**KWAME NKRUMAH UNIVERSITY OF SCIENCE AND TECHNOLOGY,**

**KUMASI**

**COLLEGE OF SCIENCE**



# PREDATOR-PREY MODEL OF HIV PROPAGATION IN A HETEROSEXUAL COMMUNITY

A THESIS SUBMITTED TO THE DEPARTMENT OF MATHE MATICS

IN PARTIAL FULFILMENT OF THE

REQUIREMENTS FOR THE AWARD OF THE DEGREE OF

MASTER OF PHILOSOPHY IN

MATHEMATICS

BY

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## DECLARATION

I hereby declare that this work is my own. It is towards the award of Master of Philosophy in Mathematics Degree and that to the best of my knowledge, it contains no materials previously published by another person nor material which has been accepted for the award of any other degree of the University, and that due acknowledge has been made to such text.



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**ABSTRACT**

The objective of this project is to apply the predator –prey model to come out with a model for the propagation of HIV, taking into consideration the population of newly infected males and newly infected females at a given time. This particular model is conjectured because it has been observed that there is an almost sinusoidal rising and falling of the time series trajectories of newly infected male and female cases of the Ghana data.

With some few assumptions made the model is formulated and the analysis shows that it conforms to the predator – prey model. Even though parameters in respect of newly infected males and females were not directly available for the simulation, with some assumptions, the parameters of the model are estimated and the simulation of the model for various scenarios using MATLAB is done. These simulations give the typical almost sinusoidal trajectories for both the populations of the newly infected males and newly infected females. This appears to confirm that the propagation of HIV follows the predator – prey model. It also shows that the rate of infections of HIV keeps rising and falling with time. The curves also show that more females are infected at any given time than males. Some recommendations regarding the eradication of HIV or the curtailment of the spread of HIV are offered.

## DEDICATION

I dedicate this project to the Lord of lords, the Alpha and the Omega, the giver of life.

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## LIST OF ABBREVIATIONS

| | |
|---|---|
| AIDS | - Acquired Immunodeficiency Syndrome |
| CCR5 | - Cysteine-Cysteine Chemokine Receptor5 |
| CD4 | - Cluster of Differentiation 4 cells |
| CXCR4 | - C-X-C Chemokine Receptor type 4 |
| DNA | - Deoxyribonucleic Acid |
| GEATM | - The Global Environmental Atmospheric Transport Model |
| HIV | - Human Immunodeficiency Virus |
| HSX | - Heterosexuals |
| MSM | - Men who have Sex with Men |
| NGO | - Non Governmental Organization |
| RNA | - Ribonucleic Acid |
| SGA | - Single Genome Amplification |
| UNAIDS | - United Nations program on HIV/AIDS |
| WHO | - World Health Organization |

**ACKNOWLEDGEMENT**

It is an undeniable fact that a project of this nature cannot contain any valuable material without the help and contribution of certain personalities in the mathematics department (KNUST) that are either directly or indirectly involved in issues pertaining to the subject matter of HIV/AIDS Epidemic, predator-prey model or the others.

My first and foremost thanks goes to the almighty God for bringing me this far.

I am also very grateful to my supervisor, Dr F. T. Oduro for patiently reading through my scripts and his valuable suggestions and corrections; without his inspirations and encouragement this project would have not reach its ultimate quality.

My special thanks go to Mr. Isaac Talley, a student and a friend of this department for his support in one way or the other in this project.

However, I am wholly responsible for any shortcoming that may be detected in this work.

# CHAPTER 1

# INTRODUCTION

## 1.1 BACKGROUND OF THE STUDY

Mathematics has been a useful tool in epidemiology; Sir Ronald Ross founded the field of mathematical epidemiology. He came out with a Mathematical model for malaria in 1911. His mathematical work has been used widely not only in the study of malaria but also in the study of other diseases. ( Macdonald, 1957)



HIV/AIDS is a disease which scientist have been trying to find a lasting solution to some of the attempts includes; education on the need to abstain from unprotected sex, being, faithful to ones' partner, the use of condoms and delay in early sex. Antiretroviral drugs are also used for those who have been infected, to prolong their lives but not to cure the disease. Although all these attempts are good, the disease is still spreading. Some mathematicians have also come out with useful models on the spread of HIV/AIDS, but there is still need to come out with a model which will help explain, predict and stop or further curtail the spread of the disease. These leads to the idea of applying the predator prey model to the propagation of HIV.

The population will be classified into a class of newly infected males (predators) and a class of newly infected females (prey).

The model which takes the form of the predator- prey model will be thoroughly analyzed. The stability of the critical points will be analyzed and the trajectories found

## 1.2 STATEMENT OF THE PROBLEM

HIV/AIDS epidemic has become one of the most critical challenges facing public health in Ghana and the world at large, particularly, the sub- Saharan African countries. African with just over 10% of world's population carries well above 75% of the burden of this epidemic UNAIDS, 2004. Prevalence and incidence rates in East Africa and South Africa include some of the highest in the world with prevalence rate exceeding 35% in Botswana and Swaziland but in West African sub – region, prevalence rates have remained lower with no country having a rate above 10% and most having a rate between 1% and 5.



Currently, the HIV/AIDS statistics show that Ghana has prevalence rate of 3-4% and that 500000 persons are infected. This number is expected to rise and as a result, scientist and researchers have been working hard to find a lasting solution to curb the propagation of HIV/AIDS in the country.
(Nasidi et al, 2004)

In an effort to combat the propagation of the disease, there is the need for a continuous quantitative monitoring of the disease to make sure it is brought under controls and this can be effectively done within the field of mathematical epidemiology. Although most mathematical models have been proposed, the virus still continuous to spread. Other control efforts includes education on abstinence, being faithful to ones' partner, the use of condoms etc. the main problem is the group that should be targeted during educational programs. Hence a predator-pray model of HIV propagation could help identify such a group.

## 1.3 OBJECTIVES OF THE STUDY

A mathematical model for the propagation of HIV in a heterosexual community will is proposed .The model can be applied to any heterosexual community, the community will be put into two classes; newly infected males and newly infected females. The model will be applied to determine whether it is the males or the females who transmit the disease more.



The model will also be applied to advise health personnel as well as the Ghana AIDS commission and all those concerned in bringing the spread of HIV under control as to which group of the society that educational campaigns should be targeted most.

## 1.4 METHODOLOGY

The methods employed in this project includes;

i. A review of differential equations as applied to epidemiology was done

ii. The possible transmission of HIV /Aids in a community was looked at.

iii. A predator-prey model for the transmission of HIV / AIDS was formulated as a system of differential equations and the equilibrium points determined

iv. The stability of the equilibrium points was also determined.

v. Simulation using MATLAB was also done.

## 1.5 JUSTIFICATION OF THE STUDY

Focusing on the epidemiology of HIV/AIDS in Ghana, several people including NGO's have tried to find solution to the problem or curtail the spread of the virus and for that matter the disease AIDS.

The Ghana AIDS Commission is currently reviewing the National Strategic Framework II, covering 2006 – 2010 with stakeholders and bilateral and multilateral partners. The first national strategic plan focused on live themes, prevention of new infections, care and support for people living with HIV/AIDS, creation of an enabling environment for the national response, decentralization of implementation of HIV/AIDS activities through international arrangements, research and monitoring.

A consequence of these features is that the analysis of data is usually most effective when it is based on a model that describes aspects of the infection process (Becker et al, 1991).



Solomon, Gakielon and Murray (2001) also stated that the understanding of the magnitude and trajectory of HIV/AIDS epidemic, as well as the uncertainty around the parameters is critically important both for planning and evaluating control strategies and for preparing for vaccine efficacy trials. Mathematical models can become very useful tools in this area. Apart from that modeling exercise are aimed at making use of the available data (no matter how little) to provide information about the trend inherent in the course of the epidemic. (Solomon et al, 2001)

## 1.6 THESIS STRUCTURE.

This thesis consists of five chapters. Chapter One covered the background of the study, statement of the problem, the objectives of the study, the methodology applied in the study, the justification of the study and the structure of the thesis/.

Chapter Two covered a review of related literature on the stability of equilibrium points as well as Mathematical and Statistical models of HIV/AIDS transmission.

Chapter Three took us through some of the various methods of modeling HIV transmissions including the predator prey model on which this thesis is based.

In chapter Four a predator- prey model of HIV propagation was formulated. The equilibrium points were determined and the stability analysis of the points was done. The parameters of the differential equations were also determined and Simulations of the model equations using MATLAB was done for the phase portrait as well as for the trajectories.

In chapter Five, the final chapter, the conclusion was made and this was followed by recommendations.

# CHAPTER 2

# REVIEW OF RELATED LITERATURE

## 2.0 INTRODUCTION

In this chapter related literature to the topic were reviewed. This includes statistical modeling of HIV/AIDS epidemic in the Northern central zone of Nigeria, the global overview of the epidemic, HIV/AIDS in sub Saharan Africa, HIV/AIDS prevalence in Ghana and some models of HIV propagation.



## 2.1 GLOBAL OVERVIEW ON THE EPIDEMIC.

'AIDS is far more than a medical problem. AIDS is far more than a national problem, AIDS is far more than over' (Kofi Annan,+ 2009). The rate at which new HIV infections are growing and the pace at which HIV infection progresses to AIDs, have to slow down in certain populations in some parts of the world.

This is happening at the same time as people adopt safer behavior and prevention and care services expanding. Partially richer countries owe the change to the availability of antiretroviral therapies.

There is abundant evidence globally that well-designed prevention programs and other successful public health measures can reduce and stabilize the epidemic. In societies where services and programs were already well equipped before the epidemic of HIV/AIDS, the creation of new initiatives and the re-orientation of others, led to a gradual decline in the incidence of HIV. We can observe a similar trend in certain sections of the population even in resource –oriented settings at least partly because of rigorous prevention efforts.

Nevertheless, the dynamics of the epidemic differ strongly between countries due to biological, epidemiological and socio –cultural factors, with continuing low prevalence in some of the northern and western countries, stabilizing epidemics in others and staggering increases in many countries in the south.

Like many developing countries which are experiencing exponential growth of HIV/AIDS cases. At the same time global spending on HIV/AIDS care, research and prevention reflects this disparity- developing countries receive only about 12% of such resources despite having 95% of the cases. In addition, the resources allocated to combat the epidemic are grossly inadequate – AIDS is spreading three times faster than the funding to control it.( UNAIDS, 2009).

## 2.2 HIV/AIDS IN SUB SAHARAN AFRICA.

Like the industrialized world, Africa is struggling with an epidemic that is now entering its third decade. However, while a few African countries have succeeded in stabilizing or reversing HIV infection rates, the epidemic is out of control especially in the Southern part of the continent. Socio economic factors like poverty, illiteracy, gender inequality, increased mobility of population and rapid industrialization involving the movement of workers from villages to cities, are still contributing to the spread of HIV/AIDS.

Today when there are over 20 million Africans infected, the situation is almost catastrophic. The fact that there were over two million deaths due to AIDs during 1998 including nearly half a million deaths occurring among children and four million new infections in 1997 prove that the epidemic is an unprecedented crisis for the continent.

In the most severely affected countries, a quarter of the adult population is infected. Hard-won gains in life expectancy and child survival are being wiped out. The AIDS related suffering of individuals, families and societies is enormous. Education and health systems are staggering under the burden as they lose trained professionals and incur higher cost because of the epidemic.

In Africa, sex between men and women is the primary means of HIV transmission although in almost every country there are also at least a few cases resulting from sex between men. Transmission of intravenous drugs is growing rapidly in southern Africa and this method of transmission is probably going to be relevant in other African countries as well. Transmission through blood transmission are still occurring where screening of blood used for transfusion is not efficient.

Approximately two thirds of people infected are living in sub- Saharan Africa and half of them are women. The trend is that in Sub –Saharan countries, more women than men are infected and worldwide, women are infected at a faster rate than men are. In some countries 20 – 45 % of pregnant women are HIV positive and one million children are living with HIV, while another twelve million have lost their mother or parents. Life expectancy is dropping to levels not seen since 1960; hard –won gains in child survival are being reversed.( UNAID, 2009).

## 2.3 STATISTICAL MODELING OF HIV/AIDS EPIDEMIC IN THE NORTH CENTRAL ZONE OF NIGERIA

Here the UNAIDs estimation and projection package(EPP) to HIV/AIDS epidemic in the north central zone of Nigeria was applied to propose a statistical model for the course of

the epidemic and to generally investigate the level of trend inherent in the epidemic over the years. HIV/AIDS surveillance data was used to model the situation for the rural and urban sentinel sites in the zone. Using EPP as a point of reference, a statistical model was proposed for the course of HIV/AIDS epidemic in the zone. The result shows that the UNAIDS package is a great Aid to the HIV/AIDS modeling in Nigeria.

The epidemic rate was estimated to be 0.91 in 1997, 0.7% in 2000, and projected to be 0.63 in 2010. Also an estimated 378,870 people were expected to die due to the epidemic in the year 2010. The prevalence peaked later than the incidence which peaked around 1997, but this was expected to rise slowly after 2007. The mortality rate was relatively low among site inside major towns than those outside major towns, but the situation was generally still rising. (Akpa et al, 2008)



## 2.4 HIV/AIDS PREVALENCE IN GHANA.

The HIV/AIDS epidemic in Ghana seems to be progressing slowly. The government of Ghana estimated the number of adults and children living with HIV as of 2004 at 404,000. The joint United Nations program of HIV/ AIDS (UNAIDS) estimated the HIV prevalence in adults to be 3.1 % at the end of 2003, with an estimated 350,000 people living with HIV/ AIDS. Ghana's 2003 Demographic and health survey reported prevalence at 2.2% among the 9,000 people who agreed to be.

Ghana's system of HIV surveillance for women attending antenatal clinics has functioned well since its establishment in 1994. Sentinel surveys of 21 antenatal clinics in 2002 reported a range from 3.2 % to 9.1% in prevalence among pregnant women. In 2002, the

median HIV prevalence at four of these sites in Accra was 4.1%; elsewhere in Ghana, prevalence in antenatal clinics ranged from 3.2% to 3.4 ( Addo, 2009)

HIV prevalence is highest in the Eastern Region of Ghana and Lowest in the Northern Regions of the country. Prevalence is generally higher in urban areas, in mining and border tows, and along main transportation routes. HIV -1 accounts for 92% of HIV cases in Ghana; another 7.4% of reported HIV cases are dual infections with HIV -1 and HIV -2. Only 0.55 of HIV cases were exclusively HIV- 2. Heterosexual intercourse is the mode of transmission for about 80% of HIV cases, with mother –to-child transmission accounting for another 15%. According to the 2003 Demographic and health Survey, HIV prevalence is very low among most younger age groups, as relatively few are infected during their youth ( with the exceptions of infants through their mothers). The infection peaks late, compared to other countries, at 35 – 39 years for women and 40 – 45 years for men. The infection levels are highest in middle income and middle educational groups, with the poor and unemployed less affected.

Though evidence is still being gathered for program decisions, some populations thought to be at risk include sex workers, transport workers, prisoners, sexual partners of people living with HIV/ AIDS, and men who have sex with men and their female sexual partners. HIV prevalence uniformed services is not fully established.

Approximately 9,600 children under age 15 are living with HIV/AIDs, and at the end of 2003, nearly 170,000 children under age 17 had lost one or both parents to AIDs. At that time only a few thousand of these children had received assistance such as food aid, health care, protection services, or educational or psychosocial support.

Ghana's goal is to prevent new HIV infections as well as to mitigate the socioeconomic and psychological effects of HIV/AIDS on individuals, communities and the nation. The first national strategic plan focused on five themes; prevention of new infections; care and support for people living with HIV/ AIDS; creation of an enabling environment for a national response, decentralization of implementation of HIV/ Aids activities through institutional arrangements, research and monitoring and evaluation of programs. The second national strategic plan, currently in progress, focuses on; policy advocacy, and enabling environment; coordination and management of the decentralized response; mitigating the economic, socio-cultural and legal impacts, prevention and behavior change communication; treatment, care and support; research and surveillance; and monitoring and evaluation.

Multilateral and bilateral partners, nongovernmental organizations (NGOs), and civil society organizations actively participate in the national response, with more than 2,500 community –based organizations and NGOs reportedly implementing HIV/AIDS activities in Ghana. Substantial funding for HIV/AIDS activities is received from the United States, the United Kingdom, the Netherlands, Denmark, Japan, Canada and the United Nations Agencies. Activities include the five –country, World –Bank –led HIV? AIDS. Abidjan - Lagos Transport Corridor project, the World Bank –funded Treatment Acceleration Program for public –private partnership in HIV/AIDS management, the World health Organization (WHO) initiative; the United Fund to Fight AIDS, Tuberculosis and Malaria (GFATM)

Following the Declaration of Commitment of the United Nations General Assembly, Special Session on HIV/AIDs in 2001, the Government earmarked 15% of its health

budget for HIV/AIDs activities, and all ministries were asked to create an HIV/AIDs budget line. Available funding to support Ghana's response to the HIV/Aids epidemic includes about $6.7million from GFATM; about $12 million from multilateral partners, including the World Bank; and about $8 million from bilateral donors. Based on the level of funding already committed by the national government and its donors, WHO estimates a $ 5 to $12.8 million funding gap for HIV/AIDs activities in Ghana for the period 2004 – 2005. (WHO, 2009)



## 2.5. APPLICATION OF PREDATOR-PEY MODEL FOR THE PROPAGATION OF HIV/AIDS.

In a research paper presented by B D.Aggarwala 2001. At university of Calgary, He came out with two models on the spread of HIV.

In first part, he discussed a ratio dependent predator – pray model and applied it to the spread of HIV/AIDS in a society. For this model, he divided the population into two classes; the HIV positive individuals and the HIV negative individuals. The model was then applied to the data available for the Canadian society obtained from Health Canada and statistics Canada, it was predicted that the number of HIV positive as individuals would go up for the next five years. The results were compared with actual numbers and the comparison was satisfactory.

In the second part, a considerably more detailed density dependent model for the propagation of HIV/AIDS was developed. This model divides the society into three classes; HIV negative individuals, HIV positive individuals who have not developed AIDS disease and those who have developed AIDS. This model was also applied to the data

available from Health Canada. It was also established that the model was physically relevant by showing that in the model the number of both HIV positive and HIV negative people stay positive and finite for all $t > 0$

In this paper, Aggarwala considered only HIV positive, HIV negative and AIDS patients. It is therefore reasonable to consider the predator – prey model in relation to the groups of males and females infected people.( Aggarwala, 2001)



## 2.6 HIV MODEL BY RICK QUAX

In a thesis presented to the academic faculty college of competing, Georgia institute of technology, Rick Quax, 2008 et al simulated two complex models for HIV epidemic and found a remarkable qualitative fit to reported data for AIDS incidence and prevalence. He remarked that the most important result is that the mere dynamics of HIV epidemic is sufficient to produce rather complex trends in the incidence and prevalence statistics.

He also highlighted some previous work (based on traditional mathematical models) that attempted to explain distinctive trends in the reported data, e.g. by the introduction of particularly effective treatments that were considered unsupported. As a corollary he also substantiated the much debated paradox that the availability of Highly Active Anti-Retroviral Treatment likely causes and increased HIV incidence. That is, the introduction of treatment could be counter effective and found indeed that it likely results in higher stabilized HIV incidence.( Quax, 2008)

## 2.7 A STUDY ON MEN WHO HAVE SEX WITH MEN

In a study involving men who have sex with their fellow men (gays) and heterosexuals, it was realized that elucidating virus-host interactions responsible for HIV-1 transmission is

important for advancing HIV-1 prevention stages. To this end, Single Genome Amplification (S G A) and sequencing of HIV-1 within the context of a model of random virus evolution has made it possible for the first time an unambiguous identification of transmitted / founder viruses and a precise estimation of their members. Hence, this approach was applied to HIV-1 env analysis in a cohort of acutely infected men who have sex with men (MSM) and found that a high proportion (10 of 28; 36%) had been productively infected by more than one virus. In subject with multivariate transmission, the minimum number of transmitted virus ranges from 2 to 10 with viral recombination leading to rapid and extensive genetic shuffling among virus lineages. A combine analysis of this results together with recently published findings based on identical S G A methods in largely heterosexual (HSX) cohort revealed a significantly higher frequency of multivariate transmission in men having sex with men than in heterosexuals (19 of 50 subjects 38%) versus 34 of 175 subjects (19%); Fisher's exact P = 0.008.

To further evaluate the SGA strategy for identifying transmitted / founder viruses, Analysis of 239 overlapping 5 and 3 half genome or env – only sequences from plasma viral RNA (v RNA) and blood mononuclear cell DNA in an MSM subject who had a particularly well documented virus explosive history 3 – 6 days before symptom onset and 14 – 17 days before peak plasma viremia (47, 600 000 v RNA molecules / m) was done. All 239 sequences coalesced to a single transmitted / founder virus genome in a time frame consistent with the clinical history and a molecular done of this genome encoded replication competent virus in accord with model prediction. High multiplicity of HIV-1 infection in MSM compared with HSX is consistent with the demonstrably higher epidemiological risk of virus acquisition in MSM and could indicate a greater challenge for HIV-1 vaccines than previously recognized.

( Li H et al 2010 )

## 2.8 EVOLUTIONARY DYNAMICS OF HIV INFECTION

Luca Sguanci et al also came out with a model on the evolutionary dynamics of HIV infection. That is, within-patient evolutionary process during HIV infection. It was realized that during the HIV infection, several mutants of the virus a rise, which are able to use di- erent chemokine receptors, in particular the CCR5 and CXCR4 co receptors (termed R5 and X4 phenotypes respectively). Phylogenetic inference of chemokine receptors suggest that virus mutational pathways away generate R5 variants able to interact with a wide range of chemokine receptors di erent from CXC. Using the chemokine tree topology as a conceptual framework for HIV viral speciation,a model of viral phenotypic mutation from R5 to X4 strains which reflect HIV late infection dynamics. The model also investigated the action of Tumor Necrosis Factor in AIDS progression and made suggestions on better design of Highly Active Anti-retroviral treatment therapy.

(Luca et al, 2006).

## 2.9 A CELLULA AUTOMATION MODEL OF VIRAL PROPAGATION

In a paper, strain MC et' al introduced a cellular automation model of viral propagation based on the known biophysical properties of HIV. In particular, they include the competition between viral ability and Brownian motion. The model predicted three testable effects not present in previous descriptions; First, they found a profound dependence of viral infectivity on cell concentration, vision instability decreases infectivity more than 100 fold typical experimental conditions resulting in misleading estimate of the number of infections particles. Second, it was found that in large parameter

regime infections extinguishes itself due to insufficient target cell replenishment. Finally, it was also found that propagation is limited by viral stability at low cell density and by Geometry at high cell density. The geometry – limited region can be modulated by drown regulation of CD4. These different properties are analyzed quantitatively and compared with previous experimental results.

(Strain et al, 2002).



## 2.10 MODELING AND CONTROL OF HIV POPAGATION

In a study on the modeling and control of HIV/AIDS propagation, a case study of the Ashanti Region of Ghana, F. T. Oduro and P. Aboagye Sarfo (2007) came out with a model on the population, age and gender structure dynamic of the reported cases of HIV/AIDS in the Ashanti Region of Ghana for the period 1982 – 2001 with a view of assessing the level and impact of the pandemic as well as the effectiveness of existing control measures. They used statistical method of system identification based on vector autoregressive time series analysis. This led in most cases to deterministic discrete – time linear autonomous models. The population dynamics of reported. HIV/AIDS cases for females and males were found to be of second order, unstable growing linearly in the mean but with a sinusoidal oscillation of period 4.2 years. Three age groups with common dynamical characteristics were identified; the 0 – 19, 20 – 49 and the 50+ years age groups. Each of these age groups however had first order dynamics, which were stable reaching equilibrium levels in a few decades. Further analysis involving the computation of the controllability matrix revealed that condom utilization as a method of controlling HIV/AIDS has no significant impact in the control of the number of reported case (Oduro et al, 2007).

# CHAPTER 3

# METHODOLOGY

## 3.1 INTRODUCTION

In this chapter, certain key concepts and methods of analyzing differential equations that are central to this project have been presented. Related models such as epidemics, competition and the predator, prey have been also discussed



### 3.1.1 DEFINITION (CRITICAL POINT)

A critical point or equilibrium position of a system of differential equations is the set of points for which $X^1(t)=0$

### 3.1.2 DEFINITION (STABLEPOINT)

Suppose $x \in W$ is an equilibrium of the differential equation $X^1= f(x)$, where $f:W \rightarrow E$ is a $C^1$ map from an open set W of the vector space E into E. then $x$ is a stable equilibrium if for every neighborhood U of $x$ in W there is a neighborhood $U_1$ of $x$ in U such that every solution x(t) with x(0) in $U_1$ is defined and in U for all $t > 0$( Morris, 1974)

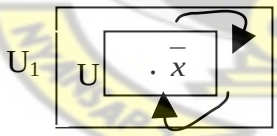


**FIGURE 3.1: A STABLE POINT**

### 3.1.3 STABILITY

**Definition 2**

If $U_1$ can be chosen such that in addition to the properties described in definition 1,

$\lim_{t \rightarrow \infty} x(t) = \bar{x}$, then $\bar{x}$ is asymptotically stable. See figure 3.2 below

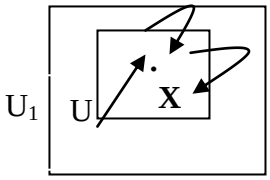


**FIGURE 3.2: ASYMTOTIC STABILITY**

### 3.1.4 DEFINITION 3

An equilibrium $\dot{x}$ that is not stable is called unstable. This means there is a neibourhood U of $\bar{x}$ such that for every neighbourhood $U_1$ of $\bar{x}$ in U, there is at least one solution x(t) starting at x(o) € $U_1$ which does not lie entirely in U. See figure 3.3 below.

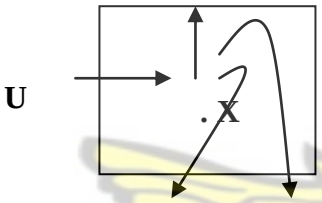


**FIGURE 3.3 : USTABLE POINT**

### 3.1.5 INSTABILITY

A sink is asymptotically stable and therefore stable. An example of an equilibrium that is stable but not asymptotically stable is the origin in $R^2$ for a linear equation $x^1$= Ax…….2 where A has pure imaginary eigenvA stalues. The orbits are all ellipses.

See fi:gure 3.4 Below.

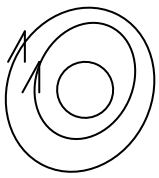

**FIGURE 3.4 : A STABLE BUT NOT ASYMTOTICALLY STABLE**

### 3.1.6 THEOREM:

The trivial solution of (1) is asymptotically stable if and only if all the eigenvalues of A have negative real parts.( Morris et al, 1974)

### 3.1.7 THEOREM:

If the eigenvalues of A with zero real parts are simple and all other eigenvalues have negative real parts then the trivial solution of (2) is stable. (Morris et al, 1974)



## 3. 2 LINEARIZATION AND STABILITY

From the definitions, stability properties depend on the behavior of the system near the equilibrium point, Hence in conducting an analysis of stability, it is convenient to replace the full nonlinear description by a simpler description that approximates the system near the equilibrium point, often a linear approximation is enough to clarify the stability properties, this idea of checking stability by examinations of a linearised version of the system is referred to as Liapunov's First Method. This is usually the first step in the analysis of any equilibrium point. The linearization of the non linear system is based on linearization of the non linear function F in its description. An nth – order system is defined by n functions, each of which depends on the n variables. In this case each function is approximated by the relations $f_1(\bar{x}_1 + y_1, \bar{x}_2 + y_2, \ldots\ldots, \bar{x}_n + y_n) = f_1(\bar{x}_1, \bar{x}_2 \ldots\ldots \bar{x}_n) +$

$\partial/\partial x_1 f_1(\bar{x}_1, \bar{x}_2 \ldots\ldots \bar{x}_n)\, y + \partial/\partial x_2 f_1(\bar{x}_1, \bar{x}_2 \ldots\ldots \bar{x}_n)\, y_2 + \ldots\ldots +$

$\partial/\partial x_n f i;(\bar{x}_1, \bar{x}_2 \ldots\ldots \bar{x}_n)\, y_n \ldots\ldots\ldots$

The linear approximation for the vectors f(x) is made up of the n separate approximations for each component function. The complete result is expressed compactly in vector notation as $f(\bar{x} + y) = F(\bar{x}) + F(y)$

In this expression F is n x n matrix

$$\begin{pmatrix} \frac{\partial f_1}{\partial x_1}, \frac{\partial f_1}{\partial x_2}, \ldots\ldots\ldots\ldots\ldots\ldots \frac{\partial f_1}{\partial x_n} \\ \frac{\partial f_2}{\partial x_1}, \frac{\partial f_2}{\partial x_2}, \ldots\ldots\ldots\ldots\ldots\ldots \frac{\partial f_2}{\partial x_n} \\ \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots \\ \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots \\ \frac{\partial f_n}{\partial x_1}, \frac{\partial f_n}{\partial x_2}, \ldots\ldots\ldots\ldots\ldots\ldots \frac{\partial f_n}{\partial x_n} \end{pmatrix}$$



This is called the Jacobian matrix.

To determine the stability properties of a linear system, we determine the location of the eigenvalues of the system matrix and the stability properties of the linear version of a nonlinear system can be determined that way.

The importance of this technique is that except for the boundary situation, the eigenvalues of the linearised system completely exposes the stability properties of an equilibrium point of a system. This is because, for small deviations from the equilibrium point, the performance of the equilibrium is approximately governed by the linear terms. These terms dominate and that determine the stability provided that the linear terms do not vanished.( Waltman,199)

### 3.3 THE PHASE PLANE

Consider the linear system with constant coefficients

$X^1 = ax + by$

$Y^1 = cx + dy$ …… 4

They can be solved explicitly by linear system; this can be regarded as the first approximation of the nonlinear system

$X^1 = f(x, y)$

$Y^1 = g(x, y)$……(5)



Where f(x, y) and g(x, y) satisfy f(0,0) =g(0,0)=0 and have continuous partial derivatives, which at the origin are labeled as

$\frac{df(0,0)}{dx} = a, \frac{df(0,0)}{dy} = b, \frac{dg(0,0)}{dx} = c, \frac{df(0,0)}{dx} = d$

It can be observed that every exact knowledge of the behaviour of solution of (4) can often give qualitative knowledge of the behaviour of solutions of (5) near the origin. To avoid complications, we will assume that ad - bc $\neq$ 0 (that is the jacobian of the RHS of (5) is not zero). the assumption that f and g have continuous derivatives implies that if a set of initial conditions $x(t_o) = \alpha$ $y(t_o) = \beta$ is added to the system (5) then the exultance of a unique solution is guaranteed.

(Morris et al, 1974)

#### 3.3.1 THEOREM:

Let f(x ,y), g(x, y) be continuously differentiable. Then there is a solution of the initial value problem

$X^1 = f(x, y)$

$Y^1 = f(x, y)$

$X(t_o) = \alpha$

$Y(t_o) = \beta$

Valid on the interval I= $(t_o - y, t_0 + y)$ if this is denoted by $x(t, \alpha \beta)$, $y(t, \alpha \beta)$ are continuous function for $\alpha$ and $\beta$.the solution above is defined for all t Є R In the case of (4) but for (5), it is necessary to make additional assumptions on f and g to guarantee that a solution exist for all t € R.



Points along the solution of (5) can be viewed as a triple in $R^3$ (x(t), y(t), t) , a path traced out in three dimensions consisting of a time coordinate t and a two dimensional space coordinate (x ,y). the absence of the independent variable t in the RHS of (5) makes another interpretation useful. Solutions may be regarded in the plane as a parametric curve given by (x(t), y(t) with t as the parameter. This curve is simply the projection of the triple (t, x(t) ,y(t) in three dimensional space onto the plane of the space variables. The curve (x(t), y(t) is called a trajectory or an orbit and the plane is called the PHASE PLANE.

We shall explore the bases of the highly geometric approach with a view toward the appreciation that will follow later on. To see how the phase plane is a useful concept, let us note first an elementary property of the solution (5)

Morris et al, 1974

### 3.3.2 LEMMA

If $(\psi_1(t), \psi_2(t))$ is a solution of (5), so is $U_1(t - \tau), U_2(t - \tau)$ for any real numbers $\tau$

**Proof**

Define $\varphi_1(t) = \varphi(t- \tau)$ and $\varphi_2(t - \tau)$ then

$\varphi_1^1(t) = \psi_1^1(t - \tau) = f(\psi_1 t - \tau), \psi_2(t- \tau) = f(\varphi_1(t), \varphi_2(t))$.

$\varphi_2(t) = \psi_2(t-\tau) = g(\psi_1(t-\tau), \psi_2(t-\tau) = g(\varphi_1(t), \varphi_2(t)$ and therefore $\varphi_1(t), \varphi_2(t)$ solves (5).

Note that $(\psi_1(t), \psi_2(t)) \; t \in R$ and $\Psi_1(t - \tau), \Psi 2(t - \tau), t \in R$

Describe the same set of points in the plane and hence the same trajectory.

Now, if the solution is viewed as points in $R^3$ as (t, x(t), y(t)) representing time and two spaces coordinates, there is a unique solution through each points.

If we project these solutions onto the phase plane by using only (x(t), y(t)) as coordinates, might not a tangle of curves result? The fact that this is not the case, when t does not appear explicitly in f and g is stated in the theorem below. (William et al, 1992)

### 3.3.3 Theorem

Let f and g be continuously differentiable. Through each point $(x_0, y_0)$ of the plane, there is a unique trajectory

$X^1 = f(x , y)$

$Y^1 = g(x , y)$

(Morris, 1974)

Proof: suppose, to the contrary, there are two different trajectories ($\psi_1(t)$ , $\psi_2(t)$, passing through ($x_0, y_0$) that is $\psi(t_o) = x_o = \psi_1(t_1)$, $\psi_2(t_o) = y_o = \psi_2(t_1)$, where necessarily $t_o \neq t_1$

[By the uniqueness of the solutions of initial value theorem]

By the above lemma, the functions $x_1(t) = \psi_1(t-t_1+ t_o)$ and

$X_2(t) = \psi_2(t-t_o = t_o)$ form a solution of (4) yet $x_1(t_1) = \psi_1(t_o) = x_o = \psi(t_1)$ and $x_2(t_1) = \psi_2(t_o) = y_o = \psi(t_1)$ for all t. Hence $\psi_1(t)$, $\psi_2(t)$ and $\psi_1((t)$, $\psi_2(t)$ are the same trajectories (uses different parameterization)

On the other hand, consider that if $f(x_o, y_o) \neq 0$ in (5), then the initial value problem

$$\frac{dy}{dx} = \frac{g(x,y)}{f(x,y)} \quad \ldots\ldots\ldots\ldots\ldots(6)$$

$Y(x_o) = y_o$ has a unique solution since $\frac{dy}{dx} = \frac{y^1(t)}{x^1(t)} = \frac{g(x(t), y(t)}{f(x(t), y(t)}$

We now use the plane technique to analyze the system in (4). This system is in the form $x^1 = AX$ that makes computation of the eigenvalues and the eigenvectors and the conversion to polar coordinates easy. The analysis of this simply system provides guidelines as to what sorts of behaviour are possible in the following cases

### 3.3.4 CASE 1 (Real distinct eigenvalues with the same sign)

Let the eigenvalues of A be real distinct and of the same sign: take as a representative $A = \begin{pmatrix} \lambda & 0 \\ 0 & \mu \end{pmatrix}$ the system in (4) is then $x^1 = \lambda x$

$$Y^1 = \mu y$$

This can be solve to obtain $x(t) = x_0 e^{\lambda t}$

$y(t) = y_0 e^{\mu t}$

(a) if $\lambda$ and $\mu$ are negative, $\lim_{t \to \infty} x(t) = 0$ and $\lim_{t \to \infty} y(t) = 0$ since the coverage is monotone, the origin is an asymptotically stable critical point

(b) if $\lambda$ and $\mu$ are positive, then $\lim_{t \to \infty} x(t) = \pm\infty$ and $\lim_{t \to \infty} y(t) = \pm\infty$

Since the limiting behaviour is the same no matter how close, the initial conditions are to the origin, this is sufficient to show that the origin is unstable for a nonlinear system, we are interested only in the behavior near the critical point and such detail global behaviour will not generally be known, so the following idea is useful. The instability of the origin follows from the fact that the trajectory tends to the origin as time runs backward. For further explanation consider the system

$x^1 = f(x,y)$

$y^1 = g(x, y)$

if a change of variable $\tau = -t$ is made, the system becomes $\frac{-dx}{\partial\tau} = f(x, y)$

$\frac{dy}{\partial\tau} = g(x, y)$ the signs of all derivatives are reversed but $\frac{dx}{dy} = \frac{g(x,y)}{f(x,y)}$

So the differential equation of the trajectories is the same for both systems. The curve is the same but the parameterization is reversed. This is what is meant by 'time running' backward.

Initiatively if backward time must repel'

Hence the critical point is unstable if either $x_o=0$ or $y_o= 0$, the corresponding component remains zero for all t since $\theta(t) = \tan^{-1}\left(\frac{y(t)}{x(t)}\right) = \tan^{-1}\left(\frac{y_0 e^{\mu t}}{x_0 e\lambda t}\right) = \tan^{-1}\left(\frac{y_0}{x_0}\right) e^{(\mu-\lambda)}\, t$

Then $\lim_{t \to \infty} \theta(t) = 0 \quad if \lambda \geq \mu \quad and \lim_{t \to \infty} \theta(t) = \pm \frac{\Pi}{2} \quad if \quad \mu > \lambda$ except for solution corresponding to $x_0=0$, $y_0=0$ respectively. Limits for $t \to -\infty$ are reversed. The distinction between $\lambda$ and $\mu$ are arbitrary. It merely determines how the axes are labeled.

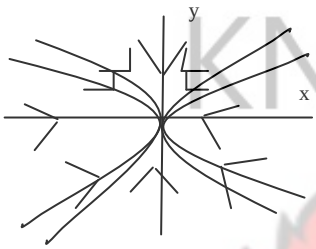


**FIGURE 3.5 : AN UNSTABLE POINT**

$0 < \mu < \lambda$ (case 1)

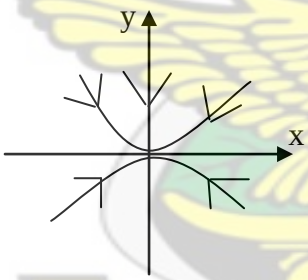


**FIGURE 3.6 : A STABLE NOTE**

$0 < \mu < \lambda$ (case 1)

Solution along the axes correspond to $x_0 = 0$ or $y_0$i this case

The origin is said to be the node.

### 3.3.5 CASE II (Real eigenvalues with opposite signs let the eigenvalues be real with opposite signs.

Also, assuming without lost of generality that $\lambda < 0 < \mu$ then the solution are

$X(t) = x_0 e^{\lambda t}$, $y(t) = y_0 e^{\mu t}$ and $r(t) = (x^2_0 e^{2\lambda t} + y_0 e^{2\mu t})^{1/2}$

$\mu >$ if $y_0 \neq 0$ then r(t) satisfies $\lim_{t \to 0} r(t) = \infty$ further again if $y_0 = 0$ $\theta(t) = \tan^{-1}\left(\frac{y_0 e^{\mu t}}{x_0 e^{\lambda t}}\right)$

satisfies

$\left(\lim_{t \to \infty} \theta(t) = \pm \frac{\Pi}{2}\right)$ if $y_0=0$, then $\lim_{t \to \infty} r(t) = 0$ and the trajectory approaches the origin

with $\theta$(t)=0 for all t. in this case the origin is said to be a **saddle point**

The equation $\frac{\partial y}{\partial x} = \frac{y^1}{x^1} = \frac{\mu y}{\lambda x}$ can be solved to yield $yx^{\frac{-\mu}{\lambda}} = c$ this give the hyperbolic-looking curve.

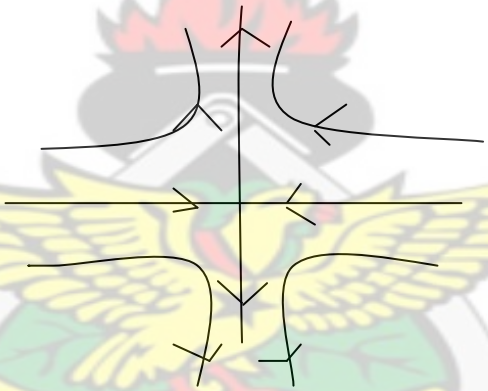

**FIGURE 3.7 A SADDLE $\lambda$ <0< $\mu$**

### 3.3.6 CASE III (complex conjugate eigenvalues) let the eigenvalues be complex conjugate with nonzero real parts

Consider A= $A = \begin{bmatrix} \alpha & -\beta \\ \beta & \alpha \end{bmatrix}$, $\alpha\beta \neq 0$ so that the eigenvalues are $\lambda = \alpha \pm \beta i$ where without

loss of generality, we take $\beta$ the system is $\begin{aligned} x^1 &= \alpha x + \beta y \\ y^1 &= \beta x + \alpha y \end{aligned}$

Hence polar coordinates are especially useful. The transformation to polar coordinates functions yield $r^1 = \alpha r,\ \theta^1 = -\beta$ this system may be solve to obtain r(t)=$r_o e^{\alpha t}$, $\theta(t) = \theta_0 - \beta t \quad as \quad t \to \infty, \theta(t) \to \infty$ so that the solution wind around the origin arbitrary many times. The polar radius tends to zero as $t \to \infty$. if $\alpha$ is negative and in this case the critical point is asymptotically stable, the polar radius tends monotonically to $+\infty \quad as \quad t \to \infty \quad and \quad to \quad zero \quad as \quad t \to -\infty \quad if \quad \alpha$ is positive. Hence, n this case the critical point is unstable.

The shape of the curve can be easily obtained in polar coordinates since $\frac{\partial r}{\partial \theta} = \frac{r^1}{\theta^1} = \frac{ar}{-\beta}$ it follows that $\log\left(\frac{r}{r_0}\right) = \left(\frac{\alpha}{-\beta}\right)(\theta - \theta_0)$ this curve is a logarithmic spiral so that trajectory in the phase plane is logarithmic spiral. This type of critical point is called a spiral or focus point or vortex plane

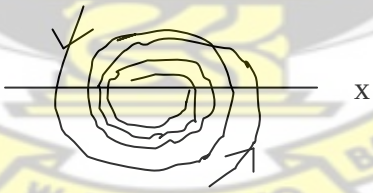


**FIGURE 3.8: A STABLE SPIRAL SINK ( $\beta > 0 > \alpha$)**

### 3.3.7 CASE IV (EIGENVALUES PURELY IMAGINARY)

Let the eigenvalues be purely imaginary. This is the same as the previous case except that $\alpha$=0

The corresponding representative of the class is A=$\begin{bmatrix} 0 & -\beta \\ \beta & 0 \end{bmatrix}$

The equation for the polar functions are as follows $r_1 = 0$ $\Theta_1 = -\beta$ and may be solved to obtain $r = r_0$, $\Theta = -\beta t + \Theta_0$ the trajectories are circles of radius $r_0$ about the critical point. This type of critical point is said to be a center. Since the trajectories in the phase plane are closed curves, the corresponding solutions are periodic since trajectories circles that begin near the origin remain there, the center is stable, but not asymptotically stable,

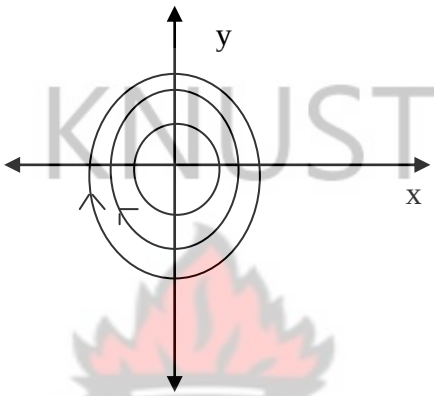


**FIGURE 3.9: A CENTER $\beta$ >0**

### 3.3.8 CASE V. (EQUAL EIGENVALUES)

Suppose the eigenvalues are coincident since the eigenvalues are equal, they are necessarily real. Here, there are two possible representative elements(depending on whether there are one or two linearly independent eigenvectors corresponding to the repeated eigenvalues)

First consider

$A = \begin{pmatrix} \lambda & 0 \\ 0 & \lambda \end{pmatrix}$ The system ( ) then becomes $x^{1} = \lambda x$

$$Y^{1} = \lambda y$$

And the equations are 'uncoupled' that is not related. A solution is

$x(t) = X_O e^{\lambda t}$, $y(t) = y_o e^{\lambda t}$

thus $r(t) = (x^2(t) + y^2(t))^{1/2}$ and $\lambda = 0$ asymptotic stability and if $\lambda > 0$, $\lim_{t \to \infty} r(t) = +\infty$

the polar angle is $\theta(t) = \tan^{-1}(y_0 e^{\lambda t} / x_0 e^{\lambda t}) = \tan^{-1}(y_0/x_0) = \theta_0$ and the direction is constant, that is, the trajectories are half way approaching or leaving the origin.

The solutions are depicted for $\lambda < 0$.

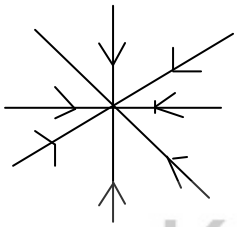



**FIGURE 3.10 : A DEGENERATED NOTE**

This critical point is referred to as a degenerate node

(Morris et al, 197)

### 3.4 LIAPUNOV FUNCTION

Earlier stability and the various types of stability including asymptotic stability of an equilibrium x of a dynamic system $x^1 = f(x)$ ..... (1) where

$F : W \longrightarrow R^n$ is a c1 map on an open set $W \subset R^n$. if $\overline{x}$ is a sink, stability can be detected by examining the eigenvalues of the part Df(x). other than that however, we have to find all solutions to (1) which may be difficult if not impossible.

The Russian mathematician and engineer A.M Liapunov, in his 1892 doctoral thesis, found a very useful criterion for stability. It is a generalization of the idea that for a sink there is a norm on $R^n$ such that $\left| x(t) - \overline{x} \right|$ decreases for solutions x(t) near x. he showed that certain other functions could be used instead of the norm to guarantee stability. —

Let $V : U \longrightarrow R$ be a differentiable function defined in a neighborhood $U \subset W$ of $\overline{x}$. we denote by $V : U \longrightarrow R$ the function defined by $V(X) = DV(x)(f(x))$. Here the right – hand side is simply the operator DV (x) applied to the vector f(x). Then if $\phi_t(x)$ is the

solution to (1) passing through x when t =0, V(x) = d/dt V ($\phi_t x$) by the chain rule. Consequently, if V(x) is negative, then V decreases along the solution of (1) through x. We can now state Liapunov's stability theorem.

Let x Є W be an equilibrium for

(1). Let V : U ⟶ R be continuous function defined on a neighborhood U ⊂W of x, differentiable on U - x, such that

a) V(x) = 0 and V(x) > 0 if $\overline{x} \neq x$;

b) V < 0 in u – $\overline{x}$. then $\overline{x}$ is stable

furthermore, if also

c) V < 0 in u –x , then x is asymptotically stable.

A function v satisfying (a) and (b) is called a Liapunov function for x. if (c) also holds , we call v a strict Liapunov function. The only equilibrium is the origin x = y = o ( waltman, 1995).

### 3.4.1 Theorem

**2**: Let $\overline{x}$ Є w be equilibrium of the dynamical system (1) and let V: U ⟶ R be a Liapunov function for $\overline{x}$, u a neighborhood of x. Let P ⊂U be a neighborhood of $\overline{X}$ which is closed in W. Suppose that P is positively invariant, and that there is no entire orbit in P ⟶ x on which V is constant. Then X is a asymptotically stable and PC B(X).

(Morri et al, 1974)

**Proof of Theorem 2**:

Imagine a trajectory X (t), 0≤t<∞ in the positive invariant set P. Suppose $\overline{x}$ (t) does not tend to x as t⟶ ∞. Then there must be a point a ≠ X in P and a sequence $t_n$ ⟶ ∞ such that

Lim X ($t_n$) = a

$N \rightarrow \infty$

If $\propto = V(a)$, then $\propto$ is the greatest lower bound of $\{V(x(t))]\ \ t \geq 0\}$ ; this follows from continuity of V and the fact that V decreases along trajectories. Let $\propto$ be the set of all such points a in w:

$L = \{ a \in w$ There exist $t_n \rightarrow \infty$ with $X(t_n) \rightarrow a\}$, where X(t) is the trajectory postulated above. Since every point of $\propto$ is a limit of points in P, and P is closed in w, it follows that $\propto \subset P$. Moreover, if $a \in \propto$, then the entire orbit of a is in $\propto$; that is $\emptyset_t(a)$ is defined and in $\propto$ for all $t \in R$.

For $\emptyset_t(a)$ is defined for all t in the interval $[-t_n, 0]$; since $X(t_n) \rightarrow$ a and we may assume $t_1 < t_2 < \ldots\ldots$ , it follows from Fundamental Theory that $\emptyset_t(a)$ is defined for all $t \in [-t_n, 0]$, n=1,2, …… since $-t_n \rightarrow -\infty$, $\emptyset_t(a)$ is defined for all $t \leq 0$. To see that $\emptyset_s(a) \in \propto$, for any particular $S \in R$, note that if $X(t_n) \rightarrow$ a, then $X(t_n+S) \rightarrow {}_s\emptyset$ (a).

We reach a contradiction, for v (a) $\propto$ for all $a \in L$; hence v is constant on an entire orbit in P. this is impossible, hence $\lim_{t\to\infty}(t)$ = x for all trajectories in P. this proves that x is asymptotically stable and so that $P \subset B(\pi)$.

Thus the proof for theorem 2.

(Morris et al, 1974)

## 3.5 PERIODIC SOLUTION AND LIMIT CYCLES

The existence of closed orbit, or periodic solutions often play an important role in physical problems because they represent phenomena that occurs repeatedly. In many situations periodic solutions represent a final state toward which neighboring solutions tend as the transients due to the initial condition die out.

A special case of periodic solution is a constant solution $x=x^o$ which corresponds to a critical point of an autonomous system. Such a solution is clearly periodic with any period in this section when we speak of periodic solution; we mean a non constant periodic solution.

Remember that the solutions of the linear autonomous system $x^1=Ax$ are periodic. If and only if the eigenvalues of A are purely imaginary. Then every solution of the linear system above is periodic, while if the eigenvalues are not purely imaginary, then there are no (non constant) periodic solutions Example, discuss the solution of the system

$$\begin{pmatrix} x^1 = y + x - x(x^2 + y)^2 \\ y^1 = -x + y - (x^2 + y^2) \end{pmatrix} \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots 10$$

It can be seen clearly that (0,0) is the only critical point of the system and also that the system is almost linear in the neighborhood of the origin if the system is liberalized by using the Jacobian matrix, we have $\begin{pmatrix} x \\ y \end{pmatrix}^1 = \begin{pmatrix} 1 & 1 \\ -1 & 1 \end{pmatrix} \ldots\ldots\ldots 11$

This has eigenvalues $1\pm i$. therefore the origin is an unstable spiral point both for the linear system (11) and for the non linear system (10). Thus any solution that starts near the origin in the phase plan will spiral away from the origin. Since there are no other critical points, we might think solution of equation (10) correspond to trajectories that spiral out to infinity. However, it can be shown that this is incorrect because far away from the origin the trajectories are directed outward. It is convenient to introduce polar coordinate r and $\theta$ where $x = r\cos\theta$, $y = r\sin\theta$ and $r \geq 0$ if we multiply the first equation by x and second equation by y and add, we then obtain $x\frac{dx}{dy} + y\frac{dy}{dt} = (x^2 + y^2) - (x^2 + y^2)^2 \ldots\ldots 12$

Since $r^2 = x^2 + y^2$ and $\frac{rdr}{dt} = x\frac{dx}{dt} + y\frac{dy}{dt}$ it follows that $r\frac{dr}{dt} = r^2\left(1 - r^2\right) \ldots\ldots\ldots\ldots 13$

Then the critical point for r≥0 are the origin and the point r=1, which correspond to the unit circle in the phase plane from equation (12) it follows that$\frac{dr}{dt} > 0$ if r<1 and $\frac{dr}{dt} < 0$ if r > 1

Thus the trajectories are directed outward, inside the circle. Whilst outside the unit circle they are directed inward. Clearly, the circle r=1is a limiting trajectory for the system. To determine an equation for$\theta$, we multiply the first equation by y and the second by x and subtracting to obtain. y $\frac{dx}{dt}$-$x\frac{dy}{dt}$ =$x^2$+$y^2$……14

By using x= rcos$\theta$ and y=-rsin$\theta$ the left side of equation (14) is –$r^2\frac{d\theta}{dt}$ equation if reduces to $\frac{d\theta}{d\theta} = 1$……..15

The system of equations 14 and (15) for $\theta$and r is equivalent to original system (10). One solution of the system (14) and (15) is r=1, $\theta$= -t+$t_0$……..16

Where $t_o$ is an arbitrary constant. As t increases, a point satisfying equation (16) moves clockwise around the unit circle. Thus the autonomous system (10) has a periodic solution. Many other periodic solutions. Many other periodic solutions can be obtained by solving (14) by separation of variables. Hence in this example, the circle r= 1 does not only correspond to periodic solution of the system (a) but also, other unenclosed trajectories spiral toward it as$t \to \infty$. Generally, a closed trajectory in a phase plane such that other unenclosed trajectories spiral toward it, either from the inside or the outside as $t \to \infty$ , is called a limit cycle.

In other wards, periodic orbits that are omega limit or alpha limit set of other orbits are called limit cycles for the system (10).

If all trajectories that start near a closed trajectory (both inside and outside) spiral toward closed trajectory as $t \to \infty$, then the limit cycle is stable since the limiting trajectory is

itself a periodic orbit rather than an equilibrium point. This type of stability is referred to as orbital stability. Closed orbits corresponds to periodic solutions and in this example, each of the periodic solution is a limit cycle.

The following theorem gives the conditions guarantee existence of a closed trajectory (Periodic Solution).( William et al, 1992).

### 3.6 POINCARE-BENDIXSON THEOREM:

Let the functions F and G have continuous first partial derivatives in a domain D of the xy plane. Let D, be a bound sub domain in D and Let R be the region that consist of D, plus its boundary (all points of R are in D). Suppose that R contains no critical point of the system $x^1=f(x, y)$ $y^1= g(x, y)$

If there exits a constant such that $x=\emptyset(t)$, $y=\emptyset(t)$ is a solution of the system that exist and stays in R for all $t\geq t_0$, then either $x=\emptyset(t)$, $y=\emptyset(t)$ is a periodic solution (closed trajectory) or $x=\emptyset(t)$, $y=\emptyset(t)$ spiral towards a closed trajectory as $t \to \infty$. In either case, the system has a periodic solution in R.

#### 3.6.1 THEOREM

Let the function f and g have continuous first partial derivatives in the main D of the xy-plane. A closed trajectory of the system $x1=f(x, y)$ $y^1= g(x, y)$ must necessarily enclosed at least one critical (equilibrium) point. The critical point cannot be a saddle point if it enclosed one critical point Note that if R does contain a closed trajectory, and then necessarily from the latter theorem, this trajectory must enclose a critical point.

However, the critical point cannot be in R. thus R cannot be simply connected it must have a hole.

The pointcare-Bendixson theorem does not hold for a system of 3 dimensions or more.

(Waltman, 1995).

### 3.6.2 THEOREM (NON-EXISTENCE OF CLOSED ORBIT (BENDIXSON-DULAC CRITERION)

Supposed there exits a continuously differentiable function B(x, y) defined on a simply connected domain D. Suppose that, the function $\frac{\partial(Bf)}{\partial x} + \frac{\partial Bg}{\partial y}$ does not change sign in G, then there are no periodic solutions $x^1$=f(x, y) and $y^1$= g(x,y) in D. for instance, consider the system $x^1$=y  $y^1$=-x-(1+$x^2$)y. The origin is a stable spiral using $\beta$=1, $\frac{\partial f}{\partial x} = 0, \frac{\partial g}{\partial y} = -(1 = x^2)$ it follows that $\frac{\partial f}{\partial x} + \frac{\partial g}{\partial y} = 0 - (1 + x^2) < 0$

Hence in conclusion there are no periodic orbits.

Example consider $x^1$=$y^1$  $y^1$=-x-y+$x^2$+$y^2$ chose $\beta$(x, y) =,$e^{-2x}$ then $\frac{\partial}{\partial x}(ye^{-2x}) = -2ye^{-2x}$

and d[ - x – y + $x^2$ + $y^2$ ) e $^{-2x}$ +2y$e^{-2x}$  thus $\frac{\partial}{\partial x}(\beta f) + \frac{\partial(\beta g)}{\partial y} = -e^{-2x} < 0$

Hence from the above theorem, there is no periodic solution in the plan. The bendixson-Dulac criterion introduce here is not flexible, that is it cannot be applied in more general cases, it is restricted to 2-dimensional cases. Hence there is the need to introduce a more general version of this result. (Morris et al, 1974)

### 3.6.3 THEOREM

Let f: $R^3 \rightarrow R^3$ be a Lipschitz continuous vector field and let r(t) be a closed piecewise smooth curve which is the boundary of an orientable smooth surface

S$\subset R^3$.Suppose that g: $R^3 \rightarrow R^3$ is defined and smooth in a neighborhood of S, and that it satisfies gγr(t)fγ(t)] ≤0 (≥0)……17 for all t and( curl g). n≥0(≤0) on S and (curlg). n>0 (<0)  for some point on S. where n is a unit normal to S, then r(t) is not the finite union of solution trajectories of $x^1$= f(x)……19

Which are traversed in the positive sense relative to the direction of n.

Proof

We first note that y (t) is an orbit of solution of (19) if and only if, it is an orbit of the system $x^1$=-f(x), which is transverse in the opposite direction. Thus, the two sets of an inequality in (17) and (18) are equivalent to the first set. By stroke's theorem and by $0<\iint \left(\frac{cur}{g}\right) ndA = \int g[r(t)]r(t)dt$ ... ... .20. Now f(r (t)) is piecewise smooth with $r^1$ (t) = f(r(t), except for finite number of points. Then from (a) . These contradictions (20) and the theorem are proved.( William et al, 1992).



## 3.7 COMPETING SPECIES (THE STRUGGLE FOR EXISTENCE)

Here, we consider a two-species ecosystem in which both species compete for the same limited food supply. We start by considering what happens if only one of the species is present, in this case we assume the logistic model $\frac{dx}{dt}$ =ax-$bx^2$ if y=0 where x and y are the species populations. Similarly $\frac{dy}{dt}$ = cy-$dy^2$ if x=0.

Assuming the growth rate is reduced by a factor proportional to the other species population. Thus the governing equations for this competing species are $\frac{dx}{dt}$ =x(a-bx-my)……21 $\frac{dx}{dt}$ =y(c-dy-nx)……...22

Where a, b, c, d, m and n are positive constants. this system of couple differential equations does not have an analytical solution, so we first find the critical points where $\frac{dx}{dt} = \frac{dy}{dt} = 0$. this gives the three points (0,0), (0, c/d), (a/b,0); and also the solution of bx+my =a nx+dy+c which provided bd-mn≠0 is given by $(\frac{ad-bm}{bd-mn}, \frac{cb-an}{bd-mn})$……23

This critical point could be located in any of the four quadrants of the phase plane depending on the values of the parameters.

We will continue the analysis in the case of the two competing species which are virtually identical; that is we take a=c, b=d it is also assumed that one of the two species is more suitable for competition; for example we take n>m which means that x is stronger. The equations are now

$\frac{dx}{dt}$ =x(a-bx-my)……24

$\frac{dx}{dt}$ = y(a-by-nx)……..25

And we have critical points (0,0), (0,a/b), (a/b, 0), [b(m-b)/(mn-$b^2$), a(n-b)/(mn-$b^2$)

The fourth critical point is in the positive xy quadrant of the phase plane. The critical points are illustrated in the figure below. We also note that $\frac{dy}{dx} = \frac{y(a-by-nx)}{x(a-bx-my)}$ … … … … .26

(i) $\frac{dy}{dx} = 0 \; on \; y = o \; and \; by + nx = a$

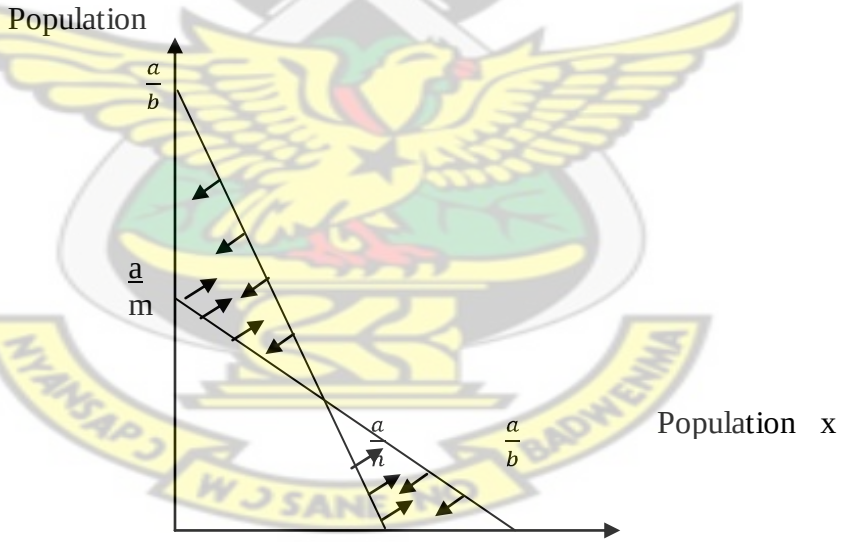


**FIGURE3. 11: COMPETING SPECIES**

(ii) $\frac{dy}{dx} = \infty$ on x=0 and bx+my=a. the directions of the trajectories are sketched on the figure above fig (19).We also note that for x>0 dx > 0 if a > bx + my

dt < 0 if a < bx + my

and so we can put arrows on the trajectories as shown. We can also use the analysis of the critical points to obtain sketches of the trajectories near the critical points, and so we are able to obtain a complete sketch of the trajectories as shown in the figure below. Fig (20)

$\beta > 0$

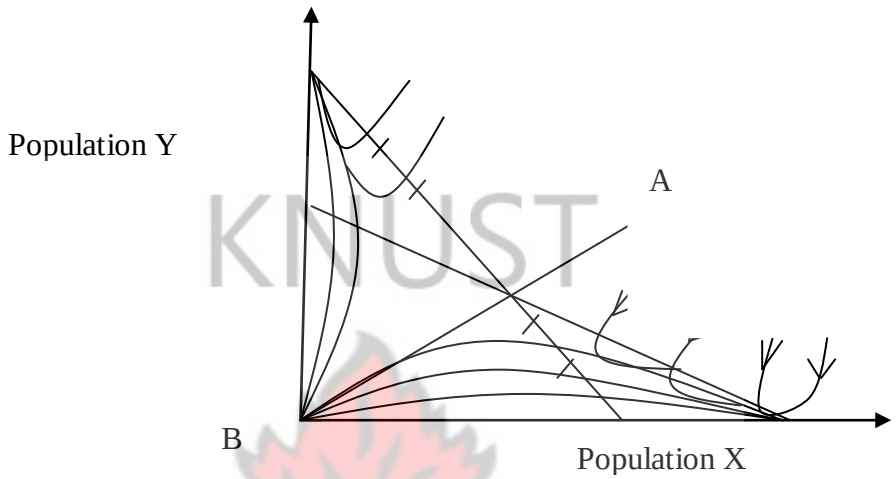


**FIGURE 3.12: TRAJECTORIES OF THE MODEL OF COMPETING SPECIES**

The equilibrium point is unstable, so that coexistence is not possible on the other hand both equilibrium points A and B in which one of the species is extinct are stable. So we conclude that in time one or other of the population will become extinct. This is an example of the principle of competitive exclusion only one of the species can in the long run survive; and although one species, x, is stronger, the trajectories in Fig 20 show that it is still possible for it to be extinct.

(Burgles et al, 1981)

## 3.8 PREDATOR-PREY MODEL

This is the situation where two species live together and one feeds on the other.

Mathematically, we define the prey and predator populations as x and y respectively. Now the governing differential equations for two species interaction can be written as

$$\frac{dx}{dt} = f(x, y)$$
$$\frac{dy}{dt} = g(x, y)$$

For this example, it is assumed that in the absence of predators, the prey will grow unlimited according to dx/dt = $\alpha x$, whilst in the absence of prey, the predators will die out according to dy/dt = - ry

The interaction term is model by xy, positive for the predator, negative for the prey, resulting in the model

$$\frac{dx}{dt} = \alpha x - \beta xy \quad \text{.....38}$$
$$\frac{dy}{dy} = -ry + \delta xy \quad \text{.....39}$$

There are a number of ways one can go about solving these equations. For example we can write $\frac{dy}{dx} = \frac{dy}{dt} / \frac{dx}{dt} = \frac{(-r+\delta x)y}{(\alpha-\beta y)x}$ .....40

Which is a first order variable separable differential equation; which can be solved to give

$$\int \frac{\alpha-\beta y}{y} dy = \int \frac{-r+\delta x}{x} dx$$

That is $\alpha \ln y - \beta y = -r \ln x + \delta x + k$

Where k is constant of integration. The solution can be rewritten as

$$\frac{y^{\alpha} x^{\gamma}}{e^{\beta y + \delta x}} = k^1$$

Where $K^1$ is a constant. This equation defines the x-y solution trajectories but it is not clear what they look like as y(or x) cannot be express as a function of x or y so we use the

phase plane techniques we first note that there are two critical points (0,0) and

$\left(\frac{\gamma}{\delta}\frac{\alpha}{\beta}\right)$.....42

Near the point (0,0), we can approximate (38) and (39) by

$\frac{dx}{dt} = \alpha x, \frac{dy}{dt} = -ry$ *let* $\begin{pmatrix} a & b \\ c & d \end{pmatrix}$ be the jacobian matrix then a= $\alpha$, b=0, c=0 d=-r

$r^2 - \alpha r - \alpha r = 0$ where r is the eigenvalue

$r = \frac{1}{2}\alpha \pm \frac{1}{2}\left(\alpha^2 + 4\alpha r\right)^{1/2}$ that is the case $r_2<0<r_1$ which gives a saddle point at (0,0) For

points near

Where u and v are small in (38) and (39)

We obtain

$\frac{d}{dt}\left(\frac{\gamma}{\delta}+u\right) = \alpha\left(\frac{\gamma}{\delta}+u\right) - \beta(\frac{r}{\delta}+u)(\frac{\alpha}{\beta}+v)$ *that is* $\frac{du}{dt} = -\left(\frac{\beta\gamma}{\delta}\right)v$.....45

That is

Neglecting the 'uv' term. Similarly (39) gives

$\frac{dv}{dt} = \left(\frac{\delta v}{\beta}\right)u$.....(46)

Using the same jacobian $a = 0, b = -\beta\gamma/\delta, c = \delta\alpha/\beta, d = 0$

The eigenvalue $r^2 + \alpha\gamma = 0$ $r = \pm i(\alpha\gamma)^{\frac{1}{2}}$

That is imaginary values hence we have a center

The trajectories are sketched as shown below

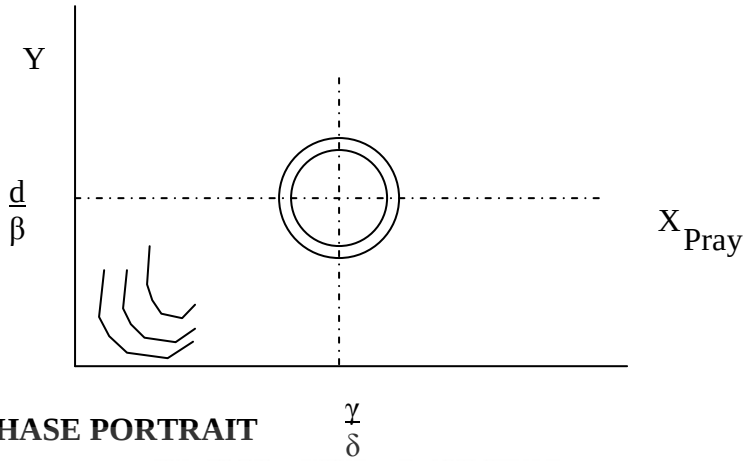


**FIGURE 3.13a : PHASE PORTRAIT**



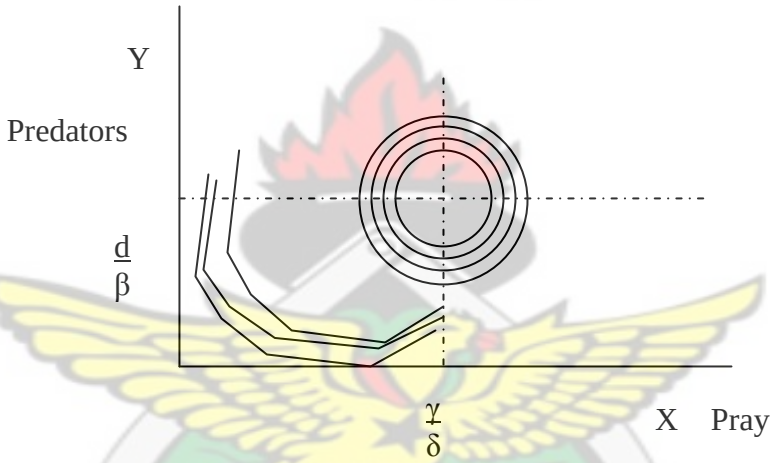


**FIGURE 3. 13: PHASE PORTRAIT**

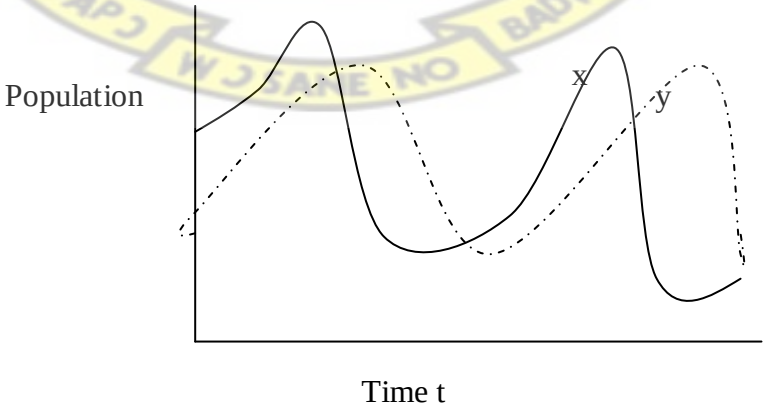


**FIGURE 3.14: TRAJECTORIES FOR PREDATOR-PREY MODEL**

The first property of the trajectories to notice is that they are closed (fig 23aand 23b) so that the solutions are periodic with time and predict that neither predator nor pray ever become extinct.

Also, each trajectory can be divided into four distinct regions (see fig 23b) in this region, the number of predators decrease because of lack of prey, whereas the prey population can increase due to lack of predators in region 11, the population of prey has increased so much that the predator population can also increase.

In region 1 the predator population can also increase.

In region 111 the predator population has increased so much that the prey population is in decline.

In the forth region, due to lack of prey, both predator and prey are in decline.

From fig 24, the average value of the prey and predator over a whole cycle is evaluated as follows

$$\bar{x} = \frac{1}{T}\int_0^T X(t)dt, \qquad \bar{y}\frac{1}{T} = \int_o^T y(t)dt \ldots\ldots\ldots 42$$

Where T is the period of the cycle, now from equation (38)

$$\frac{1}{x}\frac{dx}{dt} = \alpha - \beta y$$

And integrating from t=o to t=T

$$\int_0^T \frac{1}{X}\frac{dx}{dt}dx = \int_0^T (\alpha - \beta y)dt$$

$$\text{i.e} \quad \int_0^T \frac{1}{X}dx = \alpha T - \beta\int_0^T y(t)dt$$

$$N[x(T)/x(0)] = \quad \alpha T - \beta\int_0^T y(t)dt$$

But x(T)= x(0), since it is a complete cycle; so that we obtain

$$0 = \quad \alpha T - \beta \int_0^T y(t)dt$$

Hence from (46) $\bar{y} = \frac{\alpha}{\beta}$.....47

And similarly using $\bar{x} = \frac{\gamma}{\delta}$.....48

So the average value of the predator and prey are in fact their equilibrium values.

(Burgles et al, 1981)



### 3.9 A RATIO DEPENDENT MODEL FOR HIV / AIDS

Ratio dependent Predator-Prey systems arise mainly in ecology. Similar systems, however, may arise (guerrilla) war situations as well. The main feature of such systems is that the response of the predator to the presence of the prey (and vice-versa) depends upon the ratio of the number of prey to the number of the predators. This is in contrast to the situation where such response is dependent on the density of the prey. The ratio dependent model is realistic when the predator has to seek for the prey because then the probability of finding a prey depends upon this ratio. This is also true in guerrilla warfare where again the predator (a regular army) has to search for the prey (the guerrillas) before they can engage them in battle. In the case of infection, let x(t) and y(t) denote the number of HIV negative and HIV positive individuals respectively at any time t in the society.

Now if an HIV negative individual x has sexual contact with other people, then in any one encounter, his chances of having with an HIV positive individual y are proportional to y/(x+y) so that the spread of infection is ratio dependent as well. Such dependent systems may be modeled by differential equations of the type [1]

$$x'(t) = \alpha x(1-x)-xy/(x+y), \quad \text{.....(1.1a)}$$

and $$y'(t) = -ay+kxy/(x+y), \quad (1.1b)$$

for some non-negative parameters a, α, and k. The parameter k is often called the conversion factor. The quantities x (t) and y (t) denote the number (or density per unit area in the x-y plane) of the prey and the predators respectively at any time t. In this system the prey (or the HIV negative individuals) x(t), left to themselves, grow according to the logistic equation $x'(t) = \alpha x(\beta-x)$ where the growth factor of the prey and b, the carrying capacity of the environment, has been non-dimensionalised to one in equations (1.1). In the presence of the predator (or the HIV positive individuals), the prey die (or the HIV negative people become infected) according to the second term $-xy/(x+y) = -y/(1+y/x)$, so that the probability of any one predator finding a prey is equal to k1/(1) for some positive constant k1 which has been non-dimensionalised to one in our equations. If x = 0, this probability is zero which reasonable. Also, this probability increases with x and if x is infinitely large, this probability assumes its maximum value of which is again reasonable. Also, left to themselves, i.e. if x = 0, the predators die according to the equation $y'(t) = -ay$ because have no food or, in the case of the army, they have nothing to do and are withdrawn. In a similar manner, people with AIDS die with the disease. The system of equations (1.1) has been analysed by Kuang and Beretta . A similar system of equations has been proposed by Thompson for the spread of HIV/AIDS disease.

In this section, we look at the system of equations (1.1) in the (x,y) plane. Since the term xy/(x+y) is not defined at (x,y) = (0,0), system cannot be linearised around this point and we do not know the eigenvalues of these equations at this point. The main question we want to ask is, under what conditions on the parameters a, k, and a do the solutions of the system (1.1) approach point (0,0)? In the case of HIV/AIDS

application, the point (0,0) represents the annihilation of the society and our question clearly a pertinent question to ask. We write the system (1.1) as x'(t) = F(x,y), y'(t) = G(x,y) and arbitrarily put F(0,0) = G(0,0) = 0. Notice also that if, as we show later, (x(t),y(t)) stays in the first quadrant for all $t \geq 0$, then the limits of F(x,y) and G(x,y) as (x,y)Æ(0,0) are both zero, so F(x,y) and G(x,y) are continuous in $x \geq 0$, $y \geq 0$. Now the point P1(0,0) is an equilibrium point of this system. The other equilibrium points are $P_2(1,0)$ and $P_3(x_1,y_1)$ where $x_1 = (k\alpha-k+a)/(k\alpha)$ and $y_1 = (k-a)x_1/a$. The eigenvalues at these 'other' equilibrium points, $P_2(1,0)$ and $P_3(x_1,y_1)$, are $(-\alpha,k-a)$ and the two roots$\lambda$ of $\lambda^2+B\lambda+C = 0$ where $B = (a+\alpha-1) - a^2 (k - 1)$ and $C = a(k-a)(k\alpha-k+a)/k^2$, respectively.( Aggarwala, 2001)



### 3.10 A DENSITY DEPENDENT MODEL FOR HIV/AIDS DEVELOPMENT

In this section, a considerably more detailed (than in the previous section), but density dependent, model for propagation of HIV/AIDS in Canada (or in any other community) is developed. This time the society is divided into three groups, those who are HIV negative, those who are HIV positive but have not developed AIDS, and those who have AIDS. It was assumed that without this disease, the present population of Canada, which is at 31 million today, will grow to 50 million at logistic rate, according to the law, $x'(t) = A_1 x-A_2 x_2$, with $A_1 = .05$. This gives a growth rate of .6% when the population is 30 million. consider this to be a reasonable hypothesis. The parameters for the propagation of AIDS are as follows (all population figures are millions. The word 'healthy' in this section means HIV negative);

x(t) = Number of healthy people in Canada at any time t,

y(t) = Number of people who are HIV positive but do not have AIDS at any time t,

z(t) = Number of people with AIDS at any time t,

$A_1$ = Rate of birth of healthy babies near x=0,

$A_1/A_2$ = Maximum number of healthy people that the country can support,

$A_3$ = Rate at which healthy people become infected with HIV by contacting other people who are HIV positive (whether having AIDS or not)

$A_5$ = Rate at which infected (i.e. HIV positive) people develop AIDS disease

$A_6$ = Rate at which infected people die before developing AIDS.

$A_7$ = Rate at which infected babies are born,

k = Rate at which sick people (having AIDS) die.



We assume the values of these parameters as follows:

$A_2 = A_1/50$, which says that without the disease, the population of Canada will grow to a maximum of 50 million people, $A_1$ = .05, which gives the rate of increase of healthy population at .6% when the population is 30 million.

$A_5$ = .09 which says that 90% of the infected people develop the disease within 10 years, so that 10% of them (i.e. 9% of the research paper #811 (2001).nb 15 y's) develop the disease every year, $A_6$ =.005 which says that 10% of the people with HIV never develop the disease and that their life expectancy, from the time contact the immune deficiency, is 20 years.

$A_7$ = .001 which says that if one of the partners is infected, then some of the babies born are infected at birth. The number of babies born every year in the general population being $A_7(y(t)+z(t))$,k = .5 which says that a person who has developed AIDS has a life expectancy of 2 years so that 50% of them die every year.

We believe the values of these parameters to be reasonable. While these values may change with time as technology advances, investigate in this section the effect of a very critical parameter which can be controlled without any further advancement technology. This is the spread of disease through sexual contact and/or sharing of

drug needles. The parameter controlling variable in our model is $A_3$. We show that if $A_3$ is less than a certain critical value $A_3$ cr, then the disease will eventually be wiped out, while if $A_3$ is greater than this critical value (but less than another critical number Acr2, which number we calculate), then number of HIV positive people in the Canadian society will reach a certain equilibrium value which value we calculate, and if larger still, then the society will eventually be almost wiped out, but then grow again.

**The Model:** If x, y, and z are the number of healthy people, the people infected with HIV but not having AIDS, and people with AIDS respectively at any time t, our model is

$$x'(t) = A_1x - A_2x^2 - A_3x(y+z), \quad (2.1a)$$

$$y'(t) = A_3x(y+z) - (A_5+A_6)y + A_7(y+z), \quad (2.1b)$$

$$z'(t) = A_5y - kz. \quad (2.1c)$$

We write u = y+z and treat x, u, and z as the independent variables. Now our equations are

$$x'(t) = A_1x - A_2x_2 - A_3xu, \quad (2.2a)$$

$$u'(t) = A_3xu - (A_6-A_7)u + (A_6-k)z, \quad (2.2b)$$

$$z'(t) = A_5u - (A_5+k)z. \quad (2.2c).$$

Alternatively, we may put v = x+y, w = x+y+z and use x,v, and w as the independent variables. Our equations now become

$$x'(t) = A_1x - (A_2-A_3)x^2 - A_3xw, \quad (2.3a)$$

$$v'(t) = (A_1+A_5+A_6-A_7)x - A_2x^2 - (A_5+A_6)v + A_7w, \quad (2.3b)$$

$$w'(t) = (A_1+A_6-A_7)x - A_2x^2 + (k-A_6)v - (k-A_7)w. \quad (2.3c)$$

We shall work with equations (2.1) or equations (2.2) or equations (2.3) as appropriate. We notice that in the values we have assumed for the constants A's and k, all of them are non-negative. We shall assume this property of these parameters in this

section. Data on AIDS also suggest that usually $A_7$ is quite small (there have been very few HIV positive babies born in Canada during last few years and this number is decreasing) and that k is quite large. We shall assume that $A_7 < A_6 < k$ .

The equilibrium points: The equilibrium points of equations (2.2) are seen to be $P_1$ (0, 0, 0), $P_2$ ($A_1$ ê $A_2$, 0, 0

$P_3$ ($x_0$, $u_0$,$z_0$) where

$x_0 = ((A_6 - A_7)\, k + A_5\, (-A_7 + k))$ ê $(A_3\, (A_5 + k))$, (2.4a)

$u_0 = (A_1 - A_2\, x_0)\, /A_3$ , (2.4b)

and $z0 = A_5\, u_0 / (A_5 + k)$. (2.4c)

These three points correspond to the society being annihilated, the disease being annihilated, and the disease becoming endemic respectively.

**Positivity of the solution**: Using equations (2.1), we shall show that if x(0), y(0), and z(0) are positive, then x(t), y(t) and are non-negative for all t≥0. Notice that if x(t) = 0, then x'(t) = 0, which shows that if the moving point (x(t),y(t),z(t)) hits the plane x = 0, it cannot move away from it. Since x(0)>0, this gives x(t)≥0 for all t≥0. Equation (2.1c) gives

$$z(t) = z(0)e^{-kt} + A_5\, e^{-kt} \int_0^t e^{ks}\, y\,(s)\, ds \qquad (2.5)$$

Now at y = 0, equation (2.1b) gives y'(t) = $(A_3x(t)+A_7)z(t)$. Since y(0)>0, there is a first time t = $t_1$ say, when $y(t_1) = 0$.

implies that y(t)≥0 in 0£t£t1. Since z(0)>0, this in turn implies from equation (2.5) that $z(t_1)>0$. Also $x(t_1)\geq 0$. This gives $y'(t_1)>0$ so that the moving particle must bounce back into the first octant. Repeating the argument, we get y(t)≥0 for all t≥0, then from equation(2.5), z(t)≥0 for all t≥0. This proves the positivity of the solution.

However, notice that if in equations (2.2), x and u are both small (near the origin), then the first term on the right hand side becomes small of second order and u'(t) may become negative in a computer solution. This may make u(t) negative (due to a finite step numerical calculations). This difficulty is somewhat overcome by using equations (2.3) in a numerical solution.

**Boundedness of the solution**: Equations (2.2) give $(x(t)+u(t))' = A_1 x - A_2 x^2 - (A_6 - A_7) u - (k - A_6) z$. Since $A_7 < A_6 < k$, $z(t) \geq 0$, and $A1 > 0$, this gives $(x+u)' < 0$ if $(x+u) > N1$, where N1 is some finite number. To see this, we draw parabola $A_1x - A_2x^2-(A_6 - A_7)u = 0$ in the x-u plane. The constant $N_1$ is any number which places the line $x+u = N_1$ outside to the right of) this parabola (see Fig.10). Since u = y+z and x,y, and z are all non-negative, this proves that the solution of equations is bounded in $t \geq 0$.

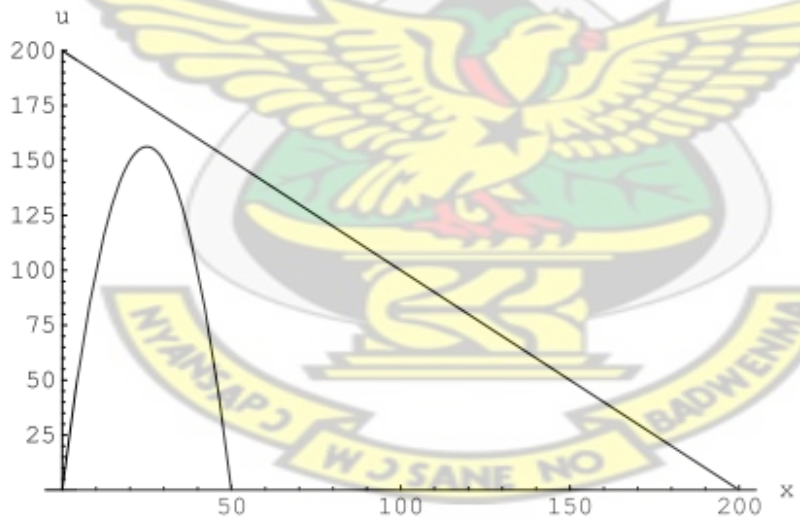


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**FIGURE 3.15: THE PARABOLA AND THE LINE $x+u = N_1$**

This diagram shows a triangle bounded by x=0, y=0 and a line $x+y=N_1$ for a suitable number $N_1$. All solutions of equations (2.1) are bounded by this triangle.

Stability of equilibrium points: Since $A_1 > 0$, the point P1 is always unstable. The point $P_2$ will be unstable

$$a_0 = A_3 x_0 (A_1 - A_2 x_0) (k + A_5)$$
$$a_1 = A_2 x_0 (k + A_5 + A_6 - A_7) + A_1 A_3 x_0 - 2 A_2 A_3 x_0^2$$
$$a_2 = (A_2 - A_3) x_0 + k + A_5 + A_6 - A_7, \quad \text{and where}$$

$$x_0 = ((A_6 - A_7) k + A_5 (-A_7 + k)) / (A_3 (A_5 + k)).$$

It can be easily shown that $(A_5+k)(A_6 - A_7 - A_1 A_3 \hat{e} A_2)-A_5(A_6-k) = -(A_3 2/A_2)(A_5 + k)u0$, so that if $u0 \geq 0$, the condition (b) satisfied and the point $P_2$ is unstable. Also if condition (a) is satisfied, then so is condition (b) and therefore $u0 \geq 0$. To see suppose condition (a) is satisfied and we write $A_6-A_7-(A_1A_3/A_2) = -b$ for some $b \geq A_5+k > 0$, then condition (b) requires $(A_5+k)(-b)-A_5(A_6-k)£0$, or $b(A_5+k) \geq kA_5-A_5A_6$, which is true since $(A_5 + k)^2 > kA_5$ for non-negative numbers $A_5$ and follows that $P_2$ is unstable if and only if $u0 \geq 0$.

The equilibrium point $P_3$ is not reachable by the moving point (x,u,z) if $u0 < 0$. If $u0 > 0$, we must determine the stability otherwise of the point (x0, u0, z0) from the characteristic equation of equations (2.2) at this point. This equation turns out to

$a_0 + a_1 \lambda + a_2 \lambda^2 + \lambda^3 = 0$ where,

$a_0 = A_3 x_0 (A_1 - A_2 x_0) (k + A_5)$

$a1 = A_2 x0 (k + A_5 + A_6 - A_7) + A_1 A_3 x0 - 2 A_2 A_3 x02$

$a_2 = (A_2 - A_3) x0 + k + A_5 + A_6 - A_7,$ and where

$x0 = ((A_6 - A_7) k + A_5 (-A_7 + k)) \hat{e} (A_3 (A_5 + k))$.

Under the assumption $k > A_6 > A_7$, the constant a1 is seen to be positive. The condition for stability of the equilibrium point now becomes $a1\ a_2 - a_0 > 0$. For the values of various parameters given in the beginning, the equation $a1\ a_2 - a_0 = 0$ two roots, namely $A_3 = A_3\,cr1$ and $A_3 = A_3\,cr2$ where the critical number $A_3\,cr1 = -.000125692$ and $A_3\,cr2 = .0728806$ approximately. However for $A_3$ less than a certain critical number

A3 cr, we get u0<0 and the point (x0,u0,z0) is not reachable from the first octant and ($A_1/A_2$, 0, 0) is the proper (stable) equilibrium point. For values of the various parameters that we assumed, we get $A_3$ cr= .00159017 approximately. In $A_3$ cr < $A_3$ <$A_3$ cr2, the point ($x_0$,$u_0$,$z_0$) is reachable and is the relevant (stable) equilibrium point. For $A_3$>$A_3$ cr2, the equilibrium point ($x_0$, $u_0$, $z_0$) becomes unstable. All the three equilibrium points are now unstable and computer experiments show that the solution approaches the origin in the beginning, i.e. the society tends annihilate itself. However the origin is unstable (where one eigenvalue is positive and the other two are negative), so that solution runs away from the origin and comes back to it repeatedly. We show the path of (x,v,w) in one particular case. It appears that the solution runs into a limit cycle.

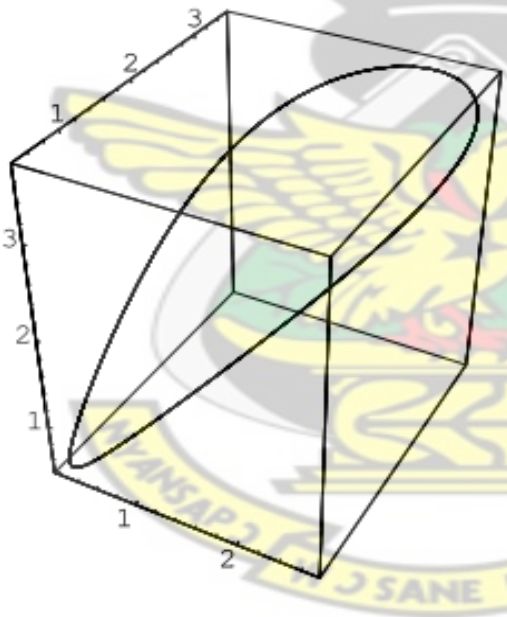



**FIGURE 3.16 : THE SOLUTION TO EQUATION 2.3**

For $A_2 = A_1/50$ , $A_5 = .09$, $A_6 = .005$, $A_7 = .001$, k = .5, $A_1 = .05$ and $A_3 = .08$ from t = 9000 to t = 10,000 starting at $(x_0,v_0,w_0) = (2.63,2.95,3.00)$.

Our model therefore gives two equilibrium values of the dynamic. In one case, the disease is eradicated, $u_0 = y_0 + z_0 = 0$, and point (A1 ê $A_2$,0,0) is a stable equilibrium point. In the other case, the disease persists, $u_0 > 0$, and the point ($x_0$, $u_0$, $z_0$ (possible) stable equilibrium point. In this scenario, the healthy (i.e. HIV negative) and HIV positive people will approach a stage stable coexistence so that public policies can be designed to cope with the situation. But this happens only for some (moderate) range of values of $A_3$, namely $A_3$ cr$<A_3<A_3$ cr2, where the values of these constants are given above. If the sexual (and/or needlesharing) attitudes of the Canadian population become such that $A_3>A_3$ cr2, then, according to this model, the Canadian society eventually perish but will be replenished later because healthy babies keep getting born. At present time, A3 is approximately equal to .0023, the disease is endemic in Canada, and if we wish to eradicate the epidemic of AIDS from our society, this constant has come down considerably. In the case when u0 > 0, and the point (x0, u0, z0) is a stable equilibrium point, the moving point (x,y,z) approaches the point an oscillatory manner (two of the eigenvalues are complex conjugate). To explore the nature of these oscillations, we notice that large values of t (and near this equilibrium point), the quantity $A_5u-(A_5 + k)z$ is small (because this quantity is zero at equilibrium point). In Canada at present time for example, while x' and y' are in hundreds of thousands and thousands respectively,

z' is in hundreds and dropping and we may take z' to be small. To explore such oscillations, we take $A_5u-(A_5 + k)z = e$ and ignore the small quantity e. Now the other two equations become

$x'(t) = A_1x-A_2x^2-A_3xu,$ (2.4a)

$u'(t) = A_3xu-(A_6-A_7)u+(A_6-k)(A_5u/(A_5 + k)).$ (2.4b)

We write $x = x0\ (1 + \bar{x})$, and $u = u0\ (1 + u)$. Our equations now become

$x'(t) = -A_1\ (x+ x^2)+A_3\ u0(x\ uxu+ x^2),$ (2.5a)

$'(t) = A_3\ x_0\ x(1 + u).$ (2.5b)

Near the equilibrium point, x and u are small and the linearised form of equations (2.5) gives

$x^2+(A_2\ x_0)x'+(A_3^2\ x_0\ u_0)x = 0.$ (2.6)

which represents the motion of a (linear) damped pendulum as expected.

(Aggarwala.2001)

# CHAPTER 4

# FORMULATION OF PREDATOR-PREY MODEL OF HIV PROPAGATION

## 4.0 INTRODUCTION

In this chapter, some behavioral and biological assumptions leading to the formulation of a predator -prey model of HIV propagation is made, analysis of the model is done especially in terms of the determination of the stability of the equilibrium points and the identification of types of trajectories. Parameter determination is also done and finally, simulation using matlab is done to see how the theoretical model works practically.



## 4.1 MODEL ASSUMPTIONS

In the formulation of the envisaged HIV model, the following assumptions are made.

1. HIV virus is transmitted mainly through heterosexual intercourse.
2. There exist assymetry of transmission in that transmission from males to females is much higher than from females to males. This is due to two reasons. One being behavioral based and the other being biologically.
    - Males tend to be more promiscuous than females in other words males tend to have more sexual partners than females.
    - Biologically in any sexual encounter between an infected male and an uninfected female, the probability of infecting an uninfected partner is higher than that between an infected female and an uninfected male.
3. For the old infected cases of HIV the rate of infection is equal to the rate of removal. The number of old infected cases is constant.

## 4.2 MODEL FORMULATION

Let x(t) be the number of newly infected males and

y(t) be the number of newly infected females.

Y(t) be the number of old infected female

X(t) be the number of old infected male

Then Y(t) + y(t) = N(y) is old and new HIV infected females cases at time t

X(t) + x(t) = N(x) is old and new HIV infected males cases at time t



LINEAR TERM

In the absence of newly infected females, newly infected males tend to decrease exponentially. Thus

$\frac{dx}{dt} = -bx$ this is because the old cases of infected females are not active enough to cause significant increase in the number of newly infected males.

In the absence of newly infected males, newly infected females tend to increase exponentially. Thus

$\frac{dy}{dt} = ay$ this is because old cases of infected males are active enough in causing significant increase in newly infected females.

NON LINEAR TERM:

The non linear terms are obtained through mass action law:

In the absence of old cases of infected females, new cases of infected males tend to increase because new cases of infected females are active in causing new cases of infected males. Thus $\frac{dx}{dt} = cxy$

In the absence of old cases of infected males, new cases of infected females tend to decrease. This is because new cases of infected males do not cause significant increase in newly infected cases of females. Thus

$\frac{dy}{dt} = -dxy$ Where d is a constant.

Combining the linear and non linear parts of the model we get :

$\frac{dy}{dt} = ay - dxy$ and

$\frac{dx}{dt} = -bx + cxy$

This is a typical Lotka- Volterra predator – prey model.

On the other hand, x(t) +X(t) = Nx

$$X(t) = Nx - X(t)$$

$$Y(t) + Y(t) = Ny$$

$$Y(t) = Ny - Y(t)$$

$\frac{d(Ny-Y)}{dt} = a(Ny - Y) - d(Nx - X)(Ny - Y)$

$\frac{dNx-X}{dt} = -b(Nx - X0 + c(Nx - X)(Ny - Y)$ . Thus we obtained the model in terms of old cases of HIV infection

## 4.3 ANALYSIS OF THE MODEL

At the equilibrium point

$\frac{dx}{dt} = 0$ and $\frac{dy}{dt} = 0$

$\Rightarrow -bx + cxy = 0$

$-x(b - cy) = 0$

X=0 or y=0

Also ay-dxy=0 $\Rightarrow$ y = 0 or $X = \frac{a}{d}$

Hence the critical points are (0,0) and $\left(\frac{a}{d}, \frac{b}{c}\right)$

Using the Jacobean matrix

$$J(x, y) = \begin{pmatrix} -b+cx & cx \\ -dy & a-dx \end{pmatrix}$$



$$At\ (0,0) = \begin{pmatrix} -b & 0 \\ 0 & a \end{pmatrix}$$

The eigenvalue are $\lambda_1 = -b$ and $\lambda_2 = a$

Hence the origin is a saddle point and therefore unstable. This point is also unstable because in the absence of new cases of males and females, the old cases of males are still aggressive enough to sustain increase in new cases of females.

For the critical point $\left(\frac{a}{d}, \frac{b}{c}\right)$

$$J\left(\frac{a}{d}, \frac{b}{c}\right) = \begin{pmatrix} 0 & \frac{ac}{d} \\ \frac{-bd}{c} & 0 \end{pmatrix}$$

Hence the eigenvalues are $\lambda^2 + ba = 0$ $\Rightarrow$ $\lambda = \pm i\sqrt{ab}$

Thus the eigenvalues are imaginary; hence the critical point is a centre (stable) of the linear system. This stable point is obtained when new cases of males interact with new cases of females. Here both males and females are infected therefore there will be no increase in either sex but after some time then we begin to see increase or decrease in new cases

To find the trajectories to the system, we consider the critical point

$$\left(\frac{a}{d}, \frac{b}{c}\right)$$

and $x^1 = 0$, $y^1 = 0$ and draw the two axes as shown below

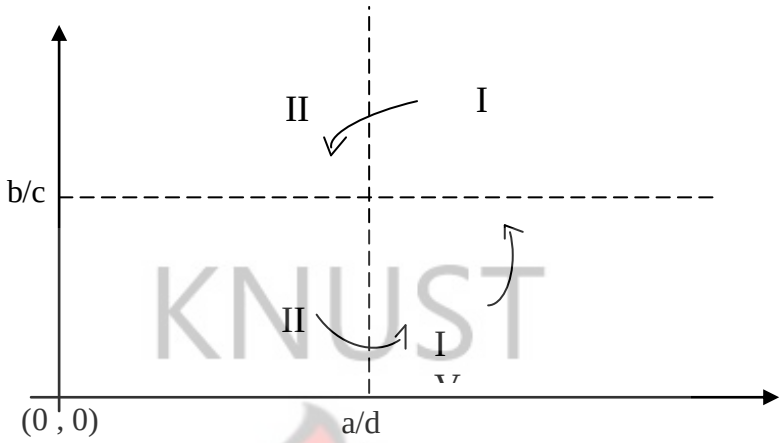



**FIGURE 4.1: A DIAGRAM, SHOWING THE TRAJECTORIES IN THE VARIOUS QUADRANTS.**

These divide the region x. > o, y > o into four quadrants as shown on the diagram

The signs of $x^1$ and $y^1$ are constant as indicated in each quadrant. The positive x – axis and the positive y – axis are each trajectories as indicated in fig 4.1. Each solution curve (x(t),) y(t)) moves anticlockwise around z from one quadrant to the other. Consider for example a trajectory (x(t)), y (t) ) starting at a point. x (0) = u > a/d > 0

y(0) = v > b/c > 0 in quadrant 1

There is a maximal interval say (o, α) = J such that ) x (t)), y (t) ) Є quadrant 1 for $0 \leq t, < 1$ ( perhaps α = ∞) put b- cv = - r < 0, du – a = S > o as long as tЄ J, x(t) is decreasing and y (t) is increasing. Hence

$$\frac{dlogy(t)}{dt} = \frac{y1}{y} = dx \; -a > s$$

$$\frac{dlogx(t)}{dt} = \frac{x1}{x} = b - cy < -r$$

therefore a/d $\leq$ x(t) $\leq$ ue $^{-rt}$ ..... 2

b/c $\geq$ y (t) $\geq$ ve $^{st}$ .....3

For o $\leq$ t α. from the second inequality of (2) we see that α is finite.

From (2) and (3) we see that for t $\epsilon$ a/d $\leq$ x(t) $\leq$ u

b $\leq$ y (t) = ve $^{s\alpha}$

Therefore (x(α), y (α.) ) is defined and in the boundary of the region since x (t) is decreasing x (α) = a/d thus the trajectory enters quadrant II. Similarly for other quadrants however, it is not clear whether trajectories spiral in toward z, spiral toward a limit cycle or spiral out towards infinity and the coordinate axes. We find a liapunov function H

Thus $H(x,y) = F(x) + G(y)$

We want $H < 0$ $where$ $H(x,y) = \frac{dH}{dt}(x(t), y(t))$

$$= \frac{dF}{dx}x^1 + \frac{dG}{dy}y^1$$

Hence $H(x,y) = \frac{xdF}{dx}(b - cy) + \frac{ydG}{dy}(cx - a)$

We obtain $H = 0$ $provided$ $\frac{\frac{xdF}{dx}}{dx-a} = \frac{\frac{ydG}{dy}}{cy-b}$

Since x and y are independent variables this is possible if and only if

$$\frac{\frac{xdF}{dx}}{dx-a} = \frac{\frac{ydG}{dy}}{cy-b} \qquad = \text{constant}$$

Let the constant be equal to one (1) then, $\begin{pmatrix} \frac{dF}{dx} & d-\frac{a}{x} \\ \frac{dG}{dy} & c-\frac{b}{y} \end{pmatrix}$ ……..4

Integrating we have $F(x) = dx - a\log x$

$G(y) = cy - b\log y,$ thus the function

$H(x,y) = dx - a\log - b\log y \; defined \; for \; x > 0, y > 0$

Is constant on the solution curves of ……. ( 1)

By considering the signs of $\frac{\partial H}{\partial X}$ $and$ $\frac{\partial H}{\partial y}$ we see that the equilibrium

$Z = \left(\frac{a}{d}, \frac{b}{c}\right)$ is an absolute

Minimum for H, it follows that H (specifically) H(t) is a Liapunov function. Therefore z is a stable equilibrium. We also note that H is not constant on any open set therefore there are no limit cycles

Also in moving from quadrant I to II it is observed that new cases of females tend to increase whiles new cases of males tend to decrease until one gets into quadrant II where new cases of both males and females tend to decrease until one gets to quadrant III where new cases of females continue to decrease, whiles that of males tend to increase. In quadrant IV, there is an increase in both new cases of males and that of females until one gets into quadrant I, and the cycle is repeated. From this it can be concluded that it is advisable to aim control programme at quadrant IV, where new cases in both males and

females are increasing and work towards quadrant II, where both cases of HIV is on the decrease.

Theorem: Every trajectory of the volterra Lotka equations (1) is a close orbit (except the equilibrium z and the coordinate axes) .

Proof: consider a point w = (u,v), u >. o  v > o; ω ≠ z then there is a doubly infinite sequence …….< $t_{-1}$ < , $t_o < t_1$ <…… such that Ø $t_n$ (w) is one the line x = a and

$$Ø\, t_n\,(\omega) \longrightarrow z \quad as \quad n \longrightarrow \infty \quad or$$

$$Ø\, t_n\,(\omega) \longrightarrow z \quad as \quad n \longrightarrow -\infty$$

Since H is constant on the trajectory of w, this implies that H(W) = H (z) but this contradicts minimality of H(Z) Hence we have the following schematic phase portrait below.

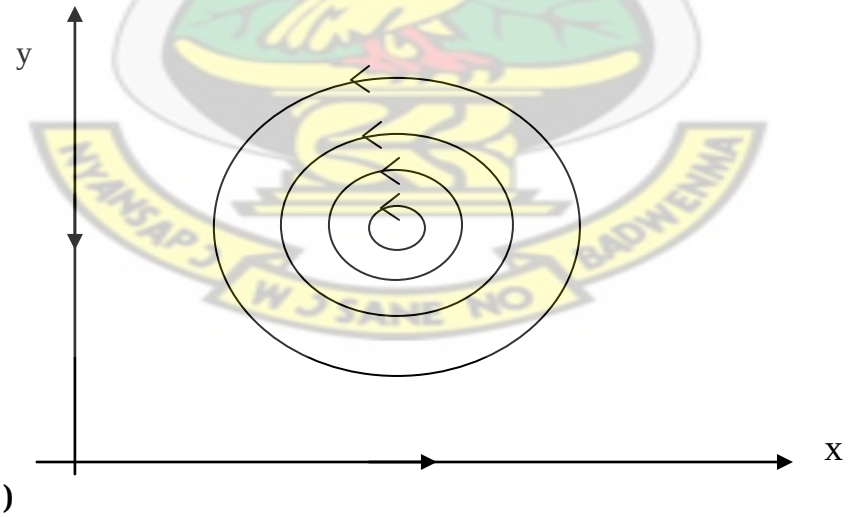



**FIGURE 4.2: PHASE PORAIT 0F THE MODEL**

Therefore, for any initial population (x(o), y(o)) with x (o) ≠ 0 and y (o) ≠ 0 other than z, the operations of newly infected males and newly infected females will oscillate cyclically. /no matter what the numbers of infected and susceptible are, neither species will die out nor will it grow indefinitely.

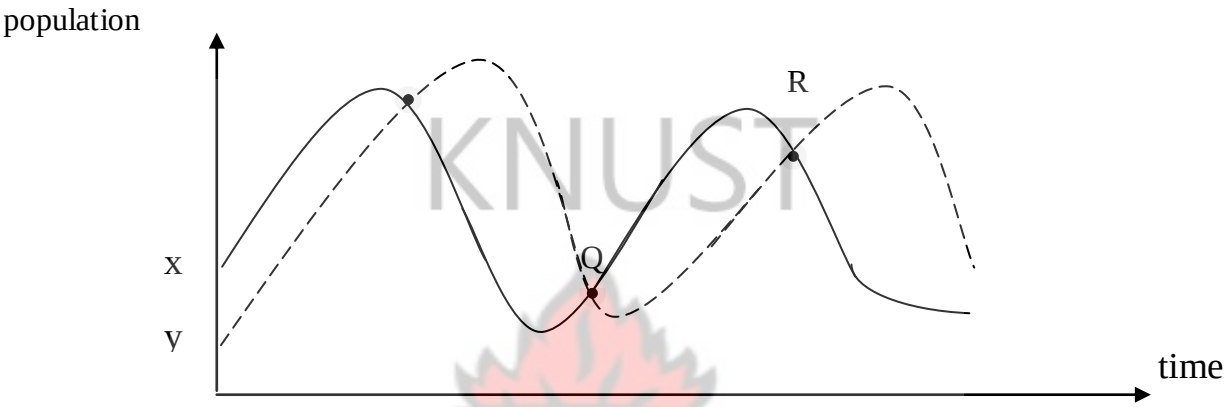



**FIGURE 4.3: TRAJECTORIES OF THE MODEL**

On the other hand except for the state z, which is improbable the populations will not remain constant.

From the graph of the populations of newly infected males and females against time, it is observed that both new cases of males and females begin to rise, but just before the intersection at P, new cases of males tend to decrease whilst new cases of females continue to increase. This can be related to the movement of quadrant IV to quadrant I in figure 1. New cases of females continue to increase until it attains its maximum and begins to decrease as that of males until new cases of males attain it maximum value first and begins to increase again until another equilibrium point is reached at Q.

After the point Q, new cases of males continue to increase whilst that of the females continue to decrease until it get to its maximum point and also begins to increase as that of the males. This can also be compared to the movement from quadrant III to quadrant IV on figure 1 as explained earlier and the cycle is repeated.

## 4.4 PARAMETER DETERMINATION

At the stable point

$(x, y) = (0, 0)\ and\ \left(\frac{a}{d}, \frac{b}{c)}\right)$ as obtained earlier in chapter four. Also considering the linearised form of the model

$$J(x, y) = \begin{pmatrix} -b+cy & cx \\ -dy & -dxa \end{pmatrix}$$

At the point $\left(\frac{a}{d}, \frac{b}{c}\right)$

$$J\left(\frac{a}{d}, \frac{b}{c}\right) = \begin{pmatrix} 0 & \frac{ac}{d} \\ \frac{-bd}{c} & 0 \end{pmatrix}$$

hence the eigenvalues are given by $\lambda^2 + ab = 0$

$$ab = -\lambda^2$$

That is constant in the form $ab = \omega^2$ where $\omega$ is a constant , the frequency of oscillation of some observed HIV/AIDS data of Ashanti region. ω = 1.45.

Therefore $ab = 1.45^2$

$$ab = 2.1$$

now assuming $b = 0.9$

$$0.9a = 2.1$$

$$a = \frac{2.1}{0.9} = 2.3$$

Also assuming at the stationary point $(a/d, b/c)$

$$a/d = b/c = 0.5$$

$$\frac{2.3}{d} = 0.5$$

$$d = \frac{2.3}{0.5} = 4.2$$

And

$$\frac{0.9}{c} = 0.5$$

$$c = \frac{0.9}{0.5} = 1.8$$

Hence, the model is

$$\frac{dy}{dt} = 2.3y - 4.6xy$$

$$\frac{dx}{dt} = -0.9x + 1.8xy$$

Assuming a = 1.1 , b = 1.9, $\frac{a}{d} = 0.3$ , $\frac{b}{c} = 0.7$

Then $\frac{1.1}{d} = 0.3$

d = $\frac{1.1}{0.3}$ ,d =3.7

also $\frac{1.9}{c} = 0.7, c = \frac{1.9}{0.7}, c = 2.7$

therefore the model is $\frac{dx}{dt} = -1.9x + 2.7xy$

$$\frac{dy}{dt} = 1.1y - 3.7xy$$

Also assuming a =1.1 , b = 1.9 , $\frac{a}{d} = 0.5$ , $\frac{b}{c} = 0.3$

Then $\frac{1.1}{d} = 0.5, d = \frac{1.1}{0.5} = 2.$

$\frac{1.9}{c} = 0.3, c = \frac{1.9}{0.3} = 6.3$

Therefore the model is also written as $\frac{dx}{dt} = -1.9x + 6.3xy$

$$\frac{dy}{dt} = 1.1y - 2.2xy$$

Taking a = 1.9 , b = 1.1 , $\frac{a}{d}$ = 0.3, $\frac{b}{c}$ = 0.7

Then $\frac{1.9}{d}$ = 0.3 , d = $\frac{1.9}{0.3}$ =6.3

$\frac{1.1}{c}$ = 0.7 , c = $\frac{1.1}{0.7}$ = 1.6

Therefore the model is written as $\frac{dx}{dt}$ = -1.1x + 1.6xy

$\frac{dy}{dt}$ = 1.9y – 6.3xy

Also assuming a =0.9 , b = 2.3 , $\frac{a}{d} = \frac{b}{c}$ = 0.5

Then $\frac{0.9}{d}$ = 0.5 , d = $\frac{0.9}{0.5}$ = 1.8 . $\frac{2.3}{c}$ = 0.5 , c = $\frac{2.3}{0.5}$ = 4.6.

Therefore the model is also written as $\frac{dx}{dt}$ = -2.3x + 4.6xy , $\frac{dy}{dt}$ = 0.9y – 1.8xy

## 4.5 SIMULATION

Phase portrait for higher initial male AIDS rate

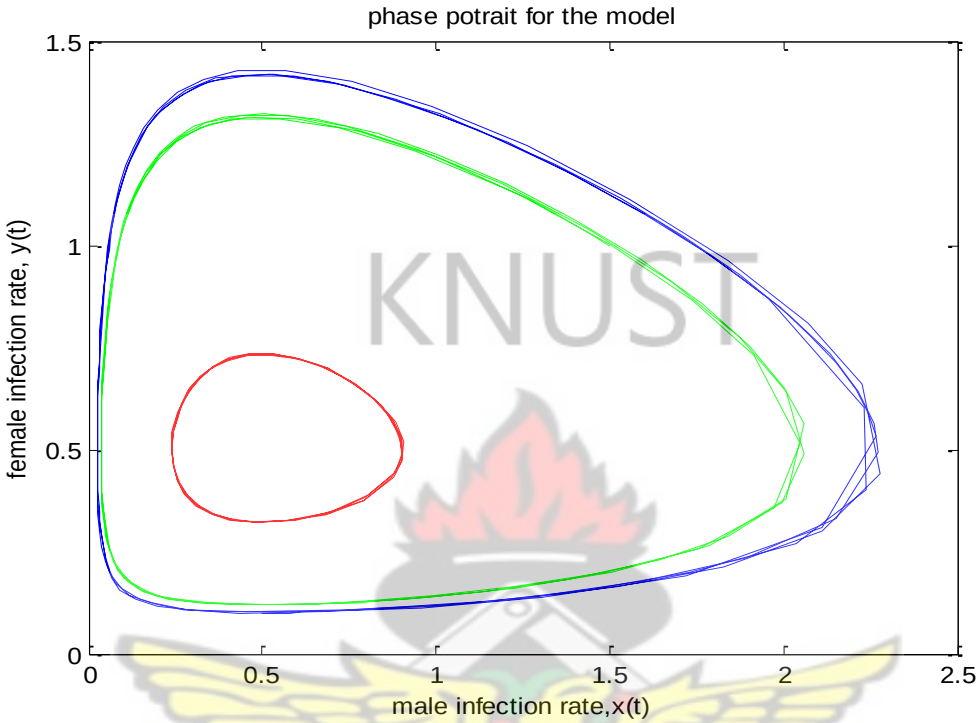



FIGURE 4.4: PHASE PORTRAIT OF THE MODELSHOWING DIFFERENT INITIAL VALUES

```
% Matlab code for solving the Predator-Prey system
% Coefficients for the equations
a=2.3; %male AIDS growth rate
b=4.6; %male rate decline coefficient
c=1.8; %female AIDS growth coefficient
d=0.9; %female decline rate
% Variables used as input in the ODE-solver
maleIni=0.9;
femaleIni=0.5;
% Solve ODE system
[T,Y] = ode45(@male_female,[0 20],[maleIni femaleIni]);
male_rate=Y(:,1);
female_rate=Y(:,2);
% plot for male rate and female rate over time
figure(1)
plot(T,male_rate,'r',T,female_rate,'--b')
xlabel('Time t')
ylabel('male rate/female rate')
legend('employment rate','wage share')
title('model Solution over time')
%phase portriat plot
figure(2)
plot(male_rate,female_rate,'r')
xlabel('male rate,x(t)')
ylabel('female rate, y(t)')
title('phase potrait for the model')
%--- End of code ---
```

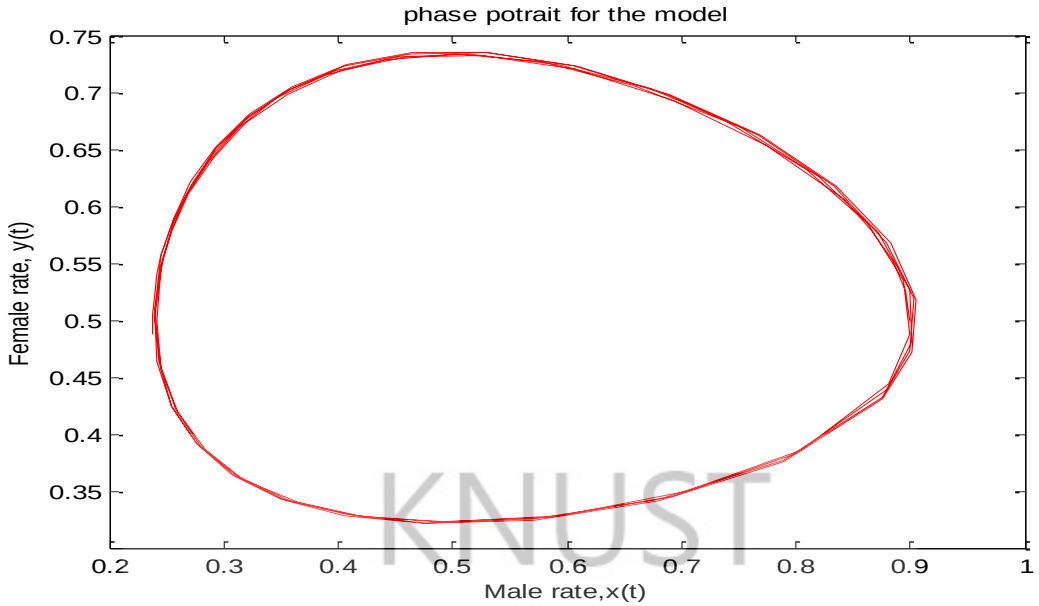



**FIGURE 4.5 : PHASE PORTRAIT FOR LOWER INITIAL MALE AIDS RATE**

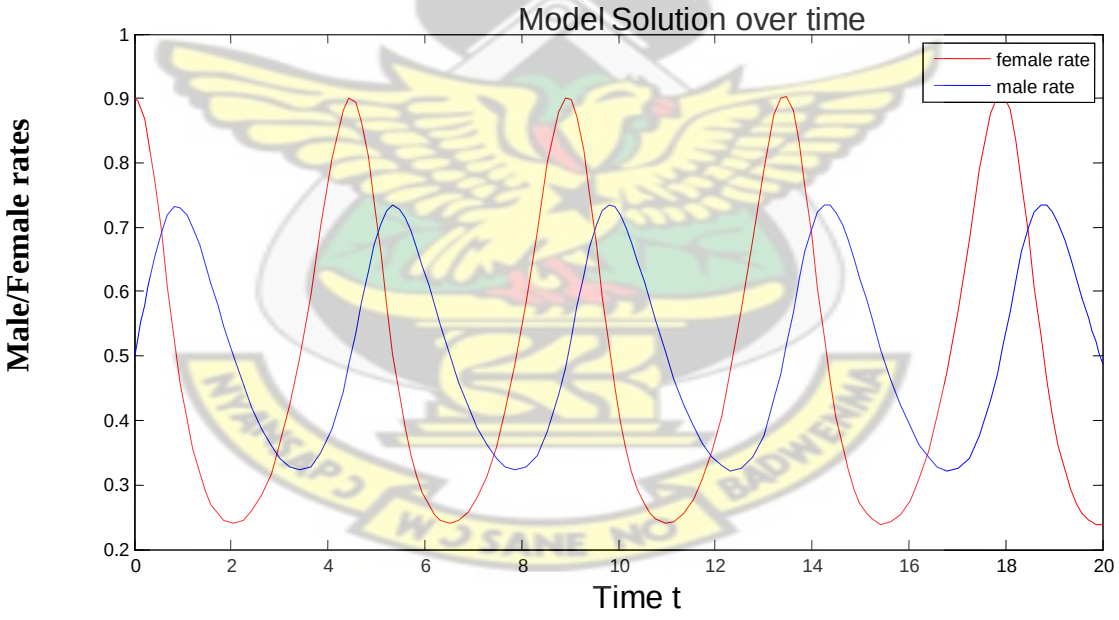



**FIGURE 4.6 : MODEL SOLUTION FOR LOWER INITIAL MALE AIDS RATE**

% Matlab code for solving the Predator-Prey system

% Coefficients for the equations

a=2.3; %male AIDS growth rate

b=4.6; %male rate decline coefficient

```
c=1.8; %female AIDS growth coefficient
d=0.9; %female decline rate
% Variables used as input in the ODE-solver
maleIni=0.5;
femaleIni=0.9;
% Solve ODE system
[T,Y] = ode45(@male_female,[0 20],[maleIni femaleIni]);
male_rate=Y(:,1);
female_rate=Y(:,2);
% plot for male rate and female rate over time
figure(1)
plot(T,male_rate,'r',T,female_rate,'--b')
xlabel('Time t')
ylabel('male rate/female rate')
legend('employment rate','wage share')
title('model Solution over time')
%phase portriat plot
figure(2)
plot(male_rate,female_rate,'r')
xlabel('male rate,x(t)')
ylabel('female rate, y(t)')
title('phase potrait for the model')
%--- End of code ---
```

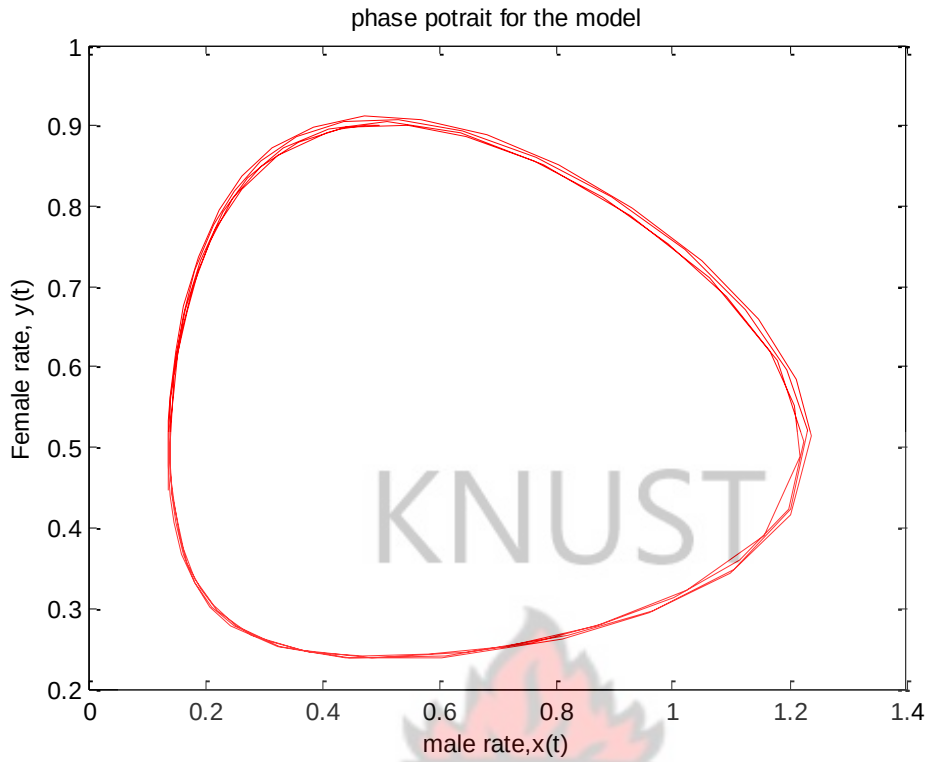



**FIGURE 4.7 : PHASE PORTRAIT FOR HIGHER INITIAL MALE AIDS RATE**

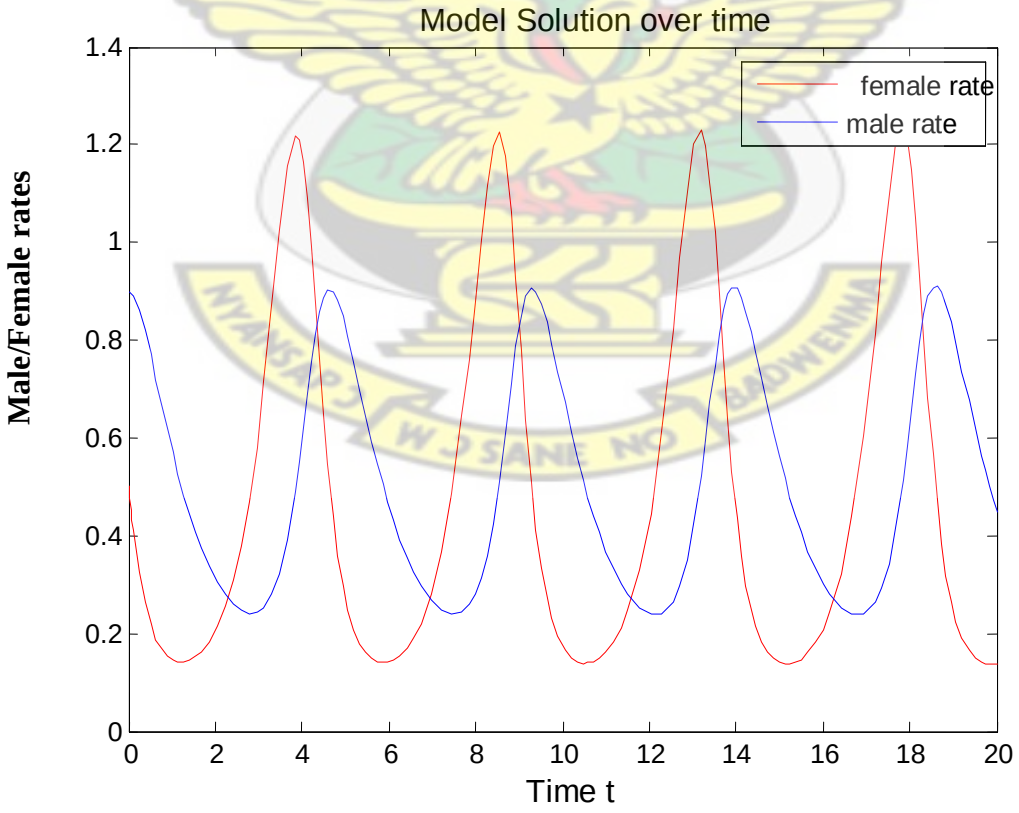



**FIGURE 4.8 : TRAJECTORIES 0F THE MODEL**

Model solution for higher initial male AIDS rate

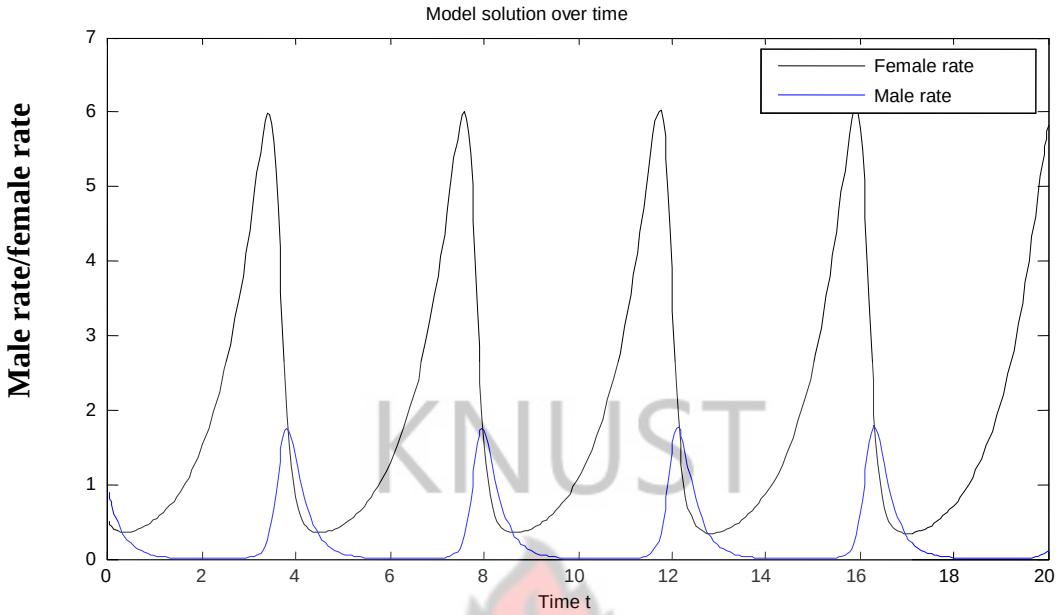



**FIGURE 4.9: TRAJECT0RIES OF THE MODLE**

$$\frac{dy}{dt} = 1.1y - 3.7xy$$

$$\frac{dx}{dt} = -1.9x + 3.8xy$$

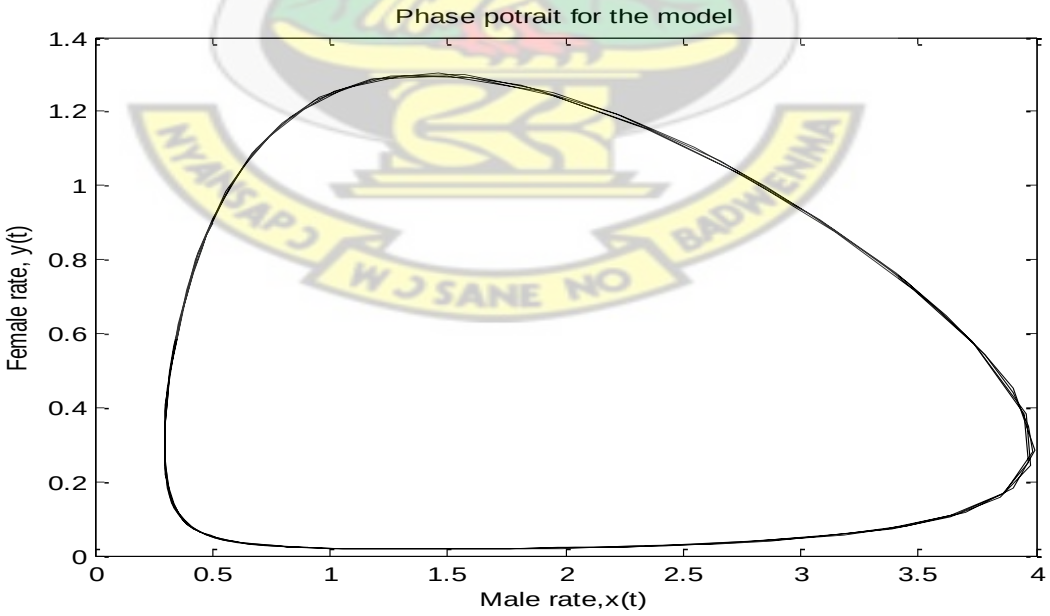



**FIGURE 4.10 : PHASE PORTAIT OF THE MODEL**

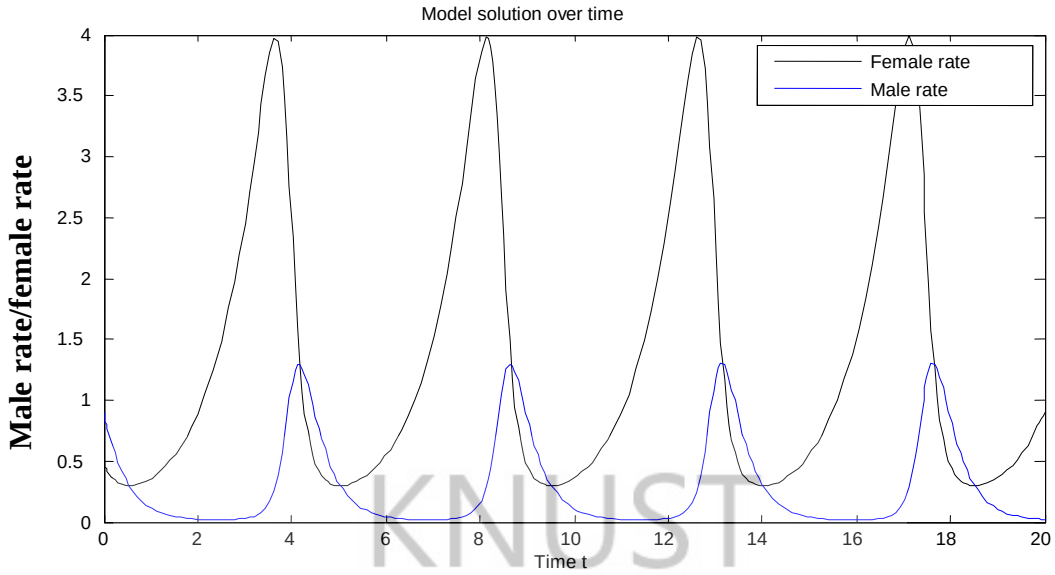
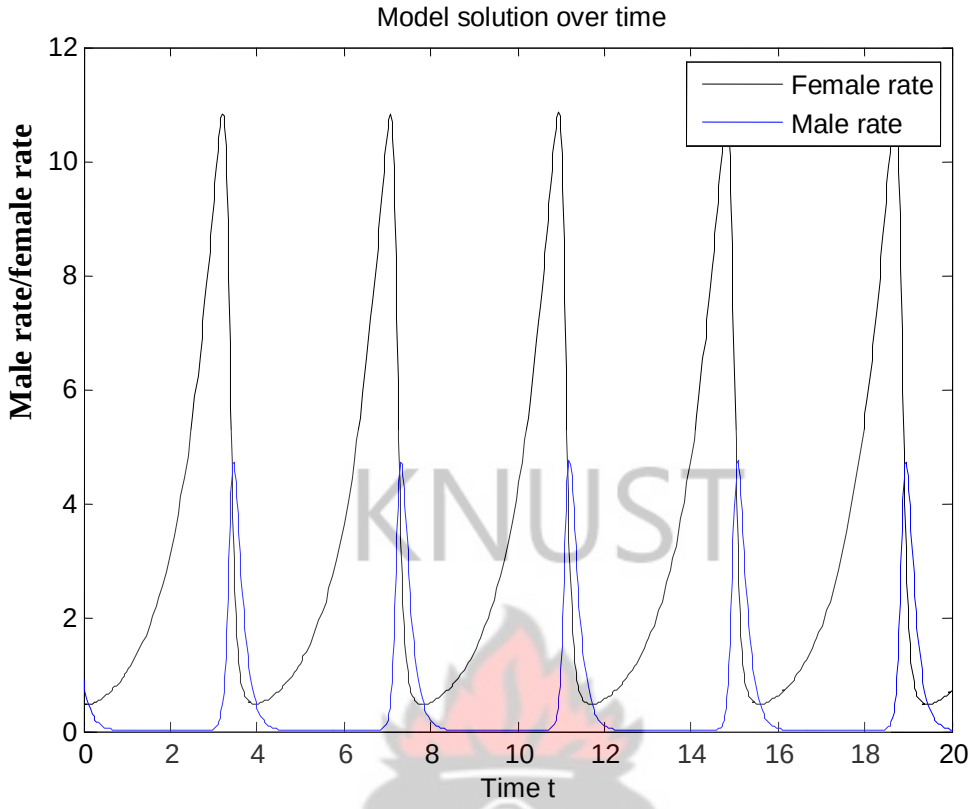





**FIGURE 4.11: TRAJECTORIES OF THE MODEL**

$$\frac{dy}{dt} = 1.1y - 3.7xy$$

$$\frac{dx}{dt} = -1.9x + 2.7xy$$




$$\frac{dy}{dt} = 1.1y - 2.2xy$$

$$\frac{dx}{dt} = -1.9x + 6.3xy$$

**FIGURE 4.12: TRAJECTORIES OF THE MODEL**

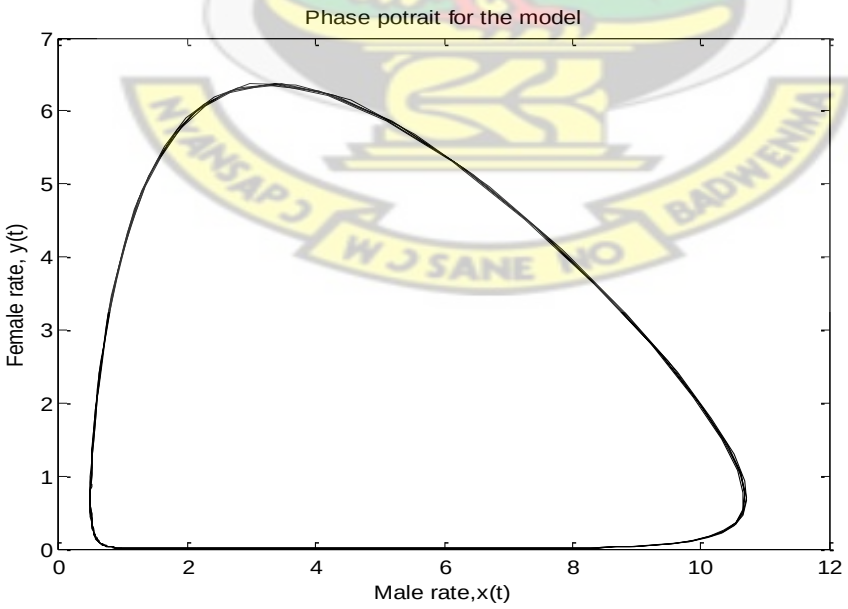
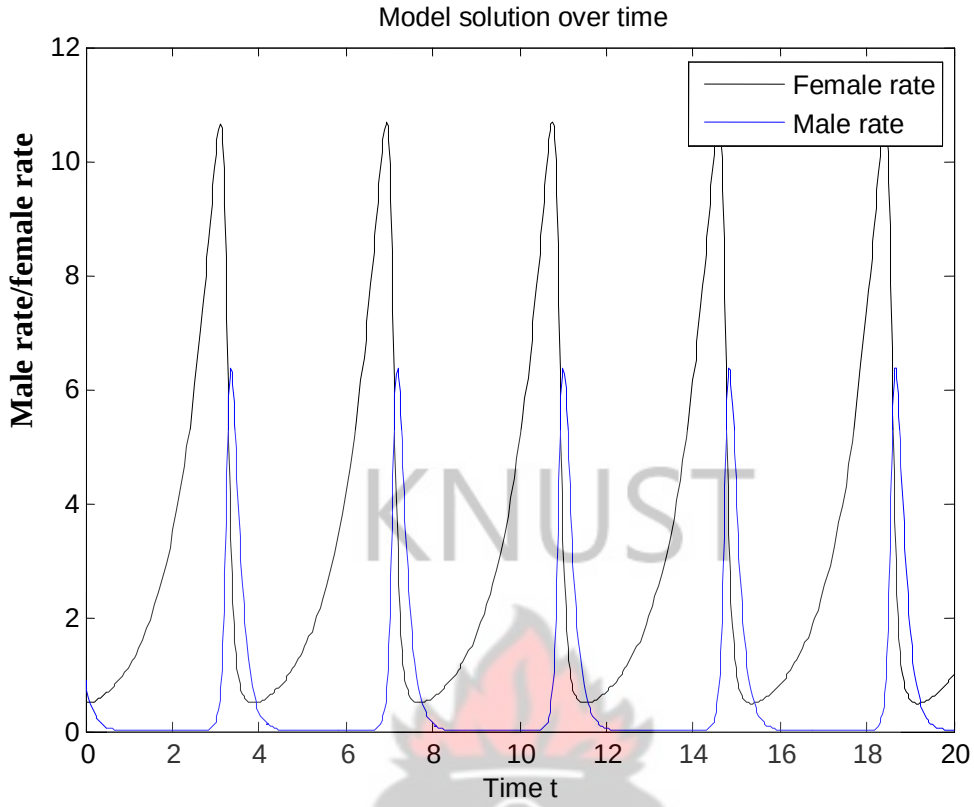



**FIGURE 4.13: PHASE PORTRAIT OF THE MODEL**




$$X' = -1.1X + 1.6XY$$

$$Y' = 0.9Y - 6.3XY$$

**FIGURE 4.14: TRAJECTORIES OF THE MODEL**

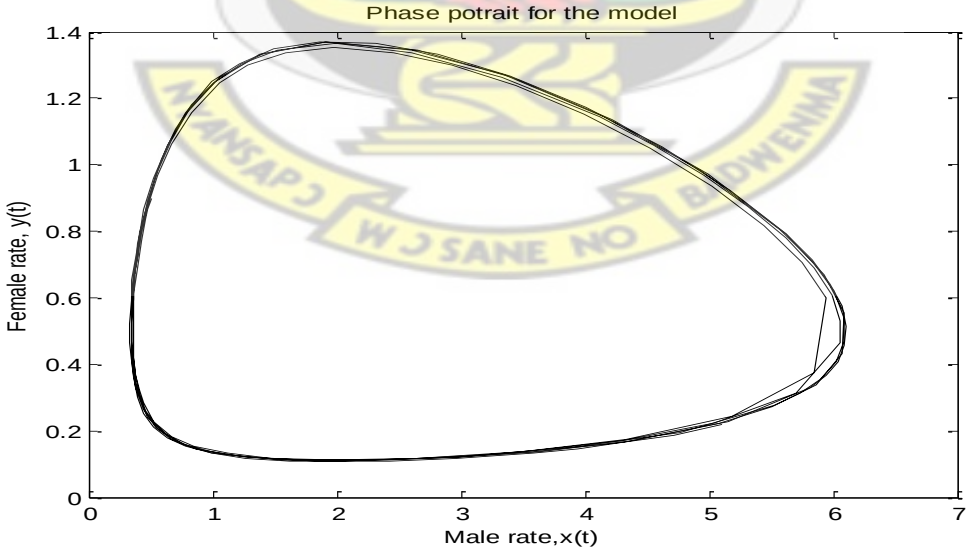



**FIGURE 4.15 : PHASE PORTRAIT OF THE MODEL**

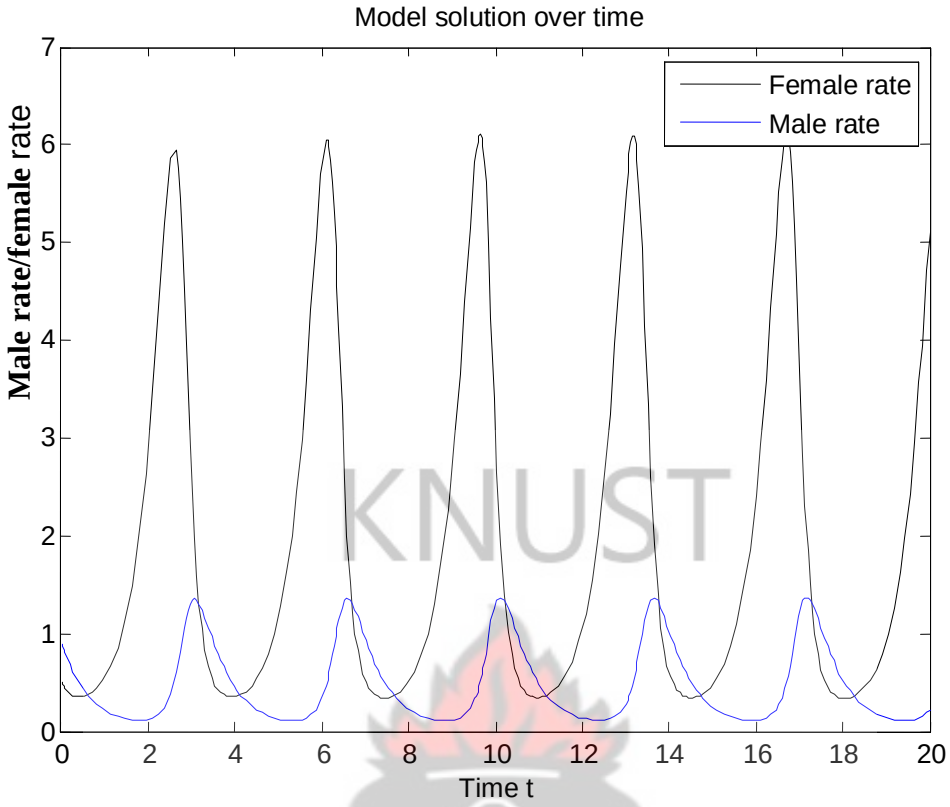



FIGURE 4.16: TRAJECTORIES OF THE MODEL

# CHAPTER FIVE

# CONCLUSION AND RECOMMENDATION

## 5.1 CONCLUSION

This project reviews the predator –prey model in ecology. However, it is applied here as an epidemiological model for the propagation of HIV.

The analysis of the model shows that it has two equilibrium points, these are : ( 0, 0) and ( a/d , b/c). However, the point (0, 0) which shows that in the absence of both newly infected males and females is an unstable point. This is due to the fact that old cases of males are still aggressive enough to sustain increase in new cases of females.

The point ( a/d , b/c ) is stable and this may be explained as having the same newly infected males interacting with the same newly infected females.

The simulation shows the sinusoidal curve for the change in the population of newly infected males and females with time as in the predator –prey model. It also shows that the curve for females is higher than that of males which implies the rate at which females contract HIV is higher than that of males. These curves also show that although the new cases of HIV keeps on rising and falling, it never gets to zero ( that is total eradication of HIV cases).

The graph of the phase portrait of the model ( that is plotting the population of new cases of females against that of males), gives circular curves as always seen in the case of plotting predator population against the prey population. The circles have a common center (0.5, 0.5) which is the stable point. $\left(\frac{a}{d}, \quad \frac{b}{c}\right)$

## 5.2. RECOMMENDATION

### 5.2.1 EDUCATION

Programmes of education on the transmission of HIV should be more targeted on the females especially the young ladies, this is because when females abstain from sex or stick to their partners only, the rate of infection of HIV in both males and females will be reduced. This is also because most of the young ladies are not married and are therefore prone to be lured into having sex with both married and unmarried men.



### 5.2.2 FURTHER STUDIES

It is worthy to explore further the application of the predator –prey model on the transmission of HIV. This will give a more comprehensive idea on how this model is applied on the transmission of HIV. If possible, data on new cases of HIV should be collected and simulation done to see how feasible this model is.

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