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EVALUATING THE DETERMINISTIC SEIRUS MODEL FOR DISEASE CONTROL IN AN AGE-STRUCTURED POPULATION

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ABSTRACT

This paper focuses on the development and analysis of the endemic model for disease control in an aged-structured population in Kenya. Upon the model framework development, the model equations were transformed into proportions with rate of change of the different compartments forming the model, thereby reducing the model equations from twelve to ten homogenous ordinary differential equations. The model exhibits two equilibria, the endemic state and the disease-free equilibrium state while successfully achieving a Reproductive Number $R_0 = 0$. The deterministic endemic SEIRUS model is analyzed for the existence and stability of the disease-free equilibrium state. Numerical simulations were carried to complement the analytical results in investigating the effect treatment rate and the net transmission rate on recovery for both juvenile and adult sub-population in an age-structured population.

Keyword: Susceptible, Exposed, Latent, Infectious, Removed, Recovery, Undetectable.

1. INTRODUCTION

This study aims to develop and evaluate the new deterministic endemic age-structured SEIRUS compartmental model of the HIV/AIDS dynamics. As a result, a two-age-structured population framework for a deterministic endemic model is constructed for the development of an endemic deterministic model with Undetectable=Untrasmittable viral load compartment.

Previous studies (Oduwole, H. K. and Kimbir, A. R. (2018) and Mugisha, J.Y.T. and Luboobi, L.S. (2003)) have focused on the epidemiology of HIV/AIDS using various models like SIR, SEIR, SIRS, SICA models which are formulated for epidemic case of disease control. These models however, do not take into account the endemic nature/state of the diseases therefore making unrealistic to effectively control the further spread and eventual eradication of the disease. This

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has led to the setbacks of various interventions for the eradication of HIV/AIDS which is also a focal point of the global Sustainable Development Goals (SGDs).

However, in this paper the endemic nature of the disease with a new endemic deterministic model is investigated with an inclusion of the Undetectable=Untrasmitable (U=U) viral load compartment of the age-structured population.

2. THE MODEL VARIABLES AND PARAMETERS

The model variables and parameters for the investigation of the stability analysis of the equilibrium state for the new deterministic endemic model which is a motivation from [1] is given by;

Variable	Description
$S_1(t)$	Number of susceptible juveniles at time t
$S_2(t)$	Number of susceptible adult at time t
$E_1(t)$	Number of exposed juvenile at time t
$E_2(t)$	Number of exposed adults at time t
$I_1(t)$	Number of infected juveniles at time t
$I_2(t)$	Number of infected adults at time t
$R_1(t)$	Number of infected juveniles receiving HAART at time t
$R_2(t)$	Number of infected adults receiving HAART at time t
$U_1(t)$	Number of recovered juveniles satisfying U=U case at time t
$U_2(t)$	Number of recovered adults satisfying U=U case at time t
$A_1(t)$	Number of AIDS cases in the juvenile sub-population at time t
$A_2(t)$	Number of AIDS cases in the adult sub-population at time t
Parameter	**Description**
ν_1	The rate at which HIV infected juveniles becomes AIDS patient.
ν_2	The rate at which HIV infected adults becomes AIDS patient.
λ	Birth rate of the adult sub-population
μ_1	Natural death rate of the juvenile sub-population
μ_2	Natural death rate of the adult sub-population
α_0	Maximum death rate due to AIDS. $(\alpha_i \leq \alpha_0), i = 1, 2$
α_1	Death rate of infected juvenile sub-population due to AIDS
α_2	Death rate of infected adult sub-population due to AIDS
φ_1	Disease induced death rate of infected juveniles not receiving HAART
φ_2	Disease induced death rate of infected adults not receiving HAART
ϖ_1	Disease induced death rate of infected juveniles receiving HAART
ϖ_2	Disease induced death rate of infected adults receiving HAART
τ_1	Disease induced death rate of recovered juvenile not receiving HAART
τ_2	Disease induced death rate of removed adults not receiving HAART
T	Maximum lifespan after infection ($T = 10$ years)
k	Efficacy of HAART $(0 \leq k \leq 1)$
c	Average number of sexual partners of adult members of class I_2
c'	Average number of sexual partners of adult members of class R_2

β	Probability of transmission by adult members of class I_2
β'	Probability of transmission by adult members of class R_2
ρ_1	Probability of secondary infection by recovered juveniles population in U=U
ρ_2	Probability of secondary infection by recovered adults population in U=U
σ_1	Proportion of infected juveniles receiving HAART per unit time (Treatment rate)
σ_2	Proportion of infected adults receiving HAART per unit time (Treatment rate)
π_1	Proportion of juvenile population from susceptible to exposed/latent class
π_2	Proportion of adult population from susceptible to exposed/latent class
ε_1	Proportion of removed juveniles still receiving treatment and being moved to susceptible class
ε_2	Proportion of removed adults still receiving treatment and being moved to susceptible class
$1-\xi$	Proportion of healthy newborns from infected mothers
ξ	Proportion of infected newborns from infected mothers
$B_1(t)$	Incidence rate in the juvenile sub-population. $B_1(t) = 0$ (no sexual contact)
$B_2(t)$	Incidence rate or force of infection in the adult sub-population
η_j	Maturation rate from Juveniles to adults (where $j = s$ denotes the susceptible population, $j = e$ denotes the exposed/latent population, $j = i$ denotes the infected population, $j = r$ denotes the removed population and $j = u$ denotes the undetectable=untransmitable population)
m	Fixed ratio of adults to juveniles, $m = \frac{N_2}{N_1}$

2.2 MODEL ASSUMPTIONS

The following assumptions would help in the derivation of the model:

1. There is no emigration from the total population and there is no immigration into the population.
2. The susceptible population are first exposed to a latent class where they can infected or not.
3. Some infected individuals move to the removed class when counseled and are placed under highly active antiretroviral therapy (HAART).
4. Newborns are not of the same class as their progenitor. A fraction $(1-\xi)$ of newborns from infected mothers are healthy, while the remaining fractions ξ are born with the virus.
5. The rate of progression from HIV to AIDS is different for both juvenile and adult sub-populations.
6. The AIDS cases have full-blown symptoms and are therefore not sexually active.
7. The recruitment into the S-class is only through birth for the juvenile sub-population and through maturation for the adult sub-population.
8. The recruitment from the S-class into the E-class is through birth for newborns and through heterosexual activities for adults. This is done at a rate π_1 and π_2 for the juvenile and adult sub-population respectively.

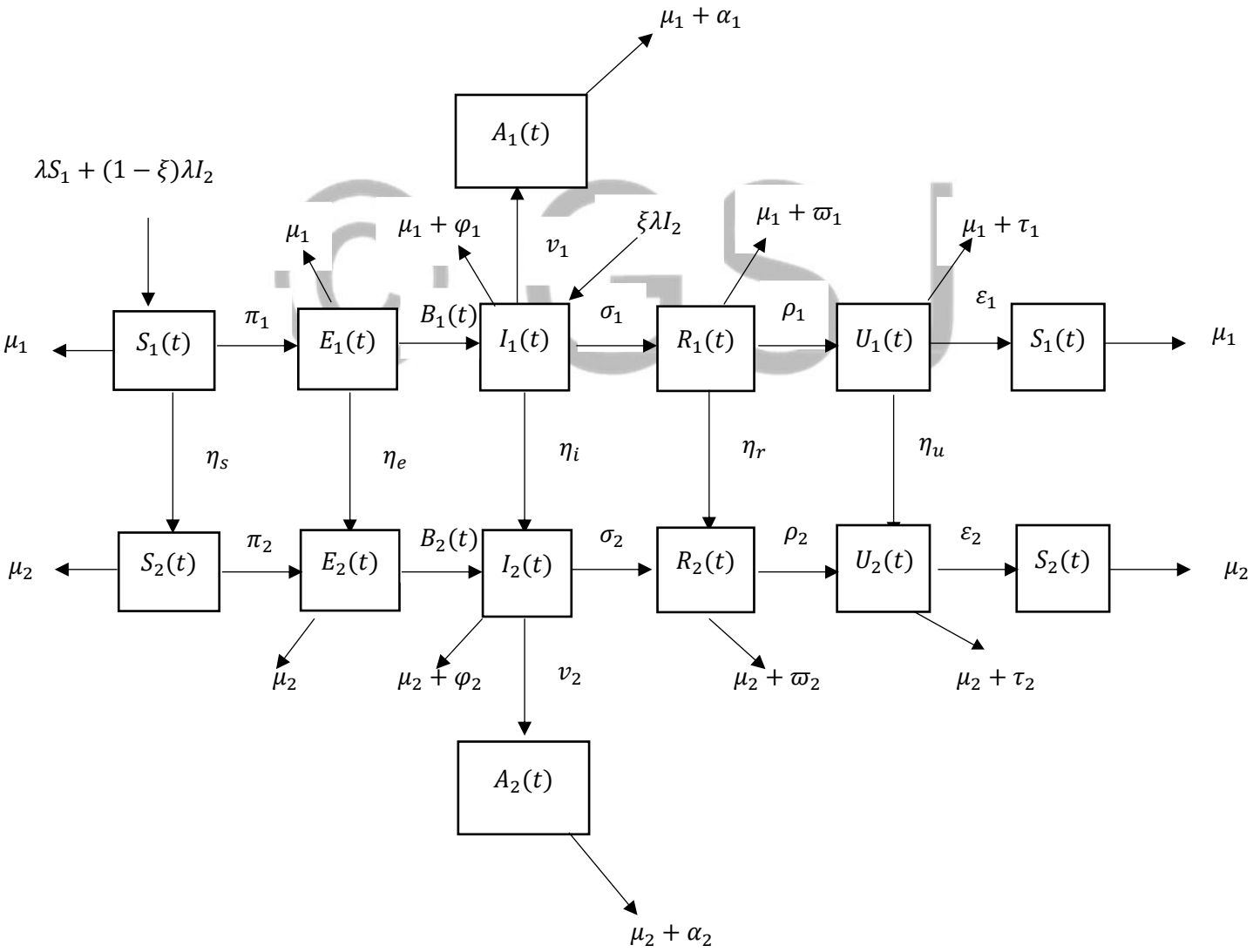
9. The recruitment into the R-class from the I-class depends on the effectiveness of public campaign and counselling. This is done at a rate σ_1 and σ_2 for the juvenile and adult sub-populations, respectively.
10. The recruitment into the U-class from the R-class depends on the effectiveness of the HAART and the change in social behavior of the recovered population. This is done at a rate ρ_1 and ρ_2 for the recovered juvenile and adult sub-population respectively.
11. The recruitment into the S-class over again from the U-class depends on how long the population in the U-class remain in the class while actively receiving treatment. This stage it is assumed that the compartment is filled with fully removed population whose viral load is less than 1% and have 0% chance of secondary infection. This is done at a rate ε_1 and ε_2 for juvenile and adult sub-population respectively.
12. There is a chance of infection by the juvenile and adult population in the U=U class at ρ_1 and ρ_2 probability if the administration of HAART is discontinued at any given time.
13. Death is implicit in the model and it occurs in all classes at constant rate μ_i, where $i = 1,2$ represents the juvenile and adult sub-population respectively. However, there is an additional death rate in the I and R classes due to infection for both juvenile and adult sub-population denoted by φ_i and ϖ_i respectively, where $i = 1, 2$ represents the juvenile and adult sub-population respectively.

2.3 MODEL DESCRIPTION

The susceptible-exposed-infected-removed-undetectable=untransmissible-susceptible (SEIRUS) model that considers an open age-structured population of juvenile and heterosexual individuals is formulated based on the McKendrick-von-Foerster type two-age-structured SIR model as formulated by Oduwole and Kimbir (2018) in studying the effect of antiretroviral therapy. The population is sub-divided based on demographic structure and epidemiological structure. Under a demographic structure, the population is divided into classes, the juvenile class (0 – 14 years) and the adult class (15 years and above), while under the epidemiological structure of this study is divided into six classes, namely; susceptible (S), exposed (E), infected (I), removed (R), Undetectable=Untransmitable (U=U) and those infected progressing to AIDS (A). A susceptible is an individual that is yet to be infected, but is open to infection as he or she interacts with members

of the I-class. An infected individual is one who has contracted HIV and is at some stage of infection. A removed individual is one that is confirmed to be HIV positive, counseled, and is receiving treatment via highly active antiretroviral therapy (HAART). A member of the Undetectable=Untransmitable class is one that has been removed and has been actively receiving treatment through HAART and has been satisfied by the UN-MDG 6's standard to be in the U=U class (CDC, 2017). A member of the A-class is an individual who is HIV positive, and has progressed to full blown AIDS.

The following diagram describes the dynamic of SEIRUS framework, and will be useful in the formulation of model equations.



3 THE MODEL EQUATIONS

From the assumptions and the flow diagram above, the following model equations are derived.

For the Juvenile sub-populations:

$$\frac{dS_1(t)}{dt} = \lambda[S_2 + (1-\xi)I_2] - (\mu_1 + \eta_s + \pi_1)S_1 \tag{3.1}$$

$$\frac{dE_1(t)}{dt} = \pi_1 S_1 - (\mu_1 + \eta_e + B_1)E_1 \tag{3.2}$$

$$\frac{dI_1(t)}{dt} = B_1 E_1 + \xi\lambda I_2 - (\nu_1 + \eta_i + \sigma_1 + \mu_1 + \varphi_1)I_1 \tag{3.3}$$

$$\frac{dR_1(t)}{dt} = \sigma_1 I_1 - (\eta_r + \rho_1 + \mu_1 + \varpi_1)R_1 \tag{3.4}$$

$$\frac{dU_1(t)}{dt} = \rho_1 R_1 - (\mu_1 + \tau_1 + \varepsilon_1 + \eta_u)U_1 \tag{3.5}$$

For the Adult sub-populations:

$$\frac{dS_2(t)}{dt} = \eta_s S_1 - [\pi_2 + \mu_2]S_2 \tag{3.6}$$

$$\frac{dE_2(t)}{dt} = \pi_2 S_2 + \eta_e E_1 - (\mu_2 + B_2)E_2 \tag{3.7}$$

$$\frac{dI_2(t)}{dt} = B_2 E_2 + \eta_i I_1 - (\nu_2 + \sigma_2 + \mu_2 + \varphi_2)I_2 \tag{3.8}$$

$$\frac{dR_2(t)}{dt} = \sigma_2 I_2 + \eta_r R_1 - (\rho_2 + \mu_2 + \varpi_2)R_2 \tag{3.9}$$

$$\frac{dU_2(t)}{dt} = \rho_2 R_2 + \eta_u U_1 - (\mu_2 + \tau_2 + \varepsilon_2)U_2 \tag{3.10}$$

For those progressing to AIDS:

$$\frac{dA_1}{dt} = \nu_1 I_1 - (\mu_1 + \alpha_1)A_1 \tag{3.11}$$

$$\frac{dA_2}{dt} = \nu_2 I_2 - (\mu_2 + \alpha_2)A_2 \tag{3.12}$$

$$\left.\begin{aligned} N_1(t) &= S_1(t) + E_1(t) + I_1(t) + R_1(t) + U_1(t) + A_1(t) \\ N_2(t) &= S_2(t) + E_2(t) + I_2(t) + R_2(t) + U_2(t) + A_2(t) \end{aligned}\right\} \tag{3.13}$$

$$N(t) = N_1(t) + N_2(t) \tag{3.14}$$

The incidence rate or force of infection at time t denoted by $B_2(t)$ in the adult population is given as

$$B_2(t) = \frac{c\beta I_2 + c'\beta' R_2 + \sigma_2 \rho_2 U_2}{N_2} \tag{3.15}$$

3.1 MODEL EQUATIONS IN PROPORTIONS

To simplify the model, it is reasonable to assume that infected juvenile and adult who progress to full blown AIDS are isolated and sexually inactive; hence they are not capable of producing children (vertical transmission) and they do not contribute to viral transmission horizontally (from adult to adult) (Contag, *el. al.*, 1997).

To achieve this, we normalize the model by transforming the model equations into proportions and eliminate the AIDS class $A(t)$, which invariably reduces the number of model equations from twelve to ten. The derive model equations in proportion of infected juveniles and adults define prevalence of infection, which has biological meaning.

The model equations are transformed into proportions as follows;

$$\frac{dN_1(t)}{dt} = \lambda(S_2 + I_2) - \mu_1 N_1 - \eta_s S_1 - \eta_e E_1 - (\eta_i + \sigma_1) I_1 - (\eta_r + \varpi_1) R_1 - (\tau_1 + \varepsilon_1 + \eta_u) U_1 - \alpha_1 A_1 \tag{3.16}$$

$$\frac{dN_2(t)}{dt} = \eta_s S_1 - \mu_2 N_2 + \eta_e E_1 + (\eta_i - \varphi_2) I_2 + \eta_r R_1 - \varpi_2 R_2 - (\tau_2 - \varepsilon_2) U_2 - \alpha_2 A_2 \tag{3.17}$$

Let

$$s_1 = \frac{S_1}{N_1}, e_1 = \frac{E_1}{N_1}, i_1 = \frac{I_1}{N_1}, r_1 = \frac{R_1}{N_1}, u_1 = \frac{U_1}{N_1}, a_1 = \frac{A_1}{N_1} \tag{3.18}$$

similarly,

$$s_2 = \frac{S_2}{N_2}, e_2 = \frac{E_2}{N_2}, i_2 = \frac{I_2}{N_2}, r_2 = \frac{R_2}{N_2}, u_2 = \frac{U_2}{N_2}, a_2 = \frac{A_2}{N_2} \tag{3.19}$$

and

$$m = \frac{N_2}{N_1} = \frac{S_2(t) + E_2(t) + I_2(t) + R_2(t) + U_2(t) + A_2(t)}{S_1(t) + E_1(t) + I_1(t) + R_1(t) + U_1(t) + A_1(t)} \tag{3.20}$$

Then the normalized system is follows,

$$\frac{ds_1}{dt} = m\lambda s_2 + m\lambda(1-\xi)i_2 - (\eta_s + \pi_1)s_1 - m\lambda(s_1 s_2 + s_1 i_2) + \eta_s s_1^2 + \eta_e s_1 e_1 + (\eta_i + \sigma_1)s_1 i_1 + (\eta_r + \varpi_1)s_1 r_1 + (\tau_1 + \varepsilon_1 + \eta_u)s_1 u_1 + s_1 \alpha_1 a_1 \tag{3.21}$$

$$\frac{de_1}{dt} = \pi_1 s_1 - (\eta_e + B_1)e_1 - m\lambda(e_1 s_2 + e_1 i_2) + \eta_s e_1 s_1 + \eta_e e_1^2 + (\eta_i + \sigma_1)e_1 i_1 + (\eta_r + \varpi_1)e_1 r_1 + (\tau_1 + \varepsilon_1 + \eta_u)e_1 u_1 + e_1 \alpha_1 a_1 \tag{3.22}$$

$$\frac{di_1}{dt} = B_1 e_1 + \xi\lambda i_2 - (\nu_1 + \eta_i + \sigma_1 + \varphi_1)i_1 - m\,\lambda(i_1 s_2 + i_1^2) + \eta_s i_1 s_1 + \eta_e i_1 e_1 + (\eta_i + \sigma_1)i_1^2 + (\eta_r + \varpi_1)i_1 r_1 + (\tau_1 + \varepsilon_1 + \eta_u)i_1 u_1 + \alpha_1 i_1 a_1 \tag{3.23}$$

$$\frac{dr_1}{dt} = \sigma_1 i_1 - (\eta_r + \rho_1 + \varpi_1)r_1 - m\lambda(r_1 s_2 + r_1 i_2) + r_1 \eta_s s_1 + r_1 \eta_e e_1 + (\eta_i + \sigma_1)r_1 i_1 + (\eta_r + \varpi_1)r_1^2 + (\tau_1 + \varepsilon_1 + \eta_u)r_1 u_1 + r_1 \alpha_1 a_1 \tag{3.24}$$

$$\frac{du_1}{dt} = \rho_1 r_1 - (\tau_1 + \varepsilon_1 + \eta_u)u_1 - m\lambda(u_1 s_2 + u_1 I_2) + u_1 \eta_s s_1 + u_1 \eta_e e_1 + (\eta_i + \sigma_1)u_1 i_1 + (\eta_r + \varpi_1)u_1 r_1 + (\tau_1 + \varepsilon_1 + \eta_u)u_1^2 + u_1 \alpha_1 a_1 \tag{3.25}$$

$$\frac{da_1}{dt} = \nu_1 i_1 - \alpha_1 a_1 - m\lambda(a_1 s_2 + a_1 I_2) + \eta_s a_1 s_1 + \eta_e a_1 e_1 + (\eta_i + \sigma_1)a_1 i_1 + (\eta_r + \varpi_1)a_1 r_1 + (\tau_1 + \varepsilon_1 + \eta_u)a_1 u_1 + \alpha_1 a_1^2 \tag{3.26}$$

Similarly for the adult sub-population, the normalized system is follows:

$$\frac{ds_2}{dt} = \frac{\eta_s s_1}{m} - \pi_2 s_2 - \frac{s_2 \eta_s s_1}{m} - \frac{s_2 \eta_e e_1}{m} - (\eta_i - \varphi_2)s_2 i_2 - \frac{s_2 \eta_r r_1}{m} + s_2 \varpi_2 r_2 + (\tau_2 - \varepsilon_2)s_2 u_2 + s_2 \alpha_2 a_2 \tag{3.27}$$

$$\frac{de_2}{dt} = \pi_2 s_2 + \frac{\eta_e e_1}{m} - B_2 e_2 - \frac{e_2 \eta_s s_1}{m} - \frac{e_2 \eta_e e_1}{m} - (\eta_i - \varphi_2)e_2 i_2 - \frac{e_2 \eta_r r_1}{m} + e_2 \varpi_2 r_2 + (\tau_2 - \varepsilon_2)e_2 u_2 + e_2 \alpha_2 a_2 \tag{3.28}$$

$$\frac{di_2}{dt} = B_2e_2 + \frac{\eta_i i_1}{m} - (\nu_2 + \sigma_2 + \varphi_2)i_2 - \frac{i_2\eta_s s_1}{m} - \frac{i_2\eta_e e_1}{m} - (\eta_i - \varphi_2)i_1^2 - \frac{i_2\eta_r r_1}{m} + i_2\varpi_2 r_2 + (\tau_2 - \varepsilon_2)i_2u_2 + i_2\alpha_2 a_2 \quad (3.29)$$

$$\frac{dr_2}{dt} = \sigma_2 i_2 + \frac{\eta_r r_1}{m} - (\rho_2 + \varpi_2)r_2 - \frac{r_2\eta_s s_1}{m} - \frac{r_2\eta_e e_1}{m} - (\eta_i - \varphi_2)r_2i_2 - \frac{r_2\eta_r r_1}{m} + \varpi_2 r_2^2 + (\tau_2 - \varepsilon_2)r_2u_2 + r_2\alpha_2 a_2 \quad (3.30)$$

$$\frac{du_2}{dt} = \rho_2 r_2 + \frac{\eta_u u_1}{m} - (\tau_2 + \varepsilon_2)u_2 - \frac{u_2\eta_s s_1}{m} - \frac{u_2\eta_e e_1}{m} - (\eta_i - \varphi_2)u_2i_2 - \frac{u_2\eta_r r_1}{m} + u_2\varpi_2 r_2 + (\tau_2 + \varepsilon_2)u_2^2 + u_2\alpha_2 a_2 \quad (3.31)$$

$$\frac{da_2}{dt} = \nu_2 i_2 - \alpha_2 a_2 - \frac{a_2\eta_s s_1}{m} - \frac{a_2\eta_e e_1}{m} - (\eta_i - \varphi_2)a_2i_2 - \frac{a_2\eta_r r_1}{m} + a_2\varpi_2 r_2 + (\tau_2 - \varepsilon_2)a_2u_2 + \alpha_2 a_2^2 \quad (3.32)$$

However,

$$\left.\begin{aligned} s_1 + e_1 + i_1 + r_1 + u_1 + a_1 = 1 \\ s_2 + e_2 + i_2 + r_2 + u_2 + a_2 = 1 \end{aligned}\right\} \quad (3.33)$$

Gives the following governing equations of the model below:

For the Juvenile sub-population:

$$\frac{ds_1}{dt} = m\lambda s_2 + m\lambda(1-\xi)i_2 - (\eta_s + \pi_1)s_1 - m\lambda(s_1s_2 + s_1i_2) + \eta_s s_1^2 + \eta_e s_1e_1 + (\eta_i + \sigma_1)s_1i_1 + (\eta_r + \varpi_1)s_1r_1 + (\tau_1 + \varepsilon_1 + \eta_u)s_1u_1 + s_1\alpha_1\big(1 - (s_1 + e_1 + i_1 + r_1 + u_1)\big) \quad (3.34)$$

$$\frac{de_1}{dt} = \pi_1 s_1 - \big(\eta_q + B_1\big)e_1 - m\lambda(e_1s_2 + e_1i_2) + \eta_s e_1s_1 + \eta_e e_1^2 + (\eta_i + \sigma_1)e_1i_1 + (\eta_r + \varpi_1)e_1r_1 + (\tau_1 + \varepsilon_1 + \eta_u)e_1u_1 + e_1\alpha_1\big(1 - (s_1 + e_1 + i_1 + r_1 + u_1)\big) \quad (3.35)$$

$$\frac{di_1}{dt} = B_1e_1 + \xi\lambda i_2 - (\nu_1 + \eta_i + \sigma_1 + \varphi_1)i_1 - m\,\lambda(i_1s_2 + i_1^2) + \eta_s i_1s_1 + \eta_e i_1e_1 + (\eta_i + \sigma_1)i_1^2 + (\eta_r + \varpi_1)i_1r_1 + (\tau_1 + \varepsilon_1 + \eta_u)i_1u_1 + \alpha_1 i_1\big(1 - (s_1 + e_1 + i_1 + r_1 + u_1)\big) \quad (3.36)$$

$$\frac{dr_1}{dt} = \sigma_1 i_1 - (\eta_r + \rho_1 + \varpi_1)r_1 - m\lambda(r_1 s_2 + r_1 i_2) + r_1\eta_s s_1 + r_1\eta_e e_1 + (\eta_i + \sigma_1)r_1 i_1 + (\eta_r + \varpi_1)r_1^2 + (\tau_1 + \varepsilon_1 + \eta_u)r_1 u_1 + r_1\alpha_1\big(1 - (s_1 + e_1 + i_1 + r_1 + u_1)\big) \quad (3.37)$$

$$\frac{du_1}{dt} = \rho_1 r_1 - (\tau_1 + \varepsilon_1 + \eta_u)u_1 - m\lambda(u_1 s_2 + u_1 i_2) + u_1\eta_s s_1 + u_1\eta_e e_1 + (\eta_i + \sigma_1)u_1 i_1 + (\eta_r + \varpi_1)u_1 r_1 + (\tau_1 + \varepsilon_1 + \eta_u)u_1^2 + u_1\alpha_1\big(1 - (s_1 + e_1 + i_1 + r_1 + u_1)\big) \quad (3.38)$$

For the Adult sub-population:

$$\frac{ds_2}{dt} = \frac{\eta_s s_1}{m} - \pi_2 s_2 - \frac{s_2\eta_s s_1}{m} - \frac{s_2\eta_e e_1}{m} - (\eta_i - \varphi_2)s_2 i_2 - \frac{s_2\eta_r r_1}{m} + s_2\varpi_2 r_2 + (\tau_2 - \varepsilon_2)s_2 u_2 + s_2\alpha_2\big(1 - (s_2 + e_2 + i_2 + r_2 + u_2)\big) \quad (3.39)$$

$$\frac{de_2}{dt} = \pi_2 s_2 + \frac{\eta_e e_1}{m} - B_2 e_2 - \frac{e_2\eta_s s_1}{m} - \frac{e_2\eta_e e_1}{m} - (\eta_i - \varphi_2)e_2 i_2 - \frac{e_2\eta_r r_1}{m} + e_2\varpi_2 r_2 + (\tau_2 - \varepsilon_2)e_2 u_2 + e_2\alpha_2\big(1 - (s_2 + e_2 + i_2 + r_2 + u_2)\big) \quad (3.40)$$

$$\frac{di_2}{dt} = B_2 e_2 + \frac{\eta_i i_1}{m} - (\nu_2 + \sigma_2 + \varphi_2)i_2 - \frac{i_2\eta_s s_1}{m} - \frac{i_2\eta_e e_1}{m} - (\eta_i - \varphi_2)i_1^2 - \frac{i_2\eta_r r_1}{m} + i_2\varpi_2 r_2 + (\tau_2 - \varepsilon_2)i_2 u_2 + i_2\alpha_2\big(1 - (s_2 + e_2 + i_2 + r_2 + u_2)\big) \quad (3.41)$$

$$\frac{dr_2}{dt} = \sigma_2 i_2 + \frac{\eta_r r_1}{m} - (\rho_2 + \varpi_2)r_2 - \frac{r_2\eta_s s_1}{m} - \frac{r_2\eta_e e_1}{m} - (\eta_i - \varphi_2)r_2 i_2 - \frac{r_2\eta_r r_1}{m} + \varpi_2 r_2^2 + (\tau_2 - \varepsilon_2)r_2 u_2 + r_2\alpha_2\big(1 - (s_2 + e_2 + i_2 + r_2 + u_2)\big) \quad (3.42)$$

$$\frac{du_2}{dt} = \rho_2 r_2 + \frac{\eta_u u_1}{m} - (\tau_2 + \varepsilon_2)u_2 - \frac{u_2\eta_s s_1}{m} - \frac{u_2\eta_e e_1}{m} - (\eta_i - \varphi_2)u_2 i_2 - \frac{u_2\eta_r r_1}{m} + u_2\varpi_2 r_2 + (\tau_2 + \varepsilon_2)u_2^2 + u_2\alpha_2\big(1 - (s_2 + e_2 + i_2 + r_2 + u_2)\big) \quad (3.43)$$

Equations (3.34) to (3.43) are the model equations in proportions, which define prevalence of infection.

3.2 EXISTENCE AND UNIQUENESS OF DISEASE FREE EQUILIBRIUM STATE (E_0) OF THE SEIRUS MODEL

The disease-free equilibrium (DFE) state of the endemic SEIRUS model is obtained by setting the left hand sides of equations (3.34) – (3.43) to zero while setting the disease components $e_1 =$

$0, e_2 = 0, i_1 = 0,\ i_2 = 0, r_1 = 0,\ r_2 = 0$ and $u_1 = 0,\ u_2 = 0$ leading to equations (3.44) – (3.45) below

For the Juvenile sub-population:

$$0 = m\lambda s_2^* - \eta_s s_1^* - \pi_1 s_1^* - m\lambda s_2^* s_1^* + \eta_s s_1^{*2} + s_1^* \alpha_1 - s_1^{*2}\alpha_1 \tag{3.44}$$

For the Adult sub-population:

$$0 = \frac{\eta_s s_1^*}{m} - \pi_2 s_2^* - \frac{s_2^* \eta_s s_1^*}{m} + s_2^* \alpha_2 - s_2^{*2}\alpha_2 \tag{3.45}$$

Factorizing s_1^* From Equation (3.45) and substituting into (3.44) gives;

$$\begin{aligned} m\lambda s_2^* - \eta_s m\left(\frac{s_2^*\alpha_2 - s_2^{*2}\alpha_2 - \pi_2 s_2^*}{s_2^*\eta_s - \eta_s}\right) - \pi_1 m\left(\frac{s_2^*\alpha_2 - s_2^{*2}\alpha_2 - \pi_2 s_2^*}{s_2^*\eta_s - \eta_s}\right) \\ - \lambda s_2^* m^2\left(\frac{s_2^*\alpha_2 - s_2^{*2}\alpha_2 - \pi_2 s_2^*}{s_2^*\eta_s - \eta_s}\right) + \eta_s m^2\left(\frac{s_2^*\alpha_2 - s_2^{*2}\alpha_2 - \pi_2 s_2^*}{s_2^*\eta_s - \eta_s}\right)^2 \\ + \alpha_1 m\left(\frac{s_2^*\alpha_2 - s_2^{*2}\alpha_2 - \pi_2 s_2^*}{s_2^*\eta_s - \eta_s}\right) - \alpha_1 m^2\left(\frac{s_2^*\alpha_2 - s_2^{*2}\alpha_2 - \pi_2 s_2^*}{s_2^*\eta_s - \eta_s}\right)^2 = 0 \end{aligned} \tag{3.46}$$

Multiplying through by $(s_2^*\eta_s - \eta_s)^2$, gives

$$\begin{aligned} m\lambda s_2^*(s_2^*\eta_s - \eta_s)^2 - \eta_s m(s_2^*\alpha_2 - s_2^{*2}\alpha_2 - \pi_2 s_2^*)(s_2^*\eta_s - \eta_s) \\ - \pi_1 m(s_2^*\alpha_2 - s_2^{*2}\alpha_2 - \pi_2 s_2^*)(s_2^*\eta_s - \eta_s) \\ - \lambda s_2^* m^2(s_2^*\alpha_2 - s_2^{*2}\alpha_2 - \pi_2 s_2^*)(s_2^*\eta_s - \eta_s) + \eta_s m^2(s_2^*\alpha_2 - s_2^{*2}\alpha_2 - \pi_2 s_2^*)^2 \\ + \alpha_1 m(s_2^*\alpha_2 - s_2^{*2}\alpha_2 - \pi_2 s_2^*)(s_2^*\eta_s - \eta_s) - \alpha_1 m^2(s_2^*\alpha_2 - s_2^{*2}\alpha_2 - \pi_2 s_2^*)^2 \\ = 0 \end{aligned} \tag{3.47}$$

Simplifying further and collecting like terms in s_2^{*4}, s_2^{*3}, s_2^{*2} and s_2^* gives,

$$As_2^{*4} + Bs_2^{*3} + Cs_2^{*2} + Ds_2^* = 0 \tag{3.48}$$

where

$$\left.\begin{aligned} A &= m\lambda\eta_s^2 + m^2\lambda\alpha_2\eta_s + m^2\alpha_2\eta_s - m\alpha_1\alpha_2 \\ B &= m\alpha_2\eta_s^2 - 2\lambda\eta_s^2 + m\pi_1\alpha_2\eta_s - 2m^2\lambda\alpha_2\eta_s + m^2\lambda\pi_2\eta_s - 2m^2\alpha_2^2\eta_s + 2m\alpha_2\pi_2\eta_s - m\alpha_1\alpha_2\eta_s + \\ &\quad 2m\alpha_1\alpha_2^2 - 2m\alpha_1\alpha_2\pi_2 \\ C &= m\lambda\eta_s^2 - 2m\alpha_2\eta_s^2 + 2m\pi_2\eta_s^2 - 2m\pi_1\alpha_2\eta_s + m\pi_1\pi_2\eta_s + m^2\lambda\alpha_2\eta_s - m^2\lambda\pi_2\eta_s + m^2\alpha_2^2\eta_s - \\ &\quad 2m^2\alpha_2\pi_2\eta_s + m^2\pi_2\eta_s + 2m\alpha_1\alpha_2\eta_s - m\alpha_1\pi_2\eta_s - m\alpha_1\alpha_2^2 - 2m\alpha_1\alpha_2\pi_2 - m\alpha_1\pi_2 \\ D &= m\alpha_2\eta_s^2 + m\pi_2\eta_s^2 + m\alpha_2\pi_1\eta_s + m\pi_1\pi_2\eta_s - m\alpha_1\alpha_2\eta_s + m\alpha_1\pi_2\eta_s \end{aligned}\right\} \quad (3.49)$$

Therefore, the solution for the simultaneous equations (3.48) is given by

$$(s_1^*, s_2^*) = \left\{(0,0), (1,1), \left(\frac{m\lambda}{(\eta_s - \alpha_1)}, \frac{(m\alpha_2 - \eta_s)}{m\alpha_2}\right), \left(\frac{(\pi_1 + \eta_s - \alpha_1)}{(\eta_s - \alpha_1)}, -\frac{\pi_2}{\alpha_2}\right)\right\} \quad (3.50)$$

Ignoring the native values of s_1^* and s_2^* and other stringent conditions, there exist a unique trivial and disease-free equilibrium states at (s_1^*, s_2^*) given by $(0,0)$ and $(1,1)$ respectively.

The solution (3.50) satisfies equations (3.47) identically.

3.3 COMPUTATION OF THE BASIC REPRODUCTIVE NUMBER (R_0) OF THE MODEL

The Basic Reproductive number (R_0) is define as the number of secondary infections that one infectious individual would create over the duration of the infectious period, provided that everyone else is susceptible. $R_0 = 1$ is a threshold below which the generation of secondary cases is insufficient to maintain the infection in human community. If $R_0 < 1$, the number of infected individuals will decrease from generation to next and the disease dies out and if $R_0 > 1$ the number of infected individuals will increase from generation to the next and the disease will persist.

To compute the basic reproductive number (R_0) of the model (3.33) – (3.43), we employ the next generation method as applied by Oduwole, H. K. and Kimbir, A. R. (2018), Diekmann, *el. al.*, (2009) and Van den Driessche, P. and Watmough, J. (2002).

$$\mathcal{F}_i = \begin{pmatrix} B_1 e_1 + \xi\lambda i_2 \\ B_2 e_2 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}$$

and

$\mathcal{V}_i$

$$= \begin{pmatrix} -(\nu_1 + \eta_i + \sigma_1 + \varphi_1)i_1 - m\,\lambda(i_1 s_2 + i_1^2) + \eta_s i_1 s_1 + \eta_e i_1 e_1 + (\eta_i + \sigma_1)i_1^2 + (\eta_r + \varpi_1)i_1 r_1 + (\tau_1 + \varepsilon_1 + \eta_u)i_1 u_1 + \alpha_1 i_1\big(1 - (s_1 + e_1 + i_1 + r_1 + u_1)\big) \\ \frac{\eta_i i_1}{m} - (\nu_2 + \sigma_2 + \varphi_2)i_2 - \frac{i_2\eta_s s_1}{m} - \frac{i_2\eta_e e_1}{m} - (\eta_i - \varphi_2)i_1^2 - \frac{i_2\eta_r r_1}{m} + i_2\varpi_2 r_2 + (\tau_2 - \varepsilon_2)i_2 u_2 + i_2\alpha_2\big(1 - (s_2 + e_2 + i_2 + r_2 + u_2)\big) \\ \pi_1 s_1 - (\eta_e + B_1)e_1 - m\lambda(e_1 s_2 + e_1 i_2) + \eta_s e_1 s_1 + \eta_e e_1^2 + (\eta_i + \sigma_1)e_1 i_1 + (\eta_r + \varpi_1)e_1 r_1 + (\tau_1 + \varepsilon_1 + \eta_u)e_1 u_1 + e_1\alpha_1\big(1 - (s_1 + e_1 + i_1 + r_1 + u_1)\big) \\ \pi_2 s_2 + \frac{\eta_e e_1}{m} - B_2 e_2 - \frac{e_2\eta_s s_1}{m} - \frac{e_2\eta_e e_1}{m} - (\eta_i - \varphi_2)e_2 i_2 - \frac{e_2\eta_r r_1}{m} + e_2\varpi_2 r_2 + (\tau_2 - \varepsilon_2)e_2 u_2 + e_2\alpha_2\big(1 - (s_2 + e_2 + i_2 + r_2 + u_2)\big) \\ \sigma_1 i_1 - (\eta_r + \rho_1 + \varpi_1)r_1 - m\lambda(r_1 s_2 + r_1 i_2) + r_1\eta_s s_1 + r_1\eta_e e_1 + (\eta_i + \sigma_1)r_1 i_1 + (\eta_r + \varpi_1)r_1^2 + (\tau_1 + \varepsilon_1 + \eta_u)r_1 u_1 + r_1\alpha_1\big(1 - (s_1 + e_1 + i_1 + r_1 + u_1)\big) \\ \sigma_2 i_2 + \frac{\eta_r r_1}{m} - (\rho_2 + \varpi_2)r_2 - \frac{r_2\eta_s s_1}{m} - \frac{r_2\eta_e e_1}{m} - (\eta_i - \varphi_2)r_2 i_2 - \frac{r_2\eta_r r_1}{m} + \varpi_2 r_2^2 + (\tau_2 - \varepsilon_2)r_2 u_2 + r_2\alpha_2\big(1 - (s_2 + e_2 + i_2 + r_2 + u_2)\big) \\ \rho_1 r_1 - (\tau_1 + \varepsilon_1 + \eta_u)u_1 - m\lambda(u_1 s_2 + u_1 i_2) + u_1\eta_s s_1 + u_1\eta_e e_1 + (\eta_i + \sigma_1)u_1 i_1 + (\eta_r + \varpi_1)u_1 r_1 + (\tau_1 + \varepsilon_1 + \eta_u)u_1^2 + u_1\alpha_1\big(1 - (s_1 + e_1 + i_1 + r_1 + u_1)\big) \\ \rho_2 r_2 + \frac{\eta_u u_1}{m} - (\tau_2 + \varepsilon_2)u_2 - \frac{u_2\eta_s s_1}{m} - \frac{u_2\eta_e e_1}{m} - (\eta_i - \varphi_2)u_2 i_2 - \frac{u_2\eta_r r_1}{m} + u_2\varpi_2 r_2 + (\tau_2 + \varepsilon_2)u_2^2 + u_2\alpha_2\big(1 - (s_2 + e_2 + i_2 + r_2 + u_2)\big) \\ m\lambda s_2 + m\lambda(1 - \xi)i_2 - (\eta_s + \pi_1)s_1 - m\lambda(s_1 s_2 + s_1 i_2) + \eta_s s_1^2 + \eta_e s_1 e_1 + (\eta_i + \sigma_1)s_1 i_1 + (\eta_r + \varpi_1)s_1 r_1 + (\tau_1 + \varepsilon_1 + \eta_u)s_1 u_1 + s_1\alpha_1\big(1 - (s_1 + e_1 + i_1 + r_1 + u_1)\big) \\ \frac{\eta_s s_1}{m} - \pi_2 s_2 - \frac{s_2\eta_s s_1}{m} - \frac{s_2\eta_e e_1}{m} - (\eta_i - \varphi_2)s_2 i_2 - \frac{s_2\eta_r r_1}{m} + s_2\varpi_2 r_2 + (\tau_2 - \varepsilon_2)s_2 u_2 + s_2\alpha_2\big(1 - (s_2 + e_2 + i_2 + r_2 + u_2)\big) \end{pmatrix}$$

where $\mathcal{F}_i$ and $\mathcal{V}_i$ are the rate of appearances of new infections in compartment i and the transfer of individuals into and out of compartment i by all means respectively. Using the linearization method, the associated matrices at disease-fee equilibrium (E_0) and after taking partial derivatives as defined by

$$D\mathcal{F}_i(E_0) = \begin{pmatrix} F & 0 \\ 0 & 0 \end{pmatrix} \text{ and } D\mathcal{V}_i(E_0) = \begin{pmatrix} V & 0 \\ J_3 & J_4 \end{pmatrix}$$

where F is nonnegative and V is a non-singular matrix, in which both are the $m \times m$ matrices defined by

$$F = \left[\frac{\partial \mathcal{F}_i}{\partial x_i}(E_0)\right]$$

and

$$V = \left[\frac{\partial \mathcal{V}_i}{\partial x_i}(E_0)\right]$$

with $1 \leq i, j \leq m$ and m is the number of infected classes.

In particular $m = 2$, we have

$$F = \begin{pmatrix} B_1 & \xi\lambda \\ 0 & B_1 \end{pmatrix}$$

and

$$V = \begin{pmatrix} \eta_s + \eta_e - \nu_1 - \eta_i - \sigma_1 - \varphi_1 - m\,\lambda & 0 \\ \dfrac{\eta_i}{m} & -\nu_2 - \sigma_2 - \varphi_2 - \dfrac{\eta_s}{m} - \dfrac{\eta_e}{m} \end{pmatrix}$$

If the inverse of V is given as

$$V^{-1} = \begin{pmatrix} \dfrac{1}{\eta_s + \eta_e - \nu_1 - \eta_i - \sigma_1 - \varphi_1 - m\,\lambda} & -\dfrac{\eta_i}{(\eta_s + \eta_e - \nu_1 - \eta_i - \sigma_1 - \varphi_1 - m\,\lambda)(-\nu_2 m - \sigma_2 m - \varphi_2 m - \eta_s - \eta_e)} \\ 0 & \dfrac{m}{-\nu_2 m - \sigma_2 m - \varphi_2 m - \eta_s - \eta_e} \end{pmatrix}$$

Then the next matrix denoted by FV^{-1} is given as

$$FV^{-1} = \begin{pmatrix} \dfrac{B_1}{\eta_s + \eta_e - \nu_1 - \eta_i - \sigma_1 - \varphi_1 - m\,\lambda} & -\dfrac{B_1\eta_i + m\xi\lambda(\eta_s + \eta_e - \nu_1 - \eta_i - \sigma_1 - \varphi_1 - m\,\lambda)}{(\eta_s + \eta_e - \nu_1 - \eta_i - \sigma_1 - \varphi_1 - m\,\lambda)(-\nu_2 m - \sigma_2 m - \varphi_2 m - \eta_s - \eta_e)} \\ 0 & \dfrac{mB_1}{-\nu_2 m - \sigma_2 m - \varphi_2 m - \eta_s - \eta_e} \end{pmatrix}$$

We find the eigenvalues of FV^{-1} by setting the determinant $|FV^{-1} - \gamma I| = 0$

$$|FV^{-1} - \gamma I| = \begin{vmatrix} \dfrac{B_1}{\eta_s + \eta_e - \nu_1 - \eta_i - \sigma_1 - \varphi_1 - m\,\lambda} - \gamma & -\dfrac{B_1\eta_i + m\xi\lambda(\eta_s + \eta_e - \nu_1 - \eta_i - \sigma_1 - \varphi_1 - m\,\lambda)}{(\eta_s + \eta_e - \nu_1 - \eta_i - \sigma_1 - \varphi_1 - m\,\lambda)(-\nu_2 m - \sigma_2 m - \varphi_2 m - \eta_s - \eta_e)} \\ 0 & \dfrac{mB_1}{-\nu_2 m - \sigma_2 m - \varphi_2 m - \eta_s - \eta_e} - \gamma \end{vmatrix}$$

$$|FV^{-1} - \gamma I| = 0$$

with characteristics polynomial

$$\rho(\gamma) = \gamma^2 - \left(\frac{B_1}{\eta_s + \eta_e - \nu_1 - \eta_i - \sigma_1 - \varphi_1 - m\,\lambda} + \frac{mB_1}{-\nu_2 m - \sigma_2 m - \varphi_2 m - \eta_s - \eta_e}\right)\gamma + \frac{mB_1^2}{(\eta_s + \eta_e - \nu_1 - \eta_i - \sigma_1 - \varphi_1 - m\,\lambda)(-\nu_2 m - \sigma_2 m - \varphi_2 m - \eta_s - \eta_e)}$$

and characteristics equation given as

$$\gamma^2 - \left(\frac{B_1(-\nu_2 m - \sigma_2 m - \varphi_2 m - \eta_s - \eta_e) + mB_1(\eta_s + \eta_e - \nu_1 - \eta_i - \sigma_1 - \varphi_1 - m\,\lambda)}{(\eta_s + \eta_e - \nu_1 - \eta_i - \sigma_1 - \varphi_1 - m\,\lambda)(-\nu_2 m - \sigma_2 m - \varphi_2 m - \eta_s - \eta_e)}\right)\gamma + \frac{mB_1^2}{(\eta_s + \eta_e - \nu_1 - \eta_i - \sigma_1 - \varphi_1 - m\,\lambda)(-\nu_2 m - \sigma_2 m - \varphi_2 m - \eta_s - \eta_e)} = 0$$

Solving the characteristics equation for the eigenvalues $\gamma_{1,2}$, where R_0 is the maximum of the two eigenvalues $\gamma_{1,2}$. Hence the Basic Reproductive number is the dominant eigenvalues of FV^{-1}. Thus we have that

$$R_0 = \frac{B_1\left(\nu_2 + \sigma_2 + \varphi_2 + \frac{\eta_s}{m} + \frac{\eta_e}{m}\right) - mB_1(\eta_s + \eta_e - \nu_1 - \eta_i - \sigma_1 - \varphi_1 - m\,\lambda)}{(\eta_s + \eta_e - \nu_1 - \eta_i - \sigma_1 - \varphi_1 - m\,\lambda)\left(\nu_2 + \sigma_2 + \varphi_2 + \frac{\eta_s}{m} + \frac{\eta_e}{m}\right)} \quad (3.51)$$

Because the incidence rate in the juvenile population is zero, that is, there is no transmission of the disease between children to children, and $B_1(t) = 0$ from table 1, then equation (3.51) hence

$$R_0 = 0 \quad (3.52)$$

The Basic Reproductive number (R_0) by Equation (3.52) shows that the number of secondary infections that one infectious individual would create over the duration of the infectious period, provided that everyone else is susceptible is zero and this implies there is no secondary infection in an endemic situation.

4. DESCRIPTION AND VALIDATION OF BASELINE PARAMETERS

The population of Kenya was reported by the Kenyan National Bureau of Statistics as of 2019 was estimated at 47,564,296 with females scooping a total of 50.31% of the population and the males holding at 49.69%.

This population size ranks Kenya as the 7th most populous country in Africa and the 27th in the world with a total of 0.68% of the World's population and a growth rate of 2.30% as of 2018.

Over the past 20 years, Kenya's population has doubled. Although Kenya has sustained population growth, but it has both high birth and infant mortality rates. This is consistent with Africa as a whole. There has been marked improvement in life expectancy, particularly in recent years. In 2006, the average level stood at 48.9 years. This figure rose, however, to around 59 years in 2016. This has increased to 64 years of age in 2018. The current median age is only 19.7 years of age in Kenya.

Although Kenya's extreme growth is expected to slow in the coming years, it will still be significant. The current rate of change of 2.52% annually is predicted to drop to 2.20% by 2030.

During this time, however, the population should grow from 53,491,697 in 2020 to 66,959,993 in 2030.

While pregnancy is obviously not a disease by any means, a lot of expectant mothers in Kenya die from pregnancy complications every year. A Kenyan woman's chances of death during pregnancy or childbirth is 1 in 12. However, the components of population change in Kenya shows that one birth occurs every 21 seconds and one death occurs every 2 minutes due to the various safety measures and advance health systems. Meanwhile, the population is also affected by the rate of migration into the country as there is one net migrant every 53 minutes into Kenya due to the strict border rules and immigrations which means a net gain of one person every 26 seconds either from immigration or birth and this speaks more about the rapid population growth in the country.

With an estimated population of about 47 million people, Kenya has a total of 1,600,000 people living with HIV as at 2018 and there are 1.02 infections among all people of all ages which are the number of new HIV infections among the uninfected population over a year. The percentage of people living with HIV – among adults (15 – 49 years) was recorded to be 4.7% with a total of 46,000 newly infected people and 25,000 cases of death due to AIDS-related illness.

Although there has been progress in the number of AIDS-related deaths in Kenya since 2010, with a 55% decrease, from 56,000 deaths to 25,000 deaths. Hence, the number of new HIV infections has also decreased from 66,000 to 46,000 in the same period (UNAID, 2017). With the 90-90-90 targets vision for 2020, which implies a 90% of people living with HIV knowing their HIV status, 90% of people who know their HIV-positive status being able to access treatment and 90% of people being placed on treatment to have suppressed viral loads, it was observed based on the 90-90-90 target that in 2018 a record high of 89% of people living with HIV knew their status and of the 81% of people living with HIV who are supposed to be on treatment, only 68% of them were on treatment

According to UNAID factsheet (2017), of all adults aged 15 years and over living with HIV, 69% were on treatment as at 2018 while 61% of children/juvenile aged 0 – 14 years living with HIV were on treatment. Also, 19% of pregnant women living with HIV accessed antiretroviral medicine to prevent transmission of the virus to their baby (MTC), which has helped to prevent about 11,000 new HIV infections among new-borns. The report further shows that women are disproportionally affected by HIV in Kenya as of the 1,400,000 adults living with HIV, 910,000 (65%) were women.

Whereby new HIV infections among young women aged 15 – 24 years were more than double those among young men with 11,000 new infections among young women, compared to 5,000 among young men. More so, HIV treatment was found to be higher among women than men, however, with 75% of adult women living with HIV on treatment, compared to 59% of adult men.

The baseline parameters for Kenya include total population (both juvenile and adult), birth rate **(λ)**, natural death rate for juvenile (μ_1) and natural death rate for adult (μ_2) as estimate gotten from UNAID (2017). See Table 1 for details.

4.1 DESCRIPTION AND VALIDATION OF OTHER ESTIMATED PARAMETERS

(a) Proportion of susceptible, exposed, infected, removed and undetectable=untrasmittable individuals in both the Juvenile and Adult sub-population ($s_1, s_2, e_1, e_2, i_1, i_2, r_1, r_2, u_1, and\ u_2$)

The United Nations AIDS (UNAID, 2017) and the World Population Review (2019) estimates there are about was 1,600,000 people living with HIV in Kenya, ranking 5th in countries with highest burden of HIV infection in Africa and the World at large after Mozambique and India respectively. Females constitute almost three-fourth (65%) of people living with HIV in Kenya, making a total of about 910,000 women and girls are infected with HIV and a total of 690,000 males. The estimated overall HIV prevalence rate is approximately 1.02% among the Kenyan Population. For adults aged between 15 – 49 years, an estimated 4.7% of the population is HIV positive.

For the purpose of this study we use $47{,}564{,}296$ as the estimated population for South Africa, with a total of $19{,}201{,}631$ juveniles (0 – 14 years) and $28{,}362{,}665$ adults (15 years and above) according World Population Review (2019) updates.

This put the proportion of susceptible juveniles and adult to be $s_1(0) = 0.40369$ and $s_2(0) = 0.5963$ respectively.

However, in this study we take the proportion of susceptible population to also start for the proportion of exposed with the assumption that all the susceptible class are equally vulnerable to be exposed to being infected in an ideal situation where MTC and adult behaviours are left

unsupervised. Therefore, this put the proportion of exposed juvenile and adult to be $e_1(0) = 0.4$ and $e_2(0) = 0.6$ respectively.

Current estimate according to UNAID (2017) put the number of infected juveniles and adult in Kenya to be about 1,600,000, with a total of 120,000 juveniles and 1,480,000 adults. Using similar approach as in the susceptible compartment where $N_1 = 19{,}201{,}631$ juveniles (0 – 14 years) and $N_2 = 28{,}362{,}665$ adults (15 years and above), we therefore have the proportion of infective to be $i_1(0) = 0.00625$ for the juveniles and $i_2(0) = 0.05218$ for adult populations.

According to UNAID factsheet (2017), about 68% of people living with HIV who need Highly Active Antiretroviral Treatment (HAART) have access to it making a total of about 1,088,000 people having assess to HAART in 2018 with about 976,000 juveniles and 1,104,000 adults where $N_1 = 19{,}201{,}631$ juveniles (0 – 14 years) and $N_2 = 28{,}362{,}665$ adults (15 years and above). We therefore have the proportion of infective receiving treatment in the juvenile and adult sub-population as $r_1(0) = 0.05093$ and $r_2(0) = 0.038924$ respectively.

Also, with a success rating in achieving the 90-90-90 targets envision for 2020, Kenya has seen a great rise to the achievement of 89% of infected people being aware of their HIV status and need HAART and 68% of them being actively under treatment of HAART. As a result of that about 65% of the 1,088,000 having assess to HAART in 2018 were virally suppressed, that is, the HIV in the 707,200 people is undetectable and the HIV treatment brings the level of the HIV in the body to such a low level that tests cannot detect it. As long as the HAART is adhered to and viral load remains undetectable (and monitored) they remain untrasmitable and hence cannot transmit to others and their health is not affected by HIV. Therefore, according to UNAID (2018) 68% of the Kenya infective population (69% and 61% adults and juvenile respectively) were on HAART, hence, 65% of that 69% adults and 61% juvenile are undetectable and untransmitable which makes up a total of 431,392 and 487,968 undetectable=untransmitable juvenile and adult sub-populations respectively with $N_1 = 19{,}201{,}631$ juveniles (0 – 14 years) and $N_2 = 28{,}362{,}665$ adults (15 years and above). We therefore have the proportion of virally suppressed population in the juvenile and adult sub-population as $u_1(0) = 0.022466$ and $u_2(0) = 0.017205$ respectively.

(b) Death rate due to AIDS (α_1, α_2) and maximum death rate due to AID (α_0)

These are all gotten from UNAID (2017). See Table 1

(c) Disease induced death rate of the infected juveniles and infected adults not receiving HAART (φ_1, φ_2).

These are all gotten from UNAID (2017). See Table 4.3

(d) Disease induced death rate of the infected juveniles and infected adults (ϖ_1, ϖ_2) receiving HAART.

To get the value for ϖ_1 and ϖ_2, we use the formula $\varpi_i = \varphi_i e^{-kT}$ where $i = 1, 2$ represent the juvenile and adult sub-population respectively. ϖ_1 and ϖ_2 represents the death rate of the juvenile and adult sub-population who are not receiving HAART. k is the efficacy of the drug and T is the maximum lifespan after infection, as provided by UNAIDS (2012). See Table 1.

(e) The rate of progression from HIV to AIDS in the juvenile and adult sub-population (ν_1, ν_2).

According to Oduwole and Kimbir (2017), without loss of generality, the rate of progression from HIV to AIDS in the juvenile and adult sub-population is taken to be $\nu_1 = 0.125$ and $\nu_2 = 0.070$ as early diagnosis of HIV infection in children is essential because due to weaker immune systems, the infection in infants and children tends to progress faster than in adults.

(f) Maturation rate of susceptible, exposed, infected, removed and undetectable juvenile to adults ($\eta_s, \eta_e, \eta_i, \eta_r$ and η_u)

Maturation is achieved by transferring a portion of the susceptible juvenile to its corresponding susceptible adult sub-population. In the susceptible juvenile compartment, we estimate the number of children alive, for each distinct age between 0 and 14, based on the annual mortality and population growth rate of Kenya. We then divide the number of 14 years old by the total size of the juvenile sub-population (Oduwole and Kimbir 2017). This will result in the rate of children who will turn 13 and will thus enter the sexually active adult class. The maturation rate for susceptible is thought to be higher than that for the infected population, which in turn is higher for the removed class receiving treatment. In the current research work, the estimated value for the maturation rate for each compartment is given as $\eta_s = 0.05$, $\eta_e = 0.04$, $\eta_i = 0.03$, $\eta_r = 0.02$ and $\eta_u = 0.01$.

(g) Probability of transmission by adult members of class I_2 and class R_2 (β, β')

The term β and β' are referred to as probabilistic terms that lies between 0 and 1 and it is expected that $\beta' < \beta$. In this research work, we choose to adopt probability of transmission values from Oduwole and Kimbir (2017) which states that probability of transmission is low if it falls within

the range ($\beta \leq 0.015,\ \beta' \leq 0.00136$) and it is high when it falls within the range ($\beta \geq 0.150$, $\beta' \geq 0.010$). For example in every 1000 adults, 15 transmit the disease in the infected compartment and 1 transmit the disease in the removed compartment is regarded as low transmission rate. Similarly, in every 1000 adults, 150 transmit the disease in the infected compartment and about 10 transmit the disease in the removed compartment is regarded as high transmission rate.

(h) Probability of secondary infection by recovered juveniles and adult populations in $U = U$ compartment (ρ_1, ρ_2)

The recruitment into the U-class from the R-class depends on the effectiveness of the HAART and the change in social behavior of the recovered population. This is done at a rate ρ_1 and ρ_2 for the recovered juvenile and adult sub-population respectively.

The recruitment into the S-class over again from the U-class depends on how long the population in the U-class remain in the class while actively receiving treatment. This stage it is assumed that the compartment is filled with fully removed population whose viral load is less than 1% and have 0% chance of secondary infection. This is done at a rate ε_1 and ε_2 for juvenile and adult sub-population respectively.

There is a chance of infection by the juvenile and adult population in the U=U class at ρ_1 and ρ_2 probability if the administration of HAART is discontinued at any given time.

The term ρ_1 and ρ_2 are referred to as probabilistic terms that lies between 0 and 1 and it is expected that $\rho_1 < \rho_2$ the probability of re-infection by juvenile is almost negligible but for the purpose of accuracy we take $\rho_1$into consideration no matter how small . In this research work, probability of transmission is low if it falls within the range ($\rho_2 \leq 0.25,\ \rho_1 \leq 0.0016$) and it is high when it falls within the range ($\rho_2 \geq 0.35$, $\rho_1 \geq 0.012$).

(i) Treatment rate of the juvenile and adult sub-population (σ_1, σ_2)

The term σ_1 and σ_2 are referred to as the proportion of those receiving treatment in comparison with the juvenile and adult sub-population respectively. It expressed as $\sigma_i = \frac{n(I_i)}{N_i}$. The treatment rate is low when it falls within the range ($\sigma_1 \leq 0.25\ ,\ \sigma_2 \leq 0.25$) for the juvenile and adult sub-population. Similarly treatment rate is high when it falls within the range ($\sigma_1 \geq 0.85\ ,\ \sigma_2 \geq 0.85$). For example in every 100 juveniles or adults that are infected, when 25 or less receive treatment,

then it is regarded as low treatment rate, while in every 100 juveniles or adults, when 75 and above receive treatment, it is regarded as high treatment rate.

(j) Rate of exposure or latency rate of juvenile and adult sub-population (π_1, π_2)

The recruitment from the S-class into the E-class is through birth for newborns and through heterosexual activities for adults. This is done at a rate π_1 and π_2 for the juvenile and adult sub-population respectively. Due to the care given to pregnant mothers and proper vaccination during pregnancy the rate of latency for juvenile will be low as compared to that of adults. For adults, a lot of factors exposes them or makes them more latent than the juvenile and some of this factors could include heterosexual relationships, use of unsterilized syrings for drugs and medication, and other uncultured behaviours hence in this research, $\pi_1 < \pi_2$.

(k) Average number of sexual partners in the I_2 and R_2 class (c, c')

The average number of sexual partners in the infected class and removed class is 1 respectively. Although it is expected that $c' < c$ since $\beta' < \beta$.

(l) Proportion of infected newborn (ξ) and healthy $(1-\xi)$ newborn

The term ξ and $(1-\xi)$ are referred to as the proportion of those children born with the disease and those born healthy. Hence this parameter must lies between 0 and 1 $(0 \leq \xi \leq 1)$. It expressed as

$$\xi = \frac{\text{Numbers of healthy newborn}}{\text{Total Number of babies born by infected and susceptable mothers}}.$$

$$1-\xi = \frac{\text{Numbers of Infected newborn}}{\text{Total Number of babies born by infected and susceptable mothers}}$$

(m) Incidence rate in juvenile and adult sub-population $(B_1(t), B_2(t))$

The incidence rate in the juvenile sub-population is negligible because there is no sexual contact and hence $B_1(t) = 0$ and that of the adult sub-population, because there is actively a force of infection, $B_2(t) > 0$.

(n) Probability of induced death of juvenile and adult sub-population not receiving HAART (τ_1, τ_2)

These are all gotten from UNAID (2017). See Table 1.

(o) Proportion of removed juveniles and adult still receiving treatment and being moved to susceptible class $(\varepsilon_1, \varepsilon_2)$.

These are all gotten from UNAID (2017). See Table 1.

4.2 NUMERICAL EXPERIMENTS OF THE MODEL

The age-structured deterministic model (3.34) – (3.43) was solved numerical using Runge-Kutta-Fehllberg 4-5th order method and implemented using Maple 15 Software (Maplesoft, Waterloo Maple Inc, 2012). The model equations were first transformed into proportions, thus reducing the model equations to ten differential equations. The parameters used in the implementation of the model are shown in Table 1 below. Parameters were chosen in consonance with the threshold values obtained in the stability analysis of the disease free equilibrium state of the model.

Table 1. Estimated values of the parameters used in the Numerical experiments

Parameters	Values	Source	Parameters	Values	Source
$N(0)$	47,564,296	UNAID (2018)	$\boldsymbol{c'}$	1.00**	Assumed
$N_1(0)$	19,201,631	UNAID (2018)	$B_2(t)$	0.0867194*	Author's computation
$N_2(0)$	28,362,665	UNAID (2018)	T	10	UNAID & WHO (2019)
$s_1(0)$	0.4	UNAID (2018)	α_0	0.0408	UNAID & WHO (2019)
$e_1(0)$	0.28998572	UNAID (2018)	α_1	0.0144	UNAID (2018)
$i_1(0)$	0.00625	UNAID (2018)	ρ_1	0.012	UNAID (2018)
$r_1(0)$	0.05093	UNAID (2018)	α_2	0.0122	UNAID (2018)
$u_1(0)$	0.022466	UNAID (2018)	φ_1	0.0760	UNAID (2018)
ν_1	0.125	Kgosi .. (2006)	ρ_2	0.35	UNAID (2018)
s_2**(0)**	0.5963	UNAID (2018)	φ_2	0.0520	UNAID (2018)
$e_2(0)$	0.6	UNAID (2018)	μ_1	0.0890	UNAID (2018)
i_2**(0)**	0.05218	UNAID (2018)	π_1	0.025**	Assumed
r_2**(0)**	0.038924	UNAID (2018)	μ_2	0.01512	UNAID (2018)
$u_2(0)$	0.017205	UNAID (2018)	λ	0.028595	WHO (2019)
ν_2	0.070	Kgosi .. (2006)	π_2	0.05**	Assumed
$\boldsymbol{\eta_s}$	0.05***	Assumed	m	1.4770966	UNAID (2018)
$\boldsymbol{\eta_e}$	0.04***	Assumed	K	0.5	UNAID (2019)
$\boldsymbol{\eta_i}$	0.03***	Assumed	β	0.150	Assumed
$\boldsymbol{\eta_r}$	0.02***	Assumed	β'	0.010	Assumed
$\boldsymbol{\eta_u}$	0.01***	Assumed	σ_1	0.25**	Assumed
ϖ_1	0.0005121*	Computed	σ_2	0.25**	Assumed
ϖ_2	0.0003503*	Computed	$1-\xi$	0.85**	Assumed
c	1.00**	Assumed	ξ	0.15**	Assumed
ε_1	0.09142	Assumed	τ_1	0.04529	Assumed
ε_2	0.18425	Assumed	τ_2	0.36217	Assumed
a_1	0.006124	Computed	a_2	0.0057117	Computed

Computed based on parameter values*: $\varpi_i = \varphi_i e^{-kT} B_2(t) = (c\beta I_2 + c'\beta' R_2 + \sigma_2\pi_2 E_2)/N_2$

Assumed:** Hypothetical data use for research purpose (See section 4.4.1) **Assumed***: Based on [1].

4.3 GRAPHICAL REPRESENTATION OF RESULTS

Experiment 1: The effect of treatment on recovery in the juvenile sub-population when the probability of secondary transmission is low ($\rho_1 = 0.012,\ \rho_2 = 0.35$) in the adult sub-population

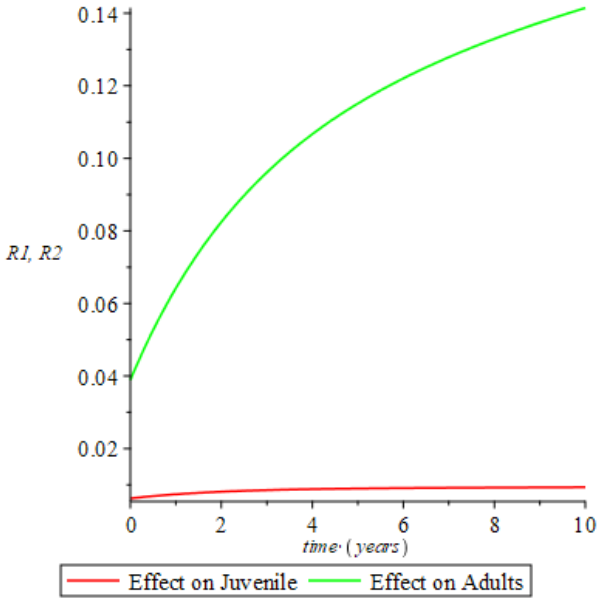


Figure 4.1 Recovery in the Juvenile sub-population when the probability of secondary transmission is low in the adult sub-population ($\sigma_1 = 0.25,\ \sigma_1 = 0.85,\ \rho_1 = 0.012,\ \rho_2 = 0.35$).

Experiment 2: The effect of treatment on recovery in the adult sub-population when the probability of secondary transmission is low ($\rho_1 = 0.012,\ \rho_2 = 0.35$) in the adult sub-population

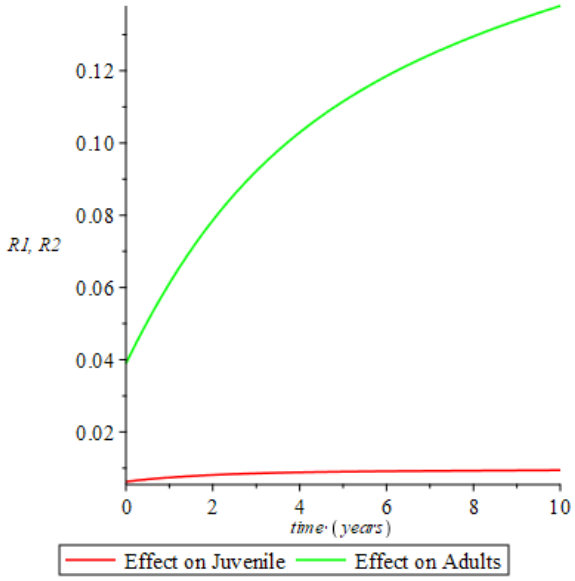


Figure 4.2 Recovery in the Adult sub-population when the probability of transmission is low in the adult sub-population ($\sigma_2 = 0.25,\ \sigma_2 = 0.75,\ \rho_1 = 0.012,\ \rho_2 = 0.35$)

Experiment 3: The effect of treatment on recovery in the juvenile sub-population when the probability of secondary transmission is high ($\rho_1 = 0.150,\ \rho_2 = 0.010$) in the adult sub-population

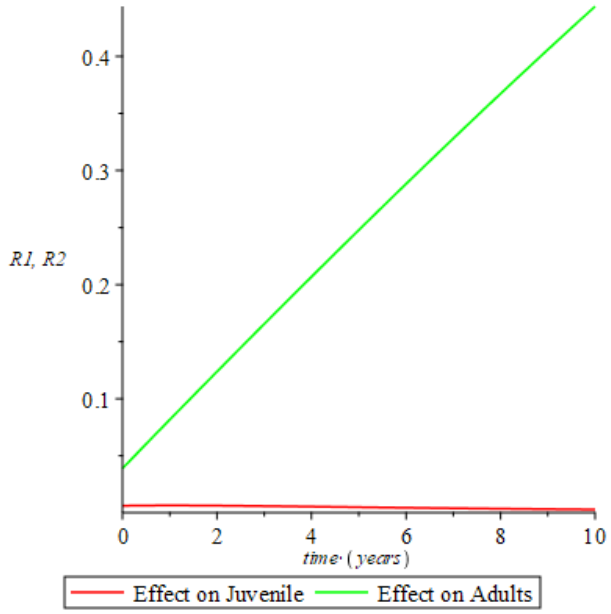


Figure 4.3 Recovery in the Juvenile sub-population when the probability of transmission is high in the adult sub-population ($\sigma_1 = 0.25,\ \sigma_1 = 0.85,\ \rho_1 = 0.150,\ \rho_2 = 0.010$)

Experiment 4: The effect of treatment on recovery in the adult sub-population when the probability of recovery transmission is high ($\rho_1 = 0.150,\ \rho_2 = 0.010$)

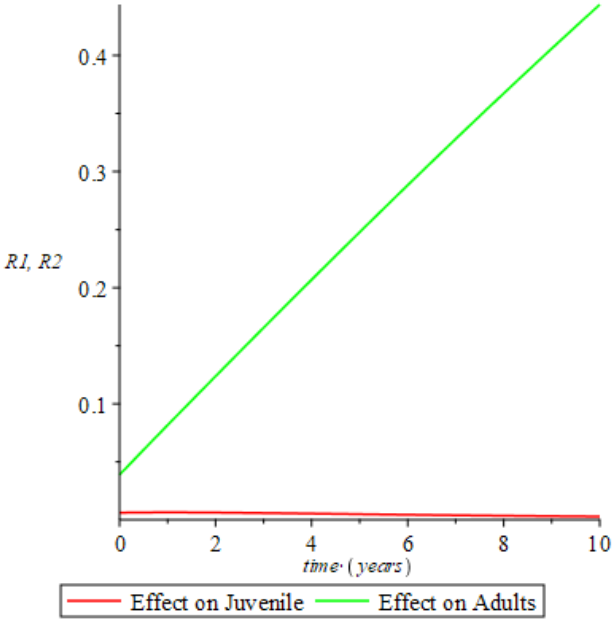


Figure 4.4 Recovery in the Adult sub-population when the probability of transmission is high in the adult sub-population ($\sigma_2 = 0.25,\ \sigma_2 = 0.85,\ \rho_1 = 0.150,\ \rho_2 = 0.010$)

Experiment 5: The effect of low treatment rate ($\sigma_1 = \sigma_2 \leq 0.25$) recovery in the juvenile and adult sub-population when the probability of secondary transmission is low ($\rho_1 = 0.012,\ \rho_2 = 0.35$)

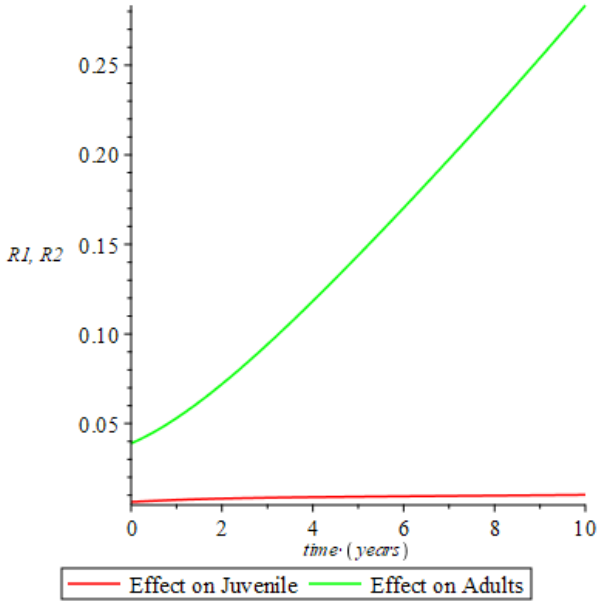


Figure 4.5 Recovery when the treatment rate is low and the probability of transmission is low ($\sigma_1 = 0.25,\ \sigma_2 = 0.25,\ \rho_1 = 0.012,\ \rho_2 = 0.35$)

Experiment 6: The effect of high treatment rate ($\sigma_1 = \sigma_2 \geq 0.85$) on recovery in the juvenile and adult sub-population when the probability of secondary transmission is low ($\rho_1 = 0.012,\ \rho_2 = 0.35$)

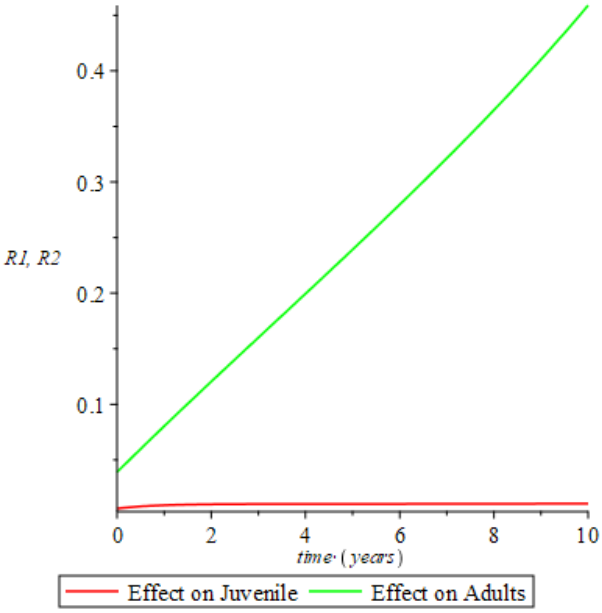


Figure 4.6 Recovery when the treatment rate is high and the probability of transmission is low ($\sigma_1 = 0.85,\ \sigma_2 = 0.85,\ \rho_1 = 0.012,\ \rho_2 = 0.35$).

Experiment 7: The effect of low treatment rate ($\sigma_1 = \sigma_2 \leq 0.25$) on recovery in the juvenile and adult sub-population when the probability of secondary transmission is high ($\rho_1 = 0.150$, $\rho_2 = 0.010$)

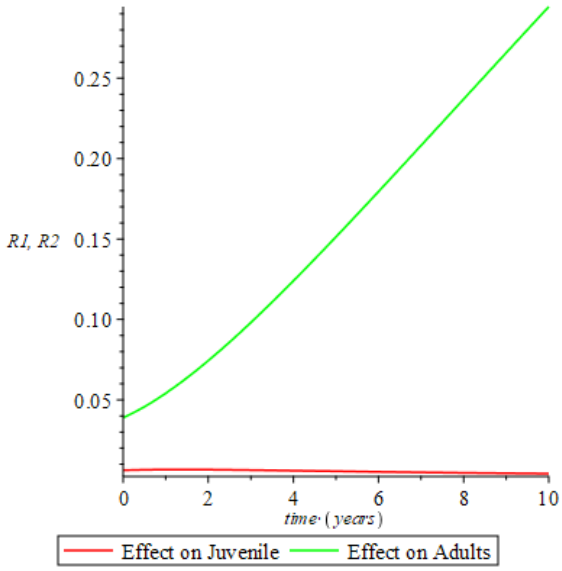


Figure 4.7 Recovery when the treatment rate is low and the probability of transmission is high and ($\sigma_1 = 0.25$, $\sigma_2 = 0.25$, $\rho_1 = 0.150$, $\rho_2 = 0.010$).

Experiment 8: The effect of high treatment rate ($\sigma_1 = \sigma_2 \geq 0.85$) on recovery in the juvenile and adult sub-population when the probability of secondary transmission is high ($\rho_1 = 0.150$, $\rho_2 = 0.010$)

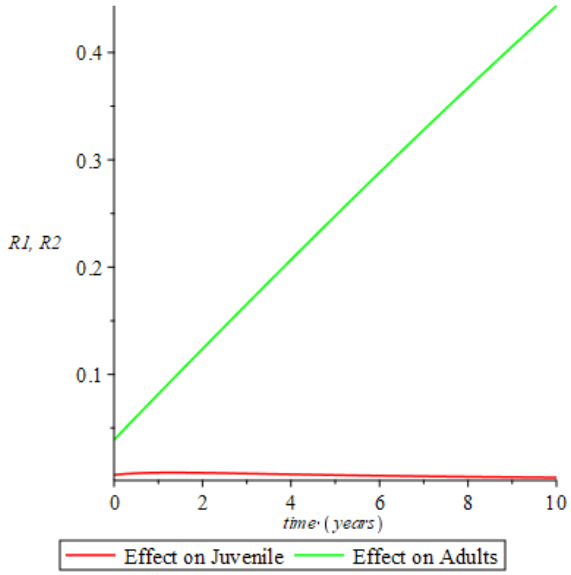


Figure 4.9 Recovery when the treatment rate is high and probability of secondary transmission is high ($\sigma_1 = 0.85$, $\sigma_2 = 0.85$, $\rho_1 = 0.150$, $\rho_2 = 0.010$)

Experiment 10: The effect of low treatment rate ($\sigma_2 \leq 0.25$) on recovery when the probability of secondary transmission is low ($\rho_1 = 0.012,\ \rho_2 = 0.35$) and the juvenile sub-population is left untreated

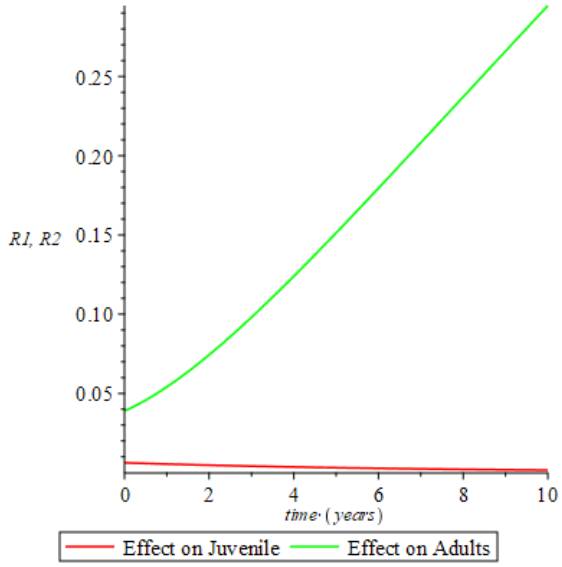


Figure 4.10 Recovery with low treatment rate when the probability of transmission is low and the juvenile sub-population is left untreated ($\sigma_1 = 0.00,\ \sigma_2 = 0.25, \rho_1 = 0.012,\ \rho_2 = 0.35$).

Experiment 11: The effect of high treatment rate ($\sigma_2 \geq 0.85$) on recovery when the probability of secondary transmission is low ($\rho_1 = 0.012,\ \rho_2 = 0.35$) and the juvenile sub-population is left untreated

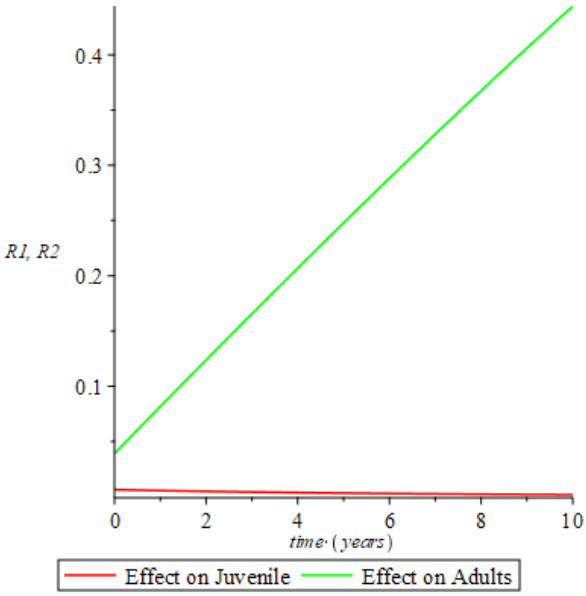


Figure 4.11 Recovery with high treatment rate when the probability of transmission is low and the juvenile sub-population is left untreated ($\sigma_1 = 0.00,\ \sigma_2 = 0.85, \rho_1 = 0.012,\ \rho_2 = 0.35$).

Experiment 12: The effect of low treatment rate ($\sigma_2 \leq 0.25$) on prevalence of infection when the probability of secondary transmission is high ($\rho_1 = 0.150$, $\rho_2 = 0.010$) and the juvenile sub-population is left untreated

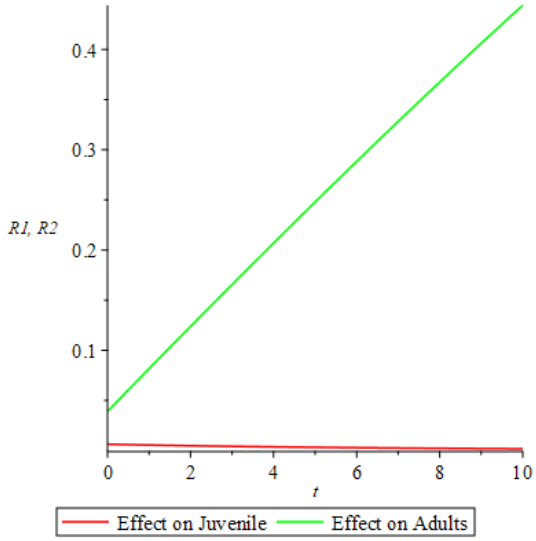


Figure 4.12 Recovery with low treatment rate when the probability of transmission is high and the juvenile sub-population is left untreated ($\sigma_1 = 0.00$, $\sigma_2 = 0.25$, $\rho_1 = 0.150$, $\rho_2 = 0.010$)

Experiment 13: The effect of high treatment rate ($\sigma_2 \geq 0.85$) on recovery when the probability of secondary transmission is high ($\rho_1 = 0.150$, $\rho_2 = 0.010$) and the juvenile sub-population is left untreated

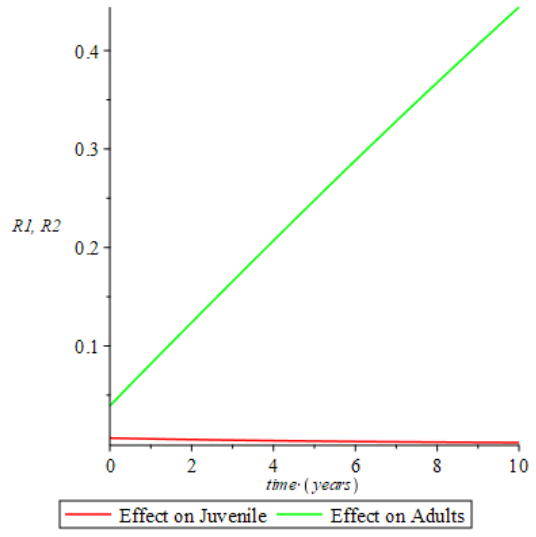


Figure 4.13 Recovery with high treatment rate when the probability of transmission is high and the juvenile sub-population is left untreated ($\sigma_1 = 0.00$, $\sigma_2 = 0.85$, $\beta = 0.150$, $\beta' = 0.010$).

Experiment 14: The effect of low treatment rate ($\sigma_1 \leq 0.25$) on recovery when the probability of secondary transmission is low ($\rho_1 = 0.012,\ \rho_2 = 0.35$) and the adult sub-population is left untreated

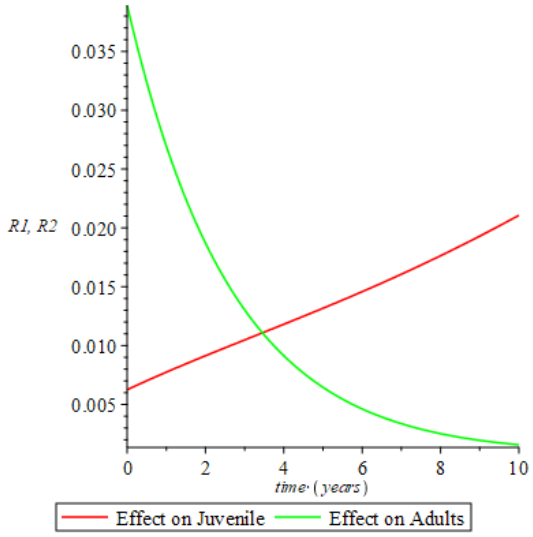


Figure 4.14 Recovery with low treatment rate when the probability of transmission is low and the adult sub-population is left untreated ($\sigma_1 = 0.25,\ \sigma_2 = 0.00, \rho_1 = 0.012,\ \rho_2 = 0.35$)

Experiment 15: The effect of high treatment rate ($\sigma_1 \geq 0.85$) on recovery when the probability of secondary transmission is low ($\rho_1 = 0.012,\ \rho_2 = 0.35$) and the adult sub-population is left untreated

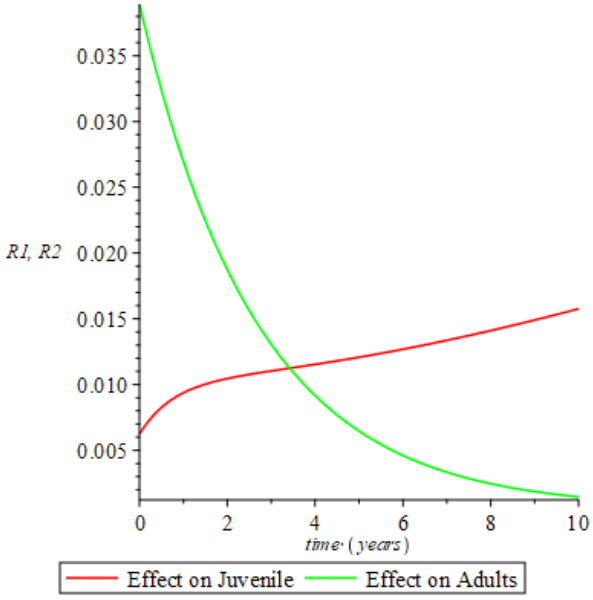


Figure 4.15 Recovery with high treatment rate when the probability of transmission is low and the adult sub-population is left untreated ($\sigma_1 = 0.85,\ \sigma_2 = 0.00, \rho_1 = 0.012,\ \rho_2 = 0.35$).

Experiment 16: The effect of low treatment rate ($\sigma_1 \leq 0.25$) on recovery when the probability of secondary transmission is high ($\rho_1 = 0.150,\ \rho_2 = 0.010$) and the adult sub-population is left untreated

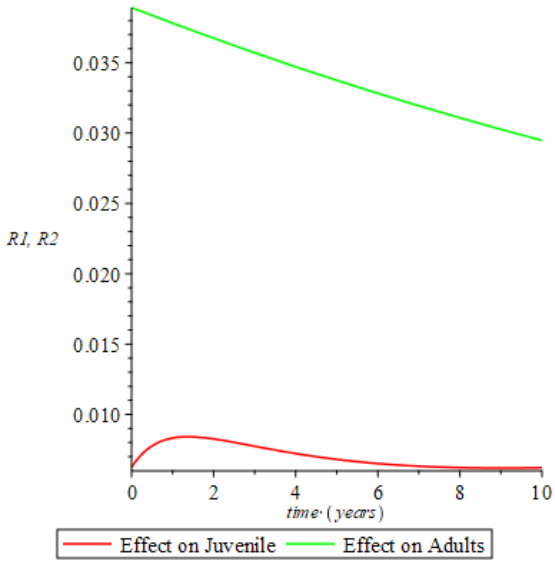


Figure 4.16 Recovery with high treatment rate when the probability of transmission is high and the adult sub-population is left untreated ($\sigma_1 = 0.85,\ \sigma_2 = 0.00, \rho_1 = 0.150,\ \rho_2 = 0.010$).

Experiment 17: The effect of high treatment rate ($\sigma_1 \geq 0.85$) on recovery when the probability of secondary transmission is high ($\rho_1 = 0.150,\ \rho_2 = 0.010$) and the adult sub-population is left untreated

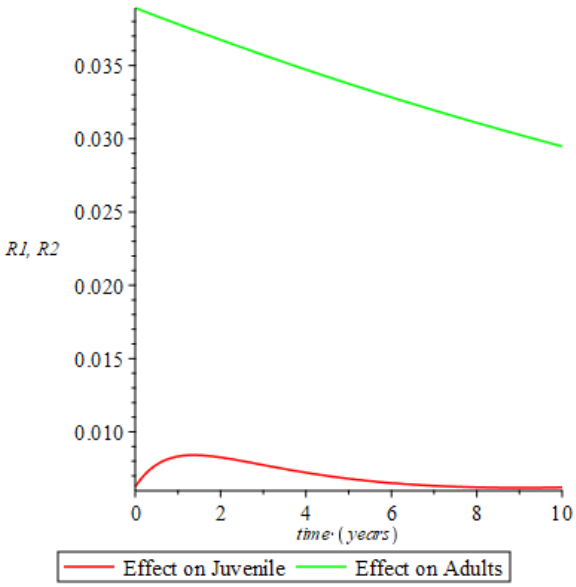


Figure 4.17 Recovery with high treatment rate when the probability of transmission is high and the adult sub-population is left untreated ($\sigma_1 = 0.85,\ \sigma_2 = 0.00, \rho_1 = 0.150,\ \rho_2 = 0.010$).

Experiment 18: The effect of low treatment rate ($\sigma_1 = \sigma_2 \leq 0.25$) on recovery in the juvenile and adult sub-population when all newborns from infected mothers are healthy ($\xi = 0.0$)

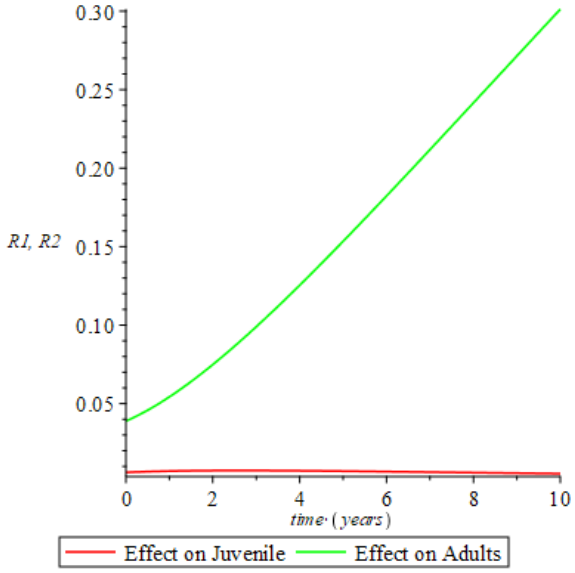


Figure 4.18 Recovery in the juvenile and adult sub-population when all newborns from infected mothers are healthy and treatment rate is low ($\sigma_1 = 0.25,\ \sigma_2 = 0.25,\ \rho_1 = 0.075,\ \rho_2 = 0.005,\ \xi = 0.0$).

Experiment 18: The effect of high treatment rate ($\sigma_1 = \sigma_2 \geq 0.85$) on recovery in the juvenile and adult sub-population when all newborns from infected mothers are healthy ($\xi = 0.0$)

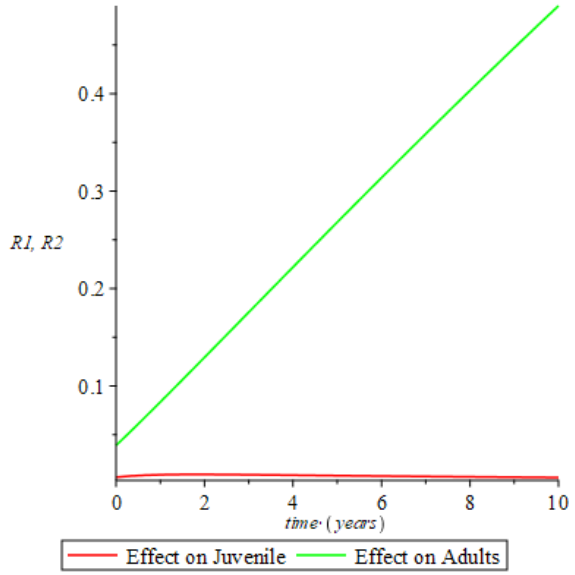


Figure 4.18 Recovery in the juvenile and adult sub-population when all newborns from infected mothers are healthy and treatment rate is high ($\sigma_1 = 0.85,\ \sigma_2 = 0.85,\ \rho_1 = 0.06,\ \rho_2 = 0.008,\ \xi = 0.0$)

Experiment 19: The effect of low treatment rate ($\sigma_1 = \sigma_2 \leq 0.25$) on recovery in the juvenile and adult sub-population when all newborns from infected mothers are HIV positive ($\xi = 1.0$)

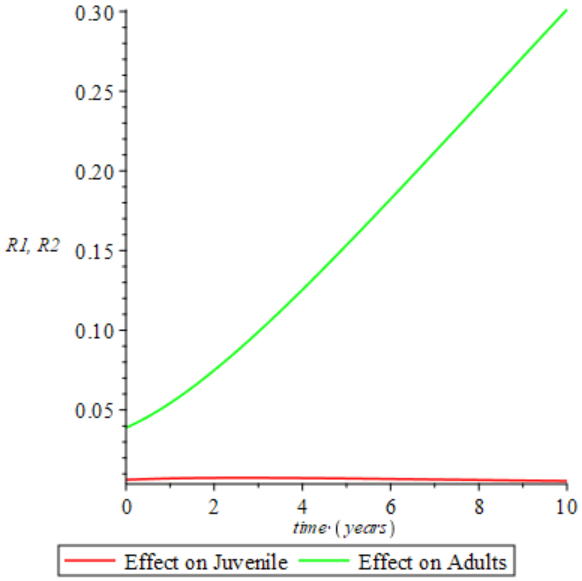


Figure 4.19 Recovery in the juvenile and adult sub-population when all newborns from infected mothers are HIV positive and treatment rate is low. ($\sigma_1 = 0.25,\ \sigma_2 = 0.25,\ \rho_1 = 0.075,\ \rho_2 = 0.005,\ \xi = 1.0$).

Experiment 20: The effect of high treatment rate ($\sigma_1 = \sigma_2 \geq 0.85$) on recovery in the juvenile and adult sub-population when all newborns from infected mothers are HIV positive($\xi = 1.0$)

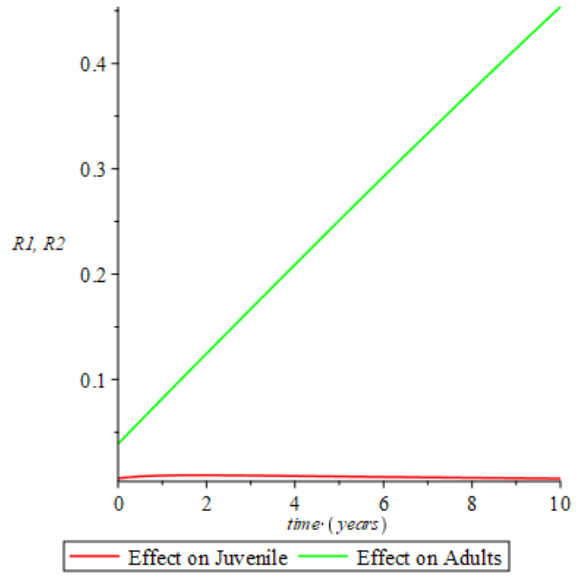


Figure 4.20 Recovery in the juvenile and adult sub-population when all newborns from infected mothers are HIV positive and treatment rate is high ($\sigma_1 = 0.85,\ \sigma_2 = 0.85\ , \rho_1 = 0.075,\ \rho_2 = 0.005,\ \xi = 1.0$).

<u>Experiment 21</u>: The effect of vertical transmission on the recovery in the juvenile and adult sub-population when newborns from infected mothers are HIV positive at different proportion ($\xi_+ = 1.0,\ \xi_- = 0.0$)

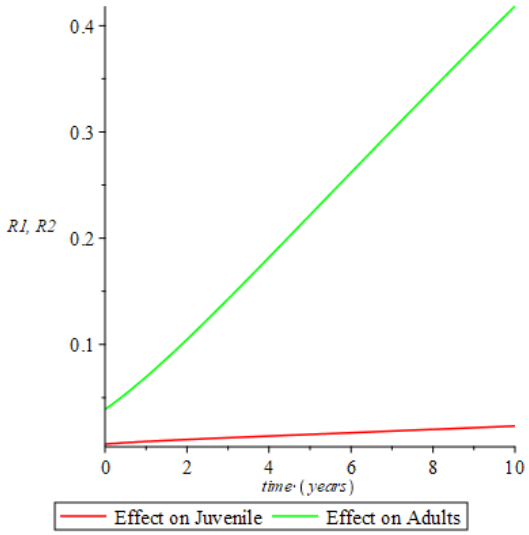


Figure 4.21 Recovery in the juvenile sub-population showing the effect of vertical transmission when all newborns from infected mothers are HIV negative and HIV positive respectively. ($\sigma_1 = 0.5,\ \sigma_2 = 0.5\ ,\ \rho_1 = 0.075,\ \rho_2 = 0.005,\ \xi_+ = 1.0,\ \xi_- = 0.0$).

4.4 DISCUSSION OF RESULTS

In experiment 1 and 3, the effect of HAART treatment on the recovery compartment to the undetectable=untransmitable compartment in the juvenile sub-population was investigated as shown in Figure 4.1 and Figure 4.3 when the probability of secondary transmission is low and high respectively. From Figure 4.1 as the probability of secondary transmission in the adult sub-population seems to decline progressively ($\sigma_1 = 0.25$) due to the administration of HAART to the recovered compartment, the recovery rate in the juvenile sub-population is seen to remain normal in a declining form over a longer period of time while that of the adult sub-population seems to increase sharply. It therefore means if the treatment is administered progressively mostly among women and there is less MTC vertical transmission, there will continually be a stiff rise in the recovery rate among juvenile as the secondary transmission among adults, especially women continuous to decline after recovery especially after proper administration of HAART ($\sigma_1 = 0.25,\ \sigma_1 = 0.85,\ \rho_1 = 0.012,\ \rho_2 = 0.35$). However, when the probability of secondary transmission

among adult sub-population is high, the recovery rate is high upon the administration of HAART treatment as can be seen in Figure 4.3 because as much as efforts is made to control the spread of HIV/AIDS in children who have no incidence rate ($B_1(t) = 0$) there is need to control the secondary transmission rate of the disease in adults before treatment is embarked upon. However, failure to control the secondary transmission among children will render the efforts to curb the rise in prevalence among children useless. Also, recovered juvenile sub-population would be exposed to the disease further instead of moving to the undetectable=untransmitable compartment.

Meanwhile, in Experiment 2 and 4, the effect of treatment on the recovery in the adult sub-population was investigated as shown with a bell curve in Figure 4.2 and Figure 4.4 when the probability of transmission is low and high respectively. Result shows clearly that the recovery rate rises over a long period in the adult sub-population when the probability of secondary transmission is low from the recovered compartment actively administering HAART over a short period of time as seen in Figure 4.2 and rises persistently over a long period of time when the probability of secondary transmission is high from the recovered compartment actively administering HAART over a long period of time and even after recovery as seen in Figure 4.4. Therefore, to enhance the movement of adult sub-population in an endemic situation from the recovered compartment to the undetectable compartment over a long period of time, the probability of transmission must be reduced to the barest minimum or possibly negligible. This can be achieved by adequately administering the HAART treatment as well creating awareness among adult sub-population on safe sex behaviors and other behavioral changes as required to reduce the risk of contracting or re-contacting the disease. In other to achieve the goal of CDC (2017), the rate of secondary transmission after recovery by the recovered compartment actively administering HAART must be zero and hence according to this study, it is found that the Reproductive number is zero ($R_0 = 0$) since there is a zero incidence rate among the juvenile sub-population.

Comparing Figure 4.5 and Figure 4.6 with Figure 4.7 and Figure 4.8 while investigating the effect of treatment when the secondary transmission rate is low and high respectively. As shown in Figure 4.5 and Figure 4.6 when the probability of secondary transmission is low and treatment rate is high, then the proportion of recovery in the adult sub-population is high over a considerably long period of time. Therefore, when the probability of secondary transmission is high and treatment

rate is high then recovery rate will increase accordingly as seen in Figure 4.7 and 4.8 respectively. Although rate of incidence among juvenile sub-population is zero, the recovery rate among adult sub-population is more applauding due to the low transmission rate and the high treatment that enhances recovery among adults. So in other to attain the goal of the CDC (2018) for undetectable=untransmittable in the entire population, both the adult and juvenile sub-population must actively be on HAART to prevent infected juvenile to mature with the disease into the adult sub-population.

Similarly, the effect of treatment on recovery of adult sub-population when the juvenile sub-population is left untreated is presented as Experiment 9, 10, 11 and 12 shows that the treatment of adult sub-population enhances recovery in both adult and juvenile sub-population even specifically when treatment rate is high and transmission rate is low. This is evident that with the active administration of the HAART treatment to both groups, the adult sub-population will move steadily into the undetectable=untransmitable compartment while the treated juveniles mature into undetectable adults as seen in Figure 4.9 and Figure 4.10. And in Figure 4.11 and 4.12, it is evident that as treatment rate increase in the adult sub-population there is sharp rise of recovery in a long run. Hence, it is significant that both juvenile and adult sub-population undergo active vaccination and preventive procedures at the same time to ensure the full control of cases of secondary infection from the recovered compartment and as a result, the possible eradication of the disease even in an endemic situation. In Experiment 13 when the rate of treatment is low and the probability of secondary transmission after recovery is low and the adult sub-population is left untreated, the recovery rate experiences a sharp decline in a short run as shown in Figure 4.13. Similarly, when the treatment rate is high as in Experiment 14 with low probability of secondary transmission and the adult sub-population is left untreated, the rate of secondary infection accumulates in small proportions and the recovery rate declines rapidly and proportionately. Meanwhile, as in Experiment 15 and 16, when the treatment rate is high and the probability of secondary transmission is high when the adult sub-population is left untreated the juvenile recovery rate declines swiftly and it therefore means the secondary infection rate of the juvenile population also increases (see Figure 4.15 and Figure 4.16).

However, the effect of vertical transmission (also referred to as Mother-to-Child transmission) was also captured in Experiment 17, 18, 19, 20 and 21. Figure 4.17 and Figure 4.18 shows that when

all newborns from infected mothers are healthy ($\xi = 0$), the recovery rate in both juvenile and mostly adult sub-population increases drastically and the disease is eradicated when treatment is sustained after recovery and the probability of secondary infection is zero. However, when all the newborns from infected mothers are all HIV positive ($\xi = 1$) as shown in Figure 4.19 and Figure 4.20, the recovery rate drops slowly among juvenile but increases among adult sub-population when treatment rate is both low and high respectively for both age-structures.

In conclusion, in other to understand the impact of different proportions of recovered juveniles moving to the undetectable compartment in the juvenile sub-population when treatment rate is low and high in Kenya, Experiment 21 in Figure 4.21 illustrates that high treatment rate is necessary to control vertical secondary transmission of the disease, especially when the probability of transmission is negligible.

5. CONCLUSIONS

In this research, the existence and uniqueness of the endemic equilibrium state of the new compartmental deterministic mathematical SEIRUS model of HIV/AIDS endemic that embodied vertical and heterosexual transmission, as well as adult and juvenile age-structured with the effect of Highly Active Antiretroviral Therapy (HAART) was developed, implemented and analyzed with very interesting results for Kenya. Results from the implementation of the model data on all three countries shows that there is a need to enhance the availability and active use of the HAART treatments to both infected and recovered juvenile and adult members of the *I*-class and *R*-class respectively as this would not only increase the prevalence of infection but also the recovery rate if risky sexual behavious among heterosexual adults is not controlled. Behavioral interventions is key based on findings from this research as well as active administration of the HAART treatment in other to control secondary re-infection in an endemic situation where vertical transmission is in control and the assumption of zero immigration is true.

If the model threshold parameters are satisfied without loose of generality, the spread and secondary transmission of HIV/AIDS among heterosexual adults and Mother-to-Child transmission can be controlled and possibly eradicated as this study suggests with the secondary Reproductive Number being zero ($R_0 = 0$) there would not be further infection in the adult sub-

population and the juvenile sub-population incidence rate is also zero ($B_1(t) = 0$). Also, as much as efforts are on the way to attain U=U in Kenya, the active administration of HAART treatment on juvenile and adults sub-population is very key and when sufficiently and actively administered would ensure that the transmission rate between infective and recovered individuals is zero. An eradication of secondary transmission in a close population to attain a zero reproductive number can be achieved through active administration of HAART, high and target health education, counselling and testing as well as behavioral change of all the classes of the SEIRUS compartment.

Also, as much as the rate of vertical transmission has been considerably reduce to about 5% in Africa, the role of Mother to Child (MTC) transmission cannot be ignored because the rate of maturation from juvenile to adult in the susceptible class η_s, exposed class η_e, infectious class η_i, and the recovered class η_r, affects the endemic equilibrium state of the entire population. With the new Reproductive Number $R_0 = 0$ then the probability of transmission from adult to adult and mother to child is reduced to zero and the susceptible juvenile must retain their HIV negative status as they mature to adults, also the recovered juvenile must retain their HIV negative status as they mature to adult in other to move into the undetectable class of the adult sub-population. The maturation rate of susceptible and recovered juvenile must be taken into consideration when planning strategies for control and eradication of the HIV/AIDS disease in Africa as well as retaining the HIV-negative status of both the juvenile and adult sub-populations.

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