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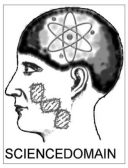


# A Deterministic Model of ART on HIV Spread Outcomes: A Case Study in Tanzania

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***Authors' contributions***

*This work was carried out in collaboration between both authors. Both authors read and approved the final manuscript.*

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## Abstract

This is about understanding in a mathematical perspective, how the introduction of antiretroviral therapy (ART) is shaping the spread of HIV. A non-linear mathematical model for HIV transmission in a variable size population is formulated in this paper. The model is about analysis and simulation of HIV spread along with treating infected individuals with ARV therapy. Thus, the model is constructed by including individuals who are under ARV therapy as transmitters of HIV. This paper includes studying the speed of spread and how best could be controlled by including the concept of doubling time. The model's point of equilibria have been found and their stability have been investigated. The model has two points of equilibria, the disease free and the endemic equilibrium. It has been found that if basic reproduction number, $R_0 < 1$ the disease free equilibrium is asymptotically stable under some conditions. On the other hand if $R_0 > 1$ the disease free equilibrium is not stable. In addition, when $R_0 > 1$ there exist a unique endemic equilibrium, which is found to be both locally and globally stable under some conditions.

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Simulations of the model have been conducted, taking Tanzania as a case study for the year 2015 onwards. Initial values for the population size start in 2015, and the endemic equilibrium has been estimated. The measures to control the spread of HIV have been suggested to ensure that $R_0 < 1$. One of the case simulated is that $R_0 = 0.6875 < 1$, in which the epidemic diminishes. When $R_0 > 1$ the disease grows, and this has been simulated for $R_0 = 1.3 > 1$.

*Keywords: ART in Tanzania; doubling time; equilibrium point stability; model simulation.*

**2010 Mathematics Subject Classification:** 53C25, 83C05, 57N16.

# Abbreviations

| *Acronym* | *Acronym description* |
|---|---|
| ART | Antiretroviral therapy |
| ARV | Antiretroviral |
| MTCT | Mother-to-child-transmission |
| AIDS | Acquired Immuno-deficiency Syndrome |
| HIV | Human Immuno-deficiency Syndrome |
| HAART | Highly active antiretroviral therapy |

# 1 Introduction

## 1.1 Historical Background

HIV (human immunodeficiency virus) is a virus that attacks the body immune system, the body's natural defense. Without a strong immunity, the body has trouble fighting off diseases. Both the virus and infection it causes are called HIV. White blood cells are an important part of the immune system. HIV infects and destroys certain white blood cells called CD4+. If too many CD4+ cells are destroyed, the body can no longer defend itself against infections [1]. The last stage of HIV infection is AIDS (Acquired Immune-Deficiency Syndrome). People with AIDS have a low number of CD4+ cells and get infections or cancers that rarely occur in healthy people. For example, Tuberculosis remains the leading cause of death among people living with HIV, accounting for around one in three AIDS-related deaths. AIDS has become a severe infectious disease in both the developed and developing nations, and is considered a world pandemic, [2]. HIV transmissions among individuals is due to non-infected individual allowing blood contamination, unsafe sexual contacts with infected individuals [3], and vertical (mother-to-child) transmission [4].

Since the beginning of HIV in 1981, to the end of 2015, more than 70 million people have been infected with the HIV virus and about 35 million people have died of AIDS. That is, globally, 36.7 (34.0 - 39.8) million people were living with HIV at the end of 2015. In 2015, around 46% of all people living with HIV had access to treatment, of which 77% pregnant women living with HIV had access to antiretroviral medicines preventing transmission of HIV to their babies, [5].

Nevertheless, there were some improvement against fighting the pandemic. In 2015 statistics by UNAIDS estimates that AIDS-related deaths have fallen by 45% since the peak in 2005, this does not exclude the leading killer Tuberculosis. Tuberculosis-related deaths among people living with HIV have fallen by 32% since 2004 globally. There has been good news following new HIV infections in children, new HIV infection have declined by 50% since 2010. Worldwide, 150 000 children became

newly infected with HIV in 2015, down from 290 000 in 2010. However, despite these improvements fighting against this pandemic, there still has a long way to go. New HIV infection among adults is still a big challenge, since 2010 there have been no declines in new HIV infections among adults. That is every year, since 2010, around 1.9 million adults have become newly infected with HIV. It is observed though that the number of deaths including adults has decreased, this can be accounted for due to antiretroviral therapy. For instance, in 2014, the percentage of identified HIV-positive tuberculosis patients who started or continued on antiretroviral therapy reached 77%. More funds has been continuing being invested to combat the pandemic since its invention. For instance, at the end of 2015, US$ 19 billion was invested in the AIDS response in low-and middle-income countries. Recent updated UNAIDS estimates indicate that US$ 26.2 billion will be required for the AIDS response in 2020, with US$ 23.9 billion required in 2030, [5].

Worldwide, Africa is the worst hit continent by the pandemic and Sub-Saharan Africa remains most severely affected. In 2015 nearly 1 out of 25 adults (4.4%) were living with HIV in sub-Saharan Africa, accounting for nearly 70% of the people living with HIV worldwide (who.int). This is a bit higher than two thirds of the 2007. In 2007, it was estimated that two thirds of the global total of 32.9 million people with HIV were living in this region, and three quarters of all AIDS deaths in 2007 were from the sub-Saharan region. It was estimated that by 2020 the nine most severely hit Sub-Saharan countries may lose 13 to 26% of their agricultural labor force to AIDS. Those who are dying, not limited to agricultural workers. They are household heads, mothers and fathers of young children and adolescents, caregivers for the old and sick, transmitters of agricultural and livelihood knowledge, skills and custodians of social safety nets.The gross domestic product in countries with HIV prevalence rates of 10% or higher could drop by 18% in 2020 [6].

## 1.2 Mathematical background

Mathematical models have been used extensively in research into the epidemiology of HIV, to help improve the understanding of major contributing factors in the pandemic. From initial models by Anderson, RM *et al* 1986 [7] and May, RM *et al.* 1987 [8]. Various refinements have been added into modeling frameworks, and specific issues have been addressed by researchers [9, 10, 11]. In recent years, more researches have been conducted to study how ART have influenced the HIV infections. Although treatment with antiviral agents has proven a very effective way to the health and survival of infected individuals, the epidemic will continue to grow unless greatly improved prevention strategies can be developed and implemented [12].

Granich, RM *et al* [13] constructed the mathematical model for HIV including ART. There are four categories in each of the HIV infected class, the two classes are those under ART and those who are not. The model excludes individual under 15 years old. WHO [14, 15] in 2010 developed a paper which is about procedures as how to be treated with ARV for children and adults. Others researchers include Cole, SR *et al* [16] about "Effect of highly active antiretroviral therapy (HAART) on time to AIDS or death using marginal structure model." Greub, G *et al* [17], [18] in 2000, for Swiss HIV cohort studied the clinical progression, survival and immune recovery during ART in patients with HIV-1 and hepatitis C virus co-infection. It can be observed that many of the mentioned publications are about how to maintain a safer ART.

In recent years, studies of vertical transmission have been conducted to describe the effects of various epidemiological and demographical factors [19]. In particular [20], discussed a variety of diseases that transmit both horizontally and vertically, gave a comprehensive survey of the formulation and mathematical analysis of compartmental models that also incorporate mother-to-child spread. Brauer, F [21] considered the model for disease with vertical transmission, non-linear population dynamics and finite carrying capacity, and analyzed the stability of equilibrium in the special case

in which the overall birth rate does not depend on infective population. Li, MY *et al* [19] proposed a model for an infectious disease that spreads in the host population through both horizontal and vertical transmission.

Agarwalar, BD [22] developed a density dependent HIV spread model for Canadian population by taking into account the vertical transmission and by using simple mass action type interaction. Also Ram, N *et al* 2006 [23] examined a similar model to Agarwalar, BD; both included vertical transmission, but excluded individual with severe AIDS symptoms in the spread of disease. Kibona, IE *et al* in 2011 with a similar model to Naresh et al, just a bit refined the model by incorporating immigrants [24]. Granich, RM *et al* in 2009 developed a mathematical model that simulates HIV transmission that includes consideration of ARV therapy for individuals above 15 years old [13].

Blower, S *et al* in 2003 reviewed how mathematical models have been used to evaluate potential impact of HIV epidemics both combination ART and imperfect vaccines. Most of the model based on numerical simulations [25]. Similar review has been done by Rivadeira, PS *et al* but is a bit different as they reviewed the potential ground-breaking impact that mathematical tools may have in the analysis and understanding of the HIV dynamics [26]. Gray, RH *et al* simulated stochastically, the impact of ART and HIV vaccines on transmission; Rikai, Uganda. They concluded that ART alone cannot control the HIV epidemic in mature epidemics such as Rakai, and persons in need of therapy will increase over time. ART in combination with a low efficacy vaccine could control the epidemic, if behavioral dis-inhibition is prevented [27]. William, BG *et al* [28] came up with a research paper titled modeling the Impact of Antiretroviral Therapy on the Epidemic of HIV" in 2011. Briefly, the study is embracing the ART, how would it lead to control HIV including preventing the transmission provided that some conditions are fulfilled on usage. Not limited to that but also did some numerical simulations for some countries including South Africa.

In this paper however, the model includes individuals of all ages by incorporating mother-to-child transmission. People who are already brown AIDS symptoms are considered to be under ART, assuming that their CD4 count is overdue threshold. They are categorized into two classes, non and sexually active individuals. That is some people under ARV treatment do not participate in the spread of HIV due to acute situation of disease. Acute in the sense that they are hospitalized or the likes. On other hand, the purpose is to formulate a model that involves individual under ART, and for all ages. To generalize, this can be considered as a refinement of Kibona, IE *et al* (2011) [24] article by incorporated individuals with full brown AIDS symptoms in the transmission of HIV.

# 2 Model Formulation

A population of size ($N$) at time,$t$ with constant inflow of susceptible at a rate $Q$ is studied. The population size is divided into four subclasses. A susceptible class ($S$), this is a class with individuals not infected but are liable to be infections. An Infectious class ($I$), this is a class in which individuals have infections but not under ARV treatment. Infectious class under ART ($T$), one taking ARV dosage and sexually active. Removed class, $R$, this is a class with individual who are under ARV dosage and are assumed not able to participate in the transmission of HIV. Both $I$ and $T$ classes are assumed to be infectious, meaning that they are both sexually active, females in these classes may become pregnant and bear children. The reason is that they are taking advantage of the ART. Natural mortality rate is $\nu$ in all classes and the disease induced death rate is $\alpha$ in the removed class ($R$).

In addition, $\beta_1$ and $\beta_2$ are contact rates due to interactions that may lead to HIV transmissions between Infectious classes ($I$ and $T$) against the susceptible class, $S$ respectively. $c_1$ and $c_2$ is the

average number of partners in $I$ and $T$ classes respectively per individual. $\mu$ is the rate of transfer from $T$ individuals to $R$. Presumably, the susceptible $S$ become HIV infected via sexual contacts with infectious classes ($I$ or $T$) which may also lead to the birth of infected newborns. It is assumed that a fraction of newborn are infected at birth, and hence are directly recruited into the $I$ class with a rate $(1-\epsilon)\theta$ and others die effectively at birth $0 < \epsilon < 1$. It is also assumed that some of the infectives ($I$) move to join $T$ class, depending on the viral counts, with a rate $\sigma\delta$ and others with serious infection directly join the removed class ($R$) with a rate $(1-\sigma)\delta$ , where $0 < \sigma < 1$. The interaction between susceptible and infectious classes is assumed to be of standard mass action type. Therefore, the system of equations (2.1) is the model.

$$\begin{aligned}
\frac{dS}{dt} &= Q - \beta_1 c_1 S\tfrac{I}{N} - \beta_2 c_2 S\tfrac{T}{N} - \nu S;\\
\frac{dI}{dt} &= \beta_1 c_1 S\tfrac{I}{N} + \beta_2 c_2 S\tfrac{T}{N} + (1-\epsilon)\theta I - (\delta+\nu)I;\\
\frac{dT}{dt} &= \sigma\delta I - (\mu+\nu)T;\\
\frac{dR}{dt} &= (1-\sigma)\delta I + \mu T - (\alpha+\nu)R.
\end{aligned} \tag{2.1}$$

In this study it is assumed that $\beta_1 c_1 = \beta_2 c_2 = \beta c$ for the reason that an individual in the $T$ class is considered sexually active as a result of the ART dosage. Table 1 is a summary of parameters and their definitions. That is the model can now be written as system (2.2).

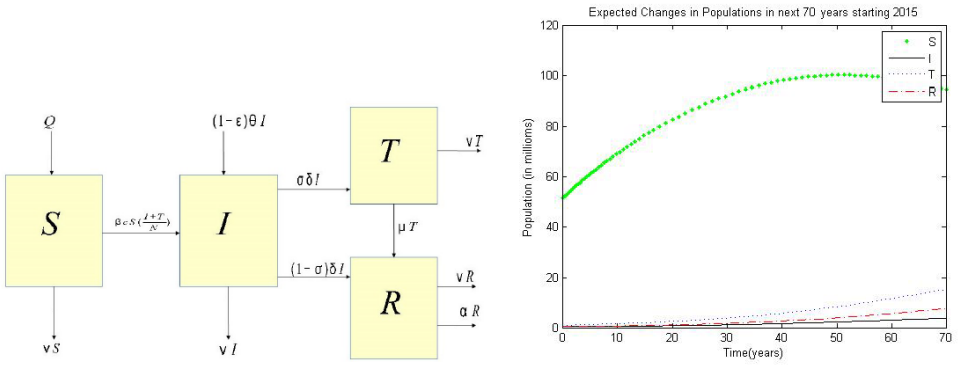
**Table 1. The Summarized Definition of Parameters**

| Parameter | Definition |
|---|---|
| $\nu$ | Natural mortality rate |
| $\alpha$ | AIDS induced death rate |
| $\beta$ | contact rates factor |
| $c$ | Average number of partners per individual |
| $\theta$ | Rate of infected newborns |
| $\epsilon$ | Fraction of babies who die immediately after birth due to HIV/AIDS |
| $\delta$ | Rate of transfer from $I$ to $T$ |
| $\sigma$ | Fraction of individual who remain in the $T$ class from $I$ |

$$\begin{aligned}
\frac{dS}{dt} &= Q - \beta c S\tfrac{(I+T)}{N} - \nu S;\\
\frac{dI}{dt} &= \beta c S\tfrac{I+T}{N} + (1-\epsilon)\theta I - (\delta+\nu)I;\\
\frac{dT}{dt} &= \sigma\delta I - (\mu+\nu)T;\\
\frac{dR}{dt} &= (1-\sigma)\delta I + \mu T - (\alpha+\nu)R.
\end{aligned} \tag{2.2}$$

$S_{t=0} = S_0$, $I_{t=0} = I_0$, $T_{t=0} = T_0$, $R_{t=0} = \Re_0$ are initial values notations.

The model can be expressed in the flow diagram as in Fig 1(a) and 1(b) is an example, which is a simulation of model(2.2) for the next 70 years starting 2015 in Tanzania. The data values for simulation mostly include reliable assumptions and approximations.




(a) Model Flow Diagram (b) A case of model simulation in Tanzania

**Fig. 1. Pictorial illustrations of the model(2.2)**

## 2.1 Basic reproduction number, $R_0$

For estimating the effects of outbreak in emerging epidemic diseases, basic reproduction number ($R_0$) is the most vital quantity in mathematical modeling. $R_0$ is basically the average number of new infectious cases caused by single infected individual in totally susceptible population. To find out $R_0$, it is first considered to rearrange the model (2.2) by adding all differential equations, this rearranges the system as bellow [[23]]: Add all equations in model (2.2), then use $N = S+I+T+R$, so that $S = N - I - T - R$.

$$\begin{aligned}
\frac{dN}{dt} &= Q - \nu N - \alpha R + (1-\epsilon)\theta I; \\
\frac{dI}{dt} &= \beta c(N - I - T - R)\tfrac{(I+T)}{N} + (1-\epsilon)\theta I - (\delta+\nu)I; \\
\frac{dT}{dt} &= \sigma\delta I - (\mu+\nu)T; \\
\frac{dR}{dt} &= (1-\sigma)\delta I + \mu T - (\alpha+\nu)R.
\end{aligned} \tag{2.3}$$

In order to evaluate the basic reproduction number, $R_0$, some assumption are made basing on the earliest stage of the epidemic. That is in the beginning stage of disease, the number of individuals in the $T$ and $R$ classes are assumed to be negligible in number, such that $T \approx 0$, $R \approx 0$ and $S \approx N$. These assumptions being substituted into the $I$ class then, $\frac{dI}{dt} = (\beta c + (1-\epsilon)\theta - (\delta+\nu))I$, of which is solved for $I$:

$$I = I_0 exp((\beta c - (\delta+\nu) + (1-\epsilon)\theta)t). \tag{2.4}$$

If $\beta c - (\delta+\nu) + (1-\epsilon)\theta < 0$ then the number of infectives die exponentially with time of which there will be no infection as $t$ approaches to infinity. That is $\beta c - (\delta+\nu) + (1-\epsilon)\theta < 0 \leftrightarrow \frac{\beta c + (1-\epsilon)\theta}{\delta+\nu} < 1$. That means $R_0 = \frac{\beta c + (1-\epsilon)\theta}{\delta+\nu}$. Define $D = \frac{1}{\delta+\nu}$ as average duration of which individual remain infectives. Then in terms of $D$ and $R_0$, $\beta c - (\delta+\nu) + (1-\epsilon)\theta = \frac{R_0 - 1}{D}$. Equation (2.4) for $I$ in terms of $D$ and $R_0$ is given as

$$I = I_0 exp((\frac{R_0 - 1}{D})t). \tag{2.5}$$

Equation (2.5) suggests that if $R_0 < 1$ the epidemic dies, where as when $R_0 > 1$ the it grows exponentially. When $R_0 = 1$, then initial number of infections is maintained for all generations.

## 2.2 Doubling time of the epidemic

In this context, doubling time is defined as the time taken to double the number of infected individual. Consider doubling time, $t = t_d$ of the epidemic be substituted in equation (2.5) so that $I = 2I_0$. Then $2I_0 = I_0 exp((\frac{R_0 - 1}{D})t_d)$, where $R_0 = D[\beta c + (1 - \epsilon)\theta]$. It follows that

$$t_d = \frac{D}{R_0 - 1} ln2. \tag{2.6}$$

Equation (2.6) suggests that doubling time, $t_d$ of the epidemic depends on the basic reproduction number, $R_0$, as in Fig 2. That doubling time, $t_d$ depends on $R_0$ in such a way that when $R_0 \leq 1$, then $t_d$ does not exist, implying that the epidemic diminishes. On the other hand, when $R_0 > 1$, $t_d$ exists and the epidemic grows exponentially as depicted by equation (2.5). Moreover, doubling time, $t_d$ is inversely proportional to $R_0$, that is the smaller $R_0$ leads to the larger doubling time. Therefore, in order to suppress the spread of epidemic, $R_0$ has to be small. It is interesting that even if $D$ changes so long as $R_0$ is maintained the doubling time is not affected. On the other hand, for a given $R_0$ a change in $D$ shifts the severeness of relationship between $R_0$ and $t_d$. That is individuals under ART are likely to have longer infectious period, this has nothing to do with HIV transmission so long as $R_0$ is kept small enough, Fig 2.

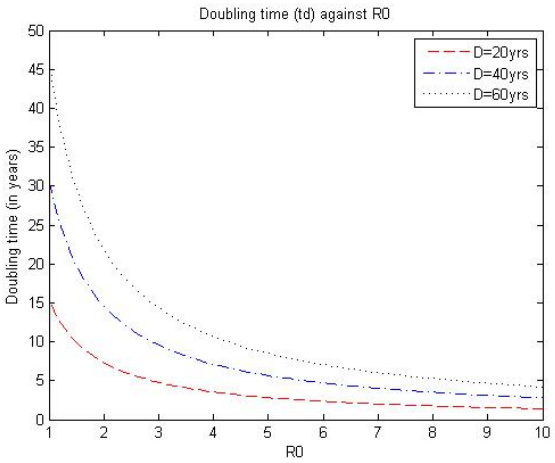



**Fig. 2. Plot of Doubling time, $t_d$ vs $R_0$**

The fact is that, $R_0$ depends not only on infected newborns but also on the constant $\beta c$ which proportional to unsafe interactions and number of partners ($c$). Under ART dosage, an infectious individual is likely to maintain, or even increase number of sexual partners for being sexually active. It is as well possible that an infected, pregnant female individual may transmit the infection to newborn through vertical transmission leading to greater $R_0$. Therefore, it is clear that while the population is enjoying the advantage of ARV dosage, at the same time it is very important to pay attention than ever prevention against HIV transmission. The best way would be to abstain sexual relationships, of which practically is unworkable to most individuals. Therefore, other protective means against transmission are strongly recommended including to reduce number of partners to one.

# 3 Equilibrium Stability of the Model

## 3.1 Disease free equilibrium of the model

Consider the disease free equilibrium for the model (3), then assign

$$\begin{aligned}
\frac{dN}{dt} &= Q - \nu N - \alpha R + (1-\epsilon)\theta I; && ...(i)\\
\frac{dI}{dt} &= \beta c(N - I - T - R)\tfrac{(I+T)}{N} + (1-\epsilon)\theta I - (\delta+\nu)I; && ...(ii)\\
\frac{dT}{dt} &= \sigma\delta I - (\mu+\nu)T; && ...(iii)\\
\frac{dR}{dt} &= (1-\sigma)\delta I + \mu T - (\alpha+\nu)R. && ...(iv)
\end{aligned}$$

The disease free equilibrium exists when $\frac{dN}{dt} = \frac{dI}{dt} = \frac{dT}{dt} = \frac{dR}{dt} = 0$ and it is practically valid to consider $I = T = R = 0$ so that when substituted, the system is reduced to $0 = Q - \nu N ...(i)$ the rest equations become trivial. Solve the later, disease free equilibrium, $E^0$ is

$$E^0(N^0 = \frac{Q}{\nu},\ I^0 = 0,\ T^0 = 0,\ R^0 = 0). \tag{3.1}$$

Thus, the total population in the susceptible class at disease free equilibrium is equal to the total population. Therefore, $N^0 = S^0 = \frac{Q}{\nu}$.

## 3.2 Endemic equilibrium of the model

The endemic equilibrium point, $E^*(N = N^*, I = I^*, T = T^*, R = R^*)$ is obtained by solving for $I \neq 0$ at the equilibrium point of the system (2.3).

$$\begin{aligned}
0 &= Q - \nu N^* - \alpha R^* + (1-\epsilon)\theta I^*; && ...(i)\\
0 &= \beta c(N^* - I^* - T^* - R^*)\frac{I^* + T^*}{N^*} + (1-\epsilon)\theta I^* - (\delta+\nu)I^*; && ...(ii)\\
0 &= \sigma\delta I^* - (\mu+\nu)R^*; && ...(iii)\\
0 &= (1-\sigma)\delta I^* + \mu T^* - (\alpha+\nu)R^*. && ...(iv)
\end{aligned}$$

After some algebraic manipulations for the endemic equilibrium, it is found that there exists only one endemic equilibrium, $E^*$:

$$E^*(N^* = \frac{I^*}{l_0 m_0},\ I^* = (R_0 - 1)m_1,\ T^* = \sigma\delta(\mu+\nu)m_2 I^*,\ R^* = (\mu + \nu(1-\sigma))\delta m_2 I^*) \tag{3.2}$$

$$\begin{aligned}
l_0 &= [\beta c(\alpha+\nu) + 2\sigma\delta(\alpha+\nu) + (\sigma\delta)^2(\alpha+\nu) + \beta c\delta(\mu+\nu(1-\sigma))(\mu+\nu) + \sigma\delta^2(\mu+\nu(1-\sigma))]^{-1};\\
m_0 &= [(\delta+\nu)(\mu+\nu)(R_0-1) + \beta c\sigma\delta](\alpha+\nu)(\mu+\nu);\\
m_1 &= \frac{Q l_0[(\delta+\nu)(\mu+\nu) + \frac{\beta c\sigma\delta}{R_0-1}][(\alpha+\nu)(\mu+\nu)]^2}{l_0 m_0[\alpha\delta(\mu+\nu(1-\sigma)) - (1-\epsilon)\theta(\alpha+\nu)(\mu+\nu)] + \nu(\alpha+\nu)(\mu+\nu)};\\
m_2 &= [(\alpha+\nu)(\mu+\nu)]^{-1}.
\end{aligned}$$

Be noted that $l_0 > 0$, provided that $R_0 > 0$, and $m_2 > 0$ unconditionally, since the parameters defining it are nonnegative. $m_1$ exists and positive if $R_0 > 1$ and $l_0 m_0[\alpha\delta(\mu + \nu(1-\sigma)) - (1-\epsilon)\theta(\alpha+\nu)(\mu+\nu)] + \nu(\alpha+\nu)(\mu+\nu) > 0$. In this case we assume that $(1-\epsilon)\theta$ which is infected newborns through vertical transmission is too small enough such that $l_0 m_0[\alpha\delta(\mu+\nu(1-\sigma)) - (1-\epsilon)\theta(\alpha+\nu)(\mu+\nu)] + \nu(\alpha+\nu)(\mu+\nu) > 0$. In this case $I^*$ exists and is positive when $R_0 > 1$. Nevertheless, the endemic equilibrium does not exist when $R_0 < 1$.

## 3.3 Local stability of equilibrium points

In order to understand the local stability of the model around equilibrium point, the Jacobian matrix, $J$ is determined from the model(2.3) and used to study the stability of disease free and endemic equilibria. That is Jacobian matrix, $J$ is given from the formula

$$J = \begin{bmatrix} \frac{\partial}{\partial N}(\frac{dN}{dt}) & \frac{\partial}{\partial I}(\frac{dN}{dt}) & \frac{\partial}{\partial T}(\frac{dN}{dt}) & \frac{\partial}{\partial R}(\frac{dN}{dt}) \\ \frac{\partial}{\partial N}(\frac{dI}{dt}) & \frac{\partial}{\partial I}(\frac{dI}{dt}) & \frac{\partial}{\partial T}(\frac{dI}{dt}) & \frac{\partial}{\partial R}(\frac{dI}{dt}) \\ \frac{\partial}{\partial N}(\frac{dT}{dt}) & \frac{\partial}{\partial I}(\frac{dT}{dt}) & \frac{\partial}{\partial T}(\frac{dT}{dt}) & \frac{\partial}{\partial A}(\frac{dT}{dt}) \\ \frac{\partial}{\partial N}(\frac{dR}{dt}) & \frac{\partial}{\partial I}(\frac{dR}{dt}) & \frac{\partial}{\partial T}(\frac{dR}{dt}) & \frac{\partial}{\partial R}(\frac{dR}{dt}) \end{bmatrix}.$$

so that after evaluation

$$J = \begin{bmatrix} -\nu & (1-\epsilon)\theta & 0 & -\alpha \\ \beta\frac{(I+T)}{N}(1-\frac{(N-I-T-R)}{N}) & \frac{\beta c}{N}(N-R) - (\delta+\nu) + (1-\epsilon)\theta & \frac{\beta c}{N}(N-R) & -\beta\frac{(I+T)}{N} \\ 0 & \sigma\delta & -(\mu+\nu) & 0 \\ 0 & (1-\sigma)\delta & \mu & -(\alpha+\nu) \end{bmatrix}.$$

### 3.3.1 Stability of the disease free equilibrium point

The Jacobian matrix at the disease free equilibrium, $J_0$ is

$$J_0 = \begin{bmatrix} -\nu & (1-\epsilon)\theta & 0 & -\alpha \\ 0 & \beta c - (\delta+\nu) + (1-\epsilon)\theta & \beta c & 0 \\ 0 & \sigma\delta & -(\mu+\nu) & 0 \\ 0 & (1-\sigma)\delta & \mu & -(\alpha+\nu) \end{bmatrix}.$$

Of which the *characteristic polynomial* is given by

$$\chi(\lambda) = \begin{vmatrix} -\nu-\lambda & (1-\epsilon)\theta & 0 & -\alpha \\ 0 & \beta c - (\delta+\nu) + (1-\epsilon)\theta - \lambda & \beta c & 0 \\ 0 & \sigma\delta & -(\mu+\nu)-\lambda & 0 \\ 0 & (1-\sigma)\delta & \mu & -(\alpha+\nu)-\lambda \end{vmatrix}.$$

That is $\chi(\lambda) = (\nu+\lambda)(\alpha+\nu+\lambda)[(\beta c - (\delta+\nu) + (1-\epsilon)\theta - \lambda)(\mu+\nu+\lambda) + \beta c\sigma\delta] = 0$ is the characteristic equation. Therefore, the eigenvalues are given as:
$\lambda = \lambda_1 = -\nu$, $\lambda_2 = -(\alpha+\nu)$, and $\lambda_3 = \frac{-a_1 - \sqrt{a_1^2 - 4a_0}}{2}$, $\lambda_4 = \frac{-a_1 + \sqrt{a_1^2 - 4a_0}}{2}$ from $\lambda^2 + a_1\lambda + a_0 = 0$;
where $a_1 = (1-R_0)(\delta+\nu) + (\mu+\nu)$, $a_0 = (1-R_0)(\delta+\nu)(\mu+\nu) - \beta c\sigma\delta$. The values of $a_0$ and

$a_1$ follows from $R_0 = \dfrac{\beta c + (1-\epsilon)\theta}{\delta + \nu} \Leftrightarrow R_0(\delta+\nu) = \beta c + (1-\epsilon)\theta$. Locally asymptotically stable equilibrium point requires that all the eigenvalues have negative real parts. Therefore, provided that $R_0 < 1$ and $a_1^2 \leq 4a_0$ the disease free equilibrium point is locally asymptotically stable, and unstable when $R_0 > 1$.

### 3.3.2 Stability of the endemic equilibrium point

Now then consider the Jacobian matrix, $J^*$ at the endemic equilibrium point of the model (2.3)

$$J_* = \begin{bmatrix} -\nu & (1-\epsilon)\theta & 0 & -\alpha \\ \beta\frac{(I^*+T^*)}{N^*}(1-\frac{(N^*-I^*-T^*-R^*)}{N^*}) & \frac{\beta c}{N^*}(N^*-R^*)-(\delta+\nu)+(1-\epsilon)\theta & \frac{\beta c}{N^*}(N^*-R^*) & -\beta\frac{(I^*+T^*)}{N^*} \\ 0 & \sigma\delta & -(\mu+\nu) & 0 \\ 0 & (1-\sigma)\delta & \mu & -(\alpha+\nu) \end{bmatrix}.$$

Where as $N^* = \dfrac{I^*}{l_0 m_0}$, $I^* = (R_0-1)m_1$, $T^* = \sigma\delta(\mu+\nu)m_2 I^*$, $R^* = [\mu+\nu(1-\sigma)]\delta m_2 I^*$. The endemic equilibrium point can also be written in terms of $R_0$ as: $N^* = \dfrac{(R_0-1)m_1}{l_0 m_0}$, $I^* = (R_0-1)m_1$, $T^* = \sigma\delta(\mu+\nu)(R_0-1)m_1 m_2$, $R^* = [\mu+\nu(1-\sigma)]\delta(R_0-1)m_1 m_2$. Substitute the later into Jacobian matrix $J$, so that the *characteristic polynomial* is given by

$$\chi(\lambda) = \begin{vmatrix} -\nu-\lambda & (1-\epsilon)\theta & 0 & -\alpha \\ \beta c(l_0 m_0)^2 l_1 & (R_0-1)(\delta+\nu)-\beta c l_0 l_2 m_0 - \lambda & \beta c(1-l_0 l_2 m_0) & -\beta c l_0 l_3 m_0 \\ 0 & \sigma\delta & -(\mu+\nu)-\lambda & 0 \\ 0 & (1-\sigma)\delta & \mu & -(\alpha+\nu)-\lambda \end{vmatrix} = 0.$$

$$l_1 = m_2^2(\mu+\nu+\sigma\delta)[(\alpha+\nu)(\mu+\nu)+(\alpha+\nu)\sigma\delta+\delta(\mu+\nu(1-\sigma))];$$
$$l_2 = m_2\delta(\mu+\nu(1-\sigma)); \qquad l_3 = m_2(\mu+\nu+\sigma\delta)(\mu+\nu).$$

and be noted that $l_1,\ l_2,\ l_3 > 0$. That is

$$\chi(\lambda) = \lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4 = 0 \tag{3.3}$$

is the characteristic equation of which

$$\begin{aligned}
a_1 &= \alpha + 3\nu + \mu + l_0 m_0 m_2 \beta c\delta(\mu+\nu(1-\sigma)) - 1 \\
a_2 &= (R_0-1)(\delta+\nu)l_0 m_0 (m_2)^2 \beta c[(\mu+\nu)\sigma\delta][(\alpha+\nu)(\mu+\nu)+(\alpha+\nu)\sigma\delta+\delta(\mu+\nu(1-\sigma))](1-\epsilon)\theta \\
&+ \beta c l_0 m_0 m_2[\delta(\mu+\nu(1-\sigma))(\alpha+3\nu+\sigma\delta)+(1-\sigma)\delta(\mu+\nu+\sigma\delta)(\alpha+\nu)] \\
&+ (\alpha+\nu)(\mu+\nu)+\nu(\alpha+2\nu+\mu)-(\alpha+3\nu+\mu)-\beta c\sigma\delta \\
a_3 &= \beta c l_0 m_0 m_2\delta(\mu+\nu(1-\sigma))[(\mu+\nu)(\alpha+\nu)+\sigma\delta(\alpha+\nu)+(\alpha+2\nu+\mu)+\nu\sigma\delta] \\
&+ l_0[\beta c m_0 m_2(\alpha+\nu)(\mu+\nu+\sigma\delta)[\sigma\delta\mu+(1-\sigma)\delta(\mu+\nu)]+m_0 m_2(\alpha+\nu)] \\
&+ \frac{\beta c m_0^2 m_2}{\mu+\nu}(\mu+\nu+\sigma\delta)[(\alpha+\nu)[\mu+\nu+\sigma\delta]+\delta(\mu+\nu(1-\sigma))][\alpha(1-\sigma)\delta-(1-\epsilon)\theta(\mu+\alpha+2\nu)] \\
&+ \nu(\mu+\nu)(\alpha+\nu)-\beta c\sigma\delta(\alpha+2\nu)-(R_0-1)[(\delta+\nu)(\mu+\nu)(\alpha+\nu)+\alpha+2\nu+\mu] \\
a_4 &= m_0^2 m_2 \beta c[(\mu+\nu)+\sigma\delta][\alpha\sigma\delta\mu+(1-\sigma)\delta(\mu+\nu)-(\mu+\nu)(\alpha+\nu)(1-\epsilon)\theta] \\
&+ \beta c\nu l_0 m_0 m_2[(\mu+\nu)+\sigma\delta][(\alpha+\nu)[\mu+\nu+\sigma\delta]+\delta(\mu+\nu(1-\sigma))][(\mu+\nu)(\alpha+\nu)+\sigma\delta(\alpha+\nu)] \\
&+ \beta c\nu l_0 m_0(\mu+\nu\sigma\delta)(\sigma\delta\mu+(1-\sigma)\delta)-(R_0-1)(\delta+\nu)(\mu+\nu)(\alpha+\nu)\nu-\beta c\sigma\delta\nu(\alpha+\nu)
\end{aligned}$$

Depending on the choice of parameters, the characteristic equation is not always Hurwitz. $a_1, a_2, a_3, a_4 > 0$ for some choices of the parameters. This is suggesting that until we have the right choice of parameters, the system is not locally stable at the endemic equilibrium.

## 3.4 Global stability

The endemic equilibrium, $E^*$ exists in a bounded region of attraction $\Omega$.

***Lemma:***
Define $\Omega = \{(N, I, T, R) : 0 < N < \overline{N}, 0 < I < \overline{I}, 0 < T < \overline{T}, 0 < R < \overline{R}\}$ of which it can be shown that $\Omega$ is the region of attraction provided that the basic reproduction number, $R_0 > 1$, where $\overline{N} = \frac{Q + (1-\epsilon)\theta\overline{I}}{\nu}$, $\overline{I} = (R_0 - 1)\frac{Q}{\nu}\frac{(\mu+\nu)(\delta+\nu)}{\beta c(\mu+\nu+\sigma\delta)}$, $\overline{T} = \frac{\sigma\delta}{\mu+\nu}\overline{I}$, $\overline{R} = \frac{\delta(\mu+\nu(1-\sigma))}{(\alpha+\nu)(\mu+\nu)}\overline{I}$.

***Theorem:***
Given that the endemic equilibrium $E^*$ exists, then it is globally stable provided the following conditions are satisfied in $\Omega$, condition (3.5):

$$0 < k_2, k_3 < m \quad and \quad \frac{k_3}{k_2} < \frac{2(\mu+\nu)(\alpha+\nu)}{3\mu^2}$$

$$m = min\{\frac{2\beta c k_1(\mu+\nu)}{3\sigma\delta}, \ \frac{4\beta c k_1(\alpha+\mu)}{9(1-\sigma)\delta}\}, \qquad k_1 > \frac{3[(1-\epsilon)\theta]^2}{2\beta c\nu}.$$

*Proof*: Consider the following positive definite function, to be Lyapunov about $E^*$

$$V = \frac{1}{2}(N - N^*)^2 + k_1\left(I - I^* - I^* ln\frac{I}{I^*}\right) + \frac{1}{2}k_2(T - T^*)^2 + \frac{1}{2}k_3(R - R^*)^2 \tag{3.4}$$

where the constants $k_1$, $k_2$ and $k_3$ can be chosen suitably. The derivative of $V$ along with the solution of dynamical system about $E^*$ is

$$\begin{aligned}\frac{dV}{dt} &= (N - N^*)\tfrac{dN}{dt} + k_1(\tfrac{I-I^*}{I})\tfrac{dI}{dt} + k_2(T - T^*)\tfrac{dT}{dt} + k_3(R - R^*)\tfrac{dV}{dt} \\ &= (N - N^*)(Q - \nu N - \alpha R + (1-\epsilon)\theta)I + k_1(\tfrac{I-I^*}{I})(\beta c(N - I - T - R)(\tfrac{I+T}{N})(\delta+\nu)I + (1-\epsilon)\theta I) \\ &\quad + k_2(T - T^*)(\sigma\delta I - (\mu+\nu)T) + k_3(R - R^*)((1-\sigma)\delta I + \mu T - (\alpha+\nu)R).\end{aligned}$$

$\frac{dv}{dt}$ can be written as conditionally negative definitive function

$$\begin{aligned}\frac{dV}{dt} = \ & -\tfrac{1}{2}a_{11}(N - N^*)^2 + a_{12}(N - N^*)(I - I^*) - \tfrac{1}{2}a_{22}(I - I^*)^2 \\ & -\tfrac{1}{2}a_{11}(N - N^*)^2 + a_{14}(N - N^*)(R - R^*) - \tfrac{1}{2}a_{44}(R - R^*)^2 \\ & -\tfrac{1}{2}a_{22}(I - I^*)^2 + a_{13}(I - I^*)(T - T^*) - \tfrac{1}{2}a_{33}(T - T^*)^2 \\ & -\tfrac{1}{2}a_{22}(I - I^*)^2 + a_{24}(I - I^*)(R - R^*) - \tfrac{1}{2}a_{44}(R - R^*)^2 \\ & -\tfrac{1}{2}a_{33}(T - T^*)^2 + a_{34}(T - T^*)(R - R^*) - \tfrac{1}{2}a_{44}(R - R^*)^2 \\ & +\tfrac{3}{2}a_{22}(I - I^*)[(I - I^*) + S\frac{(I+T)}{IN} - S^*\frac{(I^*+R^*)}{I^*N^*}]\end{aligned}$$

where $a_{11} = \nu$, $a_{12} = -(1-\epsilon)\theta$, $a_{22} = \frac{2}{3}\beta c k_1$, $a_{24} = (1-\sigma)\delta k_3$, $a_{14} = -\alpha$, $a_{44} = \frac{2}{3}k_3(\alpha+\nu)$, $a_{13} = k_2\sigma\delta$, $a_{33} = k_2(\mu+\nu)$ and $a_{34} = \mu k_3$.

$\frac{dV}{dt}$, may be short written as $\frac{dV}{dt} = \frac{dV_1}{dt} + \frac{dV_2}{dt}$ following the assignment that

$$\begin{aligned}\frac{dV_1}{dt} = \quad & -\tfrac{1}{2}a_{11}(N-N^*)^2 + a_{12}(N-N^*)(I-I^*) - \tfrac{1}{2}a_{22}(I-I^*)^2 \\ & -\tfrac{1}{2}a_{11}(N-N^*)^2 + a_{14}(N-N^*)(R-R^*) - \tfrac{1}{2}a_{44}(R-R^*)^2 \\ & -\tfrac{1}{2}a_{22}(I-I^*)^2 + a_{13}(I-I^*)(T-T^*) - \tfrac{1}{2}a_{33}(T-T^*)^2 \\ & -\tfrac{1}{2}a_{22}(I-I^*)^2 + a_{24}(I-I^*)(R-R^*) - \tfrac{1}{2}a_{44}(R-R^*)^2 \\ & -\tfrac{1}{2}a_{33}(T-T^*)^2 + a_{34}(T-T^*)(R-R^*) - \tfrac{1}{2}a_{44}(R-R^*)^2\end{aligned}$$

and $\dfrac{dV_2}{dt} = \dfrac{3}{2}a_{22}(I-I^*)[(I-I^*) + S\dfrac{(I+T)}{IN} - S^*\dfrac{(I^*+R^*)}{I^*N^*}]$.

$\dfrac{dV_1}{dt} < 0$ when $a_{12}^2 - a_{11}a_{22}$, $a_{14}^2 - a_{11}a_{44}$, $a_{13}^2 - a_{22}a_{33}$, $a_{24}^2 - a_{22}a_{44}$, $a_{34}^2 - a_{33}a_{44} < 0$.

For $a_{12}^2 - a_{11}a_{22}$, $a_{14}^2 - a_{11}a_{44}$, $a_{13}^2 - a_{22}a_{33}$, $a_{24}^2 - a_{22}a_{44}$, $a_{34}^2 - a_{33}a_{44} < 0$, choose

$$\begin{aligned} & 0 < k_2, k_3 < m \quad and \quad \frac{k_3}{k_2} < \frac{2(\mu+\nu)(\alpha+\nu)}{3\mu^2} \\ m = min\{\frac{2\beta c k_1(\mu+\nu)}{3\sigma\delta},\ & \frac{4\beta c k_1(\alpha+\mu)}{9(1-\sigma)\delta}\}, \qquad k_1 > \frac{3[(1-\epsilon)\theta]^2}{2\beta c\nu}. \end{aligned} \tag{3.5}$$

$\dfrac{dV_2}{dt} = \dfrac{3}{2}a_{22}(I-I^*)[(I-I^*) + S\dfrac{(I+T)}{IN} - S^*\dfrac{(I^*+R^*)}{I^*N^*}] = 0$ at equilibrium point.

When $I^*$ is very large, $\dfrac{dV_2}{dt} \approx \dfrac{3}{2}a_{22}(I-I^*)^2$ as it can be shown that $[S\dfrac{(I+T)}{IN} - S^*\dfrac{(I^*+R^*)}{I^*N^*}]$ = *constant* when $I^*$ is very large. So that the term $\dfrac{dV_2}{dt} \approx \dfrac{3}{2}a_{22}(I-I^*)^2$ is absorbed by the three terms containing $\frac{1}{2}a_{22}(I-I^*)^2$ in the expression for $\dfrac{dV_1}{dt}$. Therefore, to sum up $\dfrac{dV}{dt} < 0$ by the condition (3.5) of which the global equilibrium exists, hence proving the theorem.

# 4 Simulation of the Model

The parameters are all constants, however, the practicality is that most of the parameter are not constant. Tanzania is one of the case where parameters such as rate of new HIV infections per year are not constant. Most of the parameter in order to simulate the situation for Tanzania involved some reliable assumptions and approximations. On the other hand, the parameters are subject to change in future researches especially along with glowing technology on simulation tools. Therefore, simulations may not give the exact picture of HIV situation in Tanzania starting year 2015. Nevertheless, the simulation can help to convey some reliable information as how to control HIV for the next generation. Table 2 lists all the parameter and their sources. The ode45 from Mat-Lab has taken major part in the model simulation.

The 2015 demographical records indicates that of all people living with HIV at the time, most of them were under ART treatment [[5]]. That is with the total population of about 53 million in 2015, 2.6% were living with HIV of which 53% adults were under ART. It is recommended that people living with HIV should start ART right away, but in many places, the decision about when to start treatment is still dependent upon CD4+ count test,[[29]]. Whatever the case, it seems that the population under ART treatment has grown growing over people with HIV but not under ART. Our effort should be to ensure that the number of people under therapy does not grow over those living free of HIV infections as shown in the Fig 3(a).

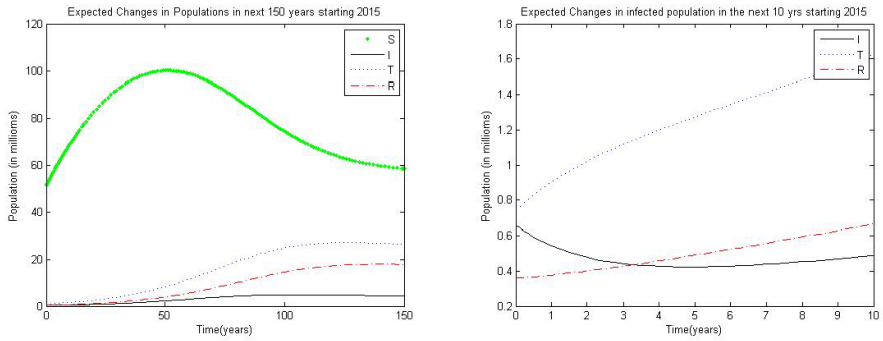
Numerical simulation has been conducted, considering Tanzania as a case study. This paper is about what if the attention to controlling HIV transmission is not enforced. Therefore, this paper should not be misquoted of what is simulated. Assuming 2015 as initial populations ($P_0$) in millions, then $P_0(S_0 = 51.24, I_0 = 0.658, T_0 = 0.742, \Re_0 = 0.36)$ with the total population of $N_0 = 53$. Starting with $P_0$, the model predicts endemic equilibrium point as $P^*(S^* = 58, I^* = 5, T^* = 25, R^* = 16)$

**Table 2. Data values for model and their sources for year 2015 in Tanzania**

| Parameter | Value(% per year) | Source |
|---|---|---|
| $Q$ | 3.19 | countrymeters.info/en/Tanzania |
| $\nu$ | $\frac{1}{53} \approx 0.02$ | countrymeters.info/en/Tanzania |
| $\alpha$ | 0.07 | estimated from UNAIDS Gap Report 2016 |
| $\beta c$ | 0.125 | assumptions |
| $\theta$ | 0.10 | estimated from UNAIDS Gap Report 2016 |
| $\epsilon$ | 0.40 | [23] |
| $\delta$ | 0.50 | assumptions |
| $\mu$ | 0.04 | assumptions |
| $\sigma$ | 0.70 | assumptions |
| $N_0$ | 53 million | UNAIDS Gap Report 2016 |
| $S_0$ | 51.24 million | UNAIDS Gap Report 2016 |
| $I_0$ | 0.658 million | UNAIDS Gap Report 2016 |
| $T_0$ | 0.742 million | UNAIDS Gap Report 2016 |
| $\Re_0$ | 0.36 million | UNAIDS Gap Report 2016 |

with the total population of $N^0 = 104$ Fig 3(b). Initially, out of the approximated 53 million population only 2.6% are people living with HIV. After one generation, the equilibrium is reached with a total population of approximately 104 million, 44.23% living with HIV.

Initially as of 2015, people in severe hit of AIDS and under ART were the least in size of all classes, but at equilibrium people infected but not under therapy are the least in number. Fig 3(b), is about to illustrate how the infected population in turn surpass one another in the course of about 5 years.



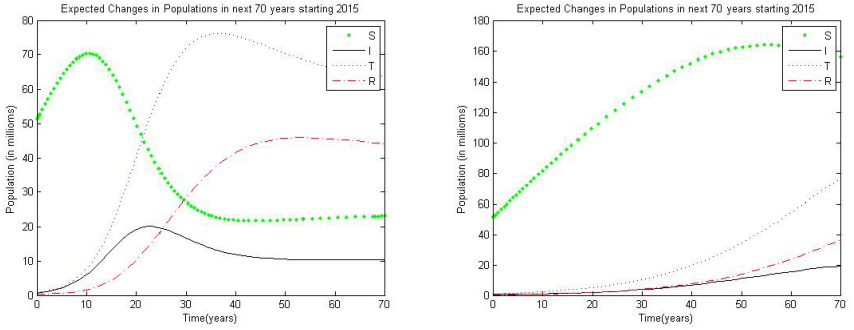

(a) $R$ is the least populated of all infected classes as of 2015 (b) $R$ may surpass the infectious ($I$) in 5 years

**Fig. 3. Current Status of HIV populations in Tanzania as predicted by the Model**

## 4.1 Impact of unsafe interactions and Number of Sexual Partners ($\beta c$)

Unsafe interaction in this context includes insecure sexual contacts, blood contamination and the likes. These are represented by a parameter $\beta c$ in the model. Increasing $\beta c$ blows up spread of the epidemic. Prediction by the model, Fig 4(a) suggests that until safe interactions and number of

partners are strictly controlled, the individual under ART are likely to be majority of all classes in two a decades. The fact is that the number of individual under ART are increasing, therefore, this may avert the growth of susceptible class after a decades.




(a) large $\beta c$ value as $\beta c = 0.26$ explodes transmissions

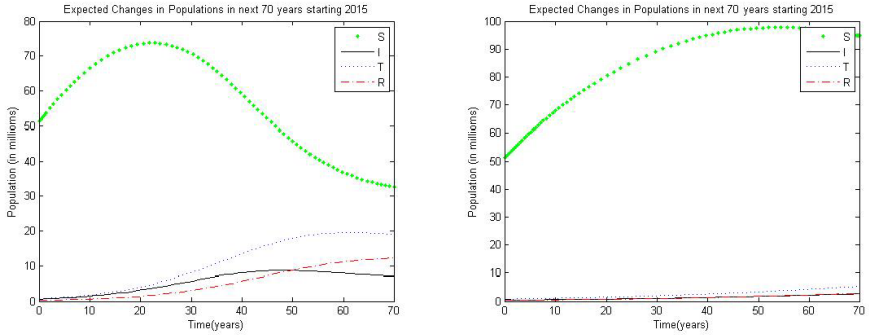
(b) Small $\beta c$ value as $\beta c = 0.08$ lowers transmissions

**Fig. 4. Variation in $\beta c$ changes the dynamics of model**

Maintaining secure sexual contacts against HIV patients may save the next generation, in this case people has to take it seriously using condoms, strict on one sexual partner and safe blood contamination. Under safer restrictions, the model in Fig 4(b) predicts a safer future against HIV.

## 4.2 Impact of Mother-to-child-transmission ($\theta$)

Mother-to-child-transmissions has a indispensable impact in futurity of HIV transmission. In 2015, out of 54,000 new HIV infections in one-fifth is due to MTCT [5]. Therefore, in no serious measure to prevent MTCT the model, Fig 5(a) predicts that susceptible population will stop growing after the next two decades.




(a) $\theta = 0.25$ lessens the growth of $S$ after 20 years

(b) $\theta \approx 0$ improves the growth of $S$

**Fig. 5. Variation in $\theta$ changes the dynamics of model**

However, if the current efforts in Tanzania to prevent MTCT [30, 31] is maintained or made better, the future is in the safer HIV world, Fig 5(b).

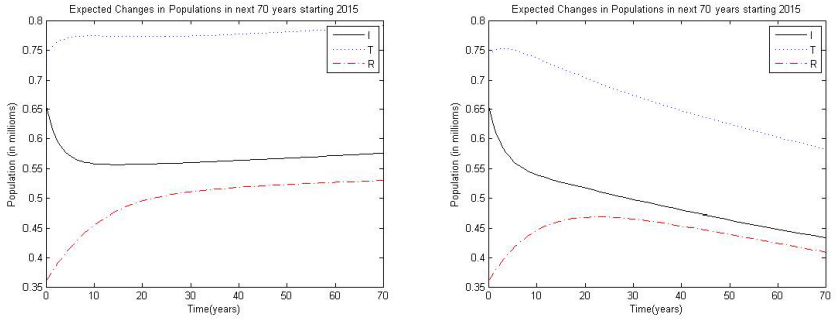
## 4.3 The Value of $R_0$ to Control the Pandemic

For the purpose of disease elimination, the value of $R_0$ has to be controlled so that always $R_0 < 1$. When $R_0 \geq 1$ the disease never diminish.

For example when $R_0 = 1.038$ as for the case of Tanzania, Fig 6(a) the disease is almost not growing such that even in the next one generation, populations of individual living with HIV remain almost constant. Nevertheless, there is even better news when $R_0 = 0.6875 < 1$ Fig 6(b) in which the disease dies after some years.

Elimination of the disease as indicated in Fig 6(b), requires not only prevention of MTCT but also individuals need to live free of insecure blood contamination and sexual contacts. In addition to that the number of partners sexually need be reduced to one. Secured sexual contact includes use of condoms and circumcision. All is about lowering both $\beta c$ and $\theta$ to ensure that $R_0$ is small enough.

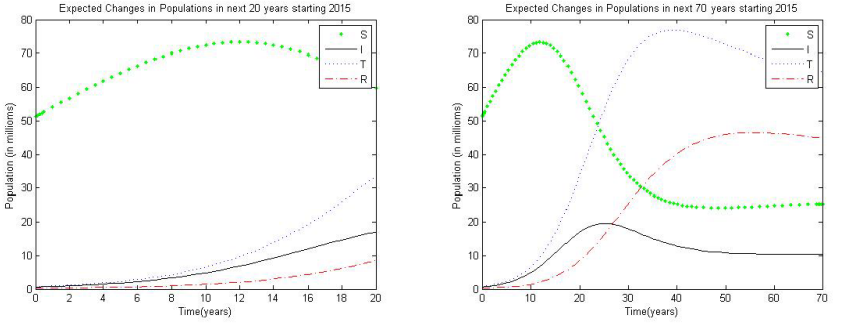
If the value of $R_0$ is not controlled such that it goes beyond unit then expect growth of the pandemic Fig 7, of which $R_0 = 1.3 > 1$. That means the future will be occupied by majority under ART. It is even disappointing that most of the working class in those days should be under ART. That is people living with HIV will be groomed on themselves. Therefore, $R_0$ has to be controlled ensuring that $R_0 < 1$ in order to bring the future in safer hands, if possible HIV free society.




(a) $R_0 = 1.038 \approx 1$ keeps total number of infections unchanged

(b) HIV/AIDS diminishes when $R_0 = 0.6875 < 1$

**Fig. 6. Variation in $R_0$ accordingly may control the pandemic**




(a) Dynamics in the first 20 years

(b) Dynamics in the next generation (70 years)

**Fig. 7. HIV/AIDS grows when $R_0 = 1.3 > 1$**

# 5 Conclusion

The fundamental part in the analysis of model (2), is the basic reproduction number ($R_0$). $R_0$ has been analyzed both analytically and at least numerically. $R_0 = D[\beta c + (1-\epsilon)\theta]$, increases if the number of infected newborns ($\theta$) or the transmission constant ($\beta c$) or both increase. When $R_0 > 1$ the number of infected individuals grow and when $R_0 < 1$ it diminishes. $R_0$ as well depends on $D$ which is the average duration an individual living with HIV remain infectious. However, in this analysis $D$ is assumed to be constant. In addition to $R_0$, Doubling time ($t_d$) refers to a period during which the epidemic may double the number of infections. $t_d$ exists only when $R_0 > 1$. Thus, $t_d$ is another way to explain how fast is the spread of disease if $R_0 > 1$. In this model, $t_d$ is more realistic in the earliest stage of epidemic. The stability of equilibrium points have been realized to be conditional both local and globally. The model has only two points of equilibria, one HIV free and one not. A HIV free equilibrium point requires Hurwitz conditions for stability analysis. Lyapunov function has been constructed in order to prove the global stability of endemic equilibrium.

Numerical simulation has been conducted, considering Tanzania as a case study. This paper is about what if the attention to controlling HIV transmission is not enforced. Therefore, this paper should not be misquoted of what is simulated. Assuming the 2015 demographic data as initial population in millions for Tanzania, then $S_0 = 51.24$, $I_0 = 0.658$, $T_0 = 0.742$, $\Re_0 = 0.36$) with the total population of about $N_0 = 53$. From this initial classes of populations, the model predicts endemic equilibrium point as $S^* = 58$, $I^* = 5$, $T^* = 25$, $R^* = 16$ amounting to the total of $N^* = 104$ Fig 3(a). Initially, out of the approximated 53 million population only 2.6% were people living with HIV. After one generation or 70 years, the equilibrium is reached with a total population of approx 104 million, 44.23% living with HIV.

It has been estimated that the epidemic would diminish if $R_0 = 0.6875 < 1$, Figure 6(b) and gets severe or catastrophic if $R_0 = 1.3 > 1$, Figure 7. Therefore, measures to ensure that $R_0 < 1$ are to be observed. Recent studies have a lot better to be taken care of as updates to controlling HIV spread including the ability to survive healthier than ever. It has been found that, levels of viral load suppression can be achieved with the best available ARV drugs. And, the relationship between plasma viral load and transmission of HIV along with effective and early ART, HIV transmissions between sexual partners or from mothers to their children can be greatly reduced [32]. ARV drugs reduce viral replication and can minimize mother-to-child transmission of HIV either by lowering plasma viral load in pregnant women or through post-exposure prophylaxis in their newborns. Not only limited to ART but there is extended ART forms, a highly active antiretroviral therapy (HAART) which usually comprises three drugs, can reduce mother-to-child-transmission rates down to around 1-2% from about 15 - 45% [33]. Because of HAART being expensive, various simpler and less costly ARV regimens can be offered to pregnant women who can not afford the price. Researches have supported the view that early ART, in combination with other methods of control, including male circumcision, vaginal microbicide, behavior change interventions, counseling and support should reduce the incidence of HIV to low levels within ten years and the prevalence of HIV to low levels within forty years. Unlike in the beginning with ART of which a lot of side effects were mentioned, now days ART is more stable, side effects are fewer, adherence is better, and resistance does not increase with earlier initiation of therapy, so that many arguments for delaying treatment are no longer valid [28].

# Acknowledgement

Basing on the assumption included in the model formulation, therefore, this study is open for improvement. Nevertheless, we greatly appreciate the reviewer who without hesitations, have spent their time to ensure that third part readers get the intended objective from the findings.

## Competing Interests

Authors have declared that no competing interests exist.

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