

**AN INFECTION AGE-STRUCTURED MATHEMATICAL
MODEL OF THE DYNAMICS OF HIV/AIDS PANDEMIC.**

BY

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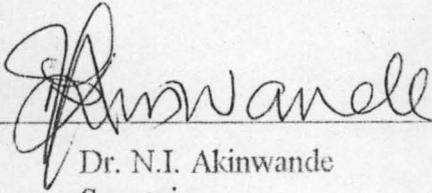
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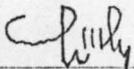
CERTIFICATION

This thesis titled: AN INFECTION AGE-STRUCTURED MATHEMATICAL MODEL OF THE DYNAMICS OF HIV/AIDS PANDEMIC by ABDULRAHMAN, MUHAMMEAD ALFA meets the regulation governing the award of the degree of Master of Technology in Mathematics, Federal University of Technology, Minna and is approved for its contribution to knowledge and literary presentation.



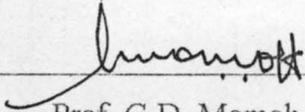
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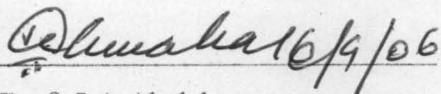
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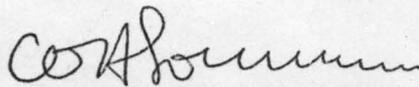
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DEDICATION

DEDICATED

TO

MY PARENTS AND FAMILY.

ACKNOWLEDGEMENT

My utmost gratitude goes to God, the Master of the Universe.

I appreciate the guidance, protection; strength and love from Him to enable me complete this project work successfully.

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ABSTRACT

In this thesis, we propose an infection age-structured mathematical model of the dynamics of HIV/AIDS pandemic in a community partitioned into two distinct compartments of susceptible and infected classes. The infected class is structured by age of infection, giving rise to a pair of model equations with one an ordinary differential equation and the other a partial differential equation. The equilibrium states are obtained and analyzed for stability using the modified version of the Bellman and Cooke's Theorem [5]. The Math-Cad Computing Application is used to obtain the tables from which the conclusions about the stability or instability of the system are drawn.

CHAPTER ONE

INTRODUCTION

1.1 Background to the study:

The need for the application of mathematical concepts to every aspect of human life in the present age of Science and Technology cannot be over-emphasized. Numerous phenomena can be conceptualized and transformed into mathematical problems. The solutions to such problems will then provide insights into such phenomena and also serve as bases for further investigation. The approaches to the solutions bring about special schemes which may be numerical or analytical in nature. Problems such as the existence of equilibrium states and their stability are of great interest in the mathematical models of population dynamics as pointed out in Akinwande [1], because they serve as the bases for analysis and to draw conclusions.

In this work we propose an infection age-structured mathematical model of HIV/AIDS dynamics. The model is a system of ordinary and partial differential equations. The population $P(t)$ is partitioned into two compartments of Susceptible $S(t)$, this is the class in which members are virus-free but are prone to infection as they interact with the members of

the infected class. The second class is the infected class $I(t)$, this is the class of those that have contracted the virus, they are at various stages of infection. They exhibit the symptoms of full blown AIDS.

The infected class is structured by the infection age with a density function $\rho(t, a)$ where 't' is the time and 'a' is the infection age. There is a maximum infection age 'T' at which a member must leave the compartment via death that is when $a=T$. This is notwithstanding that a member of both the susceptible class and the infected class still die by natural causes.

This work has been divided into five chapters. Introduction is the first chapter. Related literature reviewed in chapter two. The model equations with all the boundary conditions, the definition of parameters, the equilibrium states and corresponding characteristics are all presented in chapter three. In chapter four, we analyze and interpret the equilibrium states for stability or otherwise and finally, the conclusion and recommendations are presented in chapter five.

1.2 Aims and Objectives of the Study

The aims and objectives of this study are:

- (i) To propose a 2-dimensional mathematical model of HIV/AIDS

dynamics for a community.

(ii) To analyze the equilibrium states for stability or instability.

(iii) To draw conclusions on the spread and pattern of the dynamics.

1.3 Significance of the Study:

The interplay between the Human Immunodeficiency Virus (HIV) and the body system turns out to be significantly more dynamic than most biological scientists would have suspected. The weakening of the immune system of the infected person by HIV, day by day has grave social, economic and political implications to the entire nations and regions as the productive segments of the population are been incapacitated by the disease.

The study of population has been of great relevance to the growth of any nation or community over time because of the practical influence it has on human life. "Population plays a vital role in the economic success of a nation" Odekunle [23], to the extent that she cannot survive without adequately understanding the dynamism of her population.

Population studies is also significant for both short term and long term planning in the fields of education, health, employment, social security and environmental preservation. The studies in addition, provide handy

information for formulation of government policies so as to achieve economical and social objectives.

Since the study of population is so relevant in the existence of any nation, the formulation of mathematical models of real life phenomena has made immense and reliable contribution in the study of population dynamics in our lives as people.

This research work is not an exception, but of its unique feature is that it is an infected age-structured population model of the dynamics of HIV/AIDS pandemic. An attempt is made to test the effectiveness of anti-retroviral drugs with infection age for a longer life expectancy.

1.4 Scope and Limitation of the Study:

The population $P(t)$ of this model is partitioned into two compartments: susceptible class and infected class. Hence, it is a two dimensional mathematical model. The effectiveness of anti-retroviral drugs application in slowing down the death of the infected victims of the disease is paramount in this study. One of the limitations of this work is the in-availability of data in a locality like Minna which would have been of great interest for case study, hence the use of hypothetical values for the parameters.

1.5 A brief overview on HIV/AIDS pandemic:

HIV is an abbreviation, which stands for Human Immunodeficiency Virus. HIV is a retrovirus, the causative agent of AIDS pandemic. AIDS on the other hand, stands for Acquired Immuno-Deficiency Syndrome. It is a syndrome which refers to a group of symptoms that collectively indicate or characterize a disease.

It was discovered that HIV causes AIDS by destroying a certain kind of blood cell (CD4 T-cell), which is crucial to the normal functioning of the human immune system. In fact, loss of these cells in people with HIV is an extremely powerful predictor of the development of AIDS. Studies have revealed that most people infected by HIV carry the virus for 5 -10 years before enough damage is done to the immune system for AIDS to develop.

Patients are generally said to have crossed to the term AIDS when the helper cells come to zero functioning, in which a healthy individual whose CD+4 measure 2000 cells per micro-litre of blood fall short by 200, Ibinaiye [16]. During this stage the viral level climbs sharply and the measure of immune activity drops sharply.

Once AIDS develops, people rarely survive for more than two years, but the persistence of a good immune response in the face of constant attack by HIV raises the issue of why a patient lives marginally longer. Kudos to scientific discovery of anti-retroviral drugs, its application according to doctors can slow down dramatically the destruction of the victim's immune system which will consequently give a longer life-span.

Scientists have identified two types of HIV: HIV-1 and HIV-2. The HIV-1 is the earliest known primary cause of AIDS world-wide while HIV-2 is prevalent mostly in West Africa. In persons infected with HIV-2, the Immunodeficiency seems to develop more slowly compared to persons infected with HIV-1. HIV-2 is less infectious in the early stage of infection. The earliest known case of HIV-1 in a human was from a blood sample collected in 1959 from a man in Kinshasa, Democratic Republic of Congo, (How he became infected was unknown) CDC[9]. The genetic analysis of his blood sample suggested that HIV-1 may have stemmed from a single virus in the late 1940s or early 1950s. The virus was believed to have existed in the United States since, at least in the mid to late 1970s.

From 1979–1981 rare types of opportunistic infections such as pneumonia, cancer, and other illnesses were being reported by doctors in Los Angeles and New York among a number of patients who were homosexuals, these were conditions not clearly found in people with healthy immune systems. i.e. people with high number of CD4 T- cells.

In 1983, scientists discovered the virus that causes AIDS, it was first named HTLV (Human T-Cell Lymphotropic Virus type 111) by an International Scientific Committee and this name was later changed to HIV (Human Immunodeficiency Virus). When HIV enters the body it infects the lymphocytes, the white blood cells (CD4 T-cells) of the immune system which fights against infections in the body. It was in 1982, that public health officials began to use the term ‘Acquired Immuno-Deficiency Syndrome’ or AIDS to describe the occurrences of opportunistic infections.

AIDS is a medical diagnosis made by doctors based on specific criteria established by the CDC [9] (Centre for Diseases Control and prevention) in New-York City. Its symptoms are similar to the symptoms of many other diseases. The only way to determine whether one is infected is to go for an HIV- status-test, i.e. to be tested for HIV

infection. You cannot rely on symptoms or not, to know whether or not you are infected with HIV. Many people who are infected with HIV do not have any symptoms at all for many years. The following may be warning signs and symptoms of infection with HIV.

- Rapid weight loss.
- Dry cough.
- Recurring fever or profuse night sweat.
- Profound and unexplained fatigue.
- Swollen lymph glands in the arm-pits, groin or neck.
- Diarrhea that lasts for more than a week.
- Pneumonia.
- White spots or unusual blemishes on the tongue, in the mouth and in the throat.
- Memory loss, depression and other neurological disorders.
- Red, brown, pink, purplish blotches on or under the skin or inside the mouth, nose or eye-lids.

Similarly, you cannot rely on symptoms to establish that a person has AIDS. The symptoms of AIDS are similar to the symptoms of many other illnesses.

CHAPTER TWO

LITERATURE REVIEW

2.1 Introduction:

The purpose of this chapter is to review some of the literature on the dynamics of HIV/AIDS pandemic. Thousands of mathematical models have been developed on this topic from different points of view, which can be grouped as follows:

- **HIV: Its Transmission / Spread.**
- **HIV: Its Prevention and Treatment.**

2.2 Mathematical Modeling:

Benyah [6], defines Mathematical Modeling as “the process of creating a mathematical representation of some phenomenon in order to gain a better understanding of that phenomenon”, mathematical modeling has become a crucial engineering technique over a long period of time before now. This is because of its attempts to match observation with symbolic statement.

Mathematical models stimulate our intuitive knowledge to generate new hypothesis, suggest experiments and measure crucial parameters. Essentially, any real life-situation in the physical and biological world,

whether natural or involving technology and in human intervention is subject to analysis by modeling if it can be put in quantitative terms. Thus, optimization and control theory may be used to model industrial processes, traffic patterns, sediment transport in streams and other situations; information and communication theory may be used to model message transmission; linguistic characteristics and the likes. Also dimensional analysis and computer simulation may be used to model atmospheric circulation patterns, stress distribution in engineering structures, the growth and development of land forms and a host of other processes in science and engineering.

Once, a model has been developed and used to answer questions; it should be critically examined and often modified to obtain a more accurate reflection of the observed reality of that phenomenon. Generally, the success of a model depends on how easily it can be used and how accurate are its predictions.

As emphasized by Benyah [6] "Mathematical modeling is an evolving process, as new insight is gained the process begins as additional factors are considered".

Steps to Mathematical Modeling:

In building a mathematical model for a real-life situation, it requires a thorough understanding of the underlying principles of the system to be modeled. During the process of building a mathematical model, the modeler will decide what factors can be de-emphasized. Different problems may require very different methods of approach.

Benyah [6], outlined the following steps as a general approach to the formulation of real-life problem in mathematical terms:

- (a) Identify the problem.
- (b) Identify the important variables and parameters.
- (c) Determine how they relate to each other, stating the assumptions.
- (d) Develop the equation(s) that express the relationship between the variables and constants.
- (e) Analyze and solve the resulting mathematical problem.

Epidemiology is the mathematical study of the spread of disease and one of the most pressing problems in this area of recent has to do with HIV/AIDS. It is estimated that over 40 million people live with HIV/AIDS, and of these over 29 million are Africans, about 2.6 million of children aged 1-14 years are infected; about 11 million children are

orphaned world-wide by the scourge of which 950 000 or more are resident in Nigeria and the rate of infection in most of the developing countries is high” Farai [13].

2.3 HIV: Its Transmission / Spread.

It is established that a lot of research has been conducted on the transmission HIV/AIDS. Consequently, there is a great deal of valuable scientific and public health information about the ways in which HIV is transmitted.

CDC[9] (Centre for Disease Control and prevention) in New-York City explains that HIV is spread by sexual contact with an infected person, by sharing needles and/or syringes (primarily among drug users) and less commonly through transfusion of infected blood or blood clotting factors. Babies born to HIV infected women may become infected before or during birth or through breast-feeding after birth.

On the fears of some people that HIV might be transmitted in other ways such as through air, water or insects. CDC pointed out that no scientific evidence to support any of the these fears has been found, adding that if HIV were being transmitted through other routes, the pattern of the reported cases would have been much different from what

has been observed. For example, if mosquitoes could transmit HIV infection, many more young children and pre-adolescents would have been diagnosed with AIDS.

It is also important to note that infection with HIV has been the sole common factor shared by AIDS cases throughout the world among homosexuals, blood transfusion recipients, persons with hemophilia, sex partners of infected persons, children born to infected women and occupationally exposed health care workers. The conclusion after more than 20 years of scientific research is that people, if exposed to HIV through sexual contact or injecting drug users for example, may become infected with HIV. If they become infected, most of them will eventually develop AIDS.

In an attempt to find out if genetic mutation influences the spread of HIV/AIDS, Krischner et al [19] designed a model that compares the rate of HIV transmission in two populations. All the individuals in one group have two copies of normal genetic mutation that protects persons from HIV infection (CCRS gene). The second group was a combination of individuals some with two mutated CCRS alleles, some with one mutated and one normal mutated allele. They found out that persons with two

copies or alleles of their mutation and one normal copy can be infected, but they carry lower levels of the virus and take two years longer, on average to develop AIDS.

People with two normal copies of the CCR5 gene are almost susceptible to HIV infections. In the model, population without the protective mutation, the researchers found out that HIV/AIDS prevalence increased logarithmically for the first 35yrs of the epidemic, reaching 18 percent before leveling off. While, in the model population with mutated CCR5 gene, the prevalence reached approximately 12 percent. Prevalence began to decline after 70 years.

Finally, their result suggested that the CCR5 mutation limits the epidemic by decreasing the probability of infection due to lower viral load found with one copy of the mutated gene.

2.4 HIV: Its Prevention and Treatment.

Prior to 1996, scientists estimated that about half the people with HIV would develop AIDS within 10years after becoming infected. The time varied from person to person and depended on many factors, including a person's health status, social status and his or her health related behaviours.

Joseph [17] examined the increasing of the HIV in the human body; his model gives an answer to the question why the space of time between HIV infection and outbreak of AIDS differs to a great extent. It also gives insight into the phenomenon that our immune system, generally cannot root out HIV completely once it sets into human system.

Since 1996, the introduction of powerful anti- retroviral therapies has dramatically changed the progression time between HIV infection and the development of AIDS.

Dominick and Martins [12] reviewed of mathematical models of HIV dynamics, disease progression and therapy started by introducing a basic model of virus infection and demonstrated how it was used to study HIV dynamics and to measure crucial parameters that lead to the new understanding of the disease process. Finally, they showed how mathematical models can be used to understand the correlation of long - term immunological control of HIV and the design of therapy regimes that convert a progressing patient into a state of long - term non- progression.

There are also other medical treatments that can prevent or cure some of the illnesses associated with AIDS, though the treatments do not cure AIDS itself. But, because of the advances in drugs therapies and other

medical treatments, estimates of how many people will develop AIDS and how soon are being recalculated, revised, or are currently under study. As with other diseases, early detection of infection allows for more options for treatment and preventative health care.

David et al [11] examined the impact of condom use on the sexual transmission of Human Immunodeficiency Virus (HIV) and Acquired Immune Deficiency Syndrome (AIDS) amongst a homosexual population.

First, they derived a multi-group $S \rightarrow I \rightarrow R$ -type model of HIV/AIDS transmission where the homosexual population is split into sub-groups according to frequency of condom use. Both susceptible and infected individual can transfer between the different groups. They discussed in detail an important special case of their model-which includes two risk groups and perform an equilibrium and stability analysis for this special case. Their analysis shows that the model exhibits the result which was far from expectations.

Michael et al [20] introduced a mathematical model to study accelerating impact of HIV infection on the incidence rates of tuberculosis (TB) disease. A sexually active population aged from 15 to

49years is followed cross-sectional over a period of time, beginning with the year in which an HIV infection was probably first present in the population. The model calculates the growing incidence rates of new TB disease in HIV-positive and in HIV-negative individuals. Model equations derived by an actual method, are developed recursively. Input information required for the calculations includes the age distribution of the study population, pre-HIV annual TB infection rates, annual HIV-infection and mortality rates and estimates of annual TB disease breakdown rates in the absence and in the presence of the HIV infection. With correct input data the model provides a useful blueprint for health agencies in designing effective programme for curbing in the future, the course of similar dual epidemics in the population.

Research works on risk and benefits of medication for Human Immuno- deficiency Virus (HIV) shows that infected patients are at the risk of developing fungal and bacterial infections that take the "opportunity" provided by the patient's weakened Immune System to attack the body.

Moore et al [21] found out that the ability of zidovudine (ZDV) therapy to prolong survival in HIV-infected patients is limited to 1 and 2

years in patients with CD+ 4 cell-count of $500/\text{mm}^3$ or less, perhaps due to the emergence of ZDV which is HIV resistant. Beyond one year of ZDV use, changing to an alternative therapy - such as didanosine, zalcitabine, stavudine or combination of therapy may be appropriate, concluded the authors.

They determined the duration of ZDV benefit in 393 patients receiving HIV care at a large urban clinic, 57 percent of the patients were injecting drug users. They compared the 235 patients who use ZDV with 158 non-users and found out that the risk of dying was reduced two-thirds when ZDV was used for less than one year.

However, this hazard declined only 25 percent by the second year. These findings demonstrate an early, though limited benefit of ZDV in an urban predominantly black population with a relatively high proportion of women and injecting drug users. This study proceeded current work on combination of therapies and contributed to the evolving knowledge based on treatment options for HIV-infected patients.

Using a mathematical method called "Uncertainty analysis" in the University of California - Los Angeles (UCLA)[9] researchers led by Blower, calculated the impact of a range of variables on HIV infection in

San-Francisco. The researchers assumed that anti-retroviral treatments lower the amount of virus contained in the blood stream by at least half and possibly up to 100-fold: meaning that widespread use of anti-retroviral drugs will make it more difficult for HIV-positive persons to transmit the virus to sex-partners.

The mathematicians also assumed that drug resistant strains of HIV will develop but will be less infectious even with "worst-case assumptions" such as the evolution of drug resistant HIV that is infectious and an increase in unprotected sexual activity among persons using anti-retroviral drugs are factored in the model and predict that anti-retroviral treatment will stop the HIV/AIDS epidemic in San Francisco "well before" the end of the century. Their prediction met with reactions from various researchers.

Ganges [14] pointed out that other studies conducted in Uganda and United States have shown that anti-retroviral treatment "only marginally affects a person's ability to infect" others with the virus. He added that the main reason why anti-retroviral treatment will not slow the epidemic is that "in the real world, most patients manage to sustain a 50 percent or less reduction" in viral load, a smaller reduction than calculated in the

UCLA study.

The authors in [9] replied that their model was based on San Francisco data, where most patients on anti-retroviral therapy have experienced decrease in viral load of more 50 percent. Therefore, the pandemic could only be slowed down through the widespread usage of anti-retroviral drugs because treating only a small percentage of the HIV-positive population would have "very little impact" on HIV transmission. They concluded that regardless of whether anti-retroviral can reverse the HIV/AIDS pandemic or not, the drugs "should be available to all who need them, regardless of their social status "because (the drugs) help extend life expectancy of the infected persons.

CHAPTER THREE

THE MODEL EQUATIONS

3.1 Introduction:

In this work, we propose an infection age-structured mathematical model of HIV/AIDS dynamics. The population is partitioned into two compartments of susceptible class $S(t)$, this is the class in which members are virus-free but are prone to infection as they interact with the members of the infected class; The second class is the infected class $I(t)$, this is the class of those that have contracted the virus, they are at various stages of infection, including that of full blown AIDS.

The infected class is structured by the infection age with density function $\rho(t, a)$ where 't' is the time and 'a' is the infection age. There is a maximum infection age 'T' at which a member of the infected class must leave the compartment via death i.e. when $a=T$; this, notwithstanding a member of the class could still die by natural causes at a rate μ , this is also applicable to the susceptible class $S(t)$.

We let the death rate via infection be $\sigma(a) = \delta \tan\left(\frac{\pi a}{2kT}\right)$ where δ is an additional burden due to infection while k is a control parameter which could be associated with the measure of slowing down the death of the

infected such as the effectiveness of the application of anti-retroviral drugs which give the victims longer life-span. A high value of k will imply high effectiveness of such measure and vice-versa. It is assumed that while the new births in $S(t)$ are born therein, the offspring of $I(t)$ are shared between $S(t)$ and $I(t)$ in the proportion θ and $1-\theta$, $0 \leq \theta \leq 1$.

3.2 Definition of parameters:

The parameters used in the model equations of the population are defined as follows:-

β = natural birth rate.

μ = natural death rate.

α = rate of contracting the HIV.

$\sigma(a)$ = death rate from infection where a = age of infection.

$h(a)$ = the gross death rate of the infected class.

δ = an additional burden from infection which is being regulated by environmental factors such as the social status of the infected person; $0 \leq \delta < 1$.

k = measure of the effectiveness of efforts at slowing down the death of infected victims. It is the control parameter of the death

rate via infection. If the level of k is high, that indicates low death rate of infected persons and vice-versa.

θ = the proportion of the offspring of the infected class which are virus-free at birth, $0 \leq \theta \leq 1$.

$1-\theta$ = the proportion of the off-spring of the infected class which have contracted the virus.

T = maximum infection age, but it is assumed that with effectiveness of k as 'a' approaches T , $\sigma(a)$ tends to infinity.

Let the population $\rho(t) = S(t) + I(t)$ where $S(t)$ is the susceptible class and $I(t)$, is the infected class. We now, consider the system of the model equation as:

$$\frac{dS^1}{dt} = (\beta - \mu)S(t) + \theta\beta I(t) - \alpha S(t)I(t) \quad (3.1)$$

And

$$I(t) = \int_0^T \rho(t,a) da, \quad 0 \leq a < T \quad (3.2)$$

$$\frac{\partial \rho(t,a)}{\partial t} + \frac{\partial \rho(t,a)}{\partial a} + h(a)\rho(t,a) = 0 \quad (3.3)$$

Where,

$$h(a) = \mu + \sigma(a) \quad (3.4)$$

And

$$\sigma(a) = \delta \tan\left(\frac{\pi a}{2kT}\right), \quad 0 \leq a < kT \quad (3.5)$$

$$\sigma(a) \rightarrow \infty \quad \text{as } a \rightarrow kT$$

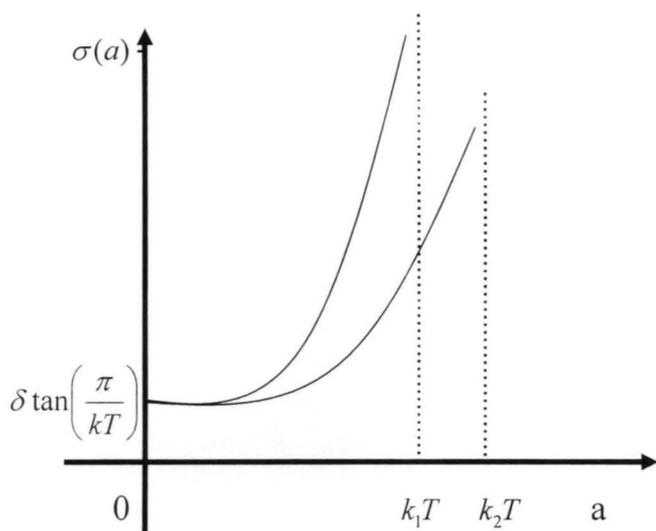


Fig. 1: The Graph of Infection age against $\sigma(a)$, the Death rate via Infection.

It is assumed that $k_1 < k_2 < \dots < k$.

Let

$$\rho(t,0) = B(t) = \alpha S(t)I(t) + (1-\theta)\beta I(t) \quad (3.6)$$

And,

$$\rho(0,a) = \phi(a) \quad (3.7)$$

$$S(0) = S_0, \quad I(0) = I_0 \quad (3.8)$$

3.3 Equilibrium State:

At an equilibrium state, $\frac{dS}{dt} = 0$ and $\frac{\partial \rho}{\partial t} = 0$

Let

$$(S(t), I(t)) = (x, y) \quad (3.9)$$

And

$$\rho(t, a) = \phi(a) \quad (3.10)$$

From (3.2),

$$y = \int_0^{\tau} \phi(a) da \quad (3.11)$$

From (3.6)

$$\phi(0) = B(0) = \alpha xy + (1 - \theta)\beta y \quad (3.12)$$

Substituting (3.9) – (3.11) into (3.1) and (3.3) become

$$(\beta - \mu)x + \theta\beta y - \alpha xy = 0 \quad (3.13)$$

And,

$$\frac{d\phi(a)}{da} + h(a)\phi(a) = 0 \quad (3.14)$$

\Rightarrow

$$\frac{d\phi(a)}{d(a)} = -h(a)d(a) \quad (3.15)$$

Integrating (3.15) gives

$$\phi(a) = \phi(0) \exp\left\{-\int_0^a h(s) ds\right\} \quad (3.16)$$

If

$$\omega(a-s) = \exp\left\{-\int_s^a h(s) ds\right\} \quad (3.17)$$

Then

$$\omega(a) = \exp\left\{-\int_0^a h(s) ds\right\} \quad (3.18)$$

Therefore (3.16) takes the form:

$$\phi(a) = \phi(0)\omega(a) \quad (3.19)$$

From (3.11)

$$y = \phi(0) \int_0^T \omega(a) da = \phi(0)\varpi \quad (3.20)$$

Where

$$\varpi = \int_0^T \omega(a) da \quad (3.21)$$

Using (3.12) in (3.20), gives

$$y = (\alpha x y + (1-\theta)\beta y)\varpi \quad (3.22)$$

Solve (3.13) and (3.22) simultaneously for x and y .

From (3.22), either

$$y = 0 \quad \text{or} \quad (\alpha x + (1 - \theta)\beta)\varpi = 1 \quad (3.23)$$

\Rightarrow

$$x = \frac{1}{\alpha\varpi}(1 - (1 - \theta)\beta\varpi) \quad (3.24)$$

From (3.13), if $y = 0$, $x = 0$, thus the point $(x, y) = (0, 0)$ is the zero equilibrium state. And substituting (3.24) into (3.12) we have,

$$y = \frac{(\beta - \mu)(1 - (1 - \theta)\beta\varpi)}{\alpha(1 - \beta\varpi)} \quad (3.25)$$

So, the point

$$(x, y) = \left[\frac{1}{\alpha\varpi}(1 - (1 - \theta)\beta\varpi), \frac{(\beta - \mu)(1 - (1 - \theta)\beta\varpi)}{\alpha(1 - \beta\varpi)} \right] \quad (3.26)$$

given by (3.24) and (3.25) respectively is the non-zero equilibrium state.

3.4 The Characteristic Equations:

In order to obtain the characteristic equation, we shall perturb as follow:

$$S(t) = x + p(t), \quad p(t) = \bar{p}e^{\lambda t} \quad (3.27)$$

$$I(t) = y + q(t), \quad q(t) = \bar{q}e^{\lambda t} \quad (3.28)$$

And,

$$\rho(t, a) = \phi(a) + \eta(a)e^{\lambda t} \quad (3.29)$$

\Rightarrow

$$\bar{q} = \int_0^T \eta(a) da \quad (3.30)$$

Substituting, (3.27) and (3.28) into the model equation (3.1)

$$\begin{aligned} \frac{d}{dt}(x + \bar{p}e^{\lambda t}) &= (\beta - \mu)(x + \bar{p}e^{\lambda t}) + \theta\beta(y + \bar{q}e^{\lambda t}) - \alpha(x + \bar{p}e^{\lambda t})(y + \bar{q}e^{2\lambda t}) \\ &= (\beta - \mu)x + (\beta - \mu)\bar{p}e^{\lambda t} + \theta\beta y + \theta\beta\bar{q}e^{\lambda t} - \alpha xy - \alpha x\bar{q}e^{\lambda t} - \alpha y\bar{p}e^{\lambda t} - \alpha\bar{p}\bar{q}e^{2\lambda t} \end{aligned} \quad (3.31)$$

Considering (3.12) and neglecting term of order 2 (i.e. $\bar{p}\bar{q}$) gives the above equation as:

$$\lambda\bar{p}e^{\lambda t} = (\beta - \mu)\bar{p}e^{\lambda t} + \theta\beta\bar{q}e^{\lambda t} - \alpha x\bar{q}e^{\lambda t} - \alpha y\bar{p}e^{\lambda t}$$

Or

$$(\beta - \mu - \alpha y - \lambda)\bar{p} + (\theta\beta - \alpha x)\bar{q} = 0 \quad (3.32)$$

And, substituting (3.29) into (3.3) which is given by

$$\frac{\partial \rho(t, a)}{\partial t} + \frac{\partial \rho(t, a)}{\partial a} + h(a)\rho(t, a) = 0,$$

We have,

$$\frac{\partial}{\partial t} [\phi(a) + \eta(a)e^{\lambda t}] + \frac{\partial}{\partial a} [\phi(a) + \eta(a)e^{\lambda t}] + h(a)[\phi(a) + \eta(a)e^{\lambda t}] = 0 \quad (3.33)$$

Differentiating with respect to 'a'

$$\lambda\eta(a)e^{\lambda t} + \frac{d}{da}\phi(a) + e^{\lambda t}\frac{d}{da}\eta(a) + h(a)\phi(a) + h(a)\eta(a)e^{\lambda t} = 0 \quad (3.34)$$

But, (3.14) is given by $\frac{d\phi(a)}{da} + h(a)\phi(a) = 0$.

So equation (3.34) reduces to:

$$\lambda\eta(a)e^{\lambda t} + e^{\lambda t} \frac{d}{da}\eta(a) + h(a)\eta(a)e^{\lambda t} = 0 \quad (3.35)$$

\Rightarrow

$$\frac{d}{da}\eta(a) + (h(a) + \lambda)\eta(a) = 0 \quad (3.36)$$

Solving the ordinary differential equation (3.36), we have

$$\frac{d\eta(a)}{\eta(a)} = -(h(a) + \lambda)da \quad (3.37)$$

$$\eta(a) = \eta(0) \exp\left\{-\int_0^a (\lambda + h(s))ds\right\} \quad (3.38)$$

Integrating (3.38) over $[0, T]$ gives

$$\bar{q} = \eta(0) \int_0^T \left[\exp\left\{-\int_0^a (\lambda + h(s))ds\right\} \right] da \quad (3.39)$$

Or

$$\bar{q} = \eta(0)b(\lambda) \quad (3.40)$$

Where

$$b(\lambda) = \int_0^T \exp\left\{-\int_0^a (\lambda + h(s))ds\right\} da \quad (3.41)$$

Since $\bar{q} = \eta(0)b(\lambda)$, what is $\eta(0)$ then?

From (3.12), $\phi(0) = \alpha xy + (1 - \theta)\beta y$

And from (3.29), $\rho(t, a) = \phi(a) + \eta(a)e^{\lambda t}$

Therefore,

$$\rho(t, 0) = B(t) = \phi(0) + \eta(0)e^{\lambda t} \quad (3.42)$$

And, from (3.6)

$$\rho(t, 0) = B(t) = \alpha S(t)I(t) + (1-\theta)\beta I(t) \quad (3.43)$$

Substituting (3.27) and (3.28) into (3.6), and using (3.12) and (3.42).

$$\begin{aligned} B(t) &= \alpha \left(x + \bar{p}e^{\lambda t} \right) \left(y + \bar{q}e^{\lambda t} \right) + (1-\theta)\beta \left(y + \bar{q}e^{\lambda t} \right) \\ &= \alpha \left[xy + x\bar{q}e^{\lambda t} + y\bar{p}e^{\lambda t} + \bar{p}\bar{q}e^{2\lambda t} \right] + (1-\theta)\beta y + (1-\theta)\beta \bar{q}e^{\lambda t} \\ &= \alpha xy + \alpha x\bar{q}e^{\lambda t} + \alpha y\bar{p}e^{\lambda t} + \alpha \bar{p}\bar{q}e^{2\lambda t} + (1-\theta)\beta y + (1-\theta)\beta \bar{q}e^{\lambda t} \end{aligned} \quad (3.44)$$

Comparing (3.44) with (3.42) and using (3.12) for $\phi(0)$ we have,

$$\begin{aligned} \alpha xy + (1-\theta)\beta y + \eta(0)e^{\lambda t} &= \alpha xy + \alpha y\bar{p}e^{\lambda t} + \alpha x\bar{q}e^{\lambda t} + \alpha \bar{p}\bar{q}e^{2\lambda t} \\ &\quad + (1-\theta)\beta y + (1-\theta)\beta \bar{q}e^{\lambda t} \end{aligned}$$

(Neglecting the term of order 2)

$$\eta(0) = \alpha y\bar{p} + \alpha x\bar{q} + (1-\theta)\beta \bar{q} \quad (3.45)$$

Substituting $\eta(0)$ in (3.40)

$$\bar{q} = \left(\alpha y\bar{p} + \alpha x\bar{q} + (1-\theta)\beta \bar{q} \right) b(\lambda) \quad (3.46)$$

\Rightarrow

$$\alpha y\bar{p}b\lambda + \left[(\alpha x + (1-\theta)\beta)b(\lambda) - 1 \right] \bar{q} = 0 \quad (3.47)$$

So from equations (3.32) and (3.47) the Jacobian determinant for the system with the eigenvalue λ is:

$$\begin{vmatrix} \beta - \mu - \alpha\gamma - \lambda & \theta\beta - \alpha x \\ \alpha\gamma b(\lambda) & (\alpha x + (1 - \theta)\beta)b(\lambda) - 1 \end{vmatrix} = 0 \quad (3.48)$$

and the characteristic equation is thus given by :

$$(\beta - \mu - \alpha\gamma - \lambda)[(\alpha x + (1 - \theta)\beta)b(\lambda) - 1] - \alpha\gamma b(\lambda)(\theta\beta - \alpha x) = 0 \quad (3.49)$$

CHAPTER FOUR

STABILITY OF EQUILIBRIUM STATES

4.1 The Zero Equilibrium State:

At the zero equilibrium state $(x, y) = (0, 0)$, the characteristic equation becomes:

$$(\beta - \mu - \lambda) [(1 - \theta) \beta b(\lambda) - 1] = 0 \quad (4.1)$$

So either

$$\beta - \mu - \lambda = 0 \quad (4.2)$$

Or

$$(1 - \theta) \beta b(\lambda) - 1 = 0 \quad (4.3)$$

From (4.2),

$$\lambda < 0 \text{ if } \beta < \mu \quad (4.4)$$

But (4.3) is a transcendental equation. We shall apply the Bellman and Cooke Theorem for the stability or otherwise of equilibrium states. The equation can be expressed in the form $H(iy) = F(y) + iG(y)$, it is required that the real $F(y)$ and imaginary $G(y)$ parts of $H(iy)$ will satisfy the condition if: (a) All the zeros of $F(y)$ and $G(y)$ are real and alternating.

(b) The inequality $F(y) G'(y) - F'(y) G(y) > 0$ be satisfied

for at least one value of y -(see Appendix).

From (3.40)

$$b(\lambda) = \int_0^T \exp \left\{ - \int_0^a (\lambda + h(s)) ds \right\} da \quad (4.5)$$

Where

$$h(s) = \mu + \sigma(s) \quad (4.6)$$

We let $\sigma(s) = \delta \tan \left(\frac{\pi s}{2kT} \right)$, for the purpose of computation,

so that $0 \leq a < kT$ and $0 \leq k \leq 1$.

\Rightarrow

$$\int_0^a (\lambda + h(s)) ds = \int_0^a (\lambda + \mu + \sigma(s)) ds \quad (4.7)$$

$$= (\lambda + \mu)a + \delta \int_0^a \tan \left(\frac{\pi s}{2kT} \right) ds \quad (4.8)$$

$$= (\lambda + \mu)a + \frac{2kT\delta}{\pi} \text{Log sec} \left(\frac{\pi a}{2kT} \right) \quad (4.9)$$

Therefore,

$$b(\lambda) = \int_0^T \exp \left\{ - \left[(\lambda + \mu)a + \left(\frac{2kT\delta}{\pi} \right) \text{Log sec} \left(\frac{\pi a}{2kT} \right) \right] \right\} da \quad (4.10)$$

$$b(\lambda) = - \left[\left(\lambda + \mu + \delta \tan \left(\frac{\pi a}{2kT} \right) \right)^{-1} \exp \left\{ - (\lambda + \mu)a - \frac{2kT\delta}{\pi} \text{Log sec} \left(\frac{\pi a}{2kT} \right) \right\} \right]_0^T \quad (4.11)$$

$$= -\left(\lambda + \mu + \delta \tan\left(\frac{\pi}{2k}\right)\right)^{-1} \exp\left\{-\left((\lambda + \mu)T + \frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right)\right)\right\} \\ + (\lambda + \mu)^{-1} \quad (4.12)$$

$$= -\frac{1}{\left(\lambda + \mu + \delta \tan\left(\frac{\pi}{2k}\right)\right)} \exp\left\{-\left((\lambda + \mu)T + \frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right)\right)\right\} + \frac{1}{(\lambda + \mu)} \quad (4.13)$$

$$= \frac{1}{(\lambda + \mu)} - \frac{1}{\left(\lambda + \mu + \delta \tan\left(\frac{\pi}{2k}\right)\right)} \exp\left\{\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - \lambda T\right\} \quad (4.14)$$

Substituting (4.14) into (4.3)

$$(1 - \theta)\beta \left\{ \frac{1}{(\lambda + \mu)} - \frac{1}{\left(\lambda + \mu + \delta \tan\left(\frac{\pi}{2k}\right)\right)} \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - \lambda T\right) \right\} - 1 = 0$$

$$(1 - \theta)\beta \left\{ \frac{\left(\lambda + \mu + \delta \tan\left(\frac{\pi}{2k}\right)\right) - (\lambda + \mu) \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - \lambda T\right)}{(\lambda + \mu) \left(\lambda + \mu + \delta \tan\left(\frac{\pi}{2k}\right)\right)} \right\} - 1 = 0$$

$$(1 - \theta)\beta \left\{ \left(\lambda + \mu + \delta \tan\left(\frac{\pi}{2k}\right)\right) - (\lambda + \mu) \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - \lambda T\right) \right\} \\ - (\lambda + \mu) \left(\lambda + \mu + \delta \tan\left(\frac{\pi}{2k}\right)\right) = 0 \quad (4.15)$$

Let

$$h(\lambda) = 0 \quad (4.16)$$

$$h(\lambda) = (1-\theta)\beta \left\{ \left(\lambda + \mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) - (\lambda + \mu) \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - \lambda T\right) \right\} \\ - (\lambda + \mu) \left(\lambda + \mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) \quad (4.17)$$

$$= (1-\theta)\beta \left(\lambda + \mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) - (1-\theta)\beta (\lambda + \mu) \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - \lambda T\right) \\ - \lambda \left(\lambda + \mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) - \mu \left(\lambda + \mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) \quad (4.18)$$

$$= (1-\theta)\beta \lambda + (1-\theta)\beta \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) \\ - (1-\theta)\beta \lambda \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - \lambda T\right) \\ - (1-\theta)\beta \mu \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - \lambda T\right) \\ - \lambda^2 - \lambda \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) - \lambda \mu - \mu \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) \quad (4.19)$$

$$= ((1-\theta)\beta - \mu) \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) + \left(\beta - \theta\beta - 2\mu - \delta \tan\left(\frac{\pi}{2k}\right) \right) \lambda - \lambda^2$$

$$- (1-\theta)\beta \lambda \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - \lambda T\right)$$

$$-(1-\theta)\beta \mu \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - \lambda T\right) \quad (4.20)$$

If we set $\lambda = iw$, we have that

$$h(iw) = f(w) + ig(w) \quad (4.21)$$

The condition for $\text{Re } \lambda < 0$ will then be given by the inequality

$$f(0)g'(0) - f'(0)g(0) > 0 \quad (4.22)$$

Therefore,

$$\begin{aligned} h(iw) = & ((1-\theta)\beta - \mu) \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) + \left(\beta - \theta\beta - 2\mu - \delta \tan\left(\frac{\pi}{2k}\right) \right) iw \\ & + w^2 - (1-\theta)\beta iw \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - iwT\right) \\ & - (1-\theta)\beta \mu \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - iwT\right) \end{aligned} \quad (4.23)$$

Resolving into real and imaginary parts, we have

$$f(w) = ((1-\theta)\beta - \mu) \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) + w^2 \quad (4.24)$$

$$\begin{aligned} g(w) = & \left(\beta - \theta\beta - 2\mu - \delta \tan\left(\frac{\pi}{2k}\right) \right) w \\ & - (1-\theta)\beta w \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - wT\right) \\ & - (1-\theta)\beta \mu \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - wT\right) \end{aligned} \quad (4.25)$$

From equations (4.24) and (4.25),

$$f'(w) = 2w \tag{4.26}$$

$$\begin{aligned} g'(w) = & \left(\beta - \theta\beta - 2\mu - \delta \tan\left(\frac{\pi}{2k}\right) \right) \\ & - (1-\theta)\beta \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - wT\right) \\ & + (1-\theta)\beta wT \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - wT\right) \\ & + (1-\theta)\beta \mu T \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - wT\right) \end{aligned} \tag{4.27}$$

$$f(0) = ((1-\theta)\beta - \mu) \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) \tag{4.28}$$

$$g(0) = -(1-\theta)\beta \mu \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T\right) \tag{4.29}$$

$$f'(0) = 0 \tag{4.30}$$

$$\begin{aligned} g'(0) = & \left(\beta - \theta\beta - 2\mu - \delta \tan\left(\frac{\pi}{2k}\right) \right) \\ & - (1-\theta)\beta \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T\right) \\ & + (1-\theta)\beta \mu T \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T\right) \end{aligned} \tag{4.31}$$

From (4.28) to (4.31) the inequality then gives

$$f(0)g'(0) > 0 \quad (4.32)$$

Let

$$J_1(k) = f(0)g'(0) \quad (4.33)$$

So the zero equilibrium state will be stable when

$$\beta < \mu \text{ and } J_1(k) > 0 \quad (4.34)$$

and unstable if otherwise.

$$J_1(k) = \left[((1-\theta)\beta - \mu) \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) \right] \left[\left(\beta - \theta\beta - 2\mu - \delta \tan\left(\frac{\pi}{2k}\right) \right) \right. \\ \left. - (1-\theta)\beta \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T\right) \right. \\ \left. + (1-\theta)\beta \mu T \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T\right) \right]$$

Using a Math Cad Software, hypothetical parameter values were used to generate a table of values for $J_1(k)$ so as to analyze the result, some of the values obtained are presented in table 4.1 below.

Table 4.1: The Table of values for the Zero equilibrium state.

$$\delta = 1.0 \quad \theta = 0.4 \quad T = 10$$

k	$J_1(k)$ $\beta = 0.5,$ $\mu = 0.2$	Remarks	$J_1(k)$ $\beta = 0.25,$ $\mu = 0.50$	Remarks	$J_1(k)$ $\beta = 0.15,$ $\mu = 0.20$	Remarks	$J_1(k)$ $\beta = 0.20,$ $\mu = 0.15$	Remarks
0.2	0.27	stable	0.796	stable	0.04	stable	0.003	stable
0.3	- 0.003	Unstable	0.514	stable	0.06	stable	0.012	stable
0.4	- 0.013	Unstable	0.395	stable	0.043	stable	0.008	stable
0.5	- 0.012	Unstable	0.333	stable	0.033	stable	0.006	stable
0.6	- 0.010	Unstable	0.296	stable	0.027	stable	0.005	stable
0.7	- 0.009	Unstable	0.272	stable	0.024	stable	0.004	stable
0.8	- 0.008	Unstable	0.254	stable	0.021	stable	0.004	stable
0.9	- 0.007	Unstable	0.214	stable	0.019	stable	0.003	stable
1.0	- 0.006	Unstable	0.230	stable	0.017	stable	0.003	stable

4.2 The Non-zero Equilibrium state:

At the non – zero equilibrium state

$$x = \frac{1 - (1 - \theta)\beta\varpi}{\alpha\varpi}$$

$$y = \frac{(\beta - \mu)(1 - (1 - \theta)\beta\varpi)}{\alpha(1 - \beta\varpi)}$$

Where

$\varpi = \int_0^r \omega(a) da$; $\omega(a) = \exp\{-h(s)ds\}$ and, from (4.16), we have that

$$b(\lambda) = \varpi = \frac{1}{(\lambda + \mu)} - \frac{1}{\left(\lambda + \mu + \delta \tan\left(\frac{\pi}{2k}\right)\right)} \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - \lambda T\right) \quad (4.35)$$

Consider the characteristic equation (3.49)

$$(\beta - \mu - \alpha\gamma - \lambda) \left[(\alpha x + (1 - \theta)\beta)b(\lambda) - 1 \right] - \alpha\gamma(\theta\beta - \alpha x)b(\lambda) = 0$$

Substituting $b(\lambda)$ into (3.49), we have

$$(\beta - \mu - \alpha\gamma - \lambda)$$

$$\left\{ \begin{array}{l} (\alpha x + (1 - \theta)\beta) \left[\frac{1}{(\lambda + \mu)} - \frac{1}{\left(\lambda + \mu + \delta \tan\left(\frac{\pi}{2k}\right)\right)} \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - \lambda T\right) \right] - 1 \\ - \alpha\gamma(\theta\beta - \alpha x) \end{array} \right\}$$

$$\left\{ \frac{1}{(\lambda + \mu)} - \frac{1}{\left(\lambda + \mu + \delta \tan\left(\frac{\pi}{2k}\right) \right)} \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - \lambda T\right) \right\} = 0$$

Simplifying, we have

$$(\beta - \mu - \alpha\gamma - \lambda)$$

$$\left\{ (\alpha x + (1 - \theta)\beta) \left[\left(\lambda + \mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) - (\lambda + \mu) \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - \lambda T\right) \right] \right.$$

$$\left. - (\lambda + \mu) \left(\lambda + \mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) - \alpha\gamma(\theta\beta - \alpha x) \right.$$

$$\left. \left[\left(\lambda + \mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) - (\lambda + \mu) \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - \lambda T\right) \right] = 0 \quad (4.36)$$

Let equation (4.36) takes the form

$$H(\lambda) = 0 \quad (4.37)$$

$$H(\lambda) = (\beta - \mu - \alpha\gamma - \lambda)$$

$$\begin{aligned} & \left\{ (\alpha x + (1 - \theta)\beta)\lambda + (\alpha x + (1 - \theta)\beta) \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) \right. \\ & \left. - (\alpha x + (1 - \theta)\beta)\lambda \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - \lambda T\right) \right. \\ & \left. - (\alpha x + (1 - \theta)\beta) \mu \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - \lambda T\right) \right\} \end{aligned}$$

$$\begin{aligned}
& -\lambda^2 - \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) \lambda - \mu\lambda - \mu \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) \Big\} \\
& - \alpha y (\theta\beta - \alpha x) \lambda - \alpha y (\theta\beta - \alpha x) \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) \\
& + \alpha y (\theta\beta - \alpha x) \lambda \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - \lambda T\right) \\
& + \alpha y (\theta\beta - \alpha x) \mu \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - \lambda T\right) \tag{4.38}
\end{aligned}$$

Setting $\lambda = iw$

$$H(iw) = (\beta - \mu - \alpha y - iw)$$

$$\begin{aligned}
& \left\{ (\alpha x + (1 - \theta)\beta) iw + (\alpha x + (1 - \theta)\beta) \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) \right\} \\
& - (\alpha x + (1 - \theta)\beta) iw \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - iw T\right) \\
& - (\alpha x + (1 - \theta)\beta) \mu \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - iw T\right) \\
& + w^2 - \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) iw - \mu iw - \mu \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) \Big\} \\
& - \alpha y (\theta\beta - \alpha x) iw - \alpha y (\theta\beta - \alpha x) \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) \\
& + \alpha y (\theta\beta - \alpha x) iw \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - iw T\right)
\end{aligned}$$

$$+ \alpha\gamma(\theta\beta - \alpha x)\mu \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - iwT\right) \quad (4.39)$$

$$H(iw) = (\beta - \mu - \alpha\gamma)$$

$$\begin{aligned} & \left\{ (\alpha x + (1 - \theta)\beta)iw + (\alpha x + (1 - \theta)\beta) \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) \right. \\ & - (\alpha x + (1 - \theta)\beta)iw \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - iwT\right) \\ & - (\alpha x + (1 - \theta)\beta) \mu \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - iwT\right) \\ & \left. + w^2 - \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) iw - \mu iw - \mu \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) \right\} \\ & + (\alpha x + (1 - \theta)\beta)w^2 - (\alpha x + (1 - \theta)\beta) \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) iw \\ & - (\alpha x + (1 - \theta)\beta)w^2 \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - iwT\right) \\ & + (\alpha x + (1 - \theta)\beta)\mu iw \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - iwT\right) \\ & - iw^3 - \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) w^2 - \mu w^2 + \mu \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) iw \\ & - \alpha\gamma(\theta\beta - \alpha x)iw - \alpha\gamma(\theta\beta - \alpha x) \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) \\ & + \alpha\gamma(\theta\beta - \alpha x) iw \exp\left(\frac{2kT\delta e^{-kT}}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - iwT\right) \end{aligned}$$

$$+ \alpha y (\theta \beta - \alpha x) \mu \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - iwT\right) \quad (4.40)$$

Resolving into real and imaginary parts

$$H(w) = F(w) + iG(w) \quad (4.41)$$

$$\begin{aligned} F(w) = & (\beta - \mu - \alpha y) \left\{ (\alpha x + (1 - \theta)\beta) \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) - \mu \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) + w^2 \right\} \\ & + (\alpha x + (1 - \theta)\beta) w^2 - \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) w^2 - \mu w^2 \\ & - \alpha y (\theta \beta - \alpha x) \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) \end{aligned} \quad (4.42)$$

$$G(w) = (\beta - \mu - \alpha y)$$

$$\begin{aligned} & \left\{ (\alpha x + (1 - \theta)\beta) w - (\alpha x + (1 - \theta)\beta) w \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - wT\right) \right. \\ & - (\alpha x + (1 - \theta)\beta) \mu \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - wT\right) \\ & \left. - \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) w - \mu w \right\} \\ & - (\alpha x + (1 - \theta)\beta) \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) w \\ & - (\alpha x + (1 - \theta)\beta) w^2 \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - wT\right) \end{aligned}$$

$$\begin{aligned}
& + (\alpha x + (1 - \theta)\beta)\mu w \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - wT\right) \\
& - w^3 + \mu\left(\mu + \delta \tan\left(\frac{\pi}{2k}\right)\right)w - \alpha y(\theta\beta - \alpha x)w \\
& + \alpha y(\theta\beta - \alpha x)w \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - wT\right) \\
& + \alpha y(\theta\beta - \alpha x)\mu \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - wT\right)
\end{aligned} \tag{4.43}$$

$$\begin{aligned}
F^1(w) & = 2w(\beta - \mu - \alpha y) + 2w(\alpha x + (1 - \theta)\beta) \\
& - 2w\left(\mu + \delta \tan\left(\frac{\pi}{2k}\right)\right) - 2w\mu
\end{aligned} \tag{4.44}$$

$$\begin{aligned}
G^1(w) & = (\beta - \mu - \alpha y) \\
& \left\{ (\alpha x + (1 - \theta)\beta) - (\alpha x + (1 - \theta)\beta) \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - wT\right) \right. \\
& + (\alpha x + (1 - \theta)\beta)wT \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - wT\right) \\
& + (\alpha x + (1 - \theta)\beta)\mu T \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - wT\right) \\
& \left. - \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right)\right) - \mu \right\} \\
& - (\alpha x + (1 - \theta)\beta)\left(\mu + \delta \tan\left(\frac{\pi}{2k}\right)\right)
\end{aligned}$$

$$\begin{aligned}
& -2w(\alpha x + (1-\theta)\beta) \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - wT\right) \\
& + (\alpha x + (1-\theta)\beta)w^2T \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - wT\right) \\
& + (\alpha x + (1-\theta)\beta)\mu \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - wT\right) \\
& - (\alpha x + (1-\theta)\beta)\mu wT \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - wT\right) \\
& - 3w^2 + \mu\left(\mu + \delta \tan\left(\frac{\pi}{2k}\right)\right) - \alpha y(\theta\beta - \alpha x) \\
& + \alpha y(\theta\beta - \alpha x) \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - wT\right) \\
& - \alpha y(\theta\beta - \alpha x) wT \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - wT\right) \\
& - \alpha y(\theta\beta - \alpha x)\mu T \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T - wT\right)
\end{aligned} \tag{4.45}$$

Setting $w = 0$

$$\begin{aligned}
F(0) = & (\beta - \mu - \alpha y) \left\{ (\alpha x + (1-\theta)\beta) \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) - \mu \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) \right\} \\
& - \alpha y(\theta\beta - \alpha x) \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right) \right)
\end{aligned}$$

⇒

$$F(0) = (\beta - \mu - \alpha\gamma) \left(\mu + \delta \tan\left(\frac{\pi}{22k}\right) \right) \{(\alpha x + (1 - \theta)\beta) - \mu\} \\ - \alpha\gamma(\theta\beta - \alpha x) \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) \quad (4.46)$$

$$G(0) = -(\beta - \mu - \alpha\gamma) \left\{ (\alpha x + (1 - \theta)\beta) \mu \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T\right) \right\} \\ + \alpha\gamma(\theta\beta - \alpha x) \mu \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T\right) \quad (4.47)$$

$$F'(0) = 0 \quad (4.48)$$

$$G'(0) = (\beta - \mu - \alpha\gamma)$$

$$\left\{ (\alpha x + (1 - \theta)\beta) - (\alpha x + (1 - \theta)\beta) \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T\right) \right\} \\ + (\alpha x + (1 - \theta)\beta) \mu T \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T\right) \\ - \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) - \mu \left\{ \right. \\ - (\alpha x + (1 - \theta)\beta) \left(\mu + \delta \tan\left(\frac{\pi}{2k}\right) \right) \\ \left. + (\alpha x + (1 - \theta)\beta) \mu \exp\left(\frac{2kT\delta}{\pi} \text{Log sec}\left(\frac{\pi}{2k}\right) - \mu T\right) \right\}$$

$$\begin{aligned}
& + \mu \left(\mu + \delta \tan \left(\frac{\pi}{2k} \right) \right) - \alpha y (\theta \beta - \alpha x) \\
& + \alpha y (\theta \beta - \alpha x) \exp \left(\frac{2kT\delta}{\pi} \text{Log sec} \left(\frac{\pi}{2k} \right) - \mu T \right) \\
& - \alpha y (\theta \beta - \alpha x) \mu T \exp \left(\frac{2kT\delta}{\pi} \text{Log sec} \left(\frac{\pi}{2k} \right) - \mu T \right)
\end{aligned} \tag{4.49}$$

From (4.46) to (4.49)

$$F(0)G'(0) > 0 \tag{4.50}$$

Let

$$J_2(k) = F(0)G'(0) \tag{4.51}$$

So the non- zero equilibrium state will be stable when

$$J_2(k) > 0 \tag{4.52}$$

And unstable if otherwise, so:

$$\begin{aligned}
J_2(k) = & \left[(\beta - \mu - \alpha y) \left(\mu + \delta \tan \left(\frac{\pi}{2k} \right) \right) \left[(\alpha x + (1 - \theta)\beta) - \mu \right] \right. \\
& \left. - \alpha y (\theta \beta - \alpha x) \left(\mu + \delta \tan \left(\frac{\pi}{2k} \right) \right) \right] \left[(\beta - \mu - \alpha y) \right. \\
& \left. \left\{ (\alpha x + (1 - \theta)\beta) - (\alpha x + (1 - \theta)\beta) \exp \left(\frac{2kT\delta}{\pi} \text{Log sec} \left(\frac{\pi}{2k} \right) - \mu T \right) \right\} \right. \\
& \left. + (\alpha x + (1 - \theta)\beta) \mu T \exp \left(\frac{2kT\delta}{\pi} \text{Log sec} \left(\frac{\pi}{2k} \right) - \mu T \right) \right]
\end{aligned}$$

$$\begin{aligned}
& - \left(\mu + \delta \tan \left(\frac{\pi}{2k} \right) \right) - \mu \} \\
& - (\alpha x + (1 - \theta)\beta) \left(\mu + \delta \tan \left(\frac{\pi}{2k} \right) \right) \\
& + (\alpha x + (1 - \theta)\beta) \mu \exp \left(\frac{2kT\delta}{\pi} \text{Log sec} \left(\frac{\pi}{2k} \right) - \mu T \right) \\
& + \mu \left(\mu + \delta \tan \left(\frac{\pi}{2k} \right) \right) - \alpha \gamma (\theta \beta - \alpha x) \\
& + \alpha \gamma (\theta \beta - \alpha x) \exp \left(\frac{2kT\delta}{\pi} \text{Log sec} \left(\frac{\pi}{2k} \right) - \mu T \right) \\
& - \alpha \gamma (\theta \beta - \alpha x) \mu T \exp \left(\frac{2kT\delta}{\pi} \text{Log sec} \left(\frac{\pi}{2k} \right) - \mu T \right) \Big]
\end{aligned}$$

Using the Math Cad Software: hypothetical parameter values were used to generate a table of values for $J_2(k)$, so as to analyze the result; some of the values obtained are presented in table 4.2 below.

Table 4.2: The table of values of the Non – zero equilibrium state.

$$\alpha = 0.3 \quad \delta = 1.0 \quad \theta = 0.4 \quad T = 10$$

k	$J_2(k)$ $\beta = 0.5,$ $\mu = 0.2$	Remarks	$J_2(k)$ $\beta = 0.25,$ $\mu = 0.50$	Remarks	$J_2(k)$ $\beta = 0.15,$ $\mu = 0.20$	Remarks	$J_2(k)$ $\beta = 0.25,$ $\mu = 0.15$	Remarks
0.2	0.261	Stable	0.061	Stable	-0.007	Unstable	-0.003	Unstable
0.3	0.003	Stable	0.029	Stable	-0.0002	“	-0.002	Unstable
0.4	-0.001	Unstable	0.020	Stable	0.0002	Stable	-0.004	Unstable
0.5	-0.001	Unstable	0.016	Stable	0.0001	Stable	-0.048	Unstable
0.6	-0.0007	Unstable	0.014	Stable	0.0001	Stable	-0.039	Unstable
0.7	-0.0004	Unstable	0.013	Stable	0.00009	Stable	-0.005	Unstable
0.8	-0.0003	Unstable	0.012	Stable	0.00007	Stable	-0.002	Unstable
0.9	-0.0002	Unstable	0.011	Stable	0.00007	Stable	-0.001	Unstable
1.0	-0.0001	Unstable	0.010	Stable	0.00006	Stable	-0.0008	Unstable

4.3 Analysis and Interpretation of the Equilibrium States

Analysis:

From table 4.1, we note that:

- (a) $J_1(k) < 0$, when $\beta \gg \mu$ (much greater than) and k is high.
- (b) $J_1(k) > 0$, when $\beta \ll \mu$ (much less than) and k is low.
- (c) $J_1(k) > 0$, when $\beta < \mu$ (a little less than) and k is low.
- (d) $J_1(k) > 0$, when $\beta > \mu$ (a little) and k is high or low.

And, from table 4.2, we note that:

- (a) $J_2(k) > 0$, when $\beta \gg \mu$ (much greater than) and k is low.
- (b) $J_2(k) > 0$, when $\beta \ll \mu$ (much less than) and k is high.
- (c) $J_2(k) < 0$, when $\beta < \mu$ (a little less than) and k is low.
- (d) $J_2(k) < 0$, when $\beta > \mu$ (greater than) and k is high.

Note however that the results presented in table 4.1 and table 4.2 above are for $\delta=0.3$, $\theta=0.4$ in each case and $\alpha=0.3$ in table 4.2. The profile remains the same as these values range from 0.2 to 1.

Interpretation:

From table 4.1 the zero equilibrium state is unstable only when the natural birth rate (β) is very much greater than the natural death rate (μ) even as the value of k is increasing (i.e. with the increase in drug

application) with the infection age. The population is stable when the natural birth rate (β) is much less than or a little less than the natural death rate (μ) even when the value of k is increasing. This is because naturally, the population will be decreasing with the additional burden of the scourge and stable also when the natural birth rate (β) is a little greater than the natural death rate (μ) even as the value of k increases, if otherwise the natural birth rate (β) must be strictly greater than the natural death rate (μ).

From table 4.2, the non-zero equilibrium state (which is the state of population sustenance) is stable only when the natural birth rate (β) is much less than or a little less than the natural death rate (μ) even as the value of k increases. But unstable when the natural birth rate (β) is much greater than and a little greater than the natural death rate (μ) as the value of k continue to increase. Interestingly, the zero-equilibrium state is stable when the natural birth rate (β) is a little greater than the natural death rate (μ) even when the value of k is increasing, this is due to a little difference between the former and the later despite the burden of the disease on the community.

CHAPTER FIVE

5.0 CONCLUSION AND RECOMMENDATION

5.1 Conclusion:

From the observations, we conclude that the zero equilibrium state will be stable when the natural birth rate (β) is less than the natural death rate (μ); while, the non – zero equilibrium state will be unstable when the natural birth rate (β) is greater than the natural death rate (μ) even as the value of k is increasing.

A low value of k indicates that the effectiveness of effort in curbing with the scourge of the disease is low resulting in high death rate among the infected, thus leading to the stability of the zero equilibrium state; and consequent wiping out of the population. This implies that once the disease–AIDS is introduced into a community, the dynamics of the system faces likely extinction of the populace except for intervention.

It follows therefore, that the application of anti – retroviral drugs can at best slow down the eventual extinction of the infected population as illustrated in fig.1- the graph of infection age against the death rate via infection and also as observed in table (4.2). This may suggest that the ideal control may be to completely remove the infected group and prevent

other members of the population that are virus free from contacting the virus.

5.2 Recommendation:

The obvious stability of the zero equilibrium state explains strongly why the pandemic tends to wipe out communities once it sets in. We therefore recommend that:

1. Public enlightenment should be intensified, so that more people would go for HIV–Status–Test to prevent further spread of the virus.
2. Also, with awareness campaign stigmatization and discrimination of HIV / AIDS victims would be minimized.
3. Government should put in place potent legislations to punish individuals who in any way contribute to the spread, this will invariably reduce the spread of the HIV among the populace.
4. The anti–retroviral drug should be easily accessible and affordable to those HIV patients, whom inadvertently would have the hope of living a normal life and possibly live longer after infection.
5. Any effort aimed at prevention still stands out as the optimal way out of the pandemic.

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APPENDIX

Bellman and Cooke's Theorem for Stability of Characteristic

Equations

Theorem:

Let

$$\Delta(z) = P(z, e^z) \quad (\text{A.1})$$

where $P(z, w)$ is a polynomial with principal term. Suppose that $\Delta(iy)$, for every $y \in \mathbb{R}$ is separated into its real and imaginary parts,

$\Delta(iy) = F(y) + iG(y)$. If all zeros of $\Delta(z)$ have negative real parts, then the zeros of $F(y)$ and $G(y)$ are real, simple, alternate and

$$G^1(y) F(y) - G(y) F^1(y) > 0 \quad (\text{A.2})$$

for $y \in \mathbb{R}$. Conversely, all zeros of $\Delta(z)$ will be in the left half - plane provided that either of the following conditions is satisfied.

(i) All the zeros of $F(y)$ and $G(y)$ are real, simple and alternate and inequality (A.2) is satisfied for at least one y .

(ii) All the zeros of $F(y)$ are real and, for each zero, Relation (A.2) is satisfied.

(iii) All the zeros of $G(y)$ are real and, for each zero, Relation (A.2) is satisfied.