A MATHEMATICAL MODEL OF THE DYNAMICS OF HIV/AIDS EPIDEMIC WITH LATENT AGE-STRUCTURE

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OCTOBER, 2005

CERTIFICATION

This thesis titled: A MATHEMATICAL MODEL OF THE DYNAMICS OF HIV/AIDS EPIDEMICS WITH LATENT AGE-STRUCTURE by KINGSLEY, CHINANUEKPERE OGBONNA 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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DECLARATION

I hereby declare that this thesis has been written by me and that it is a record of my research work. All published and unpublished sources of information have been appropriately acknowledged.

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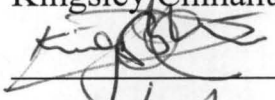
Signature: 

Date: 05/10/05

DEDICATION

This research thesis is dedicated to my brother Simeon Oziri Ogbonna for giving me the necessary financial and moral support during this programme.

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I want to thank God for his love and mercy towards me throughout this programme, and also for giving me understanding and the strength to complete this programme in record time. I am also grateful to my brother Simeon Oziri Ogbonna for his invaluable financial and moral support during this period. His advice and encouragement motivated me greatly to achieve nothing but success in this study. My thanks also go to every member of my family for their care and support, which contributed immensely to the success of this work.

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CONTENTS

ABSTRACT

We propose a mathematical model of the dynamics of HIV/AIDS epidemic with the population of the community partitioned into three compartments of Susceptible, Latent and Infected classes. The latent class is structured into "latent age", which results in a set of three equations with one partial differential equation and two ordinary differential equations. The model equations are then analysed for stability of equilibrium state. The results indicate that the origin will always be stable except where the birth rate is greater than death rate of the population. It was also observed that the model shows a tendency for the epidemic to stabilise with the application of control or mitigation measures. We also discovered that the non-zero equilibrium state will always be unstable except where the birth rate is much greater than the death rate, where the rate of contracting the virus as well as death burden due to infection is particularly low.

CHAPTER ONE

INTRODUCTION

1.1 BACKGROUND TO THE STUDY

A mathematical model is a mathematical description of a real life situation. It is a process of creating a mathematical representation of some phenomenon in order to gain better understanding of that phenomenon. This work therefore, seeks to apply mathematical modelling as a tool for analysing the impact of HIV/AIDS epidemic on any giving population with time. The objective of this study is to investigate the sustainability or otherwise of any given population made up of the susceptible, the latent, and the infected classes.

In this work, we propose a mathematical model of the dynamics of HIV/AIDS epidemic with Latent Age-Structure. The model comprises a system of ordinary differential equations, partial differential equations and integral equations, which we shall solve to obtain the equilibrium states of the model. The stability analysis of the zero and non-zero equilibrium states is then carried out to determine the conditions for stability or otherwise of the equilibrium states.

For the purpose of this study, the population is divided into three classes of Susceptible S(t), which is the class whose members are HIV free but are prone to the virus infection as they interact with members of the other two classes; the second class is the Latent L(t) whose members have contracted the HIV virus but have no symptoms of the AIDS infection; the third class is the Infected I(t): this is the class of those that exhibit the symptoms of the full blown AIDS disease at various stages of infection.

The Latent class is structured by the latent age 'τ' with a density function $\rho(t,\tau)$, where t is the time . The rate of flow from the latent class into the infected class is given by $\sigma(\tau)=\tan\left(\frac{\pi\tau}{2kT}\right)$, which tends to infinity as 'τ' approaches its maximum 'T' where k is a control parameter which represents measures taken to slow down the rate of infection. These measures include public enlightenment campaign on behavioural change and safe sexual and medical practices, public health education, voluntary HIV testing, and use of antiretroviral drugs to slow down infection. We have assumed a maximum latent age T with $0 \le \tau < T$ and $0 < k < 1$. The population has a natural birth rate 'β' and death rate 'μ', while the infected class has an additional death rate due to AIDS infection δ.

It is also assumed that new births in S(t) are born into S(t), the new births in I(t) are born into I(t), while the offspring of L(t) are divided between S(t) and L(t) in the proportion θ and 1-θ respectively with $0 \le \theta \le 1$. This is because HIV infected pregnant women can be medically treated to keep the unborn baby free from HIV infection.

The relevance of this research work cannot be over emphasized considering the disastrous effect of the HIV/AIDS epidemic on the development of many poor countries in various parts of the world. Therefore, in this first chapter we give a general introduction to the study as well as objectives of this research work. Also presented in this chapter are the scope and limitations of this research work.

In chapter two, we present a review of the literature of epidemiological models of HIV/AIDS, and the impact 0f HIV/AIDS epidemic on Africa with particular emphasis on the Nigerian case and the response strategies for prevention and management of the epidemic.

Chapter three provides the model equations using letters and symbols to represent the parameters and factors considered in this model including the initial conditions. The introduction of age-structure in the Latent class gave rise to a system of ordinary differential, partial differential and integral equations. We then solve these equations to obtain the equilibrium states of the model. The solution shows that the origin is an equilibrium state; the non-zero equilibrium state is also obtained.

In chapter four, the zero equilibrium state is analysed for stability. Assuming a perturbation in the model equations, we evaluate the resulting equations to obtain the characteristic equation. We applied numerical method in evaluating some functions that emerged in the course of our analysis. The analysis shows that both the zero and non-zero equilibrium states will be stable for some conditions on natural birth and death rates of the population.

Finally, chapter five contains suggestions and conclusion based on the result of the stability analysis. MathLAB was used to generate table of values for the stability functions with hypothetical values for the parameters. Useful suggestions on the sustainability of population are also given based on the results obtained.

1.2 SCOPE AND LIMITATIONS OF THE STUDY

We shall consider an age-structure for the Latent class of the population and this gives rise to a system of integral and partial differential equations while the Susceptible and Infected classes are represented by ordinary differential equations. Age-structure was eliminated from the Infected class for ease of analysis. Also introduced is a control/mitigation parameter 'k', which is a measure of the effectiveness of responses both preventive and control to the HIV/AIDS epidemic.

The model equations were evaluated to obtain the equilibrium states of the model and the characteristic equation. We also carried out the stability analysis of the equilibrium states using the characteristics equation. We have covered the stability analysis of the zero equilibrium state (i.e. origin) and the non-zero equilibrium state. We also compared the results obtained from the stability analysis of both the zero and non-zero equilibrium states to establish the consistency or otherwise our result.

CHAPTER TWO

LITERATURE REVIEW

2.1 INTRODUCTION

This chapter presents a literature review of HIV/AIDS and Mathematical epidemiology with emphasis on HIV/AIDS models. This includes the impact of HIV/AIDS epidemic on Africa.

2.2 IMPACT OF HIV/AIDS EPIDEMIC ON AFRICA

In recent times, HIV/AIDS epidemic is considered as the greatest health problem threatening the human race, where the burden is greatest in Sub-Saharan Africa. It has become one of the most important challenges to the continuing existence and development of many poor countries in various parts of the world. AIDS has wiped out over four decades of development progress in the worst hit countries (DFID HIV/AIDS Strategy Report, May 2001). According to the *Joint United Nations Programme on HIV/AIDS*, over 40 million people had been infected with the virus by the end of 2003 of which over 28 million were in Sub-Saharan Africa. In Sub-Saharan Africa, HIV/AIDS has shattered many families and placed extraordinary burden on the extended family and village system that have been the backbone of African child rearing tradition. In countries where prevalence is lower; the impact on many highly vulnerable groups will be severe.

The levels of infection are particularly high in India and South-East Asia. *A DFID HIV/AIDS Strategy Report of May 2001* gave the global estimates for HIV/AIDS epidemic as at the end of 2000 as follows: people living with HIV/AIDS, 36.1 million; new HIV infection in 2000, 5.3 million; death due to HIV/AIDS, 3.0 million; cumulative death due to HIV/AIDS since the start of epidemic, 21.8 million; life expectancy reduced up to 20 – 30 years; 15 million AIDS orphans (with an estimated 30.2 million by 2010); 1 -2% loss in GDP per year in hardest hit countries. In many counties, especially sub-Saharan Africa, HIV/AIDS is putting an enormous strain on health and education services by drastically reducing the number of staff, the number of new trainees, and the length of service of current staff with HIV/AIDS. The instructional capacity of services is being seriously affected. At the same time the health care sector is overwhelmed with a massive increase in patients (up to 80% of hospital beds being occupied by patients with HIV/AIDS related illness). Although latest reports show a downward trend in the HIV/AIDS figures, it is not yet time to relax in the fight against this deadly pandemic.

HIV/AIDS has many interconnected causes and consequences. Therefore, any response to the epidemic must be rooted on an understanding of why people are at risk of infection. A USAID report gave the means and proportion of HIV transmission globally as: Sexual transmission (both heterosexual and homosexual) – 80%; Injecting drug use (sharing needles) – 5%; Unsafe blood transfusions – 5%; Mother to child transmission – 10%. The risk of sexual transmission is substantially increased by the presence of other sexually transmitted infections (STI), high rates of partner exchange, lack of health information and other high risk behaviours. Alcohol is associated with high risk

behaviour in men, while biological vulnerability and female genital mutilation place women at high risk of infection.

There are two main virus types, HIV1 and HIV2. HIV1 is more easily transmitted than HIV2 and contributes more to the global pandemic. There are ten different virus subtypes each of HIV1 and HIV2. This poses substantial technical problems for vaccine development. The time between infection with the virus and death varies, but can be more than 10 years, in developing countries it is frequently less. "AIDS" refers to a clinical definition of when an individual's immune system has become progressively weak, which makes the individual prone to opportunistic infections. In developing countries, technology and systems do not exist to monitor population's immune systems. However, Anti-retroviral (ARV) therapies can delay the onset of full blown AIDS infection, but there is not sufficient evidence to show that they also reduce transmission of HIV. There is presently no effective vaccine against HIV or cure for HIV/AIDS. Therefore, effective response to the epidemic should be based on proper understanding of factors relating to means and pattern of transmission, and vulnerability to HIV infection, with priority on strategies to promote prevention, while reducing the impact of the epidemic especially at individual, family and community levels.

2.2.1 Epidemiology of HIV/AIDS in Nigeria

The 2003 Sero-Prevalence Sentinel Survey by the National AIDS/STDs Control Programme of the Federal Ministry of Health indicates that some parts of the country are worse affected than others, but no state or community is unaffected. All the states in Nigeria have a general population HIV prevalence of over 1%.

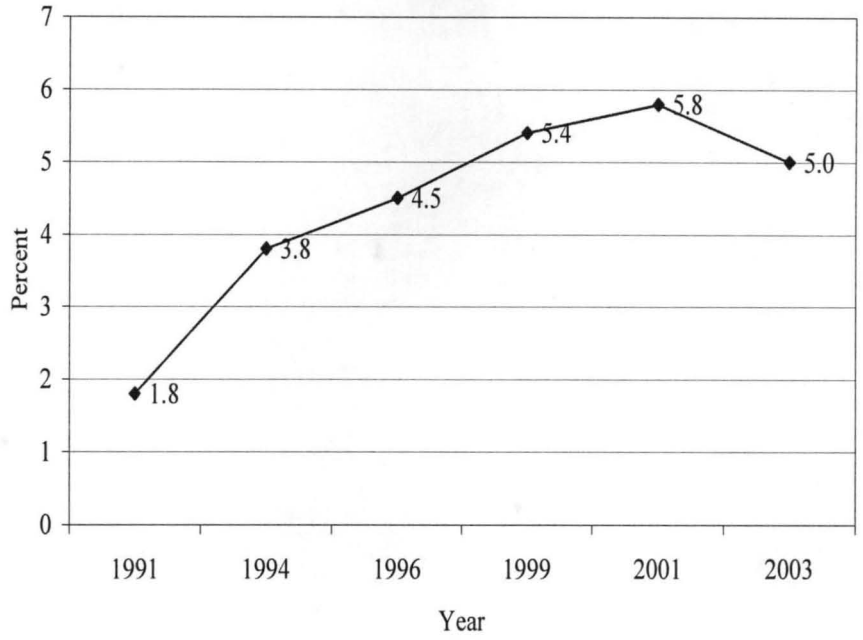
Although the first case of HIV/AIDS in Nigeria was reported in 1986, the epidemic has grown steadily since then.

The adult HIV prevalence has increased from 1.8% in 1991 to 4.5% in 1996, and 5.8% in 2001. Estimates using the 2001 HIV/Syphilis Sero-Prevalence Sentinel Survey among women attending antenatal clinics reveal that more than 3.5 million Nigerian were infected with HIV by the end of 2001, which is the highest figure in West Africa. The epidemic in Nigeria has extended beyond the commonly classified high risk groups and is now common among the general population. In 2002 alone, more than 200,000 AIDS deaths occurred, and it was estimated that there were over 1.0 million AIDS orphans in Nigeria.

With the adult HIV prevalence rate at 5.8% in 2001, the nation was said to be at the threshold of an exponential growth of the epidemic. In some communities, prevalence was higher than 10.0%. HIV/AIDS affects all age groups; but youths between the ages of 20 and 29 years are more infected, though in some zones (south-south and south-west), there was a higher prevalence in the 15-19 years age group as recorded in the 2001 Sentinel Survey.

Figure 2.1

Graphical representation of HIV Prevalence in Nigeria (1991-2003)



Source: Federal Ministry of Health, 2003 HIV Sero-prevalence Sentinel Survey (2004)

Recently, an increasing number of children are being either infected with the virus through mother-to-child transmission, or are loosing one or both parents to the disease. By all indications, the HIV/AIDS epidemic has continued to grow largely through heterosexual unprotected sexual interactions. AIDS cases are becoming more visible in communities. Although AIDS case reporting has been characterized by under-reporting, delayed reporting and under-recognition the number of reported cases has been on the increase, especially since 1996. Similarly, the HIV prevalence rate among sex workers in Nigeria remained high and on the increase from 17.5% in 1991, through 22.5% (1993) to 35.6% in 1995.

The latest data, obtained through the 2003 HIV Sero-Prevalence Sentinel Survey, estimate the national HIV prevalence for Nigeria at 5.0%, which shows a decrease from 5.8% in 2001. However, this figure conceals significant regional differences, from 2.3% in South-West to 7.0% in North-Central. State level variations are even larger, from 1.2% in Osun and 1.5% in Ogun, over 6.0% in Kaduna and 6.3% in Plateau to 9.3% in Benue and 12.0% in Cross River state. The divergence and irregular patterns of HIV prevalence rates and trends across zones and states demonstrates that the dynamics of the epidemic are different in each zone and state, which probably reflects different realities, different determinant and vulnerabilities, and different pace of the response at these levels.

According to the 2003 report, Nigeria is the third most affected country in the world, behind South Africa and India, in terms of numbers of people living with HIV/AIDS. In this context, the high prevalence rates among young people make the case for identifying prevention efforts through strengthening HIV/AIDS education in and out of school. Empowerment of young girls and women to develop the knowledge and skills to protect themselves from HIV infection also needs to be emphasized. Since the rural HIV prevalence is not markedly different from the urban prevalence, intervention strategies should equally target the rural communities.

There is the need for an increasing political commitment in the fight against this global scourge. Resource mobilization and multi-sectoral responses need to be strengthened and sustained, especially at the state and community levels. In anticipation of an increasing demand arising from the large numbers of estimated HIV/AIDS cases in the country, the existing care and support activities need to be scaled up. A comprehensive response strategy should place

particular emphasis on support for AIDS orphans and the anti-retroviral programme providing treatment to People Living with HIV/AIDS while eliminating stigma and discrimination. Other health sector interventions to be reinforced include; the National Safety programme, the provision of Voluntary Counselling and Testing services, the prevention and treatment of Sexually Transmitted Diseases and the treatment of Opportunistic Infections.

2.3 LITERATURE REVIEW OF EPIDEMIOLOGICAL MODELS HIV/AIDS

The use of mathematical models as research tools in the study of the dynamics of disease epidemiology, especially vector born diseases has been going on for a long time. As a result, the relationship between disease epidemiology and population dynamics has been studied in various models. We hereby present a review of some of these studies which are relevant to HIV/AIDS.

John Pickering, et al (1986) in a work, "Modelling the Incidence of AIDS in San Francisco, Los Angeles, and New York", presented a discrete nonlinear model, which was used to explore underlying biological and sociological characteristics of the AIDS outbreak and to forecast the number of new cases. The model dynamics assumed that AIDS is sexually transmitted and that other forms of transmission mimic sexual transmission. Its parameters reflect (1) how long AIDS takes to develop from exposure to diagnosis, (2) when during this development individuals are contagious, and (3) how changes in sexual behaviour and saturation – the removal of susceptible individuals through infection – affects the incidence of AIDS. The model was used in conjunction

with anal/rectal gonorrhoea rates from San Francisco to generate preliminary forecast of AIDS incidence, and the model's best fits to observe incidence suggesting that there would be at least 2,400, 7,200 and 2,500 cumulative cases in Los Angeles, San Francisco, and New York respectively.

Claude Lefevre and Marie-Pierre Malice, (1985) studied "A Discrete Time Model for S-I-S Infectious Disease with a Random Number of Contacts Between Individuals", in which they formulated a chain of binomial deterministic model for the spread of infectious disease of S-I-S type that accounts explicitly for the distribution of the number of contacts made by each susceptible during one time interval. Under certain hypothesis, a threshold theorem for endemicity is derived, bounds for the endemic level are constructed, and the transient behaviour of the epidemic process is investigated.

Andrea Di Liddo (1985), proposed a S-I-R Vector Disease Model with Delay in which he analysed the deterministic epidemic model with derived by the Kermack-McKendrick model. The model is suitable to describe infections transmitted by a vector. Existence and uniqueness, stability and asymptotic behaviour of the solutions were studied.

To assess the effect of random test and various factors on AIDS, W.Y. Tan and F. Shamsa (1990) published a paper "Assessing the Effect of Intervention on AIDS Development Induced by Blood Transfusion" in which they considered the situation where AIDS transmission is developed by blood transfusion and then proceeded to develop a stochastic model involving latency of AIDS

viruses. Using a computer simulation, they computed and compared the probabilities of developing AIDS for both tested and not tested individuals. The effects of various factors were also assessed by computer simulated results. The indicated that even within the Weibull family, different probability distributions of latency and incubation exert very different effects on the time from contraction of virus to manifestation of AIDS symptoms and the effects of intervention by random testing and treatment.

Ying-Hen Hsieh (1990), proposed an AIDS model with screening, in which He studied the transmission dynamics of AIDS epidemic in a male homosexual population using a compartment model along the lines of those proposed by Andrew et al (1986). The emphasis was to gain qualitative insight into the future trends of the very unique disease. Analytical results were produced with implications on the possible effects of random screening and removal on the population.

W.Y. Tan (1990) developed a stochastic model for AIDS epidemic involving several risk populations. The probability generating function (PGF) of the latent persons, infected persons and AIDS cases was derived. By using PGF, it was shown that the expected values, the variances and the covariances of the latent persons, infected persons and AIDS cases satisfy some ordinary differential equations. These equations were solved numerically to assess effects of various factors on the AIDS epidemic.

In 1990, A. Pugliese studied an S-I epidemic model with a general shape of density-dependent mortality and incidence rates. He found out the global asymptotic convergence to an endemic equilibrium above a threshold, and to disease-free equilibrium below the threshold. He also examined the effect of vaccination on the population.

Similarly, F. Brauer (1990) formulated models of S-I-R type for the spread of communicable diseases. He considered models which include non-linear population dynamics with permanent removal, 'R'. The principal result of this study is that the stability of endemic equilibrium may depend on the population dynamics and on the distribution of infective periods; sustained oscillations are possible in some cases.

Garnett G.P. and Anderson R.M. (1994) formulated a mathematical model of the transmission dynamics of HIV-1 in a heterosexual population stratified by age, sex, and sexual activity (defined by rates of sexual partner acquisition). The model represents an extension of an extension of previous studies with a special focus on patterns of mixing or contact between sexual activities and different age classes of the two sexes. A range of mixing patterns between these groups is specified for both sexes. Mixing is described on two scales from fully assortative to fully disassortative, with random defined either according to numbers of sexual partnerships or numbers of people. The sexual partnerships in the model are balanced by changes in the rates of sexual partner acquisition between particular groups and a range of changes, from only women changing behaviour to only men changing behaviour, were analysed. The pattern of mixing is most influential in determining the shape and magnitude of the

epidemic, but the manner in which people choose partners (i.e. dependent on numbers or proportions in the population) is also important. The relative importance of variation in transmission possibilities and mean rates of partner change on the course of the HIV epidemic was also illustrated. The analysis of the sensitivity of predictions to changing parameters in the force of infection term of the model provides a theoretical basis from which the influence of control strategies and the demographic effects of HIV can be analysed.

In another publication, Garnett G.P. and Anderson R.M. (1995) presented a paper titled 'Strategies for Limiting the Spread of HIV in developing Countries: Conclusions Based on Studies of the Transmission dynamics of the Virus'. They examined possible interventions to reduce the spread of HIV including actions that attempt to alter sexual behaviour, such as education aimed at reducing the rate at which individuals acquire new sexual partners, and methods that reduce the probability of transmission between partners, such as promotion of condom use and the treatment of so-called "cofactor" sexually transmitted diseases. A mathematical model of HIV transmission that is able to mimic different approaches to the control of HIV transmission was employed to study the relative values of different approaches, either used in isolation, or in combination. The nonlinear nature of the term that describes the per capita rate of transmission dictates that for a given degree of intervention, the benefit accruing in terms of reduced HIV spread depends on the prevalence on infection before the introduction of control. Benefit is greatest when HIV prevalence is low Combination approaches were predicted to be effective but the outcome is less than would be expected on the basis of simply summing the benefits resulting from each type of intervention used in isolation. The success of targeted interventions, aimed at those with high rates of sexual partner

change, depends on the heterogeneity in levels of sexual activity within populations and what proportion of the population HIV is able to establish itself in. Targeted interventions were predicted to be very cost effective but their overall success in reducing HIV spread by a significant degree depends on the timing of their introduction (within the time frame of the development of the epidemic) and the pattern of mixing between different risk groups or sexual activity classes.

M. Kakehashi (1998) analysed the spread of HIV/AIDS in Japan using a mathematical model incorporating pair formulations between adults and sexual contacts with commercial sex workers. The parameters involved in the model were carefully specified as realistically as possible to the actual situation in Japan. Plausible ranges were assigned to those parameters for which values are not known precisely. The model was used to simulate the effect of HIV infected commercial sex workers introduced into a population without HIV. It was shown that the model could generate different scenarios, an explosive infection or a temporal spread, according to different settings of the parameters. Then the condition for occasional introduction of HIV infected commercial sex workers to be able to cause an explosive spread of HIV infection was analysed. This condition was summarised in terms of the critical transmission probability so that we could easily evaluate the degree of the risk. For some unclear parameters, sensitivity to the critical transmission probability was calculated. They also calculated a plausible range of the critical transmission probability using the Latin hypercube sampling method where the parameters were distributed on the plausible ranges. According to the analyses of the model it was concluded that the actual situation of HIV spread in Japan should lie very near the critical point that determines whether the explosion HIV spread

actually takes place. This also suggests that effective action taken immediately could be useful to prevent explosive HIV infection in Japan.

Tan W.Y. and Xiang Z. (1998) developed a state space model for the HIV epidemic in homohexual population which have been divided into subpopulations according to sexual activity levels. In this model, the stochastic dynamic system model is the stochastic model of the HIV epidemic in terms of the chain multinomial whereas the observation model is a statistical model based on the observed AIDS incidences. This model was applied to the San Francisco homosexual population for estimating the number of susceptible people, infected people and AIDS cases and for estimating the probabilities of HIV transmission from infective people to susceptible people given sexual contacts. The results show that the estimated numbers of AIDS incidence trace closely the observed numbers indicating the usefulness of the model. They observed that the estimated numbers of latent people show multimodal curves and that HIV infection takes place during the primary stage and very late stage. The result further showed that there are significant differences between the observed AIDS incidences and the estimates by the embedded deterministic model. These results indicates that using embedded deterministic model to estimate the HIV-infected people and to predict future AIDS cases can be very misleading in some cases.

Hsieh Y.H. and Cooke K. (2000) considered 'Behaviour change treatment of core groups: its effect on the spread of HIV/AIDS', using a general model for treatment and behaviour change of the HIV infected in a highly sexually active core group of female commercial sex workers and a 'bridge population' of young unpartnered males. In this model, the spread of HIV/AIDS in the

community is carried out mainly through the sexual interaction between the core group and the bridge population which acts as a bridge for the spread of disease to the general population. They also considered the effect of treatment of the infected and/or the subsequent behaviour change when targeted toward the core group and the bridge population. Analytical results were given for a strategy which targets treatment and behaviour change at either the core group or the bridge population. Numerical examples were also provided to illustrate the biological significance of the treatment/behaviour change and its effect of the threshold parameter values. The results show that if the contact rates and transmission probabilities of the treated individuals are sufficiently reduced, the treatment/behaviour change can eradicate the disease provided that the level of treatment in the infected population is sufficiently high. However, an ill-planned treatment programme which fails to meet the required reductions in contact rate or transmission probability could have a detrimental effect on the spread of the epidemic.

Similarly, Auvert B. et al (2000) used a stochastic simulation model to asses the extent to which variation in sexual behaviour and transmission characteristics can explain the striking spatial heterogeneity in the prevalence of HIV among different geographical locations in Sub-Saharan Africa. Of the various parameters describing sexual behaviour the most important determinant of the spread of HIV is the proportion of men engaging in sexual relationships with people other than their spouses, including contacts with sex workers and short-term partners. Considering factors other than sexual behaviour the model shows that this heterogeneity in HIV prevalence could be the result of differences in the transmission probability of HIV or in the prevalence of other sexually transmitted diseases. These factors could play a key role in determining the

patterns of spread of HIV in sub-Saharan Africa and should be considered in the design of intervention strategies.

Nico J.D. Nagelkerke, et al (2002), in paper 'Modelling HIV/AIDS epidemics in Botswana and India: impact of interventions to prevent transmission' described a dynamic compartmental simulation model for Botswana and India, developed to identify the best strategies for preventing spread of HIV/AIDS. They considered the following interventions: a behavioural intervention focused on female sex workers; a conventional programme for the treatment of sexually transmitted infections; a programme for the prevention of mother-to-child transmission; an antiretroviral treatment programme for the entire population, based on a single regimen; and an antiretroviral treatment programme for sex workers only, also based on a single regimen. Their findings indicate that interventions directed at sex workers as well as those dealing with sexually transmitted infections showed promise for long-term prevention of HIV infection, although their relative ranking was uncertain. In India, a sex worker intervention would drive the epidemic to extinction. In Botswana none of the interventions alone would achieve this, although the prevalence of HIV would be reduced by almost 50%. Mother-to-child transmission programmes could reduce HIV transmission to infants, but would have no impact on the epidemic itself. In the long run, interventions targeting sexual transmission would be even more effective in reducing the number of HIV-infected children than mother-to-child transmission programmes. Antiretroviral therapy would prevent transmission in the short term, but eventually its effects would wane because of the development of drug resistance.

N.I. Akinwande (2005) in a study applied the theory of Laplace Transformation to carry out a stability analysis of the non-zero equilibrium state of an Age-Structured mathematical model of Yellow Fever disease dynamics. In this model, age-structures were introduced to the infective hosts' class, and the two compartments of the vectors. He also considered the urban yellow fever transmission cycle for modelling with two primary communities namely the Host and the Vector; the host community is made up of human beings while the vector community consists of aedes egypti. In his analysis, he established a sufficient condition necessary for exponential asymptotic stability of the non-zero equilibrium state of the model.

Flugentius Baryarama et al (2005) formulated an HIV/AIDS mathematical model incorporating complacency for the adult population. Complacency is assumed a function of the number of AIDS cases in a community with an inverse relation. A method to find the equilibrium state of the model is given by proving a stated theorem. An example to illustrate the application of the theorem is also given. The model analysis and simulations show that complacency resulting from dependence of HIV transmission on the number of AIDS cases in a community leads to damped periodic oscillations in the number of infective with oscillations more marked at lower rates of progression to AIDS. The implications of these results to public health with respect to monitoring the HIV/AIDS epidemic and widespread use of antiretroviral (ARV) drugs is discussed.

In another study, N.I. Akinwande (2005) proposed a mathematical model of the dynamics of HIV/AIDS pandemic and analysed the zero equilibrium state for

stability. He introduced age-structure in the infected class and the influence of drug application and public enlightenment aimed at controlling the attitude of the populace as a measure of preventing/reducing the spread of the virus. The analysis revealed that the zero equilibrium state will always be stable except for situations where the birth rate is greater than the death rates.

CHAPTER THREE

MODEL EQUATIONS AND EQUILIBRIUM STATE

3.1 INTRODUCTION

We formulate a mathematical model of the dynamics of HIV/AIDS epidemic with latent age structure by considering a population which is made up of the Susceptible S(t), the Latent L(t) and the Infected I(t) classes and we assume that the three classes interact with one another .Then the rate of contracting the HIV depends on rate and level of interaction (sexual and other risky contacts) between the first class and the other two classes. At any time t, the susceptible class comprises those born without the HIV at the rate β minus those that leave the class through death at the rate μ and by contracting the virus at the rate α. This relation is represented in equation (3.1), in which we applied the mass action law based on our assumptions. Equation (3.2) is a partial differential equation which rises from the fact that a HIV victim may die naturally at the rate μ or flow into the infected class at the rate $\sigma(\tau)$.

The infected class I(t) is made up of those born with the full blown AIDS infection at the rate β and those that flow from the latent class into the infected class at the rate $\sigma(\tau)$, and exit through a cumulative death rate from natural causes and burden of infection δ. This is represented by the differential equation (3.3). The rate of infection $\sigma(\tau)$ is defined in equation (3.4) which shows that the rate increases to infinity as the virus latent age τ approaches its maximum T.

Therefore, at any time t, and initial stage of the HIV contraction with the latent age $\tau = 0$, the latent class L(t) takes the form B(t) which consist of those born with the virus at the rate β, and those who just contracted the virus at the rate α, as shown in equation (3.5). Equation (3.6) represents the Latent class which is a transition from the susceptible class to infected class over a period τ, with $0 \le \tau < T$, While equation (3.7) represents those infected at the initial time $t = 0$. Also presented in equation (3.8) are boundary conditions of the model equations.

3.2 THE MODEL EQUATIONS

This section presents the model equations using parameters which we shall define later in this section. Our model allows for the general application of the mass action law. We have also assumed the natural birth β and death rates μ of the population as well as rate of contracting HIV α and additional death burden due to AIDS infection δ to be constants. The model equations are given by;

$$\frac{dS(t)}{dt} = (\beta - \mu)S(t) + \theta\beta L(t) - \alpha S(t)[L(t) + I(t)] \tag{3.1}$$

$$\frac{\partial \rho(t,\tau)}{\partial t} + \frac{\partial \rho(t,\tau)}{\partial \tau} + (\mu + \sigma(\tau))\rho(t,\tau) = 0 \tag{3.2}$$

$$\frac{d\mathrm{I}(t)}{dt} = (\beta - \mu - \delta)\mathrm{I}(t) + \int_0^T \sigma(\tau)\rho(t,\tau)d\tau \tag{3.3}$$

$$\text{Let } \sigma(\tau) = \tan\left(\frac{\pi\tau}{2kT}\right), \text{ where } 0 \le \tau < T \text{ and } 0 < k < 1 \tag{3.4}$$

$$\rho(t,0) = \mathrm{B}(t) = (1-\theta)\beta L(t) + \alpha S(t)[L(t) + \mathrm{I}(t)] \tag{3.5}$$

$$L(t) = \int_0^T \rho(t,\tau)d\tau : 0 \le \tau < T \tag{3.6}$$

$$\rho(0,\tau) = \Phi(\tau) \tag{3.7}$$

$$S(0) = S_0; \quad L(0) = L_0; \quad \mathrm{I}(0) = \mathrm{I}_0 \tag{3.8}$$

Equation (3.4) represents the rate of flow of people from the latent class L(t) into the infected class I(t), i.e. the rate of infection. It can be observed from this equation that the rate of infection, $\sigma(\tau)$ tends to infinity as the Latent virus age, τ approaches its maximum, T. At the point of contracting the virus, we assume an initial value for the latent age $t = 0$, so that the rate of infection is at zero. This can be seen from a graph of $\sigma(\tau)$ versus τ as shown below.

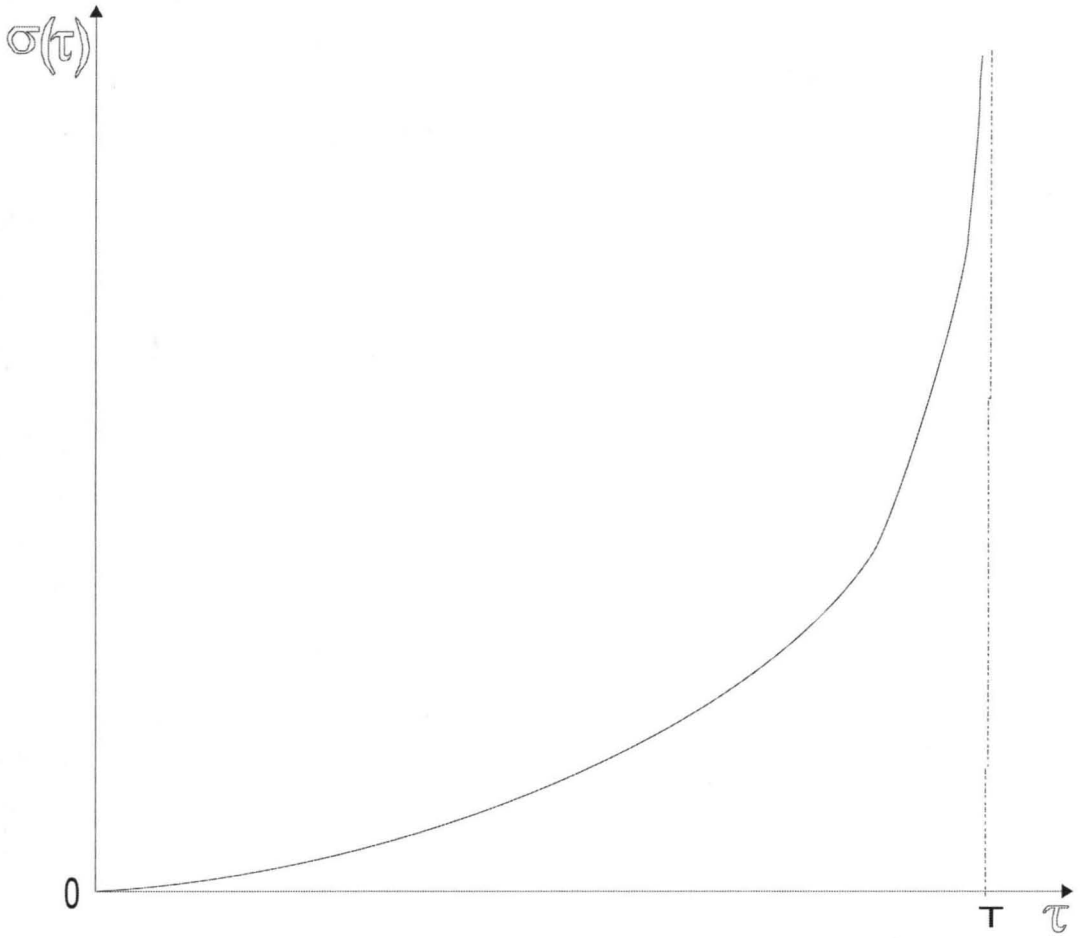


Figure 3.1 – rate of flow from the latent class into the infected class $\sigma(\tau)$

The parameters used in this model are defined as follows:

- β — Natural birth rate of the population.
- μ — Natural death rate of the population.
- α — Rate of contracting the HIV.
- $\sigma(\tau)$ — Rate of flow of people from the Latent class L(t) into the Infected class.
- τ — Latent/virus infection age.
- k — Control parameter which measures the effectiveness of efforts at slowing down the rate of infection.
- T — Maximum Latent/virus infection age.
- δ — Additional death rate due to infection.
- t — Time variable.
- θ — Proportion of new births in L(t) that are born into S(t).
- $1-\theta$ — Proportion of new births in L(t) that are born into I(t); where $0 \leq \theta \leq 1$.

3.3 EQUILIBRIUM STATE OF THE MODEL

In this section, we shall use the model equations to obtain the equilibrium states of the model.

At equilibrium state, let;

$$S(t)=x;\ \ L(t)=y;\ \mathrm{I(t)}=z;\ \ \rho(t,\tau)=\phi(\tau); \tag{3.9}$$

Hence, $y=\int_0^T \phi(\tau)d\tau$ (3.10)

$$\phi(0)=\mathrm{B}(0)=(1-\theta)\beta y+\alpha x(y+z) \tag{3.11}$$

Substituting equations (3.9) to (3.11) into the model equations, we have;

$$(\beta-\mu)x+\theta\beta y-\alpha x(y+z)=0 \tag{3.12}$$

$$\frac{d}{d\tau}\phi(\tau)+(\mu+\sigma(\tau))\phi(\tau)=0 \tag{3.13}$$

$$(\beta-\mu-\delta)z+\int_0^T \sigma(\tau)\phi(\tau)d\tau=0 \tag{3.14}$$

Solving the o.d.e (3.13), we obtain;

$$\phi(\tau)=\phi(0)\exp\left\{-\int_0^{\tau}(\mu+\sigma(s))ds\right\} \tag{3.15}$$

Let $h(\tau)=\exp\left\{-\int_0^{\tau}(\mu+\sigma(s))ds\right\}$ (3.16)

Hence, equation (3.15) becomes;

$$\phi(\tau)=\phi(0)h(\tau) \tag{3.17}$$

Substituting equation (3.17) into equation (3.10) yields

$$y = \phi(0)\int_0^T h(\tau)d\tau = \phi(0)\bar{h} \tag{3.18}$$

Where $\bar{h} = \int_0^T h(\tau)d\tau$ (3.19)

is a constant.

Putting equation (3.11) into equation (3.18), we obtain;

$$y = \{(1-\theta)\beta y + \alpha x(y+z)\}\bar{h} \tag{3.20}$$

Substituting equation (3.17) into equation (3.14) we have;

$$(\beta - \mu - \delta)z + \phi(0)\int_0^T \sigma(\tau)h(\tau)d\tau = 0 \tag{3.21}$$

Let $\pi = \int_0^T \sigma(\tau)h(\tau)d\tau$ (3.22)

Recall also from equation (3.11) that;

$$\phi(0) = (1-\theta)\beta y + \alpha x(y+z)$$

Hence, equation (3.21) becomes;

$$(\beta - \mu - \delta)z + \pi[(1-\theta)\beta y + \alpha x(y+z)] = 0 \tag{3.23}$$

We now solve equations (3.12), (3.20) and (3.23) simultaneously to obtain the values of x, y, z, which give the equilibrium states of the model.

Observe from equation (3.12), given by;

$$(\beta - \mu)x + \theta\beta y - \alpha x(y+z) = 0$$

that when $x = 0$, we have;

$\theta\beta y = 0$; $\Rightarrow y = 0$, since $\theta\beta \neq 0$.

And from equation (3.20), we observe that when $x = 0$, we have;

$$\left[(1-\theta)\beta\bar{h} - 1\right]y = 0; \tag{3.24}$$

$\Rightarrow y = 0$, since $(1-\theta)\beta\bar{h} - 1 \neq 0$

Similarly, from equation (3.23), when $x = 0$ and $y = 0$, we have;

$$(\beta - \mu - \delta)z = 0 \tag{3.25}$$

$\Rightarrow \quad z = 0$, since $\beta - \mu - \delta_2 \neq 0$

Therefore, we obtain the zero equilibrium state of the model given by; $(x, y, z) = (0,0,0)$.

For the non-zero equilibrium state of the model, we have;
when $x \neq 0$, we obtain from equation (3.12);

$$x = \frac{-\theta\beta y}{\beta - \mu - \alpha(y+z)} \tag{3.26}$$

Substituting equation (3.26) into equation (3.20), we have;

$$y = \left\{(1-\theta)\beta y - \alpha(y+z)\left[\frac{\theta\beta y}{\beta - \mu - \alpha(y+z)}\right]\right\}\bar{h} \tag{3.27}$$

$$\Rightarrow \quad [\beta - \mu - \alpha(y+z)]y = \{[\beta - \mu - \alpha(y+z)](1-\theta)\beta y - \alpha\theta\beta y(y+z)\}\bar{h}$$

$$[\beta - \mu - \alpha(y+z)]y = \{(\beta - \mu)(1-\theta)\beta y - \alpha(y+z)(1-\theta)\beta y - \alpha\theta\beta y(y+z)\}\bar{h}$$

Simplifying further we have;

$$[\beta-\mu-\alpha(y+z)]y=\{(\beta-\mu)(1-\theta)\beta-\alpha\beta(y+z)\}y\bar{h}$$

$$\beta-\mu+\alpha(y+z)(\beta\bar{h}-1)=(\beta-\mu)(1-\theta)\beta\bar{h}$$

$$\Rightarrow\quad y+z=\frac{(\beta-\mu)[(1-\theta)\beta\bar{h}-1]}{\alpha(\beta\bar{h}-1)} \tag{3.28}$$

Putting equation (3.28) into equation (3.26) yields;

$$x=\frac{-\theta\beta y}{\beta-\mu-\dfrac{\alpha(\beta-\mu)[(1-\theta)\beta\bar{h}-1]}{\alpha(\beta\bar{h}-1)}} \tag{3.29}$$

$$x=\frac{-\theta\beta y(\beta\bar{h}-1)}{(\beta-\mu)(\beta\bar{h}-1)-(\beta-\mu)[(1-\theta)\beta\bar{h}-1]}$$

$$=\frac{-\theta\beta y(\beta\bar{h}-1)}{(\beta-\mu)[\beta\bar{h}-1-\beta\bar{h}+\theta\beta\bar{h}+1]}$$

$$\Rightarrow\ x=\frac{y(\beta\bar{h}-1)}{(\beta-\mu)\bar{h}} \tag{3.30}$$

We then substitute equations (3.28) and (3.30) into equation (3.23) to obtain;

$$(\beta-\mu-\delta)z+\pi\left[(1-\theta)\beta y-\alpha\left(\frac{y(\beta\bar{h}-1)}{(\beta-\mu)\bar{h}}\right)\left(\frac{(\beta-\mu)[(1-\theta)\beta\bar{h}-1]}{\alpha(\beta\bar{h}-1)}\right)\right]=0$$

$$(\beta-\mu-\delta)z+\pi\left[(1-\theta)\beta y-\left(\frac{y[(1-\theta)\beta\bar{h}-1]}{\bar{h}}\right)\right]=0$$

$$\Rightarrow \quad z = \frac{-\pi y\left[(1-\theta)\beta\bar{h} - (1-\theta)\beta\bar{h} + 1\right]}{\bar{h}(\beta - \mu - \delta)}$$

$$z = \frac{-\pi y}{\bar{h}(\beta - \mu - \delta)} \tag{3.31}$$

Putting equation (3.31) into equation (3.28), we obtain;

$$y - \frac{\pi y}{\bar{h}(\beta - \mu - \delta)} = \frac{(\beta - \mu)\left[(1-\theta)\beta\bar{h} - 1\right]}{\alpha\left(\beta\bar{h} - 1\right)}$$

$$y\left[(\beta - \mu - \delta)\bar{h} - \pi\right] = \frac{(\beta - \mu - \delta)\bar{h}(\beta - \mu)\left[(1-\theta)\beta\bar{h} - 1\right]}{\alpha\left(\beta\bar{h} - 1\right)}$$

$$\Rightarrow \quad y = \frac{(\beta - \mu - \delta)\bar{h}(\beta - \mu)\left[(1-\theta)\beta\bar{h} - 1\right]}{\alpha\left(\beta\bar{h} - 1\right)\left[(\beta - \mu - \delta)\bar{h} - \pi\right]} \tag{3.32}$$

Again, we substitute equation (2.32) into equation (2.28) to obtain;

$$z = \frac{(\beta - \mu)\left[(1-\theta)\beta\bar{h} - 1\right]}{\alpha\left(\beta\bar{h} - 1\right)} - \frac{(\beta - \mu - \delta)\bar{h}(\beta - \mu)\left[(1-\theta)\beta\bar{h} - 1\right]}{\alpha\left(\beta\bar{h} - 1\right)\left[(\beta - \mu - \delta)\bar{h} - \pi\right]}$$

$$= \frac{(\beta - \mu)\left[(1-\theta)\beta\bar{h} - 1\right]\left[(\beta - \mu - \delta)\bar{h} - \pi\right] - (\beta - \mu)(\beta - \mu - \delta)\bar{h}\left[(1-\theta)\beta\bar{h} - 1\right]}{\alpha\left(\beta\bar{h} - 1\right)\left[(\beta - \mu - \delta)\bar{h} - \pi\right]}$$

$$z = \frac{(\beta - \mu)\left[(1-\theta)\beta\bar{h} - 1\right]\left[(\beta - \mu - \delta)\bar{h} - \pi - (\beta - \mu - \delta)\bar{h}\right]}{\alpha\left(\beta\bar{h} - 1\right)\left[(\beta - \mu - \delta)\bar{h} - \pi\right]}$$

$$\Rightarrow \quad z = \frac{-\pi(\beta - \mu)\left[(1-\theta)\beta\bar{h} - 1\right]}{\alpha\left(\beta\bar{h} - 1\right)\left[(\beta - \mu - \delta)\bar{h} - \pi\right]} \tag{3.33}$$

Similarly, putting equation (3.32) into equation (3.30), we have;

$$x = \frac{(\beta - \mu)(\beta - \mu - \delta)\bar{h}\left(\beta\bar{h} - 1\right)\left[(1-\theta)\beta\bar{h} - 1\right]}{(\beta - \mu)\bar{h}\alpha\left(\beta\bar{h} - 1\right)\left[(\beta - \mu - \delta)\bar{h} - \pi\right]}$$

$$\Rightarrow \quad x = \frac{(\beta - \mu - \delta)\left[(1-\theta)\beta\bar{h} - 1\right]}{\alpha\left[(\beta - \mu - \delta)\bar{h} - \pi\right]} \tag{3.34}$$

Therefore, the values of x, y, z given by equations (3.34), (3.32) and (3.33) give the non-zero equilibrium state of the model.

CHAPTER FOUR

STABILITY ANALYSIS OF EQUILIBRIUM STATES

4.1 INTRODUCTION

This chapter deals with the analysis of the zero and non-zero equilibrium states of our model given by; $(x.y,z)=(0,0,0)$ and (x,y,z) as given by equations (3.34), (3.32) and (3.33) respectively for stability. We shall apply the results of Bellman and Cooke, to analyse the stability or otherwise of the equilibrium states. But before then, we shall perturb the equilibrium states and from the resulting equations obtain the characteristic equation. The characteristic equation is then used to analyse the equilibrium states for stability.

To establish the results of Bellman and Cooke, we hereby present a fundamental theorem to the analysis of the stability of characteristic equations as stated by Jack Hale; Theory of Functional Differential Equations, (1977).

Theorem 4.1

Let $\mathrm{H}(z)=\mathrm{P}\left(z,e^{z}\right)$ where $\mathrm{P}(z,w)$ is a polynomial in with principal term.

Suppose $\mathrm{H}(iy)$, $y\in R$, is separated into its real and imaginary parts,

$$\mathrm{H}(iy)=F(y)+iG(y). \tag{4.1}$$

If zeros of $\mathrm{H}(z)$ have negative real parts, then the zeros of $F(y)$ and $G(y)$ are real, alternate and

$$G'(y)F(y)-G(y)F'(y)>0 \tag{4.2}$$

for $y \in R$. Conversely, all zeros of H(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 the inequality (4.2) is satisfied for at least one y.

(ii) All the zeros of $F(y)$ are real and, for each zero, the Relation (4.2) is satisfied

All the zeros of $G(y)$ are real and, for each zero, the Relation (4.2) is satisfied.

4.2 THE CHARACTERISTIC EQUATION

Let us assume a perturbation of the equilibrium states of the model as follows;

$$S(t) = x + p(t);\ p(t) = \bar{p}e^{\lambda t} \tag{4.3}$$

$$L(t) = y + q(t);\quad q(t) = \bar{q}e^{\lambda t} \tag{4.4}$$

$$\mathrm{I}(t) = z + r(t);\quad r(t) = \bar{r}e^{\lambda t} \tag{4.5}$$

$$\rho(t,\tau) = \phi(\tau) + \eta(\tau)e^{\lambda t} \tag{4.6}$$

$$\Rightarrow\ \bar{q} = \int_0^T \eta(\tau)d\tau \tag{4.7}$$

We then substitute equations (4.3) to (4.7) into the model equations;

From equation (3.1) we obtain;

$$\frac{d}{dt}\left(x+\bar{p}e^{\lambda t}\right)=(\beta-\mu)\left(x+\bar{p}e^{\lambda t}\right)+\theta\beta\left(y+\bar{q}e^{\lambda t}\right)$$

$$-\alpha\left(x+\bar{p}e^{\lambda t}\right)\left(y+\bar{q}e^{\lambda t}+z+\bar{r}e^{\lambda t}\right) \tag{4.8}$$

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

$$-\alpha y\bar{p}e^{\lambda t}-\alpha\bar{p}\bar{q}e^{2\lambda t}-\alpha xz-\alpha x\bar{r}e^{\lambda t}-\alpha z\bar{p}e^{\lambda t}-\alpha\bar{p}\bar{r}e^{2\lambda t} \tag{4.9}$$

Using equation (3.12) and neglecting terms of order two and above in equation (4.9), we have;

$$\lambda\bar{p}e^{\lambda t}=(\beta-\mu)\bar{p}e^{\lambda t}+\theta\beta\bar{q}e^{\lambda t}-\alpha x\bar{q}e^{\lambda t}-\alpha y\bar{p}e^{\lambda t}-\alpha x\bar{r}e^{\lambda t}-\alpha z\bar{p}e^{\lambda t} \tag{4.10}$$

$$\Rightarrow\ \lambda\bar{p}=(\beta-\mu)\bar{p}+\theta\beta\bar{q}-\alpha x\bar{q}-\alpha y\bar{p}-\alpha x\bar{r}-\alpha z\bar{p}$$

$$0=\left[\beta-\mu-\alpha(y+z)-\lambda\right]\bar{p}+(\theta\beta-\alpha x)\bar{q}-\alpha x\bar{r} \tag{4.11}$$

From equation (3.2), we have;

$$\frac{\partial}{\partial t}\left(\phi(\tau)+\eta(\tau)e^{\lambda t}\right)+\frac{\partial}{\partial\tau}\left(\phi(\tau)+\eta(\tau)e^{\lambda t}\right)+(\mu+\sigma(\tau))\left(\phi(\tau)+\eta(\tau)e^{\lambda t}\right)=0 \tag{4.12}$$

$$\lambda\eta(\tau)e^{\lambda t}+\frac{d}{d\tau}\phi(\tau)+e^{\lambda t}\frac{d}{d\tau}\eta(\tau)+(\mu+\sigma(\tau))\phi(\tau)+(\mu+\sigma(\tau))\eta(\tau)e^{\lambda t}=0 \tag{4.13}$$

Using equation (3.13), we obtain;

$$\lambda\eta(\tau)e^{\lambda t}+e^{\lambda t}\frac{d}{d\tau}\eta(\tau)+(\mu+\sigma(\tau))\eta(\tau)e^{\lambda t}=0 \tag{4.14}$$

$$\Rightarrow\ \frac{d}{d\tau}\eta(\tau)+(\mu+\sigma(\tau)+\lambda)\eta(\tau)=0 \tag{4.15}$$

Solving the o.d.e. (4.15), we obtain;

$$\eta(\tau) = \eta(0)\exp\left\{-\int_0^{\tau}(\mu + \sigma(s) + \lambda)ds\right\} \tag{4.16}$$

Integrating equation (4.16) over [0, T], we obtain;

$$\bar{q} = \eta(0)\int_0^T\left[\exp\left\{-\int_0^{\tau}(\mu + \sigma(s) + \lambda)ds\right\}\right]d\tau \tag{4.17}$$

$$\Rightarrow \quad \bar{q} = \eta(0)b(\lambda) \tag{4.18}$$

$$\text{where, } b(\lambda) = \int_0^T\left[\exp\left\{-\int_0^{\tau}(\mu + \sigma(s) + \lambda)ds\right\}\right]d\tau \tag{4.19}$$

To obtain the value of $\eta(0)$, we recall equation (3.11) given by;

$$\phi(0) = \mathrm{B}(0) = (1-\theta)\beta y + \alpha x(y+z)$$

And from equation (4.6), we obtain;

$$\rho(t,0) = \phi(0) + \eta(0)e^{\lambda t} = \mathrm{B}(t) \tag{4.20}$$

Again, we recall equation (3.5) given by;

$$\mathrm{B}(t) = (1-\theta)\beta L(t) + \alpha S(t)[L(t) + I(t)]$$

Substituting equations (4.3), (4.4) and (4.7) into equation (3.5), we have;

$$\mathrm{B}(t) = (1-\theta)\beta\left(y + \bar{q}e^{\lambda t}\right) + \alpha\left(x + \bar{p}e^{\lambda t}\right)\left[y + \bar{q}e^{\lambda t} + z + \bar{r}e^{\lambda t}\right] \tag{4.21}$$

$$\begin{aligned}\mathrm{B}(t) = {} & (1-\theta)\beta y + (1-\theta)\beta\bar{q}e^{\lambda t} + \alpha x(y+z) + \alpha x\bar{q}e^{\lambda t} + \alpha y\bar{p}e^{\lambda t} \\ & + \alpha\bar{\bar{p}}\bar{q}e^{2\lambda t} + \alpha x\bar{r}e^{\lambda t} + \alpha z\bar{p}e^{\lambda t} + \alpha\bar{\bar{p}}\bar{r}e^{2\lambda t}\end{aligned} \tag{4.22}$$

Comparing equations (4.22) and (4.20) using equation (3.11) and neglecting terms of order two and above in equation (4.22), we have;

$$(1-\theta)\beta y + \alpha x(y+z) + \eta(0)e^{\lambda t} = (1-\theta)\beta y + (1-\theta)\beta \bar{q} e^{\lambda t}$$

$$+ \alpha x(y+z) + \alpha x \bar{q} e^{\lambda t} + \alpha y \bar{p} e^{\lambda t} + \alpha x \bar{r} e^{\lambda t} + \alpha z \bar{p} e^{\lambda t}$$

$$\Rightarrow \quad \eta(0) = [(1-\theta)\beta + \alpha x]\bar{q} + \alpha(y+z)\bar{p} + \alpha x \bar{r} \tag{4.23}$$

Substituting equation (4.23) into (4.18), we obtain;

$$\bar{q} = \{\alpha(y+z)\bar{p} + [(1-\theta)\beta + \alpha x]\bar{q} + \alpha x \bar{r}\}b(\lambda) \tag{4.24}$$

$$\Rightarrow \quad 0 = \alpha(y+z)b(\lambda)\bar{p} + \{[(1-\theta)\beta + \alpha x]b(\lambda) - 1\}\bar{q} + \alpha x b(\lambda)\bar{r} \tag{4.25}$$

Similarly, from equation (3.3), we have

$$\frac{d}{dt}\left(z + \bar{r}e^{\lambda t}\right) = (\beta - \mu - \delta)\left(z + \bar{r}e^{\lambda t}\right) + \int_0^T \sigma(\tau)\left(\phi(\tau) + \eta(\tau)e^{\lambda t}\right)d\tau \tag{4.26}$$

$$\lambda \bar{r} e^{\lambda t} = (\beta - \mu - \delta)z + (\beta - \mu - \delta)\bar{r}e^{\lambda t} + \int_0^T \sigma(\tau)\phi(\tau)d\tau + e^{\lambda t}\int_0^T \sigma(\tau)\eta(\tau)d\tau \tag{4.27}$$

Using equation (3.14), we obtain;

$$\lambda \bar{r} e^{\lambda t} = (\beta - \mu - \delta)\bar{r}e^{\lambda t} + e^{\lambda t}\int_0^T \sigma(\tau)\eta(\tau)d\tau \tag{4.28}$$

$$\Rightarrow \quad \lambda \bar{r} = (\beta - \mu - \delta)\bar{r} + \int_0^T \sigma(\tau)\eta(\tau)d\tau \tag{4.29}$$

Substituting equation (4.16) into equation (4.29) using equation (4.23), we have;

$$\lambda\bar{r} = (\beta - \mu - \delta)\bar{r} + \int_0^T \sigma(\tau)\left[\alpha(y+z)\bar{p} + [(1-\theta)\beta + \alpha x]\bar{q}\right.$$

$$\left. + \alpha x\bar{r}\right]\exp\left[-\int_0^\tau(\mu + \sigma(s) + \lambda)ds\right]d\tau \qquad (4.30)$$

$$\lambda\bar{r} = (\beta - \mu - \delta)\bar{r} + \left[\alpha(y+z)\bar{p} + [(1-\theta)\beta + \alpha x]\bar{q}\right.$$

$$\left. + \alpha x\bar{r}\right]\int_0^T \sigma(\tau)\exp\left[-\int_0^\tau(\mu + \sigma(s) + \lambda)ds\right]d\tau$$

Let $C(\lambda) = \int_0^T \sigma(\tau)\exp\left[-\int_0^\tau(\mu + \sigma(s) + \lambda)ds\right]d\tau \qquad (4.31)$

$$\Rightarrow \lambda\bar{r} = (\beta - \mu - \delta)\bar{r} + C(\lambda)\left[\alpha(y+z) + \bar{p}[(1-\theta)\beta + \alpha x]\bar{q} + \alpha x\bar{r}\right]$$

$$0 = \alpha(y+z)C(\lambda)\bar{p} + [(1-\theta)\beta + \alpha x]C(\lambda)\bar{q} + (\beta + \alpha x C(\lambda) - \mu - \delta - \lambda)\bar{r} \qquad (4.32)$$

Since $\begin{bmatrix}\bar{p}\\ \bar{q}\\ \bar{r}\end{bmatrix} \neq 0$, we obtain the Jacobian determinant using equations (4.11), (4.25) and (4.32) as given below;

$$\begin{vmatrix} \beta - \mu - \alpha(y+z) - \lambda & \theta\beta - \alpha x & -\alpha x \\ \alpha(y+z)b(\lambda) & [(1-\theta)\beta + \alpha x]b(\lambda) - 1 & \alpha x b(\lambda) \\ \alpha(y+z)C(\lambda) & [(1-\theta)\beta + \alpha x]C(\lambda) & (\beta + \alpha x C(\lambda) - \mu - \delta - \lambda) \end{vmatrix} = 0 \qquad (4.33)$$

Hence, the characteristic equation is given by;

$$(\beta-\mu-\alpha(y+z)-\lambda)\{[((1-\theta)\beta+\alpha x)b(\lambda)-1](\beta+\alpha xC(\lambda)-\mu-\delta-\lambda)$$

$$-\alpha xb(\lambda)[(1-\theta)\beta+\alpha x]C(\lambda)\}-\alpha(y+z)b(\lambda)\{(\theta\beta-\alpha x)(\beta+\alpha xC(\lambda)-\mu-\delta-\lambda)$$

$$+\alpha x[(1-\theta)\beta+\alpha x]C(\lambda)\}+\alpha(y+z)C(\lambda)\{\alpha xb(\lambda)(\theta\beta-\alpha x)$$

$$+\alpha x([(1-\theta)\beta+\alpha x]b(\lambda)-1)\}=0 \qquad (4.34)$$

4.3 STABILITY OF THE ZERO EQUILIBRIUM STATE

In this section, the zero equilibrium state (i.e. the origin) of the model is analysed for stability using the characteristics equation.

At the zero equilibrium state $(x.y,z)=(0,0,0)$, the characteristic equation (4.34) takes the form;

$$(\beta-\mu-\lambda)(\beta-\mu-\delta-\lambda)[(1-\theta)\beta b(\lambda)-1]=0 \qquad (4.35)$$

Therefore, either;

$$(\beta-\mu-\lambda)(\beta-\mu-\delta-\lambda)=0 \qquad (4.36)$$

or

$$((1-\theta)\beta b(\lambda)-1)=0 \qquad (4.37)$$

Observe that equation (4.36) is a quadratic equation in λ, and so we have;

$$\lambda^2-(\beta-\mu)\lambda+\delta\lambda-(\beta-\mu)\lambda-(\beta-\mu)\delta+(\beta-\mu)^2=0$$

$$\Rightarrow \lambda^2-[2(\beta-\mu)-\delta]\lambda-(\beta-\mu)[\delta-(\beta-\mu)]=0 \qquad (4.38)$$

Solving the quadratic equation (4.38), we have;

$$\lambda = \frac{2(\beta-\mu)-\delta \pm \sqrt{(2(\beta-\mu)-\delta)^2 + 4(\beta-\mu)[\delta-(\beta-\mu)]}}{2}$$

$$\lambda = \frac{2(\beta-\mu)-\delta \pm \sqrt{4(\beta-\mu)^2 - 4\delta(\beta-\mu)+\delta^2+4\delta(\beta-\mu)-4(\beta-\mu)^2}}{2}$$

$$\Rightarrow \quad \lambda = \frac{2(\beta-\mu)-\delta \pm \sqrt{\delta^2}}{2}$$

$$= \frac{2(\beta-\mu)-\delta \pm \delta}{2}$$

Therefore, $\lambda_1 = \frac{2(\beta-\mu)}{2} = \beta-\mu$ (4.39)

and $\lambda_2 = \frac{2(\beta-\mu)-2\delta}{2} = \beta-\mu-\delta$ (4.40)

It can be seen clearly from equations (4.39) and (4.40) that λ_1, λ_2 are negative if and only if $\beta < \mu$. Therefore, the origin will be stable when $\beta < \mu$, and solutions of (4.37) have negative real parts.

Considering equation (4.37), we recall from equation (4.19) that;

$$b(\lambda) = \int_0^T \left[\exp\left\{ -\int_0^\tau (\mu+\sigma(s)+\lambda)ds \right\} \right] d\tau$$

From the inner integral, we have;

$$\int_0^\tau (\mu+\sigma(s)+\lambda)ds = \lambda\tau + \int_0^\tau (\mu+\sigma(s))ds \tag{4.41}$$

$$\Rightarrow \quad b(\lambda) = \int_0^T \left[\exp(-\lambda\tau) \exp\left\{ - \int_0^\tau (\mu + \sigma(s)) ds \right\} \right] d\tau$$

Again, from equation (3.16), we have;

$$b(\lambda) = \int_0^T \exp(-\lambda\tau) h(\tau) d\tau \tag{4.42}$$

We shall then use the result of Bellman and Cooke as applied by Akinwande N.I (2005), to analyse the zero equilibrium state for stability or otherwise, using equation (4.37).

Let the equation (4.37) take the form;

$$\mathrm{H}_1(\lambda) = (1-\theta)\beta b(\lambda) - 1 = 0 \equiv P\left(\lambda, e^{\lambda}\right) \tag{4.43}$$

where $b(\lambda) = \int_0^T \exp(-\lambda\tau) h(\tau) d\tau$

If we set $\lambda = i\omega$ in equation (4.43), we have;

$$\mathrm{H}_1(i\omega) = F_1(\omega) + iG_1(\omega) \tag{4.44}$$

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

$$F_1(0)G_1'(0) - F_1'(0)G_1(0) > 0 \tag{4.45}$$

From equation (4.43), we have;

$$b(i\omega) = \int_0^T \exp(-i\omega\tau) h(\tau) d\tau \tag{4.46}$$

$$= \int_0^T (\cos\omega\tau - i\sin\omega\tau) h(\tau) d\tau$$

$$b(i\omega) = \int_0^T h(\tau)\cos\omega\tau d\tau - i\int_0^T h(\tau)\sin\omega\tau d\tau$$

$$\Rightarrow \quad b(i\omega) = b_1(\omega) + i\, b_2(\omega) \tag{4.47}$$

So that,

$$b_1(\omega) = \int_0^T h(\tau)\cos\omega\tau d\tau \tag{4.48}$$

$$b_2(\omega) = -\int_0^T h(\tau)\sin\omega\tau d\tau \tag{4.49}$$

and, $$b_1(0) = \int_0^T h(\tau)d\tau = \bar{h} \tag{4.50}$$

$$b_2(0) = 0 \tag{4.51}$$

Also, $$b_1'(\omega) = -\int_0^T \tau h(\tau)\sin\omega\tau d\tau \tag{4.52}$$

$$\Rightarrow \quad b_1'(0) = 0 \tag{4.53}$$

and $$b_2'(\omega) = -\int_0^T \tau h(\tau)\cos\omega\tau d\tau \tag{4.54}$$

$$\Rightarrow \quad b_2'(0) = -\int_0^T \tau h(\tau)d\tau = -\psi \tag{4.55}$$

From equation (4.43), we have;

$$\mathrm{H}_1(i\omega) = (1-\theta)\beta b(i\omega) - 1 \tag{4.56}$$

$$= (1-\theta)\beta b_1(\omega) - 1 + i(1-\theta)\beta b_2(\omega) \tag{4.57}$$

Comparing equations (4.57) and (4.44), we obtain;

$$F_1(\omega) = (1-\theta)\beta b_2(\omega) - 1 \tag{4.58}$$

$$G_1(\omega) = (1-\theta)\beta b_2(\omega) \tag{4.59}$$

Therefore,

$$F_1(0) = (1-\theta)\beta b_1(0) - 1$$

$$\Rightarrow \quad F_1(0) = (1-\theta)\beta\bar{h} - 1 \tag{4.60}$$

$$G_1(0) = (1-\theta)\beta b_2(0) = 0 \tag{4.61}$$

since $b_1(0) = \bar{h}$ and $b_2(0) = 0$

Similarly,

$$F_1'(\omega) = (1-\theta)b_1{}'\beta(\omega) \tag{4.62}$$

$$G_1'(\omega) = (1-\theta)\beta b_2{}'(\omega) \tag{4.63}$$

so that,

$$F_1'(0) = (1-\theta)\beta b_1{}'(0) = 0 \tag{4.64}$$

$$G_1'(0) = (1-\theta)\beta b_{2'}{}'(0)$$

$$\Rightarrow \quad G_1'(0) = -(1-\theta)\beta\psi \tag{4.65}$$

since $b_1{}'(0) = 0$, and $b_2{}'(0) = -\psi$

Hence, the inequality (4.45) yields;

$$\left[(1-\theta)\beta\bar{h} - 1\right]\left[(1-\theta)\beta\psi\right] < 0 \tag{4.66}$$

Now since, $(1-\theta)\beta\psi > 0$

the inequality (4.66) holds if;

$$(1-\theta)\beta\bar{h}-1<0 \tag{4.67}$$

Recall from equations (3.16) and (3.19) that;

$$\bar{h}=\int_0^T \exp\left\{-\int_0^\tau(\mu+\sigma(s))ds\right\}d\tau \tag{4.68}$$

and from equation (3.4); $\sigma(s)=\tan\left(\frac{\pi s}{2kT}\right)$

$$\Rightarrow\ \bar{h}=\int_0^T \exp\left\{-\int_0^\tau\left(\mu+\tan\left(\frac{\pi s}{2kT}\right)\right)ds\right\}d\tau \tag{4.69}$$

Solving the inner integral, we have;

$$\int_0^\tau\left(\mu+\tan\left(\frac{\pi s}{2kT}\right)\right)ds=\mu\tau+\int_0^\tau\tan\left(\frac{\pi s}{2kT}\right)ds$$

$$=\mu\tau-\left[\frac{2kT}{\pi}\log\cos\left(\frac{\pi s}{2kT}\right)\right]_0^\tau$$

$$=\mu\tau-\frac{2kT}{\pi}\log\cos\left(\frac{\pi\tau}{2kT}\right)$$

Therefore,

$$\bar{h}=\int_0^T \exp\left\{-\left[\mu\tau-\frac{2kT}{\pi}\log\cos\left(\frac{\pi\tau}{2kT}\right)\right]\right\}d\tau \tag{4.70}$$

Carrying out the integrating, yields;

$$\bar{h}=-\left[\left[\mu+\tan\left(\frac{\pi\tau}{2kT}\right)\right]^{-1}\exp\left[-\left(\mu\tau-\frac{2kT}{\pi}\log\cos\left(\frac{\pi\tau}{2kT}\right)\right)\right]\right]_0^T$$

$$\bar{h} = -\left[\mu + \tan\left(\frac{\pi}{2k}\right)\right]^{-1} \exp\left(-\mu T + \frac{2kT}{\pi}\log\cos\left(\frac{\pi\tau}{2kT}\right)\right) + \left[\mu\right]^{-1} \tag{4.71}$$

Substituting for $\bar{h}$ in equation (4.67), we define;

$$J_1(k) = (1-\theta)\beta\left\{-\left[\mu + \tan\left(\frac{\pi}{2k}\right)\right]^{-1} \exp\left(-\mu T + \frac{2kT}{\pi}\log\cos\left(\frac{\pi\tau}{2kT}\right)\right) + \left[\mu\right]^{-1}\right\} - 1 \tag{4.72}$$

We shall then use MathLab to carry out numerical computation of $J_1(k)$ to establish the conditions for stability of the zero equilibrium. The result shows that this equilibrium state will be stable when $\beta < \mu$ and $J_1(k) < 0$.

4.4 STABILITY OF THE NON-ZERO EQUILIBRIUM STATE

For the non-zero equilibrium state (x, y, z) as given by equations (3.34), (3.32) and (3.33), we shall consider the characteristic equation (4.33) in the form;

$$\mathrm{H}_2(\lambda) = 0 \tag{4.73}$$

Hence, from equation (4.33) we have;

$$\begin{aligned} H_2(\lambda) = {} & (\beta - \mu - \alpha(y+z) - \lambda)\{[((1-\theta)\beta + \alpha x)b(\lambda) - 1](\beta + \alpha x C(\lambda) \\ & - \mu - \delta_2 - \lambda) - \alpha x b(\lambda)[(1-\theta)\beta + \alpha x]C(\lambda)\} - \alpha(y+z)b(\lambda)\{(\theta\beta - \alpha x)(\beta + \alpha x C(\lambda) \\ & - \mu - \delta_2 - \lambda) + \alpha x[(1-\theta)\beta + \alpha x]C(\lambda)\} + \alpha(y+z)C(\lambda)\{\alpha x b(\lambda)(\theta\beta - \alpha x) \\ & + \alpha x([(1-\theta)\beta + \alpha x]b(\lambda) - 1)\} \end{aligned} \tag{4.74}$$

We then apply the result of Bellman and Cooke to analyse the stability of the non-zero equilibrium state using equation (4.74).

If we set $\lambda = iw$ in equation (4.74), we have;

$$H_2(iw) = F_2(w) + iG_2(w) \tag{4.75}$$

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

$$F_2(0)G_2'(0) + F_2'(0)G_2(0) > 0 \tag{4.76}$$

Recall from equation (4.46) to (4.55) that;

$b(iw) = b_1(w) + ib_2(w)$ and ,

$b_1(0) = \bar{h}$, and $b_2(0) = 0$

$b_1'(0) = 0$, and $b_2'(0) = -\psi$

Similarly, from equation (4.31), we have that;

$$C(\lambda) = \int_0^T \sigma(\tau)\exp\left[-\int_0^\tau (\mu + \sigma(s) + \lambda)ds\right]d\tau$$

$$C(\lambda) = \int_0^T \sigma(\tau)\exp(-\lambda\tau)\exp\left[-\int_0^\tau (\mu + \sigma(s))ds\right]d\tau \tag{4.77}$$

Recall from equation (3.16) that;

$$h(\tau) = \exp\left\{-\int_0^\tau (\mu + \sigma(s))ds\right\}$$

$$\Rightarrow \quad C(\lambda) = \int_0^T \sigma(\tau)h(\tau)\exp(-\lambda\tau)d\tau \tag{4.78}$$

Thus, $C(iw) = \int_0^T \sigma(\tau)h(\tau)\exp(-iw\tau)d\tau$

$$= \int_0^T \sigma(\tau)h(\tau)(\cos w\tau - i\sin w\tau)d\tau$$

$$C(iw) = \int_0^T \sigma(\tau)h(\tau)\cos w\tau d\tau - i\int_0^T \sigma(\tau)h(\tau)\sin w\tau d\tau \quad (4.79)$$

$$\Rightarrow \quad C(iw) = C_1(w) + iC_2(w) \quad (4.80)$$

So that,

$$C_1(w) = \int_0^T \sigma(\tau)h(\tau)\cos w\tau d\tau \quad (4.81)$$

$$C_2(w) = -\int_0^T \sigma(\tau)h(\tau)\sin w\tau d\tau \quad (4.82)$$

And $C_1(0) = \int_0^T \sigma(\tau)h(\tau)d\tau = \Re \quad (4.83)$

$$C_2(0) = 0 \quad (4.84)$$

Also, $C_1'(w) = -\int_0^T \tau\sigma(\tau)h(\tau)\sin w\tau d\tau \quad (4.85)$

$$\Rightarrow C_1'(0) = 0 \quad (4.86)$$

$$C_2'(w) = -\int_0^T \tau\sigma(\tau)h(\tau)\cos w\tau d\tau \quad (4.87)$$

$$C_2'(0) = -\int_0^T \tau\sigma(\tau)h(\tau)d\tau = -A \quad (4.88)$$

Now equation (4.74) becomes;

$$H_2(iw) = (\beta - \mu - \alpha(y+z) - iw)([(1-\theta)\beta + \alpha x]b(iw) - 1)(\beta + \alpha xC(iw)$$

$$-\mu-\delta_2-iw)-\alpha x b(iw)(\beta-\mu-iw)[(1-\theta)\beta+\alpha x]C(iw)$$

$$-\alpha(y+z)b(iw)(\theta\beta-\alpha x)(\beta+\alpha x C(iw)-\mu-\delta_2-iw)$$

$$+\alpha^2 xC(iw)(y+z)(\theta\beta-\alpha x)+\alpha^2 xC(iw)(y+z)([(1-\theta)\beta-\alpha x]b(iw)-1) \quad (4.89)$$

$$H_2(iw)=(\beta-\mu-\alpha(y+z)-iw)([(1-\theta)\beta+\alpha x]b_1(w)-1+i[(1-\theta)\beta+\alpha x]b_2(w))$$

$$(\beta+\alpha x C_1(w)+i\alpha x C_2(w)-\mu-\delta_2-iw)-(\alpha x b_1(w)+i\alpha x b_2(w))(\beta-\mu-iw)$$

$$([(1-\theta)\beta+\alpha x]C_1(w)+i[(1-\theta)\beta+\alpha x]C_2(w))-(\alpha(y+z)b_1(w)$$

$$+i\alpha(y+z)b_2(w))(\theta\beta-\alpha x)(\beta+\alpha x C_1(w)+i\alpha x C_2(w)-\mu-\delta_2-iw)$$

$$+(\alpha^2 xC_1(w)(y+z)+i\alpha^2 xC_2(w)(y+z))(\theta\beta-\alpha x)+(\alpha^2 xC_1(w)(y+z)$$

$$+i\alpha^2 xC_2(w)(y+z))([(1-\theta)\beta+\alpha x]b_1(w)-1+i[(1-\theta)\beta+\alpha x]b_2(w))$$

Expanding further, we obtain;

$$H_2(iw)=\{(\beta-\mu-\alpha(y+z))([(1-\theta)\beta+\alpha x]b_1(w)-1)$$

$$+w[(1-\theta)\beta+\alpha x]b_2(w)\}(\beta+\alpha x C_1(w)-\mu-\delta_2)$$

$$-\{(\beta-\mu-\alpha(y+z))[(1-\theta)\beta+\alpha x]b_2(w)-w([(1-\theta)\beta+\alpha x]b_1(w)-1)\}(\alpha x C_2(w)-w)$$

$$+i\{(\beta-\mu-\alpha(y+z))[(1-\theta)\beta+\alpha x]b_2(w)$$

$$-w([(1-\theta)\beta+\alpha x]b_1(w)-1)\}(\beta+\alpha x C_1(w)-\mu-\delta_2)$$

$$+i\{(\beta-\mu-\alpha(y+z))([(1-\theta)\beta+\alpha x]b_1(w)-1)+w[(1-\theta)\beta+\alpha x]b_2(w)\}(\alpha x C_2(w)-w)$$

$$-(\alpha x(\beta-\mu)b_1(w)+w\alpha x b_2(w))[(1-\theta)\beta+\alpha x]C_1(w)+(\alpha x(\beta-\mu)b_2(w)$$

$$-w\alpha x b_1(w))[(1-\theta)\beta+\alpha x]C_2(w)-i\{(\alpha x(\beta-\mu)b_1(w)+w\alpha x b_2(w))$$

$$[(1-\theta)\beta+\alpha x]C_2(w)+(\alpha x(\beta-\mu)b_2(w)-w\alpha x b_1(w))[(1-\theta)\beta+\alpha x]C_1(w)\}$$

$$-\alpha b_1(w)(y+z)(\theta\beta-\alpha x)(\beta+\alpha x C_1(w)-\mu-\delta_2)+\alpha b_2(w)(y+z)(\theta\beta-\alpha x)(\alpha x C_2(w)-w)$$

$$-i\{\alpha b_1(w)(y+z)(\theta\beta-\alpha x)(\alpha x C_2(w)-w)+\alpha b_2(w)(y+z)(\theta\beta-\alpha x)(\beta+\alpha x C_1(w)-\mu-\delta_2)\}$$

$$+\alpha^2 x(y+z)(\theta\beta-\alpha x)C_1(w)+\alpha^2 x(y+z)C_1(w)([(1-\theta)\beta+\alpha x]b_1(w)-1)$$

$$-\alpha^2 x(y+z)C_2(w)[(1-\theta)\beta+\alpha x]b_2(w)$$

$$+i\{\alpha^2 x(y+z)(\theta\beta-\alpha x)C_2(w)+\alpha^2 x(y+z)C_1(w)[(1-\theta)\beta+\alpha x]b_2(w)$$

$$+\alpha^2 x(y+z)C_2(w)([(1-\theta)\beta+\alpha x]b_1(w)-1)\}\qquad(4.90)$$

Comparing equations (4.90) and (4.75), we have;

$$F_2(w)=\{(\beta-\mu-\alpha(y+z))([(1-\theta)\beta+\alpha x]b_1(w)-1)$$

$$+w[(1-\theta)\beta+\alpha x]b_2(w)\}(\beta+\alpha x C_1(w)-\mu-\delta_2)$$

$$-\{(\beta-\mu-\alpha(y+z))[(1-\theta)\beta+\alpha x]b_2(w)-w([(1-\theta)\beta+\alpha x]b_1(w)-1)\}(\alpha x C_2(w)-w)$$

$$-(\alpha x(\beta-\mu)b_1(w)+w\alpha x b_2(w))[(1-\theta)\beta+\alpha x]C_1(w)+(\alpha x(\beta-\mu)b_2(w)$$

$$-w\alpha x b_1(w))[(1-\theta)\beta+\alpha x]C_2(w)-\alpha b_1(w)(y+z)(\theta\beta-\alpha x)(\beta+\alpha x C_1(w)-\mu-\delta_2)$$

$$+\alpha b_2(w)(y+z)(\theta\beta-\alpha x)(\alpha x C_2(w)-w)+\alpha^2 x(y+z)(\theta\beta-\alpha x)C_1(w)$$

$$+\alpha^2 x(y+z)C_1(w)([(1-\theta)\beta+\alpha x]b_1(w)-1)$$

$$-\alpha^2 x(y+z)C_2(w)[(1-\theta)\beta+\alpha x]b_2(w)\qquad(4.91)$$

$$G_2(w)=\{(\beta-\mu-\alpha(y+z))[(1-\theta)\beta+\alpha x]b_2(w)$$

$$-w([(1-\theta)\beta+\alpha x]b_1(w)-1)\}(\beta+\alpha xC_1(w)-\mu-\delta_2)$$

$$+\{(\beta-\mu-\alpha(y+z))([(1-\theta)\beta+\alpha x]b_1(w)-1)+w[(1-\theta)\beta+\alpha x]b_2(w)\}(\alpha xC_2(w)-w)$$

$$-(\alpha x(\beta-\mu)b_1(w)+w\alpha xb_2(w))((1-\theta)\beta+\alpha x)C_2(w)-(\alpha x(\beta-\mu)b_2(w)$$

$$-w\alpha xb_1(w)[(1-\theta)\beta+\alpha x]C_1(w)-\alpha b_1(w)(y+z)(\theta\beta-\alpha x)(\alpha xC_2(w)-w)$$

$$-\alpha b_2(w)(y+z)(\theta\beta-\alpha x)(\beta+\alpha xC_1(w)-\mu-\delta_2)+\alpha^2x(y+z)(\theta\beta-\alpha x)C_2(w)$$

$$+\alpha^2x(y+z)C_1(w)[(1-\theta)\beta+\alpha x]b_2(w)$$

$$+\alpha^2x(y+z)C_2(w)([(1-\theta)\beta+\alpha x]b_1(w)-1) \qquad (4.92)$$

Therefore,

$$F_2(0)=(\beta-\mu-\alpha(y+z))([(1-\theta)\beta+\alpha x]b_1(0)-1)(\beta+\alpha xC_1(0)-\mu-\delta_2)$$

$$-(\beta-\mu-\alpha(y+z))[(1-\theta)\beta+\alpha x]b_2(0)\alpha xC_2(0)-\alpha x(\beta-\mu)b_1(0)[(1-\theta)\beta+\alpha x]C_1(0)$$

$$+\alpha x(\beta-\mu)b_2(0)[(1-\theta)\beta+\alpha x]C_2(0)-\alpha b_1(0)(y+z)(\theta\beta-\alpha x)(\beta+\alpha xC_1(0)-\mu-\delta_2)$$

$$+\alpha b_2(0)(y+z)(\theta\beta-\alpha x)\alpha xC_2(0)+\alpha^2x(y+z)(\theta\beta-\alpha x)C_1(0)$$

$$+\alpha^2x(y+z)C_1(0)([(1-\theta)\beta+\alpha x]b_1(0)-1)-\alpha^2x(y+z)(\theta\beta-\alpha x)C_2(0)[(1-\theta)\beta+\alpha x]b_2(0)$$

$$F_2(0)=(\beta-\mu-\alpha(y+z))([(1-\theta)\beta+\alpha x]\bar{h}-1)(\beta+\alpha\Re x-\mu-\delta_2)$$

$$-\alpha\bar{h}x(\beta-\mu)[(1-\theta)\beta+\alpha x]\Re-\alpha\bar{h}(y+z)(\theta\beta-\alpha x)(\beta+\alpha\Re x-\mu-\delta_2)$$

$$+\alpha^2\Re x(y+z)(\theta\beta-\alpha x)+\alpha^2\Re x(y+z)([(1-\theta)\beta+\alpha x]\bar{h}-1) \qquad (4.93)$$

$$G_2(0)=\{(\beta-\mu-\alpha(y+z))[(1-\theta)\beta+\alpha x]b_2(0)(\beta+\alpha xC_1(0)-\mu-\delta_2)$$

$$+(\beta-\mu-\alpha(y+z))([(1-\theta)\beta+\alpha x]b_1(0)-1)\alpha xC_2(0)$$

$$-\alpha x(\beta-\mu)b_1(0)((1-\theta)\beta+\alpha x)C_2(0)-\alpha x(\beta-\mu)b_2(0)[(1-\theta)\beta+\alpha x]C_1(0)$$

$$-\alpha b_1(0)(y+z)(\theta\beta-\alpha x)\alpha xC_2(0)-\alpha b_2(0)(y+z)(\theta\beta-\alpha x)(\beta+\alpha xC_1(0)-\mu-\delta_2)$$

$$+\alpha^2x(y+z)(\theta\beta-\alpha x)C_2(0)+\alpha^2x(y+z)C_1(0)[(1-\theta)\beta+\alpha x]b_2(0)$$

$$+\alpha^2x(y+z)C_2(0)([(1-\theta)\beta+\alpha x]b_1(0)-1)$$

$$\Rightarrow \quad G_2(0)=0 \tag{4.94}$$

Since $b_1(0)=\bar{h}$ and $b_2(0)=0$, $C_1(0)=\pi$ and $C_2(0)=0$

$$F_2'(w)=\{(\beta-\mu-\alpha(y+z))([(1-\theta)\beta+\alpha x]b_1'(w))+[(1-\theta)\beta+\alpha x]b_2(w)$$

$$+w[(1-\theta)\beta+\alpha x]b_2'(w)\}(\beta+\alpha xC_1(w)-\mu-\delta_2)$$

$$+\{(\beta-\mu-\alpha(y+z))([(1-\theta)\beta+\alpha x]b_1(w)-1)+w[(1-\theta)\beta+\alpha x]b_2(w)\}\alpha xC_1'(w)$$

$$-\{(\beta-\mu-\alpha(y+z))[(1-\theta)\beta+\alpha x]b_2'(w)-([(1-\theta)\beta+\alpha x]b_1(w)-1)$$

$$-w[(1-\theta)\beta+\alpha x]b_1'(w)\}(\alpha xC_2(w)-w)-\{(\beta-\mu-\alpha(y+z))[(1-\theta)\beta+\alpha x]b_2(w)$$

$$-w([(1-\theta)\beta+\alpha x]b_1(w)-1)\}(\alpha xC_2'(w)-1)-\{\alpha x(\beta-\mu)b_1'(w)+\alpha xb_2(w)$$

$$+w\alpha xb_2'(w)\}[(1-\theta)\beta+\alpha x]C_1(w)-(\alpha x(\beta-\mu)b_1(w)+w\alpha xb_2(w))[(1-\theta)\beta+\alpha x]C_1'(w)$$

$$+\{\alpha x(\beta-\mu)b_2'(w)-\alpha xb_1(w)-w\alpha xb_1'(w)\}[(1-\theta)\beta+\alpha x]C_2(w)+(\alpha x(\beta-\mu)b_2(w)$$

$$-w\alpha xb_1(w))[(1-\theta)\beta+\alpha x]C_2'(w)-\alpha b_1'(w)(y+z)(\theta\beta-\alpha x)(\beta+\alpha xC_1(w)-\mu-\delta_2)$$

$$-\alpha b_1(w)(y+z)(\theta\beta-\alpha x)\alpha x C_1'(w)+\alpha b_2'(w)(y+z)(\theta\beta-\alpha x)(\alpha x C_2(w)-w)$$

$$+\alpha b_2(w)(y+z)(\theta\beta-\alpha x)\left(\alpha x C_2'(w)-1\right)+\alpha^2 x(y+z)(\theta\beta-\alpha x)C_1'(w)$$

$$+\alpha^2 x(y+z)C_1'(w)([(1-\theta)\beta+\alpha x]b_1(w)-1)+\alpha^2 x(y+z)C_1(w)\left([(1-\theta)\beta+\alpha x]b_1'(w)\right)$$

$$-\alpha^2 x(y+z)C_2'(w)((1-\theta)\beta+\alpha x)b_2(w)-\alpha^2 x(y+z)C_2(w)((1-\theta)\beta+\alpha x)b_2'(w) \quad (4.95)$$

$$F_2'(0)=\{(\beta-\mu-\alpha(y+z))([(1-\theta)\beta+\alpha x]b_1'(0))+[(1-\theta)\beta+\alpha x]b_2(0)\}(\beta+\alpha x C_1(0)-\mu-\delta_2)$$

$$+(\beta-\mu-\alpha(y+z))([(1-\theta)\beta+\alpha x]b_1(w)-1)\alpha x C_1'(0)$$

$$-\left\{(\beta-\mu-\alpha(y+z))[(1-\theta)\beta+\alpha x]b_2'(0)-([(1-\theta)\beta+\alpha x]b_1(0)-1)\right\}\alpha x C_2(0)$$

$$-(\beta-\mu-\alpha(y+z))[(1-\theta)\beta+\alpha x]b_2(0)\left(\alpha x C_2'(0)-1\right)-\left(\alpha x(\beta-\mu)b_1'(0)\right.$$

$$\left.+\alpha x b_2(0)\right)[(1-\theta)\beta+\alpha x]C_1(0)-\alpha x(\beta-\mu)b_1(0)[(1-\theta)\beta+\alpha x]C_1'(0)$$

$$+\left(\alpha x b_2'(0)(\beta-\mu)-\alpha x b_1(w)\right)[(1-\theta)\beta+\alpha x]C_2(0)+\alpha x b_2(0)(\beta-\mu)((1-\theta)\beta+\alpha x)C_2'(0)$$

$$-\alpha b_1'(0)(y+z)(\theta\beta-\alpha x)\left(\beta+\alpha x C_1(0)-\mu-\delta_2\right)-\alpha b_1(0)(y+z)(\theta\beta-\alpha x)\alpha x C_1'(0)$$

$$+\alpha b_2'(0)(y+z)(\theta\beta-\alpha x)\alpha x C_2(0)+\alpha b_2(0)(y+z)(\theta\beta-\alpha x)\left(\alpha x C_2'(0)-1\right)$$

$$+\alpha^2 x(y+z)(\theta\beta-\alpha x)C_1'(0)+\alpha^2 x(y+z)C_1'(0)([(1-\theta)\beta+\alpha x]b_1(0)-1)$$

$$+\alpha^2 x(y+z)C_1(0)((1-\theta)\beta+\alpha x)b_1'(0)-\alpha^2 x(y+z)C_2'(0)((1-\theta)\beta+\alpha x)b_2(0)$$

$$-\alpha^2 x(y+z)C_2(0)((1-\theta)\beta+\alpha x)b_2'(0)$$

$$\Rightarrow \quad F_2'(0)=0 \tag{4.96}$$

Since $b_2(0)=0$, $b_1'(0)=0$, $C_2(0)=0$, **and** $C_1'(0)=0$

$$\begin{aligned}
G_2'(w) &= \{(\beta-\mu-\alpha(y+z))[(1-\theta)\beta+\alpha x]b_2'(w)-([(1-\theta)\beta+\alpha x]b_1(w)-1)\\
&-w([(1-\theta)\beta+\alpha x]b_1'(w))(\beta+\alpha xC_1(w)-\mu-\delta_2)\\
&+\{(\beta-\mu-\alpha(y+z))[(1-\theta)\beta+\alpha x]b_2(w)-w([(1-\theta)\beta+\alpha x]b_1(w)-1)\}\alpha xC_1'(w)\\
&+\{(\beta-\mu-\alpha(y+z))[(1-\theta)\beta+\alpha x]b_1'(w)+[(1-\theta)\beta+\alpha x]b_2(w)\\
&+w[(1-\theta)\beta+\alpha x]b_2'(w)\}(\alpha xC_2(w)-w)+\{(\beta-\mu-\alpha(y+z))([(1-\theta)\beta+\alpha x]b_1(w)-1)\\
&+w[(1-\theta)\beta+\alpha x]b_2(w)\}(\alpha xC_2'(w)-1)-(\alpha x(\beta-\mu)b_1'(w)+\alpha xb_2(w)\\
&+w\alpha xb_2'(w))((1-\theta)\beta+\alpha x)C_2(w)-(\alpha x(\beta-\mu)b_1(w)+w\alpha xb_2(w))((1-\theta)\beta+\alpha x)C_2'(w)\\
&-(\alpha x(\beta-\mu)b_2'(w)-\alpha xb_1(w)-w\alpha xb_1'(w))((1-\theta)\beta+\alpha x)C_1(w)-(\alpha x(\beta-\mu)b_2(w)\\
&-w\alpha xb_1(w))((1-\theta)\beta+\alpha x)C_1'(w)-\alpha b_1'(w)(y+z)(\theta\beta-\alpha x)(\alpha xC_2(w)-w)\\
&-\alpha b_1(w)(y+z)(\theta\beta-\alpha x)(\alpha xC_2'(w)-1)-\alpha b_2'(w)(y+z)(\theta\beta-\alpha x)(\beta+\alpha xC_1(w)-\mu-\delta_2)\\
&-\alpha b_2(w)(y+z)(\theta\beta-\alpha x)\alpha xC_1'(w)+\alpha^2x(y+z)(\theta\beta-\alpha x)C_2'(w)\\
&+\alpha^2x(y+z)C_1'(w)[(1-\theta)\beta+\alpha x]b_2(w)+\alpha^2x(y+z)C_1(w)[(1-\theta)\beta+\alpha x]b_2'(w)\\
&+\alpha^2x(y+z)C_2'(w)([(1-\theta)\beta+\alpha x]b_1(w)-1)\\
&+\alpha^2x(y+z)C_2(w)((1-\theta)\beta+\alpha x)b_1'(w)
\end{aligned} \tag{4.97}$$

$$G_2'(0) = \{(\beta - \mu - \alpha(y+z))[(1-\theta)\beta + \alpha x]b_2'(0)$$

$$-([(1-\theta)\beta + \alpha x]b_1(0) - 1)\}(\beta + \alpha x C_1(0) - \mu - \delta_2)$$

$$+(\beta - \mu - \alpha(y+z))[(1-\theta)\beta + \alpha x]b_2(0)\alpha x C_1'(0)$$

$$+\{(\beta - \mu - \alpha(y+z))[(1-\theta)\beta + \alpha x]b_1'(0) + [(1-\theta)\beta + \alpha x]b_2(0)\}\alpha x C_2(0)$$

$$+(\beta - \mu - \alpha(y+z))([(1-\theta)\beta + \alpha x]b_1(0) - 1)(\alpha x C_2'(0) - 1)$$

$$-(\alpha x(\beta - \mu)b_1'(0) + \alpha x b_2(0))((1-\theta)\beta + \alpha x)C_2(0)$$

$$-(\alpha x(\beta - \mu)b_1(0))((1-\theta)\beta + \alpha x)C_2'(0) - (\alpha x(\beta - \mu)b_2'(0)$$

$$-\alpha x b_1(0))((1-\theta)\beta + \alpha x)C_1(0) - \alpha x b_2(0)(\beta - \mu)((1-\theta)\beta + \alpha x)C_1'(0)$$

$$-\alpha b_1'(0)(y+z)(\theta\beta - \alpha x)(\alpha x C_2(0)) - \alpha b_1(0)(y+z)(\theta\beta - \alpha x)(\alpha x C_2'(0) - 1)$$

$$-\alpha b_2'(0)(y+z)(\theta\beta - \alpha x)(\beta + \alpha x C_1(0) - \mu - \delta_2) - \alpha b_2(0)(y+z)(\theta\beta - \alpha x)\alpha x C_1'(0)$$

$$+\alpha^2 x(y+z)(\theta\beta - \alpha x)C_2'(0) + \alpha^2 x(y+z)C_1'(0)[(1-\theta)\beta + \alpha x]b_2(0)$$

$$+\alpha^2 x(y+z)C_1(0)[(1-\theta)\beta + \alpha x]b_2'(0) + \alpha^2 x(y+z)C_2'(0)([(1-\theta)\beta + \alpha x]b_1(0) - 1)$$

$$+\alpha^2 x(y+z)C_2(0)((1-\theta)\beta + \alpha x)b_1'(0)$$

Substituting for $b_1(0) = \bar{h}$, $b_1'(0) = b_2(0) = C_2(0) = C_1'(0) = 0$, $b_2'(0) = -\psi$,

$C_1(0) = \pi$ and $C_2'(0) = -A$, we obtain;

$$G_2'(0) = -\{[(\beta-\mu-\alpha(y+z))[(1-\theta)\beta+\alpha x]\psi + ([(1-\theta)\beta+\alpha x]\bar{h}-1)](\beta+\alpha\Re x-\mu-\delta_2)$$

$$+(\beta-\mu-\alpha(y+z))([(1-\theta)\beta+\alpha x]\bar{h}-1)(\alpha Ax+1)-\alpha\bar{h}Ax(\beta-\mu)((1-\theta)\beta+\alpha x)$$

$$-(\alpha\Re x(\beta-\mu)\psi-\alpha\bar{h}x)((1-\theta)\beta+\alpha x)-\alpha\bar{h}(y+z)(\theta\beta-\alpha x)(\alpha Ax+1)$$

$$-\alpha\psi(y+z)(\theta\beta-\alpha x)(\beta+\alpha\Re x-\mu-\delta_2)+\alpha^2Ax(y+z)(\theta\beta-\alpha x)$$

$$+\alpha^2\Re x(y+z)((1-\theta)\beta+\alpha x)\psi+\alpha^2Ax(y+z)([(1-\theta)\beta+\alpha x]\bar{h}-1)\} \qquad (4.98)$$

From the inequality (4.76) we have;

$$-\{(\beta-\mu-\alpha(y+z))([(1-\theta)\beta+\alpha x]\bar{h}-1)(\beta+\alpha\Re x-\mu-\delta_2)-\alpha\bar{h}x(\beta-\mu)((1-\theta)\beta+\alpha x)\Re$$

$$-\alpha\bar{h}(y+z)(\theta\beta-\alpha x)(\beta+\alpha\Re x-\mu-\delta_2)+\alpha^2\Re x(y+z)(\theta\beta-\alpha x)$$

$$+\alpha^2\Re x(y+z)([(1-\theta)\beta+\alpha x]\bar{h}-1)\}\{[(\beta-\mu-\alpha(y+z))[(1-\theta)\beta+\alpha x]\psi$$

$$+([(1-\theta)\beta+\alpha x]\bar{h}-1)](\beta+\alpha\Re x-\mu-\delta_2)+(\beta-\mu-\alpha(y+z))([(1-\theta)\beta+\alpha x]\bar{h}-1)(\alpha Ax+1)$$

$$-\alpha\bar{h}Ax(\beta-\mu)((1-\theta)\beta+\alpha x)-(\alpha\Re x(\beta-\mu)\psi-\alpha\bar{h}x)((1-\theta)\beta+\alpha x)$$

$$-\alpha\bar{h}(y+z)(\theta\beta-\alpha x)(\alpha Ax+1)-\alpha\psi(y+z)(\theta\beta-\alpha x)(\beta+\alpha\Re x-\mu-\delta_2)$$

$$+\alpha^2Ax(y+z)(\theta\beta-\alpha x)+\alpha^2\Re x(y+z)((1-\theta)\beta+\alpha x)\psi$$

$$+\alpha^2Ax(y+z)([(1-\theta)\beta+\alpha x]\bar{h}-1)\} = F_2(0)G_2'(0) \qquad (4.99)$$

We shall then analyse equation (4.99) for stability or otherwise of the of non-zero equilibrium state by applying numerical methods and computer simulation using hypothetical values for parameters, to determine the sign (i.e. whether

positive or not) of equation (4.99). Recall the condition for stability as given by equation (4.99); $F_2(0)G_2'(0) > 0$

Recall from equation (4.71) that;

$$\bar{h} = -\left[\mu + \tan\left(\frac{\pi}{2k}\right)\right]^{-1} \exp\left(-\mu T + \frac{2kT}{\pi}\log\cos\left(\frac{\pi\tau}{2kT}\right)\right) + \left[\mu\right]^{-1}$$

And from equation (4.55) that;

$$\psi = \int_0^T \tau h(\tau) d\tau$$

Where $h(\tau) = \exp\left\{-\int_0^\tau \left(\mu + \tan\left(\frac{\pi s}{2kT}\right)\right) ds\right\}$

$$= \exp\left\{-\left[\mu\tau - \frac{2kT}{\pi}\log\cos\left(\frac{\pi\tau}{2kT}\right)\right]\right\}$$

$$\Rightarrow \psi = \int_0^T \tau \exp\left\{-\left[\mu\tau - \frac{2kT}{\pi}\log\cos\left(\frac{\pi\tau}{2kT}\right)\right]\right\} d\tau \qquad (4.100)$$

And from equation (4.83), that

$$\Re = \int_0^T \sigma(\tau) h(\tau) d\tau$$

Substituting for $\sigma(\tau)$ and $h(\tau)$ we have;

$$\Re = \int_0^T \tan\left(\frac{\pi\tau}{2kT}\right) \exp\left\{-\int_0^\tau \left(\mu + \tan\left(\frac{\pi s}{2kT}\right)\right) ds\right\} d\tau$$

$$= \int_0^T \tan\left(\frac{\pi\tau}{2kT}\right)\exp\left\{-\left[\mu\tau - \frac{2kT}{\pi}\log\cos\left(\frac{\pi\tau}{2kT}\right)\right]\right\}d\tau \tag{4.101}$$

Also, from equation (4.88);

$$A = \int_0^T \tau\sigma(\tau)h(\tau)d\tau$$

We then simplify to obtain;

$$A = \int_0^T \tau\tan\left(\frac{\pi\tau}{2kT}\right)\exp\left\{-\left[\mu\tau - \frac{2kT}{\pi}\log\cos\left(\frac{\pi\tau}{2kT}\right)\right]\right\}d\tau \tag{4.102}$$

Using the Simpson's formula for numerical integration, we evaluate to obtain approximations for ψ, $\Re$, and A for $0 \le \tau < 10$ and $0 < k < 1$. We have considered low value k as given by $k = 0.3$ and high value k given by $k = 0.9$ for $\tau = [0,1,2,3,4,5,6,7,8,9]$ and compared the stability for the low and high values of k.

Substituting for the values of x, y, z as given by equations (3.34), (3.33), and (3.32) respectively, and $\bar{h}$ given by equation (4.71), as well as the numerical approximations of ψ, $\Re$, and A into equation (4.99), we define the resulting function by $J_2(k)$ which is evaluated to determine the sign and hence the stability of the non-zero equilibrium state for different values of the natural birth rate β and death rate μ of the population. Thus, the non-zero equilibrium will be stable when $J_2(k) > 0$. We have used MathLab to solve $J_2(k)$ generating a table of values using hypothetical values for the parameters. The results are presented in the next chapter.

CHAPTER FIVE

CONCLUSION RECOMMENDATIONS

5.1 INTRODUCTION

This section presents discussions/remarks based on the results obtained in the work. Due to non availability of accurate statistical data for HIV/AIDS, we shall use MathLab to generate tables of values for $J_1(k)$ and $J_2(k)$ with hypothetical values for the parameters. Some of the results obtained are presented in Table 5.1 and Table 5.2. Also presented is a graphical representation $J_1(k)$ for given values of k, and different values of the death rate μ and birth rate β as shown in Figure 5.1. We also presented some recommendations based on our results.

5.2 DISCUSSION OF RESULTS

This section presents the results using tables and graphs as tools for illustration. Tables 5.1 and 5.2 are profiles for $J_1(k)$ and $J_2(k)$ respectively. Similarly, Figure 5.1 is the trajectory for $J_1(k)$, which represents the characteristic of the model for the zero equilibrium state at different values of the birth rate β and death rate μ. The trajectory for $J_2(k)$, which represents the characteristic of the model for non-zero equilibrium state is not shown.

Table 5.1 – Stability Profile for $J_1(k)$

$\delta_1 = 0.3,\ \theta = 0.4,\ T = 10$

K	$J_1(k)$ $\mu = 0.15, \beta = 0.45$	$J_1(k)$ $\mu = 0.25, \beta = 0.15$	$J_1(k)$ $\mu = 0.20, \beta = 0.10$	$J_1(k)$ $\mu = 0.15, \beta = 0.15$
0.1	0.6059	-0.6580	-0.7225	-0.4647
0.2	0.6808	-0.6521	-0.7145	-0.4397
0.3	0.7266	-0.6478	-0.7092	-0.4245
0.4	0.7599	-0.6444	-0.7051	-0.4134
0.5	0.7826	-0.6420	-0.7023	-0.4058
0.6	0.7948	-0.6406	-0.7007	-0.4017
0.7	0.7992	-0.6401	-0.7001	-0.4003
0.8	0.8000	-0.6400	-0.7000	-0.4000
0.9	0.8000	-0.6400	-0.7000	-0.4000
Remarks	Unstable	Stable	Stable	Stable

Table 5.1 gives the profile for $J_1(k)$ and we observe from the table that $J_1(k) > 0$ only when $\beta >> \mu$; i.e. when the birth rate β is much greater than the death rate μ of the population. We also found out that the profile follows the same trend for all values of $\delta_1, \theta \leq 1$, where δ and θ are death modulus due to AIDS infection and proportion of new births of the latent class that are

susceptible respectively. We observe that the zero equilibrium state will always be stable except for situations where the birth rate β is much higher than the death rate μ of the population. This situation is unusual considering the fact that the young and sexually active groups of the population are the worst affected by the pandemic. This means that for the population to be sustained once HIV/AIDS epidemic manifest, there should be a way of obtaining a speedy replenishment of the population. This could be achieved through accelerated birth rate and friendly immigration policies.

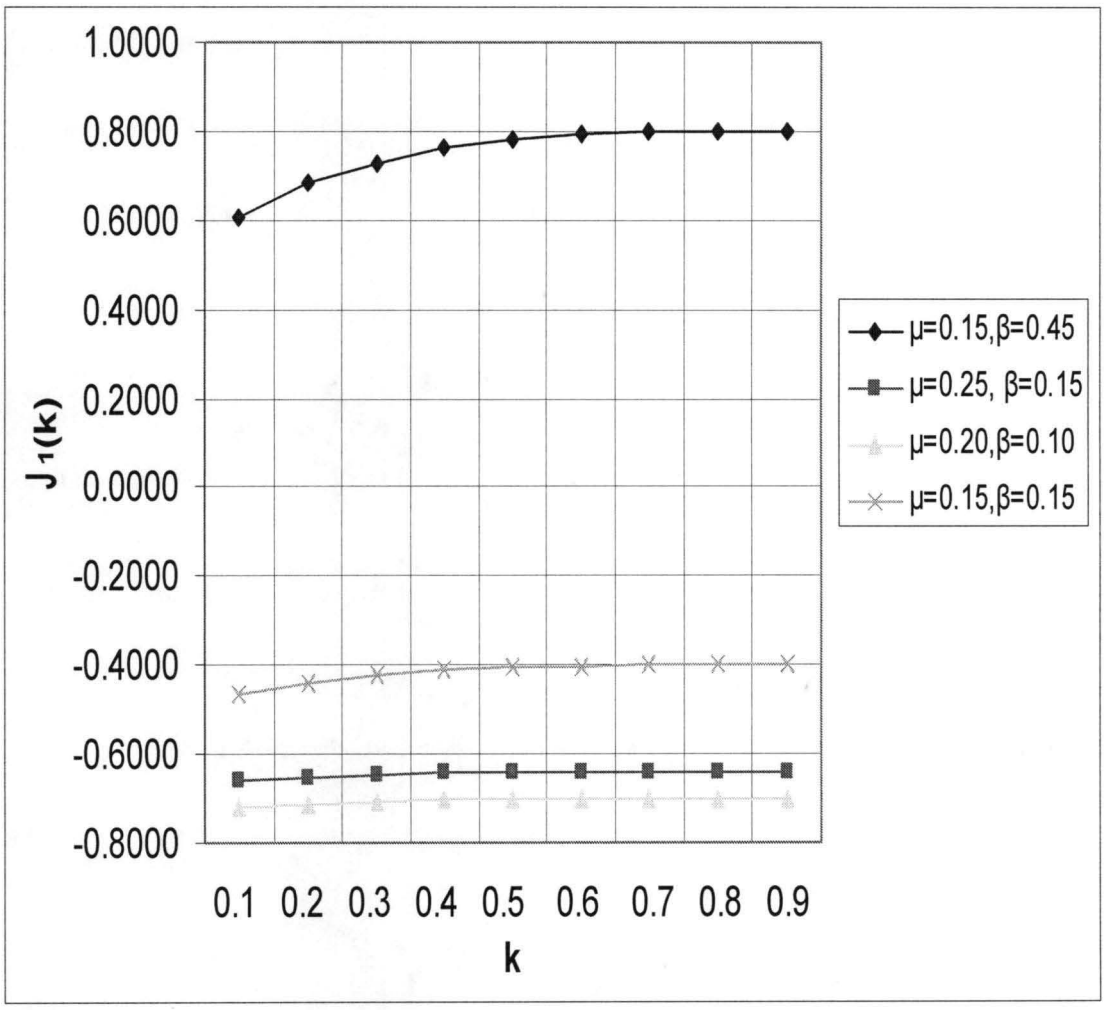


Figure 5.1 – Graphical Representation of the profile for $J_1(k)$

The figure above is a graphical demonstration of our result. It can be seen that the graph $J_1(k)$ for which the birth rate β is greater than the death rate μ lies entirely on the positive plane, while when β is less than or equal to μ, $J_1(k)$ lies on negative plain. We also observe fro figure 5.1 that $J_1(k)$ approaches a maximum value as k increases. This observation is consistent with the application of antiretroviral drugs which only differs the eventual infection with full blown AIDS once an individual has contracted the virus. This also shows that with preventive and control measures such as public awareness campaign, promotion of abstinence among unmarried people and faithfulness among sexual partners, use of condoms, public health education; the effectiveness of control increases as the effort and level of response increases. However, the epidemic situation tends to stabilise with time even with more application of mitigation and control measures such as antiretroviral drugs. This could be due to complacency, which could lead to a return to high risk behaviours as shown by Flugentius Baryarama et al (2005).

Table 5.2 – Stability Profile for $J_2(k)$

$\delta_1 = 0.3$, $\theta = 0.4$, $T = 9$, $\alpha = 0.05$, $\delta = 0.025$

K	$J_2(k)$ $\mu = 0.15$ $\beta = 0.45$	$J_2(k)$ $\mu = 0.10$, $\beta = 0.20$	$J_2(k)$ $\mu = 0.15$ $\beta = 0.15$	$J_2(k)$ $\mu = 0.20$ $\beta = 0.10$	$J_2(k)$ $\mu = 0.25$, $\beta = 0.15$	$J_2(k)$ $\mu = 0.45$ $\beta = 0.15$
0.3	-0.6563	0.0004	0.0000	0.0054	0.0049	0.1226
0.9	-0.5787	0.0000	0.0000	0.0053	0.0048	0.1186
Remarks	Unstable	Stable	Stable	Stable	Stable	Stable

Table 5.2 gives the profile for $J_2(k)$ from which we observe that $J_2(k) < 0$ only when $\beta >> \mu$ i.e. when the birth rate β is much greater than the death rate μ of the population for both low and high values of the control parameter k. This indicates deteriorating epidemic situation, which may lead to eventual extinction of the population. This situation could be attributed to complacency, which is used to mean revert to high risk behaviours such as multiple sexual partners, sex with prostitutes, ignoring safe sex messages such as use of condoms when the HIV prevalence reduces to very low level, with the rate AIDS infection becoming less in the community. This result is particularly significant in most communities especially in sub-Saharan Africa where HIV/AIDS interventions are mainly donor-funded through programmes with specific duration where complacency could result from withdrawal of intervention or expiration of programmes duration. In this case, the progress made during the period of interventions might fade away as soon as the programmes end with high tendency for people to go back to risky behaviours that expose them to infection. The results also show that $J_2(k)$ is non-negative whenever $\beta \leq \mu$ i.e. when the birth rate β is less than or equal to the death rate μ of the population for both low and high values of the control parameter k.

From the results, we observe that the non-zero equilibrium state will be predominantly stable as long as the rate of contracting the HIV virus α and the rate of AIDS infection $\sigma(\tau)$ as well as the death modulus due to infection δ are very small for all values of the control parameter k, except for situations where $\beta >> \mu$ in a community. Thus we observe that the non-zero equilibrium state is predominantly stable especially with the application of effective control and improved mitigation measures aimed at checking the prevalence rate (i.e. the

rate of contracting HIV) of the virus and reducing death burden due the full blown AIDS infection. This explains the reason why many infected communities have been able to control the spread of the virus and manage the epidemic. This means that an infected population can actually be sustained with effective applications of control and mitigation measures.

5.3 CONCLUSION

From the analysis of equations (4.35) and (4.36) we note that the zero equilibrium state of the model will always be stable when $\beta < \mu$, since $J_1(k) < 0$. The stability of the origin implies that once the virus is introduced into a population, there is an imminent danger of eventually extinction. On the other hand, the analysis of equation (4.74) shows that the non-zero equilibrium state of the model is unstable with $J_2(k) \geq 0$ when $\beta \leq \mu$, and will only be stable when $J_2(k) < 0$ and $\beta >> \mu$. This indicates the tendency for rapid and wide spread of the epidemic once it is introduced into any community. This explains observed cases in some African countries where the epidemic nearly wiped out infected communities.

Therefore, in order to forestall such tragedy there should be a means of replenishing the population and providing improved and effective application of control parameters especially to reduce the HIV/AIDS prevalence rate. We therefore conclude from our results that a virus infected population or community can only be sustained for conditions that make the origin unstable; of particular importance is the unusual situation where the birth rate is greater

than the death rate, with high level of preventive and control measures particularly to reduce the rate of contracting the virus.

5.4 RECOMMENDATIONS

By implication, the introduction of the virus into a population poses definite health threat, hence the need to adopt a preventive approach and intensify response to the fight against the pandemic. The preventive approach is particularly recommended because once the epidemic is introduced into any population, all known remedies including the use of antiretroviral drugs and prompt medical treatment of opportunistic infections can only delay the inevitable eventual extinction of the population. Similarly, efforts should be made to combat complacency in HIV/AIDS interventions when there is a reduction in the number of AIDS cases as well as when intervention programmes expire or are withdrawn, which could lead to periodic behaviour of the HIV/AIDS epidemic. Thus, it has become imperative to sustain all HIV/AIDS interventions even with significant reduction in HIV prevalence and the number of AIDS cases in a community.

Globally, especially in developing countries, HIV/AIDS has remained a major health problem, and the young age-groups are usually most affected. Therefore, health service intervention programmes need to be age-specifically oriented to protect the population highly exposed to risk. Considering the cultural and socioeconomic factors affecting the transmission, control and management of the disease, any effective response should adopt a holistic approach. This will help to correct erroneous myths as well as eliminate stigmatization associated

with the epidemic, and eliminate discrimination against those living with HIV/AIDS especially in the rural areas.

REFERENCES

Adomain G., Adomain G.E. (1985): Solution of the Marchuk Model of Infectious Disease and Immune Response; *Math. Modelling (Special Issue: Math. Models in Medicine – Diseases and Epidemics) Vol. 7, 803 – 807.*

Akinwande N.I. (1995): Local Stability Analysis of Equilibrium State of a Mathematical Model of Yellow Fever Epidemics; *J. Nig. Math. Soc. Vol. 14, 73 – 79.*

Akinwande N.I. (1996): A Mathematical Model of Yellow Fever Epidemics; *Afrika Mathematika Series 3, Vol.6, 49 -59 (Mathematical Reviews: 97j; 92012; 92D40)*

Akinwande N.I. (2005): Application of Laplace Transform to the Stability Analysis of the Non-Zero Equilibrium State of a yellow Fever Disease Dynamics Model; *Science Focus, LAUTECH, (in Press).*

Akinwande N.I. (2005): Stability Analysis of the Zero Equilibrium State of a Mathematical Model of the Dynamics of HIV/AIDS Pandemic.

Anderson R.M., May R.M. Population Biology and Infectious Diseases; *Nature Vol. 280, (1979)361 – 367.*

Anderson R.M., Jackson H.C., May R.M., Smith A.M.: Population Dynamics of Fox Rabies in Europe; *Nature Vol. 289 (1981) 765 – 771.*

Andrea Di Liddo (1985): A S-I-R Vector Disease Model with Delay; *Math Modelling (Special Issue: Math. Models in Medicine – Diseases and Epidemics) Vol. 7, 793 – 802.*

Auvert B, Buonamico G, Lagarde E, Williams B. (2000): Sexual Behaviour Heterosexual Transmission, and the Spread of HIV in sub-Sahara Africa: A Simulation Study; *Comput Biomed Res. Vol. 1, 84 – 96.*

Bellman R. and Cooke K.L. (1963): Differential Difference Equations; *Academic Press, London.*

Brauer F. (1990): Models for the Spread of Universally Fatal diseases; *J. Math. Biol., Springer-Verlag; 451 – 462.*

Chichia Chiu (1990): A Numerical Method for Nonlinear Age Dependent Population Models; *Differential and integral Equations, vol. 3 (4), 767 – 782.*

Claude Lefevre and Marie-Pierre Malice (1985): A Discrete Time Model for S-I-S Infectious disease with a Random Number of Contacts Between Individuals; *Math. Modelling (Special Issue: Math. Models in Medicine – Diseases and Epidemics); Vol. 7, 785 – 792.*

Clive L. Dym and Elizabeth S. Ivey (1980): Principles of Mathematical Modelling; *Academic Press, Inc. New York.*

Cvjetanovic B., Grab G. and Dixon H. (1985): Computerised Epidemiological Model of Typhoid Fever with Age Structure and its Use in Planning and Evaluation of Anti-typhoid Immunization and Sanitation Programmes; *Math. Modelling (Special Issue: Math. Models in Medicine – Diseases and Epidemics) Vol. 7, 719 – 744.*

David A. Sanchez (1967): Ordinary Differential Equations and Stability Theory - An Introduction.

David Greenhalgh (1985): Simple Model for Control of Epidemics; *Math. Modelling (Special Issue: Math. Models in Medicine) Vol. 7, 753 – 764.*

Department for International development (DFID) (May 2001): HIV/AIDS Strategy Report.

Flugentius Baryarama, Livingstone S. Luboobi and Joseph Y.T. Mugisha (2005): Periodicity of the HIV/AIDS Epidemic in a Mathematical Model that Incorporates Complacency; *American Journal of Infectious Diseases Vol. 1(1), 55 – 60.*

Garnett G.P, Anderson R.M. (1994): Balancing Sexual Partnerships in an Age and Activity Stratified Model of HIV Transmission in Heterosexual Populations; *IMA J Math Appl Med Biol. Vol. 11 (3), 161 – 192.*

Garnett G.P, Anderson R.M. (1995): Strategies for Limiting the Spread of HIV in Developing Countries: Conclusions Based on Studies of the Transmission Dynamics of the Virus; *J Acquir Immune Defic Syndr Hum Retrovirol Vol.15 (5); 500- 513.*

Gurtin M.E. and Maccamy R.C.: Nonlinear Age-Dependent Population Dynamics; *Arch. Rat. Mech. Anal. Vol. 54 (1974) 281 – 300).*

Hsieh Y.H, Cooke K. (2000): Behaviour Change and Treatment of Core Groups: Its Effect on the Spread of HIV/AIDS; *IMA J Math Appl Med Biol. Vol. 17(3), 213 – 241).*

Hubbard J.H. and West B.H. (1991): Differential Equations – A Dynamical Approach Part I; *Springer-Verlag New York Inc.*

Jack Hale (1977): Theory of Functional Differential Equations;

John Pickering, James A. Willy, Nancy S. Padian, Loren E. Lieb, Dean F. Echenberg and Juliette Walker (1986): Modelling the Incidence of AIDS in San Francisco, Los Angeles, and New York; *Math. Modelling (Special Issue: Math. Models in Medicine – Diseases and Epidemics) Vol. 7, 661 – 688.*

M. Kakehashi (1998): A Mathematical Analysis of the Spread of HIV/AIDS in Japan; *Mathematical Medicine and Biology Vol. 15(4), 299 – 311.*

Nico J.D. Nagelkerke, Prabhat Jha, Sake J. de Vlas, Eline L. Korenromp, Stephen Moses, James F. Blanchard, & Frank A. Plummer (2002): Modelling HIV/AIDS epidemics in Botswana and India: impact of interventions to prevent transmission; *Bulletin of the World Health Organization (Bull World Health Organ v.80 n.2).*

Murray J.D. (1989): Mathematical Biology; *Springer-Verlag New York Inc.*

National AIDS/STDs Control Programme (2004): 2003 HIV Sero-prevalence Sentinel Survey; *Department of Public Health, Federal Ministry of Health, Nigeria.*

Norman T.J. Bailey (1985): Macro Modelling and Prediction of Epidemic Spread at Community Level; *Math. Modelling (Special Issue: Math. Model in Medicine – Diseases and Epidemics) Vol. 7, 689 – 719.*

Pugliese A. (1990): Population Models for Diseases with no Recovery; *J. Math. Biol. Vol. 28, 65 – 82; Springer-Verlag.*

Sowunmi C.O.A. (1993): A Model of Heterosexual Population Dynamics with Age Structure and Gestation Period; *J. Math. Ana. App., Vol.172(2), 390 – 411.*

Sowunmi C.O.A. (2000): Stability of Steady State and Boundedness of a 2-Sex Population Model; *Nonlinear Analysis Vol. 39, 693 – 709).*

Tan W.Y. and Shamsa F. (1990): Assessing the Effect of Intervention on AIDS Development Induced by Blood Transfusion; *Math and Comp. Modelling, Vol. 14, 635 – 639.*

Tan W.Y, Xiang Z. (1998): A State Space Model for the HIV Epidemic in Homosexual Populations and Some Applications; *Math Biosci. Vol. 152(1), 29 – 61.*

Ying-Hen Hsieh (1990): An AIDS Model with Screening; *Math and Comp. Modelling Vol. 14, 640 – 643.*