needles; blood transfusion etc. is omitted and not considered.

In to $I_1(t)$ compartment some people will enter from S(t) after getting infected, some others will enter by immigrations from other places and some more will enter by vertical transmission. From $I_1(t)$ compartment some people will go to $I_2(t)$ after becoming aware of the infection, some will go to A (t) after conformation of full-fledged AIDS disease, some people will die with natural reasons, and others will stay back in $I_1(t)$ compartment itself.

In to $I_2(t)$ compartment some people will enter from $I_1(t)$ after getting aware of the infection and some others will enter by immigrations from other places. From $I_2(t)$ compartment some people will go to A (t) after conformation of full-fledged AIDS disease, some people will die with natural reasons, and others will stay back in $I_2(t)$ compartment itself.

In to A (t) compartment people will enter from both $I_1(t)$ and $I_2(t)$ compartments after conformation of full-fledged aids disease. From A (t) compartment people will leave when they die naturally or die due to AIDS disease.

2.3. Description of the Model Parameters

We assume that the people are recruited into susceptible class at a constant rate of Q_0. This recruitment into the susceptible class is due to natural births. The people of susceptible class are likely to become infected through sexual contact with the people of $I_1(t)$ and $I_2(t)$ classes. Thus, people from $S(t)$ will go to $I_1(t)$ with a rate of $[(\beta_1 I_1 + \beta_2 I_2)(S/N)]$. Here the parameters β_1 and β_2 are the probabilities per one contact with which the disease transmits to susceptible people by unaware and aware infective humans respectively. Note that in this model we consider $\beta_1 > \beta_2$ .That is, the probability of transferring the disease to susceptible population by unaware infected person is more than by aware infected person. People of $S(t)$ after getting infected will initially go to $I_1(t)$ but not to $I_2(t)$. This is because, all the infected people are assumed to be initially unaware of the infection. Further, the people of $S(t)$compartment are assumed to die naturally with a rate of μ.

People will enter into $I_1(t)$ compartment from $S(t)$ with a rate of $[(\beta_1 I_1 + \beta_2 I_2)(S/N)]$, some others will enter due to immigrations from other places at a rate of p_1 and some others will enter due to vertical transmission at a rate of $(1-\epsilon)\,\phi\, I_1$. It is assumed that the sexual contact between susceptible and unaware infected persons lead to the birth of infected children with a rate of ϕ. Of these newly born but infected children a fraction ϵ dies during the birth due to infection and the remaining complementary fraction $(1-\epsilon)$ will enter into I_1 class. From $I_1(t)$ compartment some people will go to $I_2(t)$ after becoming aware of the disease at a rate of θ and some others will go to $A(t)$ compartment after confirmation of full AIDS disease at a rate of δ_1. People of $I_1(t)$compartment are assumed to die with natural reasons and leave the compartment at a rate of .

People will enter into $I_2(t)$ compartment from $I_1(t)$ after becoming aware of the disease with a rate of θ and some others will enter due to immigrations from other places at a rate of p_2 . People will go to $A(t)$ compartment after confirmation of full AIDS disease at a rate ofδ_2. People of $I_2(t)$compartment are assumed to die with natural reasons and leave the compartment at a rate of μ.

People will enter into $A(t)$ compartment from $I_1(t)$ and $I_2(t)$ compartments at a rate of δ_1 and δ_2 respectively. Further, in this study we assume that $\delta_1 > \delta_2$ since the unaware infected people grow to AIDS much faster than the aware infected people. People of $A(t)$ compartment are assumed to die with natural reasons at a rate of μ anddie with AIDS disease at a rate of α and leave the compartment.

2.4. Flow Diagram of the Model

Here in what follows we have given the flow diagram of the model. The compartments of the model are represented by rectangular boxes. The flow directions of the people among the compartments are represented by directed arrows.

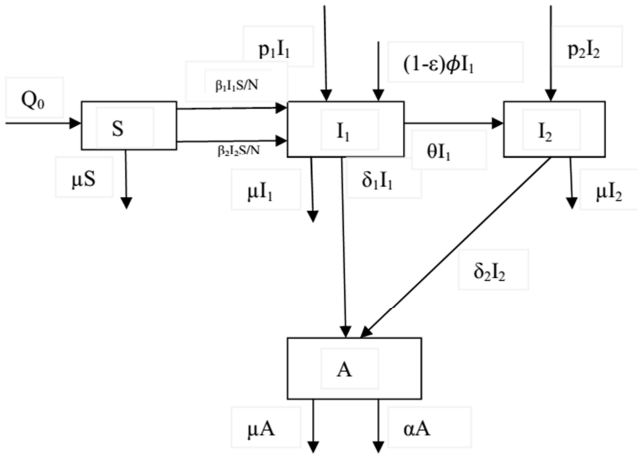

Figure 2. *Flow diagram of the present model.*

2.5. Model Assumptions

We here in the present study develop a mathematical model to describe the population dynamics of HIV / AIDS disease based on the following assumptions:

i. The population under study is heterogeneous and varying with time.
ii. The whole human population is divided in to four classes.

iii. The HIV can only transmitted by the sexual intercourse with infective peoples.
iv. The full blown AIDS class is sexually inactive.
v. All the new infected people are assumed to be initially unaware of the infection
vi. The probability of transferring the disease to susceptible population by unaware infected person is more than by aware infected person i. e. $\beta_1 > \beta_2$.
vii. The unaware infected people grow to AIDS much faster than the aware infected people i. e. $\delta_1 > \delta_2$.

2.6. Model Equations

Based on the assumptions given in Section 2.5 and the flow diagram given in Section 2.4, the dynamics of the HIV / AIDS transmission is governed by a system of Non linear ordinary differential equations as given follows:

$$\frac{dS}{dt} = Q_0 - \left(\frac{\beta_1 I_1 + \beta_2 I_2}{N}\right) S - \mu S \tag{5}$$

$$\frac{dI_1}{dt} = \left(\frac{\beta_1 I_1 + \beta_2 I_2}{N}\right) S + p_1 I_1 + (1-\epsilon)\phi I_1 - (\theta + \delta_1 + \mu) I_1 \tag{6}$$

$$\frac{dI_2}{dt} = p_2 I_2 + \theta I_1 - (\delta_2 + \mu) I_2 \tag{7}$$

$$\frac{dA}{dt} = \delta_1 I_1 + \delta_2 I_2 - (\alpha + \mu) A \tag{8}$$

Here in the system of equations from (5) to (8), the initial conditions are considered to be $S(0) = S_0, I_1(0) = I_{10}, I_2(0) = I_{20}$ and $A(0) = A_0$. Further, in what follows we call the system of these four equations as 'model equations'.

2.7. Mathematical Analysis of the Model

Here we present the mathematical analysis of the model as described by the system (5) to (8). The total population size $N(t)$ given by $N(t) = S(t) + I_1(t) + I_2(t) + A(t)$ implies using the equations (5) to (8) that $dN/dt = (dS/dt) + (dI_1/dt) + (dI_2/dt) + (dA/dt) = Q_0 + p_2 I_2 + (p_1 + (1-\epsilon)\phi) I_1 - \mu N - \alpha A$.

3. Positivity of Solutions

The model equations (5) to (8) are to be epidemiologically meaningful and well posed, we need to prove that all the state variables are non-negative. This requirement is stated as a theorem and provided its proof as follows:

Theorem 1: If $S(0) > 0, I_1(0) \geq 0, I_2(0) \geq 0$ and $A(0) \geq 0$, then the solution region $\{S(t), I_1(t), I_2(t), A(t)\}$ of the system of equations (5) to (8)is always non negative for $t > 0$.

Proof: To show the positivity of the solution of the dynamical system comprising the equations (5) to (8), we have to consider and verify each differential equation and show that their solution is positive.

First let us consider the differential equation (5) of the dynamical system and that can be rewritten as $(dS/dt) + [\mathrm{q} + \mu]S = Q_0$ where $q(t) = [(\beta_1 I_1 + \beta_2 I_2)/N(t)]$. This is a first order linear ordinary differential equation and can be solved to obtain a particular solution as $S(t) = S(0)e^{-Q(t)+Q(0)-\mu t} + \int_0^t Q_0 e^{(Q(s)-Q(t))+\mu(s-t)} ds$. Here the anti – derivative of $q(t)$ is represented by $Q(t)$. It is clear from the solution that $S(t)$ is positive since $S(0) > 0, Q_0 > 0$ and the exponential function always positive and.

Secondly let us consider the differential equation (6) and that can be rewritten as $(dI_1/dt) + [k - (\beta_1 S/N)]I_1 = (\beta_2 I_2 S/N)$ where $k = \theta + \delta_1 + \mu - p_1 - (1-\epsilon)\phi$. This is a first order linear ordinary differential equation and can be solved to obtain a solution as $I_1(t) = I_1(0)e^{-kt+\beta_1 Q(t)-\beta_1 Q(0)} + e^{-kt+\beta_1 Q(t)-\beta_1 Q(0)} \int_0^t (\beta_2 I_2 S/N) e^{ks-\beta_1 Q(s)-\beta_1 Q(0)} ds$. It is clear from this solution that $I_1(t)$ is a nonnegative quantity.

Thirdly, let us consider the differential equation (7) and that can be expressed as $(dI_2/dt) + hI_2 = \theta I_1$ where $h = (\delta_2 + \mu - p_2)$. This is a first order linear ordinary differential equation and can be solved to obtain a particular solution as $I_2(t) = I_2(0)e^{-ht} + e^{-ht} \int_0^t \theta I_1(s) e^{hs} ds$. From this solution we see that $I_2(t)$ is also nonnegative.

Finally let us consider the differential equation (8) and that can be expressed as $(dA/dt) + (\alpha + \mu)A = \delta_1 I_1 + \delta_2 I_2$. This is a first order linear ordinary differential equation and can be solved to obtain a particular solution as $A(t) = A(0)e^{-(\alpha+\mu)t} + e^{-(\alpha+\mu)t} \int_0^t (\delta_1 I_1 + \delta_2 I_2) e^{(\alpha+\mu)s} ds$. From this solution we see that $A(t)$is nonnegative

Boundedness of the solution region.

Here we note that the solution region of the system of model equations (5) to (8) is bounded. Summing up all the four equations from (5) to (8) and assuming the inequality $p_2 I_2 + (p_1 + (1-\epsilon)\phi) I_1 \leq \alpha A$ we obtain $(dN/dt) \leq [Q_0 - \mu N]$. The latter differential inequality has a solution of the form $N(t) \leq [(Q_0/\mu) - N_0 e^{-\mu t}]$ or equivalently it implies that $0 < \mathrm{N(t)} \leq (Q_0/\mu)\ as\ t \to \infty$. Therefore the solutions of system are bounded

4. Stability Analysis of the Model

In this section we identify the equilibrium points of the model developed in this study and provided as a system of equations from (5) to (8). We also analyze their stability conditions and present the results. The system exhibits two types of equilibrium points viz., disease free equilibrium points and endemic equilibrium points.

Disease free equilibrium point.

The disease free equilibrium of the model, (5) to (8), is obtained by setting $(dS/dt) = (dI_1/dt) = (dI_2/dt) = (dA/dt) = 0$. Further at the disease free equilibrium point there are neither infective people nor AIDS patients. That is $I_1 = I_2 = A = 0$. Up on substituting these, (5) implies that $[Q_0 - \mu S] = 0$ or equivalently $S = [Q_0/\mu]$. Thus the disease free equilibrium of the model is given by $E_0 = (Q_0/\mu, 0, 0, 0)$.

Reproduction number R_0.

The reproduction number is defined as the average number of secondary cases produced by a typical infected individual during his or her entire life as infectious or infectious period when introduced or allowed to live in a population of susceptible [19]. We shall now compute the basic

reproduction number $R_0$of the present model using the next generation method [20]. The basic reproduction number is a threshold quantity used to study the spread of an infection disease in epidemiological modeling and it is the spectral radius (i. e. the dominant Eigen value) of the next generation matrix [19]. It is defined as$R_0 = \rho(FV^{-1})$. Here $\rho\ (FV^{-1})$ represents the spectral radius of the matrix FV^{-1} and the matrix is given by $FV^{-1} = \left[(\partial/\partial x_j)F_i\ (x_0)\right]\left[(\partial/\partial x_j)V_i\ (x_0)\right]^{-1}$. Here F_i is the rate of appearance of new infections in the compartment i; V_i is the transfer of individuals in and out of compartment i and E_0 is the disease free equilibrium point. Consequently we obtain $[F_{I1}\quad F_{I2}]^T = [f_1\quad f_2]^T = [q(t)S\quad 0]^T$. Here the superscript T denotes the transpose of a matrix. By linearization approach, the associated matrix F at the disease free equilibrium point E_0 is given by $F = \begin{bmatrix} \frac{\partial f_1}{\partial I_1} & \frac{\partial f_1}{\partial I_2} \\ \frac{\partial f_2}{\partial I_1} & \frac{\partial f_2}{\partial I_2} \end{bmatrix} at\ E_0 = \begin{bmatrix} \beta_1 & \beta_2 \\ 0 & 0 \end{bmatrix}$ and $\begin{bmatrix} v_{I1} \\ v_{I2} \end{bmatrix} = \begin{bmatrix} v_1 \\ v_2 \end{bmatrix} = \begin{bmatrix} k\ I_1 \\ -\ dI_2/dt \end{bmatrix}$. Again by linearization, we get $V = \begin{bmatrix} \frac{\partial v_1}{\partial I_1} & \frac{\partial v_1}{\partial I_2} \\ \frac{\partial v_2}{\partial I_1} & \frac{\partial v_2}{\partial I_2} \end{bmatrix}_{E_0} = \begin{bmatrix} k & 0 \\ -\theta & h \end{bmatrix}$ and $V^{-1} = (kh)^{-1}\begin{bmatrix} h & 0 \\ \theta & k \end{bmatrix}$ and finally $FV^{-1} = (kh)^{-1}\begin{bmatrix} \beta_1 h + \beta_2\theta & k\beta_2 \\ 0 & 0 \end{bmatrix}$.

We find the Eigen values of FV^{-1} by solving the characteristic equation $|FV^{-1} - \lambda I| = 0$ as $\lambda_1 = \frac{\beta_1 h + \beta_2\theta}{kh}$ and $\lambda_2 = 0$. Thus, the spectral radius of FV^{-1} is given by $R_0 = Max\ [\lambda_1, \lambda_2] = \lambda_1$. Further, the reproduction number R_0'in the absence of vertical transmission modeled in [5] is given by $R_0' = \{(\beta_1 h + \beta_2\theta)/[k + (1-\epsilon)\varphi]h\}$.

Here at this stage we point out that $R_0 > R_0^{'}$. This shows that the basic reproduction number of the present model is greater than the one which is obtained from the model modeled without vertical transmission in [5]. This fact implies that HIV/AIDS spreads faster due to vertical transmission from infected mother to child. Hence the birth of infected newly born children by unaware infected immigrants has a significant contribution to propagation of the infection and it keeps the disease endemic in the population.

In order to assess the contribution of unaware and aware infected population on the dynamics of HIV/AIDS, let us divide the reproduction number R_0 of the present modelinto the reproduction numbers of both unaware R_{0U} and aware R_{0A}infected populations independently i. e. $R_0 = R_{0U} + R_{0A}$. Here we further observe that $R_{0U} > R_{0A}$.

$$R_{0U} = \frac{\beta_1}{k} \text{and } R_{0A} = \frac{\beta_2\theta}{kh} \tag{9}$$

We see from (9) that the contribution of vertical transmission$[(1-\epsilon)\phi]$ from infected mother to child has a significant effect on the increment of the reproduction numbers of both unaware R_{0U} and aware R_{0A} infected populations. Therefore the transmission of HIV infection increases by aware and unaware infected populations through vertical transmission. From the fact $R_{0U} > R_{0A}$ it can be understood that the unaware infectives contribute more to the transmission than the aware infectives. We now investigate the local stability of the disease free equilibrium pointE_0.

Theorem – 1 The disease free equilibrium point E_0 of the system of ordinary differential equations (5) to (8) is locally asymptotically stable if $R_0 < 1$ and unstable if$R_0 > 1$.

Proof Initially at t=0, $S\ (0) > 0, I_1\ (0) \geq 0, I_2\ (0) \geq 0$this means initially there is no AIDS patient. Hence, we only consider the subsystem of three equations (5), (6) and (7). The Jacobian matrix associated with the subsystem equations at the disease free equilibrium point $E_0 = (Q_0/\mu\quad, 0\quad, 0)$ is given by:

$$J\ (E_0) = \begin{bmatrix} -\mu & -\beta_1 & -\beta_2 \\ 0 & -k + \beta_1 & \beta_2 \\ 0 & \theta & -h \end{bmatrix}$$

We can expand the characteristic equation$|J(E_0) - \lambda I| = 0$ as $f\ (\lambda) = (-\mu - \lambda)\ (\lambda^2 + B\lambda + C) = 0.$ Here $B = (-k + \beta_1 - h)$ and $C = (hk - \beta_1 h - \beta_2\theta)$. We note that the first root of characteristic equation is$\lambda_1 = -\mu$.

If $R_0 < 1 or\ equivalently\ [(\beta_1 h + \beta_2\theta)/hk] < 1$ which is again equivalent to $C > 0$ and hence $(k - \beta_1 + h) > 0$, therefore $B > 0$ then by applying Routh – Hurwitz criteria E_0 is locally asymptotically stable.

If $R_0 > 1$ then the characteristic equation will have positive Eigen value so E_0 is unstable.

Endemic Equilibrium Point.

Similarly here we also consider the subsystem equations (5), (6) and (7). At the endemic equilibrium point $E^*$the disease persists or exists. It is given by $E^* = (S^*, I_1^*, I_2^*)$. We set each right hand side in subsystem equations to zero and express each dependent variable in terms of I_1^* at the equilibrium point and we obtain

$$S^* = [N\ h\ k/(\beta_1 h + \beta_2\theta)] = N/R_0 \tag{10}$$

$$I_1^* = [(Q_0 - \mu S^*)/k] = (Q_0/k)[1 - (\mu S^*/Q_0)] = (Q_0/k)[1 - (1/R_0)] \tag{11}$$

$$I_2^* = [\theta\ I_1^*/h] = (\theta\ Q_0/hk)[1 - (1/R_0)] \tag{12}$$

From (11), we see that I_1^* will be positiveif $R_0 > 1$. We also note that $E^* = (S^*, I_1^*, I_2^*)$ is a unique endemic equilibrium point which exists and is positive whenever $R_0 > 1, k > 0$ andh > 0. We now investigate the local stability of the endemic equilibrium point E^*. For the investigation the Lemma – 1 as stated below is useful [21].

Lemma -1 Let M is a $3\ by\ 3$ real matrix. If $tr\ (M), det\ (M)$ and $det\ (M^{[2]})$ are all negatives then all the Eigen values of the matrix M have negative real parts.

Definition – 1 (The second additive compound Matrix).

Let $M = (a_{ij})$ be an $n \times n$ real matrix. The second additive compound of M is the matrix denoted by $M^{[n]} = (b_{ij})$ and defined by (i) $M^{[2]} = [a_{11} + a_{22}]$ for $n = 2$ and (ii) $M^{[2]} = \begin{pmatrix} a_{11} + a_{22} & a_{23} & -a_{13} \\ a_{32} & a_{11} + a_{33} & a_{12} \\ -a_{31} & a_{21} & a_{22} + a_{33} \end{pmatrix}$ for $n = 3$.

Theorem – 2 The positive endemic equilibrium pointE^* of the system of equations (5) to (8) is locally asymptotically stable if $R_0 > 1$.

Proof Here we also consider the subsystem of three equations (5) to (7). Linearization of this subsystem of three equations at the

$$J(E^*) = \begin{bmatrix} -r-\mu & -(\beta_1/N)S^* & -(\beta_2/N)S^* \\ r & -k+(\beta_1/N)S^* & (\beta_2/N)S^* \\ 0 & \theta & -\mathrm{h} \end{bmatrix}$$

Here $r = \{[\beta_1 {I_1}^* + \beta_2 {I_2}^*]/N\}$.

We now show that the trace of the matrix $J(E^*)$is negative quantity. Note that the sum of the diagonal elements of a square matrix is known as trace of that matrix. It can be verified that the trace of the matrix $Tr\,J(E^*) = (-r-\mu-k+(\beta_1/N)S^*-\mathrm{h}) = (-r-\mu-k+hk\beta_1/h\beta_1+\theta\beta_2-\mathrm{h})$is a negative quantity since$(\beta_1 h) < [\beta_1 h + \beta_2\theta]$. Hence we observe that $Tr[J(E^*)] < 0$.

We now show that determinant of the matrix $J(E^*)$is a negative quantity. The determinant of the matrix $[J(E^*)]$can be obtained as $det[J(E^*)] = (-r-\mu)[hk-(h\beta_1/N)S^*-(\beta_2/N)\theta S^*]-r[(h\beta_1/N)S^*+(\beta_2/N)\theta S^*]$. But, the first term vanishesbecause $[hk-(h\beta_1/N)S^*-(\beta_2/N)\theta S^*] = (hk)\{1-(S^*/N)[(h\beta_1+\theta\beta_2)/\mathrm{h}\,k]\} = (hk)[1-(NR_0/R_0N)] = 0$. Therefore, $det[J(E^*)] = -r\left[\frac{\beta_1 h}{N}S^* + \frac{\beta_2\theta}{N}S^*\right] < 0$ We now show that the determinant of the matrix $J^{[2]}(E^*)$ is a negative quantity. We construct the second additive compound matrix $J^{[2]}(E^*)$as

$$J^{[2]}(E^*) = \begin{pmatrix} -r-\mu-k+(\beta_1/N)S^* & (\beta_2/N)S^* & (\beta_2/N)S^* \\ \theta & -r-\mu-\mathrm{h} & -(\beta_1/N)S^* \\ 0 & r & -k-\mathrm{h}+(\beta_1/N)S^* \end{pmatrix}$$

Now we obtain the determinant as $\det\left[J^{[2]}(E^*)\right] = [a+(\beta_1/N)S^*][bc+(\beta_1/N)rS^*]+(\beta_2/N)\theta S^*[\mathrm{r}-c]$. Here we have introduced the notations as$a = -r-\mu-k$, $b = -r-\mu-\mathrm{h}$ and $c = -k-\mathrm{h}+(\beta_1/N)S^*$. Thus $\det\left[J^{[2]}(E^*)\right] = -(r+\mu+k)[(r+\mu+\mathrm{h})(k+\mathrm{h})+(r\beta_1/R_0)]+(r+\mathrm{h}+k)(\theta\beta_2/R_0) = -(r+\mu+k)(r+\mu+\mathrm{h})(k+\mathrm{h})-(r+\mu+k)(r\beta_1/R_0)+(r+\mathrm{h}+k)(\theta\beta_2/R_0)$ and is a negative quantity. Therefore, $\det\left[J^{[2]}(E^*)\right] < 0$.

Thus, by statement of Lemma – 1, the equilibrium point$E^*$is locally asymptotically stable, when $R_0 > 1$. Hence, Theorem – 2is proved.

5. Numerical Simulation

Here for numerical simulation, we consider the scaling of variables and parameters in the system of equations (5) to (8) as follows: $s = S/N, i_1 = I_1/N, i_2 = I_2/N$, $a = A/N$ and $b = Q_0/N$. Then, we get $[(1/N)(dN/dt)] = [(Q_0/N)+p_2 i_2+(p_1+(1-\epsilon)\phi)i_1-\mu-\alpha a]$. After some simplifications the system of equations in terms of the scaled variables takes the following form:

$$ds/dt = b-bs-\beta_1 i_1 s-\beta_2 i_2 s-s\eta+p_2 i_2 s+\alpha a s \quad (13)$$

$$di_1/dt = \beta_1 i_1 s+\beta_2 i_2 s-(\theta+\delta_1)i_1+\eta\,[1+i_1]-\,(b+p_1 i_1+p_2 i_2-\alpha a)i_1 \quad (14)$$

$$di_2/dt = \theta i_1+p_2 i_2-\delta_2 i_2-\,(b+(\eta+p_2 i_2-\alpha a)i_2 \quad (15)$$

$$da/dt = \delta_1 i_1+\delta_2 i_2-\alpha a-\,(b+\eta+p_2 i_2-\alpha a)a \quad (16)$$

Here we have used $\eta = p_1 i_1+(1-\epsilon)\phi i_1$. To study the dynamical behavior of the model equations (13) to (16) numerically; the system of equations is integrated by using ode45 of Matlab. The following values for the parameters are selected: $b = 0.04, \beta_1 = 0.9, \beta_2 = 0.7, \theta = 0.3, \delta_1 = 0.3, \delta_2 = 0.02, \alpha = 0.9, \epsilon = 0.2, \phi = 0.03, p_1 = 0.1$ and$p_2 = 0.2$. The initial conditions selected are$s(0) = 0.65$, $i_1 = 0.20$, $i_2 = 0.10$ and $a(0) = 0.05$ for the period of 30 years.

From the Fig – 1 The distribution of the population with time is shown for all classes. It is found that susceptible population decreases with time due to inflow of infective immigrants and vertical transmission leading to an increase in the rate on infection. Unaware infective class decreases with time and then reaches its equilibrium position. The aware infective class increases with time due to screening. We also observe that the AIDS population decreases

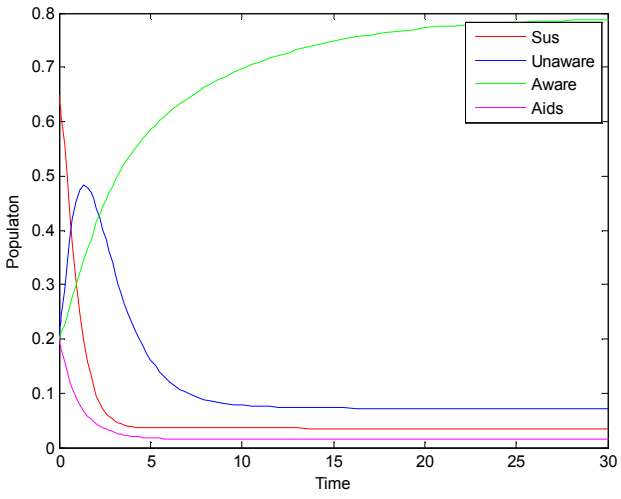

Figure 3. *Variation of population in different classes for the given parametric values.*

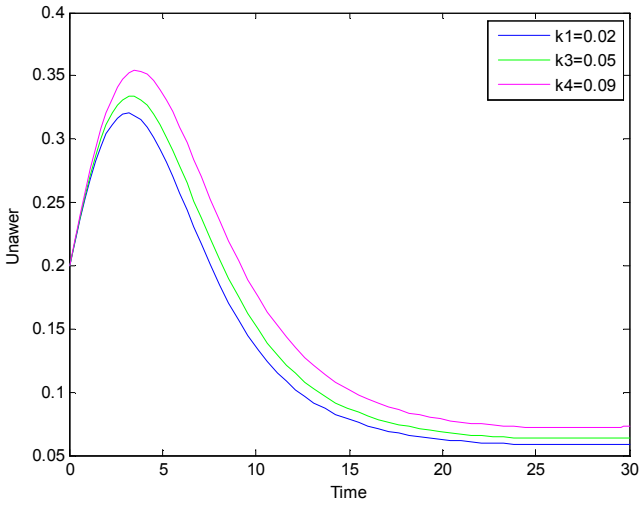

Figure 4. *The effect of the unaware infective immigrants on unaware infective class.*

Fig-2 shows the effect of the immigration rate of unaware infective immigrants on unaware infective class. It is found unaware infective population increase initially but as time goes on it decreases, this simply means that the proportion of unaware HIV infective is becoming aware infective through screening and it will come to its equilibrium position. More over we can see that as the rate of unaware infective immigrants increases, the unaware infective population also increase. This will result in increasing on the transmission of HIV/AIDS.

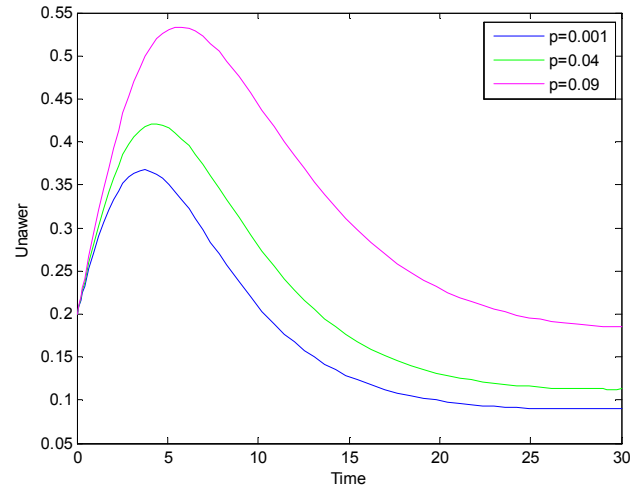

Figure 5. *The effect of the vertical transmission on unaware infective class.*

Fig-3, shows the effect of the birth rate of new born (vertical transmission) on unaware infective class. It is found unaware infective population increase initially but as time goes on it decreases, this simply means that the proportion of unaware HIV infective is becoming aware infective through screening and it will come to its equilibrium position. More over we can see that as the rate of new born increases then unaware infective population also increase. This will result in increasing on the transmission of HIV/AIDS.

Fig-4, shows the effect of the immigration rate of unaware infective immigrants and the birth rate of new born on unaware infective class. It is found unaware infective population increase initially but as time goes on it decreases, this simply means that the proportion of unaware HIV infective is becoming aware infective through screening and it will come to its equilibrium position. More over we can see that as the rate of unaware infective immigrants and the birth rate of new born increases, the unaware infective population also increase. This will result in increasing on the transmission of HIV/AIDS.

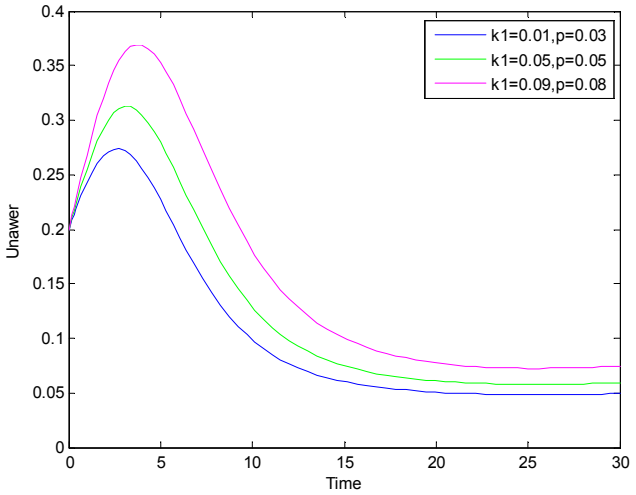

Figure 6. *The combined effect of the unaware infective immigrants and vertical transmission on unaware infective class.*

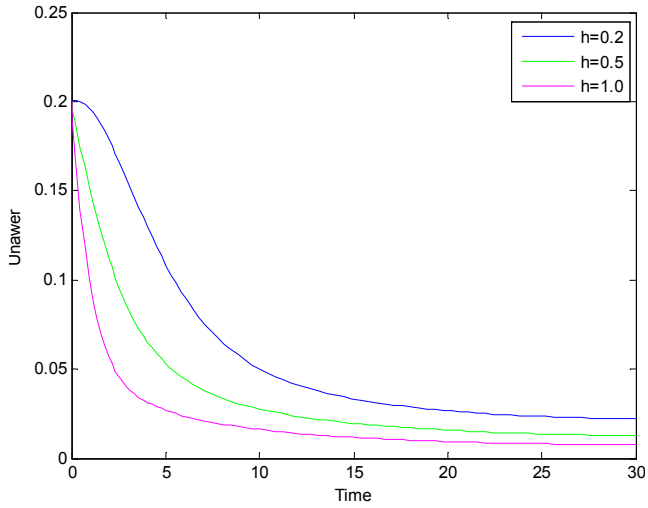

Figure 7. *The effect of screening on unaware infective class.*

Fig-5 shows the effect of screening rate, we observe that as the rate of screening increases, the unaware infective population decreases because of the transfer of some peoples to aware infective class as expected, further it reduces the spread of the disease.

Fig-6 shows the effect of screening rate, we observe that as the rate of screening increases, the aware infective population also increases as expected.

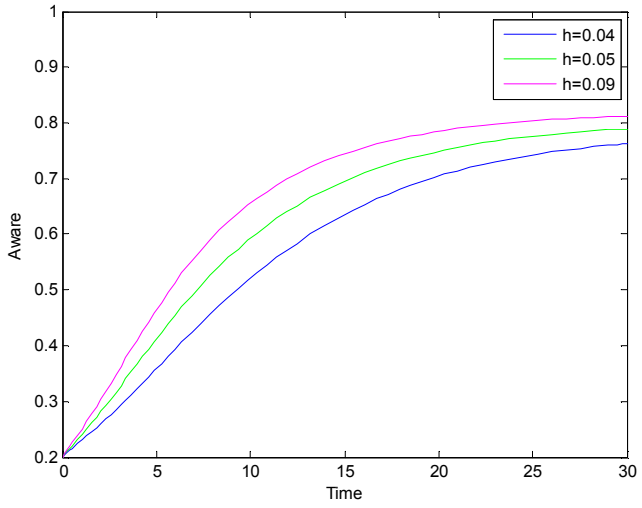

Figure 8. *The effect of screening on Aware infective class.*

6. Conclusions

In this paper, we proposed an improvement of the model [5], that is to show the combined effect of unaware infective immigrants, vertical transmission and aware infective immigrants on the dynamics of HIV/AIDS. A non-linear differential equation was formulated to represent the model. The stability analysis on the model shows that the disease free equilibrium point (E_0) is shown to be locally asymptotically stable when $R_0 < 1$ and the positive endemic equilibrium point (E^*) is shown to be locally asymptotically stable when $R_0 > 1$.

In this paper we also point out that the basic reproduction number R_0 of the present model is greater than the basic reproduction number (R_0') obtained from the model, modeled without vertical transmission in [5]. This fact implies that HIV/AIDS spreads more faster due to vertical transmission from infected mother to child. Results from Numerical simulation show that as the rate of unaware infective immigrants and the birth rate of new born increases, the unaware infective population also increase. This will result in increasing on the transmission of HIV/AIDS.

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