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Existence and Uniqueness of Solution of an HIV/AIDS Model Considering Counseling, Vaccination and Antiretroviral Therapy (ART)

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Abstract
The control of HIV/AIDS is not yet over. Recently, the main method of control is the use of counseling and antiretroviral therapy (ART). In this study, a mathematical model of HIV/AIDS transmission dynamics considering counseling, vaccination and ART is formulated and the existence of its unique solution is investigated.
Key words: HIV/AIDS, Mathematical Model, Existence and Uniqueness of Solution, Counselling, Vaccination, Antiretroviral Therapy

1. Introduction

For more than three decades now, the human immunodeficiency virus (HIV) which causes acquired immune deficiency syndrome (AIDS) has remained one of the world's serious health challenges. Since the initial reporting of the AIDS pandemic in 1981 in the United States of America (USA) by the Centers for Disease Control and prevention (CDC, 1982), the worldwide spread of the pandemic has been far reaching. Global and regional estimates of HIV have been provided by the Joint United Nations Programme on HIV/AIDS (UNAIDS) and the World Health Organization (WHO) since the late 1980s and country specific estimates since 1996 (UNAIDS, 2009; Garcia-Calleja et al., 2006). While the early 1980s AIDS cases were confined mostly to the homosexual men, hemophiliacs, and intravenous (IV) drug users in North America and Europe (CDC, 1982), today there is no geographical area, class, and cultural group of the world untouched by this pandemic (Koob and Harvan, 2003). It is common to the young and adults. Individuals aged 15 years and above are the most susceptible group. This is because they are more sexually active.

HIV is associated with severe break down of the immune system of the affected person, rendering the body to be immune-deficient, resulting into catastrophic susceptibility of the infected person to opportunistic infections like Tuberculosis (TB), Pneumonia, meningitis, cancers and the gastrointestinal tract infections, which may result into illness and death (Putzel, 2003; Mugisha, 2005). Without treatment, the average survival time after infection with HIV is estimated to be 9 to 11 years, depending on the HIV subtype (UNAIDS/ WHO, 2007).

The African Continent is the worst hit by the HIV pandemic, with Sub- Saharan Africa remaining the most severely affected, with nearly 1 in every 20 adults (4.9%) living with HIV and accounting for 69% of the people living with HIV worldwide (UNAIDS, 2012). The impact of the epidemic has also badly affected households, schools, working places and economies (UNAIDS, 2008; AVERT, 2010).

After Sub–Saharan Africa, the regions most heavily affected are the Caribean and Eastern Europe and Central Asia, where 1.0% of adults were living with HIV in 2011 (UNAIDS 2012).

Since the start of the epidemic in 1981, about 75 million people have become infected with HIV; and out of this number, about 36 million people have died of AIDS – related illnesses. In 2012 alone, about 1.6 million people died from AIDS - related causes worldwide (UNAIDS, 2013). Similarly, as at the end of 2012, surveillance data compiled by the Joint United Nations Programme on HIV/AIDS estimates that about 35.3 million people were living with HIV globally (UNAIDS, 2013); although the burden of the epidemic continues to vary considerably between countries and regions.

Although the number of people newly infected with HIV is declining worldwide, the number of people (adults and children) who acquired HIV infection in 2012 was about 2.3 million (UNAIDS, 2013), lower than that of 2011, which stood at about 2.5 million people (UNAIDS, 2012). The sharpest declines in the number of people acquiring HIV infection have occurred in the Caribean (42%) and Sub-Saharan Africa (25%). Despite these gains, Sub–Saharan Africa accounted for 71% of the adults and children newly infected in 2012 (UNAIDS, 2013), underscoring the importance of continuing and strengthening prevention efforts in the region and other regions of the world.

Global solidarity in the HIV/AIDS response during the past decade has continued to generate extraordinary health gains, with the emergence of powerful new tools to prevent people from becoming infected and from dying from AIDS - related causes. Although much of the news on the successes against HIV/AIDS is encouraging, challenges remain. National epidemics continue to expand in many parts of the world. Further, declines in the numbers of children dying from AIDS-related causes and acquiring HIV infection, although substantial, need to be accelerated to achieve global AIDS target.

The virus (HIV) can be transmitted in the following ways: horizontal transmission (from one adult to another adult through heterosexual, bisexual or homosexual contact), vertical transmission (that is, from an infected mother to fetus), or during childbirth, or during breastfeeding, and exposure to contaminated needles/blood products. However, large scale prevention of Mother to Child Transmission Programmes have greatly reduced the number of new infections globally through this route (Omar and Naresh, 2010; UNAIDS, 2008). Out of these modes of HIV transmission, heterosexual contact is the major mode of transmission, contributing about more than 75% of all the world's HIV infections (UNAIDS, 2009).

Presently, there is no known medical cure for HIV infection nor is there a vaccine to prevent HIV infection. The most plausible HIV control measure involves prevention. These include condom use, abstinence, male circumcision and faithfulness. Condom use and male circumcision were shown to have a big impact on HIV with male circumcision alone reducing HIV transmission rate by 37% (Williams et al., 2006; Mukandavire et al., 2007). Treatment of HIV consists of a combination of antiretroviral therapies (ARTs). ART blocks the replication of the virus and thus increases the life-span of HIV-infected individuals. It does not lead to viral eradication within individuals and hence does not cure (Miranda et al., 2007; Velasco-Hernandez et al., 2002). The effect of ART on HIV largely depends on the stage of infection at which treatment is initiated, levels of coverage, and the scale and stage of HIV epidemic that the community is experiencing. ART has been shown to have a big impact on HIV if the coverage is high (Velasco-Hernandez et al., 2002; Udoo, 2010).

Presently, extensive researches for the discovery of anti-HIV preventive and therapeutic vaccines are currently going on in different parts of the world. However, of the over 190 vaccine trials that have been completed to date against HIV/AIDS disease (EGPAF, 2013), only the results from the phase-3 clinical trials in Thailand of an anti-HIV preventive vaccine was released in late 2009 (UNAIDS, 2010; Kai Sun et. al., 2010). It revealed 31.2% reduction in the risk of HIV infection, making this the first HIV vaccine to have a statistically significant effect. The results showed that this vaccine may be valuable in a community setting with largely heterosexual risk (Kai Sun et. al., 2010).

The spreading of diseases has been the subject of intense research for some time now. On the other hand, epidemiologists have developed mathematical models for the spread of infectious diseases in human populations that can be used to gain insights into the spread and control of specific infectious diseases and to design control/prevention, immunization and vaccination policies (see, for example, Gomez-Gardeńes et al., 2008; Anderson et al., 1992; Daley, Gani et al., 1999; Murray, 2002) dating back to the pioneering work of Bernoulli (1760) and the likes of Ross (1911), Kermack and McKendrick (1927) and others (see, for example, Anderson and May, 1991; Hethcote, 2000). The basic reference is the book by Bailey (1957), which contains a description of both stochastic and deterministic models (Hethcote and Waltman, 1973). According to Bailey (1975), disease modeling started as far back as the ancient Greeks with the epidemic of Hippocrates [459 – 377 BC].

In this direction, mathematical models for the control of HIV/AIDS have been formulated as far back as 1987, when Medley et al. (1987), developed simple mathematical functions for the growth in the number of individuals who will ultimately develop AIDS and for the distribution of incubation period of those individuals. Other models for the control of HIV include the following aspects: random screening, contact tracing, use of the condom etc. (see, for example, Greenhalgh et al., 2001; Hyman and Stanley, 2003; Kimbir and Aboiyar, 2003; Kimbir, 2005; Kimbir et al., 2006). Mathematical models to investigate the effect of treatment and vaccination on the spread of HIV/AIDS can be found (see, for example, Swanson et al., 1994; Velasco-Hernandez and

Hsieh, 1994; Hsieh, 1996; Yang and Ferreira, 1999; Hsu-Schmitz, 2000; Gumel et al., 2002; Corbett et al., 2004; Kgosimore and Lungu, 2004; Kimbir and Oduwole, 2008; Udoo, 2010).

An important aspect of controlling HIV/AIDS is the combined strategies of Counseling, vaccination and ART. Evidence for counseling and ART modelling studies has already been reported (see, for example, Kimbir and Oduwole, 2008; Udoo, 2010). In particular, Udoo (2010) proposed and studied a deterministic mathematical model of HIV/AIDS transmission dynamics considering counseling and antiretroviral therapy (ART) on a heterosexual (two-sex) population. The analysis of his model, qualitatively and numerically, revealed that the control and eradication of HIV/AIDS in heterosexual populations is feasible and is dependent on the net transmission rates of infection, the effectiveness of counseling and efficacy of ART, and the proportion of infected people receiving ART for each sex. In this study, however, the counselling and ART model by Udoo (2010) is being extended by incorporating vaccination of susceptible males and females against the acquisition and spread of HIV infection in heterosexual populations; and the existence of its unique solution is investigated. Vaccination has been found to have induced permanent immunity against infectious diseases, like Measles, Smallpox, Rubella, to name but a few. Moreover, it is believed that a vaccine against HIV/AIDS, even one that is partially effective, could have a tremendous impact on the control of the infection in developing countries (IAVI, 2011; Anzala, 2012). Furthermore, a number of mathematical models have been developed specifically to estimate the impact of a vaccine on the AIDS epidemic (IAVI, 2005).

This study is organized as follows. The model assumptions, formulation and the basic properties of the model are presented in the following subsections. The proof of the existence and uniqueness of solutions is carried out, and the concluding remarks in section 2.

1.1 Assumptions of the Model

The following assumptions are made for our modeling activity in this research.

i) The population is a homogeneously-mixing heterosexual one.
ii) Recruitment into the infected sub-populations is through heterosexual contacts only.
iii) An effective proportion each of susceptible males and females are vaccinated against HIV infection.
iv) A proportion each of infected males and females receive ART.
v) Infected individuals apart from dying naturally, die due to the disease.
vi) No recovery from the disease, that is, infected individuals remain infected till death as the disease has no known medical cure for now.
vii) Individuals in the different sub-populations are actively engaged in reproduction.
viii) Vertical transmission and Age-structure are ignored.

1.2 Formulation of the Model: Parameters and Variables of the Model

The functions (parameters/variables) used in the model are defined below.

$S_m(t)$ = Number of susceptible males at time t;

$S_f(t)$ = Number of susceptible females at time t;

$V_m(t)$ = Number of vaccinated susceptible males at time t;

$V_f(t)$ = Number of vaccinated susceptible females at time t;

$I_m(t)$ = Number of infected males at time t;

$I_f(t)$ = Number of infected females at time t;

$R_m(t)$ = Number of infected males receiving ART at time t;

$R_f(t)$ = Number of infected females receiving ART at time t;

$N_m(t) = S_m(t) + V_m(t) + I_m(t) + R_m(t)$ = Total population of males at time t;

$N_f(t) = S_f(t) + V_f(t) + I_f(t) + R_f(t)$ = Total population of females at time t;

$B_m(t)$ = The rate at which males are infected per unit time (incidence rate);

$B_f(t)$ = The rate at which females are infected per unit time (incidence rate);

b = Natural birth rate for both sexes, b >0;

μ = Natural death rate for both sexes, μ >0;

α_o = Death rate of infected individuals not receiving ART of both sexes;

α = Death rate of infected individuals receiving ART of both sexes, $\alpha_o > \alpha$;

δ_m = The effective proportion of vaccinated susceptible males per unit time;

δ_f = The effective proportion of vaccinated susceptible females per unit time;

c_m = Average number of sexual contacts by infected males with females per unit time;

c_f = Average number of sexual contacts by infected females with males per unit time;

c_m^* = Average number of sexual contacts by infected males receiving ART with females per unit time;

c_f^* = Average number of sexual contacts by infected females receiving ART with males per unit time;

β_m = Probability of transmission by an infected male not receiving ART;

β_f = Probability of transmission by an infected female not receiving ART;

β_m^* = Probability of transmission by an infected male receiving ART;

β_f^* = Probability of transmission by an infected female receiving ART;

σ_m = Proportion of infected males receiving ART per unit time;

σ_f = Proportion of infected females receiving ART per unit time;

T = Maximum life span after infection;

k = Efficacy of ART.

1.3 Flow diagram

As a consequence of the model assumptions, the model will be of eight compartments. These compartments and the movement of individuals from one compartment to the other are presented in figure 1. All parameters/variables are as defined in sub-section 1.2.

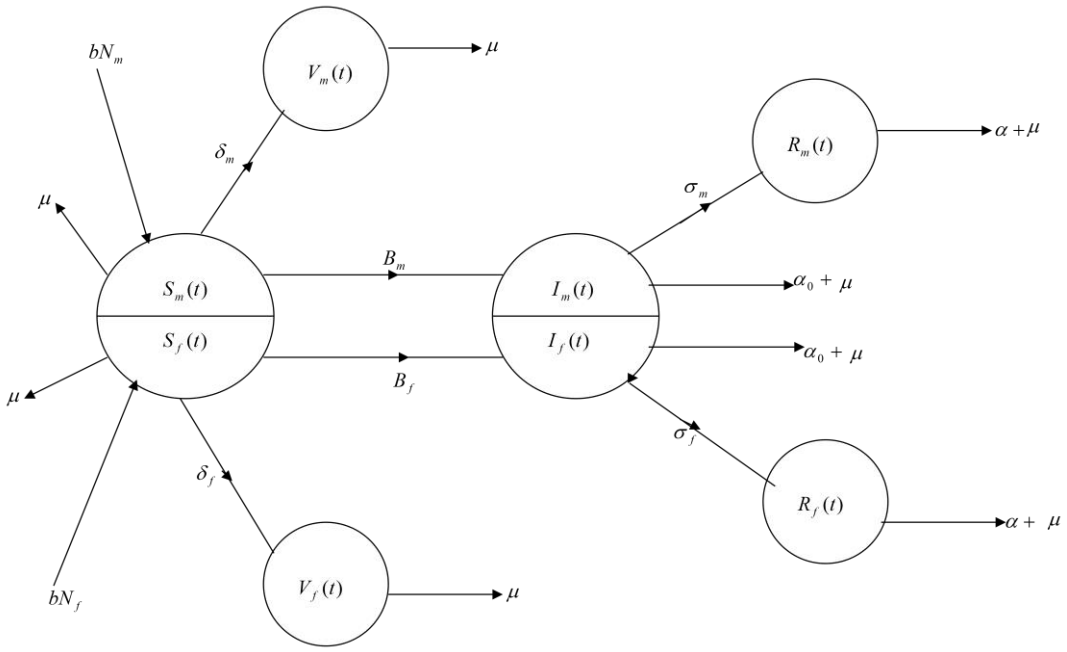

Figure 1. The Model Flow Diagram

1.4 Model Equations

Using the flow diagram we formulate the following model equations:

$$S_m^{'} = bN_m - B_m S_m - (\mu + \delta_m)S_m \quad (1.1)$$

$$S_f^{'} = bN_f - B_f S_f - (\mu + \delta_f)S_f \quad (1.2)$$

$$I_m^{'} = B_m S_m - (\mu + \alpha_0 + \sigma_m)I_m \quad (1.3)$$

$$I_f^{'} = B_f S_f - (\mu + \alpha_0 + \sigma_f)I_f \quad (1.4)$$

$$V_m^{'} = \delta_m S_m - \mu V_m \quad (1.5)$$

$$V_f^{'} = \delta_f S_f - \mu V_f \quad (1.6)$$

$$R_m^{'} = \sigma_m I_m - (\mu + \alpha) R_m \tag{1.7}$$

$$R_f^{'} = \sigma_f I_f - (\mu + \alpha) R_f \tag{1.8}$$

where

$$N_m = S_m + I_m + V_m + R_m \tag{1.9}$$

$$N_f = S_f + I_f + V_f + R_f \tag{1.10}$$

$$B_m = \frac{c_m \beta_f I_f + c_m^* \beta_f^* R_f}{N_f} \tag{1.11}$$

$$B_f = \frac{c_f \beta_m I_m + c_f^* \beta_m^* R_m}{N_m} \tag{1.12}$$

$$\alpha = \alpha_0 e^{-KT} < \alpha_0 \tag{1.13}$$

1.5 Model Equations in Proportions

The above model equations are now transformed into proportions. These equations into proportions have biological meaning, as they define prevalence of infection. They are also the governing equations of the model.

Let,

$$s_m = \frac{S_m}{N_m}, i_m = \frac{I_m}{N_m}, v_m = \frac{V_m}{N_m}, r_m = \frac{R_m}{N_m}, \tag{1.14}$$

and

$$s_f = \frac{S_f}{N_f}, i_f = \frac{I_f}{N_f}, v_f = \frac{V_f}{N_f}, r_f = \frac{R_f}{N_f}, \tag{1.15}$$

From the first equation in (1.14), we have

$$s_m^{'} = \frac{1}{N_m}\left[S_m^{'} - s_m N_m^{'} \right]$$

$$= \frac{1}{N_m}\left[bN_m - B_m S_m - (\mu + \delta_m) S_m - s_m \left\{ (b - \mu) N_m - \alpha_0 I_m - \alpha R_m \right\} \right]$$

or

$$s_m^{'} = b - \left(c_m \beta_f i_f + c_m^* \beta_f^* r_f \right) s_m - (b + \delta_m) s_m + \alpha_0 i_m s_m + \alpha r_m s_m. \tag{1.16}$$

A similar approach gives the following equations in proportions,

$$i_m^{'} = \left(c_m \beta_f i_f + c_m^* \beta_f^* r_f \right) s_m - (b + \alpha_0 + \sigma_m) i_m + \alpha_0 i_m^2 + \alpha r_m i_m, \tag{1.17}$$

$$v_m^{'} = \delta_m s_m - b v_m + (\alpha_0 i_m + \alpha r_m) v_m \tag{1.18}$$

$$r_m^{'} = \sigma_m i_m - (b+\alpha) r_m + \alpha_0 i_m + \alpha r_m^2 . \tag{1.19}$$

$$s_f^{'} = b - \left(c_f \beta_m i_m + c_f^* \beta_m^* r_m\right) s_f - \left(b + \delta_f\right) s_f + \alpha_0 i_f s_f + \alpha r_f s_f , \tag{1.20}$$

$$i_f^{'} = \left(c_f \beta_m i_m + c_f^* \beta_m^* r_m\right) s_f - \left(b + \alpha_0 + \sigma_f\right) i_f + \alpha_0 i_f^2 + \alpha r_f i_f , \tag{1.21}$$

$$v_f^{'} = \delta_f s_f - b v_f + \left(\alpha_0 i_f + \alpha r_f\right) v_f , \tag{1.22}$$

$$r_f^{'} = \sigma_f i_f - (b+\alpha) r_f + \alpha_0 i_f + \alpha r_f^2 . \tag{1.23}$$

Since

$$s_m + i_m + v_m + r_m = 1, \tag{1.24}$$

and

$$s_f + i_f + v_f + r_f = 1, \tag{1.25}$$

we have the following governing equations of the model in proportions:

$$\begin{aligned}
i_m^{'} &= (c_m \beta_f i_f + c_m^* \beta_f^* r_f)(1 - i_m - v_m - r_m) - i_m \left[b + \sigma_m - \alpha_0 (1 - i_m) - \alpha r_m\right] \\
i_f^{'} &= (c_f \beta_m i_m + c_f^* \beta_m^* r_m)(1 - i_f - v_f - r_f) - i_f \left[b + \sigma_f - \alpha_0 (1 - i_f) - \alpha r_f\right] \\
v_m^{'} &= \delta_m (1 - i_m - v_m - r_m) - v_m \left[b - (\alpha_0 i_m + \alpha r_m)\right] \\
v_f^{'} &= \delta_f (1 - i_f - v_f - r_f) - v_f \left[b - (\alpha_0 i_f + \alpha r_f)\right] \\
r_m^{'} &= \sigma_m i_m - r_m \left[b - \alpha_0 i_m + \alpha (1 - r_m)\right] \\
r_f^{'} &= \sigma_f i_f - r_f \left[b - \alpha_0 i_f + \alpha (1 - r_f)\right].
\end{aligned} \tag{1.26}$$

2. Existence and Uniqueness of Solution of the Model

In this section we establish conditions for the existence and uniqueness of a solution of our model. We shall apply Picard's theorem to achieve this.

Theorem 2.1: Picard's Theorem

Suppose

$$y' = f(t, y), y(t_0) = y_0 \tag{2.1}$$

is given system of ordinary differential equations and suppose $f(t, x)$ is continuous and satisfies a Lipschitz condition in the closed and bounded domain $\|x - x_0\| \le \varphi, \ \|t - t_0\| \le \tau$. Let $\|f(t, x)\| \le M$ there. Then the IVP (2.1) has a unique solution in the interval $\|t - t_0\| \le h$, where $h = \min\left\{\tau, \varphi/M\right\}$.

For more on Picard's theorem, see for example, Hu and Li (2004) and Muscat (2008).

Consider our transformed system of equations (1.26) above. Let

$$x = \left[i_m, i_f, v_m, v_f, r_m, r_f \right]^T,$$
$$f(x) = \left[f_1(x), f_2(x), f_3(x), f_4(x), f_5(x), f_6(x) \right]^T, \tag{2.2}$$

where, on expanding each equation in the system of equations (1.26), we get

$$f_1(t,x) = c_m\beta_f i_f + c_m^*\beta_f^* r_f - c_m\beta_f i_f i_m - c_m^*\beta_f^* r_f i_m - c_m\beta_f i_f v_m - c_m^*\beta_f^* r_f v_m - c_m\beta_f i_f r_m$$
$$-c_m^*\beta_f^* r_f r_m - bi_m - \sigma_m i_m + \alpha_0 i_m - \alpha_0 i_m^2 + \alpha r_m i_m,$$

$$f_2(t,x) = c_f\beta_m i_m + c_f^*\beta_m^* r_m - c_f\beta_m i_m i_f - c_f^*\beta_m^* r_m i_f - c_f\beta_m i_m v_f - c_f^*\beta_m^* r_m v_f - c_f\beta_m i_m r_f$$
$$-c_f^*\beta_m^* r_m r_f - bi_f - \sigma_f i_f + \alpha_0 i_f - \alpha_0 i_f^2 + \alpha r_f i_f,$$
$$f_3(t,x) = \delta_m - \delta_m i_m - \delta_m v_m - \delta_m r_m - bv_m + \alpha_0 i_m v_m + \alpha r_m v_m,$$
$$f_4(t,x) = \delta_f - \delta_f i_f - \delta_f v_f - \delta_f r_f - bv_f + \alpha_0 i_f v_f + \alpha r_f v_f,$$

$$f_5(t,x) = \sigma_m i_m - br_m + \alpha_0 i_m r_m - \alpha r_m + \alpha r_m^2,$$

and

$$f_6(t,x) = \sigma_f i_f - br_f + \alpha_0 i_f r_f - \alpha r_f + \alpha r_f^2,$$

so that our system of equations has the form (i.e., the vector-valued functional form)

$$x' = f(t,x) = f(x), \qquad x(t_0) = x_0. \tag{2.3}$$

Define

$$D = \left\{ x = (i_m, i_f, v_m, v_f, r_m, r_f) : i_m, i_f, v_m, v_f, r_m, r_f \le 1 \right\}, \tag{2.4}$$

and let

$$\left\| x - x_0 \right\| \le \varphi, \ \left\| t \right\| \le \tau, \text{ with } t_0 = 0, x_0 = \left(i_{m0}, i_{f0}, v_{m0}, v_{f0}, r_{m0}, r_{f0} \right).$$

We shall prove using Picard's theorem that (2.3) has a unique solution, by proving the following:

(1) f is continuous;
(2) f satisfies a Lipschitz condition; and
(3) $\left| f \right| \le M$.

Now, by the assumptions in our model, the vector-valued function $f(t,x)$ is continuous as each component $f_i, i = 1,2,3,4,5,6$ of $f(t,x)$ is a continuous function of the variable

$$x = (i_m, i_f, v_m, v_f, r_m, r_f)^T.$$

Let us establish the Lipschitz condition. We do this by showing that each component of $f_i, i = 1,2,...,6$ satisfies a Lipschitz condition.

Let $y=(i_{m1}, i_{f1}, v_{m1}, v_{f1}, r_{m1}, r_{f1})^T$,

Then $f(y)=(f_1(y), f_2(y), f_3(y), f_4(y), f_5(y), f_6(y))^T$.

Now, noting that $i_m, i_f, v_m, v_f, r_m, r_f \le 1$, we have

$$\begin{aligned}
|f_1(x)-f_1(y)| &= \Big|(c_m\beta_f(i_f-i_{f1})+c_m^*\beta_f^*(r_f-r_{f1})+(-c_m\beta_f)\{i_m(i_f-i_{f1})+i_{f1}(i_m-i_{m1})\}\\
&+(-c_m^*\beta_f^*)\{i_m(r_f-r_{f1})+r_{f1}(i_m-i_{m1})\}+(-c_m\beta_f)\{v_m(i_f-i_{f1})+i_{f1}(v_m-v_{m1})\}\\
&+(-c_m^*\beta_f^*)\{v_m(r_f-r_{f1})+r_{f1}(v_m-v_{m1})\}+(-c_m\beta_f)\{r_m(i_f-i_{f1})+i_{f1}(r_m-r_{m1})\}\\
&+(-c_m^*\beta_f^*)\{r_m(r_f-r_{f1})+r_{f1}(r_m-r_{m1})\}+(-b)(i_m-i_{m1})+(-\sigma_m)(i_m-i_{m1})\\
&+\alpha_0(i_m-i_{m1})+(-\alpha_0)(i_m+i_{m1})(i_m-i_{m1})+\alpha\{r_m(i_m-i_{m1})+i_{m1}(r_m-r_{m1})\})\Big|\\
&\le c_m\beta_f|i_f-i_{f1}|+c_m^*\beta_f^*|r_f-r_{f1}|+c_m\beta_f|i_m||i_f-i_{f1}|+c_m\beta_f|i_{f1}||i_m-i_{m1}|\\
&+c_m^*\beta_f^*|i_m||r_f-r_{f1}|+c_m^*\beta_f^*|r_{f1}||i_m-i_{m1}|+c_m\beta_f|v_m||i_f-i_{f1}|+c_m\beta_f|i_{f1}||v_m-v_{m1}|\\
&+c_m^*\beta_f^*|v_m||r_f-r_{f1}|+c_m^*\beta_f^*|r_{f1}||v_m-v_{m1}|+c_m\beta_f|r_m||i_f-i_{f1}|+c_m\beta_f|i_{f1}||r_m-r_{m1}|\\
&+c_m^*\beta_f^*|r_m||r_f-r_{f1}|+c_m^*\beta_f^*|r_{f1}||r_m-r_{m1}|+b|i_m-i_{m1}|+\sigma_m|i_m-i_{m1}|\\
&+\alpha_0|i_m-i_{m1}|+\alpha_0|i_m+i_{m1}||i_m-i_{m1}|+\alpha|r_m||i_m-i_{m1}|+\alpha|i_{m1}||r_m-r_{m1}|\\
&=(c_m\beta_f+c_m^*\beta_f^*+b+\sigma_m+3\alpha_0+\alpha)|i_m-i_{m1}|+4c_m\beta_f|i_f-i_{f1}|\\
&+(c_m\beta_f+c_m^*\beta_f^*)|v_m-v_{m1}|+0|v_f-v_f|+(c_m\beta_f+c_m^*\beta_f^*+\alpha)|r_m-r_{m1}|\\
&+4c_m^*\beta_f^*|r_f-r_{f1}|\\
&=l_{11}|i_m-i_{m1}|+l_{21}|i_f-i_{f1}|+l_{31}|v_m-v_{m1}|+l_{41}|v_f-v_{f1}|+l_{51}|r_m-r_{m1}|+l_{61}|r_f-r_{f1}|
\end{aligned}$$

Therefore,

$$|f_1(x)-f_1(y)| \le L_1\|x-y\| \qquad (2.4.1)$$

where $L_1=\max\{l_{11}, l_{21}, l_{31}, l_{41}, l_{51}, l_{61}\}$, and

$l_{11}=\left(c_m\beta_f+c_m^*\beta_f^*+b+\sigma_m+3\alpha_o+\alpha\right)$, $l_{21}=4c_m\beta_f$, $l_{31}=\left(c_m\beta_f+c_m^*\beta_f^*\right)$, $l_{41}=0$,
$l_{51}=\left(c_m\beta_f+c_m^*\beta_f^*+\alpha\right)$, $l_{61}=4c_m^*\beta_f^*$,

are constants depending on the parameters of the model.

Similarly,

$$\begin{aligned}|f_2(x)-f_2(y)| &\le 4c_f\beta_m|i_m-i_{m1}|+(c_f\beta_m+c_f^*\beta_m^*+b+\sigma_f+3\alpha_0+\alpha)|i_f-i_{f1}|+0|v_m-v_{m1}| \\ &+(c_f\beta_m+c_f^*\beta_m^*)|v_f-v_{f1}|+4c_f^*\beta_m^*|r_m-r_{m1}|+(c_f\beta_m+c_f^*\beta_m^*+\alpha)|r_f-r_{f1}| \\ &= l_{12}|i_m-i_{m1}|+l_{22}|i_f-i_{f1}|+l_{32}|v_m-v_{m1}|+l_{42}|v_f-v_{f1}|+l_{52}|r_m-r_{m1}|+l_{62}|r_f-r_{f1}| \\ &\le L_2\|x-y\| \end{aligned} \quad (2.4.2)$$

where $L_2=\max\{l_{12},l_{22},l_{32},l_{42},l_{52},l_{62}\}$, and

$l_{12}=4c_f\beta_m,\ l_{22}=\left(c_f\beta_m+c_f^*\beta_m^*+b+\sigma_f+3\alpha_o+\alpha\right),\ l_{32}=0,\ l_{42}=\left(c_f\beta_m+c_f^*\beta_m^*\right),$
$l_{52}=4c_f^*\beta_m^*,$

$l_{62}=\left(c_f\beta_m+c_f^*\beta_m^*+\alpha\right),$

$$\begin{aligned}|f_3(x)-f_3(y)| &\le (\delta_m+\alpha_0)|i_m-i_{m1}|+0|i_f-i_{f1}|+(\delta_m+b+\alpha_0+\alpha)|v_m-v_{m1}|+0|v_f-v_{f1}| \\ &+(\delta_m+\alpha)|r_m-r_{m1}|+0|r_f-r_{f1}| \\ &= l_{13}|i_m-i_{m1}|+l_{23}|i_f-i_{f1}|+l_{33}|v_m-v_{m1}|+l_{43}|v_f-v_{f1}|+l_{53}|r_m-r_{m1}|+l_{63}|r_f-r_{f1}| \\ &\le L_3\|x-y\| \end{aligned} \quad (2.4.3)$$

where $L_3=\max\{l_{13},l_{23},l_{33},l_{43},l_{53},l_{63}\}$, and

$l_{13}=\left(\delta_m+\alpha_o\right),\ l_{23}=0,\ l_{33}=\left(\delta_m+b+\alpha_o+\alpha\right),\ l_{43}=0,\ l_{53}=\left(\delta_m+\alpha\right),\ l_{63}=0,$

$$\begin{aligned}|f_4(x)-f_4(y)| &\le 0|i_m-i_{m1}|+(\delta_f+\alpha_0)|i_f-i_{f1}|+0|v_m-v_{m1}|+(\delta_f+b+\alpha_0+\alpha)|v_f-v_{f1}| \\ &+0|r_m-r_{m1}|+(\delta_f+\alpha)|r_f-r_{f1}| \\ &= l_{14}|i_m-i_{m1}|+l_{24}|i_f-i_{f1}|+l_{34}|v_m-v_{m1}|+l_{44}|v_f-v_{f1}|+l_{54}|r_m-r_{m1}|+l_{64}|r_f-r_{f1}| \\ &\le L_4\|x-y\| \end{aligned} \quad (2.4.4)$$

where $L_4=\max\{l_{14},l_{24},l_{34},l_{44},l_{54},l_{64}\}$, and

$l_{14}=0,\ l_{24}=\left(\delta_f+\alpha_o\right),\ l_{34}=0,\ l_{44}=\left(\delta_f+b+\alpha_o+\alpha\right),\ l_{54}=0,\ l_{64}=\left(\delta_f+\alpha\right),$

$$\begin{aligned}|f_5(x)-f_5(y)| &\le (\sigma_m+\alpha_0)|i_m-i_m|+0|i_f-i_{f1}|+0|v_m-v_{m1}|+0|v_f-v_{f1}| \\ &+(b+\alpha_0+3\alpha)|r_m-r_{m1}|+0|r_f-r_{f1}| \\ &= l_{15}|i_m-i_{m1}|+l_{25}|i_f-i_{f1}|+l_{35}|v_m-v_{m1}|+l_{45}|v_f-v_{f1}|+l_{55}|r_m-r_{m1}|+l_{65}|r_f-r_{f1}| \end{aligned}$$

$$\le L_5 \|x-y\| \tag{2.4.5}$$

where $L_5 = \max\{l_{15}, l_{25}, l_{35}, l_{45}, l_{55}, l_{65}\}$, and

$$l_{15} = (\sigma_m + \alpha_o),\ l_{25} = 0,\ l_{35} = 0,\ l_{45} = 0,\ l_{55} = (b + \alpha_o + 3\alpha),\ l_{65} = 0,$$

and

$$\begin{aligned} |f_6(x) - f_6(y)| &\le 0|i_m - i_{m1}| + (\sigma_f + \alpha_0)|i_f - i_{f1}| + 0|v_m - v_{m1}| + 0|v_f - v_{f1}| \\ &\quad + 0|r_m - r_{m1}| + (b + \alpha_0 + 3\alpha)|r_f - r_{f1}|. \\ &= l_{16}|i_m - i_{m1}| + l_{26}|i_f - i_{f1}| + l_{36}|v_m - v_{m1}| + l_{46}|v_f - v_{f1}| + l_{56}|r_m - r_{m1}| + l_{66}|r_f - r_{f1}| \\ &\le L_6 \|x-y\| \end{aligned} \tag{2.4.6}$$

where $L_6 = \max\{l_{16}, l_{26}, l_{36}, l_{46}, l_{56}, l_{66}\}$, and

$$l_{16} = 0,\ l_{26} = (\sigma_f + \alpha_o),\ l_{36} = 0,\ l_{46} = 0,\ l_{56} = 0,\ l_{66} = (b + \alpha_o + \alpha).$$

Therefore,

$$\|f(x) - f(y)\| \le L\|x-y\|, \tag{2.4.7}$$

where $L = \max\{L_1, L_2, L_3, L_4, L_5, L_6\}$.

To obtain the bound for $f(t,x)$, and noting that $i_m, i_f, v_m, v_f, r_m, r_f \le 1$, we have

$$\begin{aligned} |f_1(x)| &\le c_m\beta_f|i_f| + c_m^*\beta_f^*|r_f| + c_m\beta_f|i_f||i_m| + c_m^*\beta_f^*|r_f||i_m| + c_m\beta_f|i_f||v_m| + c_m^*\beta_f^*|r_f||v_m| \\ &\quad + c_m\beta_f|i_f||r_m| + c_m^*\beta_f^*|r_f||r_m| + b|i_m| + \sigma_m|i_m| + \alpha_0|i_m| + \alpha_0|i_m|^2 + \alpha|r_m||i_m| \\ &\le 4c_m\beta_f + 4c_m^*\beta_f^* + b + \sigma_m + 2\alpha_0 + \alpha \\ &= M_1. \end{aligned}$$

Similarly,

$$\begin{aligned} |f_2(x)| &\le 4c_f\beta_m + 4c_f^*\beta_m^* + b + \sigma_f + 2\alpha_0 + \alpha \\ &= M_2, \end{aligned} \qquad \begin{aligned} |f_5(x)| &\le \sigma_m + b + \alpha_0 + 2\alpha \\ &= M_5, \end{aligned}$$

$$|f_3(x)| \le 4\delta_m + b + \alpha_0 + \alpha \qquad |f_6(x)| \le \sigma_f + b + \alpha_0 + 2\alpha$$

$= M_3,$ $= M_6.$

$$\left| f_4(x) \right| \le 4\delta_f + b + \alpha_0 + \alpha$$

$$= M_4,$$

Therefore,

$$\left\| f(x) \right\| = \max\left\{ M_1, M_2, M_3, M_4, M_5, M_6 \right\}$$

$$\le M.$$

Thus, there exists a unique solution for the IVP (2.3) in the domain, $\left| x - x_0 \right| \le \varphi$ and $\left| t \right| \le h$, where $h = \min\left\{ \tau, \varphi / M \right\}$.

This completes the proof.

3. Conclusion

In this paper, we have formulated and presented an HIV/AIDS transmission dynamics mathematical model that incorporates vaccination of susceptibles against HIV infection with counseling and ART; and have shown that our system of equations represent a useful mathematical model of a physical system by carrying out a classical qualitative proof of the existence and uniqueness of a solution to the governing system of model equations. Hence the title of paper.

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