Mathematical modelling for assessing the impact of intervention strategies on HIV/AIDS high risk group population dynamics

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ABSTRACT

The global economic importance of HIV/AIDS and inadequacy of the literature dealing with deterministic model on control strategy of HIV/AIDS that captures factors responsible for the spread of the disease in low income country is one among other factors responsible for the ill management of the disease in an endemic population. Thus, this study focuses on a novel deterministic mathematical model to assess the impact of intervention strategies on high risk group with drug quitters special case and drug users who are sexually active (duality case). Backward bifurcation, positivity of the model variables, sensitivity analysis, global stability of DFE and EEP are used to investigate the qualitative structure of the model. It was established that the duality effect of the force of infection spur by intravenous drug users who sexually active (IDUSA-special case) are responsible for backward (IDUSA) in the endemic population. Sensitivity analysis on IDU and MSM group shows that 20% decrease of the susceptible population of both classes will attenuate the transmission dynamics by 7.4% and 2.6% of the MSM and IDU class respectively. Further simulation, using demographic and epidemiological data available in Nigeria shows that the disease burden reduces with universal strategy than combined strategy.

1. Introduction

Epidemiology is the study of frequency, distribution and determinants of infectious disease (among human or animal populations, including micro-organisms) as well as their prevention [1]. An epidemiological study of an infectious disease involves its basic description through understanding the causes and mechanisms leading to spread of the disease into the host population. The host population is a group of humans, animals etc., affected by an infection outbreak.

Deterministic mathematical models have been widely used to ascertain the spread and control of emerging and re-emerging human disease, Kermack and McKendrick [1, 2]. The dynamics of these models is determined by the threshold quantity $R_0$, called the basic reproduction number. Which ascertain the number of new cases an index case can generate in a completely susceptible population [1]. The phenomenon where the disease-free equilibrium loses its stability and a stable endemic equilibrium appears as $R_0$ increases through one, is known as forward bifurcation [3]. The implication of forward bifurcation is that the epidemiological requirement $R_0 < 1$ is necessary and sufficient condition for disease extinction. It has been established that $R_0 < 1$ though necessary may not be sufficient for disease eradication. This is due to the co-existence of a stable disease free equilibrium and stable endemic equilibrium. Co-existence of such is called backward bifurcation. It is imperative to provide a brief history of HIV/AIDS, since the focus of this paper is to assess the impact of intervention strategies on HIV/AIDS high risk group population dynamics.

HIV is a virus that spread via certain body fluids such as the blood, semen, fluid from reproductive organ, breast milk, which can be contracted through the use of drugs and sex. The virus typically attacks the body immune system, especially the CD4 cells, often called the T-cells [4]. Overtime, if the HIV is left untreated, it reduces the number of CD4 cells in the body, making the infected individual vulnerable to other infection. Thereafter, it reduces the immune system of the body and makes it unable to fight off the infection of other diseases. The HIV person is said to have AIDS when the CD4 T-Cells count is less than 200. The stages of progression of HIV include; 1) Acute infection which last within two to four weeks after

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1 HIV/AIDS mathematical model on high risk groups.

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infection: During this period, one may feel flu-like symptoms called acute retro viral syndrome (ART) or primary HIV infection [5]. One has the highest likelihood to spread HIV at this stage due to the amount of viral load in the blood. After this period, the immune system begins to build again but it may not return to pre-infection level [5]. 2) Clinical latency, sometime called the asymptomatic HIV infection: At this stage, the virus reproduces but at a very low level. During this phase of infection, HIV is active within the lymph nodes. However, with anti-retroviral therapy (ART), latency can last for several decades and also reduces the tendency to transmit the infection. As time progresses, the viral load begins to increase and the CD4 cell count begins to drop and symptoms may begin to appear [5]. 3) Acquired immune deficiency syndrome (AIDS): This is the final stage. The immune system is completely damaged with the CD4 cells below 200 cells per mm³ of the blood. At this stage one becomes vulnerable to infections and the life expectancy of the patient drops. HIV has no cure or vaccination. However, treatment is available. Highly active retro viral therapy has been having resounding success at reducing morbidity and mortality of infected individuals (May and Ingle, 2011). According to world health organization (WHO), there are 35 million people living with HIV as at 2013 and 2.1 million become infected in 2013. Nineteen million do not know they are infected and 24.7 million of lives in sub-Saharan Africa [6], thirty nine million people have died since the first case was reported with about 1.5 million death in 2013 [5].

Despite control measures, HIV/AIDS sustained its exuding effect on human population, this is due to failure in administering preventive measures to high risk group. High risk group are key population that experienced a high epidemiological impact of the disease (HIV/AIDS) coupled with reduced access to health services in managing the disease (IBBS, 2014). Examples include, intravenous drug users (IDU), men who have sex with men (MSM).

Nsuami et al. (2018) design an HIV/AIDS population dynamics incorporated with ART treatment and pre-exposure prophylaxis. They considered the population $N(t)$ at a time $t$ to be homogeneous. It was found that an increase uptake of Prep from 0.01 to 0.02 decrease the basic reproduction number and further increase of the Prep uptake reduces the basic reproduction number less than one and converges $I_2(t)$ to zero. Thus, managing HIV with early treatment can decrease transmission and possibly reduce AIDS related death. On the other hand, Kaplan (1992) considers a deterministic model for the prevalence of HIV and AIDS among intravenous drug users (addicts). Kaplan assumed that the population amongst whom the disease spread is of size $n$, where $n$ is large. The population is homogeneous and drugs are injected in exhibition room (a place where individuals meet to share drug-injecting equipment). Kaplan showed that a single infected addicts in a population of uninfected needles and susceptible addicts will cause roughly $R_n$ new secondary cases of HIV infection amongst the addicts in his or her life time. In their book, Noble Malunguza and Christainah Chiyaka (2009) designed and analysed a deterministic compartmental sex structured HIV/AIDS model for assessing the effects of homosexual and bisexuals in heterosexual settings. Their model integrate control strategies (condom and ART only) as control measures. According to Noble Malunguza et al. homosexuality and bisexuality enlarge the epidemic in heterosexual settings provided the primary transmission threshold is greater than one. They observed that the system will exhibit forward bifurcation in the absence of bisexual compartment in the heterosexual population. Buseng and Cookes (1993) discussed a variety of diseases that transmit both horizontally and vertically and gave a comprehensive survey of the formulation and the mathematical analysis of compartment models that also incorporate vertical transmission.

Garba and Gumel (2010) formulated a mathematical model for HIV elimination in Nigeria. Their model is subdivided into six mutually exclusive compartments susceptible individuals $S(t)$, new infected individuals who are unaware of their HIV status $I_1(t)$, counselled infected individuals with no AIDS symptoms $I_2(t)$, non-counselled AIDS individuals $A_{nc}(t)$, counselled AIDS individuals $A_c(t)$, and infected individuals receiving drug. The model is as follows

\[
\begin{align*}
\frac{dS}{dt} &= \Delta - \lambda_S - \mu_S S \\
\frac{dI_1}{dt} &= \lambda_S - (\sigma + \eta + \mu)I_1 \\
\frac{dI_2}{dt} &= \tau I_1 - (\sigma + \mu)I_2 \\
\frac{dA_{nc}}{dt} &= \delta I_1 - (\sigma + \delta + \tau_c)A_{nc} \\
\frac{dA_c}{dt} &= \delta I_1 - (\sigma + \delta + \tau_c)A_c \\
\frac{dT}{dt} &= \tau_T A_T + \tau_C A_C - (\varphi \delta + \mu)T 
\end{align*}
\]

(1)

His end note was that, the prospects of effectively controlling HIV in Nigeria are reasonably high if some basic and affordable steps (control measures) are taken.

While their model is noteworthy for homogeneous population its lacks the merit for a heterogeneous population (IDUSA and IDUQD special case) which are the main drivers for backward bifurcation in HIV/AIDS population [7]. Hence, a comparative distinction of the former (Abba and Gumel-without heterogeneous group) and this current research, has proven that HIV/AIDS population with heterogeneous group is responsible for the disease persistence in the population and cumulative increase of disease. The model is discussed in details as follows.

2. Model formulation

The total population of the highly risked group in Nigeria at a time $t$, denoted by $N(i)$, is subdivided into twenty mutually exclusive compartments of susceptible MSM ($S_h$), susceptible IDU ($S_d$), new infected IDU unaware of their HIV ($I_{d1}$), counselled infected IDU with no AIDS symptoms ($I_{d2}$), counselled infected IDU who quit the intake of drugs ($I_{d3}$), sexually active intravenous drug users ($I_{d4}$), sexually active intravenous drug users who received counselled ($I_{d5}$), infected IDU with AIDS symptoms ($A_{d1}$), counselled infected IDU with AIDS symptoms ($A_{d2}$), Infected IDU who Received treatment ($T_{d}$). Infected intravenous drug users who quit the intake of drugs with AIDS symptoms ($A_{d3}$), infected intravenous drug users who quit the intake of drugs receiving ART ($T_{d4}$), sexual active intravenous drug users with AIDS symptoms ($A_{d4}$), sexually active intravenous drug users with AIDS symptoms who received counselled ($A_{d5}$), sexually active intravenous drug users with AIDS symptoms receiving ART ($T_{d5}$). Infected MSM with no AIDS symptoms ($I_h$), counselled infected MSM ($I_{d6}$), AIDS class of MSM who refuse counselled ($A_{h1}$), counselled AIDS class of MSM ($A_{h2}$), MSM treatment class ($T_h$). Thus the mutually exclusive compartmental class is:

\[
N(t) = S_h + S_d + I_{d1} + I_{d2} + I_{d3} + I_{d4} + I_{d5} + I_{d6} + I_{d7} + A_h + A_{h2} + A_{h3} + A_{h4} + A_{h5} + A_{d1} + A_{d2} + A_{d3} + A_{d4} + A_{d5} + T_{d1} + T_{d2} + T_{d3} + T_{d4} + T_{d5} + T_h + T_{d6} + T_{d7}. 
\]
2.0.1. Force of infection

The force of infection acquired by the MSM and IDU individuals following the effective contact with infected individuals in

\[ I_d, I_{qd}, I_{d1}, I_{d2}, I_{d3}, I_{d4}, I_{ch}, A_{d1}, A_{d2}, A_{d3}, A_{d4}, A_{ch}, A_{cd1}, A_{cd2}, T_{d1}, T_{d2}, T_{d3}, T_{d4}, T_d \]

is given by

\[ \lambda = \lambda_d + \lambda_s \]

where

\[ \lambda_s = \beta \left(1 - \epsilon \kappa \right) \left[ I_h + I_{d2} + a_{IN} A_h + a_{2N} A_h + \theta (I_{ch} + a_c A_{ch} + I_{cd} + a_{2c} A_{ch}) + \theta_2 T_h + \theta_3 T_{ch} \right] \]

\[ \lambda_d = \left[ I_d + I_{qd} + a_{1N} A_d + a_{2N} A_{qd} + \theta (I_{d1} + a_{2c} A_{cd1}) + \theta_2 T_{d1} + \theta_3 T_d \right] \]

The parameter \( \beta \) is the effective contact rate, \( 0 < \epsilon < 1 \) is the condom efficacy, \( 0 < \kappa < 1 \) measures compliance in condom use, \( a_{IN}, a_{2N}, a_N, a_c, a_{2c}, \) are modification parameters accounting for the relative risk of infectiousness of non-counselled and counselled individuals with AIDS of MSM and IDU individuals belonging to \( E_h \) and \( E_s \) class where: \( E_h = \{ E_h : \text{set of individuals who refused counselling} \} \), \( E_s = \{ E_s : \text{set of individuals who received counselling} \} \). The modification parameters, \( \theta_2, \theta_{d1}, \theta_d, \) account for the reduction in the transmission rates of counselled and treated individuals in relation to infected individuals, since treatment reduces infectiousness in treated individuals it is plausible to set \( 0 < \theta_2 < 1 \), where: \( \theta_2 = (\theta_2, \theta_{d1}, \theta_d, \theta_d) \). Similarly, it is assumed that counselled infected MSM and IDU modify their sexual behaviour and intake of drugs positively so that \( \theta_d < 1 \).

Susceptible IDU (\( S_d \)): Susceptible IDU class is assumed to be sexually active, thus the population is diminished by force of infections through sex and drugs at a rate \( \gamma_d \) and \( \lambda_d \) respectively, where \( \gamma_d \) account for the reduction modification parameter of infectiousness through sex. The population is further diminished by peer pressure and natural mortality at a rate \( \mu \) and \( \mu \) respectively. \((1 - \rho)A\) is the recruitment rate for IDU class.

\[ dS_d/dt = (1 - \rho)A - (\lambda_d + \gamma_d)S_d - \mu S_d - \Gamma_1 S_d \]

Susceptible MSM (\( S_h \)): the susceptible MSM has a recruitment rate of \( \rho A \), and is increase by the flux rate \( \Gamma_1 \) (rate at which susceptible IDU quit drug use), population diminishes by the force of infection through sex and mortality at a rate \( \lambda_h \) and \( \mu \) respectively.

\[ dS_h/dt = \rho A - \lambda_h S_h - \mu S_h + \Gamma_1 S_d \]

Non-counselled infected IDU (\( I_d \)): The population is sourced from the force of infection through drug acting on the susceptible injecting drug users (IDU) at a rate \( \lambda_d \). Some of these individuals are detected (via screening) and counselled at a rate \( \gamma \) and progress to AIDS class at a rate \( \sigma_1 \). The population is further diminished by natural mortality \( \mu \).

\[ dI_d/dt = \lambda_d S_d - \mu I_d - \sigma_1 I_d - \gamma I_d \]

Counselled infected IDU: The detected IDU who receives counselling at a rate \( \gamma \), progresses to the AIDS class at a rate \( \sigma_1 \). The population is diminished by infected IDU who quit intake of drugs but still retain their infection. Further declination in the population occurs as a result of natural mortality a rate \( \mu \).

\[ dI_{d1}/dt = \gamma I_d - \mu I_{d1} - \sigma_1 I_{d1} - \Gamma_2 I_d \]

Infected IDU who quit the intake of drugs (\( I_{d2} \)): The infected IDU individuals who quit the intake of drugs at a rate \( \Gamma_2 \) due to counselling are recruited into this class. The population is diminished by natural death at a mortality rate of \( \mu \). The \( I_{d2} \) class is further decreased as the individuals progresses to AIDS at a rate \( \sigma_2 \).

\[ dI_{d2}/dt = \Gamma_2 I_d - \mu I_{d2} - \sigma_2 I_{d2} \]

Non-counselled infected IDU (\( I_{d3} \)): The population is sourced from the force of infection through drug (\( \lambda_d \)) acting on the susceptible injecting drug users (IDU). Some of these individuals are detected (via screening) and counselled at a rate \( \gamma \) and progress to AIDS class at a rate \( \sigma_1 \). The population is further diminished by natural mortality (\( \mu \)).

\[ dI_{d3}/dt = \lambda_d S_d - \mu I_d - \sigma_1 I_d - \gamma I_d \]

Sexually active intravenous drug users (\( I_{d4} \)): The individual who quit the intake of drugs from the Susceptible IDU acquired the infection through sex at a rate \( \theta_1 \). Individual received counselled at rate \( \gamma \), and progress to the AIDS class at a rate \( \sigma_5 \). The population is further diminished by natural death.

\[ dI_{d4}/dt = \theta_1 \lambda_d S_d - \mu I_{d4} - \gamma I_{d4} - \sigma_1 I_{d4} \]

Sexually active received counselled (\( I_{d5} \)): The active sexual intravenous drug users received recruitment from the \( I_{d4} \) individuals who received counselled at the rate \( \gamma \). The population is diminished by natural death and AIDS progressive rate \( \mu \) and \( \sigma_3 \) respectively.

\[ dI_{d5}/dt = \gamma I_{d4} - \mu I_{d5} - \sigma_3 I_{d5} \]

Infected IDU with AIDS symptoms (\( A_d \)): The population of IDU AIDS class is the class of \( I_d \) who progresses at a rate \( \sigma_1 \) to the infections class. The \( A_d \) received treatment at a rate \( \tau_d \) and suffer \( \sigma_1 \) additional disease induced mortality at rate \( \delta \).
\[ dA_d/dt = \sigma_1 I_d - \mu A_d - \delta A_d - \tau_d A_d \]

Counselling infected IDU with AIDS symptoms (\( A_{cd} \)): The population of \( A_{cd} \) is the class of \( I_{cd} \) who progress to \( A_{cd} \) at a rate \( \sigma_1 \). The \( A_{cd} \) received treatment at a rate \( \tau_{cd} \) and suffer additional disease induced mortality at a rate \( \delta \).

\[ dA_{cd}/dt = \sigma_1 I_{cd} - \mu A_{cd} - \delta A_{cd} - \tau_{cd} A_{cd} \]

Intravenous drug users with AIDS symptom receiving ART: Both \( A_d \) and \( A_{cd} \) received treatment at a rate \( \tau_d \) and \( \tau_{cd} \) respectively. The treatment AIDS class suffer additional diseases induce mortality at a rate \( \delta \).

\[ dT_d/dt = \tau_d A_d + \tau_{cd} A_{cd} - \mu T_d - \phi \delta T_d \]

Intravenous drug users who quit the intake of drugs with AIDS symptom (\( A_{qd} \)): The \( A_{qd} \) class is generated from the \( I_{qd} \) class at a rate \( \sigma_1 \). The \( A_{qd} \) received treatment at a rate \( \tau_{qd} \) and suffer additional disease induced mortality at a rate \( \phi \delta \).

\[ dA_{qd}/dt = \sigma_1 I_{qd} - \mu A_{qd} - \delta A_{qd} - \tau_{qd} A_{qd} \]

Intravenous drug users who quit the intake of drugs with AIDS symptom receiving ART (\( T_{qd} \)): The population received treatment at a rate \( \tau_{qd} \). The treatment AIDS class suffer additional disease induce mortality at a rate \( \phi \delta \).

\[ dT_{qd}/dt = \tau_{qd} A_{qd} - \mu T_{qd} - \phi \delta T_{qd} \]

Sexually active intravenous drug users with AIDS symptoms (\( A_{ds} \)): The \( A_{ds} \) class is generated from the \( A_{ds} \) class at a rate \( \sigma_2 \). The \( A_{ds} \) received induced mortality at a rate \( \delta \) with natural death rate \( \mu \).

\[ dA_{ds}/dt = \sigma_2 I_{ds} - \mu A_{ds} - \delta A_{ds} - \tau_{ds} A_{ds} \]

Sexually active intravenous drug users with AIDS symptoms who received Counselling (\( A_{cds} \)): The \( A_{cds} \) class is sourced from the \( I_{cds} \) class at a rate \( \sigma_2 \). The \( A_{cds} \) received induced treatment at a rate \( \tau_{cds} \) and suffer additional disease induced mortality at a rate \( \delta \), with natural death.

\[ dA_{cds}/dt = \sigma_2 I_{cds} - \mu A_{cds} - \delta A_{cds} - \tau_{cds} A_{cds} \]

Sexually active intravenous drug users with AIDS symptoms receiving ART (\( T_{cds} \)): Both \( A_{ds} \) and \( A_{cds} \) received treatment at a rate \( \tau_{ds} \) and \( \tau_{cds} \) respectively. The treatment AIDS class suffer additional diseases induced mortality at a rate \( \phi \delta \) and suffer natural death \( \mu \).

\[ dT_{cds}/dt = \tau_{ds} A_{ds} + \tau_{cds} A_{cds} - \mu T_{cds} - \phi \delta T_{cds} \]

New infected MSM unaware of their HIV status (\( I_s \)): The \( I_s \) population is generated when the force of infection through sex is initiated into the susceptible MSM. \( I_h \) individual receives counselling at a rate \( \tau \) and progress to \( A_h \) class at a rate \( \sigma_4 \). The \( I_h \) class is further diminished by natural death.

\[ dI_h/dt = \lambda_h S_h - \mu I_h - \sigma_4 I_h - \tau I_h \]

Counselling infected MSM (\( I_{ch} \)): The counselled \( I_h \) progress to \( I_{ch} \) at a rate of \( \tau \). The population suffers natural death (\( \mu \)).

\[ dI_{ch}/dt = \tau I_h - \mu I_{ch} - \sigma_4 I_{ch} \]

AIDS class of MSM who refuse counselling (\( A_h \)): The \( A_h \) class is sourced from the \( I_h \) class at a symptomatic rate \( \sigma_4 \). Members of this class received treatment at a rate \( \tau_h \). The population of \( A_h \) is further decreased by natural death (\( \mu \)) and induced death rate \( \delta \).

\[ dA_h/dt = \sigma_4 I_h - \mu A_h - \delta A_h - \tau_h A_h \]

Counselling AIDS class of MSM (\( A_{ch} \)): The \( I_{ch} \) class is recruited at a rate \( \sigma_4 \) to the \( A_{ch} \) which received treatment at a rate \( \tau_{ch} \). The population further suffers natural death and disease induced death at rate \( \mu \) and respectively \( \delta \).

\[ dA_{ch}/dt = \sigma_4 I_{ch} - \mu A_{ch} - \delta A_{ch} - \tau_{ch} A_{ch} \]

Treatment received by \( A_h \) and \( A_{ch} \) (\( T_h \)): Both \( A_h \) and \( A_{ch} \) progress to the treatment class at a rate \( \tau_h \) and \( \tau_{ch} \). The population is diminished by natural death and induced death at a rate \( \mu \) and \( \phi \delta \) respectively.

\[ dT_h/dt = \tau_h A_h + \tau_{ch} A_{ch} - \mu T_h - \phi \delta T_h \]

2.0.2. Derivation of model

Combining all the assumptions and definitions, the mathematical model for assessing the impact of public health education, condom use and counselling on the transmission dynamics of HIV/AIDS is given by the following system of differential equations.

\[ dS_d/dt = (1-p)A - (\lambda_d + \theta_1 \lambda_2)S_d - \mu S_d - \Gamma_1 S_d \]

\[ dS_{ch}/dt = pA - \lambda_2 S_{ch} - \mu S_{ch} + \Gamma_1 S_d \]

\[ dT_d/dt = \tau_d A_d + \tau_{cd} A_{cd} - \mu T_d - \phi \delta T_d \]

\[ dA_{cd}/dt = \sigma_1 I_{cd} - \mu A_{cd} - \delta A_{cd} - \tau_{cd} A_{cd} \]

\[ dA_d/dt = \sigma_1 I_d - \mu A_d - \delta A_d - \tau_d A_d \]
Table 1. Description of parameters.

| Symbols       | Description                                      | Reference |
|---------------|--------------------------------------------------|-----------|
| μ             | Natural death                                    | [8]       |
| β_d           | Effective contact rate of MSM                    | [8]       |
| β_t           | Effective contact rate of IDU                    | New Parameter |
| F_1           | Quitting rate of susceptible MSM                 | New Parameter |
| F_2           | Quitting rate of infectious IDU                  | New Parameter |
| r_a           | Treatment rate of A_h                           | [8]       |
| r_A           | Treatment rate of A_h                           | [8]       |
| r_d           | Treatment rate of A_d                           | New Parameter |
| r_e           | Treatment rate of A_e                           | New Parameter |
| r_A_e         | Treatment rate of A_A_e                        | New Parameter |
| r_A_e_c        | Treatment rate of A_e_c                         | New Parameter |
| g_c, g_e, g_d, g_D, g_B | Modification parameters associated with T_c | New Parameter |
| θ              | Modification parameters associated with I_A     | [8]       |
| ε              | Rate of counselled infected individuals          | [8]       |
| α_A, α_A_e, α_A_e_c, α_A_e_d | Relatively risk of infectiousness of non-counselled | New Parameter |
| α_e, α_e_c    | Relatively risk of infectiousness of counselled individuals | New Parameter |
| δ              | Disease induced death rate of AIDS individuals   | [8]       |
| k              | Condom compliance rate                           | [8]       |
| k_e           | Efficacy of condom                                | [8]       |
| p              | Fractional recruitment rate                      | New Parameter |
| σ_e           | Symptomatic parameter of IDU                     | [8]       |
| σ_d           | Symptomatic parameter of counselled MSM          | New Parameter |
| σ_A           | Symptomatic parameter of counselled IDUSA        | New Parameter |
| σ_e          | Symptomatic parameter of counselled IDUSA        | New Parameter |
| q_t           | Preventability threshold                         | New Parameter |

\[
\begin{align*}
\frac{dI_{id}}{dt} &= \tau I_d - \mu I_{id} - \sigma I_{id} - \Gamma_1 I_d \\
\frac{dI_d}{dt} &= \delta S_d - \mu I_d - \sigma_1 I_d - \tau_d I_d \\
\frac{dA_{gd}}{dt} &= \sigma_1 I_d - \mu A_{gd} - \delta A_{gd} - \tau_{gd} A_{gd} \\
\frac{dI_{gd}}{dt} &= \Gamma_2 I_d - \mu I_{gd} - \sigma_2 I_{gd} \\
\frac{dT_{gd}}{dt} &= \tau_{gd} A_{gd} - \mu T_{gd} - \phi_0 T_{gd} \\
\frac{dA_{cds}}{dt} &= \sigma_2 I_{cds} - \mu A_{cds} - \delta A_{cds} - \tau_{cds} A_{cds} \\
\frac{dT_{ds}}{dt} &= \tau_{cds} A_{ds} + \tau_{cds} A_{cds} - \mu T_{ds} - \phi_0 T_{ds} \\
\frac{dA_{ds}}{dt} &= \sigma_2 I_{ds} - \mu I_{ds} - \delta A_{ds} - \tau_{ds} A_{ds} \\
\frac{dI_{cds}}{dt} &= \tau I_{cds} - \mu I_{cds} - \sigma_3 I_{cds} \\
\frac{dI_d}{dt} &= \theta_1 S_d - \mu I_d - \tau I_d - \sigma_1 I_d \\
\frac{dI_h}{dt} &= \lambda S_h - \mu I_h - \sigma_4 I_h - \tau I_h \\
\frac{dI_{ch}}{dt} &= \tau I_{ch} - \mu I_{ch} - \sigma_4 I_{ch} \\
\frac{dA_{ch}}{dt} &= \sigma_2 I_{ch} - \mu A_{ch} - \delta A_{ch} + \tau_A A_{ch} \\
\frac{dA_{ch}}{dt} &= \sigma_4 I_{ch} - \mu A_{ch} - \delta A_{ch} - \tau_{ch} A_{ch} \\
\frac{dT_{ch}}{dt} &= \tau_{ch} A_{ch} - \mu T_{ch} - \phi \delta. 
\end{align*}
\]

Description of state variables and parameters used in the model (3) are defined in Tables 1 and 2.

2.0.3. Assumption of the model formulations

- Demographic parameter such as mortality rate is constant in all classes.
- Disease induced death is peculiar to symptomatic population (AIDS class).
- Fraction of IDU population quit the intake of drug after counselling.
- Fraction of MSM population are addicted to drug (IDUSA).
- The population is open.

2.0.4. Analysis of submodel (transmission through sex only)

In this section detailed analysis of (3) with transmission through sex only, which is the class of MSM and IDUSA (sexually drug users) is discussed. Thus system (3) will be reduced to:

\[
\begin{align*}
\frac{dT_h}{dt} &= \tau_h A_h + \tau_{ch} A_{ch} - \mu T_h - \phi \delta. \\
\frac{dA_{ch}}{dt} &= \sigma_4 I_{ch} - \mu A_{ch} - \delta A_{ch} - \tau_{ch} A_{ch} \\
\frac{dA_h}{dt} &= \sigma_4 I_{ch} - \mu A_{ch} - \delta A_{ch} + \tau_A A_{ch} \\
\frac{dA_h}{dt} &= \sigma_2 I_{ch} - \mu A_{ch} - \delta A_{ch} + \tau_A A_{ch} \\
\frac{dT_{ch}}{dt} &= \tau_{ch} A_{ch} - \mu T_{ch} - \phi \delta. 
\end{align*}
\]
Table 2. Description of state variables.

| Symbols | Description | Reference |
|---------|-------------|-----------|
| $S_h$  | Susceptible MSM individuals | [6]       |
| $S_d$  | Susceptible IDU individuals  | New Variable |
| $I_h$  | Infected MSM individuals who are unaware of their status | [8]       |
| $I_d$  | Infected MSM individuals who received counselled | [8]       |
| $A_h$  | Symptomatic MSM individuals who refused counselling | [8]       |
| $A_d$  | Symptomatic counselled MSM individuals | [8]       |
| $T_h$  | MSM individuals who received treatment | [8]       |
| $I_{hu}$ | Sexually active drug individuals who are unaware of their status | New Variable |
| $I_{du}$ | Sexually active drug individuals who received counselled | New Variable |
| $A_{hu}$ | Symptomatic sexually active drug individuals | New Variable |
| $A_{du}$ | Symptomatic sexually active drug individuals who received counselled | New Variable |
| $T_{du}$ | Sexually active drug individuals who received treatment | New Variable |
| $I_{du}$ | Infected drug users who quit the intake of drug | New Variable |
| $A_{du}$ | Symptomatic infected drug users who quit the intake of drug | New Variable |
| $T_{du}$ | Infected drug users who received treatment | New Variable |
| $I_{du}$ | Infected drug individuals who are unaware of their status | New Variable |
| $I_{du}$ | Infected drug individuals who received counselled | New Variable |
| $A_{du}$ | Symptomatic infected drug users who received counselled | New Variable |
| $A_{du}$ | Symptomatic infected drug users who refuse counselled | New Variable |
| $A_{du}$ | Symptomatic infected drug users who received counselled | New Variable |
| $T_{du}$ | Infected drug users who received treatment | New Variable |

\[
\frac{dI_h}{dt} = \lambda_i S_h - \mu I_h - \sigma_d I_h - r I_h
\]

\[
\frac{dI_d}{dt} = \tau I_h - \mu I_d - \sigma_h I_d
\]

\[
\frac{dI_{du}}{dt} = \theta_1 \lambda_i S_d - \mu I_{du} - r I_{du} - \sigma_i I_{du}
\]

\[
\frac{dI_{dus}}{dt} = \tau I_{du} - \mu I_{dus} - \sigma_i I_{dus}
\]

\[
\frac{dA_{du}}{dt} = \sigma_2 I_{du} - \mu A_{du} - \delta A_{du} - \tau A_{du} A_{dus}
\]

\[
\frac{dT_{du}}{dt} = \tau A_{du} A_{dus} + \tau A_{du} A_{dus} - \mu T_{du} - \psi \delta T_{du}
\]

\[
\frac{dS_{du}}{dt} = pA - \lambda_i S_d - \mu S_d + \Gamma S_d
\]

\[
\frac{dS_{dus}}{dt} = (1 - p)A - (\lambda_d + \theta_1 \lambda_i) S_d - \mu S_d - \Gamma S_d
\]

where

\[
\lambda_i = \beta (1 - \epsilon) \left[ I_h + I_{du} + \alpha_h A_h + \alpha_d A_d + \theta_1 (I_{du} + \alpha_d A_d) + \Gamma S_d + \theta_2 T_h + \theta_3 T_{du} \right] / N
\]

2.0.5. Basic properties model (3)

For the model (4) to be well posed it must satisfy the Lasalle’s invariance principle which state that the solutions of the model (4) with positive initial data will remain positive for all $t \geq 0$. This is achieved below.

**Lemma 1. Consider the region**

\[
D_1 = \{(S_h, S_d, I_{du}, I_h, I_{dus}, I_{dus}, I_{du}, I_{dus}, T_d, T_h, R_h) : S_h + S_d + I_{du} + I_h + I_{dus} + I_{du} A_h + A_d + T_{du} + T_h \leq \frac{\Lambda}{\mu} \}
\]

*It can be shown that the region $D_1$ is positively invariant and an attractor of all positive solutions of the model.*

**Proof.** Adding the equations in the model (4) gives

\[
\frac{dN}{dt} = \Lambda - \mu N.
\]

Hence, whenever

\[
N > \frac{\Lambda}{\mu}, \text{ then } \frac{dN}{dt} < 0.
\]

Thus, it follows from the right hand side of the inequality (6) that $\frac{dN}{dt}$ is bounded by $\Lambda - \mu N$, a standard comparison theorem can be used to show that

\[
N(t) \leq N(0)e^{-\mu t} + \frac{\Lambda}{\mu} (1 - e^{-\mu t}).
\]

If $N(0) \leq \frac{\Lambda}{\mu}$, then $N(t) \leq \frac{\Lambda}{\mu}$. Thus, $D_1$ is a positively-invariant set under the flow described by model (4) so that no solution path leaves through any boundary of $D_1$. Hence it is sufficient to consider the dynamics of the model in $D_1$. In this region the model can be considered as been epidemiologically and mathematically well-posed. □
2.0.6. Asymptotic stability of disease free equilibrium (DFE)

Disease free equilibrium (DFE): The model (3) has a disease free equilibrium given by

$$
\epsilon_0 = \left[ S_h, S_d, I_{a_d}, I_h, I_{a_h}, A_{a_h}, A_h, A_{a_h}, A_{a_d}, A_{d_a}, T_{d_a}, T_h \right] = \left[ \frac{\Lambda}{\mu} \left( \frac{(1-p)F_1}{(\mu + I_1)} + P \right), \frac{(1-p)\Lambda}{(\mu + I_1)} \right] \times 0, 0, 0, 0, 0, 0, 0, 0
$$

The linear stability of $\epsilon_0$ can be established using the next generation operation method on system (12). The matrix $F$ (for the new infection terms) and $V$ (of the transition terms) are given respectively

$$
F = \begin{pmatrix}
\beta & \beta \theta_c & \beta & \beta \theta_a & \beta \theta d & \beta \theta s & \beta \theta h \\
0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 \\
\beta \theta_1 & \beta \theta_1 & \beta \theta_1 & \beta \theta_1 & \beta \theta_1 & \beta \theta_1 & \beta \theta_1 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0
\end{pmatrix}
$$

$$
V = \begin{pmatrix}
k_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0
\end{pmatrix}
$$

$$
K_1 = \mu + \sigma_1 + \tau, K_2 = \mu + \sigma_2, K_3 = \mu + \sigma_3 + \tau, K_4 = \mu + \sigma_4, K_5 = \mu + \delta + \tau_0, K_6 = \mu + \tau_0, K_7 = \mu + \tau_0, K_8 = \mu + \delta + \tau_0, K_9 = \mu + \delta + \tau_0
$$

It follows that the basic reproduction number for the model system denoted by $R_0$ is given by $R_0 = \rho(FV^{-1}) = \max \{ R_h, R_{d_a} \}$. The partial reproductive number $R_h$ defines the number of secondary infection due to purely MSM transmission and $R_{d_a}$ defines the number of secondary infection due to MSM individuals who are addicted to drug. Where:

$$
R_h = \frac{\beta(1 - \epsilon a)(1 + \theta)(k_5 k_6 k_7 k_8 + \tau \theta_1) + k_5 a_N \theta_1 (k_2 k_6 + \tau \theta_3)}{k_1 k_2 k_3 k_4 k_9}
$$

$$
R_{d_a} = \frac{\beta(1 - \epsilon a)(1 + \theta)(k_5 k_6 k_7 k_8 + \tau \theta_1) + k_5 a_N \theta_1 (k_2 k_6 + \tau \theta_3)}{k_1 k_2 k_3 k_4 k_9}
$$

Lemma 2. The disease free equilibrium, $\epsilon_0$, of the model (3) is locally asymptotically stable (LAS) if $R_h < 1$, and unstable if $R_h > 1$. The threshold quantity, $R_0(=\rho(FV^{-1})$ is the reproduction number of the disease. It represents the average number of secondary HIV cases generated by an infected MSM and IDUSA individual if introduced into a susceptible population where the aforementioned control strategies (condom use, counselling and treatment) are used. The epidemiological implication of Lemma 2 is that perturbation from MSM and IDUSA infected individual in the susceptible population will not generate large outbreak of infection, since it is sustained in the DFE region ($\epsilon_0$) by the control parameters, the disease dies out with time. Thus HIV can be effectively controlled or eliminated from the population when the reproduction number is less than unity i.e. $R_h < 1$.

Lemma 3: HIV/AIDS can be eradicated from the population if the preventability $q = \epsilon a$ exceeds the threshold value $q_c$.

$$
R_h(\epsilon, a) = (1 - \epsilon a) R_0
$$

$$
R_0(\epsilon, a) = R_0 - \epsilon \kappa R_0
$$

$$
\epsilon = 1/s(1/R_0)
$$

Where $\epsilon$ and $\kappa$ denote the male condom efficacy and compliance, respectively. We define the product $q = \epsilon a$ as the condom induced preventability of HIV/AIDS transmission per year. Thus, the per exposure risk of the infection say $\delta$, becomes $\delta = \beta(1 - \epsilon \kappa)$.

2.0.7. Global asymptotic stability of DFE

Theorem 3. Consider the Lyapunov function

$$
F = Q_I I_h + Q_2 I_{a_h} + Q_3 A_h + Q_4 A_{a_h} + T_h + Q_5 I_{d_a} + Q_6 A_{d_a} + Q_7 A_{d_a} + Q_8 A_{d_a} + T_{d_a}
$$

The disease free equilibrium of the model (3) with $(\varphi = 0)$ is globally asymptotically stable (GAS) in $D_f$ when ever $R_h = \max \{ R_h, R_{d_a} \} \leq 1$.

Proof. See Appendix A. □
2.0.8. Existence and stability of endemic equilibrium point

Firstly, consider the case of model (4) without drug users who are sexually active (IDUSA). Thorough algebraic simplification gives

\[ S^*_h = \frac{P\lambda}{(\lambda_s + \mu)} \]
\[ I^*_h = \frac{\gamma_s P}{K_1}(\lambda_s + \mu) \]
\[ I^*_ch = \frac{\gamma_s P}{K_1}\rho/K_1 K_2(\lambda_s + \mu) \]
\[ A^*_h = \frac{\gamma_s P}{K_1}(\lambda_s + \mu) \]
\[ A^*_ch = \frac{\gamma_s P}{K_1}\rho/K_1 K_2(\lambda_s + \mu) \]
\[ T^*_h = (\gamma_s^* P^\lambda K_1)/K_1 K_2 K_3(\lambda_s + \mu) + (\gamma_s^* \lambda_s^* P^\lambda)/K_1 K_2 K_3(\lambda_s + \mu) \]

The force of infection can be expressed, at steady state as:

\[ (1 + \theta)\lambda^*_h = \beta(1 - \epsilon_k)\left[I_h + I_{ch} + a_N A_h + a_2 N A_h + \theta_s(I_{ch} + a_2 A_{ch}) + \theta_2(I_{ch} + a_2 A_{ch}) + \theta_3 T_h + \theta_{2a} T_{ch}\right]/N \]  
\[ (7) \]

Where

\[ N^* = S^*_h + I^*_h + I^*_ch + A^*_h + A^*_ch + T^*_h \]

Substitute \( N^* \) expression into equation (7)

\[ \dot{x}^*_h = \beta(1 - \epsilon_k)\left[I_h + I_{ch} + a_N A_h + a_2 N A_h + \theta_s(I_{ch} + a_2 A_{ch}) + \theta_2(I_{ch} + a_2 A_{ch}) + \theta_3 T_h + \theta_{2a} T_{ch}\right]/N \]

Further simplification gives

\[ 1 = \frac{R_0\beta(1 - \epsilon_k)/K_1 K_2 K_3 K_4 + a_N A_h + a_2 N A_h + \theta_s(I_{ch} + a_2 A_{ch}) + \theta_2(I_{ch} + a_2 A_{ch}) + \theta_3 T_h + \theta_{2a} T_{ch}}{\beta(K_1 K_2 K_3 K_4 + a_N A_h + a_2 N A_h + \theta_s(I_{ch} + a_2 A_{ch}) + \theta_2(I_{ch} + a_2 A_{ch}) + \theta_3 T_h + \theta_{2a} T_{ch}} \]

With much rigorous algebraic exercise, we have:

\[ x + \lambda^*_h = 0 \]  
\[ (8) \]

Where

\[ z = (K_2 K_3 K_4 K_5 + a_2 K_2 K_4 K_5 + \epsilon K_2 K_4 K_5 + a_2 K_3 K_4 K_5 + a_2 K_4 K_3 K_5) \]
\[ B = K_1 K_2 K_3 K_4 \]
\[ x = B/z(1 - R_0) \]

It is clear that when \( x < 0 \) and the threshold parameter is greater than one, model (4) without IDUSA group has a unique positive solution given by \( \lambda^*_h = x \) whenever \( R_0 > 1 \) (equation (8)). On the other hand, when \( R_0 < 1, x > 0 \). In this case, the force of infection at steady state is negative. Since the endemic equation (8) is of degree one, it implies that model (4) with IDUSA group will undergo forward bifurcation. Hence, the epidemiological requirement \( R_0 < 1 \), is a necessary and sufficient condition for the elimination HIV/AIDS disease burden in Nigeria.

2.0.9. Existence and stability of endemic equilibrium MSM with special case IDUSA

In this section, the possibility where at least one of the infected component of the model (4) is non-zero together with the uniqueness of the probability of transmission of the disease (force of infection) shall be explored. These claims are established as follows:

\[ S^*_h = (1 - P)\lambda/(\theta_1 x^*_h + \mu + \Gamma_1) \]
\[ I^*_h = \theta_1(1 - P)\lambda/K_1(x^*_h + \mu + \Gamma_1) \]
\[ I^*_ch = \theta_1(1 - P)\lambda/K_1(x^*_h + \mu + \Gamma_1) \]
\[ A^*_h = \theta_1(1 - P)\lambda/K_1(x^*_h + \mu + \Gamma_1) \]
\[ A^*_ch = \theta_1(1 - P)\lambda/K_1(x^*_h + \mu + \Gamma_1) \]
\[ T^*_h = \theta_1(1 - P)\lambda/K_1(x^*_h + \mu + \Gamma_1) \]

Next we show the uniqueness of the force of infection by defining the basic reproduction number of the model (3) as follows

\[ R_0 = (S_h + S_d)/N \]  
\[ (9) \]

With much rigorous exercise, we obtained the uniqueness equation of the force of infection in model (3) with IDUSA special case as follows

\[ Q_1 x^*_h + Q_2 x^*_h + Q_3 = 0 \]
Where
\[
Q_2 = \theta_1[K_1K_2K_3K_4K_5K_6K_9(1 - R_c) - \rho\mu_5K_4K_5K_9K_{10} + \rho\mu_5K_3K_9K_{10}]
\]
\[
Q_1 = [(1 + \theta_2) + \Gamma_1](1 - R_c) + (\mu + \Gamma_1)\rho\mu_5K_4K_5K_9K_{10} + \theta_2\rho\mu_5K_3K_9K_{10}
\]
\[
Q_0 = \mu(\mu + \Gamma_1)(1 - R_c)
\]
\[
y = K_4K_5K_9K_{10} + \sigma_6K_5K_9K_{10} + \sigma_1\tau_5K_5K_9 + \sigma_3\tau_5K_5K_9
\]
\[
x = K_4K_5K_9K_{10} + \sigma_6K_5K_9K_{10} + \tau_5K_5K_9K_{10} + \sigma_3\tau_6K_5K_9 + \tau_6\sigma_2K_5K_9
\]

By the Descartes rule of signs, there are few cases to be considered (depending on the signs of \(Q_0, Q_1\) and \(Q_2\)) to study the number of positive roots of \(f(\lambda + a) = 0\). It is noticed that the sub-model (10) has:

i) a unique endemic equilibrium if \(Q_0 < 0\), when \(R_c > 1\);
ii) a unique endemic equilibrium if \(Q_1 < 0\), and \(Q_2 = 0\) or \(Q_1^2 - 4Q_2Q_0 = 0\) and \(R_c = 1\);
iii) two endemic equilibria if \(Q_2 > 0\), \(Q_1 < 0\) and \(Q_1^2 - 4Q_2Q_0 > 0\) and \(R_c < 1\);
iv) no endemic equilibrium otherwise.

Condition (iii) implies backward bifurcation in the sub-model (3) when \(R_c < 1\). Hence model (3) with heterogeneous group (IDUSA) is responsible for backward bifurcation.

**Lemma 4.** The model (4) has a unique endemic (positive) equilibrium given by \(E_{th}\), whenever \(R_c > 1\).

**2.0.10. Local asymptotic stability of endemic equilibrium point (EEP)**

**Theorem 5.** The unique endemic equilibrium of the model (4) when induce death parameters are null is LAS if \(\overline{R}_c > 1\). Where

\(\overline{R}_c = \text{Reproduction number of linearized system.}\)

\(R_c = \text{Reproduction number of nonlinear system.}\)

**Proof.** See Appendix. □

**2.0.11. Global stability of endemic equilibrium points (MSM only)**

A thorough analysis for the global stability of the endemic equilibrium point shall be meant. This is attainable on the assumption that there is little or no effect of the administered control measures on infectious individuals. Thus, system (4) will be in its explosive state i.e. each modification parameter takes a value of one. The epidemiological consequence is that infectious individuals with low response to control measures do not modify their sexual behaviour. Using \(N = \Lambda/\mu\) and noting that \(a_N = a_{2N} = a_e = a_{2e} = \theta_1 = \theta_2 = 1\), it follows that the force of infection \(\dot{\lambda}_S\) defined in (3), reduces to

\[
\dot{\lambda} = \beta(1 - \kappa t)(1 + \theta)[I_b + I_{ch} + A_b + A_{ch} + T_b] \Lambda/\mu
\]

The associated reproduction number for the model with (3), denoted by \(R_n^1\) is given by

\[
R_n^1 = \frac{K_2K_3K_4K_9 + K_2K_3K_9\kappa + K_3K_9\kappa + \sigma_1\tau_6K_2K_3K_4 + \sigma_1\tau_6K_2K_3K_4}{K_1K_2K_3K_4K_9}
\]

Also we consider the coefficient of infectiousness \(Q_i\) when control measures administered but the disease still persists. Thus, \(Q_i\forall i = 1, 2, 3, 4\).

\[
Q_1 = \frac{R_cK_3}{\beta}, Q_2 = \frac{K_3(K_3 + \sigma_1) + \tau_6\kappa\sigma_1}{K_1K_2}, Q_3 = \frac{(K_3 + \tau_6)}{K_5}, Q_4 = \frac{K_3 + \tau_6}{K_6}
\]

The global asymptotic stability proof is achieved by first of all establishing the following result.

**Lemma 6.** The region \(D^* = \{I_b, I_{ch}, A_b, T_b | S_b(t) \leq S_{th}\}\) is positively invariant and attracting for the model (4).

**Proof.** Assuming \(N(0) < \Lambda/\mu\), it follows that \(S_b = p\Lambda/\mu\) at DFE \((e_0)\). It follows that the equation of \(dS_b/dt\) in model (10) i.e. \(dS_b/dt = p\Lambda - \dot{\lambda}S_b - \muS_b + \Gamma_1S_b \leq p\Lambda - \dot{\lambda}S_b - \muS_b \Rightarrow S_b(t) \leq S_{th}\). Hence, \(S_b(t) \leq S_{th}\)\(\Rightarrow\) \(S_b(t) \leq S_{th}\) asymptotically, or there is some finite time after which \(S_b(t) \leq S_{th}\) Thus, the region \(D^* \subset D_b\) is positively invariant and attracting. □

**Theorem 7.** Consider the model (4) with \(\dot{\lambda}_S\). The associated unique endemic equilibrium of the model is GAS in \(D^*_b\)/Do if \(R_n^1 > 1\) and \(S_b < S_{th}\).

**Proof.** See Appendix. □

**2.0.12. Backward bifurcation analysis of model (3)**

The phenomenon of backward bifurcation which has been observed in numerous disease transmission dynamics is typically characterized by the coexistence of a stable DFE and a stable endemic equilibrium when associated reproduction number of the model is less than unity. The epidemiological implication of the backward bifurcation phenomenon is that the classical epidemiological equipment of having the reproduction number \(R_0\) to be less than unity, while necessary, is no longer sufficient for the effective control of the disease in the population in other words, the backward bifurcation property of the model (4) makes effective control of HIV in the population difficult. To investigate the possibility of backward bifurcation at \(R = 1\), we use the centre manifold theory, as presented by Castillo-Chavez and Song [3].
2.0.13. Centre manifold theory

Consider the following general system of ordinary differential equations with the parameter \( \varphi \), such that \( x(t) = f(x, \varphi, f : \mathbb{R} \times \mathbb{R} \) and \( f \in C^2(\mathbb{R} \times \mathbb{R}) \) where zero is an equilibrium point of the system i.e. \( F(0, \varphi) = 0 \), assume

1. \( A = Dx F(a, \varphi) = (\partial f / \partial x(0,0)) \) is the linearity matrix of the system around the equilibrium.
2. Zero is a simple Eigen-value of \( A \) and other Eigen-value of \( A \) have negative real part.
3. Matrix \( A \) has a right Eigen-value \( W \) and left Eigen-value \( V \) each corresponding to the zero Eigen-value let \( f_k \) be the kth component of \( f \) and \( a = \sum_{i,j=1} w_i \cdot w_j \cdot f_k(0,0) / (\partial x_i \partial x_j) \) and \( b = \sum_{i,j=1} w_i \cdot d f_k / d x / d \varphi(0,0) \).

The local dynamics of the system around the equilibrium point is totally determined by the signs of \( a \) and \( b \).

1. \( a > 0, b > 0 \), when \( \varphi < 0 \) with \( q < 1, /p / < 0 \) is unstable and there exists a negative and locally asymptotically stable equilibrium.
2. \( a < 0, b < 0 \), when \( \varphi < 0 \) with \( /p / < 0 \), is unstable, and there exists a positive unstable equilibrium.
3. \( a > 0, b < 0 \); when \( \varphi < 0 \) with \( /p / < 0 \), is unstable and there exists a locally asymptotically stable negative equilibrium when \( 0 < \varphi < 1 \) is stable and now there exists a positive unstable equilibrium \( a < 0, b > 0 \); when \( \varphi \) changes from negative to positive, it changes its stability from stable to unstable. Corresponding, negative unstable equilibrium becomes positive and locally asymptotically stable. In particular, if \( a > 0 \) and \( b > 0 \) then a backward bifurcation occurs when \( \varphi = 0 \).

Rigorous exercise for the computation \( a \) and \( b \) gives (see appendix)

\[
\begin{align*}
\frac{a}{\mu + G_1} &= \mu \left( 1 - \kappa \right) \left[ 1 - 4 \mu \frac{\theta_2 \mu}{\mu + \Gamma_1} \right] \\
\frac{b}{\mu + G_1} &= \mu \left( 1 - \kappa \right) \left[ 1 - 4 \mu \frac{\theta_2 \mu}{\mu + \Gamma_1} \right] \\
\end{align*}
\]

Since \( b > 0 \), the system will undergo a backward bifurcation if \( a > 0 \). This is possible if

\[
\Gamma_1 > \frac{1 - p \mu}{\mu + G_1} \left[ 1 - 4 \mu \frac{\theta_2 \mu}{\mu + \Gamma_1} \right]
\]

From the analysis above, we can deduce that the existence of backward bifurcation in system (3), depends on the efflux parameter \( G_1 \) which represent the duality effect of the force of infection on the susceptible classes. That is, multiple class of individuals susceptible to HIV/AIDS is responsible for backward bifurcation.

2.1. Analysis of IDU model with IDUQD special case

In this section a thorough analysis on IDU with IDUQD shall be meant. The submodel is as follows:

\[
\begin{align*}
d S_d / d t &= (1 - p) \Lambda - \lambda_d S_d - \mu S_d - G_1 S_d \\
d I_d / d t &= \lambda_d S_d - \mu I_d - \sigma_2 I_d - \tau I_d \\
d I_d / d t &= \tau I_d - \mu I_d - \sigma_2 I_d - G_2 I_d \\
d I_d / d t &= \Gamma_2 I_d - \mu I_d - \sigma_4 I_d \\
d A_d / d t &= \sigma_2 I_d - \mu A_d - \delta A_d - \tau A_d A_d \\
d A_d / d t &= \sigma_2 I_d - \mu A_d - \delta A_d - \tau A_d A_d \\
d A_d / d t &= \sigma_4 I_d - \mu A_d - \delta A_d - \tau A_d A_d \\
d T_d / d t &= \varphi T_d - \mu T_d \\
d T_d / d t &= \delta T_d - \mu T_d \\
\end{align*}
\]

2.1.1. Existence and stability equilibrium

The model (18) has a disease free equilibrium given by \( D_d = (S_d, I_d, I_d, A_d, A_d, A_d, T_d, T_d, T_d) = (\frac{1 - p \Lambda}{\mu + G_1}, 0,0,0,0,0,0,0,0) \). The linear stability can be established using the next generation operation method on the system (18). The matrices \( F \) (for new infection terms) and \( V \) (of the transition terms) are given respectively by

\[
F = \begin{bmatrix}
    g_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
    -\tau & g_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
    0 & -\Gamma_2 & g_3 & 0 & 0 & 0 & 0 & 0 & 0 \\
    -\sigma_2 & 0 & g_4 & 0 & 0 & 0 & 0 & 0 & 0 \\
    0 & -\sigma_2 & 0 & g_5 & 0 & 0 & 0 & 0 & 0 \\
    0 & 0 & -\tau_d & -\tau_d & 0 & g_7 & 0 & 0 & 0 \\
    0 & 0 & 0 & 0 & 0 & -\tau_d & 0 & g_8 & 0 \\
    0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & g_9 \\
\end{bmatrix}
\]
The associated reproduction number denoted by $R_e = \rho(FV^{-1}) = \max \{R_d, R_{qd}\}$.

\[
R_e = (A_1 + A_2)/(g_1 g_2 s_8 s_8 s_8)
\]
\[
R_d = (K_1 + K_2)/(g_1 g_2 s_8 s_8 s_8)
\]
\[
R_{qd} = X_1/(g_1 s_8 s_8)
\]
\[
X_1 = s_8 (s_8 + \alpha s_N s_8) + \theta_4 d s_4 t_{qd}
\]
\[
K_1 = g_1 s_1 (g_4 + \alpha_3 \sigma_2) + g_4 \theta_1 r_{g_3} + \alpha_2 \sigma_2
\]
\[
K_2 = \theta_3 (g_8 s_5 t_2 + r_{c_d} s_4)
\]
\[
A_2 = \beta d (g_8 s_8 s_8 s_8 t_2 + r_{c_d} s_4)
\]
\[
A_1 = g_2 s_8 s_8 s_8 s_8 (g_4 + \alpha_3 s_N) + g_4 s_8 s_8 s_8 s_8 s_8 \tau (\Gamma_1 + \theta s_8) + g_4 s_8 s_8 s_8 \tau (\Gamma_2 + \theta s_8)
\]
\[
\theta_3 s_8 \alpha_2 s_2 + \sigma_2 \Gamma_2 a_4 N s_8) + g_4 s_8 s_8 s_8 t_{qd} d \sigma_4 t \Gamma_2.
\]

**Lemma 8.** The disease free equilibrium $\epsilon_f$ of the model (17) is locally stable asymptotically (LAS) if $R_e < 1$ and unstable if $R_e > 1$. The threshold quantity represent the average number of the disease. It represents the average number of secondary HIV cases generated by IDU individuals if introduce into a susceptible population, where the afore-mentioned intervention strategies (condom, counselling and treatment) are used. The epidemiological implication is that small perturbation from HIV infected IDU in the population will not generate large outbreak of infection and the disease dies out with time. Thus HIV can be efficiently controlled or eliminated from the population when the reproduction number is less than unity.

In this section we shall be considering the equilibrium where at least one of the infected IDU class of model (17) is non-zero. The following steps are considered.

\[
\lambda_d^* = \frac{\beta (I_d^{**} + I_{d}^{**} + \theta_1 I_{d}^{**} + \alpha s_N A_{d}^{**} + \alpha a_N A_{d}^{**} + \theta_3 \alpha_2 A_{d}^{**} + \theta_3 r_{d} T_{d}^{**} + \theta_{qd} T_{d}^{**})}{N^{**}}
\]

\[
S_d^{**} = \frac{(1 - p)\Lambda}{(\lambda_d^{**} + \mu + \Gamma_1)}
\]

\[
I_d^{**} = \frac{(1 - p)\Lambda}{(\lambda_d^{**} + \mu + \Gamma_1)}
\]

\[
I_{d}^{**} = \frac{(\tau - \Gamma_1)(1 - p)\Lambda}{(\lambda_d^{**} + \mu + \Gamma_1) g_1 s_1 s_2}
\]

\[
I_{d}^{**} = \frac{(\tau - \Gamma_1)(1 - p)\Lambda}{(\lambda_d^{**} + \mu + \Gamma_1) g_1 s_1 s_2}
\]

\[
A_{d}^{**} = \frac{\sigma_1 (1 - p)\Lambda}{(\lambda_d^{**} + \mu + \Gamma_1) g_1 s_1 s_2}
\]

\[
A_{d}^{**} = \frac{\sigma_1 (1 - p)\Lambda}{(\lambda_d^{**} + \mu + \Gamma_1) g_1 s_1 s_2}
\]

\[
T_{d}^{**} = \frac{\tau_1 \sigma_2 (1 - p)\Lambda}{(\lambda_d^{**} + \mu + \Gamma_1) g_1 s_1 s_2}
\]

\[
T_{d}^{**} = \frac{\tau_1 \sigma_2 (1 - p)\Lambda}{(\lambda_d^{**} + \mu + \Gamma_1) g_1 s_1 s_2}
\]

\[
\lambda_d^* + \frac{F}{x} (1 - R_d) = 0
\]

It is clear that when $x < 0$ when $R_d > 1$. Thus, the linear system (19) has a unique positive solution, given by $\lambda_d^* = x$ whenever $R_d > 1$. On the other hand, when $R_d < 1$, $x > 0$. In this case, the force of infection at steady state is negative. Hence the model has no positive equilibrium in this case. It is imperative to note that an attempt to reduce the primary transmission parameter below the value one is a necessary and sufficient condition for the elimination HIV/AIDS disease burden in Nigeria.
2.1.2. Local stability of endemic equilibrium point (EEP)

**Theorem 9.** The unique endemic equilibrium of the model (17) when induced death parameters are null is LAS if \( \hat{R}_d - R_d > 1 \). Where:
- \( \hat{R}_d \) = Reproduction number of linearized system.
- \( R_d \) = Reproduction number of unlinearized system.

**Proof.** Omitted. □

2.1.3. Backward bifurcation

Observing Castillo-Chavez principle in section 2, computation for a and b gives

\[
a = V\frac{2\beta}[\lambda (1 - (1 - p)\mu) + u_2 w_2 \frac{\beta_3}{\lambda} \theta_3 + \frac{(1 - p)\mu}{(\mu + T_1)} + w_2 \frac{\beta_3}{\lambda} \theta_3 + \frac{(1 - p)\mu}{(\mu + T_1)} + w_2 \frac{\beta_3}{\lambda} \theta_3 \frac{(1 - p)\mu}{(\mu + T_1)}] \\
b = -\frac{(1 - p)\mu}{(\mu + T_1) + (b + \beta)}
\]

System (17) in the absence of MSM and IDUSA will not undergo backward bifurcation, since the parameter b is less than zero when \( a > 0 \). The epidemiological implication is that the threshold parameter \( R_e = \{ R_d, \hat{R}_d \} \) less than one is not just a necessary but a sufficient condition for the elimination of HIV/AIDS when the control measures are strictly observed.

**Lemma 10.** The existence of backward bifurcation solely depends on the dual or multiple effect of the force of infection in a SI model.

2.1.4. Global stability of the DFE

The DFE of the model (17) with \( \varphi = 0 \) is globally asymptotically stable in \( D_d \) whenever \( R_e = \max \{ R_d, \hat{R}_d \} < 1 \).

**Proof.** Consider the Lyapunov function

\[
F = Q_1 I_d + Q_2 I_{cd} + Q_3 A_d + Q_4 A_{cd} + T_d + Q_5 I_{cd} + Q_6 A_{cd} + T_{cd}
\]

Where

\[
Q_1, Q_2, Q_3, Q_4, Q_5, Q_6, 1, 2, 3 \ldots 6
\]

is the coefficient of infectiousness expressed as follows

\[
Q_1 = \frac{R_d \beta}{\beta d_d} \\
Q_2 = \frac{\alpha_2 N \beta}{\beta d_d} \theta_d \\
Q_3 = \frac{\alpha_3 N \beta}{\beta d_d} \theta_d \\
Q_4 = \frac{\alpha_4 N \beta}{\beta d_d} \theta_d \\
Q_5 = \frac{\alpha_5 N \beta}{\beta d_d} \theta_d \\
Q_6 = \frac{\alpha_6 N \beta}{\beta d_d} \theta_d
\]

The associated Lyapunov derivative is given by (where a dot represents differentiation with respect to time (t))

\[
\dot{F} = F_1 I_d + F_2 I_{cd} + F_3 A_d + F_4 A_{cd} + F_5 T_d + F_6 I_{cd} + F_7 A_{cd} + F_8 T_{cd}
\]

Further simplification gives

\[
\dot{F} \leq \frac{g_1}{\theta_d} [R_d - 1] I_d + \frac{g_2}{\theta_d} [R_d - 1] I_{cd} + \frac{g_3}{\theta_d} [R_d - 1] A_d + \frac{g_4}{\theta_d} [R_d - 1] A_{cd} + \frac{g_5}{\theta_d} [R_d - 1] T_d.
\]

From equation (21) we can deduce that whenever \( R_d < 1, \dot{F} \leq 0 \) and all the parameters and Variables of equation (21) are non-negative, it follows that \( \dot{F} \leq 0 \) when \( G = 0 \) iff all infectious class equal zero. Hence, \( F \) is a Lyapunov function in \( D_d \). Thus, it follows from Lasalle’s invariance principle, that every solution to the equation in (21), with initial condition in \( D_d \) approaches the DFE as \( t \to \infty \). This implies that the classical epidemiological requirement of \( R_e \leq 1 \) is a necessary and sufficient condition for HIV elimination in Nigeria. In other words, if the control strategies considered in the model are implemented in such a way that they lead to \( R_e \leq 1 \), then HIV will be effectively controlled in Nigeria. □
2.1.5. Global stability of endemic equilibrium IDU only

Consider the special case of the model (18) with \(\alpha = \alpha_2 = \alpha_3 = \alpha_4 = \theta = \theta_2 = 1\), it follows that the force \(\lambda_d\), reduces to

\[
\lambda_d = R_d \left( I_d + I_{cd} + \alpha_2 A_{cd} + \alpha_3 A_d + \alpha_4 I_{cd} + \theta_2 T_d \right)
\]

The reproduction number becomes

\[
R_d^* = \frac{g_2 g_3 \sigma_1 (\gamma + \sigma_2) + g_4 (\tau - \Gamma_2) + g_5 \sigma_2 r_d + g_6 (\Gamma_2 - \Gamma_1)}{g_7 \beta_d \sigma_4 \delta_7}
\]

The global asymptotic stability proof is achieved by first of all, establishing the following result.

**Lemma 11.** The region \(D_d^* = \{ S_d, I_d, I_{cd}, A_d, A_{cd}, T_d, D : S_d < S_d^* \}\) is positively invariant and attracting for the model (18).

**Proof.** Assuming \(N(0) \leq \Lambda/\mu\), it follows that \(S_d^* = \Lambda/\mu\) at the DFE. It follows from the equation of \(dS_d/dt\) in (14), i.e.

\[
dS_d/dt = \Lambda - \lambda_d S_d - \mu S_d \leq \Lambda - \mu S_d - (S_d^* - S_d),
\]

hence

\[
S_d \leq S_d^* - [S_d^* - S_d(0)]e^{-\mu t}
\]

and it follows that \(S_d(t)\) approaches \(S_d^*\) asymptotically, or there is some finite time after which \(S_d \leq S_d^*\). Thus the region \(D_d^* \subset D_d^*\) is positively – invariant and attracting. \(\Box\)

**Theorem.** Consider the model (10) with \(\lambda_d\). The associated unique endemic equilibrium of the model is GAS in \((D_d^*)/D_0\) if \(R_d^* \geq 1\) and \(S_d \leq S_d^*\).

**Proof.** Consider the model (20) and \(R_d^* > 1\), so that the associated unique endemic equilibrium of the model exists. Further, consider the following non-linear Lyapunov function (of Goh–Volterra type):

\[
F = S_d - S_d^{**} + S_d^{**} I_d (A_d - A_d^{**}) + \frac{\beta S_d^{**} [g_7 (\gamma + \tau_d) + g_4]}{g_7 \sigma_4 \delta_7} \left( I_{cd}^{**} - I_{cd} \right)
\]

With Lyapunov derivative, we have

\[
\dot{F} = (S_d - S_d^{**} + S_d^{**} I_d (A_d - A_d^{**}) + \frac{\beta S_d^{**} [g_7 (\gamma + \tau_d) + g_4]}{g_7 \sigma_4 \delta_7} \left( I_{cd}^{**} - I_{cd} \right))
\]

Further simplification gives:

\[
3 - \frac{S_d^{**}}{S_d^{**}} - \frac{I_{cd} I_{cd}^{**}}{I_{cd}^{**}} - \frac{I_{cd}^{**} I_d}{I_{cd}^{**}} \leq 2 - \frac{I_{cd} I_d}{I_d} - \frac{I_{cd} I_d}{I_d}
\]

Since the arithmetic mean is greater than the geometric mean, the following inequalities from (15) hold

\[
1 - \frac{S_d^{**}}{S_d^{**}} \leq 0
\]

Thus \(F \leq 0\) for \(R_d^* > 1\). Hence, \(F\) is a Lyapunov function on \(R_d^* > 1\). It follows by Lasalle’s invariance principle, that every solution to the equation of the model (10), and initial condition in \((D_d^*)/D_0\) approaches the associated unique endemic equilibrium of the model as \(t \to \infty\) for \(R_d^* > 1\). \(\Box\)
3. Numerical simulation

In this section, the impact of the control strategy with high effectiveness of the control parameters (Table 3) shall be explored using the qualitative analysis approach together with simulation (MATLAB). This is to investigate the effectiveness of the control strategies on the HIV/AIDS epidemic in the population. Also, sensitivity analysis is considered to investigate the parameters (Tables 5 and 6) responsible for the up rise of the transmission dynamics of the disease. The intervention strategies are:

(1) Condom only, (2) Counselling only, (3) Treatment only, (4) Universal strategy (combine).

Before assessing the above control strategies, it is imperative to analyse the worst case scenario, where no intervention strategy is used in the country. This is achieved by setting all control strategy parameters to zero, i.e., \( \epsilon = \tau_{ch} = \tau_h = \tau_{cd} = \tau_d = \theta_h = \theta_{qds} = \theta_c = 0 \) and substituting this into the expression for the reproduction number, \( R_c \).

Consider the set

\[ R_* = \{ R_d, R_{qds}, R_h, R_{qds} \} \]

Where,

\[ R_h = \frac{\beta_h(1 - \kappa)(1 + \theta)(Q_1 + Q_2)}{K_1K_2K_3K_6K_9} \]

\[ R_d = \frac{\beta_d[A_1 + A_2]}{S_1S_2S_3S_4S_7} \]

\[ R_{qds} = \frac{\beta_d(\theta_1 + \theta_2)[S_6S_8 + S_3S_4S_5S_4 + \sigma_{qds}\sigma_d\theta_{qds}]}{S_1S_2S_3S_4S_7} \]

\[ R_{cds} = \frac{\beta_d(1 - \kappa)(1 + \theta)(A_1 + A_2)}{K_2K_3K_4K_6K_{10}} \]

\[ A_2 = \theta_d(\tau_{qds}\sigma_3K_6 + K_3\tau_{cds}\tau), Q_2 = \theta_d\sigma_2(\tau_hK_2K_6 + K_5\tau_{qds}\tau) \]

\[ A_1 = K_7K_8K_{10}\theta_2 + K_7K_9K_{10}\theta_2(K_5 + \alpha_{qds}\sigma_3), Q_1 = K_2K_6K_9K_3(\sigma_5 + \sigma_3) + \theta_2K_3K_9K_6 + \sigma_3 \]

\[ R_{ncasecontrol} = \frac{R_{dead}}{100} * R_* = 6.38\% \]

\[ R_{ncasecontrol} = \frac{R}{100} * R_c = 4.165\% \]

where

\[ R_{ncasecontrol} = R_{ncasecontrol}^{inc} + R_{ncasecontrol}^{dec}, \quad R_* = R_h + R_d \]
### Table 4. Values for threshold parameters in the presence and absence of intervention strategies.

| Reproduction number of IDU | Reproduction number of MSM | Total |
|----------------------------|----------------------------|-------|
| \( R_w^0 = 10.8409 \)     | \( R_h^0 = 8.100 \)       | 18.9405 |
| \( R_c = 7.5973 \)        | \( R_s = 7.177 \)         | 14.775 |
| Total = 18.4382           | Total = 15.277            | 33.7155 |

Computed values of reproduction number (Table 4).

---

**Fig. 1.** Plot dynamics of HIV/AIDS indicating high compliance rate of condom.

\[
R_{\text{declination}} = R_{\text{cases without}} - R_{\text{cases with}}
\]

The expected number of infected individuals in the high risk group (MSM and IDU), produce by an infected individual, with and without intervention strategy are respectively 15 and 18 individuals (Table 4). An evaluated cumulative incidence of new case gave 6.4%, with a declination of 1.4% yearly, if the controls measures are perfectly observed. That is, Nigeria will record at least eleven million, five hundred and twenty thousand cumulative new HIV cases, with a yearly reduction of two million, five hundred and twenty seven thousand infected individuals. This model prediction agreed with the estimate, given by UNAIDS.

### 3.1. Condom only strategy

Setting counselling and treatment parameters to zero, together with counselling infectious classes i.e., \( I_{cd} = I_{ch} = A_{cd} = A_{ch} = A_{dh} = \tau = \tau_{ch} = \tau_h = \tau_{cd} = \tau_d = \theta_h = 0 \) in equation (16), (17), and taking the partial derivatives with respect to condom function \( q = \epsilon k \).

\[
\frac{\partial R_v}{\partial q} = -\frac{\beta_h(1+\theta)(K_5 + a_N \sigma_1)}{K_1 K_5}
\]

Qualitatively, we can deduce that condom only strategy will confer a beneficial impact on the population, since the partial derivative of \( R_v \) decrease with respect to the condom compliance function \( q \).

The dynamics of the total number of infected MSM individuals with condom compliance rate (Fig. 1) of 0.1 (90%) and 0.2 (80%). With 80% condom efficacies, condom use reduced the total number of infected MSM to five million in a period of twenty years. Population wave occurs within 25-40 years (Fig. 1) which is then neutralized at high condom compliance rate (80%). Thus, reducing the total number of infected MSM individuals to about 7 million. Although this intervention strategy drastically reduces HIV infections within twenty years of its initiation, it is obvious that the use of condoms as a single control measure will not be a realistic option for combating HIV in Nigeria. This is due to endemicity spur by drug users who are sexually active and the unrealistic condom compliance rate. (See Fig. 2.)

### 3.2. Counselling only strategy

The counselled only strategy is obtained by taking the partial derivative of the \( R_v \) with respect to counselling parameters by setting condom and treatment parameters to zero in equation (16) and (17).

\[
\frac{\partial R_v}{\partial \tau} = \beta_h \left[ \frac{K_5 + a_N \sigma_1}{K_1 K_5} + \frac{(1+\theta)(K_6 + a_N \sigma_1)}{K_1 K_2 K_6} \right]
\]

\[
\frac{\partial R_v}{\partial \theta_h} = -\frac{\beta_h}{g_1^2 \theta} \left[ \frac{(1+\theta)(1 - G_2) + (\tau - G_2)}{g_1^2 \theta} \right]
\]
High rate of condom compliance and condom use reduces the primary transmission of the disease below its threshold value (Fig. 2).

Fig. 2. Contour plot of condom efficacy on reproduction number.

At \( \left\{ \frac{\partial R_h}{\partial \tau}, \frac{\partial R_d}{\partial \tau} = [0, 0] \right\} \), we obtained counselling threshold parameters as

\[
\theta_{tn} = \frac{(K_1 + \alpha_N \sigma_1)}{K_2 K_5 (K_6 + \alpha_c) (K_1 - \tau) K_5}
\]

\[
\theta_{td} = \left\{ \frac{g_4 + \alpha_3 N \sigma_2}{g_2 g_5 (g_1 (1 - \Gamma_2) + (\tau - \Gamma_2))} \right\}
\]

(18)

(19)

Equations (18) and (19) imply that counselling will have a positive impact on MSM and IDU in Nigeria on the condition that \( \theta_{tn}, \theta_{td} > \theta_c > 0 \), thus the relative risk of counselled infected individuals must reduce their sexual behaviour below the thresholds, for the disease burden (HIV) to be reduced or eliminated in Nigeria.

**Lemma 12.** The positive impact of counselling on HIV infected individuals in Nigeria, must satisfy the following conditions:

1. \( \theta_{tn} > \theta_c > 0 \) \( \forall K_1 > \tau \).
2. \( \theta_{td} > \theta_c > 0 \) \( \forall \tau > \Gamma_2, 1 > \Gamma_2 \).

**Proof.** Omitted. \( \square \)

**Lemma 13.** The positivity of the treatment threshold parameters for the HIV infected population in Nigeria must satisfy the following conditions for the elimination of HIV in Nigeria:

1. \( \theta_{tn} > \theta_b > 0 \) \( \forall K_1, K_5 > \tau_b \).
2. \( \theta_{td} > \theta_d > 0 \) \( \forall g_4 > \mu \).

**Proof.** Omitted. \( \square \)

**3.2.1. Condom and counselling only strategy**

For condom and counselling only intervention strategies, we set treatment parameters to zero as follows

\[
T_h = T_d = A_{dl} = r_{hl} = r_{dl} = r_d = \theta_b = \theta_h = r_p = 0,
\]

\[
\frac{\partial R_h}{\partial \tau} = \beta_h (K_1 + \alpha_N \sigma_1) + \frac{\beta_h (1 + \theta) (K_6 + \alpha_c) \theta_c}{K_5 K_6} \left[ \frac{1}{K_1 K_2 K_5} - \frac{\tau}{K_4 K_5 K_6} \right]
\]

\[
\frac{\partial R_d}{\partial \tau} = -\beta_d \left[ \frac{g_4 + \alpha_3 N \sigma_2}{g_2 g_5} \right] + \frac{\beta_d \theta_c [g_1 (1 - \Gamma_2) + (\tau - \Gamma_2)]}{g_2 g_4}
\]

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Co-administration effect of counselling and condom neutralizes the population wave and reduces the disease burden.

**Fig. 3.** Dynamics of combined strategy effect on infected population.

Combined strategy offers a plausible cost effective recipe for effectively combating and eliminating the disease (HIV/AIDS) within 110-120 years. Intravenous drug users HIV/AIDS population converges to zeros (Fig. 4).

**Fig. 4.** Dynamics of HIV/AIDS population with combined strategy effect.

At \( \left\{ \frac{\partial R_h}{\partial \tau}, \frac{\partial R_d}{\partial \tau} = (0, 0) \right\} \), we obtained counselling threshold parameters as

\[
\theta_{cw} = \frac{(K_5 + a_N \sigma_1)}{K_2 K_0 (K_6 + a_N) (K_1 - \tau) K_5} \\
\theta_{cd} = \frac{(g_4 + \theta_{3N} \sigma_2)}{g_{2851} g_0 (1 - I_2) + (\tau - I_2) g_4}
\]

The dynamics of the MSM individuals (Fig. 3) at observed high condom compliance rate 90% and condom efficacy rate 80%. (See Fig. 4.) The counselling high values parameters include:

i) \( \theta_c = 0.5 \) (i.e. 50% of infected individuals positively modify their sexual behaviour with a counselling rate of \( \tau = 0.015 \) for MSM, \( \tau = 0.5 \) for IDU).

### 3.3. Universal strategy

When high values of intervention strategies parameters are observed in \( R_h \) and \( R_d \), the disease fades out of the population in a short time (Fig. 5).
The absence of natural death causes disease burden to grow exponentially but attains stability as the value of natural death increases (Fig. 6). Also the population of the new cases tends to decrease when the value of natural death increases.

Fig. 6. Cumulative incidence of IDU individuals with variation of death parameter.

3.4. Cumulative incidence plots

3.4.1. Sensitivity analysis of the reproduction number of MSM ($R_{hm}$) and IDU ($R_{jd}$)

Sensitivity indices allow us to measure the relative change in a state variable when a parameter changes (Figs. 6 and 7). The normalized forward sensitivity index of a variable to a parameter is the ratio of the relative change in the variable to the relative change in the parameter. When the variable is a differentiable function of the parameter, the sensitivity index may be alternatively defined using partial derivative. The normalized forward sensitivity index of a variable $y$, that depends on a parameter, $q$, is defined as:

$$\Pi_{y}^{q} = \frac{\partial y}{\partial q} \times \frac{q}{y}$$

(22)

The sensitivity index of IDU (Table 5) and MSM (Table 6) are obtained using parameters in Table 3 incorporated in (22).
Variation of mortality rate has no stabilizing effect on infectious population (Fig. 7).

**Fig. 7.** Cumulative incidence of MSM HIV/AIDS population.

| No. | Parameters | Sensitivity values |
|-----|------------|--------------------|
| 1   | $\theta_1$ | 1.25186            |
| 2   | $\alpha_1$ | 0.52016            |
| 3   | $\beta_h$  | 0.4326             |
| 4   | $\alpha_c$ | 0.11379            |
| 5   | $\theta_h$ | 0.0022038          |
| 6   | $\nu$      | 0.000000431        |
| 7   | $\mu$      | 0.00034146         |
| 8   | $\tau$     | -0.37108           |
| 9   | $r$        | -0.1082            |
| 10  | $s_1$      | -0.09785           |
| 11  | $\phi$     | -0.0032019         |
| 12  | $\delta$   | -0.002059          |

**Table 5.** Sensitivity value of base line parameters of homosexuals (MSM).

| No. | Parameters | Sensitivity values |
|-----|------------|--------------------|
| 1   | $\beta_d$  | 0.16841            |
| 2   | $\alpha_3$ | 0.05261            |
| 3   | $\sigma_3$ | 0.01777            |
| 4   | $\theta_c$ | 0.016703           |
| 5   | $\theta_d$ | 0.01256            |
| 6   | $\Gamma_2$ | 0.00005643         |
| 7   | $\mu$      | -0.0001383         |
| 8   | $\tau_d$   | -0.05023           |
| 9   | $r$        | -0.1306            |
| 10  | $\phi$     | -0.24290           |
| 11  | $\delta$   | -0.70522           |

**Table 6.** Sensitivity value of baseline parameters for intravenous drug users (IDU).

### 3.4.2. Interpreting sensitive values of baseline parameters

Contact rate ($\beta_h$ and $\beta_d$): The probabilities of disease transmission from infectious IDU and MSM to their Susceptible counter are respectively $\mathcal{R}_h = 0.4326$ and $\mathcal{R}_d = 0.1684$. Since infectious individuals make contacts with the susceptible counterpart, a 20% decrease of the susceptible individuals will imply a decrease in $R_h$ and $R_d$ by 8.6% and 3.4% respectively. This is attainable by:

1. Having a good knowledge of the growth rate of the susceptible individuals to predict the total number of susceptible individuals in the population.
2. Monitor condom used in stratified population and access the level of compliance at a given period of time (three months interval).

It is observed that the sensitivity values (Tables 5 and 6) of the intervention strategy ($r_{c,\ell}, r_h, r_d, r_c, \theta_h, \theta_2, \theta_4, \theta_6, \theta_4, \theta_6$) are negative. The epidemiological implication is that those parameters will always have negative impact on the transmission dynamics of HIV/AIDS (reproduction number) no matter how small they are administered into the population. For examples, 20% decrease of the infected MSM ($I_h$) and IDU ($I_d$) as result of counselling...
MSM individuals modify their sexual behaviour due to counselling. Counselling single strategy has the highest attenuating effect on the transmission of HIV/AIDS MSM population (Fig. 8).

Fig. 8. Bar plot of MSM baseline parameters.

will increase the potency of the modification parameter which account for the reduction of infectiousness by 0.00025 in both MSM ($I_a$) and IDU ($I_d$) and decrease the transmission dynamics of the disease by 7.4% and 2.6% of $R_h$ and $R_d$ respectively. Also, the transmission dynamics of the disease $R_h$ will reduce by 2.2% if 20% of the total number of infected individuals in MSM class comply to condom used. The sensitivity value of the relative risk of infectiousness of non-counseled individual will attenuate $R_h$ and $R_d$ by 10.4% and 1.1%, if 20% of their AIDS class $A_h$ and $A_d$ will adhere to strict compliance of the intervention strategy. Unlike the counselled case which requires only 2.3% of $R_h$ by 20% reduction of $A_h$.

Lastly, the sensitivity value of the treatment parameter $\pi_{R_h}^N$ and $\pi_{R_d}^N$ will increase the modification parameter which account for the reduction of infectiousness via treatment by 0.0005 (20% of $\theta_h$) and reduce the disease burden by 1.9% and 1.0% of MSM and IDU respectively. (See Fig. 8.)

4. Conclusion

The impact of condom use, counselling and treatment were assessed in the HIV/AIDS population in Nigeria using a deterministic model (differential infectivity model). The sub-models were thoroughly analysed to understand the intricacy of the dynamical features of the epidemic. Biological and demographic data are used to simulate the model and assess the impact of the afore-mention intervention measures. Some of the main theoretical discovery of the study are:

1. The duality effect of the force of infection sustains backward bifurcation of HIV/AIDS in the population.
2. Infected high risk group with divergent risk of transmission of HIV/AIDS (IDUSA) is the cause of endemicity.
3. Infected drug users who quit the intake of drug, relax the asymptotic curve of infected population to zero at a shorter time for combined strategy than its counterpart (Fig. 5).
4. HIV/AIDS is effectively managed in a single mode population (IDU, MSM) than bi-mode population (IDUSA).

Declarations

Author contribution statement

Stephen Onome Oyovwevotu: Conceived and designed the experiments; Performed the experiments; Analyzed and interpreted the data; Contributed reagents, materials, analysis tools or data; Wrote the paper.

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Additional information

No additional information is available for this paper.
Appendix A

Here we show the details of the computation analysis submodels.

Proof of Theorem 3.

\[
\hat{F} = k_T R_h / \theta_h \{ (I_h + a_N A_h + \theta_c (I_c + a_c A_c) + \theta_h T_h \} S_h - k_h I_h \}
\]

\[
+ (k_h \theta_2 a_c + \theta_h r_c) (I_c + a_c A_c) - \theta_2 k_h 
\]

\[
+ (k_h \theta_2 a_c + \theta_h r_c) (I_c + a_c A_c) - \theta_2 k_h 
\]

\[
+ (k_h \theta_2 a_c + \theta_h r_c) (I_c + a_c A_c) - \theta_2 k_h 
\]

Further simplification gives

\[
\frac{R_h k_h I_h}{\theta_h} S_h - \frac{R_h k_h I_h}{\theta_h} S_h - \left( \frac{k_h \theta_2 a_c + \theta_h r_c}{\theta_2} \right) A_h + \frac{(R_h k_h \theta_2 T_h)}{\theta_h} S_h - k_h T_h +
\]

\[
\frac{k_h k_h I_h}{\theta_h} S_h - \frac{k_h k_h I_h}{\theta_h} S_h - \left( \frac{k_h \theta_2 a_c + \theta_h r_c}{\theta_2} \right) A_h + \frac{(R_h k_h \theta_2 T_h)}{\theta_h} S_h - k_h T_h +
\]

\[
\frac{k_h k_h I_h}{\theta_h} S_h - \frac{k_h k_h I_h}{\theta_h} S_h - \left( \frac{k_h \theta_2 a_c + \theta_h r_c}{\theta_2} \right) A_h + \frac{(R_h k_h \theta_2 T_h)}{\theta_h} S_h - k_h T_h +
\]

\[
\frac{k_h k_h I_h}{\theta_h} S_h - \frac{k_h k_h I_h}{\theta_h} S_h - \left( \frac{k_h \theta_2 a_c + \theta_h r_c}{\theta_2} \right) A_h + \frac{(R_h k_h \theta_2 T_h)}{\theta_h} S_h - k_h T_h +
\]

\[
\frac{k_h k_h I_h}{\theta_h} S_h - \frac{k_h k_h I_h}{\theta_h} S_h - \left( \frac{k_h \theta_2 a_c + \theta_h r_c}{\theta_2} \right) A_h + \frac{(R_h k_h \theta_2 T_h)}{\theta_h} S_h - k_h T_h +
\]

\[
\frac{k_h k_h I_h}{\theta_h} S_h - \frac{k_h k_h I_h}{\theta_h} S_h - \left( \frac{k_h \theta_2 a_c + \theta_h r_c}{\theta_2} \right) A_h + \frac{(R_h k_h \theta_2 T_h)}{\theta_h} S_h - k_h T_h +
\]

\[
\frac{k_h k_h I_h}{\theta_h} S_h - \frac{k_h k_h I_h}{\theta_h} S_h - \left( \frac{k_h \theta_2 a_c + \theta_h r_c}{\theta_2} \right) A_h + \frac{(R_h k_h \theta_2 T_h)}{\theta_h} S_h - k_h T_h +
\]

\[
\frac{k_h k_h I_h}{\theta_h} S_h - \frac{k_h k_h I_h}{\theta_h} S_h - \left( \frac{k_h \theta_2 a_c + \theta_h r_c}{\theta_2} \right) A_h + \frac{(R_h k_h \theta_2 T_h)}{\theta_h} S_h - k_h T_h +
\]

\[
\frac{k_h k_h I_h}{\theta_h} S_h - \frac{k_h k_h I_h}{\theta_h} S_h - \left( \frac{k_h \theta_2 a_c + \theta_h r_c}{\theta_2} \right) A_h + \frac{(R_h k_h \theta_2 T_h)}{\theta_h} S_h - k_h T_h +
\]

\[
\frac{k_h k_h I_h}{\theta_h} S_h - \frac{k_h k_h I_h}{\theta_h} S_h - \left( \frac{k_h \theta_2 a_c + \theta_h r_c}{\theta_2} \right) A_h + \frac{(R_h k_h \theta_2 T_h)}{\theta_h} S_h - k_h T_h +
\]

\[
\frac{k_h k_h I_h}{\theta_h} S_h - \frac{k_h k_h I_h}{\theta_h} S_h - \left( \frac{k_h \theta_2 a_c + \theta_h r_c}{\theta_2} \right) A_h + \frac{(R_h k_h \theta_2 T_h)}{\theta_h} S_h - k_h T_h +
\]

\[
\frac{k_h k_h I_h}{\theta_h} S_h - \frac{k_h k_h I_h}{\theta_h} S_h - \left( \frac{k_h \theta_2 a_c + \theta_h r_c}{\theta_2} \right) A_h + \frac{(R_h k_h \theta_2 T_h)}{\theta_h} S_h - k_h T_h +
\]

\[
\frac{k_h k_h I_h}{\theta_h} S_h - \frac{k_h k_h I_h}{\theta_h} S_h - \left( \frac{k_h \theta_2 a_c + \theta_h r_c}{\theta_2} \right) A_h + \frac{(R_h k_h \theta_2 T_h)}{\theta_h} S_h - k_h T_h +
\]

\[
\frac{k_h k_h I_h}{\theta_h} S_h - \frac{k_h k_h I_h}{\theta_h} S_h - \left( \frac{k_h \theta_2 a_c + \theta_h r_c}{\theta_2} \right) A_h + \frac{(R_h k_h \theta_2 T_h)}{\theta_h} S_h - k_h T_h +
\]

\[
\frac{k_h k_h I_h}{\theta_h} S_h - \frac{k_h k_h I_h}{\theta_h} S_h - \left( \frac{k_h \theta_2 a_c + \theta_h r_c}{\theta_2} \right) A_h + \frac{(R_h k_h \theta_2 T_h)}{\theta_h} S_h - k_h T_h +
\]

\[
\frac{k_h k_h I_h}{\theta_h} S_h - \frac{k_h k_h I_h}{\theta_h} S_h - \left( \frac{k_h \theta_2 a_c + \theta_h r_c}{\theta_2} \right) A_h + \frac{(R_h k_h \theta_2 T_h)}{\theta_h} S_h - k_h T_h +
\]

\[
\frac{k_h k_h I_h}{\theta_h} S_h - \frac{k_h k_h I_h}{\theta_h} S_h - \left( \frac{k_h \theta_2 a_c + \theta_h r_c}{\theta_2} \right) A_h + \frac{(R_h k_h \theta_2 T_h)}{\theta_h} S_h - k_h T_h +
\]

\[
\frac{k_h k_h I_h}{\theta_h} S_h - \frac{k_h k_h I_h}{\theta_h} S_h - \left( \frac{k_h \theta_2 a_c + \theta_h r_c}{\theta_2} \right) A_h + \frac{(R_h k_h \theta_2 T_h)}{\theta_h} S_h - k_h T_h +
\]

\[
\frac{k_h k_h I_h}{\theta_h} S_h - \frac{k_h k_h I_h}{\theta_h} S_h - \left( \frac{k_h \theta_2 a_c + \theta_h r_c}{\theta_2} \right) A_h + \frac{(R_h k_h \theta_2 T_h)}{\theta_h} S_h - k_h T_h +
\]

\[
\frac{k_h k_h I_h}{\theta_h} S_h - \frac{k_h k_h I_h}{\theta_h} S_h - \left( \frac{k_h \theta_2 a_c + \theta_h r_c}{\theta_2} \right) A_h + \frac{(R_h k_h \theta_2 T_h)}{\theta_h} S_h - k_h T_h +
\]

\[
\frac{k_h k_h I_h}{\theta_h} S_h - \frac{k_h k_h I_h}{\theta_h} S_h - \left( \frac{k_h \theta_2 a_c + \theta_h r_c}{\theta_2} \right) A_h + \frac{(R_h k_h \theta_2 T_h)}{\theta_h} S_h - k_h T_h +
\]

\[
\frac{k_h k_h I_h}{\theta_h} S_h - \frac{k_h k_h I_h}{\theta_h} S_h - \left( \frac{k_h \theta_2 a_c + \theta_h r_c}{\theta_2} \right) A_h + \frac{(R_h k_h \theta_2 T_h)}{\theta_h} S_h - k_h T_h +
\]

\[
\frac{k_h k_h I_h}{\theta_h} S_h - \frac{k_h k_h I_h}{\theta_h} S_h - \left( \frac{k_h \theta_2 a_c + \theta_h r_c}{\theta_2} \right) A_h + \frac{(R_h k_h \theta_2 T_h)}{\theta_h} S_h - k_h T_h +
\]

\[
\frac{k_h k_h I_h}{\theta_h} S_h - \frac{k_h k_h I_h}{\theta_h} S_h - \left( \frac{k_h \theta_2 a_c + \theta_h r_c}{\theta_2} \right) A_h + \frac{(R_h k_h \theta_2 T_h)}{\theta_h} S_h - k_h T_h +
\]

\[
\frac{k_h k_h I_h}{\theta_h} S_h - \frac{k_h k_h I_h}{\theta_h} S_h - \left( \frac{k_h \theta_2 a_c + \theta_h r_c}{\theta_2} \right) A_h + \frac{(R_h k_h \theta_2 T_h)}{\theta_h} S_h - k_h T_h +
\]

\[
\frac{k_h k_h I_h}{\theta_h} S_h - \frac{k_h k_h I_h}{\theta_h} S_h - \left( \frac{k_h \theta_2 a_c + \theta_h r_c}{\theta_2} \right) A_h + \frac{(R_h k_h \theta_2 T_h)}{\theta_h} S_h - k_h T_h +
\]
Hence $F < 0$ whenever $R_e = \max \{R_h \leq 1, R_d \leq 1\}$ and all parameters and variables of the model (4) are nonnegative, $F < 0$ when $R_e = \max \{R_h \leq 1, R_d \leq 1\} < 1$ with $\tilde{F} = 0$ iff $I_h = A_h = I_d = A_d = I_{cd} = A_{cd} = T_h = T_d = T_{cd} = 0$. It follows from Lasalle’s Invariance Principle, that every solution to the equations in model (4) with initial conditions in $D_f$ converges to $\epsilon_o$ as $t \to \infty$. The epidemiology implication is that if the control strategies considered in the model (counselling, condom and ART) are implemented in such a way that they lead to $R_e < 1$, then HIV/AIDS will be effectively controlled or eliminated.  

**Proof of Theorem 5.** Set all induced death parameters to zero. System (4) reduces to $dN/dt = \Lambda - \mu N$. So that $N \to \Lambda/\mu = N^{**}$ as $t \to \infty$. Hence, using the substitution,

$$S_h = N^{**} - (I_h + I_{ds} + A_h + A_{ds} + I_{ch} + A_{ch} + I_{cd} + A_{cd} + T_h + T_{ds})$$

$$S_d = N^{**} - (I_d + I_{cd} + A_d + A_{cd} + T_d + I_{qd} + A_{qd} + T_{qd} + T_{cd})$$

The model (4) can be rewritten as

$$dI_h/dt = \beta(I_h + I_{ds} + A_h + A_{ds} + I_{ch} + A_{ch} + I_{cd} + A_{cd} + T_h + T_{ds})$$

$$dN^{**}/dt = \theta_1(I_h + I_{ds} + A_h + A_{ds} + I_{ch} + A_{ch} + I_{cd} + A_{cd} + T_h + T_{ds})$$

$$dI_{ds}/dt = \theta_2(I_h + I_{ds} + A_h + A_{ds} + I_{ch} + A_{ch} + I_{cd} + A_{cd} + T_h + T_{ds})$$

$$dA_h/dt = \sigma_2 I_h - \gamma_s A_h$$

$$dA_{ds}/dt = \sigma_2 I_{ds} - \gamma_s A_{ds}$$

$$dA_{ch}/dt = \sigma_1 I_{ch} - \gamma_s A_{ch}$$

$$dA_{cd}/dt = \sigma_1 I_{cd} - \gamma_s A_{cd}$$

$$dA_{cd}/dt = \sigma_1 I_{cd} - \gamma_s A_{cd}$$

$$dI_{ch}/dt = \tau_h A_h + \tau_{ch} A_{ch} - \gamma_s T_h$$

$$dT_{cd}/dt = \tau_{ds} A_{cd} + \tau_{ds} A_{cd} - \gamma_{00} T_{cd}$$

Linearizing the model (10) around the endemic equilibrium point gives

$$dI_h/dt = [(y_3 - y_1) - \gamma_s I_h + (\theta y_3 - y_1) I_{ch} + (y_3 - y_1) I_{ds} + (\theta y_3 - y_1) I_{cd} + \theta A_h + \theta A_{ds} + \theta A_{cd} + \theta T_h + \theta T_{ds}]$$

$$dI_{ds}/dt = [(y_3 - y_1) - \gamma_s I_{ds} + (\theta y_3 - y_1) I_{cd} + (y_3 - y_1) I_{ds} + \theta A_h + \theta A_{ds} + \theta A_{cd} + \theta T_h + \theta T_{ds}]$$

$$dA_h/dt = \sigma_2 I_h - \gamma_s A_h$$

$$dA_{ds}/dt = \sigma_2 I_{ds} - \gamma_s A_{ds}$$

$$dA_{ch}/dt = \sigma_1 I_{ch} - \gamma_s A_{ch}$$

$$dA_{cd}/dt = \sigma_1 I_{cd} - \gamma_s A_{cd}$$

$$dI_{ch}/dt = \tau_h A_h + \tau_{ch} A_{ch} - \gamma_s T_h$$

$$dT_{cd}/dt = \tau_{ds} A_{cd} + \tau_{ds} A_{cd} - \gamma_{00} T_{cd}$$

Where:

$$y_1 = \beta(1 - \epsilon_o) [N_h^{**} - \theta (I_h + I_{ds} + A_h + A_{ds} + I_{ch} + A_{ch} + I_{cd} + A_{cd} + T_h + T_{ds})] / N$$

$$y_2 = \beta(1 - \epsilon_o) N_h^{**} / N$$

The Jacobian of the system (10) evaluated at endemic equilibrium point is given by

$$J = \begin{bmatrix}
  m_3 - k_1 & m_2 & 0 & 0 & m_3 & m_4 & 0 & 0 & m_6 & 0 \\
  r & -k_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
  0 & 0 & y_3 k_3 & \theta z_3 & 0 & 0 & a_{2N} y_3 & \theta_2 z_3 & 0 & \theta_3 z_3 \\
  0 & 0 & r & -k_4 & 0 & 0 & 0 & 0 & 0 & 0 \\
  \sigma_4 & 0 & 0 & 0 & -k_5 & 0 & 0 & 0 & 0 & 0 \\
  0 & \sigma_4 & 0 & 0 & 0 & -k_6 & 0 & 0 & 0 & 0 \\
  0 & 0 & \sigma_2 & 0 & 0 & 0 & -k_7 & 0 & 0 & 0 \\
  0 & 0 & \sigma_2 & 0 & 0 & 0 & -k_8 & 0 & 0 & 0 \\
  0 & 0 & 0 & r_h & \tau_{ch} & 0 & 0 & -k_9 & 0 & 0 \\
  0 & 0 & 0 & 0 & 0 & 0 & \tau_{cd} & \tau_{cd} & 0 & -k_{10}
\end{bmatrix}$$
\[ m_1 = (y_2 - y_1), \ m_2 = (\theta y_2 - y_1), \ m_3 = (a_{2N} y_2 - y_1), \ m_4 = (\theta a_{y_2} - y_1), \ m_6 = (\theta a_{y_2} - y_1) \]

Next, established that system (10) has no solution of the form

\[ A(t) = A_0 e^{\mu t} \] \hspace{1cm} (A.3)

with \( A_0 = (A_1, A_2, A_3, A_4, A_5, A_6, A_7, A_8, A_9, A_{10}) \) and \( \omega, A_i \in \mathbb{C} \ \forall i = 1, 2, 3 \ldots 10 \). Substituting solution of the form (12) into system (10) gives

\[ \omega A_1 = d I_0 /dt = [(y_2 - y_1) - g_1] A_1 + (\theta y_2 - y_1) A_2 + (y_2 - y_1) A_3 + (\theta y_2 - y_1) A_4 + (a_{2N} y_2 - y_1) A_5 + (\theta a_{y_2} - y_1) A_6 + (\theta a_{y_2} - y_1) A_7 + (\theta a_{y_2} - y_1) A_8 + (\theta a_{y_2} - y_1) A_9 + (\theta a_{y_2} - y_1) A_{10} \]

\[ \omega A_2 = y_1 A_1 + \theta y_2 A_2 + y_2 A_3 + \theta y_3 A_4 + a_{2N} y_3 A_5 + \theta a_{y_2} A_6 + a_{N} A_7 + \theta a_{y_2} A_8 + \theta a_{y_2} A_9 + \theta a_{y_2} A_{10} \]

\[ \omega A_3 = r A_1 - K_A A_2, \]
\[ \omega A_4 = r A_2 - K_A A_3, \]
\[ \omega A_5 = r A_3 - K_A A_4, \]
\[ \omega A_6 = r A_4 - K_A A_5, \]
\[ \omega A_7 = r A_5 - K_A A_6, \]
\[ \omega A_8 = r A_6 - K_A A_7, \]
\[ \omega A_9 = r A_7 - K_A A_8, \]
\[ \omega A_{10} = r A_8 - K_A A_9. \]

(A.4)

Thorough algebraic simplification of system (13) results in the following system

\[ [1 + G_1(\omega)] A_1 = (HA)_1, \]
\[ [1 + G_2(\omega)] A_2 = (HA)_2, \]
\[ [1 + G_3(\omega)] A_3 = (HA)_3, \]
\[ [1 + G_4(\omega)] A_4 = (HA)_4, \]
\[ [1 + G_5(\omega)] A_5 = (HA)_5, \]
\[ [1 + G_6(\omega)] A_6 = (HA)_6, \]
\[ [1 + G_7(\omega)] A_7 = (HA)_7, \]
\[ [1 + G_8(\omega)] A_8 = (HA)_8, \]
\[ [1 + G_9(\omega)] A_9 = (HA)_9, \]
\[ [1 + G_{10}(\omega)] A_{10} = (HA)_{10}. \]

(A.5)

Where

\[ G_1(\omega) = 1 + (\omega - y_1) / K_3 - y_1 / K_4 [(\theta r) / (K_5 + \omega) - a_{2N} \sigma_3 / (K_5 + \omega)] + (\theta a_{y_2} \sigma_3 / (K_5 + \omega) + \theta_a A_y / (K_5 + \omega) + \theta a_{y_2} / (K_5 + \omega)] \]
\[ G_2(\omega) = 1 + (\omega + y_1) / K_3 + y_1 / K_4 [(\theta y_2 + \sigma_1) / (K_5 + \omega) + \theta a_{y_2} \sigma_1 / (K_5 + \omega)] + (\theta a_{y_2} \sigma_1 / (K_5 + \omega) + \theta a_{y_2} / (K_5 + \omega)] \]
\[ G_3(\omega) = 1 + (\omega - y_1) / K_3 - y_1 / K_4 [(\theta r) / (K_5 + \omega) - a_{2N} \sigma_3 / (K_5 + \omega)] + (\theta a_{y_2} \sigma_3 / (K_5 + \omega) + \theta a_{y_2} / (K_5 + \omega)] \]
\[ G_4(\omega) = 1 + (\omega + y_1) / K_3 + y_1 / K_4 [(\theta y_2 + \sigma_1) / (K_5 + \omega) + \theta a_{y_2} \sigma_1 / (K_5 + \omega)] + (\theta a_{y_2} \sigma_1 / (K_5 + \omega) + \theta a_{y_2} / (K_5 + \omega)] \]
\[ G_5(\omega) = 1 + (\omega - y_1) / K_3 - y_1 / K_4 [(\theta r) / (K_5 + \omega) - a_{2N} \sigma_3 / (K_5 + \omega)] + (\theta a_{y_2} \sigma_3 / (K_5 + \omega) + \theta a_{y_2} / (K_5 + \omega)] \]
\[ G_6(\omega) = 1 + (\omega + y_1) / K_3 + y_1 / K_4 [(\theta y_2 + \sigma_1) / (K_5 + \omega) + \theta a_{y_2} \sigma_1 / (K_5 + \omega)] + (\theta a_{y_2} \sigma_1 / (K_5 + \omega) + \theta a_{y_2} / (K_5 + \omega)] \]
\[ G_7(\omega) = 1 + (\omega - y_1) / K_3 - y_1 / K_4 [(\theta r) / (K_5 + \omega) - a_{2N} \sigma_3 / (K_5 + \omega)] + (\theta a_{y_2} \sigma_3 / (K_5 + \omega) + \theta a_{y_2} / (K_5 + \omega)] \]
\[ G_8(\omega) = 1 + (\omega + y_1) / K_3 + y_1 / K_4 [(\theta y_2 + \sigma_1) / (K_5 + \omega) + \theta a_{y_2} \sigma_1 / (K_5 + \omega)] + (\theta a_{y_2} \sigma_1 / (K_5 + \omega) + \theta a_{y_2} / (K_5 + \omega)] \]
\[ G_9(\omega) = 1 + (\omega - y_1) / K_3 - y_1 / K_4 [(\theta r) / (K_5 + \omega) - a_{2N} \sigma_3 / (K_5 + \omega)] + (\theta a_{y_2} \sigma_3 / (K_5 + \omega) + \theta a_{y_2} / (K_5 + \omega)] \]
\[ G_{10}(\omega) = 1 + (\omega + y_1) / K_3 + y_1 / K_4 [(\theta y_2 + \sigma_1) / (K_5 + \omega) + \theta a_{y_2} \sigma_1 / (K_5 + \omega)] + (\theta a_{y_2} \sigma_1 / (K_5 + \omega) + \theta a_{y_2} / (K_5 + \omega)] \]

If \( A \) is a solution of (13) then it is possible to find a minimal positive real number, \( \epsilon \), such that \( ||A|| \leq \epsilon E_1 \) where \( ||A|| = (||A_1||, ||A_2||, ||A_3||, \ldots, ||A_{10}||) \).

Next we show that \( \text{Re}(\omega) \geq 0 \). Assume by contradiction, that \( \text{Re}(\omega) < 0 \). There are two cases to consider.

Case 1: \( \omega = 0 \).

For the case \( \omega = 0 \), equation (19) becomes a homogeneous linear system in the variables \( A_i \ \forall i = 1, 2 \ldots 10 \). The determinant of the system is given by
\[ \Delta_{t,\text{sim}} = K_s K_c K_s K_c (R_s - 1), \Delta_{t,\text{gas}} = K_s K_c K_s K_c (R_{c,i} - 1). \]

It follows that system (13) has a unique solution given by \( A = 0 \) and this solution corresponds to the DFE, since the determinants of both classes are negative whenever \( R_s \geq 1 \) and \( R_{c,i} \leq 1 \).

Case 2: \( a \neq 0 \).

Consider the case \( Re(a) > 0 \) (by assumption), then \( |1 + G_1(a)| > 1 \). Let \( G_1(a) = \min|1 + G_1|, \) then \( G_1(a) > 1 \) and \( \epsilon / G_1 < \epsilon \). Since \( \epsilon \) is a minimal positive real number such that \( \| A \| < \epsilon \), then

\[ \| A \| > \epsilon E \langle G_0 \rangle < \epsilon \]

On the reverse, by taking the norm of both sides of the second equation in (15), we have

\[ G_2(\| A \|) \leq \| (H A) \| \leq \| H \| \| A \| \leq \epsilon H (E_1) \]

\[ = (E_1)_2 \epsilon - \epsilon \| E_1 \|^2 \]

(A.6)

It implies that \( \| A \| \leq \epsilon / G_2(\| A \|) \), which contradicts (A.6). Hence \( Re(a) < 0 \). Thus, all eigenvalues of the characteristic equation associated with the linearized system will have negative real part, so that the unique endemic equilibrium, \( E_1 \) is LAS whenever \( R_s = \{ R_s, R_{c,i} \} > 1 \). The epidemiological implication of Theorem 5 is that the disease will persist in the population if \( R_s > 1 \). Thus, the effectiveness level of control Strategies is insufficient to bring the epidemiological threshold \( R_s \), to a value less than unity (hence the disease will remain in the endemic population).

Proof of Theorem 7. Consider the model (4) with \( \lambda_i \) and \( R_i^1 > 1 \), so that the associated unique endemic equilibrium of the model exists. Further, consider the following non-linear Lyapunov function (of Goh-Voltterra type):

\[ F = S_h - S_h^* - S_h^* \ln(S_h/S_h^*) + I_h - I_h^* - I_h^* \ln(I_h/I_h^*) + \frac{\beta S_h^* (\tau S_h \sigma + K_h(\sigma_1 + \sigma_2))}{K_h K_h} + \frac{\beta S_h^* (\tau S_h + K_h)}{K_h K_h} (A_h - A_h^* - A_h^* \ln(A_h/A_h^*) + \frac{\beta S_h^* (\tau S_h + K_h)}{K_h K_h} (A_h^* - A_h^*)). \]

With Lyapunov derivative, equation

\[ \dot{F} = \frac{\partial F}{\partial S_h} (\dot{S}_h - S_h^*) \frac{\beta S_h^* (\tau S_h \sigma + K_h(\sigma_1 + \sigma_2))}{K_h K_h} + \frac{\partial F}{\partial I_h} (\dot{I}_h - I_h^*) \frac{\beta S_h^* (\tau S_h + K_h)}{K_h K_h} \]

(A.7)

Since \( \dot{S}_h \) and \( \dot{I}_h \) are non-negative and \( S_h^* \) and \( I_h^* \) are positive, and \( \beta S_h^* (\tau S_h \sigma + K_h) \) is also positive, the derivative of \( F \) is non-positive, and \( F \) is bounded above by a constant.

Proof of Theorem 8. Consider the model (4) with \( \lambda_i \) and \( R_i^1 > 1 \), so that the associated unique endemic equilibrium of the model exists. Further, consider the following non-negative Lyapunov function (of Goh-Voltterra type):

\[ F = S_h - S_h^* - S_h^* \ln(S_h/S_h^*) + I_h - I_h^* - I_h^* \ln(I_h/I_h^*) + \frac{\beta S_h^* (\tau S_h \sigma + K_h(\sigma_1 + \sigma_2))}{K_h K_h} + \frac{\beta S_h^* (\tau S_h + K_h)}{K_h K_h} (A_h - A_h^* - A_h^* \ln(A_h/A_h^*) + \frac{\beta S_h^* (\tau S_h + K_h)}{K_h K_h} (A_h^* - A_h^*)). \]

With Lyapunov derivative, equation

\[ \dot{F} = \frac{\partial F}{\partial S_h} (\dot{S}_h - S_h^*) \frac{\beta S_h^* (\tau S_h \sigma + K_h(\sigma_1 + \sigma_2))}{K_h K_h} + \frac{\partial F}{\partial I_h} (\dot{I}_h - I_h^*) \frac{\beta S_h^* (\tau S_h + K_h)}{K_h K_h} \]

(A.7)

Since \( \dot{S}_h \) and \( \dot{I}_h \) are non-negative and \( S_h^* \) and \( I_h^* \) are positive, and \( \beta S_h^* (\tau S_h \sigma + K_h) \) is also positive, the derivative of \( F \) is non-positive, and \( F \) is bounded above by a constant.
\[
- \beta S^{*}_h r_h A_h T^{*}_h \frac{r_h}{T_h} - \beta S^{*}_h r_c h I_c h T^{*}_h \frac{r_c h}{T_h} + \beta S^{*}_h T^{*}_h.
\]

It can be shown from the model (4), at endemic steady state, that

\[
p\lambda = \beta S^{*}_h (I_h + A^{*}_h + I^{*}_c h + A^{*}_c h + T^{*}_h) - \mu S^{*}_h
\]

\[
k_1 I^{*}_h = \beta S^{*}_h (I_h + A^{*}_h + I^{*}_c h + A^{*}_c h + T^{*}_h)
\]

\[
r_h I^{*}_h = k_2 I_c h
\]

\[
\sigma_1 I^{*}_h = k_3 A^{*}_c h
\]

\[
k_6 A^{*}_c h = \sigma_1 I^{*}_c h
\]

\[
k_4 T^{*}_h = r_c h A^{*}_c h + r_c h A^{*}_c h
\]

\[
\beta S^{*}_h (1 + \frac{S^{*}_h}{S^{*}_h}) + \mu S^{*}_h (2 - \frac{S^{*}_h}{S^{*}_h}) + \beta S^{*}_h (I^{*}_c h + A^{*}_c h + T^{*}_h) (4 - \frac{S^{*}_h}{S^{*}_h}) - A^{*}_c h I^{*}_c h + I^{*}_c h I^{*}_c h)
\]

Using the assumption \( S_h \leq S^{*}_h \), the terms in (14) can be written as

\[
3. \quad 3 - S^{*}_h \frac{A^{*}_h I^{*}_c h + A^{*}_h I^{*}_c h}{A^{*}_h I^{*}_c h + A^{*}_h I^{*}_c h} \leq 3 - \frac{S^{*}_h}{S^{*}_h} - \frac{A^{*}_h I^{*}_c h}{A^{*}_h I^{*}_c h} - \frac{I^{*}_c h I^{*}_c h}{I^{*}_c h I^{*}_c h}
\]

\[
4. \quad 3 - \frac{A^{*}_h I^{*}_c h}{A^{*}_h I^{*}_c h} - \frac{I^{*}_c h I^{*}_c h}{I^{*}_c h I^{*}_c h} \leq 3 - S^{*}_h \frac{A^{*}_h I^{*}_c h + A^{*}_h I^{*}_c h}{A^{*}_h I^{*}_c h + A^{*}_h I^{*}_c h} - A^{*}_h I^{*}_c h + A^{*}_h I^{*}_c h - A^{*}_h T^{*}_h
\]

\[
5. \quad 3 - \frac{A^{*}_h I^{*}_c h}{A^{*}_h I^{*}_c h} - \frac{I^{*}_c h I^{*}_c h}{I^{*}_c h I^{*}_c h} \leq 3 - S^{*}_h \frac{A^{*}_h I^{*}_c h + A^{*}_h I^{*}_c h}{A^{*}_h I^{*}_c h + A^{*}_h I^{*}_c h} - A^{*}_h I^{*}_c h + I^{*}_c h I^{*}_c h
\]

Since the arithmetic mean is greater than the geometric mean, the following inequalities are satisfied

\[
1. \quad \frac{S^{*}_h}{S_h} \leq 0
\]

\[
2. \quad \frac{S^{*}_h}{S_h} \leq \frac{S_h}{S^{*}_h} \leq 0
\]

\[
3. \quad \frac{I^{*}_c h I^{*}_c h}{I^{*}_c h I^{*}_c h} \leq \frac{S^{*}_h}{S_h} \frac{A^{*}_h I^{*}_c h}{A^{*}_h I^{*}_c h} + \frac{I^{*}_c h I^{*}_c h}{I^{*}_c h I^{*}_c h}
\]

Thus \( F \leq 0 \) for \( R_0^* > 1 \). Hence, \( F \) is a Lyapunov function on \( D^{*}_h \). It follows by Lasalle’s invariance principle, that every solution to the equation of the model (3) and initial condition in \( D^{*}_h / D_h \) approaches the associated unique endemic equilibrium of the model as \( t \to \infty \) for \( R_0^* > 1 \).

Backward bifurcation: let \( S_h = s_1, I_h = s_2, I_c h = s_3, I_d s = s_4, I_d s = s_5, A_h = s_6, A_c h = s_7, A_d s = s_8, T_h = s_9, T_d s = s_10, \). Then the linearized form of system (3) can be written as

\[
dx_1/dt = [(y_2 - y_1) x_1 + (\theta_2 y_1 - y_1) x_2 + (y_2 - y_1) x_3 + (\theta_2 y_1 - y_1) x_4 + (a_N y_2 - y_1) x_5 +
\]

\[
(\theta_2, a_N y_2 - y_1) x_4 + (a_2 y_2 - y_1) x_5 + (\theta_2, a_2 y_2 - y_1) x_6 + (a_2 y_2 - y_1) x_7 + (\theta_2, a_2 y_2 - y_1) x_8 + (a_2 y_2 - y_1) x_9] +
\]

\[
dx_2/dt = (y_1 x_1 - I_d s) + (\theta_1 y_1 x_2 - I_d s) + (y_1 x_3 - I_d s) + (\theta_1 y_1 x_4 - A_2 s) +
\]

\[
(a_N y_2 - A_2 s) + (\theta_2 y_2 - y_1) x_5 + (a_N y_2 - y_1) x_6 + (a_2 y_2 - y_1) x_7 + (\theta_2, a_2 y_2 - y_1) x_8 + (a_2 y_2 - y_1) x_9 +
\]

\[
dx_3/dt = x_1 - g_2 x_2
\]

\[
dx_4/dt = x_3 - g_3 x_4
\]

\[
dx_5/dt = x_4 - g_4 x_5
\]

\[
dx_6/dt = x_5 - g_5 x_6
\]

\[
dx_7/dt = x_7 - g_7 x_8
\]

\[
dx_8/dt = x_8 - g_8 x_9
\]

\[
dx_9/dt = x_9 - g_9 x_{10}
\]
\[
\begin{aligned}
dx_5 &= \sigma_2 x_4 - g_5 x_6 \\
dx_6 &= c_5 x_5 + r_{10} x_5 - g_9 x_9 \\
dx_{10} &= r_{10} x_1 + r_{10} x_4 - g_{10} x_{10}.
\end{aligned}
\]

\[
J = \begin{bmatrix}
c_1 - k_1 & c_2 & 0 & 0 & c_4 & 0 & 0 & c_3 & 0 \\
\tau & -k_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & y_3 & \theta_3 y_3 & 0 & 0 & a_{2N} y_3 & \theta_2 a_{2N} y_3 & 0 \\
0 & 0 & \tau & -k_4 & 0 & 0 & 0 & 0 & 0 \\
\sigma_4 & 0 & 0 & 0 & -k_5 & 0 & 0 & 0 & 0 \\
0 & \sigma_4 & 0 & 0 & 0 & -k_6 & 0 & 0 & 0 \\
0 & 0 & \sigma_2 & 0 & 0 & 0 & -k_7 & 0 & 0 \\
0 & 0 & 0 & \sigma_2 & 0 & 0 & 0 & -k_8 & 0 \\
0 & 0 & 0 & 0 & \tau_8 & \tau_{10} & 0 & 0 & -k_9 \\
0 & 0 & 0 & 0 & 0 & \tau_{10} & \tau_{10} & 0 & -k_{10}
\end{bmatrix}
\]

\[c_1 = y_2 - y_1, c_2 = \theta_6 y_2 - y_1, c_3 = a_N y_2 - y_1, c_4 = a_N y_2 - y_1, c_5 = \theta_9 y_2 - y_1\]

The matrix \(J\) has a simple zero Eigen-value (a centre) and all other Eigen-value having negative real part (hence, the centre manifold theory can be applied) we obtained the right eigenvectors associated with the zero eigenvalue, which we shall denote as \(W = [w_1, w_2, w_3, w_4, w_5, w_6, w_7, w_8, w_9, w_{10}, w_{11}]^T\).

\subsection{Right eigenvector}

The right eigenvector expression is obtained from the Jacobian matrix as follows:

\[
\begin{aligned}
-w_1 \mu - w_2 (y_2 - y_1 - k_1) - w_3 (\theta_6 y_2 - y_1) - w_4 (a_N y_2 - y_1) - w_5 (\theta_2 a_N y_2 - y_1) - w_6 (a_N y_2 - y_1) - w_7 (\theta_2 a_N y_2 - y_1) - w_8 (a_N y_2 - y_1) - w_9 (\theta_2 a_N y_2 - y_1) - w_{10} (\theta_2 a_N y_2 - y_1) = 0 \\
-w_2 (y_2 - y_1 - k_1) + w_3 (\theta_6 y_2 - y_1) + w_5 (\theta_2 a_N y_2 - y_1) + w_7 (\theta_2 a_N y_2 - y_1) + w_{10} (\theta_2 a_N y_2 - y_1) = 0 \\
-w_3 (y_2 - y_1 - k_1) + w_4 \theta_6 y_2 + w_5 (a_N y_2 - y_1) + w_7 \theta_2 a_N y_2 + w_9 \theta_2 a_N y_2 + w_{10} \theta_2 a_N y_2 = 0 \\
-w_4 \tau - k_4 w_3 = 0 \\
w_5 \sigma_4 = k_4 w_6 = 0 \\
w_5 \sigma_4 - k_4 w_7 = 0 \\
w_5 \tau = k_4 w_5 = 0 \\
w_5 \sigma_2 = k_4 w_8 = 0 \\
w_5 \sigma_2 - k_4 w_9 = 0 \\
w_5 \tau_8 + w_7 \tau_{10} - \mu w_{10} = 0 \\
w_5 \tau_{10} + w_7 \tau_{10} - \mu w_{10} = 0
\end{aligned}
\]

Thorough algebraic simplification yielded

\[
\begin{aligned}
W_5 &= (w_{20} \tau) / k_2 \\
W_7 &= (w_{20} \sigma_4 \tau) / (k_2 k_6) \\
W_9 &= (w_{20} \sigma_4) / k_5 \\
W_5 &= (w_{20} \tau) / k_4 \\
W_6 &= (w_{20} \sigma_2 \tau) / (k_4 k_4) \\
W_8 &= (w_2 3) / k_8 \\
W_{10} &= [(w_2 \sigma_2) / (k_5 + (w_{20} \tau_{10} \sigma_4)) / (k_2 k_6)] / \mu \\
W_{11} &= [(w_2 \sigma_2 \sigma_4) / k_8 + (w_{20} \tau_{10} \sigma_2) / k_4] / \mu \\
w_1 &= w_2 (y_2 - y_1 - k_1) - \tau / k_2 (\theta_6 y_2 - y_1) - \sigma_4 / k_2 (a_N y_2 - y_1) - \tau_4 / k_2 (\theta_2 a_N y_2 - y_1) - (w_2 \sigma_2) / k_8 + (\theta_2 a_N y_2 - y_1) / k_4 + (a_N y_2 - y_1) (w_4 \sigma_2) / k_4 \\
& (\theta_2 a_N y_2 - y_1) (w_4 \sigma_2) / (k_4 k_4) + ((w_2 \sigma_2 \sigma_4) / k_8 + (\theta_9 y_2 - y_1) (w_4 \sigma_2) / k_3 \mu (k_4 k_3)).
\end{aligned}
\]

\[
w_1 > 0, w_2 > 0, w_3 > 0
\]

We can also obtain the left eigenvectors associated with the zero eigenvalue, satisfying: \(V^T W = 1\)

\[-\mu w_1 = 0\]
From the analysis above, we can deduce that the existence of backward bifurcation in system (3), depends on the efflux parameter $\Gamma_1$, which represent the duality effect of the force of infection on the susceptible classes. That is, multiple class of individuals susceptible to HIV/AIDS is responsible for backward bifurcation.

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