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A STAGE STRUCTURED MODEL FOR HIV/AIDS IN THE PRESENCE OF VERTICAL TRANSMISSION: THE CASE OF GHANA

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Abstract. Vertical transmission remains a global challenge to HIV infection dynamics. It refers to the transmission of HIV from the mother-to-child during pregnancy, delivery, and breastfeeding soon after birth. In this paper, we formulate a mathematical model to determine the transmission dynamics of HIV/AIDS and the general impact of vertical transmission on HIV/AIDS in Ghana given that horizontal transmission is the only well-documented mode of transmission. The model incorporates the treatment of juveniles, adults and both vertical and horizontal transmission of HIV/AIDS. The infection-free state and the persistent state are examined. The model is analyzed via the basic reproduction number $\mathfrak{R}_o$. We prove that the infection-free state is globally stable when the reproduction number is less than one. The model is fitted to data obtained on HIV/AIDS from the Ghana Health Service to estimate the current and future prevalence of HIV/AIDS epidemics. We conclude that without treatment, pregnant women have a high risk of transmitting HIV to their babies. However, with treatment, even if the reproduction number of vertical transmission $\mathfrak{R}_v$ increases, the disease can still be kept under control and fewer babies will be born with the disease. Numerical analysis, as well as sensitivity analysis, are carried out. Results from the sensitivity analysis show that the parameters that most influence the model output were, effective transmission rate β and treatment rate τ_2. We noticed that increasing β increases $\mathfrak{R}_o$ and increasing τ_2 decreases $\mathfrak{R}_o$.

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1. INTRODUCTION

HIV (Human Immuno-Deficiency Virus) is a deadly viral disease that destroys the immune system and leaves the immune system prone to many other acute, chronic and fatal diseases, and disorders. HIV is one of the many viruses that can be transmitted by physical contact including sexual contact (horizontal transmission) and by other means (sharing of infected needles used for injection, injecting drugs, tattooing and body piercing). Transmission through sexual and other physical contacts are as a result of contacts between susceptible and infected individuals. Vertical transmission, however, occurs during pregnancy, through delivery, and after childbirth via breastfeeding by an infected mother to the juvenile (children under 15 years).

It has been noted in [20] that the number of infants newly infected with HIV globally has declined in recent years, from 370 000 in 2009 to 150 000 in 2015. However, the impact of vertical transmission of HIV/AIDS in Africa will persist to be more severe without appropriate antiretroviral therapy. Due to the low level of literacy, the poor health care system and various issues concerning our health care systems, vertical transmission of HIV/AIDS with or without treatment will continue to be a burden in our African continent. Generally, HIV-positive juveniles must be given early diagnosis and treatment to boost their chances of survival because HIV/AIDS progresses faster in infected juveniles than adults due to their underdeveloped immune system. For HIV-positive juveniles not initiated on ART, 35% are likely to die within the first year of life and 52% by 2 years of age. Early initiation of HIV-positive juveniles into treatment slows disease progression, suppresses viral load, and dramatically reduces mortality rates [20]. In 2005, an estimated 630 000 - 820 000 infants were newly infected, of whom around 280 000 - 360 000 were infected through breastfeeding [6]. Similarly, in 2006, almost 530 000 infants became newly infected with HIV, 90% of them through mother-to-child transmission (MTCT) before, during or after delivery [2, 7, 8].

Since 2005, the availability of new antiretroviral drugs (ARTs) which offer high efficacy and acceptability and less toxicity, has led to a decrease of HIV related deaths worldwide [15, 22, 29]. Although progress has been substantial, uncertainties persist concerning the best way to manage the HIV/AIDS disease [11]. For instance, in 2011, there were approximately 330 000 new childhood infections, representing a reduction of 43% since 2003. This, however, remains unacceptably high. Unfortunately, increased childhood infection rates were observed in Angola, Congo, Equatorial Guinea, and Guinea-Bissau [28].

Vertical transmission models contain information useful to public health personnel as well as other stakeholders. They will help them with information on how to regulate disease spread since a cure is still to be found. While *Ö*zalp and Demirci [25], developed a fractional-order differential equation with the vertical transmission model in a varying population, Ogala et *al*. [24], in their work described the mother-juvenile pair characteristic that contributes to the vertical transmission of HIV and to propose remedies. They assessed the various factors responsible for increasing the chance of HIV transmission in juveniles born to positive HIV infected mothers in western Kenya. Afolabi et *al*. [1], in the model they proposed emphasized on the prevention of mother-to-child transmission (PMTCT) intervention strategies by the Nigerian government to prevent vertical transmission. Strategies included providing free tests including ARV certified drugs, which will cater for mother and juveniles during childbirth at PMTCT health service centers in Nigeria, and enrolling all infected HIV-positive mothers and their families into antiretroviral treatment as well as monitoring of their immune response to therapy.

Many models such as [4, 16, 17, 31] considered different factors such as age-structure, sex-structures, the incubation period (the period from getting the disease and development of symptoms), population density, mixing patterns, variable infectivity, genetic variation, time from infection to AIDS. These models were equipped with these characteristics to answer different questions and address important issues associated with the transmission dynamics of HIV/AIDS especially in developing countries and communities where the health care systems

are poor.

Various factors such as traditional beliefs, religious beliefs and ethnic norms play a significant role in the spread of HIV/AIDS in Ghana. Gyimah et *al*. [14], highlighted that although the HIV/AIDS situation in Ghana is not as grievous as in some southern and eastern African countries, data from sentinel sites in the country indicate that prevalence has somewhat stalled at 3.4%, mostly due to high infection rates among young people.

Most models for HIV/AIDS in Ghana do not incorporate vertical transmission and as a consequence, may fail to capture the effect of vertical transmission and the actual impact of HIV/AIDS in the Ghanaian population. Cases where HIV infected juveniles would survive into early adulthood were considered rare, but now it is possible for them to survive into adulthood due to the availability of antiretroviral treatments (ARTs). However, survival to older childhood with untreated vertically acquired HIV infection which was previously considered unusual was noted in Harare, Zimbabwe [10]. Furthermore, a substantial epidemic of HIV/AIDS in older survivors from the vertical transmission is emerging in Southern Africa [9]. Since Ghana is located in the sub-Saharan region with most of these countries, we are intrigued to ask the questions: What will be the long-term transmission dynamics of HIV/AIDS in Ghana in the presence of vertical transmission? Also, what effect will progressing from the juvenile stage (under or not under treatment) to adulthood have on the transmission dynamics of HIV/AIDS in the long run? and what will be the future trend of HIV/AIDS transmission in the presence of sustained and improved treatment protocols?

Mathematical modelling plays a significant role in predicting, assessing, controlling potential outbreaks of diseases as well as analysing social, demographic and economic factors of diseases. Therefore, the main aim of this study is to formulate a mathematical model with both modes of transmission of HIV (horizontal and vertical) and together with computer simulations, we will investigate the behaviour of the model. We will also fit the model to data obtained from Ghana to determine and predict the trends and progression of HIV/AIDS

epidemic in Ghana which has not been done to the best of our knowledge.

This paper is organised into sections as follows. In Sect. 2, a mathematical model is formulated. A brief description of the model properties is given in Sec. 3. The analysis of the model is done in Sec. 4. Section 5 contains the numerical analysis of the model. In Sect. 6, the model is applied to data obtained from Ghana on HIV/AIDS. Section 7 contains a discussion, concluding remarks and future works.

2. MODEL FORMULATION

We consider a mathematical model that describes the dynamics of HIV/AIDS infection between two populations of adults and juveniles. The juvenile population is made up of susceptible juveniles (S_j), infected juveniles not under treatment (I_j) and those under treatment (T_j). Similarly, the adult population is made up of susceptible adults (S_a), infected adults not under treatment (I_a) and those under treatment (T_a) .

The total adult and juvenile populations N_a and N_j respectively are,

$$N_a = S_a + I_a + T_a, \tag{1}$$

$$N_j = S_j + I_j + T_j. \tag{2}$$

The susceptible juvenile class is generated by the initial juvenile population Λ. The susceptible juveniles are also generated by recruitment through birth at the rate $\Pi e^{-\mu_0\phi_1}(1-\varepsilon)(I_a+T_a)$, where Π is the natural birth rate, ε is the fraction of juveniles born with the virus and $e^{-\mu_0\phi_1}$ is the probability of surviving the juvenile stage, taken to be (0-15) years, with μ_0 the natural mortality rate of juveniles. We assume that the susceptible juveniles after surviving the maturity age ϕ_1 (15 years) will move to the susceptible adult population. The dynamics of the susceptible juvenile class is given by

$$\dot{S_j} = \Lambda + \Pi e^{-\mu_0\phi_1}(1-\varepsilon)(I_a+T_a) - (\mu+\phi_1)S_j. \tag{3}$$

We assume that a proportion p of juveniles born with the virus are subjected to treatment straight after birth and given they survive the maturation age, progress to the class of infected juveniles under treatment. The infected juveniles under treatment class are recruited at the rate $\Pi e^{-\mu_0\phi_1} p\varepsilon(I_a+T_a)$ and also by the rate τ_1 at which infected juveniles not under treatment transition into treatment. Individuals leave this class by maturing into the infected adults under treatment class at a rate of ϕ_1. The infected juveniles under treatment are also assumed to progress to AIDS with a rate of ρ_2. Therefore, the dynamics of the infected juvenile under treatment class is expressed as

$$\dot{T}_j = \Pi e^{-\mu_0\phi_1} p\varepsilon(I_a+T_a) + \tau_1 I_j - (\mu+\phi_1+\rho_2)T_j. \tag{4}$$

The infected juveniles not under treatment are generated by the recruitment rate $\Pi e^{-\mu_0\phi_1}\varepsilon(1-p)(I_a+T_a)$, where $(1-p)$ are the proportion born with the virus, but not subjected to treatment. We assume that susceptible juveniles are not sexually active. We also assume that a significant proportion n of infected juveniles not under treatment though symptomatic, progress to early adulthood to cause infection. Therefore the maturation rate of infected juveniles not under treatment $\phi_2 = n\phi_1$, where $0<n<1$. This assumption is supported by [9, 10], where $0\leq\phi_2\leq 1$. The infected juveniles not under treatment are also assumed to progress to AIDS at a rate of ρ_1. We, therefore, have the dynamics of the infected juvenile not under treatment class expressed as

$$\dot{I}_j = \Pi\varepsilon e^{-\mu_0\phi_1}(1-p)(I_a+T_a) - (\mu+\phi_2+\rho_1)I_j. \tag{5}$$

Adults are assumed to transmit the infection horizontally and vertically. Therefore, infected adults not on treatment (I_a) and under-treatment (T_a) are assumed to be infectious. Let c be the average number of sexual contacts susceptible adults make with individuals from both infected adult classes per unit time. Not all contacts might result in an infection. Suppose $\bar{p}$ is the probability that an infection occurs per contact with an individual from either of the infected adult class, then the effective transmission rate of β is the product $c\bar{p}$. Thus, β is given by $\beta = c\bar{p}$. Sexually transmitted diseases like HIV/AIDS are usually driven by a standard incidence force of infection. The force of infection λ is given by $\lambda = \dfrac{\beta(I_a+\eta T_a)}{N_a}$, where η is the relative infectivity of T_a with respect to I_a. It is worth noting that η is the variable

that controls or accounts for the impact that each infected adult class have on the spread of HIV/AIDS since I_a and T_a may have a different possibility on vertical transmission of the diseases.

The susceptible adult class S_a is generated by maturation of susceptible juveniles at the rate ϕ_1. Individuals in this compartment transition into the infected adult class I_a at a rate λS_a, where $\lambda = \dfrac{\beta(I_a + \eta T_a)}{N_a}$. We therefore express the dynamics of the susceptible adult class as

$$\dot{S_a} = \phi_1 S_j - (\mu + \lambda) S_a. \tag{6}$$

The class of the infected adults not under treatment I_a is generated by the maturation of infected juveniles not under treatment at the rate ϕ_2 and the infection of the adults in S_a. Similarly, the infected adults under treatment class are generated by the maturation of infected juveniles under treatment at the rate of ϕ_1. Adults not under treatment are treated at a rate of τ_2. The infected adults not under treatment and infected adults under-treatment given progress to AIDS at rates respectively by ρ_3 and ρ_4. The infected individuals (both from I_a and T_a) on the AIDS class are assumed not to be involved with disease transmission, and thus, taken as redundant also die at a per-capita death rate of μ. The dynamics of both infected adult classes are given by

$$\dot{I_a} = \lambda S_a + \phi_2 I_j - (\mu + \tau_2 + \rho_3) I_a, \tag{7}$$

$$\dot{T_a} = \tau_2 I_a + \phi_1 T_j - (\mu + \rho_4) T_a. \tag{8}$$

From equation 7 above, we note that λS_a and $\phi_2 I_j$ are different even though both denote the only way by which I_a is recruited. While λS_a denote the rate at which susceptible adults become infected through physical contacts (mainly sexual), $\phi_2 I_j$ strictly denote how infected juveniles not under treatment mature to I_a, which does not involve any physical contact. One can argue that since HIV/AIDS is mostly dependent on or driven by sexual transmission, and not many juveniles without treatment mature to I_a as clarified by the proportion ϕ_2, then λS_a contribute more to the compartment of I_a than $\phi_2 I_j$. Similarly, albeit $\tau_2 I_a$ and $\phi_1 T_j$ being the two ways by which T_a is generated in equation 8, $\tau_2 I_a$ mainly denotes how adults infected from horizontal transmission transition to be treated while $\phi_1 T_j$ denotes the maturation of juveniles

in treatment from the vertical transmission into T_a.

Let $M = \Pi e^{-\mu_0 \phi_1}$, from the description of the dynamics of each class, the following system of ordinary differential equations is used to describe the transmission dynamics of HIV/AIDS in the presence of vertical transmission.

$$\left.\begin{aligned}
\dot{S}_j &= \Lambda + M(1-\varepsilon)(I_a + T_a) - (\mu + \phi_1)S_j, \\
\dot{I}_j &= M\varepsilon q(I_a + T_a) - (\phi_2 + \mu + \tau_1 + \rho_1)I_j, \\
\dot{T}_j &= p\varepsilon M(I_a + T_a) + \tau_1 I_j - (\phi_1 + \mu + \rho_2)T_j, \\
\dot{S}_a &= \phi_1 S_j - (\mu + \lambda)S_a, \\
\dot{I}_a &= \lambda S_a + \phi_2 I_j - (\mu + \tau_2 + \rho_3)I_a, \\
\dot{T}_a &= \tau_2 I_a + \phi_1 T_j - (\mu + \rho_4)T_a,
\end{aligned}\right\} \tag{9}$$

with initial conditions $S_j(0) = S_{j(0)}$, $I_j(0) = I_{j(0)}$, $T_j(0) = T_{j(0)}$, $S_a(0) = S_{a(0)}$, $I_a(0) = I_{a(0)}$, $T_a(0) = T_{a(0)}$, where all model parameter are assumed to be positive.

3. Model Properties

For convenience sake, we can rewrite the system (9) as

$$\left.\begin{aligned}
\dot{S}_j &= \Lambda + M(1-\varepsilon)(I_a + T_a) - Q_1 S_j, \\
\dot{I}_j &= M\varepsilon q(I_a + T_a) - Q_2 I_j, \\
\dot{T}_j &= p\varepsilon M(I_a + T_a) + \tau_1 I_j - Q_3 T_j, \\
\dot{S}_a &= \phi_1 S_j - (\mu + \lambda)S_a, \\
\dot{I}_a &= \lambda S_a + \phi_2 I_j - Q_4 I_a, \\
\dot{T}_a &= \tau_2 I_a + \phi_1 T_j - Q_5 T_a,
\end{aligned}\right\} \tag{10}$$

where

$$Q_1 = \mu + \phi_1, \;\; Q_2 = \mu + \phi_2 + \tau_1 + \rho_1, \;\; Q_3 = \mu + \phi_1 + \rho_2,$$

$$Q_4 = \mu + \tau_2 + \rho_3, \;\; Q_5 = \mu + \rho_4.$$

3.1. Existence and uniqueness of solution. For the model system (10) to be biologically, mathematically and epidemiologically meaningful, we prove that all the state variables of the model system will remain positive and that the solutions with positive initial conditions will remain positive for all $t > 0$.

Theorem 3.1. *There exist a unique and bounded solution of the system in a positive invariant set that remains finite* $\forall\ t \geq 0$. *Thus, the unique solution*

$$(S_j(t)I_j(t)T_j(t)S_a(t)I_a(t)T_a(t))$$

of the model system (10) defined in the open interval $(0,T)$ *with positive initial data will remain positive for all* $T > 0$ *for the set of initial data*

$$(S_j(0), I_j(0), T_j(0), S_a(0), I_a(0), T_a(0)).$$

We now consider the biological feasible region for the system (10) which is in $\mathbb{R}^6_+$ and is represented by the invariant region

$$\Omega = \left\{(S_j,\ I_j,\ T_j,\ S_a,\ I_a,\ T_a) \in \mathbb{R}^6_+ : 0 \leq\ N \leq \frac{\Lambda}{\mu - M}\right\},$$

in which the usual local existence, uniqueness and continuity of solutions holds. We note that here, M is always less than μ_0. This result is established by Lemma 3.2. The positive invariance can be established given that

$$\begin{aligned}\dot{N} &= \Lambda + M(I_a + T_a) - \mu N \\ &\leq \Lambda + MN - \mu N \\ &= \Lambda + (M - \mu)N.\end{aligned}$$

Integration yields,

$$N(t) \leq \left(N_0 + \frac{\Lambda}{M - \mu}\right)e^{(M-\mu)t} + \frac{\Lambda}{\mu + M}.$$

Lemma 3.2. $M < \mu$.

Proof. Suppose $M > \mu$, then

$$\begin{aligned}
\frac{\Pi}{e^{\mu_0\phi_1}} &> \mu, \text{ but } \mu_0 < \mu, \\
\mu &< \frac{\Pi}{e^{\mu_0\phi_1}}, \\
e^{\mu_0\phi_1} &< \frac{\Pi}{\mu} \Rightarrow \mu e^{\mu_0\phi_1} < \Pi, \text{ but,} \\
M &= \Pi e^{-(\mu_0\phi_1)} > \Pi e^{-(\mu\phi_1)}, \text{ since } \mu_0 < \mu, \\
N(t) &\leq \frac{\Lambda}{\mu - M} + \left(N_0 - \frac{\Lambda}{\mu - M}\right) e^{-(\mu - M)t},
\end{aligned}$$

we note, as

$$\lim_{t\to\infty} N(t) \leq \frac{\Lambda}{\mu - M}.$$

This means

$$\lim_{t\to\infty} N(t) \leq \frac{\Lambda}{\mu - M} < 0,$$

which is a contradiction, therefore $M < \mu$. □

Proposition 3.3. *The domain Ω is positively invariant. That is, the solution of system (10) is positive and remains or stays in Ω for all positive time, given initial conditions in Ω.*

Proof. Applying Birkhoff's theorem [3], the right hand side (R.H.S) of the model system (10) is continuous and therefore, the partial derivatives exist and are continuous. Hence, our model has a unique solution in $\mathbb{R}^6_+$ for $t \in [0,\infty)$ and the positive initial conditions.

We thus have

$$\left.\begin{aligned}
\dot{S}_j &\geq S_j(0)e^{-Q_1 t} > 0, \\
\dot{I}_j &\geq I_j(0)e^{-Q_2 t} > 0, \\
\dot{T}_j &\geq T_j(0)e^{-Q_3 t} > 0, \\
\dot{S}_a &\geq S_a(0)e^{-(\mu+\lambda)t} > 0, \\
\dot{I}_a &\geq I_a(0)e^{-Q_4 t} > 0, \\
\dot{T}_a &\geq T_a(0)e^{-Q_5} > 0.
\end{aligned}\right\} \tag{11}$$

It follows that

$$\lim_{t\to\infty} S_j(t) \geq 0,$$

and therefore

$$S_j(t) \geq 0, \ \ \forall t > 0.$$

Similarly, as $\lim_{t\to\infty}$, $I_j(t),\ T_j(t),\ S_a(t),\ I_a(t)$ and $T_a(t)$ are all positive for all $t > 0$. Likewise, $\lim_{t\to\infty}$, $I_j(t),\ T_j(t),\ S_a(t),\ I_a(t)$ and $T_a(t) \geq 0\ \forall t > 0$. Thus, for all time $t > 0$, the solution exist. Therefore the solutions of the model system remains in Ω for all positive time. The model system (10) is, therefore mathematically, epidemiologically and biologically well posed. □

4. Model Analysis

4.1. Infectious-free state. The infection-free steady state also known as the disease-free equilibrium of the model system (10) occurs when there is no disease. Thus, when $I_a = T_a = I_j = T_j = 0$. Setting the right hand side (RHS) of the model system (10) to 0, we have the infection-free state as

$$E^0 = \left(S_j^0, I_j^0, T_j^0, S_a^0, I_a^0, T_j^0\right) = \left(\frac{\Lambda}{Q_1},\ 0,\ 0,\ \frac{\phi\Lambda}{\mu Q_1},\ 0,\ 0\right), \tag{12}$$

4.2. Basic reproduction number with next generation matrix. Considering the next generation method for analysis with the model system (10), we have

$$F = \begin{bmatrix} 0 \\ 0 \\ \lambda S_a \\ 0 \end{bmatrix}, V = \begin{bmatrix} Q_2 I_j - M\varepsilon q(I_a + T_a) \\ Q_3 T_j - \tau_1 I_j - p\varepsilon M(I_a + T_a) \\ Q_4 I_a - \phi_2 I_j \\ Q_5 T_a - \phi_1 T_j - \tau_2 I_a \end{bmatrix}.$$

Rewriting F and V with $\lambda = \dfrac{\beta(I_a + \eta T_a)}{N_a}$,

$$F = \begin{bmatrix} 0 \\ 0 \\ \dfrac{\beta(I_a + \eta T_a)}{N_a} \\ 0 \end{bmatrix}, V = \begin{bmatrix} Q_2 I_j - M\varepsilon q(I_a + T_a) \\ Q_3 T_j - \tau_1 I_j - p\varepsilon M(I_a + T_a) \\ Q_4 I_a - \phi_2 I_j \\ Q_5 T_a - \phi_1 T_j - \tau_2 I_a \end{bmatrix}.$$

Finding the derivative of the F and V matrices with respect to the state variables and at the infection-free steady state (12), we have the F and V matrices as

$$F = \begin{bmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & \beta & \eta\beta \\ 0 & 0 & 0 & 0 \end{bmatrix}, V = \begin{bmatrix} Q_2 & 0 & -M\varepsilon q & -M\varepsilon q \\ -\tau_1 & Q_3 & -p\varepsilon M & -p\varepsilon M \\ -\phi_2 & 0 & Q_4 & 0 \\ 0 & -\phi_1 & -\tau_2 & Q_5 \end{bmatrix}.$$

We note that FV^{-1} is given by

$$FV^{-1} = \begin{bmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ e_1 & e_2 & e_3 & e_4 \\ 0 & 0 & 0 & 0 \end{bmatrix}.$$

The effective reproduction number is the spectral radius of FV^{-1} given by

$$\mathfrak{R}_o = \rho(FV^{-1}) = e_3,$$

$$\mathfrak{R}_o = \frac{\beta(Q_2Q_3Q_5 - M\varepsilon\phi_1(pQ_2 + q\tau_1)) + \eta\beta(Q_2Q_3\tau 2 + M\varepsilon\phi_1(pQ_2 + q\tau_1))}{Q_4(Q_2Q_3Q_5 - M\varepsilon\phi_1(pQ_2 + q\tau_1)) - M\varepsilon qQ_3(Q_5 + \tau_2)\phi_2}. \tag{13}$$

When we simplify further

$$\mathfrak{R}_o = \frac{\psi}{Q_4 - \phi_2\nu_1(1 + \nu_3)}, \tag{14}$$

where

$$\psi = \beta(1 + \eta\nu_3), \quad \nu_1 = \frac{M\varepsilon q}{Q_2}, \quad \nu_3 = \frac{Q_2Q_3\tau 2 + M\varepsilon(pQ_2 + q\tau_1)\phi_1}{Q_2Q_3Q_5(1 - \mathfrak{R}_v)}.$$

Expressing $\mathfrak{R}_o$ in terms of $\mathfrak{R}_v$ gives us

$$\mathfrak{R}_o = \frac{\beta}{Q_4(1 - \theta_3)} + \frac{\eta\beta(\tau_2 + Q_5\theta_1)}{Q_4Q_5(1 - \mathfrak{R}_v)(1 - \theta_3)}, \tag{15}$$

where

$$\mathfrak{R}_{\mathfrak{v}} = \frac{\phi_1(p\varepsilon MQ_2 + \tau_1 M\varepsilon q)}{Q_2Q_3Q_5}, \text{ and} \tag{16}$$

$$\theta_3 = \frac{M\varepsilon q\phi_2}{Q_2Q_4(1-\mathfrak{R}_{\mathfrak{v}})} + \frac{M\varepsilon q\tau_2\phi_2}{Q_2Q_4Q_5(1-\mathfrak{R}_{\mathfrak{v}})}, \text{ with } 0 < \theta_3 < 1.$$

The reproduction number $\mathfrak{R}_{\mathfrak{o}}$ is hence expressed as a function of $\mathfrak{R}_{\mathfrak{v}}$ (reproduction number due to vertical transmission of HIV/AIDS) in (15). Clearly if $\mathfrak{R}_{\mathfrak{v}}$ increases $\mathfrak{R}_{\mathfrak{o}}$ also increases.

4.3. Sexual transmission (Horizontal transmission). We adopt a similar approach by Kgosimore and Lungu [19], to analyze the reproduction number of the model system (10) in terms of horizontal transmission.

Without mother-to-child transmission (MTCT), $\varepsilon = 0$. This implies $\mathfrak{R}_{\mathfrak{v}} = 0$ and $\theta_3 = 0$. Therefore $\mathfrak{R}_{\mathfrak{o}}$ becomes

$$\mathfrak{R}_{\mathfrak{o}}^{'} = \frac{\beta}{Q_4} + \frac{\eta\beta\tau_2}{Q_4Q_5}. \tag{17}$$

Here, $\mathfrak{R}_{\mathfrak{o}}^{'}$ is the basic reproduction number for horizontal transmission.

Let the average number of individuals the infected class not under treatment I_a infects during their duration of infectiousness be denoted by $\mathfrak{R}_{\mathfrak{1}} = \frac{\beta}{\mu+\rho_3}$ and the average number of individuals the infected class under treatment T_a infects during their duration of infectiousness be denoted by $\mathfrak{R}_{\mathfrak{2}} = \frac{\eta\beta}{\mu+\rho_4}$.
From equation (17), we have that

$$\frac{\partial\mathfrak{R}_{\mathfrak{o}}^{'}}{\partial\beta} = \frac{1}{Q_4(1-\theta_3)} + \frac{\eta(\tau_2 + Q_5\mathfrak{R}_{\mathfrak{v}})}{(Q_4Q_5(1-\mathfrak{R}_{\mathfrak{v}})(1-\theta_3))^2}, \tag{18}$$

$$\frac{\partial \mathfrak{R}_o{}'}{\partial \tau_2} = \frac{-(\mu+\rho_3)(\beta-\mathfrak{R}_2)}{(\mu+\tau_2+\rho_3)^2}. \tag{19}$$

Thus, $\mathfrak{R}_o'$ is monotonic non-decreasing function of β and a decreasing function of τ_2 if and only if $\beta-\mathfrak{R}_2>0$, which implies $\beta>\mathfrak{R}_2$. Therefore $\mathfrak{R}_o>\beta>\mathfrak{R}_2$ is a prerequisite condition that needs to be satisfied in order to slow the spread of the disease.

The optimal treatment value to reduce the reproduction number below unity is given as

$$\tau_2^* = \frac{(\mu+\rho_4)(\beta-(\mu+\rho_3))}{(\mu+\rho_4)-\eta\beta},$$

that is

$$\tau_2^* = \frac{(\mu+\rho_3)(\mathfrak{R}_1-1)}{(1-\mathfrak{R}_2)}. \tag{20}$$

4.4. Mother-to-child transmission (MTCT) (Vertical transmission). Since MTCT is mainly from infected mothers during and after childbirth, the impact of MTCT can be investigated by looking at the dependence of the basic reproduction number $\mathfrak{R}_o$ on the reproduction number through vertical transmission $\mathfrak{R}_v$. Finding the derivative of $\mathfrak{R}_o$ with respect to $\mathfrak{R}_v$, we have

$$\frac{\partial \mathfrak{R}_o}{\partial \mathfrak{R}_v} = \frac{(Q_5+\tau_2)}{Q_4(1-\theta_3)(1-\mathfrak{R}_v)^2}\mathfrak{R}_2.$$

This implies $\mathfrak{R}_o$ is strictly a monotonic non-decreasing function of $\mathfrak{R}_v$. We define

$$\mathfrak{R}_v{}^* = \frac{Q_4((1-\theta_3)-\mathfrak{R}_o)}{Q_4(1-\theta_3)+(\eta\beta-\beta)},$$

to be the critical vertical transmission threshold number which must not be surpassed if we want to control the disease. We note that $\eta\beta-\beta<0$ and $(1-\theta_3)>0$, since $\mathfrak{R}_v<1$. By assumption, it can be seen that there is an $\mathfrak{R}_v{}^*$ for $\mathfrak{R}_o<1$ and $\eta\beta<\beta$ such that the impact of MTCT would be negligible for values $\mathfrak{R}_v<\mathfrak{R}_v{}^*$.

4.5. Local stability of the infection-free steady state. Local asymptotic stability of the infection-free state can be deduced by computing the model system (10) at the infection-free state E^0 using the Jacobian matrix J. We have

$$J_{E^0} = \begin{pmatrix} -Q_1 & 0 & 0 & 0 & M(1-\varepsilon) & M(1-\varepsilon) \\ 0 & -Q_2 & 0 & 0 & M\varepsilon q & M\varepsilon q \\ 0 & \tau_1 & -Q_3 & 0 & p\varepsilon M & p\varepsilon M \\ \phi_1 & 0 & 0 & -\mu & -\beta & -\eta\beta \\ 0 & \phi_2 & 0 & 0 & \beta - Q_4 & \eta\beta \\ 0 & 0 & \phi_1 & 0 & \tau_2 & -Q_5 \end{pmatrix}$$

After expanding the determinant of the characteristic equation $|J_{E^0} - \lambda I| = 0$ around column 1 row 1 and column 4 row 4, we have two eigenvalues $\lambda_1 = -Q_1$ and $\lambda_2 = -\mu$. The remaining of the eigenvalues are determined by the 4 x 4 matrix, given by the Jacobian matrix with the infected classes only, namely $I_a,\ T_a,\ I_j,\ T_j$. The eigenvalues of the 4 x 4 matrix

$$\begin{pmatrix} -Q_2 & 0 & M\varepsilon q & M\varepsilon q \\ \tau_1 & -Q_3 & p\varepsilon M & p\varepsilon M \\ \phi_2 & 0 & \beta - Q_3 & \eta\beta \\ 0 & \phi_1 & \tau_2 & -Q_5 \end{pmatrix},$$

are given by the quartic polynomial equation

$$P(\lambda) = a_0\lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4 = 0,$$

where

$$a_0 = 1,\ \ a_1 = Q_2 + Q_3 + Q_4 + Q_5 - \beta,\ \ a_2 = w_1 - (\beta(Q_2 + \mu + Q_1 + \eta\tau_2),$$

$$a_3 = w_2 - \beta(\eta\tau_2(Q_2 + Q_3) - Q_2Q_3 + Q_5(Q_2 + Q_3),\ \ a_4 = k(1 - \mathfrak{R}_o),$$

and

$$w_1 = Q_3Q_5(1 - \psi_1) + Q_2Q_4(1 - \psi_2) + (Q_2 + Q_4)(Q_3 + Q_5).$$

Here

$$\psi_1 = \frac{M\varepsilon p\phi_1}{Q_3Q_5}, \;\; \psi_2 = \frac{M\varepsilon q\phi_2}{Q_2Q_4}, \;\; \text{and} \;\; 0 < \psi_1 < 1, \; 0 < \psi_1 < 1,$$

$w_2 = Q_2Q_3Q_5(1-\Re_\mathfrak{v})Q_2Q_4Q_5(1-\theta_3) + Q_4(Q_3Q5(1-\psi_1)) + Q_3(Q_2Q_4(1-\psi_2)) + p\varepsilon M(\beta - \eta\beta)\phi_1$.

We notice from w_2 that $\beta > \eta\beta$ since $0 < \eta < 1$,

$$k = Q_2Q_3Q_4Q_5(1-\Re_\mathfrak{v})(1-\theta_3).$$

To show that the infection-free state is locally asymptotically stable, we show that $P(\lambda) = 0$ lies in the left half plane (has only negative roots) using the Routh-Hurwitz conditions for dimension four. We notice that $\Re_\mathfrak{o} < 1$ correspond to $a_4 > 0$, where k is positive given $\Re_\mathfrak{v} < 1$. Thus when $\Re_\mathfrak{o} < 1$, $a_4 > 0$. The condition $\Re_\mathfrak{o} < 1$ also gives $Q_2 + Q_3 + Q_4 + Q_5 + \mu > \beta$, hence $a_1 > 0$. Similarly, the condition $\Re_\mathfrak{o} < 1$ also gives $w_1 > \beta(Q_2 + \mu + Q_1 + \eta\tau_2)$. We also note that $w_2 > \beta(\eta\tau_2(Q_2+Q_3) - Q_2Q_3 + Q_5(Q_2+Q_3))$ when $\Re_\mathfrak{v} < 1$ with the condition $\Re_\mathfrak{o} < 1$. It means that $a_2 > 0$ and $a_3 > 0$.

We use the Routh-Hurwitz conditions for quartic equations to make our analysis, thus $a_i > 0$ for $i = 0,1,2,3,4$, $a_1a_2 - a_0a_3 > 0$ and $a_1a_2a_3 - a_1^2a_4 - a_0a_3^2 > 0$. We note here that all the coefficients of the polynomial $P(\lambda) = 0$ are greater than zero. That is $a_1 > 0$, $a_2 > 0$, $a_3 > 0$ and $a_4 > 0$. We now state the theory of local stability of the infection-free state E^0 using the Routh-Hurwitz conditions.

Theorem 4.1. *Let $\Re_\mathfrak{v} < 1$. The infection-free state E^0, whenever it exists, is locally asymptotically stable (l.a.s) for $\Re_\mathfrak{o} < 1$ provided that $a_1a_2 - a_0a_3 > 0$ and $a_1a_2a_3 - a_1^2a_4 - a_0a_3^2 > 0$ otherwise it is unstable.*

It can also be established by considering the fact that the reproduction number was determined by the next generation matrix approach.

4.6. Global stability of the infection-free steady state. We show that the infectious-free state (12) of the model system (10) is globally asymptotically stable (g.a.s). We adapt the concept of Lyapunov in [5].

Theorem 4.2. *If $\Re_o \leq 1$, then the infectious-free state is globally asymptotically stable (g.a.s) on Ω (our invariant set).*

Proof. Let us define a Lyapunov function $\mathbf{v}$ as $\mathbf{v}(I_j, T_j, I_a, T_a) = I_a + b_1 I_j + b_2 T_j + b_3 T_a$, where

$$b_1 = \frac{\beta\eta\tau_1\phi_1(Q_3Q_5(1-\psi_1))\phi_2}{Q_2Q_3Q_5(1-\Re_v)},\ b_2 = \frac{\phi_1(\eta\beta Q_2 + M\varepsilon q\phi_2)}{Q_2Q_3Q_5(1-\Re_v)},\ b_3 = \frac{Q_3(\eta\beta Q_2 + Mq\varepsilon\phi_2)}{Q_2Q_3Q_5(1-\Re_v)}.$$

Since, the state solutions of the model system (10) are positive, we have that $Q_2Q_3Q_5(1-\Re_v) > 1$. Therefore constants b_1, b_2, b_3, are all positive for $\Re_v < 1$ at the infection-free state.

We take the partial derivative of $\mathbf{v}$ with respect to t. Thus,

$$\frac{\partial \mathbf{v}}{\partial t} = \frac{\partial \mathbf{v}}{\partial I_a}\dot{I}_a + \frac{\partial \mathbf{v}}{\partial I_j}\dot{I}_j + \frac{\partial \mathbf{v}}{\partial T_j}\dot{T}_j + \frac{\partial \mathbf{v}}{\partial T_a}\dot{T}_a.$$

We therefore have

$\dot{\mathbf{v}} = \lambda S_a + \phi_2 I_j - Q_4 Ia + b_1(M\varepsilon q(I_a + T_a) - Q_2 I_j) + b_2(P\varepsilon M(I_a + T_a) + \tau_1 I_j - Q_3) + b_3(\tau_2 I_a + \phi_1 T_j - Q_5 T_a)$,
$\dot{\mathbf{v}} = \frac{\beta(I_a + \eta T_a)}{N_a} S_a + \phi_2 I_j - Q_3 Ia + b_1(M\varepsilon q(I_a + T_a) - Q_2 I_j) + b_2(P\varepsilon M(I_a + T_a) + \tau_1 I_j - Q_3) + b_3(\tau_2 I_a + \phi_1 T_j - Q_5 T_a)$. Given that at the infection-free state, $\frac{S_a}{N_a} \leq 1$
we have an expression in only I_a after simplifying and substituting b_1, b_2, b_3,
$\dot{\mathbf{v}} = \left[\beta + \frac{\eta\beta(\tau_2 + Q_5\Re_v)}{Q_5(1-\Re_v)} - Q_4(1-\theta_3)\right] I_a$,
$\dot{\mathbf{v}} = \Gamma(\Re_o - 1) I_a$, where $\Gamma = Q_4(1-\theta_3)$.

Γ is positive since, $0 < \theta_3 < 1$, when $\Re_v < 1$. All the model parameters are positive and the state variables are non-negative, therefore it follows that $\dot{\mathbf{v}} \leq 0$ for $\Re_o \leq 1$ with $\dot{\mathbf{v}} = 0$ if and only if $I_a = 0$ or $\Re_o = 1$. Therefore $\dot{\mathbf{v}}$ is a Lyapunov function on Ω. Since, the set is compact,

positively invariant, we deduce by LaSalle's Invariance Principle [5] that

$$(I_a,\ T_a,\ I_j,\ T_j) \to (0,\ 0,\ 0,\ 0), \tag{21}$$

therefore the infectious-free state is globally asymptotically stable. Since

$$\lim_{t\to\infty} \sup I_a = 0,$$

and

$$\lim_{t\to\infty} \sup T_a = 0,$$

it suffices that for adequately small $\bar{\gamma} > 0$ there exist constant A_1 and A_2 such that

$$\lim_{t\to\infty} \sup I_a \leq \bar{\gamma}$$

for all $t > A_1$ and

$$\lim_{t\to\infty} \sup T_a \leq \bar{\gamma}$$

for all $t > A_2$. Hence, from the second equation in the model system (10), we have that for $t > \max\left\{A_1, A_2\right\}$

$$\dot{I}_j \leq 2M\varepsilon q\bar{\gamma} - Q_1 I_j, \tag{22}$$

thus by comparison theorem [30],

$$I_j^{\infty} = \lim_{t\to\infty} \sup I_j \leq \frac{2M\varepsilon q\bar{\gamma}}{Q_1}, \tag{23}$$

so that if $\bar{\gamma} \to 0$,

$$I_j^{\infty} = \lim_{t\to\infty} \sup I_j \leq 0. \tag{24}$$

Similarly, it can be shown that

$$I_{j\infty} = \lim_{t\to\infty} \sup I_j \geq 0, \tag{25}$$

by (24) and (25),

$$I_j^{\infty} \leq 0 \leq I_{j\infty}, \tag{26}$$

which implies

$$\lim_{t\to\infty} I_j = 0. \tag{27}$$

It can also be shown that

$$T_j^{\infty} \leq 0 \leq T_{j\infty}, \tag{28}$$

and that

$$\lim_{t\to\infty} T_j = 0, \tag{29}$$

likewise

$$\lim_{t\to\infty} S_{j(t)} = \frac{\Lambda}{Q_1}, \tag{30}$$

$$\lim_{t\to\infty} S_{a(t)} = \frac{\phi\Lambda}{\mu Q_1}. \tag{31}$$

Therefore it suffices that from (21), (27), (29), (30), (31) as $\lim_{t\to\infty} \Re_o < 1$, all solution of the model system (10) with positive initial conditions in Ω approaches the infection-free state E^0.

4.7. Endemic equilibrium. The persistent steady state of the model system (10) also commonly known as the endemic state is given as

$$E^* = \left(S_j^*,\, I_j^*,\, T_j^*,\, S_a^*,\, I_a^*,\, T_j^*\right), \tag{32}$$

where

$$\begin{aligned}
S_j^* &= \frac{\Lambda}{Q_1} + \frac{\nu_4(1+\nu_3)\Re_o(\Re_o-1)\phi_1\Lambda}{Q_1(\mu\Re_o(1+\nu_3)+(\Re_o-1)\beta(1+\eta\nu_3)(1-\theta_2))},\\
S_a^* &= \frac{\phi_1\Lambda\Re_o(1+\nu_3)}{Q_1(\mu\Re_o(1+\nu_3)+(\Re_o-1)\beta(1+\eta\nu_3)(1-\theta_2))},\\
I_a^* &= \frac{\Re_o(\Re_o-1)\phi_1\Lambda}{Q_1(\mu\Re_o(1+\nu_3)+(\Re_o-1)\beta(1+\eta\nu_3)(1-\theta_2))},\\
T_a^* &= \frac{\phi_1\Lambda\Re_o(\Re_o-1)\nu_3}{Q_1(\mu\Re_o(1+\nu_3)+(\Re_o-1)\beta(1+\eta\nu_3)(1-\theta_2))},\\
I_j^* &= \frac{\phi_1\Lambda\Re_o(\Re_o-1)\nu_1(1+\nu_3)}{Q_1(\mu\Re_o(1+\nu_3)+(\Re_o-1)\beta(1+\eta\nu_3)(1-\theta_2))},\\
T_j^* &= \frac{\phi_1\Lambda\Re_o(\Re_o-1)\nu_2(1+\nu_3)}{Q_1(\mu\Re_o(1+\nu_3)+(\Re_o-1)\beta(1+\eta\nu_3)(1-\theta_2))}.
\end{aligned} \tag{33}$$

$$\nu_1 = \frac{M\varepsilon q}{Q_2},\ \ \nu_2 = \frac{M\varepsilon(pQ_2+q\tau 1)}{Q_2Q_3},\ \ \nu_3 = \frac{Q_2Q_3\tau 2 + M\varepsilon(pQ_2+q\tau_1)\phi_1}{Q_2Q_3Q_5(1-\Re_\mathfrak{v})},$$

$$\nu_4 = \frac{M(1-\varepsilon)}{Q_1(1-\Re_{\mathfrak{v}})} + \frac{M(1-\varepsilon)\tau_2}{Q_1Q_5(1-\Re_{\mathfrak{v}})}, \;\; \theta_2 = \frac{M(1-\varepsilon)\phi_1}{Q_1Q_4(1-\omega)} + \frac{M(1-\varepsilon)\tau_2\phi_1}{Q_1Q_4Q_5(1-\omega)},$$

$$\omega = \frac{M\varepsilon p\phi_1}{Q_1Q_3Q_5} + \frac{M\varepsilon q\tau_1\phi_1}{Q_1Q_2Q_3Q_5} + \frac{M\varepsilon q\phi_2}{Q_1Q_2Q_4} + \frac{M\varepsilon q\tau_2\phi_2}{Q_1Q_2Q_4Q_5}.$$

We also have that

$$\lambda^* = \frac{Q_4Q_5(1-\Re_{\mathfrak{v}})(1-\theta_3)(\Re_{\mathfrak{o}}-1)}{\Re_{\mathfrak{o}}(Q_5+\tau_2)}, \;\; \text{where} \;\; \theta_3 = \frac{M\varepsilon q(Q_5+\tau_2)\phi_2}{Q_2Q_4Q_5(1-\Re_{\mathfrak{v}})},$$

and $0 < \theta_2 < 1$, $0 < \theta_3 < 1$, $0 < \omega < 1$ with $\Re_{\mathfrak{v}} < 1$.

4.8. Existence and uniform persistent of the endemic state. The model system (10) is said to be uniformly persistent if there exist a constant c such that

$$\lim_{t\to\infty} S_{j(t)} > c, \;\; \lim_{t\to\infty} I_{j(t)} > c,$$

$$\lim_{t\to\infty} T_{j(t)} > c, \;\; \lim_{t\to\infty} S_{a(t)} > c,$$

$$\lim_{t\to\infty} I_{a(t)} > c, \;\; \lim_{t\to\infty} T_{a(t)} > c,$$

provided $(S_j(0),\ S_a(0),\ I_j(0),\ T_j(0),\ I_a(0),\ T_a(0) \in \Omega^o)$, where Ω^o is the interior of Ω. The constant c does not depend on the initial conditions in Ω.

Theorem 4.3. *Assume that $\Re_{\mathfrak{o}} > 1$ then the model system (10) is persistent in Ω^o*

Proof. The theorem can be proved by applying a persistence result in [12] and by using the approach used to prove Proposition 3.3 of [21]. Given that $\Re_{\mathfrak{o}} > 1$, it follows that the model system (10) is persistent; by applying Theorem 2.8.6 in [32], we have that the model system (10), has at least one persistent state in Ω^o. We establish the following result.

Lemma 4.4. *Whenever $\Re_{\mathfrak{o}} > 1$, we have at least one persistent state given by E^**

□

5. NUMERICAL SIMULATIONS

5.1. Parameter Estimation. In this section, we give the results of numerical simulations for the model system (10). The life expectancy of Ghana from [27] is 62.4 giving us the death rate μ as 0.016. Only a handful of parameters are known, it is therefore imperative to estimate the others. The estimation process seeks to find the best concordance between the observed and computed data. To illustrate the usefulness of the model system (10), we determined the population dynamics of the juvenile and adult population when the reproduction number $\mathfrak{R}_o$ is less than one and when it is greater than one. These simulations were done in Matlab (initially with version R2018a and later with version R2018b). The least squares-curve fitting method in Matlab was used to estimate parameters that gave the best fit. The unknown parameter values in the Matlab codes are allocated upper and lower bounds. Parameter values that gave the best fit were obtained from within the bounds. The range of the parameter values and their sampled values used in the simulations and data fitting are shown in Table 1. The initial populations for juveniles and adults were estimated close to the population of Ghana [27]. That is, $S_J(0) = 10000000,\ S_A(0) = 18000000$. Recruitment Λ was taken to be 1600000.

In Figure 1, we note that the model approaches the stable infection-free HIV state. The infected populations asymptotically converge to zero while the susceptible adult population increases and susceptible juvenile population decreases as a result of progression to the susceptible adult population. This is a graphical description of the fact that the infection-free state is locally stable for $\mathfrak{R}_o < 1$ and unstable otherwise.

We observe in Figure 2 that the populations are at a stable endemic state where both susceptible populations decrease asymptotically to zero. This is also a graphical description of the fact that the persistent state is locally stable for $\mathfrak{R}_o > 1$ and unstable otherwise.

TABLE 1. Parameter values and range obtained from the best fit, where units are yr^{-1}

Parameter	**Range**	**Estimated values**
Π	0.3	[19, 23]
Λ	-	1600000
ε	(0.1, 0.5)	0.5.
p	(0.059, 0.1)	0.1
ϕ_1	(0.05, 0.5)	0.5
ϕ_2	($2.1\text{x}10^{-5}$, $9.0\text{x}10^{-5}$)	$9.0\text{x}10^{-5}$
τ_1	(0.0998, 0.4)	0.4
τ_2	(0.1, 0.9)	0.5812
μ_0	(0.001, 0.0054)	0.0054
μ	0.016	[27]
η	(0.1, 0.5)	0.5
β	(0.3, 0.89)	0.890
ρ_1	(0.010, 0.03)	0.030
ρ_2	(0.008, 0.2)	0.02
ρ_3	(0.115, 0.3)	0.1150
ρ_4	(0.088, 0.2)	0.1211

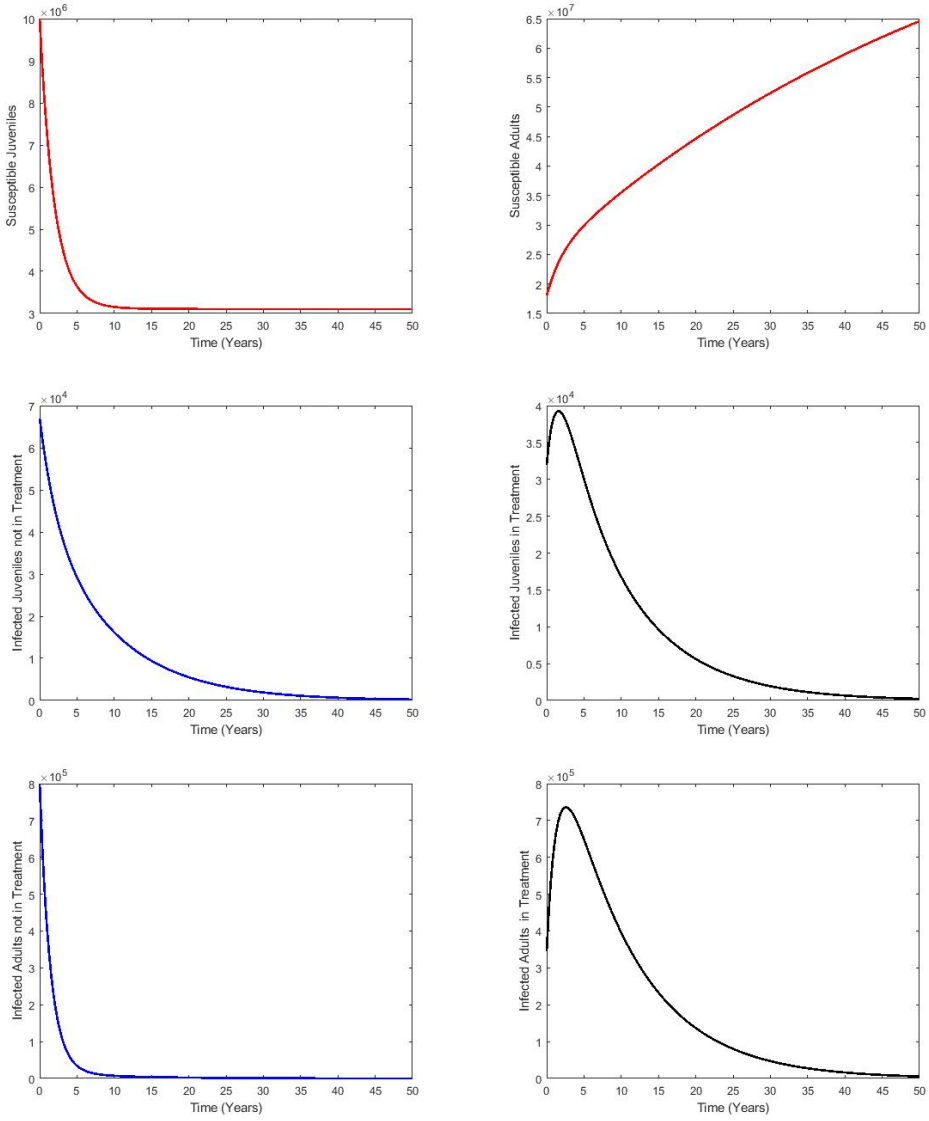

FIGURE 1. Shows the result of simulations for the given parameter values $\varepsilon = 0.5,\ \phi_1 = 0.5,\ \phi_2 = 0.00009,\ \tau_1 = 0.4,\ \tau_2 = 0.5812, p = 0.1,\ \eta = 0.5,\ \beta = 0.02, \mu_0 = 00.54,\ \rho_1 = 0.03,\ \rho_2 = 0.02,\ \rho_3 = 0.1150,\ \rho_4 = 0.1211.\ SJ0 = 10000000,\ SA0 = 18000000$ for Ghana. $\Re_o = 0.0951$. The value of $\Re_o$ here indicates a stable infection-free state, thus, $\Re_o < 1$ for the given parameter values which are further confirmed by the graphical representation.

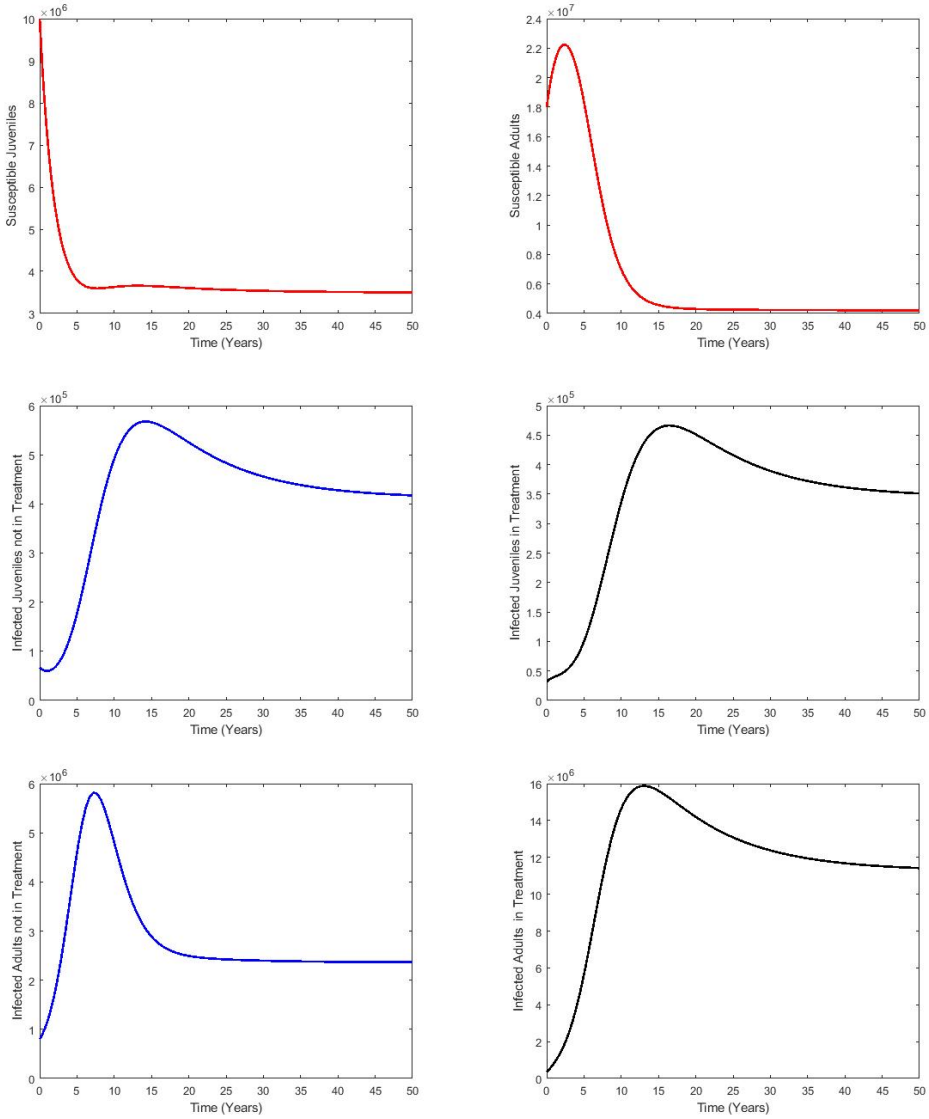

FIGURE 2. Shows the result of simulations for the given parameter values $\varepsilon = 0.5,\ \phi_1 = 0.5,\ \phi_2 = 0.00009,\ \tau_1 = 0.4,\ \tau_2 = 0.5812, p = 0.1,\ \eta = 0.5,\ \beta = 0.89, \mu_0 = 00.54,\ \rho_1 = 0.03,\ \rho_2 = 0.02,\ \rho_3 = 0.1150,\ \rho_4 = 0.1211.\ SJ0 = 10000000,\ SA0 = 18000000$ for Ghana. $\mathfrak{R}_o = 4.2315$. The value of $\mathfrak{R}_o$ here depicts a persistent state, thus, $\mathfrak{R}_o > 1$ for given parameter values and is also confirmed by the graphical representation.

5.2. Latin hypercube sampling. In this research, we examine the sensitivity of the reproduction number $\mathfrak{R}_o$ to the changes or variations in parameters. Latin hypercube sampling and partial rank correlation coefficients (PRCCs) were adopted together with 1000 simulations per

run in Matlab. Latin hypercube sampling is a statistical sampling method that allows for an effective investigation of changes in parameters across simultaneous uncertainty ranges in each parameter (Blower and Dowlatabadi 1994). PRCCs illustrate the magnitude of the impact that each parameter has on the outcome of the reproduction number $\mathfrak{R}_o$. The reproduction number $\mathfrak{R}_o$ increases when parameters having positive PRCCs increases whiles parameters having negative PRCCs decreases $\mathfrak{R}_o$ when they are increased.

The parameter values listed in the final column of Table 1 were estimated to determine the appropriate parameter ranges. Results from this indicate that τ_2 and β were the two parameters with the most significant impact on the output of the reproduction number $\mathfrak{R}_o$. Figure 3 also confirms these results.

Results from Figure 4 show the variation in $\mathfrak{R}_o$ with respect to the parameters τ_2 and β. A a positive correlation exist between $\mathfrak{R}_o$ and β while a negative correlation exists between τ_2 and $\mathfrak{R}_o$ indicating that efforts must be made to increase compliance to significant treatment protocols already in place to enroll more people living with HIV/AIDS in Ghana into treatment to reduce the burden of the disease. This is not far fetched and can be achieved since about 90% of women are already enrolled on treatment [26]. This will aid in the total elimination of vertical transmission in Ghana.

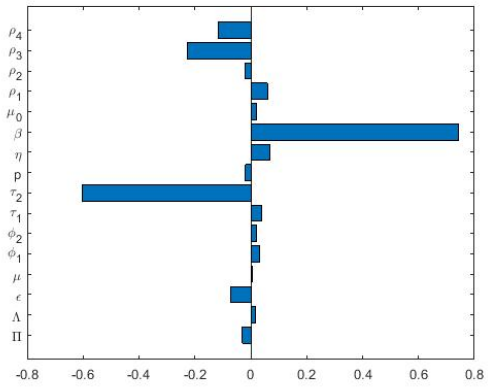

FIGURE 3. Partial Rank Correlation Coefficients (PRCCs) for the range of parameters from 1. It can be noted that the parameter with the highest potential to reduce the epidemics is τ_2. While β is the parameter with the highest potential to make the epidemic worse when it is maximized. Thus, the influence of τ_2 is critical in reducing HIV epidemics in a population.

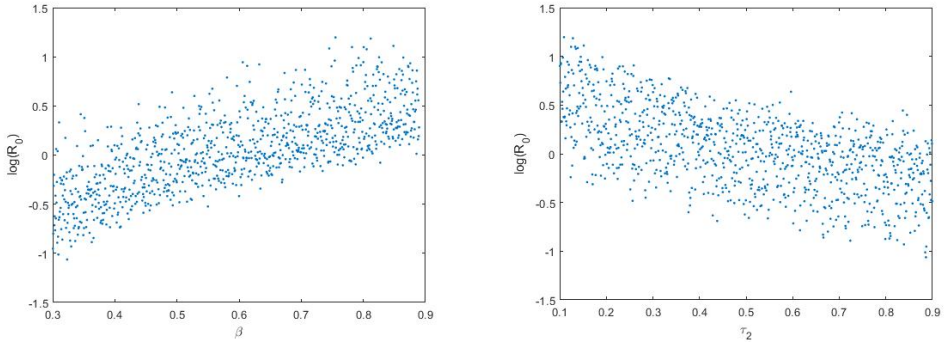

FIGURE 4. shows the scatter plot of the sensitive parameters τ_2 and β

6. APPLICATION TO DATA FROM GHANA

In this section, the model system (10) is fitted to data obtained from Ghana from 2003-2016 on the total number of individuals enrolled in HIV/AIDS antiretroviral therapy (ART). The original data set was multiplied by 1000 to get the estimated fit. Table 2 shows the data obtained from the Ghana Health Service on the number of children and adults enrolled in ART from 2003-2016.

TABLE 2. Data set for the number of people enrolled on ART in Ghana from 2003-2016

Year	**Children on ART**	**Adults on ART**	Total
2003	0	197	197
2004	27	1804	1831
2005	119	1913	2032
2006	122	3156	3278
2007	308	5783	6091
2008	450	9735	10185
2009	722	9409	10131
2010	894	12920	13814
2011	942	13441	14383
2012	684	13648	14332
2013	843	13456	14299
2014	1185	13809	14994
2015	1093	15875	16968
2016	1390	15107	16497

Our results in Figure 5 are indicative of a consistent increment of the total number of individuals who enrolled for treatment in Ghana each year. The projected HIV prevalence can be seen in Figure 6. We noticed a decline in prevalence which can be attributed to better government policies put in place on HIV/AIDS management and control. This can be justified since Ghana has the lowest population-based HIV Prevalence of 2.0% according to the Ghana Demographic Health Survey report (GDHS, 2014) [13]. There also exist many strategic plans in place by United Nations Program on HIV and AIDS (UNAIDS) to reduce the prevalence and the burden of the disease in countries by enrolling more people into treatment by 2020 [33].

In Figure 7, we noticed an increment in the reproduction number $\mathfrak{R}_o$ and the infected compartments as we increase the progression rates as seen from the contour plot and time series plot

respectively. This suggests that HIV infected individuals will continue to increase in the long run so long as these progression rates keep increasing, and decreasing when these rates decrease.

We can also notice in Figure 8 that as more HIV infected individuals are enrolled in treatment, the reproduction number, as well as the number of infected victims, decreases substantially. Particularly, we can see the effect that enrolling people into treatment has on the infected classes in the time series plot in Figure 8. The effect of vertical transmission on the treated population was also examined. We note that increment in $\mathfrak{R}_{\mathfrak{v}}$ does not significantly increase the treated populations.

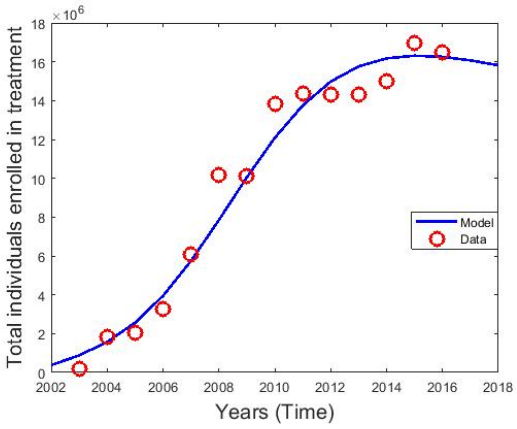

FIGURE 5. Model system (10) fitted to data for individuals enrolled in treatment for HIV/AIDS. The red circles indicate the actual data and the solid blue line indicates the model fit to the data.

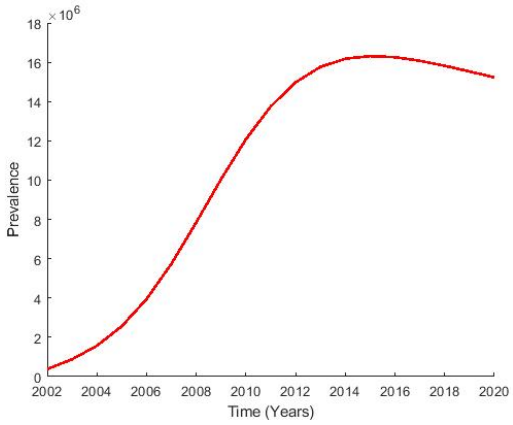

FIGURE 6. Projected Total HIV prevalence in Ghana

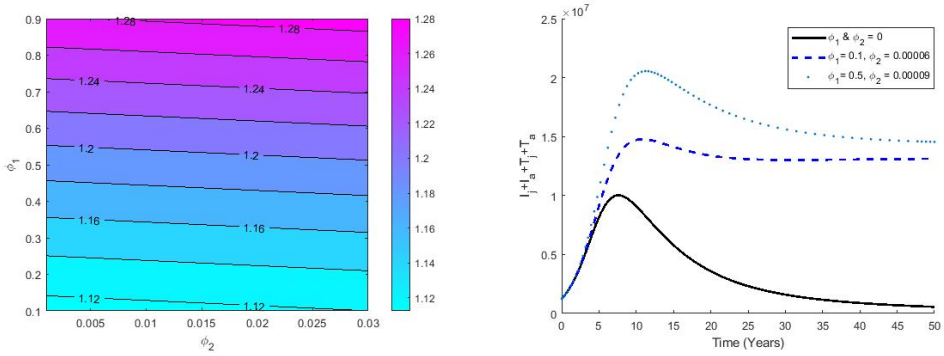

FIGURE 7. Shows the contour plot of ϕ_1 and ϕ_2 and the plot of varying of the progression parameters, ϕ_1 and ϕ_2.

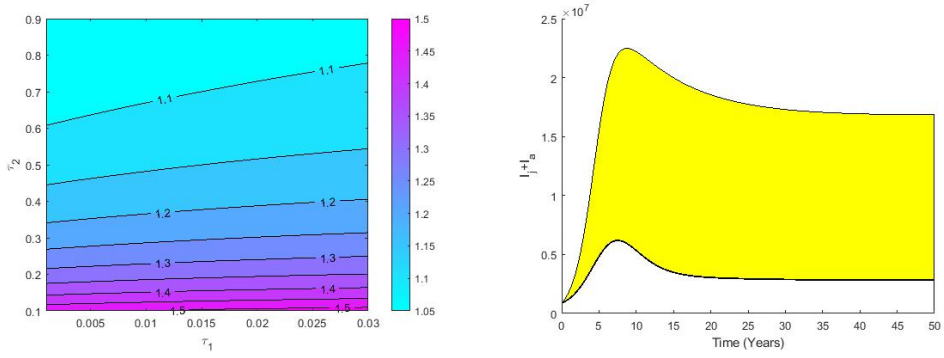

FIGURE 8. Contour plot of the basic reproduction number $\mathfrak{R}_o$ as a function of τ_1 and τ_2 and the plot of the influence of τ_1 and τ_2 on the infected non-treated population

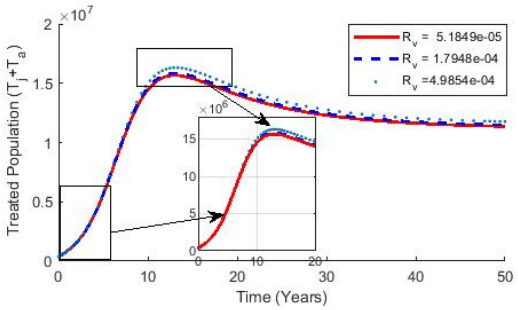

FIGURE 9. Shows the influence of reproduction number through vertical transmission $\mathfrak{R}_\upsilon$ on the treatment class, that is the HIV epidemic in the presence of vertical transmission.

7. DISCUSSION, CONCLUSION AND FUTURE WORK

In this paper, we proposed and analyzed a deterministic non-linear ordinary differential equation model for HIV/AIDS in Ghana. This model included juvenile and adult populations. Treatment of juveniles infected with HIV/AIDS via mother-to-child transmission (vertical) and treatment of adults were included in both juvenile and adult populations respectively. We studied the local stability of the infection-free state for the model system (10). From the steady-state analysis, we determined the reproduction number for the model system (10), which from the analysis of the model reproduction number was found to be an increasing function of the effective transmission rate β and a decreasing function of the treatment rate τ_2 if $\beta > \mathfrak{R}_2$ (the parameter which measures the average number of individuals that infectives in the treated adult population infect during their duration of infectiousness). We also found out that the basic reproduction number $\mathfrak{R}_o$ is an increasing function of $\mathfrak{R}_\upsilon$ (the reproduction number due to the mother-to-child-transmission of HIV/AIDS).

It was noted that without treatment programs, HIV-positive mothers have a higher risk of transmitting HIV to their child through birth or after birth. However, in the presence of treatment protocols such as early diagnosis and enrollment of infected juveniles and mothers on ART or antiretroviral prophylaxis, the burden of HIV reduces significantly as seen in both plots in Figure 8. This tends to suggest adherence to PMTCT programs needs to be intensified

and sustained to achieve this reduction.

It was determined that the progression rates of infected juveniles whether on treatment or not significantly increase the total infected classes. This suggests that the admission of infected juveniles into early adulthood can have a significant impact on the reproduction number of HIV and the over-all total number of people infected as seen in both plots in Figure 7.

It was also noted that vertical transmission of HIV/AIDS alone cannot lead to an epidemic. However, increment in vertical transmission without treatment can lead to a high burden of the disease among juveniles. With treatment, however, the disease burden among juveniles can still be kept under control even if vertical transmission increases as seen in Figure 9.

Sensitivity analysis for parameters of the model system (10) was also considered. Latin hypercube sampling and partial rank correlation coefficients (PRCCs) demonstrated that the two parameters with the most significant impact on the reproduction number $\mathfrak{R}_o$ and the behaviour of the population variables are τ_2 and β, the treatment and effective transmission rate respectively. Again, from the partial rank correlation coefficients (PRCCs) plot in Figure 3, we note that the progression rate to AIDS for both infected adults in treatment and not in treatment, ρ_3 and ρ_4 have a negative impact on $\mathfrak{R}_o$. This implies, that the HIV infected adult classes reduce as more people from these classes progress to AIDS. We also observed in Figure 8 that there is no significant correlation between the treatment rate of juveniles τ_1 and $\mathfrak{R}_o$, whiles increasing τ_2 reduces $\mathfrak{R}_o$ significantly. Epidemiologically, it means the treatment of juveniles does not have any significant impact on the secondary infection of HIV/AIDS. Treatment of adults, on the other hand, does significantly.

The model system (10) was also fitted to data on individuals enrolled in treatment. The objective was to utilize the model parameters that give the best fit to obtain the projected future prevalence of HIV/AIDS as seen in Figure 6. The model projected a decline in the prevalence of HIV in Ghana. The decline was observed from 2016 towards 2020 and gradually stabilizing

after 2020 given all intervention programs are kept in place, sustained and also more infected HIV mothers, juveniles and other HIV victims are enrolled through the intervention programs. This result is consistent with [18]. This suggests that more individuals would be enrolled in treatment towards 2020 since ARTs reduces the viral load of HIV patients, the effective transmission rate of HIV will decline, eventually reducing the reproduction number and HIV prevalence. It follows, therefore, that even though the prevalence rate of HIV in Ghana is low, persistent efforts must be intensified. These efforts must be made by the health policy-makers for continuous sustainability and compliance with treatment protocols against HIV/AIDS. The objective is to reduce the disease burden, to eliminate vertical transmission of the disease, and make the disease evade the Ghanaian population.

In conclusion, the transmission rate and the rate at which adults and juveniles are enrolled in treatment significantly reduces the infected class and the reproduction number. Hence, the need to sustain and ensure high and continuous compliance with treatment programs. Progression rates of infected juveniles either in treatment or not increases both infected adult classes and the reproduction number as well. This tends to suggest higher progression rates of infected juveniles will increase the HIV epidemics in a population. We also observed that vertical transmission of HIV alone cannot lead to HIV epidemics and the disease can be kept under control among the treated population even if $\mathfrak{R}_{\mathfrak{v}}$ increases. Finally, the model projected a fall in the prevalence rate of HIV in Ghana which is observed from 2016 towards 2020.

This work cannot be without limitations. We used estimates for some parameters due to the unavailability of published data on those parameters. Consequently, we resorted to a computational iterative method to provide the estimates of these parameters. The model could have been extended by the inclusion of some determinant factors significant to HIV epidemiology, such as variable infectivity, genetic variation, sex-structures, population density, mixing patterns, and time from infection to AIDS. These constraints raised above will be taken into account in future work to provide for a better model and a more realistic estimation of parameters.

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CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

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