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A MATHEMATICAL MODEL FOR THE EFFECTS OF DRUG ABUSE IN TRANSMISSION OF HIV/AIDS IN TURKANA COUNTY

Research Report in Mathematics, Number 08, 2020

LOKARAN ELIZABETH

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Submitted to the School of Mathematics in partial fulfilment for a degree in Master of Science in Applied Mathematics

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Master Thesis

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Abstract

Drugs and substance misuse has been recognized to have a significant impact on the spread of HIV/AIDS epidemic. We formulate a deterministic model to assess the contribution of drug and substance abuse to the spread of HIV/AIDS disease among adults in Turkana County, Kenya. The basic reproduction number of the model is determined and stability of equilibria analyzed. The disease free equilibrium point is shown to be globally asymptotically stable when its corresponding basic reproduction number is less than unity. The Lyapunov theorem is used to show that the endemic equilibrium point is Globally asymptotically stable when its corresponding reproduction number is greater than unity.

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Declaration and Approval

I the undersigned declare that this dissertation is my original work and to the best of my knowledge, it has not been submitted in support of an award of a degree in any other university or institution of learning.

Signature Date

Lokaran Elizabeth
Reg No. I56/11852/2018

In my capacity as a supervisor of the candidate's dissertation, I certify that this dissertation has my approval for submission.

Signature Date

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Dedication

I dedicate this project to God Almighty my creator, my strong pillar, my source of inspiration, wisdom, knowledge and understanding. He has been the source of my strength throughout this program and on His wings only have I soared. I also dedicate this work to my husband; Akilimo Safari who has encouraged me all the way and whose encouragement has made sure that I give it all it takes to finish that which I have started. To my children Bildad Akilimo, Monique Rapoo, Looyapus Safari and Daisy Napeyok who have been affected in every way possible by this quest. Thank you. My love for you all can never be quantified. God bless you.

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1 Introduction

1.0.1 HIV/AIDS

HIV is an abbreviation that stands for Human Immuno-deficiency Virus while AIDS stand for Acquired Immune Deficiency Syndrome which is the higher level or the phase of HIV. Human Immunodeficiency Virus attacks the immune system of the human being and weakens the body to attack by other infections. HIV/AIDS is spread from infected individual during sexual intercourse, blood transfusion, through sharp objects such as injectible needles, from mother to child during gestation period and breast suckling that are principal means of its transmission (Fact HIV/AIDS, 2015). There is no medication to treat people if the person currently has HIV status. Nevertheless, identification and active handling of the infected persons in the phase with ARV therapy will prolong time for the infected people to progress to the phase. HIV/AIDS has continued to be an health issue and has so far claimed more than 32 million lives up to now but with increasing access to care, identification, treatment and prevention of opportunistic infections, the effects of the chronic health condition can be minimized. Towards end of 2o18, 37 million people were living with HIV/AIDS (WHO.,2019).

1.0.2 Substance and Drug abuse

A drug is any substance that alters the body functioning (Galvez, 2006). Drug abuse is the wrong use of a substance that interferes with both psychological and physical functioning of the body and can disrupt societal order and patterns of behavior (WHO.,2003). Medications that have influence on human mind can include both legal and illegal drugs (NACADA., 2012). Commonly abused drugs are, and not limited to Alcohol, Heroin, Cocaine, 'Marijuana', Tobacco etc. In Kenya 1.5 million people out of which 18,327 injection material consumers were HIV positive in 2016. Currently, HIV/AIDS prevalence in injection substance users who inject drugs stands at 18.3 percent.

1.0.3 Relationship between Alcohol, other drugs and HIV/AIDS

HIV stigma can lead to drug abuse and the converse is also true. The health of the drug user is compromised negatively influenced. This behavior exposes the drug abuser to offense and infectious diseases including HIV/AIDS (CDC.,2000). Drug and alcohol misuse can alter people's judgment and they may take risks that might expose them to the disease which would otherwise not be the case. The risk of HIV/AIDS transmission can also be related to non-injection drug. A case in point is Cocaine and amphetamine-type stimulants

(ATS) that can easily lead to higher risk of irresponsible sexual behavior which consequently lead to contraction of the illness. Methamphetamine, for example was linked to increased irresponsible behavior among the LGBT and heterosexuals. (Manserg., 2005). At the same time cocaine use may promote multiple sexual partners thus increasing likelihood of contracting HIV/AIDS (Edlin et al., 1994).

1.0.4 Statement of the problem

HIV/AIDS is a global pandemic and Kenya is ranked one of the high-burden countries. Kenya together with countries like Mozambique and Uganda are among those with the joint third-largest epidemic in the World, (AVERT.,2019). According to research by WHO report through the MOH in 2019 HIV/AIDS infection in the country indicate positive progress but new infections are still a challenge. Turkana is high incidence cluster-9 of the 47 Counties that contribute 57 percent of all new infections in Kenya(County HIV profile Report, 2014). Building on the previous deterministic models and focusing on subsequent infections, this mathematical model aims at understanding the dynamics of heterosexual transmission of HIV/AIDS in relation to alcohol and substance abuse based on age of young adults in Turkana county, Kenya. We try to answer the question; "Does alcohol and drug use bear an effect on HIV/AIDS transmission among young adults in Kenya?"

1.0.5 Research Objectives

General Objective

To develop a mathematical model that describes heterosexual transmission of HIV/AIDS in presence of drugs among the adults in Turkana county.

Specific Objectives

- To formulate a model on transmission of HIV/AIDS among adults drug abusers
- To deduce ordinary differential equations from the model.
- To determine DFE and EE points
- To compute the basic reproduction number.
- To investigate the local and global stabilities of DFE and EE points
- To Simulate the model

1.0.6 Justification of the Study

The study of co-infections has become an important aspect of social epidemic given its alarming rate especially those a rising from opportunistic diseases among HIV/AIDS infected individuals. Against this backdrop we endeavor to model how substance abuse is a major contributor to non-adherence to HIV/AIDS therapy. In this research we present a non-linear model on effect of drugs and substance abuse on HIV/AIDS transmission in Turkana county. We also focus on abuse of alcohol and other drugs then apply the model to secondary data of persons seeking treatment services for HIV/AIDS at Lodwar Referral Hospital in Turkana County. This study will sensitize policy makers in Kenya to focus on integration of drug therapy into HIV/AIDS prevention, care and treatment strategies as they develop mechanism where patients exhibiting substance abuse behaviors can be referred to rehabilitation treatment programs where available to mitigate the adverse effects HIV/AIDS pandemic.

METHODOLOGY

We develop a compartmental model using epidemiological approach and carry out analysis of the model by quantitatively fitting secondary data from Turkana County and infer the model parameters. This basically involves proof of positivity and boundedness of the model solutions, computing disease-free and endemic equilibrium point and their stability analysis, basic reproduction number and carry out simulation using MATLAB ode inbuilt solvers.

2 Literature Review

According to Omondi et al. (2018), Mathematical models are intended to help comprehend how HIV/AIDS spreads in various groups of people around the world. The model explores the degree of treatment, testing and transmission among the population in Kenya. The model accounts for the mix of HIV/AIDS intervention approaches like PrEP HTC and ART therapy. The research did not not consider entry of people who ha the infection to the susceptible and also never considered other transmission means routes like in those who inject drugs. This notwithstanding, their outcomes are relevant in formulating time-dependent intervention approaches in curbinng HIV/AIDS transmission.

Kok, (2015) formulated a dynamic version of HIV/AIDS therapy continuation of therapy in Vancouver Canada. He constructed a four sub-population, among them men who have sex with men (MSM), intraveneous drug users, female sex workers and the general populace. Their work based on primary activities that focus on treatment adherence and retention among the four risky groups. Their outcome never linked optimal resource allocation to ARV therapy adherence. In conclusion they advocated for more resource allocation to testing in high prevalence settings to obtain good data .

Mathematical model developed by Mardhiyah et al., (2012) was on how HIV/AIDS may spread among a community of injecting drug users. They used the basic reproduction number to analyse stability of the model and found out that the probability of HIV/AIDS transmission among those who inject drugs was such that when the reproduction number is greater than one, or otherwise, the disorder may not exist.

Comiskey et al.,(2007) developed a simple compartmental model with non-linear ordinary differential equations to mathematically represent and ascertain the role played treatment and also the recurrence in the dynamics of alcoholism. The analysis was driven by the simple fact that when the possibility of being a user of drugs exceeds the sum of quitting drug use, leading to increase in prevalence. Sensitivity analysis identified that being a drug user was a good parameter that should be targeted for reproduction number (R0) since to prevent drug use was better than curing it. They also recommended that, increasing prevention modalities should be done frequently other than emphasizing on relying on statistics of those individuals registered on treatment.

Chersich et al., (2007), in a paper on "Heavy Episodic Drinking among Kenyan Female sex workers in Mombasa" adduced that, about a third (1/3) of sex workers were HIV/AIDS infected. The study further revealed temporal linkages between use of alcohol and unsafe

sex. These two links might have been as a result of influence of alcohol on their correct use, negotiation on whether to use a condom and their incorrect use.

Ma et al., (2015) studied a lively alcohol intake model incorporating awareness and time delay, in which they desired to understand how this awareness was restraining the alcohol problems. They assumed the susceptible inhabitants avoided being in contact with each other because awareness programs execution resulted minimal drinking. The researchers argued that most of the alcoholism studies were Ordinary Differential Equation-oriented and did not incorporate the acts of time delay. In comparison to previous research, an integral novelty of their model was on introduction of delay in describing the time needed for an individual to become infectious. They discovered that embracing these programs was the main measure in controlling the alcohol problems by raising the value of time delay though bifurcation might appear.

Blower et al.,(1991)developed a design based on data to evaluate the effects of gender, injecting drug users and perinatal transmission. Though this study had thirty-four non-linear Ordinary Differential Equations, they analyzed and clarified the link between these modes of transmission and also provided great insights into the HIV/AIDS outbreak in New York City. The confidence intervals on the forecast estimates of accumulated estimates in the future of HIV/AIDS cases were broad. However, only a couple of variables are vital in contributing to the forecasting suggesting that to reduce estimation uncertainties in the biological–behavioral transmission parameters will probably have a much bearing on elevating the forecasting of adult HIV/AIDS instances than estimating sub-group sizes.

Mukandavire et al.,(2009) devised a compartmental design on the transmission dynamics of HIV/AIDS and malaria in a given community and rigorously evaluated it. The model considered the epidemiological synergy between malaria and sexually transmitted diseases. The study consisted of twelve non-linear Ordinary Differential Equations and they examined three components i.e malaria sub-model,HIV/AIDS sub-model and Malaria-HIV/AIDS sub-model. The results of the full HIV-malaria model revealed that the two infections co-existed so long as the reproduction number of each exceeded one another irrespective of their number sizes, as well as the number of new cases of malaria at steady state appears to always exceed that of HIV/AIDS. They also recommended for more research that considers the possible repercussions of HIV-Malaria co-infection in spread of HIV/AIDS

Thomas and Lungu., (2010) formulated a gender-based model on understanding these sexes, with varying heavy use of alcohol tendencies give rise to the HIV/AIDS spread differently. They used a system of twelve non-linear Ordinary Differential Equations. The duo concluded that binge drinking was a major force for HIV/AIDS among heavy drinking infected people.

P. Small et al., (1993) in their analysis on exogenous re-infection and drug resistance among the HIV infected people, concluded that immunity to anti-TB drugs could result not just in the virus that gave rise to that the original infection, but also in a new breed that could arise due to re-infection using a nother breed of M.TB which is resistant to drugs. Even Though the analysis was completed in the USA, the results are authentic worldwide (Barberis et al, .2017),

3 Model Formulation and Analysis

3.1 Model Formulation

We design a model which entails use of drugs in the population in aiding transmission of HIV/AIDS. We also carry out estimation of parameters and threshold values that will assist in describing the disease transmission dynamics and prediction of future trends and will help in suggesting how use of illicit drugs can be reduced to minimize HIV/AIDS transmission.

The population is divided into seven compartments.These compartments consist of susceptibles (S), infected (I) and individuals under treatments (T). Further we have susceptible individuals (S), individuals with drugs and substance use habits only ($\boldsymbol{I_D}$) and individuals infected with HIV/AIDS only($\boldsymbol{I_H}$). Similarly we have dually infected individuals with HIV/AIDS and also drug users ($\boldsymbol{I_{HD}}$) in addition we have HIV/AIDS positive individuals under treatment ($\boldsymbol{T_H}$)and Drug users under treatment ($\boldsymbol{T_D}$). Finally there are dually infected individuals (both with HIV/AIDS and on drugs)under treatment ($\boldsymbol{T_{HD}}$). It's assumed that AIDS patients are left out because they are not sexually active due to their health status. Therefore, the expression for the population at anytime (t) is

$$\boldsymbol{N(t) = S + I_H + I_D + I_{HD} + T_H + T_D + T_{HD}}$$

The assumption here is that susceptible are recruited at a constant rate $\wedge$ by natural birth. Those under treatment for drug use ($\boldsymbol{T_D}$) relapse and become susceptible (at the rate $\boldsymbol{Y_{2d}}$). Susceptible acquire HIV/AIDS status following contact with individuals with HIV/AIDS(at a rate $\boldsymbol{\beta_1}$).This class may also increase by an assumption that youth under treatment for dual infection can become infected with HIV/AIDS after interacting effectively with HIV/AIDS positive youths (at the rate γ_{1d}). In addition, susceptibles acquire drug use condition majorly through effective interaction with other drug users.These can as a result of drinking friends, peer pressure, family members who also use drugs etc (at a rate $\boldsymbol{\beta_2}$). Although typically there could be asymptomatic period, in this model we do not consider that and instead we assume that people gain infectiousness upon infection with drug abuse habits. Further natural death occurs in all human sub-populations (at a rate $\boldsymbol{\mu}$). The force of infection of HIV/AIDS infection is,

$$\boldsymbol{\lambda_H = \frac{\beta_1(I_H + \pi_1 I_{HD} + \pi_2 T_H + \pi_3 T_{HD})}{N}} \qquad \textbf{(1)}$$

where β_1 is the contact rate for HIV/AIDS transmission Further, the parameters $\pi_1 \geq 1$, π_2 and $\pi_3 \leq 1$ (i.e $\pi_1 \geq \pi_2 \geq \pi_3$) account for the relative infectiousness of infected persons in the I_{HD} class, T_H and T_{HD} compartments respectively compared to those in the I_H class. That is, persons in the I_{HD} class are more infectious than those in the I_H group because of their higher viral load level, similarly, HIV/AIDS infected persons (T_H and T_{HD}) are less infectious than I_H class because being under therapy significantly reduces their viral load.

drug users acquire these habits at a rate;

$$\lambda_D = \frac{\beta_2(I_D + (1-\pi_1)I_{HD} + \pi_4 T_D + (1-\pi_3)T_{HD})}{N} \tag{2}$$

where β_2 determines the "influence" of I_{HD}, T_D and T_{HD} on S. $(1-\pi_2)$, $(1-\pi_3)$ and π_4 are modification parameters for$T_D T_{HD}$which account for the increased likelihood of I_{HD},I_D.

Individuals using illicit drugs get HIV/AIDS at a rate ω_2. Those in I_D class move to I_{HD} on being HIV/AIDS positive at the rate ω_2 ($\omega_2 \geqslant 0$). These individuals can as well undergo treatment for drug use without acquiring HIV/AIDS at the rate ξ_2. Furthermore, individuals infected with HIV/AIDS can acquire illicit drug use habits at a rate ω_1. HIV/AIDS infected individuals can also undergo treatment theraphy without necessarilly being illicit drug users at the rate ξ_1.

Finally, dually infected individuals can either receive treatment separately for each infection i.e T_H for HIV/AIDS or T_D for illicit drug use at the rate ν_1 and ν_2 respectively or be treated for both conditions at the rate π. Moreover exit through death due to the severity of AIDS has been included hence AIDS class is left out because we assume that they are not sexually active to be pertinent in the ifnfection process.

Individuals undergoing treatment singly for each infection may as well receive treatment for the subsequent infection at the rates α_1 and α_2 respectively. Further individuals in the I_H (HIV/AIDS infected) class, active I_D (those infected with drug abuse habits), I_{HD} (dinfection-induced deaths at the rates δ_h,δ_d and δ_{hd} respectively.

3.2 Model equations

$$
\begin{cases}
\dfrac{dS}{dt} = \wedge + \gamma_{2d} T_D - \lambda_H S I_H - \lambda_D S I_D - \mu S \\
\dfrac{dI_H}{dt} = \lambda_H S I_H + \gamma_{1d} T_{HD} - (\mu + \delta_h + \xi_1 + \omega_1) I_H \\
\dfrac{dI_D}{dt} = \lambda_D S I_D - (\omega_2 + \xi_2 + \mu + \delta_d) I_D \\
\dfrac{dI_{HD}}{dt} = \omega_1 I_H + \omega_2 I_D - (\nu_1 + \nu_2 + \mu + \delta_{hd}) I_{HD} \\
\dfrac{dT_H}{dt} = \nu_1 I_{HD} + \xi_1 I_H - (\alpha_1 + \mu + \delta_h) T_H \\
\dfrac{dT_D}{dt} = \nu_2 I_{HD} + \xi_2 I_D - (\alpha_2 + \mu + \delta_d + \gamma_{2d}) T_D \\
\dfrac{dT_{HD}}{dt} = \alpha_1 T_H + \alpha_2 T_D - (\gamma_{1d} + \delta_{hd} + \mu) T_{HD}
\end{cases}
\tag{3}
$$

And with the initial conditions as;

$S(0), I_H(0), I_D(0), I_{HD}(0), T_H(0), T_D(0), T_{HD}(0)$

=(S)o,$(I_H)o, (I_D)o, (I_{HD})o, (T_H)o, (T_D)o, (T_{HD})o$

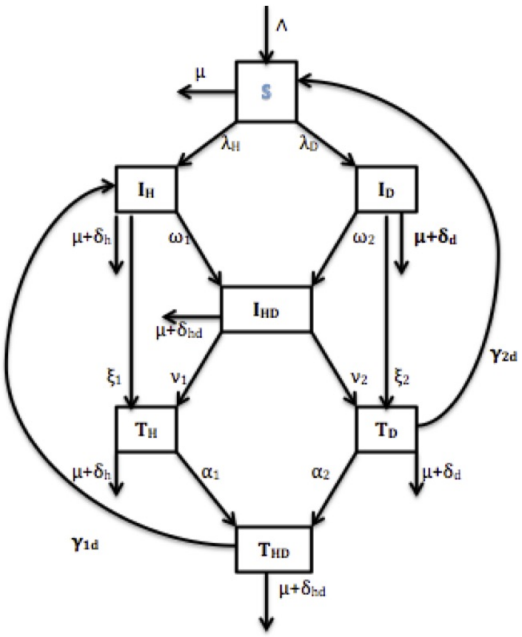

Figure 1(a). flow of individuals into different compartments

Description of variables and parameter used in the model

VARIABLE	DESCRIPTION
S	Susceptible population
$\boldsymbol{I_H}$	HIV/AIDS Infected individuals
$\boldsymbol{I_D}$	Infected Drug users
$\boldsymbol{I_{HD}}$	Infectives with HIV/AIDS and use Drug
$\boldsymbol{T_H}$	Individuals with HIV/AIDS under treatment
$\boldsymbol{T_D}$	Drug users under treatment
$\boldsymbol{T_{HD}}$	HIV/AIDS and Drug users under treatment

Table 1. Description of model variables

PARAMETER	DESCRIPTION
$\wedge$	**Recruitment rate for individuals by age**
β_1	**HIV/AIDS transmission rate**
β_2	**Drugs transmission rate**
λ_H	**Force of infection for HIV/AIDS**
λ_D	**Force of infection for drug abuse**
ω_1	**Rate at which HIV infected individuals become dually infected**
ω_2	**Rate at which drug users become dually infected**
ν_1	**Per capita treatment rate for HIV/AIDS**
ν_2	**Per capita treatment rate for Drug use**
α_1	**Rate at which T_H individuals receive treatment for dual infection**
α_2	**Rate at which T_D individuals receive for dual dual infection**
ξ_1	**Rate at which HIV/AIDS infected receive treatment for HIV/AIDS**
ξ_2	**Rate at which infected drug users receive treament for Drug use**
γ_{1d}	**Rate at which dually infected on treatment quit**
γ_{2d}	**Relapse rate for drug abusers**
μ	**Natural death rate**
δ_d	**Drug abuse induced death rate**
δ_h	**HIV/AIDS induced death rate**
δ_{hd}	**Death rate due to dual disease infection**

Table 2. Description of model parameters

3.3 Model Analysis

3.3.1 Positivity of solutions

Theorem 3.3.1. *Given* $(S(0), I_H(0), I_D(0), I_{HD}(0), T_H(0), T_D(0), T_{HD}(0))$, *the set*

$$\{S(t), I_D(t), I_H(t), I_{TD}(t), T_H(t), T_D(t), T_{HD}(t)\}$$

of model(3) is positively invariant for all $t \geq 0$.

Proof

The parameters here are non-negative and hence the system of equations in the mosel are well posed i.e For initial values $(S, I_H, I_D, I_{HD}, T_H, T_D, T_{HD})$ in R_+^7, the solutions exist, are uniqiue and remain in the region for all $t \geq 0$.

when there is no infection,the total population is given by $\dot{N} = \wedge - \mu N$. Here, N(t) approaches a carrying capacity $\frac{\wedge}{\mu}$ for any non-zero initial population size. we set

$$\lambda_H = \frac{\beta_1(I_H + \pi_1 I_{HD} + \pi_2 T_H + \pi_3 T_{HD})}{N}$$

and

$$\lambda_D = \frac{\beta_2(I_D + (1-\pi_1) I_{HD} + \pi_4 T_D + (1-\pi_3) T_{HD})}{N}$$

such that the equations of model (3) take the form

$$\begin{cases} \dot{S} = & \wedge + \gamma_{2d} T_D - \frac{\beta_1(I_H + \pi_1 I_{HD} + \pi_2 T_H + \pi_3 T_{HD})}{N} S I_H - \\ & \frac{\beta_2(I_D + (1-\pi_1) I_{HD} + \pi_4 T_D + (1-\pi_3) T_{HD})}{N} S I_D - \mu S \\ \dot{I}_H = & \frac{\beta_1(I_H + \pi_1 I_{HD} + \pi_2 T_H + \pi_3 T_{HD})}{N} S I_H + \gamma_{1d} T_{HD} - \\ & (\mu + \delta_h + \xi_1 + \omega_1) I_H \\ \dot{I}_D = & \frac{\beta_2(I_D + (1-\pi_1) I_{HD} + \pi_4 T_D + (1-\pi_3) T_{HD})}{N} S I_D \\ & -(\omega_2 + \xi_2 + \mu + \delta_d) I_D \\ \dot{I}_{HD} = & \omega_1 I_H + \omega_2 I_D - (\nu_1 + \nu_2 + \mu + \delta_{hd}) I_{HD} \\ \dot{T}_H = & \nu_1 I_{HD} + \xi_1 I_H - (\alpha_1 + \mu + \delta_h) T_H \\ \dot{T}_D = & \nu_2 I_{HD} + \xi_2 I_D - (\alpha_2 + \mu + \delta_d + \gamma_{2d}) T_D \\ \dot{T}_{HD} = & \alpha_1 T_H + \alpha_2 T_D - (\gamma_{1d} + \delta_{hd} + \mu) T_{HD} \end{cases} \tag{4}$$

The feasible region is

$$\Gamma = \{(S, I_H, I_D, I_{HD}, T_H, T_D, T_H D) \epsilon \Re_+^7 : S \leq N, 0 \leq I_H \leq N, 0 \leq I_D \leq N,$$

$$0 \leq I_{HD} \leq N, 0 \leq T_H \leq N, 0 \leq T_D \leq N, 0 \leq T_{HD} \leq N, N \geq 0\}$$

where $\Re_+^7$ denotes its non-negative cone including it's lower dimensional forces. Clearly, Γ is positively invariant with respect to model (3) and we denote the boundary of Γ by $\partial\Gamma$ and its interior by $\dot{\Gamma}$

3.3.2 Boundedness

Theorem 3.3.2. *The model (1) solutions are uniformly bounded in a set* $L = \{S, I_H, I_D, I_{HD}, T_H, T_H, T_{HD}\} \in R_+^7; (N(0) \leq N(t) \leq \frac{\wedge}{\mu})$

$$\frac{dN}{dt} \leq \frac{dS}{dt} + \frac{dI_H}{dt} + \frac{dI_D}{dt} + \frac{dI_{HD}}{dt} + \frac{dT_H}{dT_D} + \frac{dT_D}{dt} + \frac{dT_{HD}}{dt} \tag{5}$$

$$\frac{dN}{dt} \leq \wedge - \mu N - (\delta_h + \delta_d + \delta_{hd}) \tag{6}$$

When there is no infection in the population,there is no death due to the infection and hence δ_h=δ_d=δ_{hd}=0.

therefore, the RHS of (6) reduces to

$$\frac{dN}{dt} = \wedge - \mu N \text{ and solving for N,} \tag{7}$$

$$\Rightarrow dN = (\wedge - \mu N)dt \tag{8}$$

or

$$\frac{dN}{\wedge - \mu N} = dt \tag{9}$$

by separation of variables.

Integrating both sides of (9) we get

$$\int \frac{dN}{\wedge - \mu N} \leq \int dt \tag{10}$$

$$\Rightarrow -\frac{1}{\mu}\, ln\ |\wedge - \mu N| \leq t + C_1 \tag{11}$$

or

$$ln\ |\wedge - \mu N| \leq -\mu t + C_2, \text{ where } C_2 = -\mu C_1 \tag{12}$$

taking exponential on both sides of (12) we get

$$e^{\,ln\ |\wedge - \mu N|} \leq e^{C_1}.e^{-\mu t} \tag{13}$$

or

$$\wedge - \mu N \leq C\ e^{-\mu t} \tag{14}$$

$$\Rightarrow N \leq \frac{\wedge - Ce^{-\mu t}}{\mu} \tag{15}$$

$$N(t) \leq \frac{\wedge - Ce^{-\mu t}}{\mu} \tag{16}$$

as $t \to \infty$,

$$N(t) \leq \frac{\wedge}{\mu} \tag{17}$$

hence the total population is bounded and is in the feasible

$N(0) \leq N(t) \leq \frac{\wedge}{\mu}$.

Therefore,

$$L = \{(S, I_H, I_D, I_{HD}, T_H, T_D, T_{HD}) \in R_+^7 | 0 \leq S + I_H + I_D + I_{HD} + T_H + T_D + T_{HD} \geq 0, N \leq \frac{\wedge}{\mu}\}$$

and it is clear that L is positively invariant with respect to model (3). We denote the boundary and the interior of L by ∂L and $\mathring{L}$ respectively.

3.3.3 Existence and Stability of Equillibria

3.3.4 Infection-drug free equilibrium Point,E_{HD}

This is the point at which there is no infection or drug use present in the population and its obtained by taking

$$I_H = I_D = I_{HD} = T_H = T_D = T_{HD} = 0$$

hence ; $E_{HD} = (S^o, 0, 0, 0, 0, 0, 0,)$

$$E_{HD} = (\frac{\wedge}{\mu}, 0, 0, 0, 0, 0, 0)$$

3.3.5 Reproduction Number

This is defined as the average number of secondary infections produced when one infected individual is introduced into a host population where everyone is susceptible.

$R_0 = \rho(FV^{-1})$ and $1 \leq j \leq m$ for the infected compartments only. ρFV^{-1} is the spectral radius of a matrix A. F and V are $m \times m$ matrices, where m is the number of infected classes.We use the linearized infected subsystem of model (3) to compute the reproduction number since it is similar to that of the original system Diekmann and Heesterbeek.,(2000). We consider the sub-system below

$$\frac{dI_H}{dt} = \lambda_H S I_H + \gamma_{1d} T_{HD} - A_1 I_H \tag{18}$$

$$\frac{dI_D}{dt} = \lambda_D S I_D - A_2 I_D \tag{19}$$

$$\frac{dI_{HD}}{dt} = \omega_1 I_H + \omega_2 I_D - A_3 I_{HD} \tag{20}$$

$$\frac{dT_H}{dt} = \nu_1 I_{HD} + \xi_1 I_H - A_4 T_H \tag{21}$$

$$\frac{dT_D}{dt} = \nu_2 I_{HD} + \xi_2 I_D - A_5 T_D \tag{22}$$

$$\frac{dT_{HD}}{dt} = \alpha_1 T_H + \alpha_2 T_D - A_6 T_{HD} \tag{23}$$

where

$A_1 = \mu + \delta_h + \xi_1 + \omega_1$

$A_2 = \omega_2 + \xi_2 + \delta_d + \mu,$

$A_3 = \nu_1 + \nu_2 + \delta_{hd} + \mu,$

$A_4 = \alpha_1 + \delta_h + \mu$,

$A_5 = \alpha_2 + \delta_d + \gamma_{2d} + \mu$,

$A_6 = \gamma_{1d} + \delta_{hd} + \mu$

We decompose the RHS of the infected subsystem into two parts $\mathcal{F}$ and $\mathcal{V}$

where $\mathcal{F}=[\frac{\partial F_i(x_o)}{\partial x_j}]$

and

$\mathcal{V}=[\frac{\partial V_i(x_o)}{\partial x_j}]$

the matrix of the new infection

$$\mathcal{F} = \begin{bmatrix} \lambda_H S \\ \lambda_D S \\ o \\ o \\ o \\ o \end{bmatrix}$$

$$\mathcal{F} = \begin{bmatrix} \frac{S}{N}(\beta_1(I_D + \pi_1 I_{HD} + \pi_2 T_H + \pi_3 T_H D) \\ \frac{S}{N}(\beta_2(I_D + (1-\pi_1) I_H D + \pi_4 T_D + (1-\pi_3) T_{HD})) \\ o \\ o \\ o \\ o \end{bmatrix}$$

We obtain the Jacobian of $\mathcal{F}$ with respect to
$I_H, I_D, I_{HD}, T_H, T_D, T_{HD}$ and at Disease free equilibrium,

$$F = \begin{bmatrix} \beta_1 & 0 & \beta_1\pi_1 & \beta_1\pi_2 & 0 & \beta_1\pi_3 \\ 0 & \beta_2 & \beta_2(1-\pi_1) & 0 & \beta_2(1-\pi_4) & \beta_2(1-\pi_3) \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & o & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

and transfer of infection

$$\mathcal{V} = \begin{bmatrix} -\gamma_{1d}T_{HD} + A_1 I_H \\ A_2 I_D \\ -\omega_1 I_H - \omega_2 I_D + A_3 I_{HD} \\ -\nu_1 I_{HD} - \xi_1 I_H + A_4 T_H \\ -\nu_2 I_{HD} - \xi_2 I_D + A_5 T_D \\ -\alpha_1 T_H - \alpha_2 T_D + A_6 T_{HD} \end{bmatrix}$$

We obtain the Jacobian of $\mathcal{V}$ with respect to $I_H, I_D, I_{HD}, T_H, T_D, T_{HD}$ and at Disease free equilibrium,

$$V = \begin{bmatrix} A_1 & 0 & 0 & 0 & 0 & -\gamma_{1d} \\ 0 & A_2 & 0 & 0 & 0 & 0 \\ -\omega_1 & -\omega_2 & A_3 & 0 & 0 & 0 \\ -\xi_1 & o & -\nu_1 & A_4 & 0 & 0 \\ 0 & -\xi_2 & -\nu_2 & 0 & A_5 & 0 \\ 0 & 0 & 0 & -\alpha_1 & -\alpha_2 & A_6 \end{bmatrix}$$

Where

$$
\begin{aligned}
A_1 &= \mu + \delta_h + \xi_1 + \omega_1 \\
A_2 &= \nu_1 + \nu_2 + \mu + \delta_{hd} \\
A_3 &= \alpha_1 + \mu + \delta_h \\
A_4 &= \gamma_{1d} + \delta_{hd} + \mu \\
A_5 &= \omega_1 + \omega_2 + \mu + \delta_d \\
A_6 &= \alpha_2 + \mu + \delta_d + \gamma_{2d}
\end{aligned}
$$

$$
FV^{-1} = \begin{bmatrix}
\frac{y_1}{y_2} & \frac{y_3}{y_4} & \frac{y_5}{y_6} & \frac{y_7}{y_8} & \frac{y_9}{y_{10}} & \frac{y_{11}}{y_{12}} \\
\frac{y_{13}}{y_{14}} & \frac{y_{15}}{y_{16}} & \frac{y_{17}}{y_{18}} & \frac{y_{19}}{y_{20}} & \frac{y_{21}}{y_{22}} & \frac{y_{23}}{y_{24}} \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0
\end{bmatrix}
$$

where

$$
\frac{y_1}{y_2} = \frac{\beta_1(A_3A_5(A_4A_6x_1 + A_6\pi_1 + \pi_3\alpha_1)\xi_1}{A_3A_5(A_1A_4A_6 - \alpha_1\gamma_{1d}\xi_1)}
$$

$$
\frac{y_3}{y_4} = \frac{(\beta_1(\alpha_2\xi_2(A_1A_3A_4\pi_3(A_4(A_1\pi + \gamma_{1d}(A_3(A_4 + \pi_1\xi_1)))))}{(A_2A_3A_5(A_1A_4A_6 - \alpha_1\gamma_{1d}\xi_1)}
$$

$$
\frac{y_5}{y_6} = \frac{\beta_1(A_1(A_5(A_6\pi_1 + \pi_3\alpha_1)\nu_1 + A_4(A_5A_6\pi_1 + \pi_3\alpha_2\nu_2))}{A_3A_5(A_1A_4A_6 - \alpha 1d) - \gamma 1d(A_5\alpha_1\nu_1 - A_4\alpha_2\nu_2)\omega_1}
$$

$$
\frac{y_7}{y_8} = \frac{\beta_1(A_3A_5(A_1(A_6\pi_1 + \pi_3\alpha_1) + \alpha_1\gamma_{1d}) + \pi_1\gamma_{1d}(A_5\alpha_1 - \alpha_2\nu_2)\omega_1)}{A_3A_5(A_1A_4A_6 - \alpha_1\gamma_{1d}\xi_1)}
$$

$$
\frac{y_9}{y_{10}} = \frac{\alpha_2\beta_1(A_1A_3A_4\pi_3 + \gamma_{1d}(A_3(A_4 + \pi_1\xi_1) + \pi_1(A_4 + \nu_1)\omega_1))}{A_3A_5(-A_1A_4A_6 + \alpha_1\gamma_{1d}\xi_1)}
$$

$$
\frac{y_{11}}{y_{12}} = \frac{A_5\beta_1(A_1A_3A_4\pi - 3 + \gamma_{1d}(A_3(A_4 + \pi_1\xi_1) + \pi_1(A_4 + \nu_1)\omega_1))}{A_3A_5(A_1A_4A_6 - \alpha_1\gamma_{1d}\xi_1)}
$$

$$\frac{y_{13}}{y_{14}} = \frac{\beta_2(A_2A_3(-1+\pi_3)\alpha_1\xi_1 + (A_5(-1+\pi_3)\alpha_1\nu_1 + A_4(A_5A_6(-1+\pi_1)}{A_3A_5(-A_1A_4A_6+\alpha_1\gamma_{1d}\xi_1) - \gamma_{1d}(A_5\alpha_1\nu_1 + A_4\alpha_2\nu_2)\omega_1}$$

$$\frac{y_{15}}{y_{16}} = \frac{(\beta_2(A_1(A_3A_4(A_5A_6 + (A_6 - A_6\pi_4 + \alpha_2 - \pi_3\alpha_2)\xi_2)}{A_3A_5(A_1A_4A_6-\alpha_1\gamma_{1d}\xi_1) - \gamma_{1d}(A_5\alpha_1\nu_1 + A_4\alpha_2\nu_2)\omega_1}$$

$$\frac{y_{17}}{y_{18}} = \{-\beta_2(A_1(A_5(-1+\pi_3)\alpha_1\nu_1 + A_4(A_5A_6(-1+\pi_1) + (A - 6(-1+\pi_4) + (-1+\pi_3)\alpha_2)\nu_2))\}/\{A_3A_5(A_1A_4A_6-\alpha_1\gamma_{1d}\xi_1) - \gamma_{1d}(A_5\alpha_1\nu_1 + A_4\alpha_2\nu_2)\}$$

$$\frac{y_{19}}{y_{20}} = \frac{\alpha_1\beta_2(A_1A_3A_5(-1+\pi_3) + \gamma_{1d}(A_5(-1+\pi_1) + (-1+\pi_4)\pi_2)\omega_1}{A_2A_3A_5(A_1A_4A_6-\alpha_1\gamma_{1d}\xi_1) - \gamma_{1d}(A_5\alpha_1\nu_1 + A_4\alpha_2\nu_2)\omega_1}$$

$$\frac{y_{21}}{y_{22}} = \frac{\beta_2(A_1A_3A_4(-A_6(-1+\pi_4) - (-1+\pi_3)\alpha_2)}{A_3A_5(A_1A_4A_6-\alpha_1\gamma_{1d}\xi_1) - \gamma_{1d}(A_5\alpha_1\nu_1 + A_4\alpha_2\nu_2)\omega_1}$$

$$\frac{y_{23}}{y_{24}} = -\frac{\beta_2A_4(A_1A_3A_5(-1+\pi_3) + \gamma_{1d}(A_5(-1+\pi_1) + (-1+\pi_4)\nu_2)\omega_1)}{A_3A_5(A_1A_4A_6-\alpha_1\gamma_{1d}\xi_1) - \gamma_{1d}(A_5\alpha_1\nu_1 + A_4\alpha_2\nu_2)\omega_1}$$

Eigenvalues of FV^{-1} are

$$\lambda_1 = 0$$
$$\lambda_2 = 0$$
$$\lambda_3 = 0$$
$$\lambda_4 = 0$$
$$\lambda_5 = \frac{-\sqrt{A_1^2\xi_1^2 - 2A_1A_2\xi_2\omega_1 + A_2^2\omega_1^2 + 4A_1A_2\nu_1\omega_2} + A_1\xi_1 + A_2\omega_1}{2A_1A_2A_3A_5(A_1A_4A_6-\alpha_1\gamma_{1d}\xi_1) - \gamma_{1d}(A_5\alpha_1\nu_1 + A_4\alpha_2\nu_2)\omega_1}$$
$$\lambda_6 = \frac{+\sqrt{A_1^2\xi_1^2 - 2A_1A_2\xi_2\omega_1 + A_2^2\omega_1^2 + 4A_1A_2\nu_1\omega_2} + A_1\xi_1 + A_2\omega_1}{2A_1A_2A_3A_5(A_1A_4A_6-\alpha_1\gamma_{1d}\xi_1) - \gamma_{1d}(A_5\alpha_1\nu_1 + A_4\alpha_2\nu_2)\omega_1}$$

The dominant eigenvalue is λ_6 which is the basic reproduction number and hence $\mathcal{R}_{\mathcal{HD}}=$

$$\frac{+\sqrt{A_1^2\xi_1^2 - 2A_1A_2\xi_2\omega_1 + A_2^2\omega_1^2 + 4A_1A_2\nu_1\omega_2} + A_1\xi_1 + A_2\omega_1}{2A_1A_2A_3A_5(A_1A_4A_6-\alpha_1\gamma_{1d}\xi_1) - \gamma_{1d}(A_5\alpha_1\nu_1 + A_4\alpha_2\nu_2)\omega_1}$$

3.3.6 Local stability of the E_{HD}

We establish E_{HD} point i,e when there is no infection, the system has a constant solution, $E_{HD}= (\frac{\wedge N}{\mu}, 0, 0, 0, 0, 0, 0)$.

Theorem 3.3.3. *The infection-drug free equilibrium , E_{HD} is locally asymptomatically stable if $R_{HD} < 0$.*

proof

To establish this stability, we compute the Jacobian of the system at $\boldsymbol{E_{HD}}$. The stability of $\boldsymbol{E_{HD}}$ is determined by the signs of the eigenvalues of the Jacobian matrix.

$$J(E_o) = \begin{bmatrix} -\mu & 0 & 0 & 0 & 0 & \gamma_{2d} & 0 \\ 0 & -A_1 & 0 & 0 & 0 & 0 & \gamma_{1d} \\ 0 & 0 & -A_2 & 0 & 0 & 0 & 0 \\ 0 & \omega_1 & \omega_2 & -A_3 & 0 & 0 & 0 \\ 0 & \xi_1 & 0 & \nu_1 & -A_4 & 0 & 0 \\ 0 & 0 & \xi_2 & \nu_2 & 0 & -A5 & 0 \\ 0 & 0 & 0 & 0 & \alpha_1 & \alpha_2 & -A_6 \end{bmatrix}$$

$|J_{E_o} - XI| = 0$ as follows

$$J = \begin{vmatrix} -\mu - X & 0 & 0 & 0 & 0 & \gamma_{2d} & 0 \\ 0 & A_1 - X & 0 & 0 & 0 & 0 & \gamma_{1d} \\ 0 & 0 & A_2 - X & 0 & 0 & 0 & 0 \\ 0 & \omega_1 & \omega_2 & A_3 - X & 0 & 0 & 0 \\ 0 & \xi_1 & 0 & \nu_2 & A_4 - X & 0 & 0 \\ 0 & 0 & \xi_2 & \nu_2 & 0 & A5 - X & 0 \\ 0 & 0 & 0 & 0 & \alpha_1 & \alpha_2 & A_6 - X \end{vmatrix}$$

The characteristic polynomial of $\boldsymbol{J_{Eo}}$ is

$$(X-\mu)(-X-A_2)[(X^2+A_1X+A_3X+A_1A_3)(-X-A_5)(-X-A_6)+\gamma_{1d}(-(-X-A_5)$$

$$\alpha_1(X\xi_1+A_3\xi_1+\nu_1\omega_1)+\alpha_2(X\mu_2\omega_1+A_4\nu_2\omega_1)]$$

Notice that from column (1), $(-\mu - X)$ can be factored straight away, and also $X_2 = -A_2$. The other four remaining eigenvalues are obtained by solving the reduced polynomial below:

$$[(X^2+A_1X+A_3X+A_1A_3)(-X-A_5)(-X-A_6)+\gamma_{1d}(-(-X-A_5))] \tag{24}$$

and using the Routh-Hurwitz criterion, the other roots are obtained

Lemma 3.3.4. *The infection-drug free equilibrium point* $(\boldsymbol{E_{HD}})$ *is stable iff all the elements appearing in column one are all positive otherwise unstable (Routh's.,1874).*

Proof

By constructing the Routh-Hurwitz array for equation (24) and examining the elements of the column one,being negative, the system is considered unstable.

3.3.7 Global stability analysis of E_{HD}

The Global stability of infection-drug free equilibrium point $(\boldsymbol{E_{HD}})$ is investigated using the Lyapunov function for some positive parameters, $\boldsymbol{p_i}$,
$\boldsymbol{where} \quad \boldsymbol{i = 1,2,3,4,5} \ \boldsymbol{and} \ \boldsymbol{6}$.

$$\boldsymbol{V(S, I_H, I_D, I_{HD}, T_H, T_H, T_{HD}) = \left(S - S^o - S^o ln\left(\frac{S}{S^o}\right)\right)}$$

$$\boldsymbol{+p_1\left(I_H - I_H^o - I_H^o ln\left(\frac{I_H}{I_H^o}\right)\right) + p_2\left(I_D - I_D^o - I_H^o ln\left(\frac{I_D}{I_D^o}\right)\right)}$$

$$\boldsymbol{+p_3\left(I_{HD} - I_{HD}^o - I_{HD}^o ln\left(\frac{I_{HD}}{I_{HD}^o}\right)\right) + p_4\left(T_H - T_H^o - T_H^o ln\left(\frac{T_H}{T_H^o}\right)\right)}$$

$$\boldsymbol{+ P_5 T_D + P_6 T_{HD}.} \qquad \textbf{(25)}$$

satisfying the following conditions

$$\boldsymbol{1. V(S^o, I_H^o, I_D^o, I_{HD}^o, T_H^o, T_H^o, T_{HD}^o) = 0}$$
$$\boldsymbol{2. V(S, I_H, I_D, I_{HD}, T_H, T_H, T_{HD}) > 0}$$
$$\boldsymbol{3. \frac{dL}{dt}(S^o, I_H^o, I_D^o, I_{HD}^o, T_H^o, T_H^o, T_{HD}^o) = 0}$$
$$\boldsymbol{4. \frac{dl}{dt}(S, I_H, I_D, I_{HD}, T_H, T_H, T_{HD}) < 0}$$

Proof

Condition (1)

$$\boldsymbol{V(S^o, I_H^o, I_D^o, I_{HD}^o, T_H^o, T_D^o, T_{HD}^o) = \left(S^o - S^o - S^o ln\left(\frac{S^o}{S^o}\right)\right)}$$

$$+p_1\left(I_H^o - I_H^o - I_H^o ln\left(\frac{I_H^o}{I_H^o}\right)\right) + p_2\left(I_D^o - I_D^o - I_H^o ln\left(\frac{I_D^o}{I_D^o}\right)\right)$$
$$+p_3\left(I_{HD}^o - I_{HD}^o - I_{HD}^o ln\left(\frac{I_{HD}^o}{I_{HD}^o}\right)\right) + p_4\left(T_H^o - T_H^o - T_H^o ln\left(\frac{T_H^o}{T_H^o}\right)\right)$$
$$+P_5 T_D^o + P_6 T_{HD}^o = 0$$

.

and hence the proof

Condition (2)

the derivative of V with respect to t is,

$$\frac{dV(S^o, I_H^o, I_D^o, I_{HD}^o, T_H^o, T_D^o, T_{HD}^o)}{dt} = \left(1 - \frac{S^o}{S^o}\right)\frac{dS^o}{dt} + p_1\left(1 - \frac{I_H^o}{I_H^o}\right)\frac{dI_H^o}{dt}$$
$$+ p_2\left(1 - \frac{I_D^o}{I_D^o}\right)\frac{dI_D^o}{dt} + p_3\left(1 - \frac{I_{HD}^o}{I_{HD}^o}\right)\frac{dI_{HD}^o}{den}$$
$$+ p_4\left(1 - \frac{T_H^o}{T_H^o}\right)\frac{dT_H^o}{dt} + P_5\frac{dT_D^o}{dt} + P_6\frac{dT_{HD}^o}{dt}.$$

since $I_H^o = I_D^o = I_{HD}^o = T_H^o = T_D^o = T_{HD}^o$)=0 and $S^o = \frac{\wedge}{\mu}$, the time derivative of the Lyapuov function V at infection-drug free equilibrium point reduces to

$$\frac{dV(S^o, I_H^o, I_D^o, I_{HD}^o, T_H^o, T_D^o, T_{HD}^o)}{dt} = \left(1 - \frac{S^o}{S^o}\right)\frac{dS_o}{dt}$$

and since $1 - \frac{S_o}{S_o} = 0$, then $\frac{dS_o}{dt} = 0$

Hence

$$\frac{dV(S^o, I_H^o, I_D^o, I_{HD}^o, T_H^o, T_D^o, T_{HD}^o)}{dt} = 0$$

Similarly,

$$\frac{dV(S, I_H, I_D, I_{HD}, T_H, T_D, T_{HD})}{dt} = \left(1 - \frac{S^o}{S}\right)\frac{dS}{dt} + p_1\left(1 - \frac{I_H^o}{I_H}\right)\frac{dI_H}{dt}$$

$$+p_2\left(1-\frac{I_D^o}{I_D}\right)\frac{dI_D}{dt}+p_3\left(1-\frac{I_{HD}^o}{I_{HD}}\right)\frac{dI_{HD}}{dt}+p_4\left(1-\frac{T_H^o}{T_H}\right)\frac{dT_H}{dt}$$
$$+P_5\frac{dT_D}{dt}+P_6\frac{dT_{HD}}{dt}.$$

replacing for

$\frac{dS}{dt}, \frac{dI_H}{dt}, \frac{dI_D}{dt}, \frac{dI_{HD}}{dt}, \frac{dT_D}{dt}, \frac{dT_H}{dt}$ and $\frac{dT_{HD}}{dt}$, we get

$$\begin{aligned}\frac{dV(S,I_H,I_D,I_{HD},T_H,T_D,T_{HD})}{dt} &= \left(1-\frac{S^o}{S}\right)(\wedge+\gamma_{2d}T_D-\lambda_H SI_H-\lambda_D SI_D-\mu S)\\ &+p_1\left(1-\frac{I_H^o}{I_H}\right)(\lambda_H SI_H+\gamma_{1d}T_{HD}-A_1I_H)\\ &+p_2\left(1-\frac{I_D^o}{I_D}\right)(\lambda_D SI_D-A_2I_D)\\ &+p_3\left(1-\frac{I_{HD}^o}{I_{HD}}\right)(\omega_1I_H+\omega_2I_D-A_3I_{HD})\\ &+p_4\left(1-\frac{T_H^o}{T_H}\right)(\nu_1I_{HD}+\xi_1I_H-A_4T_H)\\ &+P_5(\nu_2I_{HD}+\xi_2I_D-A_5T_D)\\ &+P_6(\alpha_1T_H+\alpha_2T_D-A_6T_{HD})\end{aligned}$$

expanding and simplifying yields,

$$\begin{aligned}\frac{dV(S,I_H,I_D,I_{HD},T_H,T_D,T_{HD})}{dt} &= S^o\mu S-S^o\wedge+S\wedge-S^2\mu+(-S^o+S)T_D\gamma_{2d}\\ &+(S^o-S)SI_D\lambda D+S^oSI_H\lambda_H-S^2I_H\lambda_H\\ &+SP_1I_H\lambda_H+P_1T_{HD}\gamma_{1d}-a_1P_1I_H-SI_H^oP_1\lambda_H\\ &-\frac{P_1T_{HD}I_H^o\gamma_{1d}}{I_H}+P_1A_1I_H^o+I_D^oA_2P_2-I_DA_2P_2\\ &-I_D^oSP_2\lambda_D+I_DSP_2\lambda_D-I_{HD}A_3P_3+I_{HD}^oA_3P_3\\ &+P_3I_H\omega_1-I_{HD}\frac{P_3I_H\omega_1}{I_{HD}}+I_DP_3\omega_2-\frac{I_{HD}^oI_DP_3\omega_2}{I_{HD}}\\ &+T_H^oA_4P_4-A_4P_4T_H+I_{HD}P_4\nu_1-\frac{T_H^oI_{HD}P_4\nu_1}{T_H}\\ &+P_4I_H\xi_1-\frac{T_H^oP_4I_H\xi_1}{T_H}+P_5\nu_2I_{HD}+P_5\xi_2I_D\\ &-P_5A_5T_D+P_6\alpha_1T_H+P_6\alpha_2T_D-P_6A_6T_{HD}\end{aligned}$$

At infecton-drug free equilibrium point,

$$I_H^o = I_D^o = I_{HD}^o = T_H^o = T_D^o = T_{HD}^o) = 0$$

reducing the equation to

$$\frac{dV(S, I_H, I_D, I_{HD}, T_H, T_D, T_{HD})}{dt} =$$
$$\frac{S^o\mu S - S^o \wedge + S \wedge - S^2\mu + (-S^o + S)T_D\gamma_{2d} + (S^o - S)SI_D\lambda D + S^oSI_H\lambda_H - S^2I_H\lambda_H}{S}$$
$$+ SP_1I_H\lambda_H - A_4P_4T_H + I_{HD}P_4\nu_1 + P_4I_H\xi_1 + P_5\nu_2I_{HD} + P_5\xi_2I_D + I_D - P_5A_5T_D$$
$$+ P_6\alpha_1T_H + P_6\alpha_2T_D - P_6A_6T_{HD}$$

$$= 2\wedge - \frac{\wedge^2}{\mu S} + \frac{(S - \frac{\wedge}{\mu})T_D\gamma_{2d}}{S} + (\frac{\wedge}{\mu} - S)I_D\lambda_D + \frac{\wedge}{\mu}I_H\lambda_D + SI_H\lambda_H + SP_1I_H\lambda_H$$
$$+ P_1T_{HD}\gamma_{1d} - A_1P_1I_H - I_DA_2P_2 + I_DSP_2\lambda_D - I_HA_3P_3 + P_3I_H\omega_1 + I_DP_3\omega_2 - A_4P_4T_H$$
$$+ I_{HD}P_4\nu_1 + P_4I_H\xi_1 + P_5\nu_2I_{HD} + P_5\xi_2I_D - P_5A_5T_D + P_6\alpha_1T_H + P_6\alpha_2T_D - P_6A_6T_{HD}$$

Grouping the non-linear terms on the RHS

$$\begin{aligned}\frac{dV(S, I_H, I_D, I_{HD}, T_H, T_D, T_{HD})}{dt} &= 2\wedge - \frac{\wedge^2}{\mu S} - S\mu + (P_4 - 1)SI_H\lambda_H \\ &+ (P_2 - 1)SI_D\lambda_D + (P_3\omega_1 + P_4\xi_1 - A_1P_1)I_H \\ &+ (P_3\omega_2 + P_5\xi_2 - A_2P_2)I_D \\ &+ (P_4\nu_1 + P_5\nu_2 - A_3P_3)I_{HD} + \\ &(P_6\alpha_1 - A_4P_4)T_H \\ &+ (P_6\alpha_2 + \gamma_{2d} - \frac{\wedge}{\mu S}\gamma_{2d} - P_5A_5)T_D \\ &+ (P_1\gamma_{1d} - P_6A_6)T_{HD} + \frac{\wedge}{\mu}I_{HD\lambda_H} + \frac{\wedge}{\mu}I_D\lambda_D\end{aligned}$$

Equating the non-linear terms to zero we get,

$$P_1 = 1,\ P2 = 1,\ P_3 = \frac{A_1}{\omega_1} - \frac{\gamma_{1d}}{\omega_1A_4A_6}\alpha_1\xi_1,\ P_4 = \frac{\gamma_{1d}}{A_4A_6}$$
$$P_5 = \frac{A_2}{\xi_2} + \frac{\gamma_{1d}}{\omega_1\xi_1A_4A_6}\alpha_1\xi_1 - \frac{A_1}{\omega_1\xi_2},\ P_6 = \frac{\gamma_{1d}}{A_6}$$

Since

$$2 \wedge -\frac{\wedge^2}{\mu} S - S\mu < 0$$

thus ,

$$\frac{dV(S, I_H, I_D, I_{HD}, T_H, T_D, T_{HD})}{dt} < 0$$

hence the two conditions are satisfied for infection-free equilibrium point to be GAS

all parameters being non-positive, then $\dot{V} \leq 0$ for $R_{HD} < 1$

therefore,V is a Lyapunov function on L, and the largest compact invariant set in

$\{(S, I_H, I_D, I_{HD}, T_H, T_D, T_{HD}) \in L : \dot{V} = 0\}$ is the singleton $\{E_{HD}\}$. Therefore, by LaSalle's Invariance Principle(LaSalle.,1976), every solution of model (1), with initial conditions in L, approaches E_{HD} as $t \to \infty$, whenever $R_o \leq 0$. This completes the proof.

3.3.8 Endemic Equillibrium Point,E^*

This is usually when the infection is present in the human population. It's obtained when atleast one of the infected components is not equal to zero. For model(1) they are given by
$E^* = (S^*, I_H^*, I_D^*, I_{HD}^*, T_H^*, T_D^*, T_{HD}^*)$
For E^* *to exist*,
$S^* > 0, I_H^* > 0, I_D^* > 0, I_{HD}^* > 0, T_H^* > 0, T_D^* > 0, T_{HD}^* > 0$

We set the RHS of system of differential equations (1) to zero and obtain

$$\wedge + \gamma_{2d} T_D^* - \lambda_H S^* I_H^* - \lambda_D S^* I_D^* - \mu S^* = 0 \quad (26)$$
$$\lambda_H S^* I_H^* + \gamma_{1d} T_{HD}^* - A_1 I_H^* = 0 \quad (27)$$
$$\lambda_D S^* I_D^* - A_2 I_D^* = 0 \quad (28)$$
$$\omega_1 I_H^* + \omega_2 I_D^* - A_3 I_{HD}^* = 0 \quad (29)$$
$$\nu_1 I_{HD}^* + \xi_1 I_H^* - A_4 T_H^* = 0 \quad (30)$$
$$\nu_2 I_{HD}^* + \xi_2 I_D^* - A_5 T_D^* = 0 \quad (31)$$
$$\alpha_1 T_H^* + \alpha_2 T_D^* - A_6 T_{HD}^* = 0 \quad (32)$$

solving for S^*, I_H^*, I_D^*, I_{HD}^*, T_H^*, T_D^* and T_{HD}^* and using Mathematica software we obtain

$$S^* = \frac{N A_2 A_5}{\beta_2 (A_5 + \pi_4 \xi_2)}$$

$$I_H^*=\frac{A_4(A_4\wedge\beta_1\xi_1+\wedge\pi_2\beta_1\xi_1^2-K_2)}{2\xi_1K_4}$$

$$I_D^*=\frac{-A^2\beta_1\xi_1\wedge+A_4A_5\beta_1\xi_1\wedge-A_4\pi_2\beta_1\xi_1\wedge+A_5\pi_2\beta_1\xi_1\wedge+K_3+K_4+\frac{A_5(-\gamma_{2d}+K_5)}{K_6}}{\xi_1}$$

$$I_{HD}^*=\frac{-A_4\beta 1\xi_1\wedge+A_4A_5\beta_1\xi_1^2\wedge+A_5\pi_2\beta_1\xi_1^2+A_4K_7+A_5K_8}{2A_1A_4\beta_1\nu_1(A_4+\pi_2\xi_1)}$$

$$T_H^*=\frac{A_4\beta_1\xi_1\wedge+\pi_2\beta_1\xi_1^*K_9}{2K_{10}}$$

$$T_D^*=\frac{-\gamma_{2d}K_{11}}{2K_{12}}$$

$$T_{HD}^*=\frac{-\alpha_1(\frac{A_4\beta_1\xi_1\wedge+\pi_2\beta_1\xi_1^2\wedge+K_{13}-\frac{\alpha_2(-\gamma_{2d}+K_{14})}{2K_{15}}}{K_{16}})}{A_6}$$

where

$$K_1=\sqrt{4N\mu A1\xi_1^2(-A_1A_4^2\beta_1-A_1A_4\pi_2\beta_1\xi_1)+(\wedge A_4\beta_1\xi_1+\wedge\pi_2\beta_1\xi_1)^2}$$

$$K_2=A_1A_4^2\beta_1-A_1A_4\pi_2\beta_1\xi_1,$$

$$K_3=A_4\sqrt{\beta_1\xi_1^2(A_4+\pi_2\xi_1)(-4\mu A_1^2A_4+\wedge^2A_4\beta_1+\wedge^2\pi_2\beta_1\xi_1)}$$

$$K_4=A_5\sqrt{\beta_1\xi_1^2(A_4+\pi_2\xi_1)(-4N\mu A_1^2A_4+\wedge^2A_4\beta_1+\wedge^2\pi_2\beta_1\xi_1)}$$

$$K_5=\sqrt{\gamma_{2d}^2-4\left(\wedge\frac{N\mu A_2A_5}{\beta_2(A_5+\pi_4\xi_2)}\right)\left(\frac{-A_2A_5^3}{\xi_2^2(A_5+\pi_4\xi_2)}-\frac{A_2A_5^2\pi_4}{\xi_2(A_5+\pi_4\xi_2)}\right)},$$

$$K_6=2\left(-\frac{A_2A_5^3}{\xi_2^2(A_5+\pi_4\xi_2)}-\frac{A_2A_5^2\pi_4}{\xi_2(A_5+\pi_4\xi_2)}\right)$$

$$K_7=\sqrt{\beta_1\xi_1^2(A_4+\pi_2\xi_1)(-4N\mu A_1^2A_4+\wedge^2A_4\beta_1+\wedge^2A_4\beta_1+\wedge^2\pi_2\beta_1\xi_1)}$$

$$K_8=\sqrt{\beta_1\xi_1^2(A_4+\pi_2\xi_1)(-4N\mu A_1^2A_4+\wedge^2A_4\beta_1+\wedge^2\pi_2\beta_1\xi_1)}$$

$$K_9=\sqrt{4N\mu\xi_1^2A_1(-A_1A_4^2\beta_1-A_1A_4\pi_2\beta_1\xi_1+(\wedge A_4\beta_1\xi_1+\wedge\pi_2\beta_1\xi_1^2)^2}$$

$$K_{10} = A_1 A_4^2 \beta_1 + A_1 A_4 \pi_2 \beta_1 \xi_1,$$

$$K_{11} = \left(\wedge \frac{N\mu A_2 A_5}{\beta_2 (A_5 + \pi_4 \xi_2)}\right) \left(\frac{A_2 A_5^3}{\xi_2^2 (A_5 + \pi_4 \xi_2)} - \frac{A_2 A_5^2 \pi_4}{\xi_2 (A_5 + \pi_4 \xi_2)}\right)$$

$$K_{12} = \left(-\frac{A_2 A_5^2}{\xi_2^2 (A_5 + \pi_4 \xi_2)} - \frac{A_2 A_5^2 \pi_4}{\xi_2 (A_5 + \pi_4 \xi_2)}\right)$$

$$K_{13} = \sqrt{4N\mu A_1 \xi_1^2 (-A_1 A_4 \beta_1 - A_1 A_4 \pi_2 \beta_1 \xi_1) + (\wedge A_4 \beta_1 \xi_1 + \wedge \pi_2 \beta_1 \xi_1^2)^2}$$

$$K_{14} = 2\left(A_1 A_4^2 \beta_1 + A_1 A_4 \pi_2 \beta_1 \xi_1\right),$$

$$K_{15} = 4\left(\wedge \frac{N\mu A_2 A_5^3}{\beta_2 (A_5 + \pi_4 \xi_2}\right)\right) \left(\frac{-A_2 A}{\xi_2^2 (A_5 + \pi_4 \xi_2)} - \frac{A_2 A_5^2 \pi_4}{\xi_2 (A_5 + \pi_4 \xi_2)}\right)$$

$$K_{16} = \left(\frac{-A_2 A_5^3}{\xi_2^2 (A_5 + \pi_4 \xi_2)} - \frac{A_2 A_5^2 \pi_4}{\xi_2 (A_5 + \pi_4 \xi_2)}\right)$$

it is observed that the model has one endemic state and therefore a forward bifurcation exists.

3.3.9 Global stability of drug endemic equilibrium

Theorem 3.3.5. *If R_o >1, then the infection-drug endemic equilibrium is globally asymptotically stable.*

Proof

By considering a Lyapunov logarithmic function defined by;

$$\begin{aligned} L(S^*, I_H^*, I_D^*, I_{HD}^*, T_H^*, T_D^*, T_{HD}^*) = & \left(S - S^* - S^* ln\left(\frac{S}{S^*}\right)\right) \\ & + \left(I_H - I_H^* - I_H^* ln\left(\frac{I_H}{I_H^*}\right)\right) \\ & + \left(I_D - I_D^* - I_H^* ln\left(\frac{I_D}{I_D^*}\right)\right) \\ & + \left(I_{HD} - I_{HD}^* - I_{HD}^* ln\left(\frac{I_{HD}}{I_{HD}^*}\right)\right) \\ & + \left(T_H - T_H^* - T_H^* ln\left(\frac{T_H}{T_H^*}\right)\right) \\ & + \left(T_D - T_D^* - T_D^* ln\left(\frac{T_D}{T_D^*}\right)\right) \\ & + \left(T_{HD} - T_{HD}^* - T_{HD}^* ln\left(\frac{T_{HD}}{T_{HD}^*}\right)\right) \end{aligned}$$

computing the derivative of L along the solutions of the system of equations directly;

$$\begin{aligned} \frac{dL}{dt} = & \left(1 - \frac{S}{S^*}\right)\frac{dS^*}{dt} + \left(1 - \frac{I_H}{I_H^*}\right)\frac{dI_H^*}{dt} + \left(1 - \frac{I_D}{I_D^*}\right)\frac{dI_D^*}{dt} \\ & + \left(1 - \frac{I_{HD}}{I_{HD}^*}\right)\frac{dI_{HD}^*}{dt} + \left(1 - \frac{T_H}{T_H^*}\right)\frac{dT_H^*}{dt} \\ & + \left(1 - \frac{T_D}{T_D^*}\right)\frac{dT_D^*}{dt} + \left(1 - \frac{T_{HD}}{T_{HD}^*}\right)\frac{dT_{HD}^*}{dt} \end{aligned}$$

substituting for

$$\frac{dS^*}{dt}, \frac{dI_H^*}{dt}, \frac{dI_D^*}{dt}, \frac{dI_{HD}^*}{dt}, \frac{dT_D^*}{dt}, \frac{dT_H^*}{dt} \text{ and } \frac{dT_{HD}^*}{dt}$$

$$\frac{dL}{dt} = \left(1 - \frac{S}{S^*}\right)(\wedge + \gamma_{2d} T_D^* - \lambda_H S^* I_H^* - \lambda_D S^* I_D^* - \mu S^*)$$
$$+ \left(1 - \frac{I_H}{I_H^*}\right)(\lambda_H S^* I_H^* + \gamma_{1d} T_{HD}^* - A_1 I_H^*)$$
$$+ \left(1 - \frac{I_D}{I_D^*}\right)(\lambda_D S^* I_D^* - A_2 I_D^*)$$
$$+ \left(1 - \frac{I_{HD}}{I_{HD}^*}\right)(\omega_1 I_H^* + \omega_2 I_D^* - A_3 I_{HD}^*)$$
$$+ \left(1 - \frac{T_H}{T_H^*}\right)(\nu_1 I_{HD}^* + \xi_1 I_H^* - A_4 T_H^*)$$
$$+ \left(1 - \frac{T_D}{T_D^*}\right)(\nu_2 I_{HD}^* + \xi_2 I_D^* - A_5 T_D^*)$$
$$+ \left(1 - \frac{T_{HD}}{T_{HD}^*}\right)(\alpha_1 T_H^* + \alpha_2 T_D^* - A_6 T_{HD}^*)$$

Expanding and simplifying,

$$\frac{dL}{dt} = \wedge + \gamma_{2d} T_D^* - \lambda_H S^* I_H^* - \lambda_D S^* I_D^* - \mu S - \frac{S}{S^*} \wedge - \gamma_{2d} T_D^* \frac{S}{S^*} + \lambda_H I_H^* S$$
$$+ \lambda_D I_D^* S + \mu S + \lambda_H S^* I_H^* + \gamma_{1d} T_{HD}^* - A_1 I_H^* - \lambda_H S^* I_H$$
$$- \gamma_{1d} T_{HD}^* \frac{I_H}{I_H^*} + A_1 I_H + \lambda_D S^* I_D^* - A_2 I_D^* - \lambda_D S^* I_D - A_2 I_D$$
$$+ \omega_1 I_H^* + \omega_2 I_D^* - A_3 I_{HD}^* - \omega_1 I_H^* \frac{I_{HD}}{I_{HD}^*} - \omega_2 I_D^* \frac{I_{HD}}{I_{HD}^*} + A_3 I_{HD}$$
$$+ \nu_1 I_{HD}^* + \xi_1 I_H^* - A_4 T_H^* - \nu_1 I_{HD}^* \frac{T_H}{T_H^*} - \xi_1 I_H^* \frac{T_H}{T_H^*} + A_4 T_H + \nu_2 I_{HD}^*$$
$$+ \xi_2 I_D^* - A_5 T_D^* - \nu_2 I_{HD}^* \frac{T_D}{T_D^*} - \xi_2 I_D^* \frac{T_D}{T_D^*} + A_5 T_D + \alpha_1 T_H^*$$
$$\alpha_2 T_D^* - A_6 T_{HD}^*) - \alpha_1 T_H^* \frac{T_{HD}}{T_{HD}^*} - \alpha_2 T_D^* \frac{T_{HD}}{T_{HD}^*} + 6 A_6 T_{HD}^*$$

let $\frac{dL}{dt} = M - N$

where M are the positive values and N the negative values then, grouping negative and positive terms gives,

$$M = \wedge + \gamma_{2d}T^* + \lambda_H I_H^* S + \lambda_D I_D^* S + \mu S + q_1\lambda_H S^* I_H^* + q_1\gamma_{1d}T_{HD}^* + q_1 A_1 I_H + q_2\lambda_D S^* I_D^* + q_3\omega_1 I_H^* + q_3\omega_2 I_D^* + q_3 A_3 I_{HD} + q_4\nu_1 I_{HD}^* + \xi_1 I_H^* + q_4 A_4 T_H + q_5\nu_2 I_{HD}^* + q_5\xi_2 I_D^* + q_5 A_5 T_D + q_6\alpha_1 T_H^* + q_6\alpha_2 T_D^* + q_6 A_6 T_{HD}^*$$

$$N = -\lambda_H S^* I_H^* - \lambda_D S^* I_D^* - \mu S - \frac{S}{S^*} \wedge -\gamma_{2d}T_D^* \frac{S}{S^*} - q_1 A_1 I_H^* - q_1\lambda_H S^* I_H - \gamma_{1d}T_{HD}^* \frac{I_H}{I_H^*} - q_2 A_2 I_D^* - q_2\lambda_D S^* I_D - q_2 A_2 I_D - q_3 A_3 I_{HD}^* - q_3\omega_1 I_H^* \frac{I_{HD}}{I_{HD}^*} - q_3\omega_2 I_D^* \frac{I_{HD}}{I_{HD}^*} - A_4 T_H^* - q_4\nu_1 I_{HD}^* \frac{T_H}{T_H^*} - q_4\xi_1 I_H^* \frac{T_H}{T_H^*} - q_5 A_5 T_D^* - q_5\nu_2 I_{HD}^* \frac{T_D}{T_D^*} - q_5\xi_2 I_D^* \frac{T_D}{T_D^*} - q_6 A_6 T_{HD}^*) - q_6\alpha_1 T_H^* \frac{T_{HD}}{T_{HD}^*} - q_6\alpha_2 T_D^* \frac{T_{HD}}{T_{HD}^*}$$

if M<N, then $\frac{dL}{dt} < 0$.

$\frac{dL}{dt} = 0$, if and only if $S = S^*, I_H = I_H^*, I_D = I_D^*, I_{HD} = I_{HD}^*, T_H = T_H^*, T_D = T_D^* \; and \; T_{HD} = T_{HD}^*$.

The largest compact invariant set in $\{S, I_H, I_D, I_{HD}, T_H, T_D, T_{HD} \in: \frac{dL}{dt} = 0\}$

is a singleton E^*, where E^* is endemic equilibrium point. Therefore, the endemic equilibrium point is globally a symptomatically stable in the invariant L if M<N by Lasalle.,(1976).

3.3.10 Sensitivity analysis

This is examining the effect of control parameters on the basic reproduction number. We investigate using the Normalized forward sensitivity variable which is differentiable and depends on the control parameters. The sensitivity index of a control parameter on basic reproduction number is given by

$$\frac{\partial R_o}{\partial \phi} \times \frac{\phi}{R_o} \tag{33}$$

where

$\phi(\wedge, \omega_1, \omega_2, \nu_2, \nu_2, \alpha_1, \alpha_2, \xi_1, \xi_2, \gamma_{1d}, \gamma_{2d}, \mu, \delta_h, \delta_{2d}, \delta_{hd}, \beta_1, \beta_2)$

from analytical analysis, its evident that $\wedge, \beta_1, \beta_2, \nu_2, \delta_{hd}, \gamma_{1d}, \gamma_{2d}, \alpha_1, \alpha_2$ have no effect on the basic reproduction number. The sensitivity indices for $\omega_1, \omega_2, \nu_1, \xi_1, \xi_2, \mu, \delta_h, \delta_d)$ will be determined by numerical analysis.

4 Numerical simulations

4.0.1 Parameter Estimation

Table 4.1, shows some parameter values from the literature and others assumed for the purpose of illustration.

In 2015, Turkana county population was estimated at 1045579, comprising of 542,658 males (52 percent) and 502,921 females (48 percent)[KNBS, 2015]. Children below 15 years constituted 43 percent of the population, while youth aged 15-24 years constituted 24 percent of the population (2015 KNBS Population Projections).

We used the Kenyan demographic data to compute the death rates, HIV parameter values were obtained from the National AIDS Control Council (NACC) report of 2015, and the Kenya National Bureau of Statistics (KNBS) report of 2014 The initial values for some state variables were obtained from the Kenyan demographic data, Kenya-HIV-County-profiles 2016, NACADA 2017 and the NACC 2015 report although lack of documented data on drug and substance a buse in Turkana contributed to assumed values of some state variables and parameters on drugs

Female life expectancy in Kenya is 65.8 years and 61.1 years for male [WHO, 2015]. Therefore natural mortality rates, $\boldsymbol{\mu_f}=\frac{\mathbf{1}}{\mathbf{65.8}}$ = 0.01519757 and $\boldsymbol{\mu_m}=\frac{\mathbf{1}}{\mathbf{61.1}}$ = 0.01636661 respectively. We then calculate the natural mortality rate as the average of the two giving approximately $\boldsymbol{\mu}$=0.0158 per year.

We used the following variables;

$$\begin{aligned} &\boldsymbol{S = 50,000}(\text{assumed}), && \boldsymbol{I_H = 20396} \\ &\boldsymbol{I_D = 30,000}(\text{estimated}), && \boldsymbol{I_{HD} = 10,000}(\text{estimated}) \\ &\boldsymbol{TH = 4684} && \boldsymbol{T_D = 500}(\text{assumed}) \\ &\boldsymbol{T_{HD} = 5200(assumed).} \end{aligned}$$

PARAMETER	VALUE	SOURCE
$\wedge$	**0.0397**	**World Bank 2017**
β_1	**0.04**	**Kenya HIV estimates(2015)**
β_2	**0.06**	**NACADA(2017)**
λ_H	**0.0039**	**Computed**
λ_D	**0.0116**	**Computed**
ω_1	**0.1700**	**Computed**
ω_2	**0.8333**	**Computed**
ν_1	**0.0390**	**Computed**
ν_2	**0.0042**	**Computed**
α_1	**0.9008**	**Estimated**
α_2	**0.0962**	**Estimated**
ξ_1	**0.2297**	**Computed**
ξ_2	**0.0050**	**Assumed**
γ_{1d}	**0.0199**	**Estimated**
γ_{2d}	**0.0005**	**Literature**
μ	**0.0200**	**WHO 2015**
δ_d	**0.015**	**Estimated**
δ_h	**0.0308**	**Computed**
δ_{hd}	**0.0229**	**Estimated**

Table 3. parameter values

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4.0.2 Numerical results

Susceptible

The figure below shows what happens in the susceptible population in the system with time. Since this happenings take a lot of time to be realised, the period of the simulation is taken as five years. The susceptible population decreases steadily with time and this explains why the graph in fig.2 increases as time goes on since increase in infected individuals decreases the susceptible.

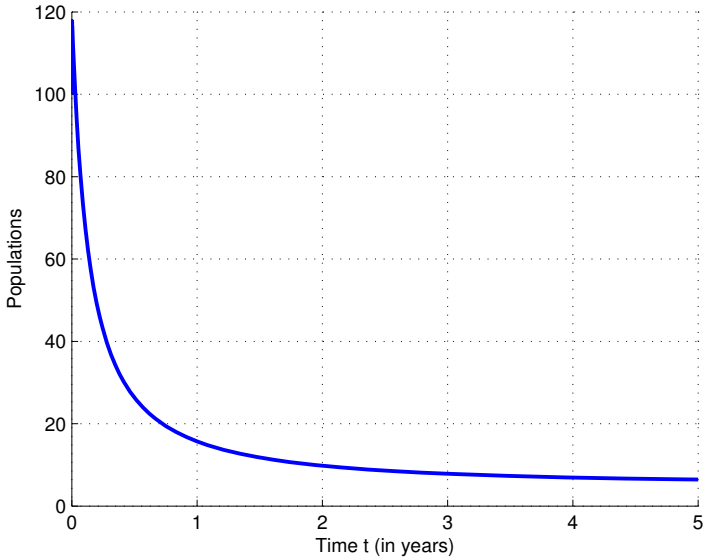

Figure 1. Population of susceptible

HIV infected individuals

The rate at which susceptible individuals in fig.2 are getting infected with HIV increases steadily hence decreasing the susceptible population.

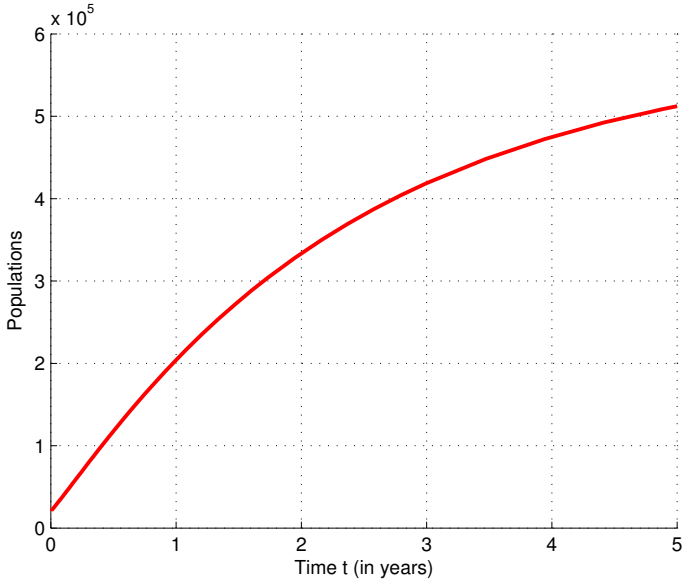

Figure 2. HIV infected individuals

Infected drug users

The population of drug users in fig.3 increases for a short while and gradually decreases. This can be viewed in the context that, the the rate at which individuals interact with those who use substances i.e susceptible drug users is lower compared to the rate of HIV infection.

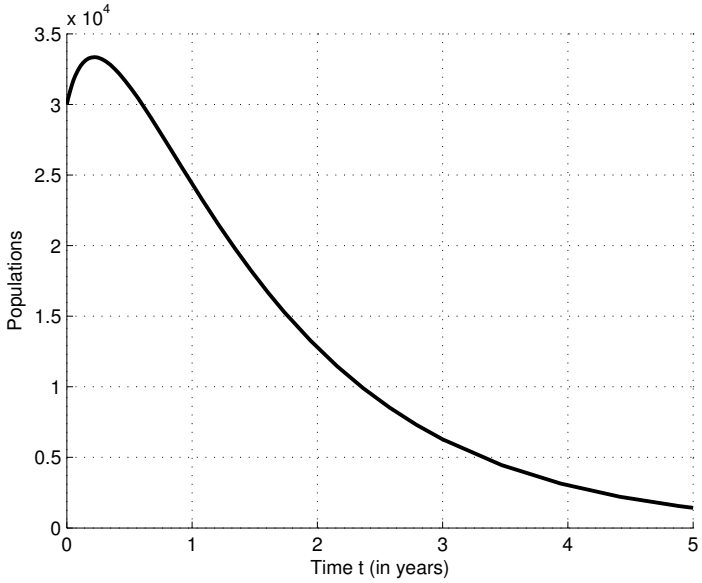

Figure 3. Infected drug users, I_H

Dually infected individuals

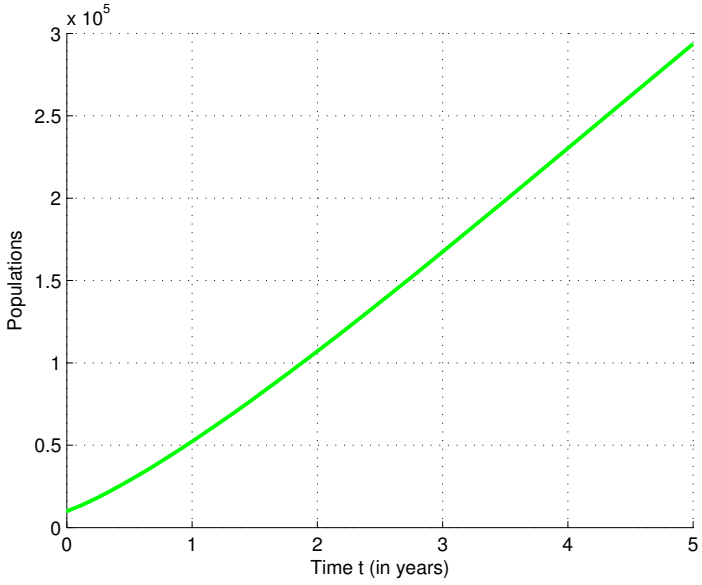

Figure 4. Dually infected individuals, I_{HD}

Number of dually infected individuals in fig.4 above increases as the number of those infected singly with HIV and drug abuse habits increases too.

HIV infectives under treatment

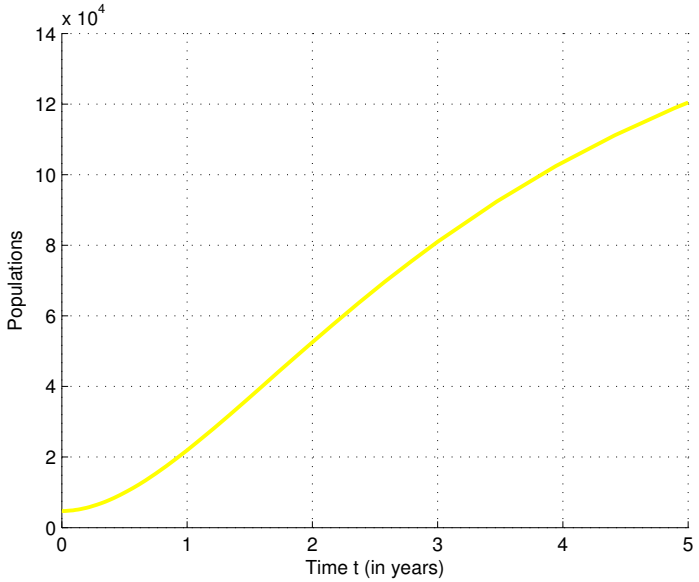

Figure 5. HIV infectives under treatment, I_{HD}

Demand for HIV therapy treatment increases with increase in HIV infection in fig.2. This steady increase may be as a result of awareness information on importance of Antiretroviral therapy in suppressing the viral load leading to prolonged lifespan of HIV positive individuals.

Infected drug users on treatment

Figure(6) shows the rate at which drug abusers are seeking rehabilitation services or gradually getting informed on drug use dangers is increasing. Fig.3 explains this happenings since at a point in time, the number of those who use drugs reduces gradually. Decrease in the number of drug users corresponds to an increase in the number of people seeking drug abuse rehabilitation services or just avoiding the use of drugs.

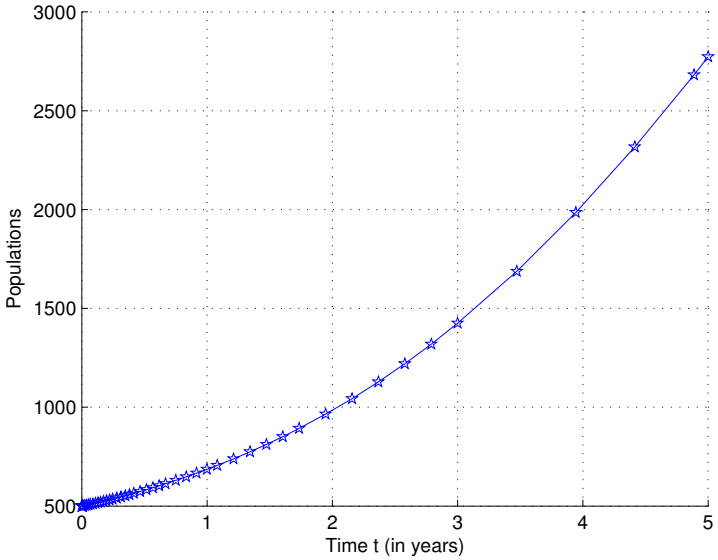

Figure 6. Infected drug users on treatment,T_D

Dually infected individuals under treatment

The population seeking treatment for both HIV infection and drug abuse habits is increasing as the number of those dually infected also increases in fig.4. This could be attributed to the fact that, managing drug abuse habits helps in avoiding drug abuse consequences related to sexual health including decision making on use of condoms, having multiple sexual partners etc that can lead to getting sexually transmitted diseases including HIV/AID.

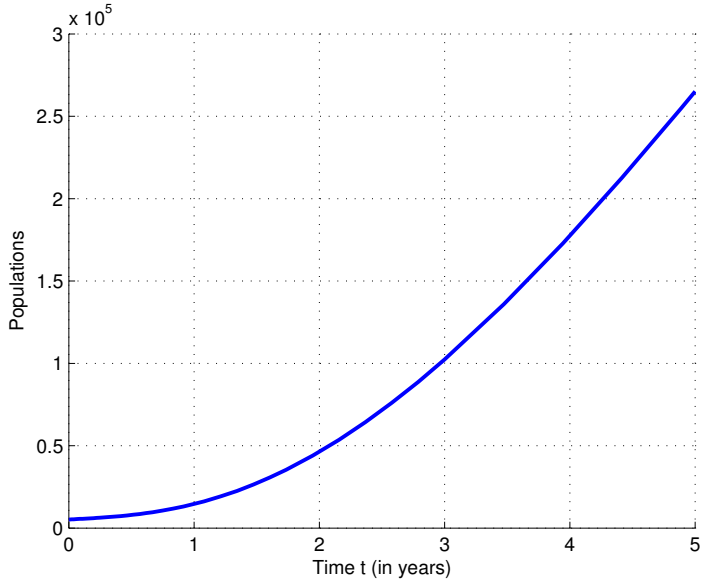

Figure 7. Dually infected individuals under treatment, T_{HD}

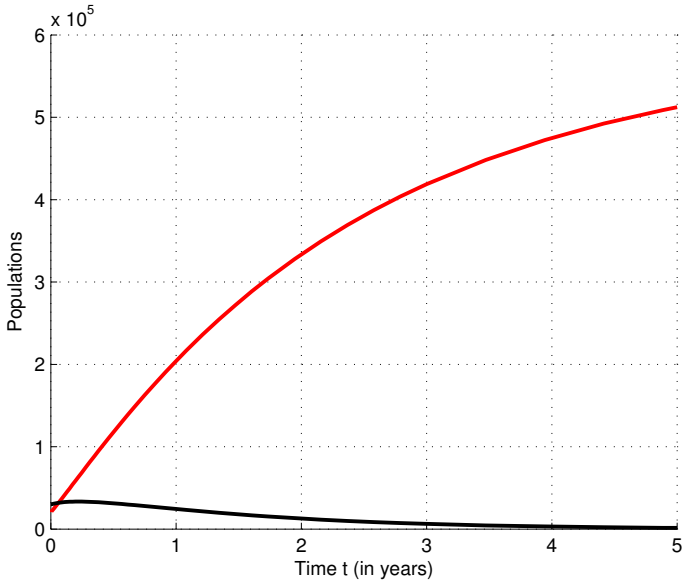

Figure 8. $I_H(red)$ **and** $I_D(black)$ **Populations**

From figure (8) above, as the number of HIV infectives increases, the population with drug abuse habits decreases showing that drug and substance abuse will have little impact on the spread of HIV/AIDS in Turkana County in the next five years.

5 Discussion and Conclusion

We designed a mathematical model on the transmission dynamics of HIV/AIDS in the context of drug and substance abuse is and investigated its mathematical properties for assessing the impact of drug abuse on the transmission of HIV/AIDS in Turkana County. The disease-free equilibrium is shown to be globally asymptotically stable when the corresponding reproduction number is less than unity. Furthermore, a sensitivity analysis of the reproduction number to the level of drug and substance abuse is carried and it showed that drug misuse has the potential to increase disease transmission through the basic reproduction number.

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Appendix

Expressions for the parameters in table 4.1 are,

$$\begin{cases} \boldsymbol{\lambda_H} = \frac{\beta_1(I_H+\pi_1 I_{HD}+\pi_2 T_H+\pi_3 T_{HD})}{N} \\ \boldsymbol{\lambda_D} = \frac{\beta_2(I_D+(1-\pi_1) I_{HD}+\pi_4 T_D+(1-\pi_3) T_{HD})}{N} \\ \boldsymbol{\omega_1} = \frac{I_H}{I_{HD}},\ \boldsymbol{\omega_2} = \frac{I_D}{I_{HD}},\ \boldsymbol{\nu_1} = \frac{I_{HD}}{T_H},\ \boldsymbol{\nu_2} = \frac{I_{HD}}{T_D}, \\ \boldsymbol{\alpha_1} = \frac{T_H}{T_{HD}},\ \boldsymbol{\alpha_2} = \frac{T_D}{T_{HD}},\ \boldsymbol{\xi_1} = \frac{T_H}{I_H},\ \boldsymbol{\xi_2} = \frac{T_H}{I_H}, \\ \boldsymbol{\gamma_{1d}} = \frac{T_{HD}}{I_H+T_H},\ \boldsymbol{\gamma_{2d}} = \frac{T_D}{S}. \end{cases} \tag{34}$$