MATHEMATICAL MODELLING OF THE AIDS EPIDEMIC

TESTING FOR SEROPOSITIVITY OF THE HUMAN IMMUNODEFICIENCY
VIRUS

By

CYNTHIA ROSE BOULANGER, B. Sc.4 yr Spec. (Brandon University)

A Thesis

Submitted to the School of Graduate Studies

in Partial Fulfilment of the Requirements

for the Degree

Master of Science

McMaster University

MASTER OF SCIENCE (1991) MCMASTER UNIVERSITY
(Mathematics) Hamilton, Ontario

TITLE: Testing for Seropositivity of the Human Immunodeficiency Virus

AUTHOR: Cynthia Rose Boulanger
B. Sc.4 yr Spec. (Brandon University)

SUPERVISOR: G.S.K. Wolkowicz

NUMBER OF PAGES: x, 197

Abstract

This paper considers a series of models and the effect of HIV antibody testing on the dynamics of the disease. We examine HIV antibody testing in conjunction with persuasive techniques designed to encourage tested infecteds to behave in a sexually responsible manner. The population under consideration is a homosexual population. Analytical methods are used to obtain information about the qualitative behaviour of the models. Areas requiring further study are discussed.

Acknowledgements

I would like to express my gratitude to my supervisor, Dr. G.S.K. Wolkowicz, for her patience and her helpful suggestions throughout the course of my thesis preparation.

I would also like to acknowledge the members of my defense committee, Professors G. Wolkowicz, J. Chadam, Z. Kovarik and H. Heinig.

I would like to thank my family, especially my son, Cory Boulanger, for his patience and understanding. A special thanks to M. Ballyk for her never ending support.

Notation

$S(t)$ -   Susceptibles

$I(t)$ -   Infecteds, not tested.

$Q(t)$ -   Infecteds, tested, sexually irresponsible.

$P(t)$ -   Infecteds, tested, sexually responsible.

$A(t)$ -   Full-blown AIDS.

$N(t)$ -   Total sexually mature homosexual population,

$$N(t) = S(t) + I(t) + Q(t) + P(t) + A(t)$$

           if P and A populations are sexually active.

$$N(t) = S(t) + I(t) + Q(t)$$

           if P and A abstain from sexual activity.

Λ -   Rate of initiation into $S(t)$ per unit time.

β -   Transmission probability per sexual act.

c -   Average number of sexual partners per individual in $S(t)$.

ρ -   Proportion of tested infecteds entering $P(t)$, that is, tested infecteds who act responsibly.

$1 - \rho$ -   Proportion of tested infecteds entering $Q(t)$, that is, tested infecteds who behave irresponsibly

ω -   Proportion of the I population who are tested at time t.

v -   Proportion of tested infecteds ($P(t)$ or $Q(t)$) entering $A(t)$ at time t.

μ -   Natural death rate at time t.

d -   AIDS induced death rate at time t.

Contents

List of Figures

Chapter 1

INTRODUCTION

1.1 Epidemiology

Epidemiology is the study of the spread of infection in a population. An epidemic is a major outbreak of a disease. A disease is called endemic if it is long term and maintained at levels of various severity. An endemic disease is usually composed of many epidemic outbreaks of the disease, with a reduced level of infection between outbreaks. Often, the terms endemic and epidemic are used interchangeably. Ideally epidemiologists would like to gain sufficient insight into the dynamics of infectious diseases to allow them to provide guidelines for their eradication. Historically, infectious diseases have placed an incredible toll on human life. In Europe, in the fourteenth century, for instance, approximately one quarter of the population was wiped out by the Black Plague. Over time, much has been learned about diseases, enabling us to apply preventative techniques like immunization and better health standards, thus providing us with tools for eradication of once rampant diseases. However, there still remains a phenomenal number of diseases in today's society which defy prevention and for which no cures have been developed.

There are several methods of studying infectious diseases including, clinical,

biological, ecological and mathematical. It is only with the combined input of these disciplines, that we can expect to reduce the prevalence of disease in society.

To gain a better understanding of what epidemiology is about we look at a very simple example. Consider one strain of the common cold virus.[1] We assume that initially, we have only a small number of individuals who are suffering from the disease. If we place these "infected" individuals into a group of people who are not yet affected by the virus, or in other words, who have not yet contracted the virus, a certain number of these "susceptibles" would become infected, given appropriate conditions for the spread of the common cold. The appropriate conditions may include adequate proximity between the infected and susceptible persons, or poor health conditions of the disease free individual. Once an individual has outlived the life-span of the microparasite involved in this particular strain of the common cold, then he has developed an immunity to the disease, and is now considered to be a member of the removed class. That is, he can no longer contribute to the spread of the disease.

We attempt to describe the spread of infectious diseases in the population, with mathematical models, using certain simplifying assumptions appropriate to the specific disease behaviour and population mixing. With the use of these models we are often able to ascertain certain information which may lead to control or even eradication of the disease. The mathematical models may provide sufficient insight into the likelihood of transmission and spread of infectious disease, as well as the information required to predict future trends of the epidemic. Mathematical models are valuable for examining biological aspects, such as degree of infectiousness in individuals at various times in the course of the disease, or of the life-cycle of the microparasite being considered. In addition, we may be able to ascertain threshold conditions which indicate what restrictions we need to impose on the population, and what biological interventions are requisite to eradicate or at least impede the spread of disease.

As an introduction to mathematical modelling of infectious diseases we look at earlier stages of development of the theory involved in epidemiology. Our example

---

[1]There are a number of different strains of the common cold but once an individual has suffered through the course of infection of a particular strain, that individual develops a life-long immunity to that particular strain.

of the common cold is very similar to some of the earliest models in epidemiology developed by Kermack and McKendrick (1927), (see [36], [37], and [50]). They divide the population into three component classes, as we have, in our common cold example. We assume that each class is disjoint from the others.

These classes are

1. Susceptible (S) – containing non-infected individuals who are capable of contracting the disease.

2. Infected (I) – containing those individuals who are infected and capable of transmitting the disease given appropriate conditions characteristic of the disease.

3. Removed (R) – containing those individuals who have developed an immunity to the disease and are no longer capable of being involved in the spread of the disease. In addition, this class could represent the number of those who have died in the course of infection or from other causes, or those individuals isolated from the susceptible population.

Appropriate conditions for the transmission of the infection usually include direct physical contact with an infected individual, inhaling infectious microorganisms, eating contaminated foods or contact with an agent such as a mosquito. Moreover, the susceptible individual may or may not possess certain resistance to a disease, through varying biological or biochemical defences. Once an individual has been infected, he generally must succumb to the effects of the parasite in the course of its life-cycle. Very often there is a latency period, in which an infected can not transmit the disease. The disease at this point is merely developing internally. At some point, however, the microparasite will have matured enough to make this infected person infectious. This person is then capable of passing the microorganism onto another unsuspecting susceptible. Eventually, the infected will exhibit recognizable symptoms. Formerly, it was at this point that an infected person might be isolated from the susceptible population until immunity had been developed or at least until communication of the parasite was no longer possible. Frequently, the natural course of the parasite would involve death of the infected person. More recently, however, due to the high cost

of medical facilities and the availability of more humane treatments, isolation is not as common. Other methods are now more frequently used to arrest the spread of disease. Most models to date incorporate these hypotheses.

Kermack and McKendrick made three basic assumptions as a basis for their model.

1. The population size is constant, or alternatively, we are only considering a closed population which excludes birth and mortality information.

2. The rate of new infections is proportional to the number of contacts between S and I where the number of contacts are given as a proportion of the product of S and I. This hypothesis follows the law of mass action, which assumes uniform mixing of the population.

3. Infecteds are removed from the infected class at a rate proportional to the number of infecteds, or in other words, recovery is equally likely among infecteds.

Under these assumptions Kermack and McKendrick derived the basic SIR model. The flow of individuals in their system can be represented schematically as

$$S \longrightarrow I \longrightarrow R.$$

It is assumed that we are dealing with a continuous-infection model involving a very large population. This allows us to treat the population as a continuum. The equations with $S(t), I(t)$, and $R(t)$ representing the respective population sizes at time t are given by,

$$
\begin{aligned}
S'(t) &= -rS(t)I(t) \\
I'(t) &= rS(t)I(t) - \gamma I(t) \\
R'(t) &= \gamma I(t)
\end{aligned}
\tag{1.1}
$$

where, the total population

$$
\begin{aligned}
N(t) &= S(t) + I(t) + R(t) \\
S(0) &= S_o > 0, \quad I(0) = I_o > 0 \text{ and } R(0) = 0.
\end{aligned}
$$

Note that $S'(t) + I'(t) + R'(t) = N'(t) = 0$, thus satisfying assumption 1. Since the population is constant, $N(t) = S_0 + I_0$ for all t. The proportionality constants r and γ are positive, where $r > 0$ is the infection rate and $\gamma > 0$ is the removal rate. The first equation in 1.1, indicates that the susceptible population will decrease at a rate proportional to the number of contacts between the infecteds and the susceptibles. The constant term r, can be determined by the history of the transmission of the microparasite. The second equation respresents the change in $I(t)$ at any time t. This class receives new members equivalent to the number of individuals leaving $S(t)$. The rate at which class $I(t)$ loses individuals is given by the parameter γ. The constant γ can be determined through data collection, and represents the rate at which the disease loses its infective power. The final equation symbolizes the flow into the recovered class $R(t)$. This amount is identical to the number of persons leaving the infected class. In addition, note that there are no terms in the above equations which include entry into $S(t)$ or exit from $R(t)$.

Kermack and McKendrick are credited with the threshold theorm, (see [36]), that has become invaluable in epidemiology. This theorem states that there exists a threshold or critical value of the susceptible population size such that if this threshold value is surpassed, an epidemic will occur. Directly related with this is what we call the reproductive number. If the reproductive number, (see [7]), generally labelled R_o, defined as the number of secondary infections produced when one infected individual is placed in a wholly susceptible population, is greater than one, than an epidemic will occur. If this reproductive number is less than one, then the disease will die out.

Although the SIR model presented here furnished researchers with important results, the model has several limitations. One of these includes the lack of vital dynamics, accounting for rates of births and deaths in populations. Generally, if a population is large enough and if the period of time under study is short, then assuming a constant population with no births or deaths simplifies the model and

is therefore useful for modelling purposes, since any contributions such as births or deaths would be negligible. However, this is not usually the case. More often we must account for births and deaths in order to improve the predictive capabilities of the model. The inclusion of vital dynamics becomes a standard issue with mathematical modellers. However, in order to maintain simplicity it is often assumed that the birth rate is identical to the mortality rate, hence maintaining a constant population size.

Kermack's and McKendrick's model is simplistic, but the form of their equations provides a good base upon which elementary decisions can be made. However, there is room for expansion of the Kermack/McKendrick model. If the population is homogeneous, (i.e. uniformly distributed), and random mixing can be assumed then model 1.1 is a relatively good approximation. However, very often there are other important factors that enter into the dynamics of the disease. If the population is not uniform or similarly, if there are geographic or demographic factors contributing to the spread of the disease, then we cannot assume random or uniform mixing. Thus, more complicated models have to be developed to account for these characteristics of the population.

With Kermack's and McKendrick's model, researchers are equipped with a noteworthy starting point. More factors can be incorporated into future models, see for example [50], [29], [30], [8], and [17]. For instance, it is not such an unmanageable task to include more precise characteristics of the infective agent as it progresses through its life cycle. Some diseases are characterized by a brief period of immunity following the period of infectivity. The immunity eventually wears off and an individual becomes a susceptible again. Tetanus, smallpox, influenza, cholera and typhoid fever are examples of a disease of this nature. (See [26]). In this case the general flow of the population can be represented schematically by

$$S \longrightarrow I \longrightarrow R \longrightarrow S$$

If no immunity is developed, then the following flowchart is appropriate.

$$S \longrightarrow I \longrightarrow S$$

A disease which falls under this category is gonnorhea, a sexually transmitted disease. Another possibility is a disease which possesses a long latency period. Chicken pox

is a disease which could be modelled within this setting. The flow chart for this progression may be given as

$$S \longrightarrow E \longrightarrow I \longrightarrow R$$

where E represents the population in the latency period.

There are also a variety of other modelling forms which are presently being used in research today. (See for example [3] and [4].) The models introduced in this paper are basically of the SIR form.

1.2   Sexually Transmitted Diseases

Gonnorhea, syphilis, and genital herpes are examples of sexually transmitted diseases. The models for sexually transmitted diseases differ from those mentioned in the previous section. Anderson *et al.*, [7] list four major variations.

1. We need only consider those individuals who are sexually active. Further, the relative size of the infected population does not determine the degree of the spread of the infection.

2. There are numerous individuals who do not display any symptoms of the disease, i.e. carriers, but they are still capable of spreading infection for lengthy periods of time.

3. Most often, suffering from a sexually transmitted disease does not induce an immunity. Thus, once an individual has been treated and recovered, the individual becomes a susceptible once more.

4. There are large differences in sexual behaviours and this often contributes to the persistence of the disease in the population.

There are many factors involved in the modelling of sexually transmitted diseases, that we are unable to fully understand. We must account for the social processes of sexual interaction as this is the fundamental means of transmission of sexually transmitted diseases. The inclusion of heterogeneity of behaviour is necessary

for a more complete and comprehensive description of the disease in nature. Initially, though, we examine a homogeneous population in order to get a grasp of the general dynamics of a system. A homogeneous population can be regarded as a population whose members have uniform behaviours. All susceptible individuals would have equal probabilities of contracting the disease. Some examples of homogeneous populations include homosexuals, intravenous drug users, or individuals in a school classroom. On the other hand, individuals in a heterogeneous population would have varying degrees of risky behaviour, or susceptibility with respect to disease transmission. For example, individuals, before marriage, are more likely to have a larger number of sexual partners than after marriage, thus putting them more at risk of contracting a sexually transmitted disease. [2] A heterosexual population is an example of a heterogeneous population. Modelling heterogeneous populations tends to be more accurate than using a homogeneous population in modelling, in light of the complexity of life. For examples of heterogeneous models see [16], [19], [24], and [45]. Adding a heterogeneous quality immediately makes the models more complicated.

Besides behavioural aspects, math modellers must also include certain biological aspects of the disease. For instance, the transmission probability per sexual contact, or variable behaviour of the virus involved in the disease are biological aspects. The life cycle of a sexually transmitted disease virus is considerably different than for a measles virus or for most of the other diseases mentioned in the previous section.

As with most epidemiological models, some factors are significant to some diseases but not to others, for example, the presence of other sexually transmitted diseases may increase the probability of transmission of another sexually transmitted disease. In the AIDS epidemic, insertive sexual contact is less likely to result in infection than is receptive contact. (See [32]). However, this factor is unlikely to be important in the transmission of the herpes virus. In modelling, one must be careful to strike an appropriate balance between complexity and accountability for crucial parameters.

Generally, due to the means of transmission of a sexually transmitted disease,

---

[2]Of course, this comparison is necessarily true only if the married couple has noninfected partners.

and hence the difficulty of surveying the population, there is an extreme lack of data available for accurately modelling these diseases or for testing the accuracy of existing models. Data to determine sexual contact frequency, average number of sexual partners per individual, sexual practices that contribute to the spread of the disease, length of incubation period, transmission probability per sexual contact, and the knowledge of other possible relevant social factors would be helpful.

Cooke and Yorke [17] developed a model describing the gonorrhea epidemic that provided modellers with an excellent base for studying other sexually transmitted diseases, including the AIDS epidemic. This model was the first mathematical model for the transmission of a venereal disease, see [46]. (See also [39] and [53].) More recently, Dietz and Hadeler [19] have presented a model involving eight differential equations that include a pair formation function incorporating female and male behaviours in a nonlinear fashion. (See also [24]). May and Anderson [5] have also been significant contributors in this area of study. They have incorporated different risk groups according to sexual activity. Many models today employ these methods. The models mentioned here represent few of many available approaches to modelling sexually transmitted diseases, see for example, [3], [4], [9], [10], [15], [20], and [33]. Schwager *et al.* [46] provide a brief background on the history of epidemiology, including the modelling of sexually transmitted diseases and AIDS.

This paper considers the sexually transmitted disease called the acquired immunodeficiency syndrome (AIDS), and the virus associated with it, the human immunodeficiency virus (HIV). In the next section we explain some of the important aspects of this debilitating disease.

1.3 Acquired Immunodeficiency Syndrome

The acquired immunodeficiency syndrome has become a major concern to society today. The occurrence and death rate due to AIDS is increasing at a phenomenal rate. It's toll on human life is incredible. Usually, once an individual displays symptoms of AIDS he can be expected to die in approximately one year (see [2]). There are new drugs, such as AZT which may assist in lengthening lifespans, but even so, death due to the disease is highly likely. Primarily, deaths are due to the decrease in immune system capabilities, thus inviting many opportunistic diseases directly related to AIDS (see [32] and [2]). These include Kaposi's Sarcoma, Pneumocystis carinii and cryptococcal meningitis.

Much progress has been made in studying the AIDS epidemic, but much still remains to be discovered. Mathematical modelling provides information about the epidemic which enables us to more fully understand the dynamics of the disease, and further, supplies us with a priority scale for future research and data collection. Allen *et al.* [1], consider many important issues in the prevention of AIDS and HIV infection, and priorize some of their suggestions. They emphasize the need for more research and serosurveillance studies.

AIDS is a sexually transmitted disease with certain unique properties. One basic difference between AIDS and other sexually transmitted diseases, is that AIDS has an extraordinarily long incubation period. During this period the infected individual displays no obvious outward symptoms, although he is still capable of transmitting the disease. This makes disease control even more difficult, since, unless we know which individuals are infected, we are often vulnerable.

There are many other unique components inherent in the AIDS epidemic, such as the high probability of death due to AIDS, whereas in other venereal or sexually transmitted diseases, death is generally avoided. The virus associated with AIDS, the HIV, is a lentivirus, or more specifically a retrovirus, (see [18]). Most viruses involved in sexually transmitted diseases are not retroviruses.

HIV is contained and transmitted through bodily fluids especially through

blood, blood products and semen. We must consider the various activities that in-
volve exchange of these bodily fluids. Contributing factors in the spread of AIDS
can be subdivided into demographic and biological components. At this point, we
consider various social components related to the spread of disease. Sexual activity is
one of the primary mediums for transmission of the HIV. See [52], [7], and [32] for the
following and additional facts concerning the characteristics and determining factors
in the spread of AIDS. Consider the heterogeneity of behaviour involved here. Some
individuals have many different partners, others have few, and still others are absti-
nent. The number of sexual partners an individual has, plays an important role in the
modelling of AIDS. Clearly, the probability of finding an infected partner increases as
the number of different sexual partners increases. A sexual contact with an infected
individual does not guarantee transmission of the HIV due to the low probability of
transmission per sexual act. Hence, the number of different partners a susceptible
individual has is not the sole determining factor in the spread of AIDS. Frequency
of sexual contacts with the same partner also has a bearing on disease transmission.
The type of sexual contact may also contribute to the probability of infection. Hy-
man and Stanley [32] consider more in depth analysis of sexual contact types. See
also [51] for a survey of sexual contact types. To date, anal receptive sex has the
highest probability of transmission of the virus. This is probably the reason for the
rapid spread of disease in the homosexual population. Further, there is a difference
in transmission probabilities between receptive and insertive sexual contacts. Now,
of course, not everyone behaves alike in sexual behaviour. For instance, most mar-
ried individuals do not behave promiscuously, (although there are exceptions to every
rule), and hence married couples are at less 'risk' of contracting the disease than other
sexually active individuals. This leads to the common modelling technique of divid-
ing the population into various risk groups. A risk group contains individuals who
have similar behaviours, and whose members tend to interact primarily within their
group. Those individuals who have sexual behaviours which invite infection, would be
considered as high risk individuals. Examples of high risk individuals would include
homosexuals and prostitutes. Many young adults, those being highly sexually active,
would fall in a moderately high risk group. High risk individuals are more likely to

contract the disease, than those individuals who are more conscientious about safe sexual practices. Safe sexual practices include the use of prophylactics, nonoxynol-9, or complete abstinence. The higher the risk level the faster the susceptible population becomes saturated since high risk groups tend to have less members than low risk groups. Once a risk group is saturated, that is, once a large proportion of the individuals in that risk group become infected, there is a tendency to pass the disease to lower risk groups. Since in the lower risk groups safer behaviour is practiced, and since the number of individuals is greater, the time to saturation is longer, and hence the slower the spread of disease to lower risk groups. For a more detailed look at saturation issues refer to [18].[3]

Sexual activity is a primary means of disease transmission but is not the sole means. Intravenous drug use has been found to be a major cause for concern. Often, IV drug users will share unsterilized needles, (this is common in shooting galleries) and since blood products are a medium for transmission of the HIV, then these drug users are at risk. Many prostitutes, and heterosexuals are drug users and hence there is great concern for the spread of disease in the heterosexual population. Considering this, at some point we must consider the interactions between various risk behaviours and the spread of AIDS. These considerations, of course, would make models very complicated and for the most part, we are not in a position to examine these combinations. Although much research and headway has been made since the first AIDS case was diagnosed, much effort is still required in understanding the basics of the AIDS epidemic.

Other social contributing factors, (see [32] and [52]), in the spread of AIDS and HIV infection include age, population density, geographical factors, ethnicity and social groups, and increased probability of infection due to other diseases. Other means of transmitting the disease is through vertical transmission (from mother to child, before or during birth) or through accidental exposure to blood or blood products by health care workers. Prior to the development of the enzyme-linked immunosorbent assay (ELISA), which accurately detects HIV antibodies, and prerequisite blood screening of blood donors, hemophiliacs were at a great risk of contracting the disease

---

[3]The concept of saturation was introduced by Hethcote and Yorke [28] in 1984.

through frequent blood transfusions.

Biological factors are more difficult to control than demographic variables. The greatest biological contribution to the annihilation of AIDS would be the development of a vaccine. However, this is unlikely to be obtained in the near future.

In modelling the AIDS epidemic we must consider the probability of trans- mission of the HIV during a sexual act or other risk behaviour. The probability of transmission of the HIV is considered by some to be within the range 0.1 to 0.2, (see [2]). Others, see [32], say that the probability of transmission per sexual act is less than 0.01.

Inherent in the AIDS epidemic is the long incubation period. Current esti- mates, (see [2], [44] and [32]), of the length of the incubation period are around an average of 7 to 8 years. These estimates are getting larger in value as time progresses and more data is collected. It is hard to determine an accurate average incubation period owing to the relative newness of the disease and the lack of time available to undertake long-term studies. To further complicate the modelling of AIDS, the infec- tiousness of individuals varies during the incubation period. According to Hyman and Stanley [32], and their references therein, there is a short period of high infectivity immediately after infection, followed by a lengthy period of low infectivity for the majority of the incubation period, with another period of high infectivity prior to a full-blown AIDS diagnosis. This variability in infectivity creates another complicated dimension in the study of AIDS.

No disease to date, has received as much attention as AIDS. Many significant individuals have devoted much of their recent years to the modelling of the AIDS epidemic. For instance, Anderson *et al.* [7] introduced one of the earlier models of the AIDS epidemic. It is upon this model that many more recent models, including those presented in this thesis, were developed. Others, such as the Los Alamos group, [48], [16], [32], [31], [18], have taken different approaches but have done so by extending the risk models of Anderson *et al.* (see also [2], [6], [5], and [42]). Anderson *et al.* have assumed exponential growth in their modelling efforts while the Los Alamos group conclude that the growth is more likely to be cubic. Observed data indicates [18] that the growth is actually cubic but early in the epidemic an exponential growth

is appropriate.

Carlos Castillo-Chavez [11] presents a review of recent modelling efforts paying particular attention to the role of long incubation periods, the effects of partnership dynamics, and the effects of multiple sexual partners. There has been much activity in studying the effects of long periods of infectivity. These include the work of Castillo-Chavez *et al.* [12], [13], [14], where a series of models are presented, allowing for long periods of infectivity while accounting for various risk groups. Thieme *et al.* [49] consider the role of variable infectivity in the spread of the HIV. The issue of risk groups is important to understand in our plight to end the AIDS epidemic. Groups such as Blythe *et al.* [9], consider like-with-like mixing in creating appropriate risk groups. Castillo-Chavez *et al.* [15] consider age structure, proportionate mixing and cross-immunity in their 1989 paper. Not necessarily restricted to the AIDS epidemic is the approach taken by Hethcote and van Ark [27], in which they consider heterogeneous population mixing. Other significant papers include those by Jacquez *et al.* [33], [34], Koopman *et al.* [38], and Sattenspiel and Simon [45].

Common to most models is the notion of a reproductive number. If this number is below one, then an epidemic will be avoided. The problem remains, what interventions are necessary to reduce the spread of disease, or that is, what must we do to bring the reproductive number below one? As we have little control over biological aspects, we must focus on behavioural factors. For instance, reducing the number of unsafe sexual practices would aid in eliminating this deadly disease. Education plays a major role here. As more individuals become aware of the risks involved, the larger the proportion of individuals practicing safe sex or other low risk behaviours, and hence there is a slow down in the growth of infected individuals. A free supply of IV needles is offered in some places to decrease the risk to drug users. Prophylactics are now being made more accessible in an attempt to promote safer sexual practices. Another potential means of reducing the spread of AIDS is the practice of HIV antibody testing. Already, all blood donors are screened for the HIV, and individuals are notified if the test results are seropositive. The study of the effects of HIV testing is an area where little research has been done. McCusker *et al.* [43] assess the effects of HIV antibody testing on future behaviours. They were

able to conclude that testing did have an affect on future behaviours, but that these conclusions could not be generalized outside of the cohort being studied. Lyter *et al.* [41] examined the desire of homosexual and bisexual men to know the results of HIV antibody testing. They concluded that demographic differences were evident between those who wanted their results and those who did not want their results, especially between ethnicity, age and educational levels. Judson *et al.* [35], have looked at the effect of the AIDS epidemic on public health policy. They consider a number of important issues, including educational target groups, control regulations and laws, confidentiality of HIV testing, and need for constant re-evaluation, as additional data is collected.

An excellent way of gaining more information about the dynamics of AIDS is to perform HIV antibody tests. More accurate estimates of numbers infected, trends of the disease, infectivity levels, and other important factors can be derived with these tests. Further, knowledge of seropositive results would help to encourage infected individuals to practice safer sex. However, lack of confidentiality of test results could deter individuals from becoming tested. The imposition of laws requiring individuals to be tested, and legal repercussions enforcing responsible behaviour may be effective but may be considered an infringement on our freedom. Laws enforcing responsible sexual behaviour may need to be imposed on those individuals who insist on behaving irresponsibly. Regarding this idea, we need more data on proportions of individuals behaving responsibly or irresponsibly.

Data collection is a major problem in controlling the AIDS epidemic. Without data, parameter estimations will be inaccurate, future trends can not accurately be predicted, and modelling in general is less effective. Modelling, however, does provide us with recognition of many of the necessary areas that require data collection. In order to most effectively aim educational attempts, we must know more about finding the target groups most likely to benefit from certain types of intervention, or what information is required, or what educational techniques are most beneficial.

1.4 Outline

In this thesis we consider the effects of HIV antibody testing in a homosexual population in conjunction with educational or other persuasive techniques to encourage responsible sexual behaviour of tested infected individuals, in reducing the spread of AIDS. We also consider some confidentiality and legal issues. The models being used are purposely simplistic in nature. Since little research has been done in this area, we require a general indication of testing significance. The models in this thesis are composed of nonlinear differential equations. Local stability properties are examined for all models, and global results are obtained in many cases. In Chapter 2 we introduce and interpret the general model. In Chapter 3 we introduce and analyze the effects of instantaneous testing, by examining the SPA, SQA, and SQPA models. Tested infected individuals either behave responsibly or irresponsibly. We are able to consider the effects of these behaviours on the outcome of the disease, prior to introducing the partial testing proportions used in Chapter 4. Chapter 4 analyzes the most realistic models in this thesis. Here we look at partial testing proportions in combination with responsible or irresponsible sexual behaviours. In Chapter 5 we provide a comparison and discussion of the models presented in this thesis. The appendices include most of the supporting calculations and proofs.

Chapter 2

THE MODELS

The main purpose of this paper is to introduce and analyze models of the AIDS epidemic with emphasis on the importance and effectiveness of testing for HIV positivity, with respect to curtailing the spread of the disease. It is important to note that we are assuming that the procedures for testing seropositivity are very accurate and that we are not studying the effectiveness of the tests for determining whether or not a person who tests positive actually is HIV positive. If testing is confidential, then more people are tested and hence there are fewer untested infectives to spread the disease unwaringly. However, testing for the HIV does not cure an individual. These tested individuals will either behave responsibly by practicing safe sexual behaviours or by abstaining from sexual activity, or will behave irresponsibly, and not use preventive techniques to reduce the spread of AIDS. As the number of individuals tested increases, the greater the number of responsible or irresponsible tested persons. If test results are not confidential between doctor and patient, then there will be fewer individuals tested, since there may be a fear of quarantine, loss of job security, negative social implications, or imposition of laws involving penalties for purposeful transmission. If there are fewer tested infecteds, then there will be fewer knowingly irresponsible infected persons. On the other hand, there will be more sexually responsible individuals due to possible repercussions for irresponsible behaviour. There is much controversy as to whether test result confidentiality should

be honoured between doctor and patient or whether laws should be created that would make it compulsory for doctors to report their patients positivity in certain situations. This selective confidentiality poses a problem in itself. If some infected individuals are reported and others not, then attempts at collecting accurate data would be in vain. To make any firm conclusions about the effect of confidentiality on the course of the epidemic would require data giving the probability of being a responsible individual rather than an irresponsible one, under voluntary and compulsory conditions, and the probability of being tested where confidentiality is practiced versus where lack of confidentiality is practiced. Further, a solution to achieve adequate data collection would need to be suggested.

Using one basic model and some variations on it, we are able to analyze many significant factors of HIV testing. The models are purposely simple to reduce unnecessary complexity in analysis, but complex enough to provide some information to indicate general trends and potential remedies or reductions in the spread of AIDS. However, our results are preliminary, since the models would require considerable refining before the predictions should be used by decision makers.

A schematic diagram providing a general framework for the flow of activity through the system, taking testing into account is displayed in figure 2.1.

The population under study is a sexually active homogeneous population that very closely resembles a homosexual population. [1] We subdivide the population into five disjoint categories:

1.) Susceptibles $S(t)$,

2.) Infecteds, not tested $I(t)$,

3.) Infecteds, tested, sexually responsible $P(t)$,

4.) Infecteds, tested, sexually irresponsible $Q(t)$,

5.) Full-blown AIDS $A(t)$.

The susceptible class, denoted by $S(t)$ represents those individuals that are

---

[1] We consider sexual activity as the medium of HIV infection but this is not necessarily required in the models presented in this paper. We do require a homogeneous population, but this may be any homogeneous population. For example, a population of IV drug users is also a homogeneous population. Since sexual activity is the major cause of the spread of AIDS in society today, we choose this to be our risk activity under study.

Figure 2.1: Schematic Diagram of Partial Testing: SIQPA
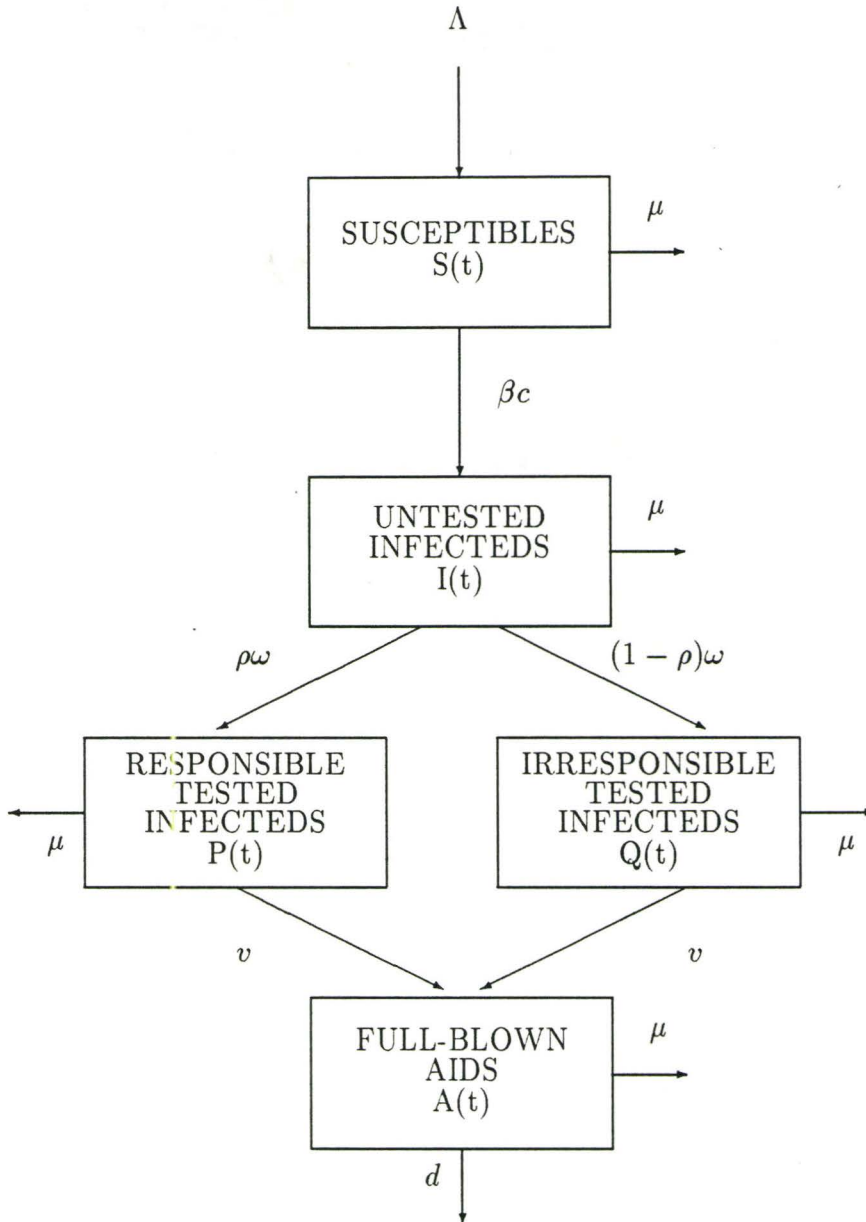
not infected but are capable of contracting the HIV under appropriate conditions. One enters the susceptible population upon one's first sexual act. The number of individuals entering the susceptible population is represented by a constant rate Λ. The assumption that Λ is constant is not too unrealistic, since the population we are considering is a homosexual population which is unlikely to produce offspring. Further, even if the population were not homosexual, any offspring, would take a considerable amount of time to mature into a sexually active individual. Individuals leave the susceptible population and enter the infected population through sexual contact with members of the infected class at a rate βc, where β represents the transmission probability and c represents the average number of sexual contacts per individual in S. We assume the law of mass action in that the susceptibles interact with the infecteds at a rate proportional to the product of the number of infecteds and susceptibles.

We have subdivided the infected population into three categories using responsibility (or lack of responsibility) in sexual activity and HIV testing as the factors that determine to which class an individual belongs. $I(t)$ represents those individuals who are infected with the HIV but have not yet been tested positive. A simplifying assumption in our models is that the latency period, in other words, the period between infection and presence of HIV antibodies in the blood, is insignificant, so that if an individual has been infected, then testing for antibodies will indicate this. In fact, data seems to indicate the latency period is less than six months. All current mathematical models of the AIDS epidemic reflect this fact. Individuals in the I(t) class will leave this class as determined by the proportions ω and μ. The proportion of the infecteds in I(t) that are tested at time t is represented by ω. The parameter μ is used in all populations to represent the natural death rate of individuals. Q(t) represents the class of infecteds who have been tested, but have not paid any heed to their condition and remain irresponsible sexually, thus putting susceptibles at risk. The last subdivision of infecteds are those individuals who have knowledge of their infectiousness and behave responsibly, that is, they practice safe sex or are abstinent. We label this class $P(t)$. Finally, we assume that all infecteds eventually develop AIDS at a rate v. Once an individual has developed full-blown AIDS, they become a member of

the class $A(t)$ and they no longer directly contribute to the epidemic. As this class is of interest to us for various reasons, including benefit cost analysis for care of AIDS patients, we consider it in our model.

If sexual activity of infecteds is confined to other infecteds only, then their contribution to the epidemic would be insignificant. But we cannot assume total knowledge in regards to who may or may not be infected. Confidentiality of testing plays a role here since the limited or complete public knowledge of an individuals infectivity would probably provide incentive to refrain from unsafe sexual practices. However, total disclosure could conceivably backfire, putting susceptibles at greater risk from those infecteds who do not know they are infected and hence seek out only those not known to be infected, as their sexual partners. As well, greater confidentiality might encourage more high risk individuals to be tested and thus might result in a smaller $I(t)$ class and a larger responsible $P(t)$ class, due to the migration of newly tested $I(t)$ individuals into the tested classes, thus reducing the spread of the disease. Unfortunately, there is insufficient data available. Moreover, the models do indicate that testing may be a key factor in reducing the incidence of AIDS, and thus data collection on this matter should be undertaken.

The responsible and irresponsible tested infected populations are determined by the proportions ρ and $1 - \rho$, respectively. The parameter ρ stipulates the percentage of tested infecteds who are responsibly sexually active and $1 - \rho$ provides the proportion of tested infecteds who remain sexually irresponsible. Hence, we should consider various alternatives including education, confidentiality issues, and as a last resort legal issues, that would increase the parameter ρ, and therefore decrease the number of tested infected sexually irresponsible individuals contributing to the spread of the disease.

The equations describing our system are:

$$\frac{dS(t)}{dt} = \Lambda - \left(\mu + \frac{\beta c(I(t) + Q(t))}{N(t)}\right) S(t)$$

$$\frac{dI(t)}{dt} = \left(\frac{\beta c(I(t) + Q(t))}{N(t)}\right) S(t) - (\mu + \omega)I(t)$$

$$\frac{dQ(t)}{dt} = (1 - \rho)\omega I(t) - (\mu + v)Q(t)$$

$$\frac{dP(t)}{dt} = \rho\omega I(t) - (\mu + v)P(t) \qquad (2.1)$$

$$\frac{dA(t)}{dt} = v(P(t) + Q(t)) - (\mu + d)A(t)$$

$$N(t) = S(t) + I(t) + Q(t) + P(t) + A(t)$$

where

$$S(0) = S_o > 0,$$
$$I(0) = I_o > 0,$$
$$Q(0) = Q_o \geq 0,$$
$$P(0) = P_o \geq 0, \text{ and}$$
$$A(0) = A_o \geq 0.$$

It follows that

$$\frac{dN(t)}{dt} = \frac{dS(t)}{dt} + \frac{dI(t)}{dt} + \frac{dQ(t)}{dt} + \frac{dP(t)}{dt} + \frac{dA(t)}{dt}$$
$$= \Lambda - \mu N(t) - dA(t).$$

This model is a generalization of the model in Anderson *et al.* [7]. Anderson *et al.* develop a continuous-infection model that includes a susceptible class, an infected class, a full-blown AIDS class and a recovered class. Their model was developed at a very early stage in the study of AIDS. We make the simplifying assumption that all individuals who become infected will eventually die of AIDS related causes and so we do not include a recovered class in our models. According to current data, a very large proportion, if not all of the infected individuals will die from AIDS related causes. Otherwise. Anderson *et al.* have a good introductory approach to modelling the AIDS epidemic. We have refined their model since we subdivide the infected individuals into the classes $I(t)$, $Q(t)$, and $P(t)$. Further, we have excluded their

removed class $Z(t)$, as it is no longer appropriate. The use of the law of mass action to model interactions between members of the different populations is retained for our model, as are the general dynamical qualities of the disease. This format allows a good introductory look at the dynamics of the AIDS epidemic as it relates to HIV testing.

The model we developed is well-posed in the sense that all solutions remain nonnegative and bounded. Refer to Appendix G for the proof.

Before further analyzing the model in equations 2.1, we look at a few variations of this model.

Chapter 3

INSTANTANEOUS TESTING

3.1 Preliminaries

All the models included in this chapter are derived, under certain simplifying assumptions, from our original system given in equations 2.1. We include a series of less mathematically complex systems in order to gain some insight into the dynamical behaviour of the AIDS disease in certain extreme cases. The models in this chapter are not mathematical subsystems of the SIQPA model. The SIQPA model, is not properly defined when $\omega = 1$ or $\omega = 0$. If $\omega = 1$, then $I'(t) = \frac{\beta c(I+Q)}{N}S - (u+1)I$, which implies that more than one hundred percent of $I(t)$ is being removed at any time. If $\omega = 0$, then this implies that $Q(t) \equiv 0$ and $P(t) \equiv 0$, so that the untested infecteds all must die of natural causes, and not AIDS related diseases. Thus, we have decided, that in order to get a reasonable grasp of the effects of HIV antibody testing we would examine the series of models using the assumption that the untested population, $I(t)$ is non-existent. In the first sub-model presented and analyzed in this thesis we disregard the non-tested infected class $I(t)$. Thus we make the simplifying assumption that testing is instantaneous, i.e., the instant a person from the susceptible class contracts the HIV, he becomes a member of the class $P(t)$ or $Q(t)$ with the respective probabilities ρ and $1 - \rho$. All models included and analyzed in this chapter employ the instantaneous testing assumption. Thus, the models are all based on

24

the simplified model, denoted in figure 3.2, a schematic diagram representing general movement between classes.

The four dimensional system, obtained by making the assumption of instantaneous testing, is:

$$\frac{dS(t)}{dt} = \Lambda - \left(\mu + \frac{\beta c Q(t)}{N(t)}\right) S(t)$$

$$\frac{dQ(t)}{dt} = (1 - \rho)\frac{\beta c Q(t) S(t)}{N(t)} - (\mu + v)Q(t)$$

$$\frac{dP(t)}{dt} = \rho\frac{\beta c Q(t) S(t)}{N(t)} - (\mu + v)P(t) \tag{3.1}$$

$$\frac{dA(t)}{dt} = v(P(t) + Q(t)) - (\mu + d)A(t)$$

$$N(t) = S(t) + Q(t) + P(t) + A(t)$$

with $\quad S_o > 0, Q_o > 0, P_o > 0$ and $A_o \geq 0.$

Therefore,

$$\frac{dN(t)}{dt} = \Lambda - \mu N(t) - dA(t).$$

The assumption of instantaneous testing is not realistic. However, understanding this extreme case will prove useful for interpreting the effects of testing on the AIDS epidemic. The value of ρ indicates the proportion of the infected population who are acting responsibly, and who have not yet developed full-blown AIDS, and $1 - \rho$ indicates the proportion that behave irresponsibly. We consider $0 \leq \rho \leq 1$. Since ρ is the coefficient representing the proportion of new individuals entering $P(t)$, of those who were tested from $I(t)$, and $1 - \rho$ the equivalent coefficient for $Q(t)$, then an increase in the value of ρ will increase the number of individuals entering $P(t)$. Similarily a decrease in ρ results in a decrease in the number of persons entering $P(t)$. Due to the fact that $\rho + (1 - \rho) = 1$, as the number of individuals entering $P(t)$

Figure 3.2: Schematic Diagram of Instantaneous Testing: SQPA
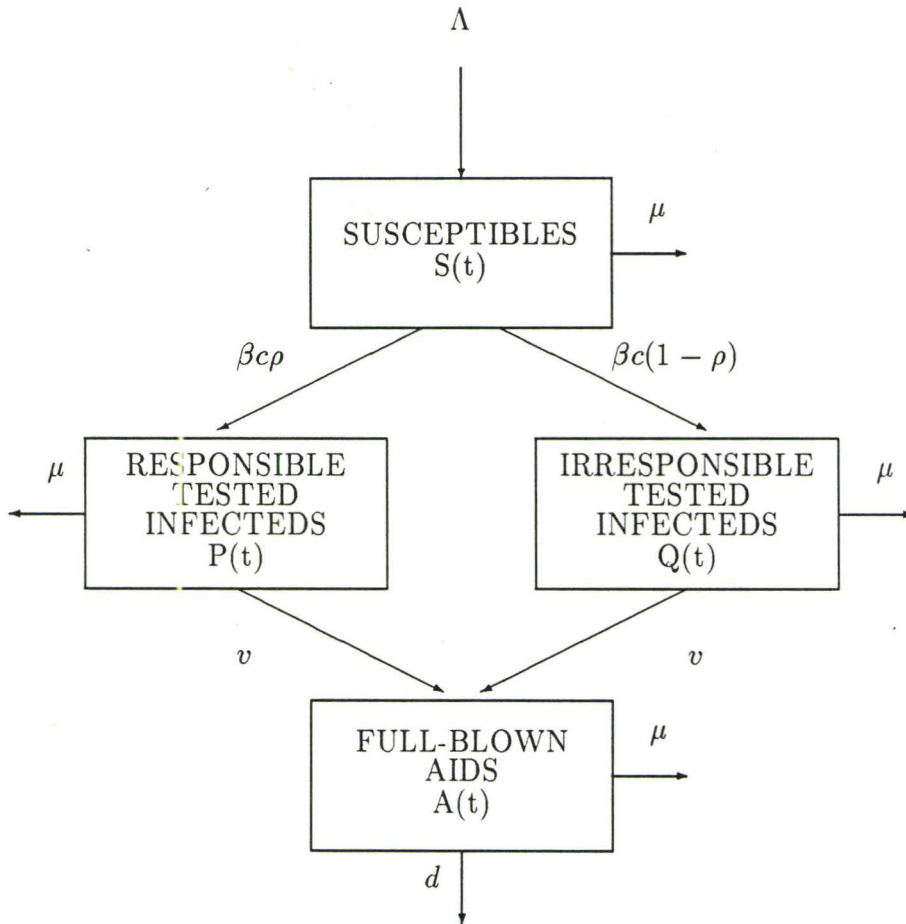
increases, the number of persons entering $Q(t)$ necessarily decreases and vice versa. Hence ultimately for a decrease in the sexually irresponsible infected population we would like to attain a large value for ρ. This may be partially achieved using different educational techniques aimed at the tested infecteds, or perhaps legal repercussions as incentives for tested infecteds to refrain from sexual activity.

We start our analysis by looking at the two extreme cases, $\rho = 1$ and $\rho = 0$. We consider the case where $\rho = 1$ first. If $\rho = 1$, then we can interpret the model as indicative of no infected sexually irresponsible individuals in the system. Hence, we would expect the disease to eventually die out, since testing is 100% effective in eliminating the source of infection. Disease elimination, of course, is the optimal situation. However, it would be very difficult to obtain. There are many factors to be taken into consideration when striving for this goal, such as the phenomenal cost involved in testing everyone,[1] as well as the task of converting peoples moral values, or imposing a quarantine on those individuals infected with HIV. Further, it is not an easy task to monitor an individual's sexual behaviours, or to collect appropriate data due to the private nature of sexual activity. Fortunately, we need not attain this state of totally responsible tested infected individuals in order for the disease to die out. We shall see in the analyses of the various models that there are scenarios in which the models predict that the disease will die out. The case where $\rho = 0$ is presented later. In this latter case the infected population consists of only those individuals who are not willing to behave responsibly.

3.2 Testing is 100% Effective: SPA and SPA$_\epsilon$

3.2.1 The SPA Model

The schematic representation for the SPA system, (i.e. when $\rho = 1$), is given

---

[1] The ELISA is a relatively low cost test, but if we consider the organization required to test the entire population, the cost can become quite large.

in figure 3.3, and the system of equations becomes:

$$\frac{dS(t)}{dt} = \Lambda - \mu S$$

$$\frac{dP(t)}{dt} = -(\mu + v)P$$

$$\frac{dA(t)}{dt} = vP - (\mu + d)A \qquad (3.2)$$

$$N(t) = S(t) + P(t) + A(t)$$

with $\quad S(0) = S_o > 0, \quad P(0) = P_o > 0 \quad$ and $A(0) = A_o \geq 0$.

Thus,

$$\frac{dN(t)}{dt} = \Lambda - \mu N(t) - dA(t).$$

Note that $Q(t)$ is not considered in this model since when $\rho = 1$, and $Q_o = 0$, no individuals enter the class $Q(t)$, and hence, $Q \equiv 0$. If there are any members in $Q(t)$ at $t = 0$, in other words if $Q_0 > 0$, these individuals will die out exponentially, so again we can consider $Q \equiv 0$.

Only one equilibrium point exists:

$$\begin{pmatrix} \hat{S} \\ \hat{P} \\ \hat{A} \end{pmatrix} = \begin{pmatrix} \frac{\Lambda}{\mu} \\ 0 \\ 0 \end{pmatrix} \qquad (3.3)$$

The Jacobian for this system, at the fixed point, is given by

$$J = \begin{pmatrix} -\mu & 0 & 0 \\ 0 & -(\mu + v) & 0 \\ 0 & v & -(\mu + d) \end{pmatrix} \qquad (3.4)$$

Figure 3.3: Schematic Diagram for 100% Effective Testing: SPA
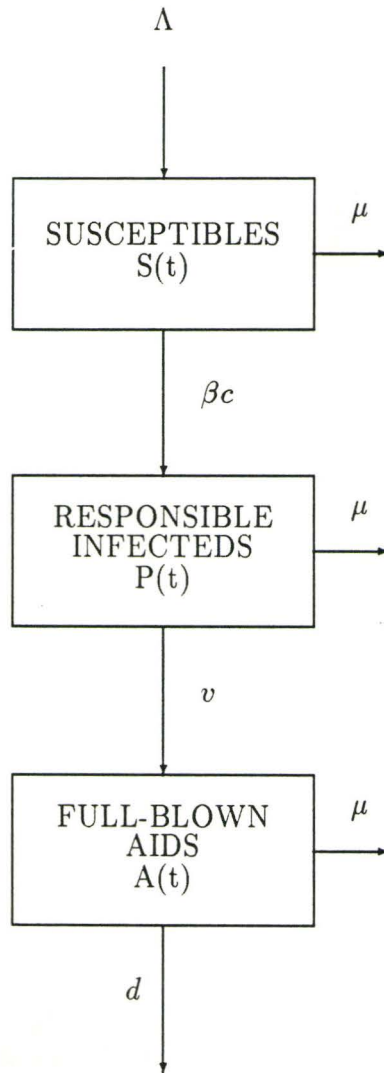
The Jacobian is a triangular matrix and hence the eigenvalues are the diagonal elements:

$$
\begin{aligned}
\lambda_1 &= -\mu \\
\lambda_2 &= -(\mu + v) \\
\lambda_3 &= -(\mu + d).
\end{aligned}
\tag{3.5}
$$

Since the eigenvalues are real and all negative and since system 3.2 is linear, $(\frac{\Lambda}{\mu}, 0, 0)$ is a globally asymptotically stable node. In fact, since the equations in 3.2 are linear, we are able to solve the system explicitly.

$$
S(t) = \frac{\Lambda}{\mu} + \left(S_o - \frac{\Lambda}{\mu} \right) e^{-\mu t}
$$

$$
P(t) = P_o e^{-(\mu + v)t}
$$

$$
A(t) = \frac{v P_o}{d - v} e^{-(\mu + v)t} + \left(A_o - \frac{v P_o}{d - v} \right) e^{-(\mu + d)t}
$$

The results we obtained are intuitively clear. If there are no sexually active irresponsible infected individuals then the disease will not spread, in fact, the disease will die out exponentially.

The assumption used in this section, i.e. $\rho = 1$, is, as mentioned previously, unrealistic. Only if we were to look at a subpopulation would we possibly have this situation arise. That is, there may be a group of individuals living in a certain community or household where sexual activity by infecteds is against moral values or is contradictory to some other factor. Even so, there still exists the possibility of extraneous sources of infection, such as IV drug use, transfusions or accidental exposures to the virus. Thus we alter our model to include these sources.

3.2.2 Extraneous Sources: SPA$_\epsilon$

The following model takes a look at the introduction of an extraneous input that is not due to a sexual transmission of the virus HIV. We label this constant rate of input ϵ. We are interested in looking at what effects $\epsilon > 0$ has in the long term on the AIDS epidemic. Our revised model is then

$$\frac{dS(t)}{dt} = \Lambda - (\mu + \epsilon)S(t)$$

$$\frac{dP(t)}{dt} = \epsilon S(t) - (\mu + v)P(t) \qquad (3.6)$$

$$\frac{dA(t)}{dt} = vP(t) - (\mu + d)A(t)$$

$$N(t) = S(t) + P(t) + A(t)$$

with $S_o > 0, \ P_o \geq 0, \text{ and } A_o \geq 0.$

Therefore,

$$\frac{dN(t)}{dt} = \Lambda - \mu N(t) - dA(t).$$

Note that we still assume that $Q(t) \equiv 0$, as we still have $\rho = 1$.

To determine equilibria we set $S'(t) = P'(t) = A'(t) = N'(t) = 0$. There is one fixed point

$$\begin{pmatrix} \bar{S} \\ \bar{P} \\ \bar{A} \end{pmatrix} = \begin{pmatrix} \frac{\Lambda}{\mu+\epsilon} \\ \frac{\epsilon\Lambda}{(\mu+\epsilon)(\mu+v)} \\ \frac{\epsilon v\Lambda}{(\mu+\epsilon)(\mu+v)(\mu+d)} \end{pmatrix}.$$

Further,

$$\bar{N} = \Lambda \left(\frac{(\mu + v)(\mu + d) + \epsilon(\mu + v + d)}{(\mu + \epsilon)(\mu + v)(\mu + d)} \right).$$

The introduction of epsilon results in an endemic equilibrium, rather than a disease free equilibrium.

The Jacobian is:

$$J_{(\bar{S},\bar{P},\bar{A})} = \begin{pmatrix} -(\mu + \epsilon) & 0 & 0 \\ \epsilon & -(\mu + v) & 0 \\ 0 & v & -(\mu + d) \end{pmatrix}$$

Again we have a triangular matrix, with eigenvalues given by the diagonal elements:

$$\lambda_1 = -(\mu + \epsilon)$$
$$\lambda_2 = -(\mu + v)$$
$$\lambda_3 = -(\mu + d)$$

As before, the eigenvalues are real and all negative and the revised system is still linear, hence the endemic fixed point is a globally asymptotically stable node. The explicit solutions are calculated in Appendix A. We obtained:

$$S(t) = \frac{\Lambda}{(\mu + \epsilon)} + \left(S_o - \frac{\Lambda}{\mu + \epsilon}\right) e^{-(\mu+\epsilon)t},$$

$$P(t) = \frac{\epsilon \Lambda}{(\mu + \epsilon)(\mu + v)} + \frac{\epsilon}{(v - \epsilon)}\left(S_o - \frac{\Lambda}{\mu + \epsilon}\right) e^{-(\mu+\epsilon)t}$$
$$+ \left(P_o - \frac{\epsilon \Lambda}{(\mu + \epsilon)(\mu + v)}\right) e^{-(\mu+v)t}$$
$$- \frac{\epsilon e^{-(\mu+v)t}}{(v - \epsilon)}\left(S_o - \frac{\Lambda}{\mu + \epsilon}\right).$$

$$A(t) = \frac{v\epsilon\Lambda}{(\mu + d)(\mu + \epsilon)(\mu + v)}$$
$$+ \left(S_o - \frac{\Lambda}{\mu + \epsilon}\right)\left(\frac{\epsilon}{(v - \epsilon)(d - \epsilon)}\right) e^{-(\mu+\epsilon)t}$$
$$+ \left(P_o - \frac{\epsilon\Lambda}{(\mu + \epsilon)(\mu + v)} - \frac{\epsilon}{(v - \epsilon)}\left(S_o - \frac{\Lambda}{\mu + \epsilon}\right)\right)\frac{e^{-(\mu+v)t}}{(d - v)}$$

$$+ \left(A_o - \frac{v\epsilon\Lambda}{(\mu + d)(\mu + \epsilon)(\mu + v)} \right) e^{-(\mu+d)t}$$

$$- \left(S_o - \frac{\Lambda}{\mu + \epsilon} \right) \left(\frac{\epsilon}{(v - \epsilon)(d - \epsilon)} \right) e^{-(\mu+d)t}$$

$$- \left(P_o + \frac{\epsilon\Lambda}{(\mu + \epsilon)(\mu + v)} + \frac{\epsilon}{(v - \epsilon)} \left(S_o - \frac{\Lambda}{\mu + \epsilon} \right) \right) \frac{e^{-(\mu+d)t}}{(d - v)}.$$

For $\epsilon > 0$, $\bar{S}$ is a decreasing function of ϵ whereas $\bar{P}$ and $\bar{A}$ are increasing functions of ϵ. From the endemic equilibrium we can deduce that the susceptible population will exceed the class of infecteds in $P(t)$, if $\mu + v > \epsilon$, that is, if the removal rate $\mu + v$ from $P(t)$ is greater than the rate of new infections ϵ. It is interesting to note that the full-blown AIDS class never exceeds the infected class, but will exceed the susceptible class if $\epsilon > \frac{(\mu+v)(\mu+d)}{v}$ since $\mu + d > v$ always. Further, we note that $\bar{P} + \bar{A} = \frac{\epsilon\Lambda(\mu+v+d)}{(\mu+v)(\mu+\epsilon)(\mu+d)}$. Thus the uninfected population $S(t)$ will exceed the total infected population if $\epsilon < \frac{(\mu+v)(\mu+d)}{\mu+v+d}$. A cost analysis of health care given ratios of healthy to infected individuals would prove interesting at this point, but we are not presently equipped to carry out an analysis of this type.

The results in this section indicate that controlling the amount of infectivity caused by external sources is important if we wish to control the spread of the disease.

3.3 Testing is 100% Inneffective: SQA

3.3.1 Local Asymptotic Stability

The SQA Model

The following model represents the extreme case where $\rho = 0$. In this case, all those who are tested and know they are infected continue to behave in a sexually irresponsible manner. Thus, testing makes no contribution to the reduction of the spread of the HIV, and in effect $P(t) \equiv 0$.

A schematic diagram of this system is provided in figure 3.4. The system of equations is given by:

$$\frac{dS(t)}{dt} = \Lambda - \left(\mu + \frac{\beta c Q(t)}{N(t)}\right) S(t),$$

$$\frac{dQ(t)}{dt} = \frac{\beta c Q(t) S(t)}{N(t)} - (\mu + v)Q(t),$$

$$\frac{dA(t)}{dt} = v Q(t) - (\mu + d)A(t), \tag{3.7}$$

$$N(t) = S(t) + Q(t) + A(t),$$

with $\quad S_o > 0, \ Q_o > 0, \text{ and } A_o \geq 0.$

Hence,

$$\frac{dN(t)}{dt} = \Lambda - \mu N(t) - dA(t),$$

Due to the law of mass action, these equations are nonlinear. This makes finding the explicit solutions of the system unlikely. Instead, we use linearization and stability analysis to determine the qualitative behaviour of the system. This will enable us to obtain local stability properties.

Equilibria

This system has two fixed points. Appendix B contains the supporting calculations for determining these fixed points. The first is,

$$\begin{pmatrix} \hat{S} \\ \hat{Q} \\ \hat{A} \end{pmatrix} = \begin{pmatrix} \frac{\Lambda}{\mu} \\ 0 \\ 0 \end{pmatrix}.$$

This fixed point represents a disease free situation, that is, the disease will die out. We call this the disease free equilibrium. The existence of the second equilibrium

Figure 3.4: Schematic Diagram for 100% Ineffective Testing

Λ
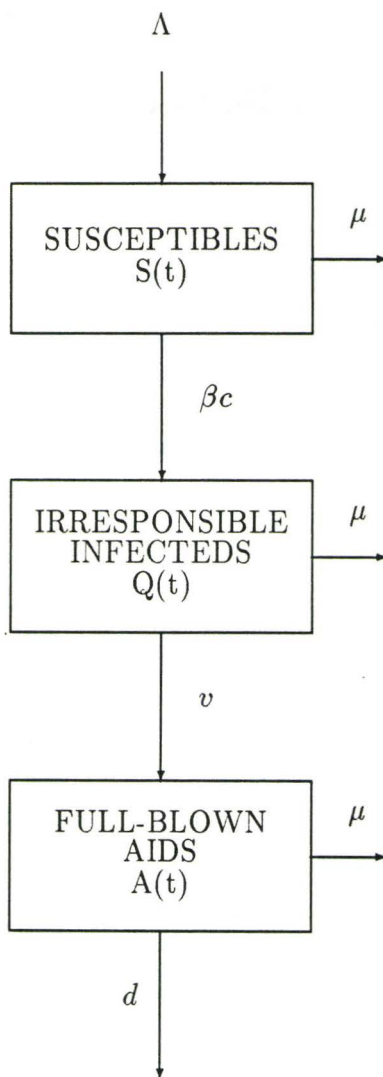
indicates that there is a possibility of an endemic outbreak of HIV. This equilibrium point is given by:

$$
\begin{pmatrix} \bar{S} \\ \bar{Q} \\ \bar{A} \end{pmatrix} = \begin{pmatrix} \frac{\Lambda(\mu+d+v)}{\beta c(\mu+d)-vd} \\ \left(\frac{\Lambda}{\mu+v}\right)\left(\frac{(\beta c-(\mu+v))(\mu+d)}{\beta c(\mu+d)-vd}\right) \\ \left(\frac{\Lambda v}{\mu+v}\right)\left(\frac{\beta c-(\mu+v)}{\beta c(\mu+d)-vd}\right) \end{pmatrix},
$$

where

$$
\bar{N} = \bar{S} + \bar{Q} + \bar{A} = \frac{\Lambda\beta c(\mu+v+d)}{(\mu+v)(\beta c(\mu+d)-vd)}.
$$

Existence of the endemic equilibrium in the positive cone, is guaranteed provided all the components of the endemic equilibrium are positive. This is satisfied if $\bar{Q} > 0$ or equivalently if $\beta c > \mu + v$. Clearly $\mu + v > \frac{vd}{\mu+d}$. Thus $\beta c > \mu + v$ implies that $\beta c(\mu+d) - vd > 0$, which shows the positivity of $\bar{N}$.

In the worst case, letting βc tend to infinity:

$$
\lim_{\beta c \to \infty} \begin{pmatrix} \bar{S} \\ \bar{Q} \\ \bar{A} \end{pmatrix} = \begin{pmatrix} 0 \\ \frac{\Lambda}{\mu+v} \\ \frac{\Lambda v}{(\mu+v)(\mu+d)} \end{pmatrix}.
$$

Note that $\bar{S}$ is a decreasing function of βc whereas $\bar{Q}$ and $\bar{A}$ are increasing functions of βc. Clearly then, the limit as βc tends to 0 is the disease free equilibrium.

Local Stability Analysis

Since the eigenvalues corresponding to the disease free equilibrium are negative and all real by the calculations in Appendix B, the disease free equilibrium is a locally asymptotically stable node. Alternatively, the disease free equilibrium is locally asymptotically stable if the endemic equilibrium does not exist in the nonnegative cone (i.e. $\beta c < \mu + v$), and is an unstable saddle point if the endemic equilibrium exists, that is, if $\beta c > \mu + v$.

The fixed point $(\bar{S}, \bar{Q}, \bar{A})$ exists in the nonnegative cone, and by the Routh Hurwitz criteria (H.2), is locally asymptotically stable provided $\beta c > \mu + v$. The

supporting calculations can be found in Appendix B. In fact, as soon as the endemic equilibrium exists it is locally asymptotically stable.

The reproductive number R_o, for this model is given by

$$R_o = \frac{\beta c}{\mu + v}.$$

If $R_o < 1$ the disease will die out asymptotically. If $R_o > 1$ an epidemic will occur. Ideally, we would like to reduce βc, in an attempt to reach the disease free status. We have no control, at least not in the near future, over the probability of transmission parameter β, as it is biological in nature. However, by instructing people to use prophylactics and/or other safe sex practices, as well as approaching issues of morality and promiscuity we can expect a drop in c, thus decreasing the value of R_o.

In summary

EQUILIBRIUM	$\beta c < \mu + v$	$\beta c > \mu + v$
$(\frac{\Lambda}{\mu}, 0, 0)$	Local asymptotic stability	Exists, but is unstable
$(\bar{S}, \bar{Q}, \bar{A})$	Does not exist in the nonnegative cone	Local asymptotic stability

3.3.2 Global Asymptotic Stability

We prove that the disease free equilibrium is globally asymptotically stable whenever it is locally asymptotically stable, that is, when $\beta c < \mu + v$.

Define

$$\mathcal{G} = \{(S, Q, A) \in \Re^3 : S > 0, Q > 0, A > 0\}$$

$$\overline{\mathcal{G}} = \{(S, Q, A) \in \Re^3 : S \geq 0, Q \geq 0, A \geq 0\}$$

Further, define $V : \Re^3 \to \Re$, by

$$V(S, Q, A) = Q.$$

Then V is $C^1(\Re^3)$ and the time derivative

$$\dot{V}(S, Q, A) = Q\left(\frac{\beta cS}{N} - (\mu + v)\right).$$

Thus $\dot{V}(S, Q, A) \leq 0$ on $\mathcal{G}$. Therefore V is a Liapunov function by definition H.5. $\dot{V}$ is equal to 0 if and only if $Q = 0$, since $\beta c < \mu + v$ and $\frac{S}{N} \leq 1$.

We define

$$
\begin{aligned}
\mathcal{E} &= \{(S, Q, A) \in \overline{\mathcal{G}} : \dot{V} = 0\} \\
&= \{(S, Q, A) : Q = 0, S \geq 0, A \geq 0\}.
\end{aligned}
$$

By the LaSalle Extension Theorem (H.6), every bounded solution of system 3.7, and hence every solution by H.1(b), converges to $\mathcal{M}$ where $\mathcal{M}$ is the largest invariant subset of $\mathcal{E}$.

Consider the system obtained if $Q(t) \equiv 0$ in 3.7.

$$
\begin{aligned}
S' &= \Lambda - \mu S \\
A' &= -(\mu + d)A
\end{aligned}
\tag{3.8}
$$

We define the largest invariant subset of $\mathcal{E}$ as

$$\mathcal{M} = \{(S, Q, A) \in \Re_+^3 : Q = 0, S \geq 0, A \geq 0 \text{ and } (S, A) \text{ satisfies } 3.8\}.$$

For every solution of 3.8,

$$S(t) \longrightarrow \frac{\Lambda}{\mu}$$
$$A(t) \longrightarrow 0.$$

Therefore, the point $(\frac{\Lambda}{\mu}, 0, 0)$ is in the omega limit set of every solution of 3.7. But, this point is a locally asymptotically stable critical point and so it must be the only

point in the omega limit set of any solution of 3.7. Hence, the disease free equilibrium is globally asymptotically stable with respect to the solutions initiating in the nonnegative cone provided $\beta c < \mu + v$.

Today's society is experiencing an endemic form of the AIDS disease. Although our model is only representative of a small portion of society it provides us with some insight into the problem. It seems that without interventions, provided the parameters are within a certain range, the disease will tend towards the endemic equilibrium.

We have looked at the four dimensional model with $\rho = 0$ and $\rho = 1$ and have gained some insight into the behaviour of the system in these extreme cases. We now move on to more realistic situations, that is, where $0 < \rho < 1$. Society today is within this range of values for ρ.

3.4 Testing is Partially Effective: SQPA

3.4.1 Local Asymptotic Stability

The SQPA Model

The model in this section represents the situation where there is instantaneous testing and an opportunity to examine the effect of the size of the parameter ρ on the outcome of the AIDS epidemic. Both classes, $P(t)$ and $Q(t)$ will be represented in the total population.

A schematic diagram is provided in figure 3.2 The system of equations is:

$$\frac{dS(t)}{dt} = \Lambda - \left(\mu + \frac{\beta c Q(t)}{N(t)}\right) S(t)$$

$$\frac{dQ(t)}{dt} = (1 - \rho)\frac{\beta c Q(t) S(t)}{N(t)} - (\mu + v)Q(t)$$

$$\frac{dP(t)}{dt} = \rho\frac{\beta c Q(t) S(t)}{N(t)} - (\mu + v)P(t) \tag{3.9}$$

$$\frac{dA(t)}{dt} = v(P(t) + Q(t)) - (\mu + d)A(t)$$

$$N(t) = S(t) + Q(t) + P(t) + A(t)$$

with $\quad S_o > 0, \ Q_o > 0, \ P_o \geq 0, \ \text{and} \ A_o \geq 0.$

Equilibria

The disease free equilibrium is:

$$\begin{pmatrix} \hat{S} \\ \hat{Q} \\ \hat{P} \\ \hat{A} \end{pmatrix} = \begin{pmatrix} \frac{\Lambda}{\mu} \\ 0 \\ 0 \\ 0 \end{pmatrix}.$$

Deriving the endemic equilibrium is somewhat more complex and the supporting calculations can be found in Appendix C. The endemic equilibrium is:

$$\begin{pmatrix} \bar{S} \\ \bar{Q} \\ \bar{P} \\ \bar{A} \end{pmatrix} = \begin{pmatrix} \frac{\Lambda(\mu+v+d)}{(\mu+d)(1-\rho)\beta c - vd} \\ \left(\frac{\Lambda(\mu+d)(1-\rho)}{\mu+v}\right)\left(\frac{(1-\rho)\beta c - (\mu+v)}{(\mu+d)(1-\rho)\beta c - vd}\right) \\ \left(\frac{\Lambda\rho(\mu+d)}{\mu+v}\right)\left(\frac{(1-\rho)\beta c - (\mu+v)}{(\mu+d)(1-\rho)\beta c - vd}\right) \\ \left(\frac{\Lambda v}{\mu+v}\right)\left(\frac{(1-\rho)\beta c - (\mu+v)}{(\mu+d)(1-\rho)\beta c - vd}\right) \end{pmatrix}$$

The endemic equilibrium exists in the positive cone if $\bar{S}, \bar{Q}, \bar{P}$, and $\bar{A}$ are positive. This is satisfied if $\bar{Q} > 0$, or equivalently if $\beta c > \frac{\mu+v}{1-\rho}$. The reproductive number is

$$R_o = \frac{\beta c(1-\rho)}{\mu + v}.$$

As before, if $R_o < 1$ the disease free equilibrium will be obtained. If $R_o > 1$, the disease will flourish.

Local Stability Analysis

In summary the stability properties are:

EQUILIBRIUM	$\beta c < \frac{\mu + v}{1-\rho}$	$\beta c > \frac{\mu + v}{1-\rho}$
$(\hat{S}, \hat{Q}, \hat{P}, \hat{A})$	Local asymptotic stability	Exists, but is unstable
$(\bar{S}, \bar{Q}, \bar{P}, \bar{A})$	Does not exist in the nonnegative cone	Local asymptotic stability

3.4.2 Global Asymptotic Stability

We prove that the disease free equilibrium is globally asymptotically stable whenever it is locally asymptotically stable, that is, when $\beta c < \frac{\mu + v}{1-\rho}$.
Define

$$\mathcal{G} = \{(S, Q, P, A) \in \Re^4 : S > 0, Q > 0, P > 0, A > 0\}$$

$$\overline{\mathcal{G}} = \{(S, Q, P, A) \in \Re^4 : S \geq 0, Q \geq 0, P \geq 0, A \geq 0\}$$

Further, define $V : \Re^4 \to \Re$, by

$$V(S, Q, P, A) = Q.$$

Then V is $C^1(\Re^4)$ and the time derivative

$$\dot{V}(S, Q, P, A) = Q\left(\frac{\beta c S}{N} - \frac{\mu + v}{1 - \rho}\right).$$

Thus $\dot{V}(S, Q, P, A) \leq 0$ on $\mathcal{G}$. Therefore V is a Liapunov function by definition H.5. $\dot{V}$ is equal to 0 if and only if $Q = 0$, since $\beta c < \frac{(\mu + v)}{(1 - \rho)}$.

We define

$$\begin{aligned}
\mathcal{E} &= \{(S,Q,P,A) \in \overline{\mathcal{G}} : \dot{V} = 0\} \\
&= \{(S,Q,P,A) : Q = 0, S \geq 0, P \geq 0, A \geq 0\}.
\end{aligned}$$

By the LaSalle Extension Theorem (H.6), every bounded solution, and hence every solution by H.1(b), of system 3.9 converges to $\mathcal{M}$ where $\mathcal{M}$ is the largest invariant subset of $\mathcal{E}$.

Consider the system obtained if $Q(t) \equiv 0$ in 3.9.

$$\begin{aligned}
S' &= \Lambda - \mu S \\
P' &= -(\mu + v)P \\
A' &= vP - (\mu + d)A
\end{aligned} \tag{3.10}$$

The solutions to 3.10 are:

$$S(t) = \frac{\Lambda}{\mu} + \left(S_o - \frac{\Lambda}{\mu}\right) e^{-\mu t},$$

$$P(t) = vP_o e^{-(\mu+v)t},$$

$$A(t) = \frac{vP_o}{d-v} e^{-(\mu+v)t} + \left(A_o - \frac{vP_o}{d-v}\right) e^{-(\mu+d)t}.$$

We define the largest invariant subset of $\mathcal{E}$ to be:

$$\mathcal{M} = \{(S,Q,P,A) \in \Re_+^4 :$$

$$Q = 0, S \geq 0, P \geq 0, A \geq 0 \text{ and } (S,P,A) \text{ satisfies 3.10}\}.$$

For every solution of 3.10,

$$S(t) \longrightarrow \frac{\Lambda}{\mu},$$

$$P(t) \longrightarrow 0,$$

$$A(t) \longrightarrow 0.$$

Therefore, the point $(\frac{\Lambda}{\mu},0,0,0)$ is in the omega limit set of every solution of 3.9. But, this point is a locally asymptotically stable critical point and so it must be the only point in the omega limit set of any solution of 3.9. Hence, the disease free equilibrium is globally asymptotically stable with respect to the solutions initiating in the nonnegative cone, provided $\beta c < \frac{\mu+v}{1-\rho}$.

Chapter 4

PARTIAL TESTING OF INFECTEDS

4.1 Preliminaries

The models in this chapter present the opportunity to analyze the effects of partial testing for HIV antibodies on the AIDS epidemic by introducing a parameter indicating the proportion of the number of untested infecteds that are tested at time t. We wish to find the minimal range of values for this parameter that will still guarantee eradication of the disease. With the introduction of the option to test different proportions of the population comes an additional infected class, $I(t)$. This class contains those infecteds who are not tested, and hence, are unaware of their seropositivity, but still consider themselves uninfected and so do not change their sexual behaviours. It is from this class that we choose those individuals who are to be tested. We label the parameter indicating the proportion of those infecteds, $I(t)$, who choose to be tested at time t, as ω. The parameter can have values in the range $0 \leq \omega \leq 1$. In the previous chapter, we examined the situation where $\omega = 1$ (and $\omega = 0$, since the dynamics of the SQA model are identical to the SIA model). In this chapter we focus on the more realistic range of values, $0 < \omega < 1$, as applied to the model found in 2.1. Note that we can realistically assume that

44

ω is significantly larger than v. The average incubation period is around seven or eight years, but AIDS related symptoms, such as thrush, yeast infections, and slowly healing wounds begin appearing towards the end of the incubation period. If testing is voluntary, untested infected individuals will probably not consider being tested until these symptoms appear. They most likely will however, be tested prior to developing full-blown AIDS. The parameter v in our models is determined by the inverse of the average length of the incubation period, i.e. $\frac{1}{v} \approx 7$ or 8 years, so $\omega > v$. If testing is mandatory, then depending on public policy and frequency of tests, we can consider $\omega >> v$.

As in the previous chapter, we will look at the extreme cases of responsible behaviourindividually, that is, where $\rho = 0$ and $\rho = 1$. The parameter ρ has no effect on the infected class $I(t)$ but as before determines what proportion of those tested enter class $Q(t)$ or $P(t)$.

4.2 Partial Testing is 100% Effective: SIPA

4.2.1 Local Asymptotic Stability

The SIPA Model

We start with the system when $\rho = 1$, which eliminates the class $Q(t)$. The schematic diagram of the SIPA model is available in figure 4.5.

The model is:

$$\frac{dS(t)}{dt} = \Lambda - \left(\mu + \frac{\beta c I(t)}{N(t)}\right) S(t)$$

$$\frac{dI(t)}{dt} = \left(\frac{\beta c I(t)}{N(t)}\right) S(t) - (\mu + \omega)I(t)$$

$$\frac{dP(t)}{dt} = \omega I(t) - (\mu + v)P(t) \tag{4.1}$$

Figure 4.5: Schematic Diagram of the SIPA Model

Λ

μ

SUSCEPTIBLES
S(t)

βc

UNTESTED
INFECTEDS
I(t)

μ

ω

RESPONSIBLE
TESTED
INFECTEDS
P(t)

μ

v

FULL-BLOWN
AIDS
A(t)

μ

d
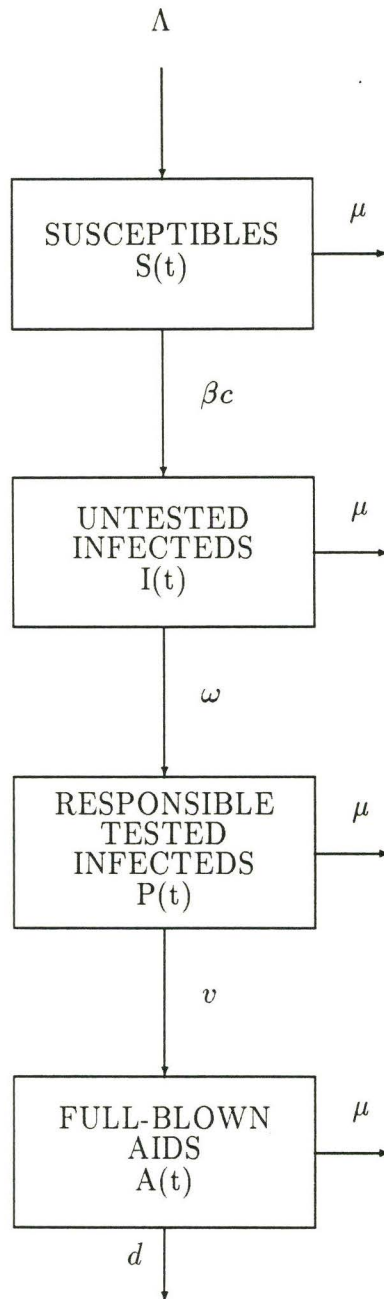
$$\frac{dA(t)}{dt} = vP(t) - (\mu + d)A(t)$$

$$N(t) = S(t) + I(t) + P(t) + A(t)$$

with $S_o > 0, \; I_o > 0, \; P_o \geq 0, \; \text{and} \; A_o \geq 0.$

Therefore,

$$\frac{dN(t)}{dt} = \Lambda - \mu N(t) - dA(t)$$

Equilibria

The equilibria are calculated in Appendix D and are provided below:

$$\begin{pmatrix} \hat{S} \\ \hat{I} \\ \hat{P} \\ \hat{A} \end{pmatrix} = \begin{pmatrix} \frac{\Lambda}{\mu} \\ 0 \\ 0 \\ 0 \end{pmatrix},$$

and

$$\begin{pmatrix} \bar{S} \\ \bar{I} \\ \bar{P} \\ \bar{A} \end{pmatrix} = \begin{pmatrix} \frac{\Lambda((\mu+v+\omega)(\mu+d)+v\omega)}{(\mu+v)(\mu+d)(\beta c - (\mu+\omega)) + \mu((\mu+v+\omega)(\mu+d)+v\omega)} \\ \frac{\Lambda((\mu+v)(\mu+d)(\beta c - (\mu+\omega)))}{\mu+\omega} \\ \frac{\Lambda\omega(\mu+d)(\beta c - (\mu+\omega))}{\mu+\omega} \\ \frac{\Lambda v\omega(\beta c - (\mu+\omega))}{\mu+\omega} \end{pmatrix}.$$

The endemic equilibrium exists if all the components are positive. This is true if $\bar{I} > 0$, or that is, if $\beta c > \mu + \omega$. The reproductive number is:

$$R_o = \frac{\beta c}{\mu + \omega}$$

If $R_o < 1$ a disease free situation will be asymptotically approached, and if $R_o > 1$ the endemic situation occurs. The supporting calculations for the determination of the equilibria are found in Appendix D.

Local Stability Analysis

The results of the linear analysis are given below in tabular form. (See Appendix D for calculations).

EQUILIBRIUM	$\beta c < \mu + \omega$	$\beta c > \mu + \omega$
$(\hat{S}, \hat{I}, \hat{P}, \hat{A})$	Local asymptotic stability	Exists, but is unstable
$(\bar{S}, \bar{I}, \bar{P}, \bar{A})$	Does not exist in the nonnegative cone	Local asymptotic stability

4.2.2 Global Asymptotic Stability

We prove that the disease free equilibrium is globally asymptotically stable whenever it is locally asymptotically stable, that is, when $\beta c < \mu + \omega$.
Define

$$\mathcal{G} = \{(S, I, P, A) \in \Re^4 : S > 0, I > 0, P > 0, A > 0\}$$

$$\overline{\mathcal{G}} = \{(S, I, P, A) \in \Re^4 : S \geq 0, I \geq 0, P \geq 0, A \geq 0\}$$

Further, define $V : \Re^4 \rightarrow \Re$, by

$$V(S, I, P, A) = I.$$

Then V is $C^1(\Re^4)$ and the time derivative

$$\dot{V}(S, I, P, A) = I\left(\frac{\beta c S}{N} - (\mu + \omega)\right).$$

Thus $\dot{V}(S, I, P, A) \leq 0$ on $\mathcal{G}$. Therefore V is a Liapunov function by definition H.5. $dotV$ is equal to 0 if and only if $I = 0$, since $\beta c < \mu + \omega$, and $\frac{S}{N} \leq 1$.

We define

$$
\begin{aligned}
\mathcal{E} &= \{(S, I, P, A) \in \overline{\mathcal{G}} : \dot{V} = 0\} \\
&= \{(S, I, P, A) : I = 0, S \geq 0, P \geq 0, A \geq 0\}.
\end{aligned}
$$

By the LaSalle Extension Theorem (H.6), every bounded solution, and hence every solution by H.1(b), of system 4.1 converges to $\mathcal{M}$ where $\mathcal{M}$ is the largest invariant subset of $\mathcal{E}$.

Consider the system obtained if $I(t) \equiv 0$ in 4.1:

$$
\begin{aligned}
S' &= \Lambda - \mu S \\
P' &= -(\mu + v)P \\
A' &= vP - (\mu + d)A
\end{aligned}
\tag{4.2}
$$

The solutions to 3.10 are:

$$
S(t) = \frac{\Lambda}{\mu} + \left(S_o - \frac{\Lambda}{\mu}\right) e^{-\mu t},
$$

$$
P(t) = vP_o e^{-(\mu+v)t},
$$

$$
A(t) = \frac{vP_o}{d-v} e^{-(\mu+v)t} + \left(A_o - \frac{vP_o}{d-v}\right) e^{-(\mu+d)t}.
$$

We define the largest invariant subset of $\mathcal{E}$ to be:

$$
\mathcal{M} = \{(S, I, P, A) \in \Re_+^4 : Q = 0, S \geq 0, P \geq 0, A \geq 0 \text{ and } (S, P, A) \text{ satisfies } 4.2\}.
$$

For every solution of 4.2,

$$
S(t) \longrightarrow \frac{\Lambda}{\mu}
$$

$$
P(t) \longrightarrow 0
$$

$$
A(t) \longrightarrow 0.
$$

Therefore, the point $(\frac{\Lambda}{\mu}, 0, 0, 0)$ is in the omega limit set of every solution of 4.1. But, this point is a locally asymptotically stable critical point and so it must be the only point in the omega limit set of any solution of 4.1. Hence, the disease free equilibrium is globally asymptotically stable with respect to the solutions initiating in the nonnegative cone, provided $\beta c < \mu + \omega$.

4.3 Partial Testing is 100% Ineffective: SIQA

4.3.1 Local Asymptotic Stability

The SIQA Model

In this section we examine the extreme case with $\rho = 0$ and in doing so, we eliminate the class P(t). The model is:

$$\frac{dS(t)}{dt} = \Lambda - \left(\mu + \frac{\beta c(I(t) + Q(t))}{N(t)}\right) S(t).$$

$$\frac{dI(t)}{dt} = \frac{\beta c(I(t) + Q(t))}{N(t)} - (\mu + \omega) I(t).$$

$$\frac{dQ(t)}{dt} = \omega I(t) - (\mu + v)Q(t). \tag{4.3}$$

$$\frac{dA(t)}{dt} = vQ(t) - (\mu + d)A(t).$$

$$N(t) = S(t) + I(t) + Q(t) + A(t)$$

with $\quad S_o > 0, \ I_o > 0, \ Q_o \geq 0, \ \text{and} \ A_o \geq 0.$

Therefore,

$$\frac{dN(t)}{dt} = \Lambda - \mu N(t) - dA(t).$$

A schematic diagram is provided for your perusal in figure 4.3.1.

Figure 4.6: Schematic Diagram of the SIQA Model
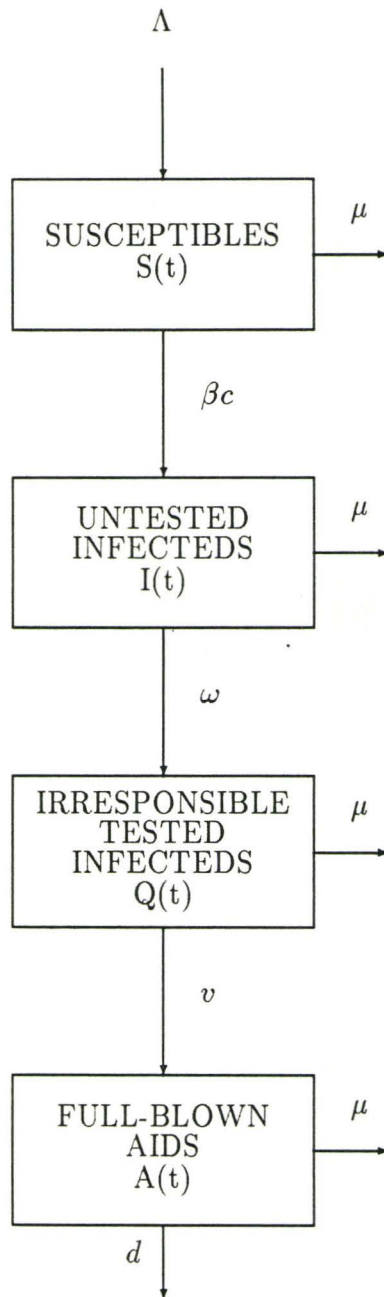
Λ

SUSCEPTIBLES
S(t)

μ

βc

UNTESTED
INFECTEDS
I(t)

μ

ω

IRRESPONSIBLE
TESTED
INFECTEDS
Q(t)

μ

v

FULL-BLOWN
AIDS
A(t)

μ

d

Equilibria

There are two equilibria, the disease free equilibrium, and the endemic equilibrium. The supporting calculations can be found in Appendix E. The disease free equilibrium is:

$$
\begin{pmatrix} \hat{S} \\ \hat{I} \\ \hat{Q} \\ \hat{A} \end{pmatrix} = \begin{pmatrix} \frac{\Lambda}{\mu} \\ 0 \\ 0 \\ 0 \end{pmatrix}.
$$

The endemic equilibrium is:

$$
\begin{pmatrix} \bar{S} \\ \bar{I} \\ \bar{Q} \\ \bar{A} \end{pmatrix} = \begin{pmatrix} \frac{\Lambda(\mu+d)(\mu+v+\omega)+v\omega}{\beta c(\mu+v+\omega)(\mu+d)-vd\omega} \\ \frac{\Lambda}{\mu+\omega}\left(\frac{\beta c(\mu+v+\omega)(\mu+d)-(\mu+\omega)(\mu+v)(\mu+d)}{\beta c(\mu+v+\omega)(\mu+d)-vd\omega}\right) \\ \frac{\Lambda\omega}{(\mu+\omega)(\mu+v)}\left(\frac{\beta c(\mu+v+\omega)(\mu+d)-(\mu+\omega)(\mu+v)(\mu+d)}{\beta c(\mu+v+\omega)(\mu+d)-vd\omega}\right) \\ \frac{\Lambda\omega v}{(\mu+\omega)(\mu+v)(\mu+d)}\left(\frac{\beta c(\mu+v+\omega)(\mu+d)-(\mu+\omega)(\mu+v)(\mu+d)}{\beta c(\mu+v+\omega)(\mu+d)-vd\omega}\right) \end{pmatrix}.
$$

The disease free equilibrium exists always, and the endemic equilibrium exists in the positive cone if its' components are positive, i.e. if $\bar{I}$ is positive. This holds true if $\beta c(\mu + v + \omega)(\mu + d) > (\mu + v)(\mu + \omega)$. The reproductive number is:

$$
R_o = \frac{\beta c(\mu + v + \omega)(\mu + d)}{(\mu + v)(\mu + \omega)}.
$$

If $R_o < 1$ then the disease free equilibrium will be asymptotically approached. If $R_o > 1$ then we will experience an endemic situation.

Local Stability Analysis

The calculations accompanying the stability analysis can be found in Appendix E. To summarize we have:

EQUILIBRIUM	βc $< \frac{(\mu+v)(\mu+\omega)}{(\mu+v+\omega)(\mu+d)}$	βc $> \frac{(\mu+v)(\mu+\omega)}{(\mu+v+\omega)(\mu+d)}$
$(\hat{S}, \hat{I}, \hat{Q}, \hat{A})$	Local asymptotic stability	Exists but is unstable
$(\bar{S}, \bar{I}, \bar{Q}, \bar{A})$	Does not exist in nonnegative cone	Local asymptotic stability

4.3.2 Global Asymptotic Stability

We prove that the disease free equilibrium is globally asymptotically stable whenever it is locally asymptotically stable, that is, when $\beta c(\mu + v + \omega)(\mu + d) < (\mu + v)(\mu + \omega)$.

Define

$$\mathcal{G} = \{(S, I, Q, A) \in \Re^4 : S > 0, I > 0, Q > 0, A > 0\}$$

$$\overline{\mathcal{G}} = \{(S, I, Q, A) \in \Re^4 : S \geq 0, I \geq 0, Q \geq 0, A \geq 0\}$$

Further, define $V : \Re^4 \to \Re$, by

$$V(S, I, Q, A) = I + \left(\frac{\mu + \omega}{\mu + v + \omega}\right) Q.$$

Then V is $C^1(\Re^4_+)$ and the time derivative

$$
\begin{aligned}
\dot{V}(S, I, Q, A) &= \left(\frac{\beta c(I + Q)S}{N} - (\mu + \omega)I\right) \\
&\quad + \left(\frac{\mu + \omega}{\mu + v + \omega}\right)(\omega I - (\mu + v)Q) \\
&= I\left(\frac{\beta cS}{N} - (\mu + \omega) + \frac{(\mu + \omega)\omega}{\mu + v + \omega}\right) \\
&\quad + Q\left(\frac{\beta cS}{N} - \frac{(\mu + v)(\mu + \omega)}{\mu + v + \omega}\right)
\end{aligned}
$$

$$= I\left(\frac{\beta c S}{N} - \frac{(\mu + v)(\mu + \omega)}{\mu + v + \omega}\right) + Q\left(\frac{\beta c S}{N} - \frac{(\mu + v)(\mu + \omega)}{\mu + v + \omega}\right)$$

Thus $\dot{V}(S, I, Q, A) \leq 0$ on $\mathcal{G}$. Therefore V is a Liapunov function by definition H.5. $\dot{V}$ is equal to 0 if and only if $I = 0$ and $Q = 0$, since $\beta c < \frac{(\mu+v)(\mu+\omega)}{(\mu+v+\omega)}$, and $\frac{S}{N} \leq 1$.

We define

$$\begin{aligned}\mathcal{E} &= \{(S, I, Q, A) \in \overline{\mathcal{G}} : \dot{V} = 0\} \\ &= \{(S, I, Q, A) : I = 0, Q = 0, S \geq 0, A \geq 0\}.\end{aligned}$$

By the LaSalle Extension Theorem (H.6), every bounded solution, and hence every solution by H.1(b), of system 4.3 converges to $\mathcal{M}$ where $\mathcal{M}$ is the largest invariant subset of $\mathcal{E}$.

Consider the system obtained if $I(t) \equiv 0$ and $Q(t) \equiv 0$ in 4.3.

$$\begin{aligned}S' &= \Lambda - \mu S \\ A' &= -(\mu + d)A\end{aligned} \qquad (4.4)$$

We define the largest invariant subset of $\mathcal{E}$ as:

$$\mathcal{M} = \{(S, I, Q, A) \in \Re^4_+ :$$

$$I = 0, Q = 0, S \geq 0, A \geq 0 \text{ and } (S, A) \text{ satisfies } 4.4\}.$$

For every solution of 4.4,

$$S(t) \longrightarrow \frac{\Lambda}{\mu}$$

$$A(t) \longrightarrow 0.$$

Therefore, the point $(\frac{\Lambda}{\mu}, 0, 0, 0)$ is in the omega limit set of every solution of 4.3. But, this point is a locally asymptotically stable critical point and so it must be the only point in the omega limit set of any solution of 4.3. Hence, the disease free equilibrium is globally asymptotically stable with respect to the solutions initiating in the nonnegative cone, provided $\beta c < \frac{(\mu+v)(\mu+\omega)}{(\mu+v+\omega)}$.

4.4   Testing is Partially Effective: SIQPA

4.4.1   Local Asymptotic Stability

The SIQPA Model

The model is:

$$\frac{dS(t)}{dt} = \Lambda - \left(\mu + \frac{\beta c(I(t) + Q(t))}{N(t)}\right) S(t),$$

$$\frac{dI(t)}{dt} = \frac{\beta c(I(t) + Q(t))}{N(t)} - (\mu + \omega)\, I(t),$$

$$\frac{dQ(t)}{dt} = (1 - \rho)\omega I(t) - (\mu + v)Q(t), \tag{4.5}$$

$$\frac{dP(t)}{dt} = \rho\omega I(t) - (\mu + v)P(t),$$

$$\frac{dA(t)}{dt} = vQ(t) - (\mu + d)A(t),$$

$$N(t) = S(t) + I(t) + Q(t) + P(t) + A(t),$$

with $S_o > 0, I_o > 0, Q_o \geq 0, P_o \geq 0,$ and $A_o \geq 0.$

Equilibria

There are two equilibria, the disease free equilibrium, and the endemic equilibrium. The supporting calculations can be found in Appendix F. The disease free

equilibrium is:

$$
\begin{pmatrix} \hat{S} \\ \hat{I} \\ \hat{Q} \\ \hat{P} \\ \hat{A} \end{pmatrix} = \begin{pmatrix} \frac{\Lambda}{\mu} \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}.
$$

The endemic equilibrium is

$$
\begin{pmatrix} \bar{S} \\ \bar{I} \\ \bar{Q} \\ \bar{P} \\ \bar{A} \end{pmatrix} = \begin{pmatrix} \frac{\Lambda((\mu+v+\omega)(\mu+d)+v\omega}{\beta c(\mu+d)(\mu+v+(1-\rho)\omega)-vd\omega} \\[2ex] \frac{\Lambda(\mu+d)(\beta c(\mu+v+(1-\rho)\omega)-(\mu+\omega)(\mu+v))}{(\mu+\omega)(\beta c(\mu+d)(\mu+v+(1-\rho)\omega)-vd\omega)} \\[2ex] \frac{\Lambda(\mu+d)(1-\rho)\omega(\beta c(\mu+v+(1-\rho)\omega)-(\mu+\omega)(\mu+v))}{(\mu+\omega)(\mu+v)(\beta c(\mu+d)(\mu+v+(1-\rho)\omega)-vd\omega)} \\[2ex] \frac{\Lambda(\mu+d)\rho\omega(\beta c(\mu+v+(1-\rho)\omega)-(\mu+\omega)(\mu+v))}{(\mu+\omega)(\mu+v)(\beta c(\mu+d)(\mu+v+(1-\rho)\omega)-vd\omega)} \\[2ex] \frac{\Lambda v\omega(\beta c(\mu+v+(1-\rho)\omega)-(\mu+\omega)(\mu+v))}{(\mu+\omega)(\mu+v)(\beta c(\mu+d)(\mu+v+(1-\rho)\omega)-vd\omega)} \end{pmatrix}.
$$

The disease free equilibrium exists always, and the endemic equilibrium exists in the positive cone if each component population is positive. This is true if $\bar{I}$ is positive, that is, if $\beta c(\mu + v + (1 - \rho)\omega) > (\mu + v)(\mu + \omega)$. The reproductive number is:

$$
R_o = \frac{\beta c(\mu + v + (1 - \rho)\omega)}{(\mu + v)(\mu + \omega)}.
$$

If $R_o < 1$ we will asymptotically approach the disease free equilibrium. If $R_o > 1$ we will encounter an endemic situation.

Local Stability Analysis

To summarize we have:

EQUILIBRIUM	$\beta c(\mu + v + (1-\rho)\omega)$ $< (\mu + v)(\mu + \omega)$	$\beta c(\mu + v + (1-\rho)\omega)$ $> (\mu + v)(\mu + \omega)$
$(\hat{S}, \hat{I}, \hat{Q}, \hat{P}, \hat{A})$	Local asymptotic stability	Exists but is unstable
$(\bar{S}, \bar{I}, \bar{Q}, \bar{P}, \bar{A})$	Does not exist in nonnegative cone	Local asymptotic stability

4.4.2   Global Asymptotic Stability

We prove that the disease free equilibrium is globally asymptotically stable whenever it is locally asymptotically stable, that is, when $\beta c < \frac{(\mu+v)(\mu+\omega)}{\mu+v+(1-\rho)\omega}$. Define

$$\mathcal{G} = \{(S, I, Q, P, A) \in \Re^5 : S > 0, I > 0, Q > 0, P > 0, A > 0\}$$

$$\overline{\mathcal{G}} = \{(S, I, Q, P, A) \in \Re^5 : S \geq 0, I \geq 0, Q \geq 0, P \geq 0, A \geq 0\}$$

Further, define $V : \Re^5 \to \Re$, by

$$V(S, I, Q, P, A) = I + \left(\frac{\mu + \omega}{\mu + v + (1 - \rho)\omega}\right) Q.$$

Then V is $C^1(\Re^5)$ and the time derivative

$$
\begin{aligned}
\dot{V}(S, I, Q, P, A) &= \left(\frac{\beta c(I + Q)S}{N} - (\mu + \omega)I\right) \\
&\quad + \left(\frac{(\mu + \omega)}{\mu + v + (1 - \rho)\omega}\right)((1 - \rho)\omega I - (\mu + v)Q) \\
&= I\left(\frac{\beta c S}{N} - \frac{(\mu + \omega)(\mu + v)}{\mu + v + (1 - \rho)\omega}\right)
\end{aligned}
$$

$$+ Q \left(\frac{\beta c S}{N} - \frac{(\mu + \omega)(\mu + v)}{\mu + v + (1 - \rho)\omega} \right)$$

Thus $\dot{V}(S, I, Q, P, A) \leq 0$ on $\mathcal{G}$. Therefore V is a Liapunov function by definition H.5. $\dot{V}$ is equal to 0 if and only if $I = 0$ and $Q = 0$, since $\beta c < \frac{(\mu+v)(\mu+\omega)}{(\mu+v+(1-\rho)\omega)}$, and $\frac{S}{N} \leq 1$.

We define

$$\begin{aligned} \mathcal{E} &= \{(S, I, Q, P, A) \in \overline{\mathcal{G}} : \dot{V} = 0\} \\ &= \{(S, I, Q, P, A) : I = 0, Q = 0, S \geq 0 P \geq 0, A \geq 0\}. \end{aligned}$$

By the LaSalle Extension Theorem (H.6), every bounded solution, and hence every solution by H.1(b), of system 4.5 converges to $\mathcal{M}$ where $\mathcal{M}$ is the largest invariant subset of $\mathcal{E}$.

Consider the system obtained if $I(t) \equiv 0$ and $Q(t) \equiv 0$ in 4.5

$$\begin{aligned} S' &= \Lambda - \mu S \\ P' &= -(\mu + v)P \\ A' &= vP - (\mu + d)A. \end{aligned} \tag{4.6}$$

The solutions to system 4.6 are:

$$S(t) = \frac{\Lambda}{\mu} + \left(S_o - \frac{\Lambda}{\mu} \right) e^{-\mu t},$$

$$P(t) = v P_o e^{-(\mu + v)t},$$

$$A(t) = \frac{v P_o}{d - v} e^{-(\mu + v)t} + \left(A_o - \frac{v P_o}{d - v} \right) e^{-(\mu + d)t}.$$

We define the largest invariant subset of $\mathcal{E}$ to be:

$$\begin{aligned} \mathcal{M} = \{(S, I, Q, P, A) &\in \Re^5_+ : \\ I = 0, Q = 0, S \geq 0, P \geq 0, &A \geq 0, \text{ and } (S, P, A) \text{ satisfies } 4.6\}. \end{aligned}$$

For every solution of 4.6,

$$S(t) \longrightarrow \frac{\Lambda}{\mu}.$$

$$P(t) \longrightarrow 0$$

$$A(t) \longrightarrow 0.$$

Therefore, the point $(\frac{\Lambda}{\mu}, 0, 0, 0, 0)$ is in the omega limit set of every solution of 4.5. But, this point is a locally asymptotically stable critical point and so it must be the only point in the omega limit set of any solution of 4.5. Hence, the disease free equilibrium is globally asymptotically stable with respect to the solutions initiating in the nonnegative cone, provided $\beta c < \frac{(\mu+v)(\mu+\omega)}{\mu+v+(1-\rho)\omega}$.

4.5 The SIQ/PA Model

4.5.1 Local Asymptotic Stability

The Model

Consider the system in equations 4.5. If we assume that those individuals in the populations P and A refrain from sexual contacts, then the total sexually active population consists of $N(t) = S(t) + I(t) + Q(t)$. This assumption is realistic according to the way we have defined our classes. Individuals from the P and A classes are assumed to practice safe sexual behaviours or complete abstinence, so there is no loss of generality in assuming they are not members of the sexually active population. Under this assumption, P and A do not appear in S', I', or Q'. Thus P' and A' can be decoupled from the remainder of the equations. We examine the equations:

$$\frac{dS(t)}{dt} = \Lambda - \left(\mu + \frac{\beta c(I(t) + Q(t))}{N(t)}\right) S(t)$$

$$\frac{dI(t)}{dt} = \left(\frac{\beta c(I(t) + Q(t))}{N(t)}\right) S(t) - (\mu + \omega) I(t)$$

$$\frac{dQ(t)}{dt} = (1 - \rho)\omega I(t) - (\mu + v)Q(t), \tag{4.7}$$

$$\frac{dP(t)}{dt} = \rho \omega I(t) - (\mu + v)P(t),$$

$$\frac{dA(t)}{dt} = v(Q(t) + P(t)) - (\mu + d)A(t),$$

$$N(t) = S(t) + I(t) + Q(t),$$

with $\quad S_o > 0, I_o > 0, Q_o \geq 0, P_o \geq 0,$ and $A_o \geq 0$

Therefore,

$$\frac{dN(t)}{dt} = \Lambda - \mu N(t).$$

Equilibria

There are two equilibria, the disease free equilibrium, and the endemic equilibrium. The supporting calculations can be found in Appendix G. The disease free equilibrium is

$$\begin{pmatrix} \hat{S} \\ \hat{I} \\ \hat{Q} \\ \hat{P} \\ \hat{A} \end{pmatrix} = \begin{pmatrix} \frac{\Lambda}{\mu} \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}.$$

The endemic equilibrium is

$$
\begin{pmatrix} \bar{S} \\ \bar{I} \\ \bar{Q} \\ \bar{P} \\ \bar{A} \end{pmatrix} = \begin{pmatrix} \frac{\Lambda c_1}{c_2} \\ \frac{\Lambda c_3}{(\mu+\omega)c_2} \\ \frac{\Lambda c_3 (1-\rho)\omega}{c_2(\mu+\omega)(\mu+v)} \\ \frac{\Lambda c_3 \rho\omega}{c_2(\mu+\omega)(\mu+v)} \\ \frac{\Lambda c_3 v\omega}{c_2(\mu+\omega)(\mu+v)(\mu+d)} \end{pmatrix}
$$

where

$$
\begin{aligned}
c_1 &= (\mu + v + (1 - \rho)\omega) \\
c_2 &= \beta c(\mu + v + (1 - \rho)\omega) - (\mu + \omega)(\mu + v) + \mu(\mu + v + (1 - \rho)\omega) \\
c_3 &= \beta c(\mu + v + (1 - \rho)\omega) - (\mu + \omega)(\mu + v).
\end{aligned}
$$

The disease free equilibrium always exists, and the endemic equilibrium exists in the positive cone if the components of the endemic equilibrium are all positive. This requirement is satisfied if $\bar{I} > 0$, that is, if $\beta c(\mu + v + (1 - \rho)\omega) > (\mu + v)(\mu + \omega)$. The reproductive number is given by:

$$
R_o = \frac{\beta c(\mu + v + (1 - \rho)\omega)}{(\mu + v)(\mu + \omega)}.
$$

As before, if $R_o < 1$ then the disease free state will be achieved and if $R_o > 1$ we will experience an endemic situation.

Local Stability Analysis

To summarize we have:

EQUILIBRIUM	$\beta c(\mu + v + (1 - \rho)\omega)$ $< (\mu + v)(\mu + \omega)$	$\beta c(\mu + v + (1 - \rho)\omega)$ $> (\mu + v)(\mu + \omega)$
$(\hat{S}, \hat{I}, \hat{Q}, \hat{P}, \hat{A})$	Local asymptotic stability	Exists but is unstable
$(\bar{S}, \bar{I}, \bar{Q}, \bar{P}, \bar{A})$	Does not exist in nonnegative cone	Local asymptotic stability

We now turn to an examination of the global properties of the SIQ/PA model.

4.5.2   Global Stability

Disease Free Equilibrium

We prove that the disease free equilibrium is globally asymptotically stable whenever it is locally asymptotically stable, that is, when $\beta c < \frac{(\mu+v)(\mu+\omega)}{\mu+v+(1-\rho)\omega}$. As P and Q can be decoupled from the remainder of the equations in 4.7 we do not directly include them in our calculations, so for the following discussion we need only consider the equations:

$$\frac{dS(t)}{dt} = \Lambda - \left(\mu + \frac{\beta c(I(t) + Q(t))}{N(t)}\right) S(t)$$

$$\frac{dI(t)}{dt} = \left(\frac{\beta c(I(t) + Q(t))}{N(t)}\right) S(t) - (\mu + \omega) I(t) \qquad (4.8)$$

$$\frac{dQ(t)}{dt} = (1 - \rho)\omega I(t) - (\mu + v)Q(t),$$

$$N(t) \;=\; S(t) + I(t) + Q(t)$$

The initial conditions corresponding to the above equations are the same as those provided in 4.7.

Define

$$\mathcal{G} = \{(S, I, Q) \in \Re^3 : S > 0, I > 0, Q > 0\}$$

$$\overline{\mathcal{G}} = \{(S, I, Q) \in \Re^3 : S \geq 0, I \geq 0, Q \geq 0.\}$$

Further, define $V : \Re^3 \to \Re$, by

$$V(S, I, Q) = I + \left(\frac{\mu + \omega}{\mu + v + (1 - \rho)\omega} \right) Q.$$

Then V is $C^1(\Re^3)$ and the time derivative

$$
\begin{aligned}
\dot{V}(S, I, Q) \;=\;& \left(\frac{\beta c(I + Q)S}{N} - (\mu + \omega)I \right) \\
&+ \left(\frac{(\mu + v)(\mu + \omega)}{\mu + v + (1 - \rho)\omega} \right) \left((1 - \rho)\omega I - (\mu + v)Q \right). \\
\;=\;& I \left(\frac{\beta c S}{N} - \frac{(\mu + \omega)(\mu + v)}{\mu + v + (1 - \rho)\omega} \right) \\
&+ Q \left(\frac{\beta c S}{N} - \frac{(\mu + \omega)(\mu + v)}{\mu + v + (1 - \rho)\omega} \right)
\end{aligned}
$$

Thus $\dot{V}(S, I, Q) \leq 0$ on $\mathcal{G}$. Therefore V is a Liapunov function by definition H.5. $\dot{V}$ is equal to 0 if and only if $I = 0$ and $Q = 0$, since $\beta c < \frac{(\mu+v)(\mu+\omega)}{(\mu+v+(1-\rho)\omega)}$, and $\frac{S}{N} \leq 1$.

We define

$$
\begin{aligned}
\mathcal{E} \;=\;& \{(S, I, Q) \in \overline{\mathcal{G}} : \dot{V} = 0\} \\
\;=\;& \{(S, I, Q) : I = 0, Q = 0, S \geq 0\}.
\end{aligned}
$$

By the LaSalle Extension Theorem (H.6), every bounded solution, and hence every solution by H.1(b), of system 4.8 converges to $\mathcal{M}$ where $\mathcal{M}$ is the largest invariant subset of $\mathcal{E}$.

Consider the system obtained if $I(t) \equiv 0$ and $Q(t) \equiv 0$ in 4.8.

$$S' = \Lambda - \mu S, \qquad (4.9)$$

We define the largest invariant subset of $\mathcal{E}$ as:

$$\mathcal{M} = \{(S, I, Q) \in \Re_+^3 : I = 0, Q = 0, S \geq 0, \text{ and } S \text{ satisfies } 4.9\}.$$

For every solution of 4.9,

$$S(t) \longrightarrow \frac{\Lambda}{\mu}.$$

Therefore, the point $(\frac{\Lambda}{\mu}, 0, 0)$ is in the omega limit set of every solution of 4.8. But, this point is a locally asymptotically stable critical point and so it must be the only point in the omega limit set of any solution of 4.8. Hence, the disease free equilibrium is globally asymptotically stable with respect to the solutions initiating in the nonnegative cone, provided $\beta c < \frac{(\mu+v)(\mu+\omega)}{\mu+v+(1-\rho)\omega}$.

In section 4.4, we looked at a Liapunov function for the SIQPA model. This function shows that the global stability of the P and A components of the disease free equilibrium follow directly.

Endemic Equilibrium

Suppose $\beta c(\mu + v + (1 - \rho)\omega) > (\mu + v)(\mu + \omega)$. Then we shall show that system 4.8 is persistent with respect to all solutions for which the initial conditions are positive. We identify the space $(S(t), I(t), Q(t))$ with $\Re_+^3$. Refer to figure 4.7 for the qualitative behaviour of the system on the boundaries of $\Re_+^3$.

Recall from our linear analysis found in Appendix G that there are five eigenvalues associated with the disease free equilibrium of system 4.7. These are listed in

Figure 4.7: Diagram of the Qualitative Behaviour of SIQ on $\partial\Re_+^3$
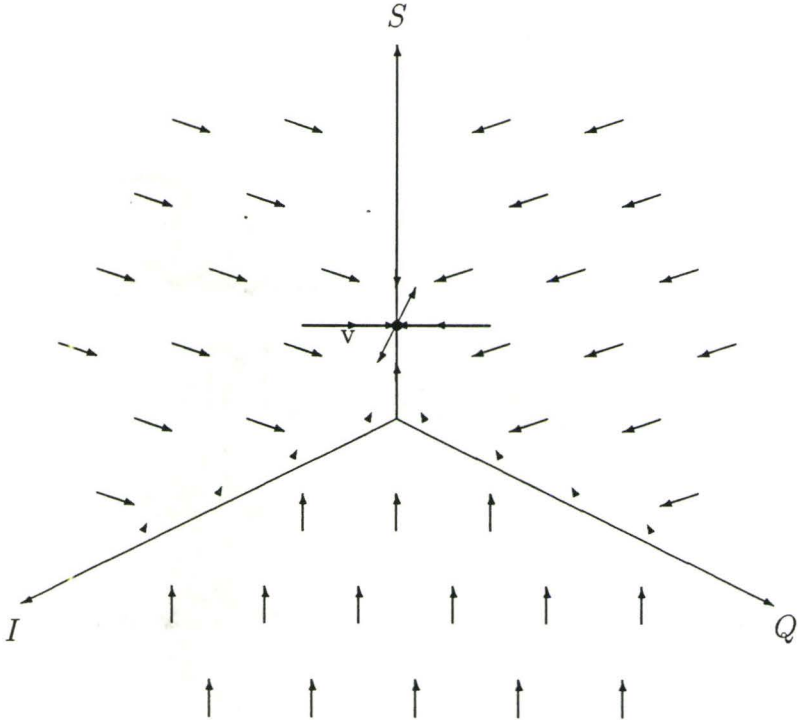
section G.2.1. To begin with, we will only be considering the system 4.8. Thus, for now we are only concerned with the characteristic equation

$$(\mu + \lambda)(\lambda^2 - (\beta c - (2\mu + v + \omega))\lambda - (\mu + v)(\beta c - (\mu + \omega)) - \beta c(1 - \rho)\omega)$$

We always have at least one negative eigenvalue, $\lambda_1 = -\mu$. To determine the stable manifold of the disease free equilibrium we must now examine the quadratic factor remaining in the characteristic equation. The constant coefficient of the characteristic equation is the product of the eigenvalues. We call this coefficient H. Recall that from section G.2.1, where we have solved for the eigenvalues, that the discriminant is always positive and the eigenvalues always real. Hence we need not consider complex conjugates in determining the sign of H. We consider the remaining two cases, firstly where H is positive and secondly where H is negative. In the first case, if $H > 0$, then either both λ_2 and λ_3 are positive or both λ_2 and λ_3 are negative, where

$$\lambda_2 = \frac{1}{2}(\beta c - (2\mu + v + \omega))$$
$$+ \frac{1}{2}\sqrt{(\beta c - (2\mu + v + \omega))^2 + 4(\mu + v)(\beta c - (\mu + \omega)) + 4\beta c(1 - \rho)\omega}$$

$$\lambda_3 = \frac{1}{2}(\beta c - (2\mu + v + \omega))$$
$$- \frac{1}{2}\sqrt{(\beta c - (2\mu + v + \omega))^2 + 4(\mu + v)(\beta c - (\mu + \omega)) + 4\beta c(1 - \rho)\omega}.$$

If both eigenvalues are positive, then we need only look at $\lambda_3 > 0$ to see a contradiction to our assumption that $\beta c(\mu + v + (1 - \rho)\omega) > (\mu + \omega)(\mu + v)$, since $\lambda_2 > \lambda_3$ and $\lambda_3 > 0$ implies that $\beta c(\mu + v + (1 - \rho)\omega) < (\mu + \omega)(\mu + v)$. If λ_2 and λ_3 are both negative, then the disease free equilibrium must be globally stable, a contradiction.

In the second possibility, $H < 0$, we must have one negative and one positive eigenvalue. Since $\lambda_2 > \lambda_3$ then the case to be considered is with $\lambda_2 > 0$ and $\lambda_3 < 0$. Solving the inequalities posed by these conditions we arrive at the result $\beta c(\mu + v + (1 - \rho)\omega) > (\mu + \omega)$ which agrees with our assumption that the disease free equilibrium is unstable. This implies that if $\beta c > \frac{(\mu + \omega)(\mu + v)}{\mu + v + (1 - \rho)\omega}$, there are always two negative eigenvalues λ_1 and λ_3 and one positive eigenvalue λ_2. Thus, the stable manifold of the

disease free equilibrium is of dimension two and the unstable manifold of dimension one.

In order to determine whether or not the stable manifold of the disease free equilibrium intersects the interior of $\Re_+^3$ we consider the eigenvector associated with λ_3. We will determine that the components of the eigenvector have opposite signs. Consider the 2x2 matrix, labelled C, representing the lower matrix of the Jacobian for system 4.8, evaluated at $(\frac{\Lambda}{\mu}, 0, 0)$,

$$C = \begin{pmatrix} \beta c - (\mu + \omega) & \beta c \\ (1 - \rho)\omega & -(\mu + v) \end{pmatrix}. \tag{4.10}$$

Then,

$$C - I\lambda_3 = \begin{pmatrix} \beta c - (\mu + \omega + \lambda_3) & \beta c \\ (1 - \rho)\omega & -(\mu + v + \lambda_3) \end{pmatrix}$$

$$= \begin{pmatrix} \frac{1}{2}\left(\beta c - (\mu + \omega) + (\mu + v) + \sqrt{B}\right) & \beta c \\ (1 - \rho)\omega & -\frac{1}{2}\left(\beta c - (\mu + \omega) + (\mu + v) - \sqrt{B}\right) \end{pmatrix}.$$

where $4B^2 = (\beta c - (2\mu + v + \omega))^2 + 4\beta c(1 - \rho)\omega + 4(\mu + v)(\beta c - (\mu + \omega))$.

Let

$$A = \frac{1}{2}(\beta c - (\mu + \omega) + (\mu + v))$$

and

$$B = \frac{1}{2}\sqrt{(\beta c - (2\mu + v + \omega))^2 + 4\beta c(1 - \rho)\omega + 4(\mu + v)(\beta c - (\mu + \omega))}.$$

Then we may rewrite the matrix $C - I\lambda_3$ in a more manageable form as:

$$\begin{pmatrix} A + B & \beta c \\ (1 - \rho)\omega & -A + B \end{pmatrix}$$

Let $\underline{v} = (v_1, v_2)$ be the eigenvector corresponding to this matrix. By definition, $(C - I\lambda_3)(\underline{v}) = 0$. That is,

$$\begin{pmatrix} A + B & \beta c \\ (1 - \rho)\omega & -A + B \end{pmatrix} \begin{pmatrix} v_1 \\ v_2 \end{pmatrix} = 0$$

Therefore

$$(A + B)v_1 + \beta c v_2 \;=\; 0 \qquad\qquad (4.11)$$

$$(1 - \rho)\omega v_1 + (B - A)v_2 \;=\; 0 \qquad\qquad (4.12)$$

Further, we know that $|C - I\lambda_3| = 0$ so that

$$(A + B)(B - A) \;=\; \beta c(1 - \rho)\omega \qquad\qquad (4.13)$$

Multiplying equation 4.11 by $(1 - \rho)\omega$ and equation 4.12 by $A + B$, we have

$$(1 - \rho)\omega(A + B)v_1 + \beta c(1 - \rho)\omega v_2 \;=\; 0$$

and

$$
\begin{aligned}
(1 - \rho)\omega(A + B)v_1 + (A + B)(B - A)v_2 \\
= \;(1 - \rho)\omega(A + B)v_1 + \beta c(1 - \rho)\omega v_2 \\
= \;0,
\end{aligned}
$$

and thus

$$v_1 \;=\; -\frac{\beta c}{A + B}v_2. \qquad\qquad (4.14)$$

Let $\hat{B} = \frac{1}{2}|2\mu + v + \omega - \beta c|$. Since $\beta c(\mu + v + (1 - \rho)\omega) > (\mu + v)(\mu + \omega)$, then $\hat{B} < B$. This follows immediately from

$$
\begin{aligned}
4\hat{B}^2 &= |2\mu + v + \omega - \beta c|^2 \\
&< \;(\beta c - (2\mu + v + \omega))^2 + 4(\mu + v)(\beta c - (\mu + \omega)) + 4\beta c(1 - \rho)\omega = 4B^2.
\end{aligned}
$$

If $A + \hat{B} > 0$ then $A + B > 0$.

$$A + \hat{B} = \frac{1}{2}(\beta c - (\mu + \omega) + \mu + v) + \frac{1}{2}|2\mu + v + \omega - \beta c|$$

Note that if $A + B > 0$ then v_1 and v_2 have opposite signs, by 4.14. If $2\mu + v + \omega - \beta c > 0$ then $\beta c < 2\mu + v + \omega$, so that

$$
\begin{aligned}
A + \hat{B} &= \frac{1}{2}(\beta c - (\mu + \omega) + \mu + v + 2\mu + v + \omega - \beta c) \\
&= \mu + v \\
&> 0
\end{aligned}
\tag{4.15}
$$

If $2\mu + v + \omega - \beta c < 0$ then $\beta c > 2\mu + v + \omega > \mu + \omega > 0$, so that

$$
\begin{aligned}
A + \hat{B} &= \frac{1}{2}(\beta c - (\mu + \omega) + \mu + v + \beta c - (\mu + \omega) - (\mu + v)) \\
&= \beta c - (\mu + \omega) \\
&> 0.
\end{aligned}
\tag{4.16}
$$

Hence by equations 4.15 and 4.16, $A + \hat{B} > 0$ which implies $A + B > 0$ and so we may conclude by 4.14 that v_1 and v_2 have opposite signs. Thus the eigenvector in $\Re^3_+$ corresponding to the eigenvalue λ_3, $(0, v_1, v_2)^t$ does not intersect $\overset{o}{\Re}{}^3_+$.

Now we are in a position to prove system 4.8 is persistent. Let $\underline{R}$ be a point in the interior of $\Re^3_+$. We examine the closed IQ face, the open SQ and SI faces, and the S axis, to determine if there are any points on the boundary of the nonnegative cone contained in $\Omega(\underline{R})$, the omega limit set of $\underline{R}$. Consider a point (S_o, I_o, Q_o) in the closed nonnegative IQ face, i.e. $(S_o, I_o, Q_o) \in \{(S, I, Q) : S = 0, I \geq 0, Q \geq 0\}$. On this face $S' = \Lambda > 0$ and hence any point on this face will leave the nonnegative cone in negative time. This implies that no point on this face is in the $\Omega(\underline{R})$.

We now consider the open nonnegative SI face, and choose some point,

$$
(S_o, I_o, Q_o) \in \{(S, I, Q) : S > 0, I > 0, Q \geq 0\}.
$$

On this face we have $Q' = (1 - \rho)\omega I > 0$. Hence no point on this face can be in $\Omega(\underline{R})$ since in negative time, any point on this face will leave the nonnegative cone.

The results for the open SQ face are similar. We choose a point,

$$
(S_o, I_o, Q_o) \in \{(S, I, Q) : S > 0, I \geq 0, Q > 0\}.
$$

On this face, $I' = \frac{\beta cQS}{N} > 0$, and so in negative time any point on this face will exit the nonnegative cone. Therefore, there are no points on the open SQ face in the $\Omega(\underline{R})$.

Now we need only examine the S-axis, to prove the remainder of the persistence argument. To this end, choose a point $\tilde{S} > S_o$ on the S-axis. Then in negative time its orbit becomes unbounded, a contradiction by H.2. Therefore $\{\tilde{S}\} \cap \Omega(\underline{R}) = \emptyset$. Similarly, if we choose a point $\check{S} < S_0$, then in negative time its orbit leaves the nonnegative cone. Thus $\{\check{S}\} \cap \Omega(\underline{R}) = \emptyset$.

Let $W^s(\mathcal{P})$ denote the stable manifold of the disease free equilibrium $\mathcal{P} = (\frac{\lambda}{\mu}, 0, 0)$. Further, let $W^u(\mathcal{P})$ denote the unstable manifold of $\mathcal{P}$. Since two of the eigenvalues of the characteristic equation associated with $\mathcal{P}$ are negative and one is positive, then $dim(W^s(\mathcal{P})) = 2$. The stable manifold is smooth and contains $\{(S_o, 0, 0) \in \Re_+^3\}$. If we are sufficiently close to $\mathcal{P}$, we are able to approximate the stable manifold with the half plane defined by:

$$\{(x_1, x_2, x_3) \in \Re_+^3 : x_2 - \frac{v_1}{v_2}x_3 = 0, \}$$

where v_1 and v_2 are the components of the eigenvector associated with the negative eigenvalue λ_3 defined in equation 4.14. We proved earlier that v_1 and v_2 were of opposite sign, and hence the stable manifold of the disease free equilibrium does not intersect the interior of $\Re_+^3$.

Suppose $\mathcal{P} \in \Omega^+(\underline{R})$. But $\mathcal{P}$ can't be the only point in the forward orbit of $\underline{R}$ since $W^s(\mathcal{P}) \cap \Re_+^3 = \emptyset$. So by the Butler McGehee Lemma, H.4, there exists points $\mathcal{P}^s$ in $W^s(\mathcal{P}) \backslash \{\mathcal{P}\}$ and $\mathcal{P}^u$ in $W^u(\mathcal{P}) \backslash \{\mathcal{P}\}$ in $\Omega(\underline{R})$. However, we have already proved that no points on the S-axis can be in the $\Omega(\underline{R})$. Thus, we conclude that there are no omega limit points on the boundary of $\Re_+^3$. Therefore system 4.8 is persistent by the definition of persistence H.3.

We now tie in the P and A portions of system 4.7 to the persistence argument. Note that, given solutions for $Q(t)$ and $I(t)$, the equations P' and A' are linear. We solve these to get:

$$P(t) = \rho \omega e^{-(\mu+v)t} \int e^{(\mu+v)t} I(t) dt$$

$$A(t) \;=\; v e^{-(\mu+d)t} \int e^{(\mu+d)t}(Q(t)+P(t))dt.$$

Thus, if we consider the endemic equilibrium to be a solution, (which is very likely), then

$$\bar{P} \;=\; \frac{\rho\omega}{\mu+v}\bar{I}$$

$$\bar{A} \;=\; \frac{v\omega}{(\mu+v)(\mu+d)}\bar{I}.$$

These values correspond to the earlier calculation of the endemic equilibrium, and since the I population is persistent then it follows that at equilibrium, so are the P and A populations. Now, if we consider the solutions $I(t) = \epsilon > 0$ and $Q(t) = \delta > 0$, then the solutions for $P(t)$ and $A(t)$ are:

$$P(t) \;=\; \frac{\rho\omega\epsilon}{\mu+v} \quad > 0$$

$$A(t) \;=\; \frac{v}{\mu+d}(\epsilon+\delta) \quad > 0$$

Hence, we can conclude that for any solutions $I(t) > 0$ and $Q(t) > 0$, the populations P and A are persistent. Thus the endemic equilibrium of system 4.7 is persistent.

Simon and Jacquez, [47] have established global stability of the endemic equilibrium in various SI models for heterogeneous populations. They state that the arguments can be extended to SIR and other models. Thus, we conjecture that the endemic equilibrium for the SIQ/PA (and other models in this thesis), is globally asymptotically stable.

4.5.3 SIQPA vs. SIQ/PA

It is interesting to note the similarity of results between the SIQPA and the SIQ/PA models. The equilibria are identical, as are the reproductive numbers. The size of the sexually active population is larger for the SIQPA model then it is for the SIQ/PA model. This difference in size changes the speed that the populations approach the equilibria. Early in the epidemic, there is little difference between the models, as the proportion of the population in the P and A populations is small. However, as the disease progresses, the difference becomes more evident. Since N is smaller for the SIQ/PA model, there is a faster movement out of the susceptible population and into the untested infected population. Hence, if the system is in an endemic situation, the endemic equilibrium will be approached faster for the SIQ/PA model. The opposite is true for the disease free equilibrium. If the reproductive number is less than 1, the SIQPA model approaches the disease free state faster than the SIQ/PA model. We can interpret these results to indicate that having a P class is beneficial to the reduction in the spread of the AIDS disease. This is plausible, since if there are more individuals around practicing safe sex, then the susceptibles are more likely to choose an uninfected partner than they are if there were only irresponsible individuals from which to choose their sexual partners.

In the SIQPA model, and all other models in this thesis excepting the SIQ/PA model, there was no distinction made between sexually active individuals in any of the populations. In the SIQ/PA model, the individual was required to engage in unsafe sexual practices, in order to be defined as sexually active. We see by our results, that the qualitative behaviour is identical between models. This makes sense because there has ultimately been no change in the number of sexual contacts in the two models. However, if one were to conduct a cost/benefit analysis of various ratios of populations, we would obtain different results for the two models. For instance, if we were interested in knowing the ratio of susceptibles to sexually active infecteds, so that we could predict future trends of the disease, then the SIQ/PA model would probably provide a more accurate estimate than the SIQPA model. This follows, since, in essence, the P and A classes pose no threat to the susceptible population

in either of the models. However, if we were to expand the models presented in this paper we might find ourselves requiring the totally sexually active population to be the same as in the SIQPA model. We leave the options available to us for future modelling efforts.

Chapter 5

DISCUSSION

5.1 Preliminaries

In this thesis we introduced a series of models examining the dynamics of the spread of the AIDS disease. We are interested in examining how HIV antibody testing in conjunction with persuasive techniques encouraging safe sexual behaviour of these tested infecteds, will influence the dynamics of the system. By first examining the extreme cases of the parameters ρ and ω we could more precisely determine the effects on the outcome of the AIDS disease of these factors. By performing local, and where possible, global analyses on this series of models, we are able to establish the importance of HIV antibody testing and responsible sexual behaviour.

We begin our discussion by displaying, in figures 5.8, and 5.9, the stability results obtained in each of the sections. Then we will compare the effects of introducing certain parameters and populations on the outcome of the AIDS epidemic. By examining the reproductive numbers of the various models, in the form of the criteria as provided in figures 5.8 and 5.9, we can determine what effects the parameter values have on the progression of the AIDS disease. We will first deal with the results of the instantaneous testing models, SPA, SQA, and SQPA, and then secondly, with the results of the partial testing models, SIPA, SIQA, SIQPA, and SIQ/PA. Throughout the discussion we will consider the SIQ/PA model as representative of the SIQPA model,

since their dynamics are very similar. In the final section, we consider shortcomings of the models, and suggest applicable future research ideas.

5.2 SPA, SQA, and SQPA Results

We first compare the results of the SPA, SQA and the SQPA models. From figure 5.8 we see that there is no need to place restrictions on the sexual activity levels, or on other parameters for the SPA model. We are guaranteed an asymptotic approach to the disease free equilibrium, because of its global stability properties, provided there are no external factors, (as we had in section 3.2.2). Any combination of values will still guarantee a disease free state. This is plausible since we have no irresponsible infecteds present.

In all the models presented in this thesis, elimination of the disease is guaranteed provided the criterion for the model is satisfied, since in all cases, we have shown global asymptotic stability of the disease free equilibrium.

After examining the SPA model we moved on to the SQA model. At this point an endemic equilibrium was discovered. Thus, introducing the class Q, while ignoring the P class, had a significant effect on the dynamics of the system. In fact we found that we were now obliged to place restrictions on the parameters to encounter a disease free situation. For the SQA model we require $\beta c < \mu + v$. Since μ and v are biological factors upon which we have little control, then in this system the only way to induce a disease free situation is to influence individuals to have less sexual acts. This however, is beyond the scope of this thesis. The effect of having a long incubation period is clear in this model, since the shorter the incubation period, i.e. the smaller the value of $\frac{1}{v}$, the more the flexibility available for the term βc. The parameters in the SQA model require more severe restrictions than for the SQPA model.

In the SQPA model, the severity of the necessary restrictions caused by the irresponsible infected class Q is offset by the effect of reintroducing the responsible infected class P. The inclusion of P with Q, and the option to choose a value of

Figure 5.8: Summary of SPA, SQA, and SQPA Models

EQUILIBRIUM	CRITERION	
SPA	No Restrictions	
$(\hat{S}, \hat{P}, \hat{A})$	Always Global Asymptotic Stability	
SQA	$\beta c < \mu + v$	$\beta c > \mu + v$
$(\hat{S}, \hat{Q}, \hat{A})$	Local asymptotic stability	Exists but is unstable
$(\bar{S}, \bar{Q}, \bar{A})$	Does not exist in the nonnegative cone	Local asymptotic stability
SQPA	$\beta c < \frac{\mu + v}{1 - \rho}$	$\beta c > \frac{\mu + v}{1 - \rho}$
$(\hat{S}, \hat{Q}, \hat{P}, \hat{A})$	Local asymptotic stability	Exists but is unstable
$(\bar{S}, \bar{Q}, \bar{P}, \bar{A})$	Does not exist in the nonnegative cone	Local asymptotic stability

Figure 5.9: Summary of SIPA, SIQA, and SIQ/PA Models

EQUILIBRIUM	CRITERION	
SIPA	$\beta c < \mu + \omega$	$\beta c > \mu + \omega$
$(\hat{S}, \hat{I}, \hat{P}, \hat{A})$	Local asymptotic stability	Exists but is unstable
$(\bar{S}, \bar{I}, \bar{P}, \bar{A})$	Does not exist in the nonnegative cone	Local asymptotic stability
SIQA	$\beta c(\mu + v + \omega)$ $< (\mu + v)(\mu + \omega)$	$\beta c(\mu + v + \omega)$ $> (\mu + v)(\mu + \omega)$
$(\hat{S}, \hat{I}, \hat{Q}, \hat{A})$	Local asymptotic stability	Exists but is unstable
$(\bar{S}, \bar{I}, \bar{Q}, \bar{A})$	Does not exist in nonnegative cone	Local asymptotic stability
SIQ/PA and SIQPA	$\beta c(\mu + v + (1 - \rho)\omega)$ $< (\mu + v)(\mu + \omega)$	$\beta c(\mu + v + (1 - \rho)\omega)$ $> (\mu + v)(\mu + \omega)$
$(\hat{S}, \hat{I}, \hat{Q}, \hat{P}, \hat{A})$	Local asymptotic stability	Exists but is unstable
$(\bar{S}, \bar{I}, \bar{Q}, \bar{P}, \bar{A})$	Does not exist in nonnegative cone	Local asymptotic stability

ρ between 0 and 1, provides more flexibilty of control. As the value chosen for ρ approaches 1, the restrictions required on parameter values to provide a disease free state, become less strict. This is evident in the graph in figure 5.10. The graph represents the change in the criterion, (see figure 5.8) as we scan through the interval $0 < \rho < 1$. The data in this graph were calculated using the fixed values $\mu = 0.02$ and $v = 0.125$, corresponding to an average life span of 50 years and an average incubation period of 8 years. For equivalent parameter values, the SQA model has a constant criterion of 0.145. Clearly, the SQPA model offers more hope. It seems that in reality, the value for ρ is larger than 0.7. Hence, if $\beta c \approx .5$ the disease will die out. In figure 5.10 we can see the definite sensitivity to the higher ρ values. This implies, that if $\rho = 0.7$ then perhaps minimal effort would be required to bring that value up to 0.8, which rather dramatically increases our flexibility for βc. However, it is conceivable that in this upper scale no amount of educational persuasion can increase the value of ρ, since perhaps the tested population can no longer benefit from further education. Conversely if there were to be a drop in the proportion of responsible individuals a significant drop in βc would be required for the disease to die out.

By these three models we can conclude that the value of ρ has significant effects on the outcome of the disease. Thus, some action, whether it be educational, legal or other action, should be imposed on the tested infected population to encourage responsible behaviour. The educational action might include educating the tested infecteds as to the effects of unsafe sex practices, and to encourage responsible sexual activity. Imposing legal repercussions on tested individuals who behave irresponsibly might provide some incentive for these individuals to practice safe sex. This approach has problems in itself. The enforcement of these laws could prove difficult, as in actuality there are many ways to become infected and pinpointing a sexual act is unlikely. Laws concerning sexual practices increase cost to society and are difficult to enforce, but perhaps we will need to expand present laws if educational efforts fail, as the disease spreads to a larger and larger population. We conclude that the percentage of individuals behaving responsibly has a significant effect on the dynamics of the AIDS disease.

The following section considers the balance of the models presented in this

Figure 5.10: Graph of SQPA Model Results
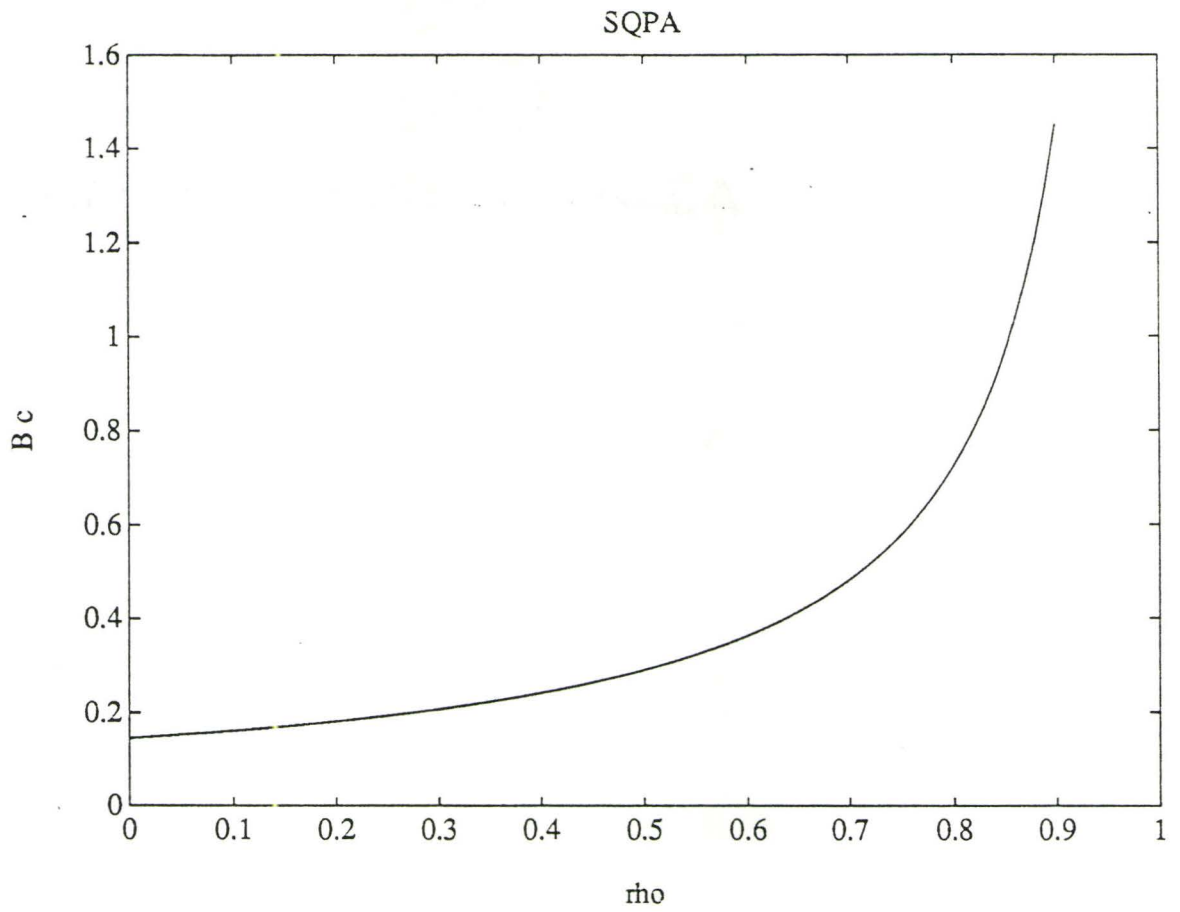
paper. In these models we examine the effects that testing for HIV antibodies have on the progress of AIDS.

5.3   SIPA, SIQA, SIQ/PA Results

The three models being summarized in this section provided one more parameter, ω, the proportion of individuals tested in a time period. On introducing ω we are no longer able to consider v as representative of the incubation period. The length of the incubation period is now

$$
\begin{aligned}
X &= \frac{1}{\omega} + \frac{1}{v} \\
  &= \frac{v + \omega}{v\omega}.
\end{aligned}
\tag{5.1}
$$

The average incubation period, labelled X, represents the average time from infection, i.e. entry into $I(t)$, to the time of development of full-blown AIDS. In the last section, we looked at an average incubation period of 8 years. We will continue using this value throughout the remainder of this discussion. However, we must keep in mind that now v is dependent on ω. We wish to find the optimal affordable value for ω, in an attempt to reduce the spread of disease. The first of these three models, the SIPA model, looks at the effect of testing under the assumption that all individuals, once tested, behave responsibly. In this case, $\rho = 1$. We note that qualitatively, the behaviour of this model, for any specific value of ω, is similar to that for the SQA model. However, since we can vary the value for ω through intervention, we have more control over the outcome of the AIDS epidemic. Refer to the graph in figure 5.11. The SIQA model provides us with the opposite extreme to the SIPA model, in that for this model we consider $\rho = 1$. The results are as expected. Regardless of the value of ω the SIPA model is more likely to attain the disease free state than the SIQA model, since $\mu + \omega > \frac{(\mu+\omega)(\mu+v)}{\mu+v+\omega}$.

Finally, we consider the SIQ/PA model, in which we combine the two parameters $0 < \rho < 1$ and $0 < \omega < 1$. The SIQPA and SIQ/PA models have similar

Figure 5.11: Graph of SIPA Model Results

CHAPTER 5. DISCUSSION   82
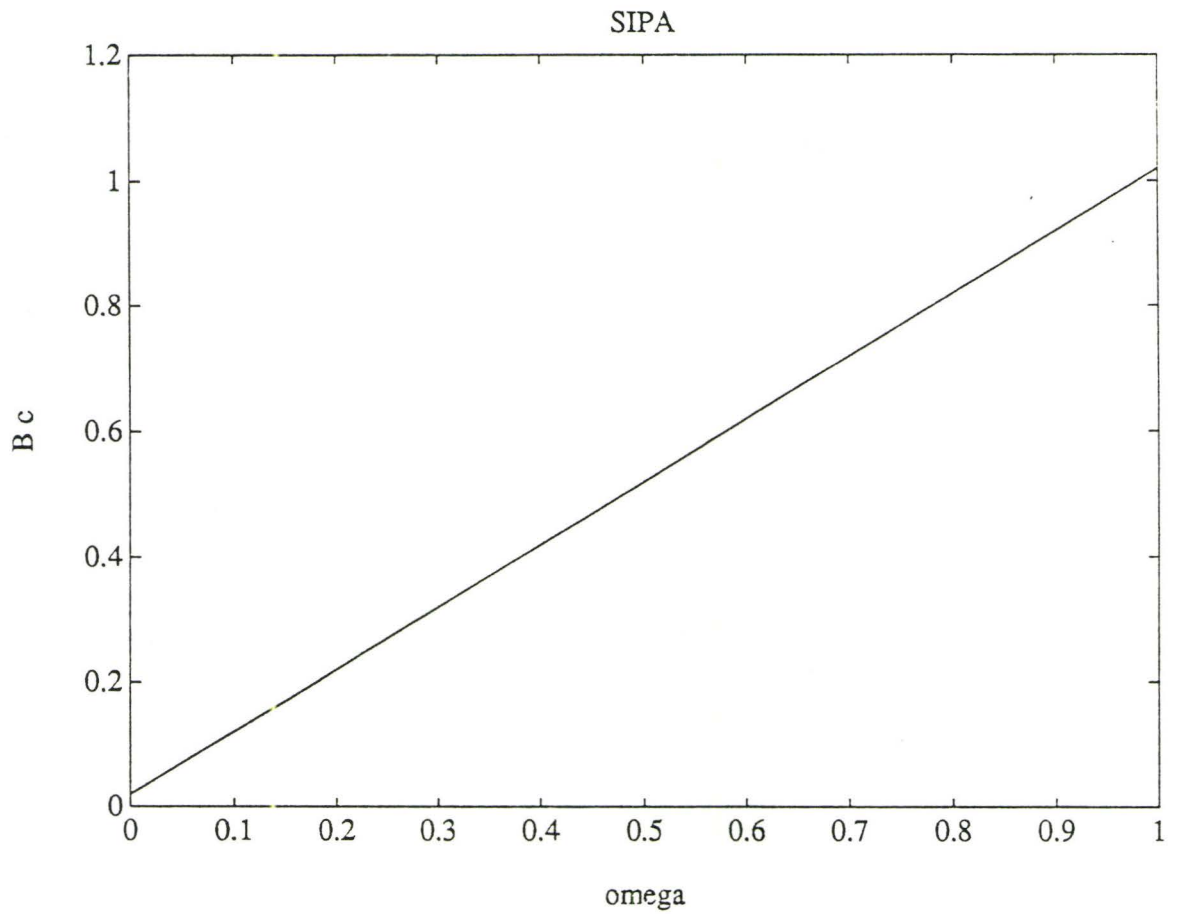
results, so we only consider the latter in this discussion, as we have stronger results for the SIQ/PA in that we proved persistence of the epidemic within a certain range of the parameters, for the SIQ/PA and not for the SIQPA. As was mentioned in the SIQ/PA section, we eliminate the P and A classes from the contributing sexually active population and hence we decouple the P and A populations from the S, I, and Q populations. This simplification was done primarily for two reasons, the first being a simplification of the analysis, and secondly for a more realistic approach in approximating the actual numbers of sexually active and contributing offenders. The criterion for the SIQ/PA model is $\frac{\beta c(\mu+v+(1-\rho)\omega)}{(\mu+v)(\mu+\omega)}$. A graph of the criterion for the SIQ/PA model is provided in figure 5.12.

The graph indicates that our chances of achieving a disease free state increase as ω and ρ increase. By more closely examining this graph, we observe that there is a greater sensitivity in flexibility as ρ approaches 1. For ω values, there is more sensitivity to changes when ω is closer to 0.2 or 0.3. This implies that perhaps we should focus on making more responsible the individuals who have been tested, rather than testing more untested individuals. This intersting observation deserves further attention in future modelling efforts.

In considering the optimal values for ρ and ω we must consider practicality issues. A benefit/cost analysis would provide us with a better idea of where the optimal practical values of these parameters would be. At this point, we are not equipped with sufficient resources to carry out an analysis of this type. But, for future uses, it is important to note that a study in this area would prove beneficial to the reduction in the spread of the AIDS epidemic. We require information about proportions of the infected populations who behave responsibly, and what effects education or other actions imposed on the tested infected populations might have on increasing the responsible infected population. On the other hand, we need information about the cost and practicality of testing large proportions of individuals. What is the likelihood of a home test being developed? Should there be target populations for testing, such as high risk groups or certain age groups? There are many factors entering into this analysis but these are well worth considering, since we have shown the importance of testing for HIV antibodies and for encouraging individuals to behave responsibly.

Figure 5.12: Graph of SIQ/PA Model Results
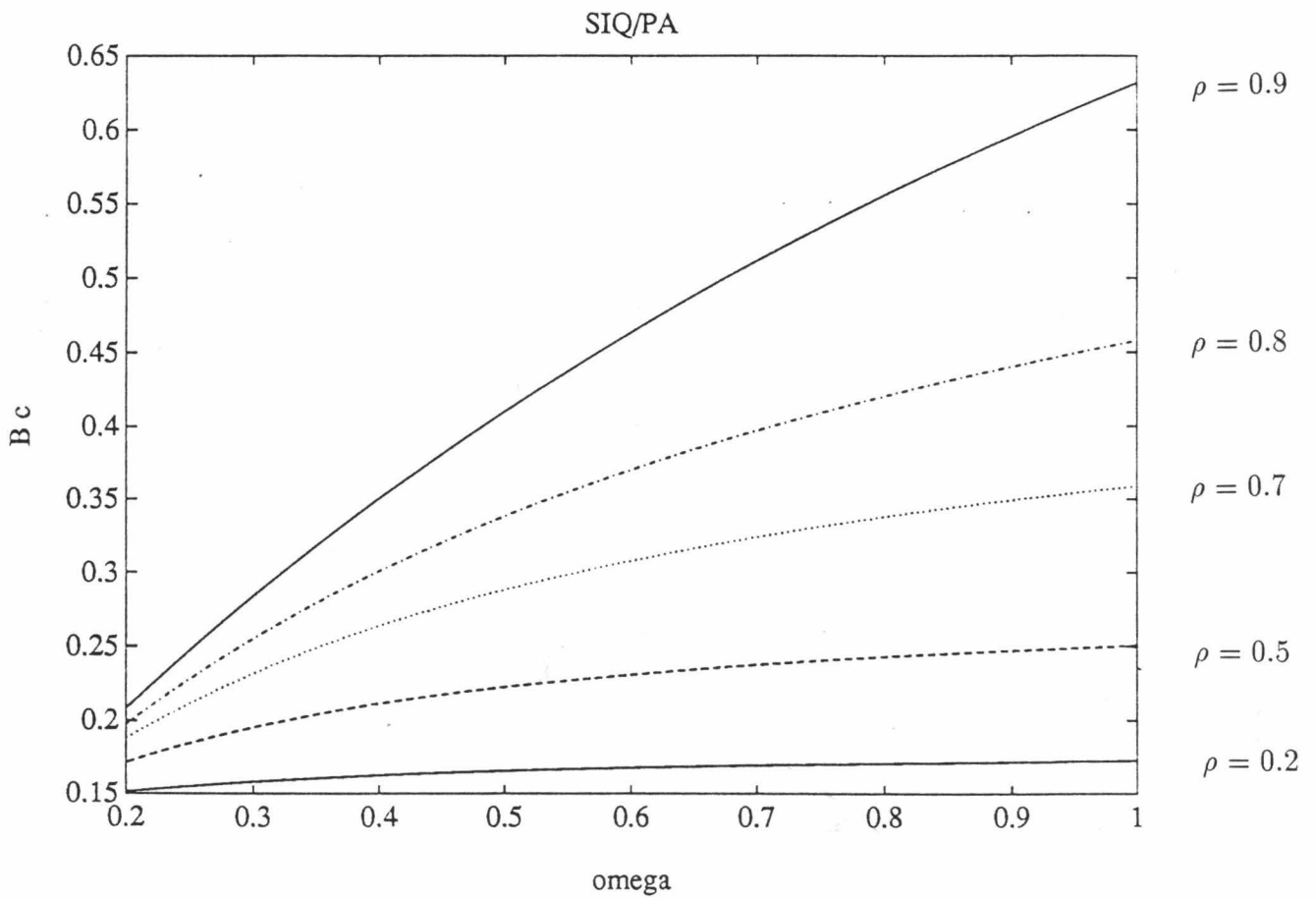
5.4   Summary

We have shown that a combination of testing for HIV antibodies and imposing persuasive techniques to encourage safe sexual behaviours of the tested infecteds plays a significant role in the reduction of the spread of AIDS. The extent of these benefits are not completely evident. However, we are better equipped to further study their effects. For a more complete analysis of the effects of HIV antibody testing in combination with various influencing techniques to increase the number of responsible tested infected individuals, we should consider the impact of various alternatives to the execution of these aspects. For instance, we would be interested in studying the outcomes of a voluntary versus compulsory nature of testing for HIV antibodies. The degree of confidentiality of test results would also contribute to the outcome of the disease. Further, means to enforce laws on individuals who fail to behave sexually responsible, should be examined for effectiveness. Many problems are intrinsic in these options. For instance, a voluntary, confidential testing regime, would probably motivate more individuals to become tested. Adding educational methods to this combination might result in increasing the responsibility of the sexually active tested population. However, if the test results are confidential between doctor and patient, then attempts at educating individuals would be in vain. This would lead to a larger Q class. If testing was compulsory and complete disclosure of results by the doctor was required, then obviously a larger P class would be achieved, but the rights of individuals would be violated, and the cost could be extreme. Limiting the compulsory testing to certain risk groups may help this situation. Only in continuing our study can we gain some insight into the effects of these alternatives.

Our models are simplistic, and hence they require some refining to more completely simulate the disease in nature. For instance, in defining our Q and P classes, we established a clear cut division between responsible and irresponsible sexual behaviour for every sexual act. Hence, the I and Q populations behaved sexually identically. This may not necessarily be accurate. In future modelling efforts we must examine the very likely case of individuals from the Q class behaving irresponsibly only part

of the time. For instance, an individual in the Q class may, on average, engage in unsafe sexual practices in only one out of every ten sexual contacts. Compensating for this would in essence have a positive effect on the disease outcome. We might solve this problem by combining the Q and P classes while considering a density function incorporating a continuous drop in activity levels as the disease symptoms become more evident. Another alternative is to divide the infecteds into progressively less sexually active groups. The number of groups would depend on the number of different activity levels required to satisfy our modelling efforts.

In future modelling efforts we must consider the effects of variable infectivity. Including this element of uncertainty, could significantly alter the optimal proportion of infecteds to be tested, and hence the outcome of the epidemic.

A natural extension of our models would also include incorporation of various risk groups. Within the susceptible population, there are individuals whose behaviour invites infection. For instance, IV drug users are at high risk of contracting the HIV virus. The inclusion of risk groups in our models would provide a more complete understanding of the actual dynamics of AIDS. If we are aware of which individuals are at highest risk, we can focus our testing efforts on these groups, thus cutting down unnecessary costs.

These and other revisions to our models can now be pursued more easily since we have an understanding of the basic dynamics of the AIDS epidemic with respect to HIV antibody testing and persuasive techniques encouraging responsible sexual behaviour of tested infecteds. With combined efforts and further study we hope that eradication of the acquired immunodeficiency syndrome will become a reality.

Appendix A

The SPA and SPA$_\epsilon$ Models

The calculations required for the SPA and most of the SPA$_\epsilon$ models are straight forward and a listing of calculations are not required. We do provide the calculations required for the solutions of the SPA$_\epsilon$ model. The model is found in equations 3.6

Applying the integrating factor technique we first find the solution for $S(t)$.

$$e^{(\mu+\epsilon)t}S(t) = \int \Lambda e^{(\mu+\epsilon)t}dt$$

$$= \frac{\Lambda}{\mu+\epsilon}e^{(\mu+\epsilon)t} + c_1.$$

Thus,

$$S(t) = \frac{\Lambda}{\mu+\epsilon} + c_1 e^{-(\mu+\epsilon)t} \qquad \text{where}$$

$$c_1 = S_o - \frac{\Lambda}{\mu+\epsilon}$$

Using the solution for $S(t)$ we now solve for $P(t)$.

$$e^{(\mu+v)t}P(t) = \int \epsilon e^{(\mu+v)t}S(t)dt$$

$$= \int \frac{\epsilon\Lambda}{\mu+\epsilon}e^{(\mu+v)t} + c_1\epsilon e^{v-\epsilon)t}dt$$

$$= \frac{\epsilon\Lambda}{(\mu+\epsilon)(\mu+v)}e^{(\mu+v)t} + \frac{c_1\epsilon}{v-\epsilon}e^{v-\epsilon)t} + c_2.$$

Thus,

$$P(t) = \frac{\epsilon\Lambda}{(\mu+\epsilon)(\mu+v)} + \frac{c_1\epsilon}{v-\epsilon}e^{-(\mu+\epsilon)t} + c_2e^{-(\mu+v)t} \qquad \text{where}$$

$$c_2 = P_o - \frac{\epsilon\Lambda}{(\mu+\epsilon)(\mu+v)} - \frac{c_1\epsilon}{v-\epsilon}$$

Combining the solutions for $S(t)$ and $P(t)$ we solve for $A(t)$.

$$e^{(\mu+d)t}A(t) = \int v\left(\frac{\epsilon\Lambda}{(\mu+\epsilon)(\mu+v)} + \frac{c_1\epsilon}{v-\epsilon}e^{-(\mu+\epsilon)t} + c_2e^{-(\mu+v)t}\right)e^{(\mu+d)t}dt$$

$$= \frac{v\epsilon\Lambda}{(\mu+v)(\mu+\epsilon)(\mu+d)}e^{(\mu+d)t} + \frac{v\epsilon c_1}{(v-\epsilon)(d-\epsilon)}e^{(d-\epsilon)t}$$

$$+\frac{vc_2}{d-v}e^{(d-v)t} + c_3$$

Therefore,

$$A(t) := \frac{v\epsilon\Lambda}{(\mu+v)(\mu+\epsilon)(\mu+d)} + \frac{v\epsilon c_1}{(v-\epsilon)(d-\epsilon)}e^{-(\mu+\epsilon)t}$$

$$+\frac{vc_2}{d-v}e^{-(\mu+v)t} + c_3e^{-(\mu+d)t} \qquad \text{where}$$

$$c_3 := A_o - \frac{v\epsilon\Lambda}{(\mu+v)(\mu+\epsilon)(\mu+d)} - \frac{v\epsilon c_1}{(v-\epsilon)(d-\epsilon)} - \frac{vc_2}{d-v}$$

Appendix B

The SQA Model

B.1 Equilibria

For simplicity, in this and all following appendices, the notation indicating dependence on t is to be assumed where appropriate. The SQA system given in equations 3.7, has two equilibrium points. We show how these points are derived. Setting $Q' = 0$ in equation 3.7 it follows that at equilibrium either

$$Q = 0$$

or

$$S = \frac{N(\mu + v)}{\beta c}. \tag{B.1}$$

B.1.1 Disease Free Equilibrium

When $Q = 0$, the equations 3.7 yield the equilibrium point

$$\begin{pmatrix} \hat{S} \\ \hat{Q} \\ \hat{A} \end{pmatrix} = \begin{pmatrix} \frac{\Lambda}{\mu} \\ 0 \\ 0 \end{pmatrix} \tag{B.2}$$

This fixed point represents a disease free situation and is called the disease free equilibrium.

B.1.2 The Endemic Equilibrium

When $Q \neq 0$, then $\bar{S} = \frac{\bar{N}(\mu+v)}{\beta c}$. Setting $S' = 0$ in 3.7 we solve for $\bar{Q}$:

$$
\begin{aligned}
S' &= \Lambda - \left(\mu + \frac{\beta c Q}{N}\right) S \\
&= \Lambda - \left(\mu + \frac{\beta c Q}{N}\right) \left(\frac{\bar{N}(\mu+v)}{\beta c}\right) \\
&= \Lambda - \frac{\mu(\mu+v)N}{\beta c} + (\mu+v)Q \\
&= 0,
\end{aligned}
$$

so that

$$
\bar{Q} = \frac{\Lambda}{\mu+v} - \frac{\mu\bar{N}}{\beta c}.
$$

Setting $A' = 0$ in equation 3.7 we obtain:

$$
\begin{aligned}
A' &= vQ - (\mu+d)A \\
&= 0
\end{aligned}
$$

and so

$$
\bar{A} = \left(\frac{v}{\mu+d}\right)\left(\frac{\Lambda}{\mu+v} - \frac{\mu\bar{N}}{\beta c}\right).
$$

The endemic equilibrium as a funtion of $\bar{N}$ is:

$$
\begin{pmatrix} \bar{S} \\ \\ \bar{Q} \\ \\ \bar{A} \end{pmatrix} = \begin{pmatrix} \frac{\bar{N}(\mu+v)}{\beta c} \\ \\ \frac{\Lambda}{\mu+v} - \frac{\bar{N}\mu}{\beta c} \\ \\ \left(\frac{v}{\mu+d}\right)\left(\frac{\Lambda}{\mu+v} - \frac{\bar{N}\mu}{\beta c}\right) \end{pmatrix} \tag{B.3}
$$

Using equation B.3 and $\bar{N} = \bar{S} + \bar{Q} + \bar{A}$ we derive an explicit expression for $\bar{N}$ in terms of the parameters of the model:

$$
\begin{aligned}
\bar{N} &= \frac{\bar{N}(\mu+v)}{\beta c} + \frac{\Lambda}{\mu+v} - \frac{\bar{N}\mu}{\beta c} + \left(\frac{v}{\mu+d}\right)\left(\frac{\Lambda}{\mu+v} - \frac{\bar{N}\mu}{\beta c}\right) \\
&= \bar{N}\left(\frac{\mu+v}{\beta c} - \frac{\mu}{\beta c} - \frac{\mu v}{(\mu+d)\beta c}\right) + \frac{\Lambda}{\mu+v}\left(1 + \frac{v}{\mu+d}\right) \\
&= \bar{N}\frac{vd}{(\mu+d)\beta c} + \frac{\Lambda}{\mu+v}\left(\frac{\mu+v+d}{\mu+d}\right)
\end{aligned}
$$

so that we obtain

$$
\bar{N} = \frac{\Lambda\beta c(\mu+v+d)}{(\mu+v)(\beta c(\mu+d) - vd)}. \tag{B.4}
$$

Substituting $\bar{N}$ in equation B.4, into equation B.3, we derive expressions for the endemic equilibrium in terms of the parameters of the system:

$$
\begin{aligned}
\bar{S} &= \left(\frac{\Lambda\beta c(\mu+v+d)}{(\mu+v)(\beta c(\mu+d) - vd)}\right)\left(\frac{\mu+v}{\beta c}\right) \\
&= \frac{\Lambda(\mu+v+d)}{\beta c(\mu+d) - vd},
\end{aligned}
$$

$$
\begin{aligned}
\bar{Q} &= \frac{\Lambda}{\mu+v} - \frac{\mu}{\beta c}\left(\frac{\Lambda\beta c(\mu+v+d)}{(\mu+v)(\beta c(\mu+d) - vd)}\right) \\
&= \left(\frac{\Lambda}{\mu+v}\right)\left(\frac{(\beta c - (\mu+v))(\mu+d)}{\beta c(\mu+d) - vd}\right),
\end{aligned}
$$

and

$$\bar{A} \ := \ \left(\frac{v}{\mu+d}\right)\left(\frac{\Lambda}{\mu+v}\frac{\mu}{\beta c}\right)\left(\frac{\Lambda\beta c(\mu+v+d)}{(\mu+v)(\beta c(\mu+d)-vd)}\right)$$

$$:= \ \left(\frac{\Lambda v}{\mu+v}\right)\left(\frac{\beta c-(\mu+v)}{\beta c(\mu+d)-vd}\right).$$

We record these in vector form:

$$\begin{pmatrix} \bar{S} \\ \\ \bar{Q} \\ \\ \bar{A} \end{pmatrix} = \begin{pmatrix} \frac{\Lambda(\mu+d+v)}{\beta c(\mu+d)-vd} \\ \\ \left(\frac{\Lambda}{\mu+v}\right)\left(\frac{(\beta c-(\mu+v))(\mu+d)}{\beta c(\mu+d)-vd}\right) \\ \\ \left(\frac{\Lambda v}{\mu+v}\right)\left(\frac{\beta c-(\mu+v)}{\beta c(\mu+d)-vd}\right) \end{pmatrix} \tag{B.5}$$

Note that the endemic equilibrium in equation B.5 exists if and only if $\beta c > \mu + v$. This inequality was derived by restricting the values of the components of the endemic equilibrium to the positive cone.

B.2   Local Stability Properties

It is useful, in the subsequent analysis, to note the relationships in the equilibrium, $(\bar{S}, \bar{Q}, \bar{A})$:

$$\bar{S} = \frac{(\mu+v)}{\beta c}\bar{N}, \quad \text{and} \quad \bar{Q} = \frac{(\mu+d)(\beta c-(\mu+v))}{\beta c(\mu+v+d)}\bar{N}.$$

Furthermore, to aid in simplifying calculations we use the substitutions,

$$\begin{aligned} m &= \mu+v, \\ n &= \mu+d, \\ M &= \beta c-(\mu+v), \\ D &= \beta c(\mu+v+d). \end{aligned} \tag{B.6}$$

We now proceed to look at the local analysis of these two fixed points. The Jacobian is:

$$J = \begin{pmatrix} \frac{\partial S'}{\partial S} & \frac{\partial S'}{\partial Q} & \frac{\partial S'}{\partial A} \\ \\ \frac{\partial Q'}{\partial S} & \frac{\partial Q'}{\partial Q} & \frac{\partial Q'}{\partial A} \\ \\ \frac{\partial A'}{\partial S} & \frac{\partial A'}{\partial Q} & \frac{\partial A'}{\partial A} \end{pmatrix}$$

$$= \begin{pmatrix} -\left(\mu + \frac{\beta c Q}{N^2}(N-S)\right) & -\frac{\beta c S}{N^2}(N-Q) & \frac{\beta c Q S}{N^2} \\ \\ \frac{\beta c Q}{N^2}(N-S) & \frac{\beta c S}{N^2}(N-Q) - (\mu+v) & -\frac{\beta c Q S}{N^2} \\ \\ 0 & v & -(\mu+d) \end{pmatrix}.$$

B.2.1 Disease Free Equilibrium

The Jacobian at the disease free equilibrium $\left(\frac{A}{\mu}, 0, 0\right)$ is:

$$J_{\left(\frac{A}{\mu},0,0\right)} = \begin{pmatrix} -\mu & -\beta c & 0 \\ 0 & \beta c - (\mu+v) & 0 \\ 0 & v & -(\mu+d) \end{pmatrix},$$

and thus,

$$|J_{\left(\frac{A}{\mu},0,0\right)} - \lambda I| = \begin{vmatrix} -(\mu+\lambda) & -\beta c & 0 \\ 0 & \beta c - (\mu+v+\lambda) & 0 \\ 0 & v & -(\mu+d+\lambda) \end{vmatrix}.$$

The eigenvalues for the disease free fixed point are:

$$\begin{aligned} \lambda_1 &= -\mu \\ \lambda_2 &= \beta c - (\mu+v) \\ \lambda_3 &= -(\mu+d) \end{aligned}$$

Since μ and d are positive, we need only be concerned with the sign of λ_2 for local stability of the point $(\frac{\Lambda}{\mu}, 0, 0)$. We conclude that the disease free equilibrium is locally asymptotically stable if

$$\beta c < \mu + v.$$

B.2.2   Endemic Equilibrium

The Jacobian at the endemic fixed point $(\bar{S}, \bar{Q}, \bar{A})$, using the substitutions B.6, is:

$$J_{(\bar{S},\bar{Q},\bar{A})} := \begin{pmatrix} -\left(\mu + \frac{\beta c \bar{Q}}{N^2}(\bar{N} - \bar{S})\right) & -\frac{\beta c \bar{S}}{N^2}(\bar{N} - \bar{Q}) & \frac{\beta c \bar{Q} \bar{S}}{N^2} \\ \frac{\beta c \bar{Q}}{N^2}(\bar{N} - \bar{S}) & \frac{\beta c \bar{S}}{N^2}(\bar{N} - \bar{Q}) - m & -\frac{\beta c \bar{Q} \bar{S}}{N^2} \\ 0 & v & -n \end{pmatrix}.$$

We continue by simplifying the components:

$$\begin{aligned} \frac{Q}{N} &= \frac{\Lambda}{mN} - \frac{\mu}{\beta c} \\ &= \frac{\Lambda}{m}\left(\frac{m(n\beta c - mn + \mu(m + d))}{\Lambda \beta c(m + d)}\right) - \frac{\mu}{\beta c} \\ &= \frac{n(\beta c - m)}{\beta c(m + d)} \\ &= \frac{nM}{D}. \end{aligned}$$

$$\begin{aligned} \frac{\beta c Q(N - S)}{N^2} &= \beta c \frac{Q}{N}\left(1 - \frac{S}{N}\right) \\ &= \beta c \frac{nM}{D}\left(\frac{\beta c - m}{\beta c}\right) \end{aligned}$$

$$= \frac{nM^2}{D}.$$

$$\frac{\beta c S(N-Q)}{N^2} = \frac{m}{D}(D-nM).$$

Therefore,

$$|J_{(S,Q,\bar{A})} - \lambda I| =$$

$$\begin{vmatrix} -(\mu + \frac{nM^2}{D} + \lambda) & -\frac{m}{D}(D-nM) & \frac{mnM}{D} \\ \\ \frac{nM^2}{D} & \frac{m}{D}(D-nM)-(m+\lambda) & -\frac{mnM}{D} \\ \\ 0 & v & -(n+\lambda) \end{vmatrix}$$

$$= -v\left(\left(\mu + \frac{nM^2}{D} + \lambda\right)\left(\frac{mnM}{D}\right) - \frac{n^2m^2M^3}{D^2}\right)$$
$$+ (n+\lambda)\left(\left(\mu + \frac{nM^2}{D} + \lambda\right)\left(\frac{m}{D}(D-nM)-(m+\lambda)\right)\right)$$
$$- (n+\lambda)\left(\frac{nM^2m}{D^2}(D-nM)\right).$$

This gives the characteristic equation

$$0 = \lambda^3 + \frac{nD + nM^2 + mnM + \mu D}{D}\lambda^2$$
$$+ \frac{vmnM + n^2M^2 + mn^2M + n\mu D + \mu mnM + nM^2m}{D}\lambda$$
$$+ \frac{vmnM\mu + \mu mn^2M + n^2M^2m}{D}.$$

We use the Routh-Hurwitz criteria (H.2) to determine the local stability of our system. The criteria for all the roots of a cubic polynomial to have their real parts negative is $a_0 > 0, a_1 > 0, a_2 > 0, a_3 > 0$ and in addition the determinant condition $a_1 a_2 - a_3 a_0 > 0$, where a_i for $i = 0, 1, 2, 3$ are the coefficients of the λ^{3-i} terms of the characteristic equation. Note that the parameters $\mu, v, d, m, n, M, D, \beta$, and c are all positive. Thus,

$$a_0 = 1 \qquad\qquad > 0,$$

$$a_1 = \frac{nD + nM^2 + mnM + \mu D}{D} \qquad\qquad > 0,$$

$$a_2 = \frac{vmnM + n^2 M^2 + mn^2 M + n\mu D + \mu mnM + nM^2 m}{D} \qquad > 0,$$

$$a_3 = \frac{vmnM\mu + \mu mn^2 M + n^2 M^2 m}{D} \qquad\qquad > 0,$$

and

$$a_1 a_2 - a_3 a_0 =$$

$$n\mu(n + \mu)$$
$$+ \frac{n^2 mM(v + n) + n^3 M^2 + 2n^2 M\mu(m + M) + n\mu mM(\mu + n + M)}{D}$$
$$+ \frac{n^2 M^3 m^2 + n^2 M^4(n + m) + 2n^3 M^3 m + n^2 m^2 M^2(M + m + n)}{D^2}$$
$$> 0.$$

Therefore the endemic equilibrium exists and is locally asymptotically stable if $\beta c > \mu + v$.

Appendix C

The SQPA Model

C.1 SQPA Equilibria

Calculations in this appendix refer to model 3.9. The equilibria for 3.9 are found by setting the differential equations to 0. Setting $Q'(t) = 0$ in 3.9 we have either

$$Q = 0,$$

or

$$S = \frac{N(\mu + v)}{(1 - \rho)\beta c}. \tag{C.1}$$

C.1.1 Disease Free Equilibrium

If $Q = 0$, then $\hat{S} = \frac{\Lambda}{\mu}$, $\hat{P} = 0$, and $\hat{A} = 0$. The disease free equilibrium is then,

$$\begin{pmatrix} \hat{S} \\ \hat{Q} \\ \hat{P} \\ \hat{A} \end{pmatrix} = \begin{pmatrix} \frac{\Lambda}{\mu} \\ 0 \\ 0 \\ 0 \end{pmatrix}.$$

C.1.2 The Endemic Equilibrium

In this section we perform the necessary calculations to determine the endemic equilibrium. If $Q \neq 0$, then by setting $S' = 0$ in 3.9, we can solve for $\bar{Q}$.

$$
\begin{aligned}
S' &= \Lambda - \left(\mu + \frac{\beta cQ}{N}\right) S \\[2ex]
&= \Lambda - \left(\mu + \frac{\beta cQ}{N}\right)\left(\frac{N(\mu + v)}{(1 - \rho)\beta c}\right) \\[2ex]
&= \Lambda - \frac{\mu N(\mu + v)}{(1 - \rho)\beta c} - \frac{Q(\mu + v)}{(1 - \rho)} \\[2ex]
&= 0,
\end{aligned}
$$

so that

$$
\begin{aligned}
\bar{Q} &= \frac{(1 - \rho)}{(\mu + v)}\left(\Lambda - \frac{\mu \bar{N}(\mu + v)}{(1 - \rho)\beta c}\right) \\[2ex]
&= \frac{(1 - \rho)\Lambda}{(\mu + v)} - \frac{\mu N}{\beta c}.
\end{aligned}
$$

Setting $P' = 0$ in equation (2.16) we obtain

$$
\begin{aligned}
P' &= \rho \frac{\beta cQS}{N} - (\mu + v)P \\[2ex]
&= \frac{\rho \beta c}{N}\left(\frac{N(\mu + v)}{(1 - \rho)\beta c}\right)\left(\frac{(1 - \rho)\Lambda}{\mu + v} - \frac{\mu N}{\beta c}\right) - (\mu + v)P \\[2ex]
&= \frac{\rho(\mu + v)}{(1 - \rho)}\left(\frac{(1 - \rho)\Lambda}{\mu + v}\right) - \frac{\rho(\mu + v)\mu N}{\beta c(1 - \rho)} - (\mu + v)P \\[2ex]
&= \rho \Lambda - \frac{\mu \rho N(\mu + v)}{\beta c(1 - \rho)} - (\mu + v)P
\end{aligned}
$$

$$= \rho \left(\frac{\Lambda\beta c(1-\rho) - \mu N(\mu+v)}{\beta c(1-\rho)} \right) - (\mu+v)P$$

$$= 0,$$

and hence

$$\bar{P} = \left(\frac{\rho}{\mu+v} \right) \left(\Lambda - \frac{\mu\bar{N}(\mu+v)}{\beta c(1-\rho)} \right)$$

$$= \rho \left(\frac{\Lambda}{\mu+v} - \frac{\mu\bar{N}}{\beta c(1-\rho)} \right).$$

$\bar{A}$ is derived by setting $A' = 0$ in equation 3.9, which gives:

$$\bar{A} = \frac{v}{\mu+d} \left(\bar{Q} + \bar{P} \right)$$

$$= \frac{v}{\mu+d} \left(\frac{(1-\rho)\Lambda}{\mu+v} - \frac{\mu\bar{N}}{\beta c} + \frac{\rho\Lambda}{\mu+v} - \frac{\rho\mu\bar{N}}{\beta c(1-\rho)} \right)$$

$$= \frac{v}{\mu+d} \left(\frac{\Lambda}{\mu+v} - \frac{\mu\bar{N}}{\beta c} \left(1 + \frac{\rho}{1-\rho} \right) \right)$$

$$= \frac{v}{\mu+d} \left(\frac{\Lambda}{\mu+v} - \frac{\mu\bar{N}}{\beta c(1-\rho)} \right).$$

Therefore, the endemic equilibrium, as a function of $\bar{N}$, is:

$$\begin{pmatrix} \bar{S} \\ \\ \bar{Q} \\ \\ \bar{P} \\ \\ \bar{A} \end{pmatrix} = \begin{pmatrix} \frac{\bar{N}(\mu+v)}{(1-\rho)\beta c} \\ \\ \frac{(1-\rho)\Lambda}{\mu+v} - \frac{\mu\bar{N}}{\beta c} \\ \\ \rho\left(\frac{\Lambda}{\mu+v} - \frac{\mu\bar{N}}{\beta c(1-\rho)} \right) \\ \\ \frac{v}{\mu+d}\left(\frac{\Lambda}{\mu+v} - \frac{\mu\bar{N}}{\beta c(1-\rho)} \right) \end{pmatrix} \qquad (C.2)$$

Using equation C.2 and the fact that $\bar{N} = \bar{S} + \bar{Q} + \bar{P} + \bar{A}$, we derive an explicit expression for $\bar{N}$ in terms of the parameters of the model:

$$
\begin{aligned}
\bar{N} &= \frac{\bar{N}(\mu + v)}{\beta c(1 - \rho)} + (1 - \rho)\frac{\Lambda}{\mu + v} - \frac{\mu \bar{N}}{\beta c} + \rho\left(\frac{\Lambda}{\mu + v} - \frac{\mu \bar{N}}{\beta c(1 - \rho)}\right) \\
&\quad + \frac{v}{\mu + d}\left(\frac{\Lambda}{\mu + v} - \frac{\mu \bar{N}}{\beta c(1 - \rho)}\right) \\
&= \bar{N}\left(\frac{\mu + v}{(1 - \rho)\beta c} - \frac{\mu}{\beta c} - \frac{\rho\mu}{\beta c(1 - \rho)} - \frac{\mu v}{\beta c(1 - \rho)(\mu + d)}\right) \\
&\quad + \frac{\Lambda}{\mu + v}\left(1 + \frac{v}{\mu + d}\right) \\
&= \bar{N}\left(\frac{(\mu + v)(\mu + d) - \mu(1 - \rho)(\mu + d) - \rho\mu(\mu + d) - \mu v}{\beta c(1 - \rho)(\mu + d)}\right) \\
&\quad + \frac{\Lambda(\mu + v + d)}{(\mu + v)(\mu + d)} \\
&= \frac{\Lambda(\mu + v + d)}{(\mu + v)(\mu + d)} + \bar{N}\left(\frac{vd}{\beta c(1 - \rho)(\mu + d)}\right)
\end{aligned}
$$

so that

$$
\bar{N}\left(1 - \frac{vd}{\beta c(\mu + d)(1 - \rho)}\right) = \frac{\Lambda(\mu + v + d)}{(\mu + v)(\mu + d)},
$$

and therefore,

$$
\begin{aligned}
\bar{N} &= \left(\frac{\Lambda(\mu + v + d)}{(\mu + v)(\mu + d)}\right)\left(\frac{(\mu + d)(1 - \rho)\beta c}{(\mu + d)(1 - \rho)\beta c - vd}\right) \\
&= \frac{\Lambda(\mu + v + d)(1 - \rho)\beta c}{(\mu + v)\left((\mu + d)(1 - \rho)\beta c - vd\right)}.
\end{aligned} \tag{C.3}
$$

We now substitute $\bar{N}$, as it is in equation C.3 into our endemic equilibrium in equation C.2, and we obtain the following expressions for the endemic equilibrium in terms of

the parameters of the model:

$$\bar{S} = \frac{\bar{N}(\mu + v)}{\beta c(1 - \rho)}$$

$$= \frac{\Lambda(\mu + v + d)}{(\mu + d)(1 - \rho)\beta c - vd}$$

$$\bar{Q} = \frac{(1 - \rho)\Lambda}{\mu + v} - \frac{\mu\bar{N}}{\beta c}$$

$$= \frac{(1 - \rho)\Lambda}{\mu + v} - \frac{\mu}{\beta c}\left(\frac{\Lambda(\mu + v + d)(1 - \rho)\beta c}{(\mu + v)((\mu + d)(1 - \rho)\beta c - vd)}\right)$$

$$= \Lambda\left(\frac{(1 - \rho)((\mu + d)(1 - \rho)\beta c - vd) - \mu(\mu + v + d)(1 - \rho)}{(\mu + v)((\mu + d)(1 - \rho)\beta c - vd)}\right)$$

$$= \Lambda(1 - \rho)\left(\frac{\mu\beta c + d\beta c - \mu\rho\beta c - d\rho\beta c - vd - \mu^2 - \mu v - \mu d}{(\mu + v)((\mu + d)(1 - \rho)\beta c - vd)}\right)$$

$$= \Lambda(1 - \rho)\left(\frac{-(\mu + d)(\mu + v) - \rho\beta c(\mu + d) + \beta c(\mu + d)}{(\mu + v)((\mu + d)(1 - \rho)\beta c - vd)}\right)$$

$$= \Lambda\left(\frac{((1 - \rho)\beta c - (\mu + v))(\mu + d)(1 - \rho)}{(\mu + v)((\mu + d)(1 - \rho)\beta c - vd)}\right)$$

$$\bar{P} =: \frac{\rho}{1 - \rho}\bar{Q}$$

$$=: \frac{\Lambda\rho}{(\mu + v)}\left(\frac{(\mu + d)((1 - \rho)\beta c - (\mu + v))}{(\mu + d)(1 - \rho)\beta c - vd}\right)$$

$$\bar{A} = \frac{v}{\rho(\mu + d)}\bar{P}$$

$$= \frac{\Lambda v}{(\mu + v)}\left(\frac{(1 - \rho)\beta c - (\mu + v)}{(\mu + d)(1 - \rho)\beta c - vd}\right)$$

Therefore, the endemic equilibrium is given by:

$$
\begin{pmatrix} \bar{S} \\\\ \bar{Q} \\\\ \bar{P} \\\\ \bar{A} \end{pmatrix} = \begin{pmatrix} \dfrac{\Lambda(\mu+v+d)}{(\mu+d)(1-\rho)\beta c - vd} \\\\ \left(\dfrac{\Lambda(\mu+d)(1-\rho)}{(\mu+v)}\right)\left(\dfrac{(1-\rho)\beta c-(\mu+v)}{(\mu+d)(1-\rho)\beta c - vd}\right) \\\\ \left(\dfrac{\Lambda\rho(\mu+d)}{\mu+v}\right)\left(\dfrac{(1-\rho)\beta c-(\mu+v)}{(\mu+d)(1-\rho)\beta c - vd}\right) \\\\ \left(\dfrac{\Lambda v}{\mu+v}\right)\left(\dfrac{(1-\rho)\beta c-(\mu+v)}{(\mu+d)(1-\rho)\beta c - vd}\right) \end{pmatrix} \tag{C.4}
$$

The endemic equilibrium exists in the positive cone if and only if all components of the endemic equilibrium are positive. This is satisfied if $\bar{I} > 0$, or equivalently if $\beta c > \frac{\mu+v}{1-\rho}$.

C.2 Local stability analysis

In the subsequent analysis we use the above information, the substitutions

$$
\begin{aligned}
m &= \mu + v \\
n &= \mu + d \\
D &= \beta c(\mu + v + d) \\
T &= \beta c(1 - \rho) - m,
\end{aligned} \tag{C.5}
$$

and the relations

$$
\begin{aligned}
\bar{S} &= \frac{\bar{N}(\mu + v)}{(1 - \rho)\beta c} \\\\
&= \frac{\bar{N}m}{(1 - \rho)\beta c}, \text{ and}
\end{aligned}
$$

$$\frac{\bar{Q}}{\bar{N}} = \left(\frac{(1-\rho)\Lambda}{(\mu+v)\bar{N}} - \frac{\mu}{\beta c} \right)$$

$$= \frac{(1-\rho)\Lambda}{m} \left(\frac{m(n(1-\rho)\beta c - vd)}{\Lambda(1-\rho)D} \right) - \frac{\mu}{\beta c}$$

$$= \frac{n(1-\rho)\beta c - vd}{D} - \frac{\mu}{\beta c}$$

$$= \frac{\beta cn(1-\rho) - vd - \mu(\mu+v+d)}{D}$$

$$= \frac{nT}{D}.$$

Therefore, we can simplify:

$$\frac{\beta c \bar{Q}(\bar{N} - \bar{S})}{\bar{N}^2} = \beta c \left(\frac{\bar{Q}}{\bar{N}} \right) \left(1 - \frac{\bar{S}}{\bar{N}} \right)$$

$$= \left(\frac{nT}{D} \right) \left(\frac{\beta c(1-\rho) - m}{1-\rho} \right)$$

$$= \frac{nT^2}{(1-\rho)D},$$

$$\frac{\beta c \bar{Q} \bar{S}}{\bar{N}^2} = \beta c \left(\frac{Tn}{D} \right) \left(\frac{m}{(1-\rho)\beta c} \right)$$

$$= \frac{Tnm}{D(1-\rho)},$$

$$\frac{\beta c \bar{S}(\bar{N} - \bar{Q})}{\bar{N}^2} = \beta c \left(\frac{m}{(1-\rho)\beta c} \right) \left(1 - \frac{Tn}{D} \right)$$

$$= \frac{m}{(1-\rho)} \left(\frac{D - Tn}{D} \right).$$

We are now able to perform a linear analysis on the system of equations in 3.9. The Jacobian is:

$$
J = \begin{pmatrix}
\frac{\partial S'}{\partial S} & \frac{\partial S'}{\partial Q} & \frac{\partial S'}{\partial P} & \frac{\partial S'}{\partial A} \\[2ex]
\frac{\partial Q'}{\partial S} & \frac{\partial Q'}{\partial Q} & \frac{\partial Q'}{\partial P} & \frac{\partial Q'}{\partial A} \\[2ex]
\frac{\partial P'}{\partial S} & \frac{\partial P'}{\partial Q} & \frac{\partial P'}{\partial P} & \frac{\partial P'}{\partial A} \\[2ex]
\frac{\partial A'}{\partial S} & \frac{\partial A'}{\partial Q} & \frac{\partial A'}{\partial P} & \frac{\partial A'}{\partial A}
\end{pmatrix}
$$

$$
= \begin{pmatrix}
-\mu - \frac{\beta c Q(N-S)}{N^2} & -\frac{\beta c S(N-Q)}{N^2} & \frac{\beta c Q S}{N^2} & \frac{\beta c Q S}{N^2} \\[2ex]
\frac{\beta c (1-\rho) Q(N-S)}{N^2} & \frac{\beta c (1-\rho) S(N-Q)}{N^2} - m & -\frac{\beta c (1-\rho) Q S}{N^2} & -\frac{\beta c (1-\rho) Q S}{N^2} \\[2ex]
\frac{\beta c \rho Q S(N-S)}{N^2} & \frac{\beta c \rho S(N-Q)}{N^2} & -\frac{\beta c \rho Q S}{N^2} - m & -\frac{\beta c \rho Q S}{N^2} \\[2ex]
0 & v & v & -n
\end{pmatrix}.
$$

C.2.1 Disease Free Equilibrium

At the disease free equilibrium $(\frac{\Lambda}{\mu}, 0, 0, 0)$ the Jacobian is:

$$
J_{(\frac{\Lambda}{\mu}, 0, 0, 0)} = \begin{pmatrix}
-\mu & -\beta c & 0 & 0 \\
0 & (1-\rho)\beta c - (\mu + v) & 0 & 0 \\
0 & \rho \beta c & -(\mu + v) & 0 \\
0 & v & v & -(\mu + d)
\end{pmatrix},
$$

so that

$$|J_{(\frac{A}{\mu},0,0,0)} - \lambda I| =$$

$$\begin{vmatrix} -(\mu + \lambda) & -\beta c & 0 & 0 \\ 0 & (1-\rho)\beta c - (\mu + v + \lambda) & 0 & 0 \\ 0 & \rho\beta c & -(\mu + v + \lambda) & 0 \\ 0 & v & v & -(\mu + d + \lambda) \end{vmatrix}$$

$$= -(\mu + \lambda) \begin{vmatrix} (1-\rho)\beta c - (\mu + v + \lambda) & 0 & 0 \\ \rho\beta c & -(\mu + v + \lambda) & 0 \\ v & v & -(\mu + d + \lambda) \end{vmatrix}.$$

This is a triangular matrix, so the eigenvalues can be determined directly from the diagonal elements. Hence the characteristic equation is

$$(\mu + \lambda)\left((1-\rho)\beta c - (\mu + v) - \lambda\right)\left((\mu + v) + \lambda\right)\left((\mu + d) + \lambda\right) = 0$$

which yields the following eigenvalues

$$\begin{aligned} \lambda_1 &= -\mu \\ \lambda_2 &= (1-\rho)\beta c - (\mu + v) \\ \lambda_3 &= -(\mu + v) \\ \lambda_4 &= -(\mu + d) \end{aligned}$$

The eigenvalues are real and with the exception of λ_3, are all negative. Therefore, we must impose a restriction that will make $\lambda_3 < 0$, to guarantee local stability of the disease free equilibrium. To this end, we require

$$\beta c < \frac{\mu + v}{1 - \rho}.$$

C.2.2   Endemic Equilibrium

The Jacobian at the endemic equilibrium $(\bar{S}, \bar{Q}, \bar{P}, \bar{A})$ is provided below using substitutions C.5.

$$
J_{(\bar{S},\bar{Q},\bar{P},\bar{A})} =
\begin{pmatrix}
-\mu - \frac{nT^2}{(1-\rho)D} & -\frac{m}{(1-\rho)}\left(\frac{D-nT}{D}\right) & \frac{mnT}{(1-\rho)D} & \frac{mnT}{(1-\rho)D} \\[2mm]
\frac{nT^2}{D} & m\left(\frac{D-nT}{D}\right) - m & -\frac{mnT}{D} & -\frac{mnT}{D} \\[2mm]
\frac{\rho nT^2}{(1-\rho)D} & \frac{\rho m}{(1-\rho)}\left(\frac{D-nT}{D}\right) & -\frac{\rho mnT}{(1-\rho)D} - m & -\frac{\rho mnT}{(1-\rho)D} \\[2mm]
0 & v & v & -n
\end{pmatrix}
$$

The characteristic equation, using row and column operations to simplify the form of the determinant, is derived as follows:

$$
\left| J_{(\bar{S},\bar{Q},\bar{P},\bar{A})} - \lambda I \right| =
$$

$$
\begin{vmatrix}
-\mu - \frac{nT^2}{(1-\rho)D} - \lambda & -\frac{m}{(1-\rho)}\left(\frac{D-nT}{D}\right) & \frac{mnT}{(1-\rho)D} & \frac{mnT}{(1-\rho)D} \\[2mm]
\frac{nT^2}{D} & m\left(\frac{D-nT}{D}\right) - m - \lambda & -\frac{mnT}{D} & -\frac{mnT}{D} \\[2mm]
\frac{\rho nT^2}{(1-\rho)D} & \frac{\rho m}{(1-\rho)}\left(\frac{D-nT}{D}\right) & -\frac{\rho mnT}{(1-\rho)D} - m - \lambda & -\frac{\rho mnT}{(1-\rho)D} \\[2mm]
0 & v & v & -n - \lambda
\end{vmatrix}
$$

$$
(C3 \leftarrow C3 - C4)
$$

$$
= -
\begin{vmatrix}
-\mu - \frac{nT^2}{(1-\rho)D} - \lambda & -\frac{m}{(1-\rho)}\left(\frac{D-nT}{D}\right) & 0 & \frac{mnT}{(1-\rho)D} \\[2mm]
\frac{nT^2}{D} & m\left(\frac{D-nT}{D}\right) - m - \lambda & 0 & -\frac{mnT}{D} \\[2mm]
\frac{\rho nT^2}{(1-\rho)D} & \frac{\rho m}{(1-\rho)}\left(\frac{D-nT}{D}\right) & -m - \lambda & -\frac{\rho mnT}{(1-\rho)D} \\[2mm]
0 & v & v + n + \lambda & -n - \lambda
\end{vmatrix}
$$

$$(R3 \leftarrow -R3 - R1)$$

$$= - \begin{vmatrix} -\mu - \frac{nT^2}{(1-\rho)D} - \lambda & -\frac{m}{(1-\rho)}\left(\frac{D-nT}{D}\right) & 0 & \frac{mnT}{(1-\rho)D} \\ \frac{nT^2}{D} & m\left(\frac{D-nT}{D}\right) - m - \lambda & 0 & -\frac{mnT}{D} \\ \mu + \lambda + \frac{nT^2}{D} & m\left(\frac{D-nT}{D}\right) & m + \lambda & -\frac{mnT}{D} \\ 0 & v & v + n + \lambda & -n - \lambda \end{vmatrix}$$

$$(R3 \leftarrow R3 - R2)$$

$$= - \begin{vmatrix} -\mu - \frac{nT^2}{(1-\rho)D} - \lambda & -\frac{m}{(1-\rho)}\left(\frac{D-nT}{D}\right) & 0 & \frac{mnT}{(1-\rho)D} \\ \frac{nT^2}{D} & m\left(\frac{D-nT}{D}\right) - m - \lambda & 0 & -\frac{mnT}{D} \\ \mu + \lambda & m + \lambda & m + \lambda & 0 \\ 0 & v & v + n + \lambda & -n - \lambda \end{vmatrix}$$

$$(C2 \leftarrow C2 - C3)$$

$$= - \begin{vmatrix} -\mu - \frac{nT^2}{(1-\rho)D} - \lambda & -\frac{m}{(1-\rho)}\left(\frac{D-nT}{D}\right) & 0 & \frac{mnT}{(1-\rho)D} \\ \frac{nT^2}{D} & m\left(\frac{D-nT}{D}\right) - m - \lambda & 0 & -\frac{mnT}{D} \\ \mu + \lambda & 0 & m + \lambda & 0 \\ 0 & -n - \lambda & v + n + \lambda & -n - \lambda \end{vmatrix}$$

$$(C2 \leftarrow C2 - C4)$$

$$= - \begin{vmatrix} -\mu - \frac{nT^2}{(1-\rho)D} - \lambda & -\frac{m}{(1-\rho)} & 0 & \frac{mnT}{(1-\rho)D} \\ \frac{nT^2}{D} & -\lambda & 0 & -\frac{mnT}{D} \\ \mu + \lambda & 0 & m + \lambda & 0 \\ 0 & 0 & v + n + \lambda & -n - \lambda \end{vmatrix}.$$

The characteristic equation and the Routh-Hurwitz criteria (H.2), are derived with the use of MAPLE. A program listing and results are provided for your perusal in figures C.13 and C.14.

Figure C.13: Program for Routh-Hurwitz criteria: SQPA

```
#
#The determinant elements, where s=1-p.
#
a1:=-u-n*T^2/(s*D)-r:
a2:=-m/s:
a3:=0:
a4:=T*n*m/(s*D):
b1:=n*T^2/D:
b2:=-r:
b3:=0:
b4:=-T*n*m/D:
c1:=u+r:
c2:=0:
c3:=m+r:
c4:=0:
d1:=0:
d2:=0:
d3:=m+d+r:
d4:=-n-r:
#
#Calculate the characteristic equation and simplify.
#We can factor out (m+r).
#
ceqn:=simplify(-d3*(c1*(a2*b4-b2*a4))+c3*(-(n+r)*(a1*b2-b1*a2))):
ceqn:=ceqn*s*D/(m+r):
ceqn:=collect(ceqn,r);
#
#The coefficients of the characteristic equation.
#
k0:=-coeff(ceqn,r,3);
k1:=-coeff(ceqn,r,2);
k2:=-coeff(ceqn,r,1);
k3:=-coeff(ceqn,r,0);
#
#The final Routh-Hurwitz condition, k1k2-k3k0.
#
rhc:=simplify(k1*k2-k3*k0);
quit
```

Note that a factor of $m + \lambda$ is contained in the characteristic equation, and so we only need to use the Routh-Hurwitz criteria for a cubic polynomial. The characters used in the program are not identical to those used in the analysis. These substitutions include those listed in C.5, the symbols ki, for $i = 0, 1, 2, 3$ corresponding to the coefficients of λ^{3-i}, as well as the substitutions

$$
\begin{aligned}
u &= \mu, \\
r &= \lambda, \text{ and} \\
s &= (1 - \rho).
\end{aligned}
$$

Since the Routh-Hurwitz criteria are satisfied, we can conclude that the endemic equilibrium is locally asymptotically stable provided $\beta c > \frac{\mu + v}{1 - \rho}$.

Figure C.14: Results for Routh-Hurwitz criteria: SQPA

```
#Calculate the characteristic equation and simplify.
#We can factor out (m+r).
#
              2          2 2
ceqn := - m  u T n - n  T  m - d u m T n

                           2                    2 2     2
   +(-d m T n - u m T n - m T n - n u s D - n T  - n T m) r

                         2          2          3
    + (- n s D - u s D - n T  - m T n) r  - s D r
#
#The coefficients of the characteristic equation.
#
                  k0 := s D
                                  2
          k1 := n s D + u s D + n T  + m T n

                            2                  2 2       2
k2 := d m T n + u m T n + m  T n + n u s D + n  T  + n T  m

              2        2 2
          k3 := m  u T n + n  T  m + d u m T n
#
#The final Routh-Hurwitz condition.
#
      2              2              2       2       2 2   2
rhc:=n s D d m T + 2 n s D u m T + n s D m T + n s D u

    3   2   2           2 2 2          2 2
 + n s D T  + u s D m T n + u s D n + 2 u s D n T

          2     2 3        2 3          2 3 2     3 4
 + u s D n T m + n T d m + n T u m + 2 n T m  + n T

     2 4      2 2 2      2 2 2      3 2 2       3 3
   + n T m + m T n d + m T n u + m T n  + m T n
#
# All terms are positive.
#
```

Appendix D

The SIPA Model

D.1   Equilibria

The calculations in this Appendix are for the model appearing in equations 4.1. We begin our linear analysis by solving for the equilibria. We use the substitutions:

$$
\begin{aligned}
m &= \mu + v \\
n &= \mu + d \\
l &= \mu + \omega \\
X &= mn + n\omega + v\omega
\end{aligned}
\tag{D.1}
$$

Setting $I' = 0$, in equation 4.1, we obtain:

$$
\left(\frac{\beta c S}{N} - (\mu + \omega) \right) I = 0
$$

so that we have only

$$
I = 0 \qquad \text{or} \qquad S = \frac{Nl}{\beta c}
$$

D.1.1   The Disease Free Equilibrium

If $I = 0$ then $\hat{P} = 0$, $\hat{A} = 0$ and $\hat{S} = \frac{\Lambda}{\mu}$ so that the disease free equilibrium is:

$$
\begin{pmatrix} \hat{S} \\ \hat{I} \\ \hat{P} \\ \hat{A} \end{pmatrix} = \begin{pmatrix} \frac{\Lambda}{\mu} \\ 0 \\ 0 \\ 0 \end{pmatrix}
$$

D.1.2   The Endemic Equilibrium

If $I \neq 0$ then $\bar{S} = \frac{Nl}{\beta c}$. Setting $S' = 0$, in equation 4.1, we have:

$$
\begin{aligned}
0 &= \Lambda - \left(\mu + \frac{\beta cI}{N} \right) \frac{Nl}{\beta c} \\[2mm]
&= \Lambda - \frac{\mu Nl}{\beta c} - Il
\end{aligned}
$$

so that,

$$
\bar{I} = \frac{\Lambda}{l} - \frac{\mu \bar{N}}{\beta c}
$$

Setting $P' = 0$, in equation 4.1, we obtain:

$$
\omega I = (\mu + v)P \tag{D.2}
$$

so that

$$
\begin{aligned}
\bar{P} &= \frac{\omega \bar{I}}{m} \\[2mm]
&= \frac{\omega}{m} \left(\frac{\Lambda}{l} - \frac{\mu \bar{N}}{\beta c} \right)
\end{aligned}
$$

Setting $A' = 0$, in equation 4.1, we have:

$$
vP = (\mu + d)A
$$

and hence

$$\bar{A} \;=\; \frac{v\omega I}{mn}$$

$$=\; \frac{v\omega}{mn}\left(\frac{\Lambda}{l} - \frac{\mu\bar{N}}{\beta c}\right)$$

The endemic equilibrium, as a function of $\bar{N}$, is then:

$$\begin{pmatrix} \bar{S} \\\\ \bar{I} \\\\ \bar{P} \\\\ \bar{A} \end{pmatrix} = \begin{pmatrix} \frac{\bar{N}l}{\beta c} \\\\ \frac{\Lambda}{l} - \frac{\mu\bar{N}}{\beta c} \\\\ \frac{\omega}{m}\left(\frac{\Lambda}{l} - \frac{\mu\bar{N}}{\beta c}\right) \\\\ \frac{v\omega}{mn}\left(\frac{\Lambda}{l} - \frac{\mu\bar{N}}{\beta c}\right) \end{pmatrix} \tag{D.3}$$

Using equation D.3, we obtain a value for $\bar{N}$ in terms of the parameters of the system:

$$\bar{N} \;=\; \bar{S} + \bar{I} + \bar{P} + \bar{A}$$

$$=\; \frac{\bar{N}l}{\beta c} + \left(1 + \frac{\omega}{m} + \frac{v\omega}{mn}\right)\left(\frac{\Lambda}{l} - \frac{\mu\bar{N}}{\beta c}\right)$$

$$=\; \bar{N}\left(\frac{l}{\beta c} - \frac{\mu}{\beta c}\left(1 + \frac{\omega}{m} + \frac{v\omega}{mn}\right)\right) + \frac{\Lambda}{l}\left(1 + \frac{\omega}{m} + \frac{v\omega}{mn}\right)$$

$$=\; \bar{N}\left(\frac{vd\omega}{\beta cmn}\right) + \frac{\Lambda}{l}\left(\frac{mn + n\omega + v\omega}{mn}\right).$$

Let X be as in equation D.1 so that:

$$\bar{N} \;=\; \frac{\Lambda}{l}\left(\frac{\beta cX}{\beta cmn - vd\omega}\right) \tag{D.4}$$

We now can solve, using equation D.4 the coordinates of the endemic equilibrium, in equation D.3 in terms of the parameters of the system.

$$
\begin{aligned}
\bar{S} &= \frac{\bar{N}l}{\beta c} \\
&= \frac{\Lambda X}{\beta cmn - vd\omega},
\end{aligned}
\tag{D.5}
$$

$$
\begin{aligned}
\bar{I} &= \frac{\Lambda}{l} - \frac{\mu\bar{N}}{\beta c} \\
&= \frac{\Lambda}{l}\left(\frac{\beta cmn - vd\omega - \mu mn - \mu n\omega - \mu v\omega}{\beta cmn - vd\omega}\right) \\
&= \frac{\Lambda mn}{l}\left(\frac{\beta c - l}{\beta cmn - vd\omega}\right),
\end{aligned}
\tag{D.6}
$$

$$
\begin{aligned}
\bar{P} &= \frac{\omega}{m}\left(\frac{\Lambda}{l} - \frac{\mu\bar{N}}{\beta c}\right) \\
&= \frac{\Lambda n\omega}{l}\left(\frac{\beta c - l}{\beta cmn - vd\omega}\right),
\end{aligned}
\tag{D.7}
$$

and

$$
\begin{aligned}
\bar{A} &= \frac{v\omega}{mn}\left(\frac{\Lambda}{l} - \frac{\mu\bar{N}}{\beta c}\right) \\
&= \frac{\Lambda v\omega}{l}\left(\frac{\beta c - l}{\beta cmn - vd\omega}\right).
\end{aligned}
\tag{D.8}
$$

Replacing the substitutions listed in equation D.1 into equations D.5, D.6, D.7, and D.8 we obtain the endemic equilibrium in terms of the parameters of the system:

$$
\begin{pmatrix} \bar{S} \\ \\ \bar{I} \\ \\ \bar{P} \\ \\ \bar{A} \end{pmatrix} = \begin{pmatrix} \frac{\Lambda((\mu+v+\omega)(\mu+d)+v\omega)}{\beta c(\mu+v)(\mu+d)-vd\omega} \\ \\ \frac{\Lambda(\mu+v)(\mu+d)(\beta c-(\mu+\omega))}{(\mu+\omega)(\beta c(\mu+v)(\mu+d)-vd\omega)} \\ \\ \frac{\Lambda\omega(\mu+d)(\beta c-(\mu+\omega))}{(\mu+\omega)(\beta c(\mu+v)(\mu+d)-vd\omega)} \\ \\ \frac{\Lambda v\omega(\beta c-(\mu+\omega))}{(\mu+\omega)(\beta c(\mu+v)(\mu+d)-vd\omega)} \end{pmatrix}.
$$

D.2 Local Stability Analysis

The Jacobian for the SIPA model is:

$$
J = \begin{pmatrix} -\mu - \frac{\beta c I(N-S)}{N^2} & -\frac{\beta c S(N-I)}{N^2} & \frac{\beta c I S}{N^2} & \frac{\beta c I S}{N^2} \\ \\ \frac{\beta c I(N-S)}{N^2} & \frac{\beta c S(N-I)}{N^2} - l & -\frac{\beta c I S}{N^2} & -\frac{\beta c I S}{N^2} \\ \\ 0 & \omega & -m & 0 \\ \\ 0 & 0 & v & -n \end{pmatrix}
$$

where we have again made the substitutions as in D.1.

D.2.1 Disease Free Equilibrium

The Jacobian calculated at the disease free equilibrium $(\frac{A}{\mu}, 0, 0, 0)$, is:

$$
J_{(\frac{A}{\mu},0,0,0)} = \begin{pmatrix} -\mu & -\beta c & 0 & 0 \\ 0 & \beta c - l & 0 & 0 \\ 0 & \omega & -m & 0 \\ 0 & 0 & v & -n \end{pmatrix}
$$

The characteristic equation is:

$$
\left| J_{(\frac{A}{\mu},0,0,0)} - \lambda I \right| = \begin{vmatrix} -\mu - \lambda & -\beta c & 0 & 0 \\ 0 & \beta c - l - \lambda & 0 & 0 \\ 0 & \omega & -m - \lambda & 0 \\ 0 & 0 & v & -n - \lambda \end{vmatrix}
$$

$$
=: -(n + \lambda) \begin{vmatrix} -(\mu + \lambda) & \beta c & 0 \\ 0 & \beta c - (l + \lambda) & 0 \\ 0 & \omega & -(m + \lambda) \end{vmatrix}
$$

$$
= (n + \lambda)(m + \lambda)(\mu + \lambda)((\beta c - l) - \lambda) = 0.
$$

Thus,

$$
\begin{aligned}
\lambda_1 &= -n, \\
\lambda_2 &= -m, \\
\lambda_3 &= -\mu, \text{ and} \\
\lambda_4 &= \beta c - l.
\end{aligned}
$$

Since the eigenvalues are real and excepting λ_4, all negative, the disease free equilibrium is locally asymptotically stable provided

$$\beta c > \mu + \omega,$$

and is unstable if

$$\beta c < \mu + \omega.$$

D.2.2 The Endemic Equilibrium

The Jacobian calculated at the endemic equilibrium, (where we omit the bar notation for convenience), is:

$$\left| J_{(\bar{S},\bar{I},\bar{P},\bar{A})} - \lambda I \right|$$

$$= \begin{vmatrix} -\mu - \frac{\beta c I(N-S)}{N^2} - \lambda & -\frac{\beta c S(N-I)}{N^2} & \frac{\beta c I S}{N^2} & \frac{\beta c I S}{N^2} \\ \frac{\beta c I(N-S)}{N^2} & \frac{\beta c S(N-I)}{N^2} - l - \lambda & -\frac{\beta c I S}{N^2} & -\frac{\beta c I S}{N^2} \\ 0 & \omega & -m - \lambda & 0 \\ 0 & 0 & v & -n - \lambda \end{vmatrix}$$

$$(R1 \leftarrow R1 + R2)$$

$$
= \begin{vmatrix}
-(\mu + \lambda) & -(l + \lambda) & 0 & 0 \\[2mm]
\frac{\beta c I(N-S)}{N^2} & \frac{\beta c S(N-I)}{N^2} - l - \lambda & -\frac{\beta c IS}{N^2} & -\frac{\beta c IS}{N^2} \\[2mm]
0 & \omega & -(m + \lambda) & 0 \\[2mm]
0 & 0 & v & -(n + \lambda)
\end{vmatrix}
$$

$$
-v \begin{vmatrix}
-(\mu + \lambda) & -(l + \lambda) & 0 \\[2mm]
\frac{\beta c I(N-S)}{N^2} & \frac{\beta c S(N-I)}{N^2} - l - \lambda & -\frac{\beta c IS}{N^2} \\[2mm]
0 & \omega & 0
\end{vmatrix}
$$

$$
-(n + \lambda) \begin{vmatrix}
-(\mu + \lambda) & -(l + \lambda) & 0 \\[2mm]
\frac{\beta c I(N-S)}{N^2} & \frac{\beta c S(N-I)}{N^2} - l - \lambda & -\frac{\beta c IS}{N^2} \\[2mm]
0 & \omega & -(m + \lambda)
\end{vmatrix}
$$

$$
= \ vw(\mu + \lambda)\frac{\beta c IS}{N^2} + (n + \lambda)\left(\omega(\mu + \lambda)\frac{\beta c IS}{N^2}\right)
$$

$$
+ (n + \lambda)(m + \lambda)(\mu + \lambda)\left(l + \lambda - \frac{\beta c S(N - I)}{N^2}\right)
$$

$$
+ (n + \lambda)(m + \lambda)(l + \lambda)\left(\frac{\beta c I(N - S)}{N^2}\right).
$$

Let

$$
R_1 \ = \ \frac{\beta c S}{N}
$$

$$= l,$$

$$
\begin{aligned}
R_2 &= \frac{\beta c I S}{N^2} \\
&= \frac{l}{\beta c} R_3, \\
&= \frac{lmn}{\beta c X} (\beta c - l)
\end{aligned}
$$

$$R_3 = \frac{\beta c I}{N}.$$

Further, let C represent the characteristic equation.

$$
\begin{aligned}
C = \ & -(R_1 - R_2)\mu mn + \mu lmn + \mu n\omega R_2 \\
& + \mu v\omega R_2 + (R_3 - R_2)lmn \\
& + (-(R_1 - R_2)(\mu(m+n) + mn) + \mu l(m+n) + mn(\mu + l))\lambda \\
& + (\omega(m+n)R_2 + (R_3 - R_2)(l(m+n) + mn))\lambda \\
& + (-(R_1 - R_2)(\mu + m + n) + \mu l + mn + (\mu + l)(m+n))\lambda^2 \\
& + (\omega R_2 + (R_3 - R_2)(l + m + n))\lambda^2 \\
& + (-(R_1 - R_2) + m + n + \mu + l + R_3 - R_2)\lambda^3 \\
& + \lambda_4
\end{aligned}
\tag{D.9}
$$

We denote the coefficients of the characteristic equation in a manner similar to previous sections, that is where a_i is the coefficient of λ^{4-i}.

By the Routh-Hurwitz criteria we require the coefficients of the characteristic equation, as well as the determinant condition, $a_3(a_1 a_2 - a_3) - a_1^2 a_4$ to be positive. To this end

$$a_0 = 1$$

$$a_1 = R_3 + \mu + m + n$$

$$
\begin{aligned}
a_2 &= -l(\mu + m + n) + R_2(\mu + m + n) + \mu l + mn + \mu(m + n) \\
&\quad + l(m + n) + \omega R_2 + R_3(l + m + n) - R_2(l + m + n) \\
&= mn + \mu(m + n) + R_3(l + m + n)
\end{aligned}
$$

$$
\begin{aligned}
a_3 &= -(R_1 - R_2)(\mu(m + n) + mn) + \mu l(m + n) + mn(\mu + l) \\
&\quad + \omega(m + n)R_2 + (R_3 - R_2)(l(m + n) + mn) \\
&= -R_1(\mu m + \mu n + mn) + R_2(\mu m + \mu n + mn) + \mu l(m + n) \\
&\quad + mn(\mu + l) + \omega(m + n)R_2 + R_3(lm + ln + mn) \\
&= -R_2(\mu m + \mu n + mn) - R_2\omega(m + n) \\
&= \mu mn + R_3(lm + ln + mn)
\end{aligned}
$$

$$
\begin{aligned}
a_4 &= -(R_1 - R_2)\mu mn + \mu lmn + \mu n\omega R_2 \\
&\quad + \mu v\omega R_2 + (R_3 - R_2)lmn \\
&= R_2(\mu mn + \mu n\omega + \mu v\omega) + (R_3 - R_2)lmn \\
&= R_2\mu X + lmn R_3\left(\frac{\beta c - l}{\beta c}\right) \\
&= lmn R_3\left(\frac{\beta c - l}{\beta c}\right) + \frac{\mu X l}{\beta c}R_3.
\end{aligned}
$$

Since all the parameters are positive then, by the above, all the components of the a_i's are positive. Thus the first conditions of the Routh-Hurwitz criteria are satisfied. That is, $a_i > 0$, for $i = 0, 1, 2, 3, 4$. The final Routh-Hurwitz condition is calculated below, beginning with the simplification of $a_1 a_2 - a_3$.

$$
\begin{aligned}
a_1 a_2 - a_3 &= (R_3 + \mu + m + n)(mn + \mu(m + n) + R_3(l + m + n)) \\
&\quad - \mu mn - R_3 l(m + n) - R_3 mn
\end{aligned}
$$

Figure D.15: Program Listing for SIPA Model

```
det:=R3*(m*n+u*(m+n)+R3*(1+m+n)):
det:=det+u*(u*(m+n)+R3*(1+m+n)):
det:=det+m*(m*n+u*(m+n)+R3*m):
det:=det+n*(m*n+u*(m+n)+R3*(m+n)):
a3:=u*m*n+R3*(l*m+l*n+m*n):
a1:=R3+u+m+n:
a4:=l*m*n*R3-l^2*m*n*R3/B+u*l*(m*n+n*w+v*w)*R3/B:
rhc:=collect(expand(det*a3-a1^2*a4),R3);
quit
```

$$
\begin{aligned}
= \ & R_3(mn + \mu(m + n) + R_3(l + m + n)) \\
& + \mu(\mu(m + n) + R_3(l + m + n)) \\
& + m(mn + \mu(m + n) + R_3 m) \\
& + n(mn + \mu(m + n) + R_3(m + n))
\end{aligned}
$$

We now deal with the determinant condition $a_3(a_1 a_2 - a_3) - a_1^2 a_4$ as required. We employ MAPLE for a simplification. The program is listed in figure D.15, and the MAPLE output follows.

```
         2 3       2 3       3 2       2 3       3 2
rhc := m   n   u + u   n   m + m   n   u + u   m   n + u   m   n

         2  2  2     3  2
    + 2 u   m   n  + u   n   m
```

```
      2 2           2 2           2 2         2 2         2 2
  + (4 m  n  u + 3 u  n  m + 3 u  m  n + u  m  l + u  n  l

        3             3             3             3         2 3
  + u m  l + 2 u m  n + u n  l + 2 u n  m + m  n

      3 2       2             2             2
  + m  n  + 2 u  m l n + u m  l n + u m n  l

      ** 1 **         ** 1 **         ** 1 **         ** 1 **

      2 2             3             3             3
      u l m n         u l m n         u l n w         u l v w
  + ------------ - ----------- - ----------- - -----------
        B               B               B               B

      ** 2 **         ** 2 **         ** 2 **

      2 2             2 2             2
      u m  l  n       u m  l  n       u  m l n w
  + 2 ----------- - 2 ----------- - 2 -------------
          B               B               B

      ** 2 **         ** 3 **         ** 3 **

        2               2
      u  m l v w       m n  u l w       m n u l v w
  - 2 ----------- - 2 ------------- - 2 -------------
          B               B               B
```

```
    ** 4 **        ** 4 **        ** 4 **          ** 4 **

     3  2           3              3               2
    n  l  m       n  u  l  m     n  u  l  w       n  u  l  v  w
+ ---------- - ----------- - ---------- - ------------
        B              B             B               B



    ** 5 **        ** 5 **        ** 5 **

     2  2           2  2           2  2
    u  n  l  m     u  n  l  m     u  n  l  w
+ 2 ----------- - 2 ----------- - 2 ------------
        B              B             B



    ** 5 **        ** 6 **      ** 6 **

     2             3  2           3
    u  n  l  v  w     m  l  n       m  u  l  n
- 2 ------------ + ---------- - ----------
        B              B             B



    ** 6 **        ** 6 **        ** 3 **
     2             2             2  2  2
    m  u  l  w     m  u  l  v  w     m  n  l
- ------------ - ------------ + 2 ----------
        B              B             B
```

$$- 2 \; \frac{\overset{** \ 3 \ **}{\overset{2 \ 2}{m \ n \ u \ l}}}{B} \Big) \quad R3$$

$$+ \Big(3 \, u \, m^{2} \, n + 3 \, u \, n^{2} \, m + 2 \, u \, n^{2} \, l + 2 \, u \, m^{2} \, l + m^{2} \, n \, l$$

$$+ m \, n^{2} \, l + u \, l \, m^{2} + u \, l \, n^{2} + 4 \, u \, l \, m \, n + 2 \, m^{2} \, n^{2}$$

$$+ m \, l^{3} + n \, m^{3} + n \, l^{3} + m \, n^{3} - 2 \; \frac{\overset{** \ 7 \ **}{\overset{2}{u \ l \ m \ n}}}{B}$$

$$- 2 \; \frac{\overset{** \ 7 \ **}{\overset{2}{u \ l \ n \ w}}}{B} - 2 \; \frac{\overset{** \ 7 \ **}{\overset{2}{u \ l \ v \ w}}}{B} + 2 \; \frac{\overset{** \ 8 \ **}{\overset{2 \ 2}{m \ l \ n}}}{B}$$

```
        ** 9 **        ** 7 **        ** 8 **
         2  2            2              2
         n  l  m        u l  m n       m  u l n
   + 2 ----------- + 2 ----------- - 2 -----------
           B              B              B



        ** 8 **        ** 8 **        ** 9 **
                                        2
        m u l n w      m u l v w       n  u l m
   - 2 ----------- - 2 ----------- - 2 -----------
           B              B              B



        ** 9 **        ** 9 **
         2
        n  u l w       n u l v w       2
   - 2 ----------- - 2 -----------) R3
           B              B



       ** 10 **    ** 10 **            ** 10 **
        u l n w     u l m n    2        u l v w     2
   + (- --------- - --------- + n  l - --------- + l  m
           B           B                  B



       ** 10 **
         2
        l  m n     2   2              2     2   3
   + -------- + l  n + m  l + 2 l m n + m  n + m n ) R3
          B
```

The numbering for the simplifications below correspond to the numbering used in the output. We begin by cancelling all negative coefficients of R_3.

$$
\begin{aligned}
1 \quad &= \quad \frac{\mu^2 l}{\beta c}(lmn - \mu mn - \mu n\omega - \mu v\omega) \\
&= \quad \frac{\mu^2 lvd\omega}{\beta c}
\end{aligned}
$$

$$
\begin{aligned}
2 \quad &= \quad 2\frac{\mu lm}{\beta c}(lmn - \mu mn - \mu n\omega - \mu v\omega) \\
&= \quad 2\frac{\mu lmvd\omega}{\beta c}
\end{aligned}
$$

$$
\begin{aligned}
3 \quad &= \quad 2\frac{lmn}{\beta c}(lmn - \mu mn - \mu n\omega - \mu v\omega) \\
&= \quad 2\frac{lmnvd\omega}{\beta c}
\end{aligned}
$$

$$
\begin{aligned}
4 \quad &= \quad \frac{ln^2}{\beta c}(lmn - \mu mn - \mu n\omega - \mu v\omega) \\
&= \quad \frac{ln^2 vd\omega}{\beta c}
\end{aligned}
$$

$$
\begin{aligned}
5 \quad &= \quad 2\frac{\mu ln}{\beta c}(lmn - \mu mn - \mu n\omega - \mu v\omega) \\
&= \quad 2\frac{\mu nlvd\omega}{\beta c}
\end{aligned}
$$

$$
\begin{aligned}
6 \quad &= \quad \frac{lm^2}{\beta c}(lmn - \mu mn - \mu n\omega - \mu v\omega) \\
&= \quad \frac{lm^2 vd\omega}{\beta c}
\end{aligned}
$$

We now simplify the negative coefficients of R_3^2.

$$
7 \quad = \quad 2\frac{\mu l}{\beta c}(lmn - \mu mn - \mu n\omega - \mu v\omega)
$$

$$= \ 2\frac{\mu l v d\omega}{\beta c}$$

$$**8** \ = \ 2\frac{lm}{\beta c}(lmn - \mu mn - \mu n\omega - \mu v\omega)$$

$$= \ 2\frac{lmvd\omega}{\beta c}$$

$$**9** \ = \ 2\frac{ln}{\beta c}(lmn - \mu mn - \mu n\omega - \mu v\omega)$$

$$= \ 2\frac{lnvd\omega}{\beta c}$$

Finally, we simplify the negative coefficients of R_3^3.

$$**10** \ = \ \frac{l}{\beta c}(lmn - \mu mn - \mu n\omega - \mu v\omega)$$

$$= \ \frac{lvd\omega}{\beta c}.$$

Therefore, since we have proved that all the negative coefficients of all the powers of R_3 simplify, the determinant condition of the Routh Hurwitz criteria is satisfied. Recall that the coefficients of the characteristic equation were also all positive. Thus, by the Routh-Hurwitz condition, the endemic equilibrium is locally asymptotically stable provided $\beta c > \mu + \omega$.

Appendix E

The SIQA Model

E.1 Equilibria

The model being analyzed in this section is found in equation 4.3. We begin our linear analysis by solving for the equilibria. We use the substitutions:

$$
\begin{aligned}
m &= \mu + v \\
n &= \mu + d \\
l &= \mu + \omega \\
f &= m + \omega
\end{aligned}
\tag{E.1}
$$

Setting $I' = 0$, in equation 4.3, we obtain:

$$
\left(\frac{\beta c S}{N} - (\mu + \omega) \right) I = 0
$$

so that we have only

$$
I = 0 \qquad \text{or} \qquad S = \frac{lmN}{\beta c (m + \omega)}
$$

E.1.1 Disease Free Equilibrium

If $I = 0$ then $\hat{Q} = 0$, $\hat{A} = 0$ and $\hat{S} = \frac{\Lambda}{\mu}$. Thus the disease free equilibrium is:

$$\begin{pmatrix} \hat{S} \\ \hat{I} \\ \hat{Q} \\ \hat{A} \end{pmatrix} = \begin{pmatrix} \frac{\Lambda}{\mu} \\ 0 \\ 0 \\ 0 \end{pmatrix}.$$

E.1.2 Endemic Equilibrium

If $\bar{I} \neq 0$ then $\bar{S} = \frac{lmN}{\beta cf}$. Setting $Q' = 0$, in equation 4.3, we obtain:

$$\omega I = (\mu + v)Q.$$

Setting $S' = 0$, in equation 4.3, we have:

$$\begin{aligned} 0 &= \Lambda - \left(\mu + \frac{\beta cf I}{mN} S \right) \\ &= \Lambda - \frac{\mu lmN}{\beta cf} - lI \end{aligned}$$

so that,

$$\bar{I} = \frac{\Lambda}{l} - \frac{\mu m \bar{N}}{\beta cf}$$

Then

$$\begin{aligned} \bar{Q} &= \frac{\omega \bar{I}}{m} \\ &= \frac{\omega}{m} \left(\frac{\Lambda}{l} - \frac{\mu m \bar{N}}{\beta cf} \right) \end{aligned}$$

Setting $A' == 0$, in equation 4.3, we have:

$$vQ = (\mu + d)A$$

and hence

$$\bar{A} = \frac{v\omega I}{mn}$$

$$= \frac{v\omega}{mn}\left(\frac{\Lambda}{l} - \frac{\mu m \bar{N}}{\beta cf}\right)$$

Therefore, the endemic equilibrium, as a function of $\bar{N}$, is:

$$\begin{pmatrix} \bar{S} \\ \\ \bar{I} \\ \\ \bar{Q} \\ \\ \bar{A} \end{pmatrix} = \begin{pmatrix} \frac{lm\bar{N}}{\beta cf} \\ \\ \frac{\Lambda}{l} - \frac{\mu m \bar{N}}{\beta cf} \\ \\ \frac{\omega}{m}\left(\frac{\Lambda}{l} - \frac{\mu m \bar{N}}{\beta cf}\right) \\ \\ \frac{v\omega}{mn}\left(\frac{\Lambda}{l} - \frac{\mu m \bar{N}}{\beta cf}\right) \end{pmatrix} \qquad (E.2)$$

Using equation E.2, we obtain a value for $\bar{N}$ in terms of the parameters of the system:

$$\begin{aligned} \bar{N} &= \bar{S} + \bar{I} + \bar{Q} + \bar{A} \\ &= \frac{lm\bar{N}}{\beta cf} + \left(1 + \frac{\omega}{m} + \frac{v\omega}{mn}\right)\left(\frac{\Lambda}{l} - \frac{\mu m \bar{N}}{\beta cf}\right) \\ &= \bar{N}\left(\frac{lm}{\beta cf} - \frac{\mu m}{\beta cf}\left(1 + \frac{\omega}{m} + \frac{v\omega}{mn}\right)\right) \\ &\quad + \frac{\Lambda}{l}\left(1 + \frac{\omega}{m} + \frac{v\omega}{mn}\right) \\ &= \bar{N}\left(\frac{lmn - \mu mn - \mu n\omega - \mu v\omega}{\beta cfn}\right) + \frac{\Lambda}{l}\left(\frac{mn + n\omega + v\omega}{mn}\right). \end{aligned}$$

so that

$$\bar{N} = \frac{\Lambda(nf + v\omega)}{l}\left(\frac{\beta cf}{mn\beta cf - lm^2n + \mu m(nf + v\omega)}\right)$$

$$= \frac{\Lambda\beta cf(nf + v\omega)}{lm(\beta cfn - lmn + \mu nf + \mu v\omega)} \tag{E.3}$$

We now can solve, using equation E.3 the coordinates of the endemic equilibrium, in equation E.2, in terms of the parameters of the system.

$$\bar{S} = \frac{lm\bar{N}}{\beta cf}$$

$$= \frac{lm}{\beta cf}\left(\frac{\Lambda\beta cf(nf + v\omega)}{lm(\beta cfn - lmn + \mu nf + \mu v\omega)}\right)$$

$$= \frac{\Lambda(nf + v\omega)}{\beta cfn - lmn + \mu(nf + v\omega)}, \tag{E.4}$$

$$\bar{I} = \frac{\Lambda}{l} - \frac{\mu m\bar{N}}{\beta cf}$$

$$= \frac{\Lambda}{l} - \frac{\mu m}{\beta cf}\left(\frac{\Lambda\beta cf(nf + v\omega)}{lm(\beta cfn - lmn + \mu nf + \mu v\omega)}\right)$$

$$= \frac{\Lambda n}{l}\left(\frac{\beta cf - lm}{\beta cfn - lmn + \mu(nf + v\omega)}\right), \tag{E.5}$$

$$\bar{Q} = \frac{\omega}{m}\left(\frac{\Lambda}{l} - \frac{\mu m\bar{N}}{\beta cf}\right).$$

$$= \frac{\Lambda n\omega}{lm}\left(\frac{\beta cf - lm}{\beta cfn - lmn + \mu(nf + v\omega)}\right), \tag{E.6}$$

and

$$\bar{A} = \frac{v\omega}{mn}\left(\frac{\Lambda}{l} - \frac{\mu m\bar{N}}{\beta cf}\right)$$

$$= \frac{\Lambda v\omega}{lm}\left(\frac{\beta cf - lm}{\beta cfn - lmn + \mu(nf + v\omega)}\right), \tag{E.7}$$

Replacing the substitutions in equation E.1 into equations E.4, E.5, E.6, and E.7 and using the fact that $\beta cfn - lmn + \mu(nf + v\omega) = \beta cfn - vd\omega$, we obtain the endemic equilibrium:

$$
\begin{pmatrix} \bar{S} \\ \bar{I} \\ \bar{Q} \\ \bar{A} \end{pmatrix} = \begin{pmatrix} \frac{\Lambda((\mu+v+\omega)(\mu+d)+v\omega)}{\beta c(\mu+v+\omega)(\mu+d)-vd\omega} \\ \frac{\Lambda(\mu+d)}{(\mu+\omega)} \left(\frac{\beta c(\mu+v+\omega)-(\mu+\omega)(\mu+v)}{\beta c(\mu+v+\omega)(\mu+d)-vd\omega} \right) \\ \frac{\Lambda\omega(\mu+d)}{(\mu+v)(\mu+\omega)} \left(\frac{\beta c(\mu+v+\omega)-(\mu+\omega)(\mu+v)}{\beta c(\mu+v+\omega)(\mu+d)-vd\omega} \right) \\ \frac{\Lambda v\omega}{(\mu+v)(\mu+\omega)} \left(\frac{\beta c(\mu+v+\omega)-(\mu+\omega)(\mu+v)}{\beta c(\mu+v+\omega)(\mu+d)-vd\omega} \right) \end{pmatrix} .
$$

The endemic equilibrium exists if $\beta c > \frac{lm}{m+\omega}$.

E.2   Local Stability Analysis

The Jacobian for the SIQA model is:

$$
J = \begin{pmatrix} -\mu - \frac{\beta c(I+Q)(N-S)}{N^2} & -\frac{\beta cS(N-(I+Q))}{N^2} & -\frac{\beta c(N-(I+Q))S}{N^2} & \frac{\beta c(I+Q)S}{N^2} \\ \frac{\beta c(I+Q)(N-S)}{N^2} & \frac{\beta cS(N-(I+Q))}{N^2} - l & \frac{\beta c(N-(I+Q))S}{N^2} & -\frac{\beta c(I+Q)S}{N^2} \\ 0 & \omega & -m & 0 \\ 0 & 0 & v & -n \end{pmatrix}
$$

where we have again made the substitutions as in E.1.

E.2.1 Disease Free Equilibrium

The Jacobian calculated at the disease free equilibrium $(\frac{\Lambda}{\mu}, 0, 0, 0)$, is:

$$J_{(\frac{\Lambda}{\mu},0,0,0)} = \begin{pmatrix} -\mu & -\beta c & -\beta c & 0 \\ 0 & \beta c - l & \beta c & 0 \\ 0 & \omega & -m & 0 \\ 0 & 0 & v & -n \end{pmatrix}$$

The characteristic equation is:

$$\left| J_{(\frac{\Lambda}{\mu},0,0,0)} - \lambda I \right| = \begin{vmatrix} -\mu - \lambda & -\beta c & -\beta c & 0 \\ 0 & \beta c - l - \lambda & \beta c & 0 \\ 0 & \omega & -m - \lambda & 0 \\ 0 & 0 & v & -n - \lambda \end{vmatrix}$$

$$= -(n + \lambda) \begin{vmatrix} -(\mu + \lambda) & \beta c & -\beta c \\ 0 & \beta c - (l + \lambda) & \beta c \\ 0 & \omega & -(m + \lambda) \end{vmatrix}$$

$$= (n + \lambda)(\mu + \lambda)((m + \lambda)(l + \lambda - \beta c) - \beta c \omega) = 0$$

Two of the eigenvalues are easily determined. They are $\lambda_1 = -n$ and $\lambda_2 = -\mu$. The other two eigenvalues are determined by solving the quadratic equation (derived from the quadratic factor of the characteristic equation) $\lambda^2 + (m + (l - \beta c))\lambda + m(l - \beta c) - \beta c \omega$. Thus

$$\begin{aligned} \lambda_{3,4} &= \frac{\beta c - l - m}{2} \pm \frac{1}{2}\sqrt{(m + (l - \beta c))^2 + 4m(\beta c - l) + 4\beta c \omega} \\ &= \frac{\beta c - l - m}{2} \pm \frac{1}{2}\sqrt{(\beta c - l + m)^2 + 4\beta c \omega}. \end{aligned}$$

Cleary, the discriminant is always positive, and hence we will always have real roots. For stability of the disease free equilibrium we require λ_3 and λ_4 to be negative. Since $\lambda_3 > \lambda_4$ then this is satisfied if $\lambda_3 < 0$. To this end, $\lambda_3 < 0$ provided

$$(\beta c - l + m)^2 + 4\beta c\omega \; < \; (m + l - \beta c)^2$$

which implies

$$\beta c \; < \; \frac{lm}{m + \omega}$$

Thus

$$\lambda_1 \;=\; -n$$

$$\lambda_2 \;=\; -\mu$$

$$\lambda_3 \;=\; \frac{\beta \dot{c} - l - m)}{2} + \frac{1}{2}\sqrt{(\beta c - l + m)^2 + 4\beta c\omega}. \qquad (E.8)$$

$$\lambda_4 \;=\; \frac{\beta c - l - m)}{2} - \frac{1}{2}\sqrt{(\beta c - l + m)^2 + 4\beta c\omega}.$$

By the above discussion and since λ_1 and λ_2 are negative, the disease free equilibrium is locally asymptotically stable provided

$$\beta c < \frac{(\mu + \omega)(\mu + v)}{\mu + v + \omega},$$

and is unstable if

$$\beta c > \frac{(\mu + \omega)(\mu + v)}{\mu + v + \omega}.$$

E.2.2 Endemic Equilibrium

The Jacobian calculated at the endemic equilibrium provides the equations leading to the characteristic equation. We assume in the following calculations that mention of the populations refers to equilibrium populations.

$$\left| J_{(\bar{S},\bar{I},\bar{Q},\bar{A})} - \lambda I \right| =$$

$$\begin{vmatrix} -\mu - \frac{\beta c(I+Q)(N-S)}{N^2} - \lambda & -\frac{\beta c S(N-(I+Q))}{N^2} & \frac{\beta c(I+Q)(N-S)}{N^2} & \frac{\beta c(I+Q)S}{N^2} \\[2mm] \frac{\beta c(I+Q)(N-S)}{N^2} & \frac{\beta c S(N-(I+Q))}{N^2} - l - \lambda & \frac{\beta c(N-(I+Q))S}{N^2} & -\frac{\beta c(I+Q)S}{N^2} \\[2mm] 0 & \omega & -m - \lambda & 0 \\[2mm] 0 & 0 & v & -n - \lambda \end{vmatrix}$$

$$(R_1 \leftarrow R_1 + R_2)$$

$$= \begin{vmatrix} -(\mu + \lambda) & -(l + \lambda) & 0 & 0 \\[2mm] \frac{\beta c(I+Q)(N-S)}{N^2} & \frac{\beta c S(N-(I+Q))}{N^2} - (l + \lambda) & \frac{\beta c(N-(I+Q))S}{N^2} & -\frac{\beta c(I+Q)S}{N^2} \\[2mm] 0 & \omega & -(m + \lambda) & 0 \\[2mm] 0 & 0 & v & -(n + \lambda) \end{vmatrix}$$

We use the following substitutions to simplify calculations:

$$W_1 = \frac{\beta c(I + Q)(N - S)}{N^2}$$

$$W_2 = \frac{\beta c S}{N^2}(N - (I + Q))$$

$$W_3 = \frac{\beta c(I + Q)S}{N^2} \tag{E.9}$$

$$U = \beta cf - lm$$

Using the substitutions in E.9 we solve for the characteristic equation, which we have labelled C:

$$
C \;=:\;
\begin{vmatrix}
-(\mu + \lambda) & -(l + \lambda) & 0 & 0 \\
W_1 & W_2 - (l + \lambda) & W_2 & -W_3 \\
0 & \omega & -(m + \lambda) & 0 \\
0 & 0 & v & -(n + \lambda)
\end{vmatrix}
$$

$$
=\; -(\mu + \lambda)
\begin{vmatrix}
W_2 - (l + \lambda) & W_2 & -W_3 \\
\omega & -(m + \lambda) & 0 \\
0 & v & -(n + \lambda)
\end{vmatrix}
$$

$$
+(l + \lambda)
\begin{vmatrix}
W_1 & W_2 & -W_3 \\
0 & -(m + \lambda) & 0 \\
0 & v & -(n + \lambda)
\end{vmatrix}
$$

$$
=\; -(\mu + \lambda)\left(-v\omega W_3 + (n + \lambda)((m + \lambda)(W_2 - (l + \lambda)) + \omega W_2)\right)
$$
$$
+W_1(l + \lambda)(m + \lambda)(n + \lambda)
$$

$$
=\; W_1(l + \lambda)(mn + (m + n)\lambda + \lambda^2)
$$
$$
+\mu v \omega W_3 - \mu(n + \lambda)(m + \lambda)W_2
$$
$$
+\mu(n + \lambda)(m + \lambda)(l + \lambda) - \mu(n + \lambda)\omega W_2
$$
$$
-\lambda(n + \lambda)(m + \lambda)W_2 + \lambda(n + \lambda)(m + \lambda)(l + \lambda)
$$
$$
-\lambda(n + \lambda)\omega W_2 + v\omega W_3
$$

$$
=\; W_1 lmn + \mu v\omega W_3 - \mu mn W_2 + \mu lmn - \mu n\omega W_2
$$
$$
+\lambda\left(W_1(l(m + n) + mn) - \mu(n + m)W_2 + \mu(l(m + n) + mn)\right)
$$
$$
+\lambda\left(lmn - (\mu\omega + nf)W_2 + v\omega W_3\right)
$$
$$
+\lambda^2\left((W_1 - W_2)(l + m + n) + \mu(l + m + n) + l(m + n) + mn\right)
$$
$$
+\lambda^3\left(W_1 + \mu + l + m + n - W_2\right)
$$
$$
+\lambda^4
$$

Therefore the coefficients a_i for $i = 0, 1, 2, 3, 4$ corresponding to the $\lambda^{(4-i)th}$ term of the characteristic equation are:

$$a_0 = 1$$

$$a_1 = W_1 + \mu + l + m + n - W_2$$

$$a_2 = W_1(l + m + n) - (l + m + n)W_2 + \mu(l + m + n) + l(m + n) + mn$$

$$a_3 = W_1(l(m + n) + mn) - \mu(n + m)W_2 + \mu(l(m + n) + mn)$$
$$-(\mu\omega + nm + \omega n)W_2 + lmn + v\omega W_3$$

$$a_4 = W_1 lmn + \mu v\omega W_3 - \mu mn W_2 + \mu lmn - \mu n\omega W_2$$

At this point we are prepared to check the Routh-Hurwitz criteria for the SIQA model. We must replace the substitutions given in E.9 in terms of the parameters of the system.

$$\frac{I}{N} = \frac{\Lambda}{lN} - \frac{\mu m}{\beta c f}$$
$$= -\frac{\mu m}{\beta c f} + \frac{\Lambda}{l}\left(\frac{lm(\beta c f n - lmn + \mu n f + \mu v\omega)}{\Lambda\beta c f(n f + v\omega)}\right)$$
$$= \frac{mn(\beta c f - lm)}{\beta c f(n f + v\omega)}$$

$$W_1 = \frac{\beta c(I + Q)(N - S)}{N^2}$$
$$= \frac{I}{N}\left(\frac{\beta c f - lm}{m}\right)$$
$$= \frac{nU^2}{\beta c f(n f + v\omega)}$$

$$W_2 = \frac{\beta cS}{N^2}(N-(I+Q))$$

$$= \frac{lm}{f} - \frac{lmnU}{\beta cf(nf+v\omega)}$$

$$= \frac{lm}{f}\left(\frac{\beta c(nf+v\omega)-n\beta cf+lmn}{\beta c(nf+v\omega)}\right)$$

$$= \frac{lm}{f}\left(\frac{\beta cv\omega+lmn}{\beta c(nf+v\omega)}\right)$$

$$W_3 = \frac{\beta c(I+Q)S}{N^2}$$

$$= \frac{lI}{N}$$

$$= \frac{lmn(\beta cf-lm)}{\beta cf(nf+v\omega)}$$

$$= \frac{lmnU}{\beta cf(nf+v\omega)}$$

The Routh-Hurwitz criteria for a four dimensional system requires that $a_i > 0$ for $i = 0, 1, 2, 3, 4$ as well as the determinant condition $a_3(a_1a_2 - a_3) - a_1^2 a_4$. We start by examining the coefficients of the characteristic equation.

$$a_0 = 1$$
$$> 0$$

$$a_1 = W_1 + \mu + l + m + n - W_2$$

$$= W_1 + \mu + m + n + l\left(\frac{\beta cf(nf+v\omega)-m\beta cv\omega-lm^2n}{\beta cf(nf+v\omega)}\right)$$

$$= W_1 + \mu + m + n + l\left(\frac{\beta cfmn+\beta cf\omega n+\beta cv\omega^2-lm^2n}{\beta cf(nf+v\omega)}\right)$$

$$= W_1 + \mu + m + n + l\left(\frac{mn(\beta cf-lm)+\beta c\omega(nf+v\omega)}{\beta cf(nf+v\omega)}\right)$$

$$= W_1 + \mu + m + n + \frac{lmnU}{\beta cf(nf+v\omega)} + \frac{l\omega}{f}$$

$$> 0.$$

$$a_2 = (W_1 - W_2)(l + m + n) + \mu(l + m + n) + l(m + n) + mn$$

$$= W_1(l + m + n) + mn + \mu(m + n)$$
$$+ \left(\mu l - \frac{\mu lm}{f} \left(\frac{\beta cv\omega + lmn}{\beta c(nf + v\omega)} \right) \right)$$
$$+ lm - \frac{vlm}{f} \left(\frac{\beta cv\omega + lmn}{\beta c(nf + v\omega)} \right)$$
$$- \frac{l^2 m}{f} \left(\frac{\beta cv\omega + lmn}{\beta c(nf + v\omega)} \right)$$
$$+ \left(ln - \frac{lmn}{f} \left(\frac{\beta cv\omega + lmn}{\beta c(nf + v\omega)} \right) \right)$$

$$= W_1(l + m + n) + mn + \mu(m + n)$$
$$+ \mu l \left(\frac{\beta cf^2 n + \beta cfv\omega - \beta cmv\omega - lm^2 n}{\beta cf(nf + v\omega)} \right)$$
$$+ lm \left(\frac{\beta cf^2 n + \beta cfv\omega - \beta cv^2\omega - lmnv - \beta clv\omega - l^2 mn}{\beta cf(nf + v\omega)} \right)$$
$$+ ln \left(\frac{\beta cf^2 n + \beta cfv\omega - \beta cmv\omega - lm^2 n}{\beta cf(nf + v\omega)} \right)$$

$$= W_1(l + m + n) + mn + \mu(m + n)$$
$$+ \frac{lmnU}{\beta cf(nf + v\omega)}(l + m + n) + \frac{ln\omega}{f} + \frac{\mu l\omega}{f}$$

$$> 0.$$

$$a_3 = W_1(l(m + n) + mn) - \mu(n + m)W_2 + \mu(l(m + n) + mn)$$
$$- (\mu\omega + nm + \omega n)W_2 + lmn + v\omega W_3$$

$$= W_1(l(m + n) + mn) + \mu mn - W_2(\mu f + nf + \mu n)$$

$$+ v\omega W_3 + lmn + \mu ml + \mu ln$$

$$
\begin{aligned}
= \ & W_1(l(m+n) + mn) + \mu mn + v\omega W_3 \\
& + \left(\mu ml - \mu ml \left(\frac{\beta cv\omega + lmn}{\beta c(nf + v\omega)} \right) \right) \\
& + \left(lmn - lmn \left(\frac{\beta cv\omega + lmn}{\beta c(nf + v\omega)} \right) \right) \\
& + \left(\mu ln - \frac{\mu lmn}{f} \left(\frac{\beta cv\omega + lmn}{\beta c(nf + v\omega)} \right) \right)
\end{aligned}
$$

$$
\begin{aligned}
= \ & \mu lm \left(\frac{\beta cnf + \beta cv\omega - \beta cv\omega - lmn}{\beta c(nf + v\omega)} \right) \\
& + lmn \left(\frac{\beta cnf + \beta cv\omega - \beta cv\omega - lmn}{\beta c(nf + v\omega)} \right) \\
& + \mu ln \left(\frac{\beta cf^2 n + \beta cfv\omega - \beta cmv\omega - lm^2 n}{\beta cf(nf + v\omega)} \right) \\
& + W_1(l(m+n) + mn) + \mu mn + v\omega W_3
\end{aligned}
$$

$$
\begin{aligned}
= \ & W_1(l(m+n) + mn) + \mu mn + \frac{\mu lmn U}{\beta c(nf + v\omega)} + \frac{lmn^2 U}{\beta c(nf + v\omega)} \\
& + \frac{\mu ln^2 m U}{\beta cf(nf + v\omega)} + \frac{\mu ln\omega}{f} + v\omega W_3
\end{aligned}
$$

$$> \ 0.$$

$$a_4 \ = \ lmnW_1 + \mu v\omega W_3 - \mu nfW_2 + \mu lmn$$

$$
= \ lmnW_1 + \frac{\mu v\omega lmn U}{\beta cf(nf + v\omega)} + \left(\mu lmn - \frac{\mu lmnf(\beta cvw + lmn)}{\beta cf(nf + v\omega)} \right)
$$

$$
= \ lmnW_1 + \frac{\mu lmn U}{\beta cf}
$$

$$> \ 0.$$

We are now in a position to examine the determinant condition. At this juncture, we employ the use of MAPLE, to perform this analysis. Included in figure E.16 and following pages, are the MAPLE program and the output. We have used the simplifications and substitutions

$$U1 = \frac{lmnU}{\beta cf(nf + v\omega)}$$

$$W = W_1$$

$$u = \mu$$

$$w = \omega$$

$$\det = a_1 a_2 - a_3$$

```
         3 2      3   2      3 2    2 3        2 3
rh := u  m  n + u  m n  + u m  n + u  m  n + u m  n

        2   3     2 2 2
    + u  m n  + 2 u  m  n


         3 2    2 3    2    2    3      2    2    3
    + W  (m  n  + m  n  + m  l u + m  l u + n  l u + n  l u

           2   2       2 2      3          3
      + 3 m  n u + 3 m n  u  + 2 m  n u + 2 m n  u

           2 2        2           2    2
      + 4 m  n  u + 2 m l u  n + m l u n  + m  l u n
```

Figure E.16: Program Results for SIQA Model

Calculate the Routh Hurwitz determinant

```
a1:=W+u+m+n+U1+l*w/f:
a2:=W*(l+m+n)+m*n+u*(m+n)+U1*(l+m+n)+l*w/f*(u+n):
a3:=W*(l*(m+n)+m*n)+u*m*n+u*f*U1+n*f*U1+u*n*U1:
a3:=a3+u*n*l*w/f+v*w*U1:
a4:=l*m*n*W+u*(n*f+v*w)*U1:
det:=collect(expand(a1*a2-a3),W):
det:=det+u*f*U1+n*f*U1+v*w*U1:
det:=det-u*m*U1-u*l*U1-n*m*U1-n*l*U1-m*l*U1:
det:=det+u^2*U1+u*n*U1+u*l*U1+u*v*U1:
```

Collect the Routh Hurwitz determinant and simplify.

```
rhc:=collect(expand(a3*det-a1^2*a4),W):
W0:=collect(coeff(rhc,W,0),U1):
W1:=collect(coeff(rhc,W,1),U1):
W2:=collect(coeff(rhc,W,2),U1):
W3:=collect(coeff(rhc,W,3),U1):
UW00:=coeff(W0,U1,0):
UW01:=coeff(W0,U1,1):
UW02:=coeff(W0,U1,2):
UW10:=coeff(W1,U1,0):
UW11:=coeff(W1,U1,1):
UW12:=coeff(W1,U1,2):
UW20:=coeff(W2,U1,0):
UW21:=coeff(W2,U1,1):
UW22:=coeff(W2,U1,2):
UW30:=coeff(W3,U1,0):
UW31:=coeff(W3,U1,1):
UW32:=coeff(W3,U1,2):
rh:=UW00+U1*UW01+U1^2*UW02+W*(UW10+U1*UW11+U1^2*UW12):
rh:=rh+W^2*(UW20+U1*UW21+U1^2*UW22):
rh:=rh+W^3*(UW30+U1*UW31+U1^2*UW32);
quit
```

$$+\ 3\,\frac{m\,l^2\,u\,n\,w}{f} + \frac{m^3\,l^2\,w\,u}{f^2} + 2\,\frac{n^2\,l^2\,u\,w}{f}$$

$$+\ 2\,\frac{n^3\,l^2\,w\,u}{f^2} + \frac{n^3\,l^2\,w}{f} + \frac{m^2\,l^2\,w\,n}{f}$$

$$+\ 2\,\frac{m^2\,l^2\,w\,u}{f} + \frac{m^2\,l^2\,w\,u}{f} + 2\,\frac{m\,n^2\,l^2\,w\,u}{f^2}$$

$$+\ \frac{m\,n^3\,l\,w}{f} + 2\,\frac{m\,n^2\,l^2\,w}{f} + 4\,\frac{m\,n^2\,l\,w\,u}{f}$$

$$+\ \frac{m^2\,n^2\,l^2\,w}{f^2} + 4\,\frac{m\,n\,l^2\,w\,u}{f} + \frac{n^2\,l^3\,w^2}{f^2}$$

$$+\ 2\,\frac{n^2\,l^2\,w\,u}{f} + 6\,\frac{m\,n^2\,u\,l\,w}{f} + \frac{u\,n^3\,l\,w}{f}$$

```
     2 2              2   2 2            2 2 2
     u n l w          u n l w            u n l w
+ 2 ----------- + ------------- + 2 -------------
        f                f^2                f^2


      2                    2       2
+ (2 u n  + 2 n f l + n  l + n l + 2 u m n


                              2   2
+ 2 m l n + 2 n f m + m n + m  l + 2 v w m


                    2       2
+ 2 v w n + m  n + 2 n  f + 2 u f m + 2 u f l


   ** 1 **                        ** 1 **
                           2               2
+ 2 v w l + 2 u n l + m l  - 2 u v w) U1


   3       3       3       3     2 2     3
+ (m  l + n  l + m  n + m n  + 2 m n  + n f


         3       2 2       2       2       2
 + u n  + 2 u n  + m l n + m l  u + m  l u


     2         2           2             2
+ m  l n + n l  u + 2 n  l u + 6 m n  u


                                2         2 2
     2                        m l w u    m l w
+ 4 n  n u + 4 m l u n + ---------- + ---------
                              f             f
```

```
    3              2            3
  m l  w        n l  w u      n l  w
+ -------- + 4 ----------- + --------
    f              f            f


      2                           2
  m l  w n      m n l w u      m  n l w
+ 2 ---------- + 4 ----------- + ----------
      f              f             f


    2              2 2            2
  m n  l w        n l  w        u n  l w
+ 2 ---------- + 2 ---------- + 4 -----------
    f              f              f


    2        2       2        2        2
+ u f m + u f n + 2 u f m + u f l + u l w


    2        2       2        2        2
+ u l  w + n f m + 2 n l w + 2 n f m + n l  w


    2        2              2        2
+ 3 u  n m + 2 u  n l + v w m + v w n


  ** 2 **              ** 2 **
        2          2
- 2 v w u  + m l u  + 2 u f m n + n f u l


                              2
                            u n l w
+ n l w m + u l w n + u l w m + ----------
                              f
```

$$
+ vwul - \frac{v^2 w^3 lu}{f} + 2\frac{v^2 w^3 ln}{f}
$$

$$
+ 2vwmn + \frac{v^2 w^2 l}{f} + \frac{v^2 w lm}{f} + mluv
$$

$$
+ nluv + mnuv)\,U1)
$$

$$
+ W(m^2 l^3 + n^3 l + m^3 n + mn^3 + 2m^2 n^2 + m^2 ln + m^2 lu
$$

$$
+ 2m^2 lu + m^2 ln + n^2 lu + 2n^2 lu + 3m^2 nu
$$

$$
+ 3m^2 nu + 4mlun + \frac{m^2 l w^2 u}{f} + \frac{m^2 l^2 w^2}{f}
$$

$$
+ \frac{m l w^3}{f} + 2\frac{n l w^2 u}{f} + \frac{n l w^3}{f} + 2\frac{m l w^2 n}{f}
$$

$$
+ 2\,\frac{m\,n\,l\,w\,u}{f} + \frac{m\,n\,l\,w^2}{f} + 2\,\frac{m\,n\,l\,w^2}{f}
$$

$$
+ 2\,\frac{n^2\,l\,w^2}{f} + \frac{u\,n\,l\,w^2}{f}
$$

$$
+ (2\,m\,l^2 + u\,n + 2\,n^2\,l + 2\,m^2\,l + 2\,m^2\,n
$$

$$
+ 2\,m\,n^2 - u\,v\,w + n^2\,f + u\,f\,l + u\,f\,m
$$

$$
+ 4\,m\,l\,n + n\,f\,l + n\,f\,m + u\,n\,l + u\,m\,n
$$

$$
+ 2\,n\,l^2 + v\,w\,l + v\,w\,m + v\,w\,n)\,U1)
$$

$$
+ 4\,\frac{u^2\,m\,n^2\,l\,w}{f} + 3\,\frac{u^2\,m\,n\,l\,w^2}{f^2} + 2\,\frac{u^3\,m\,n\,l\,w}{f}
$$

$$
+ 3\,\frac{u\,m^2\,n^2\,l\,w}{f} + 3\,\frac{u^2\,m\,n\,l\,w}{f} + 3\,\frac{u\,m^2\,n\,l^2\,w^2}{f^2}
$$

$$
+ 2\,\frac{u\ m\ n\ l\ w^{3}}{f} + \frac{u\ n\ l\ w^{3}{}^{2}}{f} + \frac{u\ n\ l\ w^{2}{}^{3}}{f}
$$

$$
+ 2\,\frac{u\ n\ l\ w^{2\ 2\ 2\ 2}}{f^{2}} + \frac{u\ n\ l\ w^{2\ 3\ 3}}{f^{3}} + \frac{u\ n\ l\ w^{3\ 2\ 2}}{f^{2}}
$$

$$
+ \frac{u\ n\ l\ w^{2\ 3\ 3}}{f^{3}} + \frac{u\ n\ l\ w^{3\ 2\ 2}}{f^{2}}
$$

$$
+ (m\ l^{2} + 2\,m\ l\ n + n\ l^{2} + n^{2} l + m^{2} l + m^{2} n + m\ n^{2})\ W^{3}
$$

$$
+ (n^{3} f + u\ n^{3} + 2\,u^{2} n^{2} + u\ n^{2} + 3\,m\ n^{3} u + 2\,m^{2} n\ u
$$

$$
+ u^{3} f + m\ l\ u\ n + 2\,\frac{n\ l\ w\ u^{2}}{f} + 2\,\frac{m\ n\ l\ w\ u}{f}
$$

$$
+ 3\,\frac{u\ n^{2} l\ w}{f} + n\ f\ u\ v + u\ f\ m^{2} + u\ f\ n^{2} + u^{2} f\ m
$$

```
        2         2         2         2          2           2
  + u   f n + u   f l + u   l w + u   f v + u l   w + n f m

          2           2           2     2           2
  + 2 n   l w + 2 n   f m + n l   w + u   n m + u   n l


            ** 6 **   ** 5 **            ** 5 **
        2           2       2   2           2
  + u   n v + v w m   + v w n   + v   w u - v w u   + u f m n

                                                      2
                                                  u   n l w
  + n f u l + n l w m + u l w n + u l w m + ----------
                                                      f

      ** 6 **   ** 7 **      ** 7 **
                              2               2
                          v w   l u       v w   l n
  - v w u m + v w u l - ---------- + 2 ----------
                              f               f

                        2   2         2
                    v w   l       v w   l m     2
  + 2 v w m n + --------- + ----------) U1
                        f               f


      3   2     2   3     2       2         2   2     3
  + ( u   n   + u   n   + 2 m   n u + 4 m n   u   + m   n u

              3           2   2           2             2
  + 2 m n   u + 3 m   n   u + m l u   n + u f m   n
```

```
        2               3  2             2  2
    m l  u n w        n l  w  u       m n l  w  u
 + ------------    + -----------   + -------------
        f               f^2              f^2


      2                          2          2    2
    m  n l w u       m n l w u        n l  w  u
 + 2 ------------ + 4 ------------ + ------------
        f                f                f


        2                3               2  2
    m n  u l w        u n  l w        u n  l w
 + 6 ------------ + 2 ---------- + 4 -----------
        f                f                f


    2   2 2          2 2 2               3
    u n l  w         u n l w          u n l w
 + 2 ------------ + 3 ------------ + 2 ----------
       f^2             f^2               f


                               2 2 2
      2            2           n l w               3
 + u  m n v + 2 n  m l w + ---------- + 2 u  m n
                               f

 ** 8 **
      3       2   2   2   2   3      3
 + u  f m + u  f m + n  f m + n  f m + n  l w
```

```
     ** 8 **
                  2 2 2
     3         u l w              2
   - u  v w + ---------- + u l w n + 2 u m l w n
                   f


       2           3          2 2            2
   + 2 u  m l w + u  l w + u f m n + u  f m n + u  n l w


                                  ** 9 **     ** 10 **
       2 2          2
     u l  w  n    u n l w v      2         2
   + ----------- + ------------ - v w u  m - v w u  n
         f             f


   ** 9 **     ** 10 **     ** 11 **
                                2  2          2
       2            2     v w  l  n     v w  m l n
   + v w m  n + v w m n + ----------- + 2 ------------
                              f              f

                    ** 11 **


       3 2          2  2
     v w  l  n    v w  l u
   + ----------- - ----------- ) U1
         2             f
         f
```

Listed below are the supporting simplifying calculations as they correspond
with the marked terms in the output.

$$** \, 1 \, ** \quad = \quad 2v\omega l - 2\mu v\omega$$
$$= \quad 2v\omega^2$$

$$** \, 2 \, ** \quad = \quad 2\mu f m n - 2v\omega \mu^2$$
$$= \quad 2\mu^2 m n + 2\mu v m n + 2\mu^2 n\omega + 2\mu v d\omega$$

$$** \, 3 \, ** \quad = \quad v\omega \mu l - \frac{v\omega^2 \mu l}{f}$$
$$= \quad \frac{\mu l v\omega m}{f}$$

$$** \, 4 \, ** \quad = \quad \mu f l - \mu v\omega$$
$$= \quad \mu l^2 + \mu^2 v$$

$$** \, 5 \, ** \quad = \quad v\omega n^2 - v\omega \mu^2$$
$$= \quad v\omega d(\mu + n)$$

$$** \, 6 \, ** \quad = \quad v\omega m^2 - v\omega \mu m$$
$$= \quad v^2 \omega m$$

$$** \, 7 \, ** \quad = \quad v\omega \mu l - \frac{v\omega^2 \mu l}{f}$$
$$= \quad \frac{\mu l v\omega m}{f}$$

$$** \, 8 \, ** \quad = \quad \mu^3 f m - \mu^3 v\omega$$
$$= \quad \mu^3 m^2 + \mu^4 \omega$$

$$** \, 9 \, ** \quad = \quad v\omega m^2 n - v\omega \mu^m$$

$$= v\omega\mu dm + v^2\omega mn$$

$$**10** \quad = v\omega mn^2 - v\omega\mu^2 n$$

$$= v^2\omega n^2 + v\omega\mu dn$$

$$**11** \quad = \frac{v\omega^2 ln^2}{f} - \frac{v\omega^2 l\mu^2}{f}$$

$$= \frac{v\omega^2 ld}{f}(\mu + n)$$

We have thus shown by the above simplifications that the Routh-Hurwitz criteria holds for the endemic equilibrium of the SIQA model, provided $\beta cf - lm > 0$. Therefore, we can conclude that the endemic equilibrium is locally asymptotically stable whenever $\beta cf - lm > 0$.

Appendix F

The SIQPA Model

F.1 Equilibria

The SIQPA system, given in equation 2.1, has two equilibrium points.

By examining $Q' = 0$, $P' = 0$, and $A' = 0$ in 2.1 we see that

$$Q = \frac{(1-\rho)\omega}{\mu + v}I,$$

$$P = \frac{\rho\omega}{\mu + v}I, \text{ and}$$

$$A = \frac{v\omega}{(\mu + v)(\mu + d)}I.$$

Setting $I' = 0$ it follows that at equilibrium, either

$$I = 0$$

or

$$S = \frac{(\mu + \omega)(\mu + v)N}{\beta c(\mu + v + (1-\rho)\omega)}.$$

F.1.1 Disease Free Equilibrium

When $I = 0$, the disease free equilibrium is

$$
\begin{pmatrix} \hat{S} \\ \hat{I} \\ \hat{Q} \\ \hat{P} \\ \hat{A} \end{pmatrix} = \begin{pmatrix} \frac{\Lambda}{\mu} \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}
$$

F.1.2 The Endemic Equilibrium

We use the substitutions found in C.5 as well as

$$
\begin{aligned}
X &= mn + n\omega + v\omega \\
k &= \mu + v + (1 - \rho)\omega.
\end{aligned}
$$

When $I \neq 0$, then $S = \frac{lmN}{\beta ck}$. Setting $S' = 0$ in 2.1 we solve for $\bar{I}$:

$$
\Lambda - \mu\bar{S} = \frac{\beta ck\bar{S}}{m\bar{N}}\bar{I}
$$

$$
\bar{I} = \frac{\Lambda}{l} - \frac{\mu m\bar{N}}{\beta ck}.
$$

It follows that

$$
\bar{Q} = \frac{(1-\rho)\omega}{m}\left(\frac{\Lambda}{l} - \frac{\mu m\bar{N}}{\beta ck}\right)
$$

$$
\bar{P} = \frac{\rho\omega}{m}\left(\frac{\Lambda}{l} - \frac{\mu m\bar{N}}{\beta ck}\right)
$$

$$
\bar{A} = \frac{v\omega}{mn}\left(\frac{\Lambda}{l} - \frac{\mu m\bar{N}}{\beta ck}\right)
$$

The endemic equilibrium as a function of $\bar{N}$ is:

$$
\begin{pmatrix}
\bar{S} \\
\\
\bar{I} \\
\\
\bar{Q} \\
\\
\bar{P} \\
\\
\bar{A}
\end{pmatrix}
=
\begin{pmatrix}
\frac{lm\bar{N}}{\beta ck} \\
\\
\frac{\Lambda}{l} - \frac{\mu m\bar{N}}{\beta ck} \\
\\
\frac{(1-\rho)\omega}{m}\left(\frac{\Lambda}{l} - \frac{\mu m\bar{N}}{\beta ck}\right) \\
\\
\frac{\rho\omega}{m}\left(\frac{\Lambda}{l} - \frac{\mu m\bar{N}}{\beta ck}\right) \\
\\
\frac{v\omega}{mn}\left(\frac{\Lambda}{l} - \frac{\mu m\bar{N}}{\beta ck}\right)
\end{pmatrix}
\tag{F.1}
$$

Using equation F.1 and $\bar{N} = \bar{S} + \bar{I} + \bar{Q} + \bar{P} + \bar{A}$ we derive an explicit expression for $\bar{N}$ in terms of the parameters of the model:

$$
\begin{aligned}
\bar{N} &= \frac{lm\bar{N}}{\beta ck} \\
&\quad + \frac{\Lambda}{l} - \frac{\mu m\bar{N}}{\beta ck} \\
&\quad + \frac{((1-\rho)\omega)}{m}\left(\frac{\Lambda}{l} - \frac{\mu m\bar{N}}{\beta ck}\right) \\
&\quad + \frac{\rho\omega}{m}\left(\frac{\Lambda}{l} - \frac{\mu m\bar{N}}{\beta ck}\right) \\
&\quad + \frac{v\omega}{mn}\left(\frac{\Lambda}{l} - \frac{\mu m)\bar{N}}{\beta ck}\right) \\
&= \frac{lm\bar{N}}{\beta ck} - \frac{\mu X\bar{N}}{\beta ckn} + \frac{\Lambda X}{lm}
\end{aligned}
$$

so that we obtain

$$
\bar{N} = \frac{\Lambda X\beta ck}{lm(\beta ckn + \mu X - lmn)}.
\tag{F.2}
$$

Substituting $\bar{N}$ into equation F.1, we derive expressions for the endemic equilibrium in terms of the parameters of the system. Note that $\mu X - lmn = -vd\omega$.

$$\bar{S} = \frac{lm}{\beta ck} \left(\frac{\beta ck}{lm} \right) \left(\frac{\Lambda X}{\beta ckn - vd\omega} \right)$$

$$= \frac{\Lambda X}{\beta ckn - vd\omega}$$

$$\bar{I} = \frac{\Lambda}{l} - \frac{\mu m}{\beta ck} \left(\frac{\beta ck}{lm} \right) \left(\frac{\Lambda X}{\beta ckn - lmn + \mu X} \right)$$

$$= \frac{\Lambda n}{l} \left(\frac{\beta ck - lm}{\beta ckn - vd\omega} \right)$$

$$\bar{Q} = \frac{\Lambda n(1 - \rho)\omega}{lm} \left(\frac{\beta ck - lm}{\beta ckn - vd\omega} \right)$$

$$\bar{P} = \frac{\Lambda n \rho \omega}{lm} \left(\frac{\beta ck - lm}{\beta ckn - vd\omega} \right)$$

and

$$\bar{A} = \frac{\Lambda v \omega}{lm} \left(\frac{\beta ck - lm}{\beta ckn - vd\omega} \right)$$

We record these in tabular form:

$$
\begin{pmatrix} \bar{S} \\ \bar{I} \\ \bar{Q} \\ \bar{P} \\ \bar{A} \end{pmatrix} = \begin{pmatrix} \frac{\Lambda X}{\beta ckn - vd\omega} \\ \frac{\Lambda n}{l}\left(\frac{\beta ck - lm}{\beta ckn - vd\omega}\right) \\ \frac{\Lambda n(1-\rho)\omega}{lm}\left(\frac{\beta ck - lm}{\beta ckn - vd\omega}\right) \\ \frac{\Lambda n\rho\omega}{lm}\left(\frac{\beta ck - lm}{\beta ckn - vd\omega}\right) \\ \frac{\Lambda v\omega}{lm}\left(\frac{\beta ck - lm}{\beta ckn - vd\omega}\right) \end{pmatrix}
$$

Note that the endemic equilibrium exists if and only if $\beta ck > lm$.

F.2 Local Stability Properties

We now proceed to look at the local analysis of these two fixed points.
The Jacobian is:

$$
\begin{pmatrix}
-\mu - Z_1 & -Z_2 & -Z_2 & Z_3 & Z_3 \\
Z_1 & Z_2 - l & Z_2 & -Z_3 & -Z_3 \\
0 & (1-\rho)\omega & -m & 0 & 0 \\
0 & \rho\omega & 0 & -m & 0 \\
0 & 0 & v & v & -n
\end{pmatrix}
$$

where

$$
Z_1 = \frac{\beta c(I+Q)(N-S)}{N^2},
$$

$$
Z_2 = \frac{\beta c(N-(I+Q))S}{N^2},
$$

$$
Z_3 = \frac{\beta c(I+Q)S}{N^2}.
$$

F.2.1 Disease Free Equilibrium

The Jacobian at the disease free equilibrium $(\frac{\Lambda}{\mu}, 0, 0, 0, 0)$ is:

$$
J_{(\frac{\Lambda}{\mu},0,0,0,0)} = \begin{pmatrix}
-\mu & -\beta c & -\beta c & 0 & 0 \\
0 & \beta c - l & \beta c & 0 & 0 \\
0 & (1-\rho)\omega & -m & 0 & 0 \\
0 & \rho\omega & 0 & -m & 0 \\
0 & 0 & v & v & -n
\end{pmatrix},
$$

and thus,

$$
|J_{(\frac{\Lambda}{\mu},0,0,0,0)} - \lambda I| =
$$

$$
\begin{vmatrix}
-(\mu + \lambda) & -\beta c & -\beta c & 0 & 0 \\
0 & \beta c - (l + \lambda) & \beta c & 0 & 0 \\
0 & (1-\rho)\omega & -(m + \lambda) & 0 & 0 \\
0 & \rho\omega & 0 & -(m + \lambda) & 0 \\
0 & 0 & v & v & -(n + \lambda)
\end{vmatrix}
$$

$$
= (n + \lambda)(\mu + \lambda)(m + \lambda)((\beta c - l - \lambda)(m + \lambda) + \beta c(1 - \rho)\omega)
$$

We can solve immediately for three of the eigenvalues, those being $\lambda_1 = -n, \lambda_2 = -m$, and $\lambda_3 = -\mu$. These three eigenvalues are negative. Solving the quadratic part of the characteristic equation we have

$$
\lambda_{4,5} = \frac{\beta c - l - m}{2} \pm \frac{1}{2}\sqrt{(\beta c - l - m)^2 + 4(\beta c - l)m + 4\beta c(1 - \rho)\omega}
$$

$$
= \frac{\beta c - l - m}{2} \pm \frac{1}{2}\sqrt{(\beta c - l + m)^2 + 4\beta c(1 - \rho)\omega}.
$$

Clearly the discriminant is positive and λ_4 and λ_5 are negative if the larger one, say λ_4, is negative. To this end,

$$
(\beta c - l + m)^2 + 4\beta c(1 - \rho)\omega < (l + m - \beta c)^2
$$

if

$$\beta c(m + (1 - \rho)\omega) < lm.$$

Thus, the eigenvalues,

$$\lambda_1 = -n,$$
$$\lambda_2 = -m,$$
$$\lambda_3 = -\mu,$$
$$\lambda_4 = \frac{\beta c - l - m}{2} + \frac{1}{2}\sqrt{(\beta c - l + m)^2 + 4\beta c(1 - \rho)\omega},$$
$$\lambda_5 = \frac{\beta c - l - m}{2} - \frac{1}{2}\sqrt{(\beta c - l + m)^2 + 4\beta c(1 - \rho)\omega},$$

are real and all negative provided $\beta ck < lm$. Therefore the disease free equilibrium is locally asymptotically provided $\beta c < \frac{lm}{m+(1-\rho)\omega}$.

F.2.2 Endemic Equilibrium

We assume that Z_1, Z_2, and Z_3 are calculated at the endemic equilibrium. We now derive the characteristic equation.

$$|J_{(S,I,Q,P,A)} - \lambda I| = \begin{vmatrix} -\mu - Z_1 - \lambda & -Z_2 & -Z_2 & Z_3 & Z_3 \\ Z_1 & Z_2 - l - \lambda & Z_2 & -Z_3 & -Z_3 \\ 0 & (1-\rho)\omega & -m - \lambda & 0 & 0 \\ 0 & \rho\omega & 0 & -m - \lambda & 0 \\ 0 & 0 & v & v & -n - \lambda \end{vmatrix}$$

$(R_1 \leftarrow R1 + R2)$

$$= \begin{vmatrix} -(\mu+\lambda) & -(l+\lambda) & 0 & 0 & 0 \\ Z_1 & Z_2-(l+\lambda) & Z_2 & -Z_3 & -Z_3 \\ 0 & (1-\rho)\omega & -(m+\lambda) & 0 & 0 \\ 0 & \rho\omega & 0 & -(m+\lambda) & 0 \\ 0 & 0 & v & v & -(n+\lambda) \end{vmatrix}$$

We simplify using MAPLE. The program listing and results can be found in figure F.17 and following pages.

```
ceqn := - u p w Z2 n - u l m n - l Z1 m n - u w v Z3

  + u w Z2 n - u p w Z3 n + u Z2 m n

  + (- p w Z2 n - l Z1 n - u p w Z2 + Z2 m n - l Z1 m

     + u Z2 m + u Z2 n - Z1 m n - u l m - u l n

     - u m n - w v Z3 + u w Z2 - l m n - u p w Z3

     - p w Z3 n + w Z2 n) r

  + (Z2 m - l n + u Z2 - u n - l Z1 - Z1 m - Z1 n

     + Z2 n - u l - p w Z3 - u m - p w Z2 - m n - l m

                2
     + w Z2) r

                         3     4
  + (Z2 - m - u - l - n - Z1) r  - r
```

Figure F.17: Program listing for characteristic equation: SIQPA

```
#
# We begin by entering the nonzero matrix elements.
#
A1:=-u-r:
A2:=-l-r:
B1:=Z1:
B2:=Z2-l-r:
B3:=Z2:
B4:=-Z3:
B5:=-Z3:
C2:=(1-p)*w:
C3:=-m-r:
D2:=p*w:
D4:=-m-r:
E3:=v:
E4:=v:
E5:=-n-r:
#
# Calculate the characteristic equation, factor out (m+r),
# and collect with respect to r.
#
ceqn:=A1*D2*(-C3)*(B4*E5-E4*B5):
ceqn:=ceqn+A1*D4*(B2*C3*E5-C2*(B3*E5-E3*B5)):
ceqn:=ceqn-A2*B1*C3*D4*E5:
ceqn:=collect(ceqn,r);
ceqn:=simplify(ceqn);
quit
```

Notice that $(m + \lambda)$ can be factored out of the characteristic equation, so we need only use the Routh- Hurwitz criteria as it applies to a quartic polynomial. The coefficients of the characteristic equation are:

$$a_0 = 1$$

$$a_1 = \mu + l + m + n + Z_1 - Z_2$$

$$a_2 = ln + \mu n + (l + m + n)(Z_1 - Z_2)$$
$$+ \mu l + \mu m + mn + lm + \rho\omega(Z_2 + Z_3)$$

$$a_3 = \rho\omega Z_2 n + ln Z_1 + \mu\rho\omega Z_2 - Z_2 mn + lm Z_1$$
$$- \mu m Z_2 - \mu n Z_2 + mn Z_1 + \mu lm + \mu ln + \mu mn$$
$$+ \omega v Z_3 - \mu\omega Z_2 + lmn + \mu\rho\omega Z_3 + \rho\omega n Z_3 - \omega n Z_2$$

$$a_4 = \mu\rho\omega n Z_2 + \mu lmn + lmn Z_1 + \mu\omega v Z_3 - \mu\omega n Z_2$$
$$+ \mu\rho\omega n Z_3 - \mu mn Z_2.$$

By the Routh-Hurwitz criteria we require these coefficients to be positive. We examine each one individually, but begin by rewriting Z_i for $i = 1, 2, 3$ in terms of the parameters of the system. It is assumed that we are working with the endemic equilibrium even though we omit the bar notation.

$$\frac{I}{N} = \frac{\Lambda}{lN} - \frac{\mu m}{\beta ck}$$

$$= \frac{\Lambda}{l}\left(\frac{lm}{\beta ck}\right)\left(\frac{\beta ckn + \mu X - lmn}{\Lambda X}\right) - \frac{\mu m}{\beta ck}$$

$$= \frac{mn(\beta ck - lm)}{\beta ck X}$$

$$Z_1 = \frac{\beta c(I+Q)(N-S)}{N^2}$$

$$= \frac{\beta ck}{m} \left(\frac{mn(\beta ck - lm)}{\beta ckX} \right) \left(1 - \frac{lm}{\beta ck} \right)$$

$$= \frac{n(\beta ck - lm)^2}{\beta ckX}$$

$$Z_2 = \frac{\beta cS(N-(I+Q))}{N^2}$$

$$= \frac{\beta clm}{\beta ck} \left(1 - \frac{kmn(\beta ck - lm)}{m\beta ckX} \right)$$

$$= \frac{lm}{k} - \frac{lmn}{\beta ckX}(\beta ck - lm)$$

$$Z_3 = \frac{\beta c(I+Q)S}{N^2}$$

$$= \frac{lmn}{\beta ckX}(\beta ck - lm)$$

$$Z_1 - Z_2 = \frac{\beta c(I+Q)(N-S)}{N^2} - \frac{\beta cS(N-(I+Q))}{N^2}$$

$$= \frac{n}{X}(\beta ck - lm) - \frac{lm}{k}$$

$$Z_1 + Z_3 = \frac{\beta c(I+Q)(N-S)}{N^2} + \frac{\beta c(I+Q)S}{N^2}$$

$$= \frac{n}{X}(\beta ck - lm)$$

Let

$$Y = \frac{n}{X}(\beta ck - lm)$$
$$s = (1 - \rho)$$

We now examine the coefficients.

$$a_1 = \mu + l + m + n + Z_1 - Z_2$$

$$= \mu + m + n + l + \frac{n}{X}(\beta ck - lm) - \frac{lm}{k}$$

$$= \mu + m + n + l\left(\frac{k-m}{k}\right) + \frac{n}{X}(\beta ck - lm)$$

$$= \mu + m + n + (1 - \rho)\frac{\omega l}{k} + \frac{n}{X}(\beta ck - lm)$$

$$= Y + \frac{ls\omega}{k} + \mu + m + n$$

$$> 0.$$

$$a_2 = ln + \mu n + (l + m + n)(Z_1 - Z_2)$$
$$+ \mu l + \mu m + mn + lm + \rho\omega(Z_2 + Z_3)$$

$$= ln + \mu n + (l + m + n)\left(Y - \frac{lm}{k}\right)$$
$$+ \mu l + \mu m + mn + lm + \frac{\rho\omega lm}{k}$$

$$= (l + m + n)Y - l(\mu + m + n)\frac{lm}{k} + l(\mu + n + m)$$

$$+\mu n + \mu m + mn + \frac{\rho\omega lm}{k}$$

$$= (l+m+n)Y + \frac{ls\omega}{k}(\mu+m+n) - \frac{\omega lm}{k}$$

$$+\mu n + \mu m + mn + \frac{\rho\omega lm}{k}$$

$$= (l+m+n)Y + \frac{ls\omega}{k}(\mu+n) + \mu(n+m) + mn$$

$$> 0.$$

$$a_3 = \rho\omega Z_2 n + ln Z_1 + \mu\rho\omega Z_2 - Z_2 mn + lm Z_1$$
$$-\mu m Z_2 - \mu n Z_2 + mn Z_1 + \mu lm + \mu ln + \mu mn$$
$$+\omega v Z_3 - \mu\omega Z_2 + lmn + \mu\rho\omega Z_3 + \rho\omega n Z_3 - \omega n Z_2$$

$$= \mu ln + \mu lm + \mu mn + lmn + Z_1(ln + lm + mn)$$
$$-Z_2(mn + \mu m + \mu n + \mu w + \omega n) + (Z_2 + Z_3)\rho\omega(\mu+n) + Z_3 v\omega$$

$$= \mu ln + \mu lm + \mu mn + lmn + v\omega Y + \frac{lm\rho\omega}{k}(\mu+n)$$
$$+(Z_1 - Z_2)(ln + mn + \mu m + \mu\omega)$$

$$= \mu ln + \mu lm + \mu mn + lmn + Y(ln + lm + mn) + \frac{lm\rho\omega}{k}(\mu+n)$$
$$-\frac{ln}{k}(ln + mn + \mu m + \mu\omega)$$

$$= \mu ln + lm(\mu+n) - \frac{lm^2}{k}(\mu+n) + \mu mn + \frac{lm\rho\omega}{k}(\mu+n)$$
$$+Y(ln + lm + mn) - \frac{lm}{k}(ln + \mu\omega)$$

$$= \quad \mu ln + \frac{lms\omega}{k}(\mu + n) + \mu mn + Y(ln + lm + mn)$$
$$-\frac{ln\mu n}{k} - \frac{lm\omega}{k}(\mu + n) + \frac{lm\rho\omega}{k}(\mu + n)$$

$$= \quad \frac{\mu ln s\omega}{k} + \mu mn + Y(ln + lm + mn)$$

$$> \quad 0.$$

$$a_4 \quad = \quad \mu\rho\omega n Z_2 + \mu lmn + lmn Z_1 + \mu\omega v Z_3 - \mu\omega n Z_2$$
$$+\mu\rho\omega n Z_3 - \mu mn Z_2$$

$$= \quad \mu lmn + lmn Z_1 + (\mu\rho\omega n - \mu\omega n - \mu mn)Z_2 + (\mu v\omega + \mu\rho\omega n)Z_3$$

$$= \quad \mu lmn + lmn Z_1 - \frac{\mu lmnk}{k} + \mu nk Z_3 + (\mu v\omega + \mu\rho\omega n)Z_3$$

$$= \quad \mu mn Z_1 + \mu\omega n Z_1 + v d\omega Z_1 + \mu v\omega(Z_1 + Z_3)$$
$$+\mu\rho\omega n Z_3 + \mu mn Z_3 + \mu ns\omega Z_3$$

$$= \quad \mu mn Y + \mu\omega n Z_1 + v d\omega Z_1 + \mu v\omega Y + \mu\omega Z_3$$

$$= \quad \mu XY + v d\omega Y \frac{\beta ck - lm}{\beta ck}$$

$$> \quad 0.$$

Thus we have shown that all the coefficients of the characteristic equation are positive. We now prove the determinant condition of the Routh-Hurwitz criteria. That is, we show $a_3(a_1 a_2 - a_3) - a_1^2 a_4 > 0$. We begin by simplifying $a_1 a_2 - a_3$.

$$a_1 a_2 - a_3 =$$

$$\mu \left((l + m + n)Y + \frac{ls\omega}{k}(\mu + n) + \mu(n + m) + mn \right)$$

$$+ m \left((l + m + n)Y + \frac{ls\omega}{k}(\mu + n) + \mu(n + m) + mn \right)$$

$$+ n \left((l + m + n)Y + \frac{ls\omega}{k}(\mu + n) + \mu(n + m) + mn \right)$$

$$+ \frac{ls\omega}{k} \left((l + m + n)Y + \frac{ls\omega}{k}(\mu + n) + \mu(n + m) + mn \right)$$

$$+ Y \left((l + m + n)Y + \frac{ls\omega}{k}(\mu + n) + \mu(n + m) + mn \right)$$

$$- \left(\frac{\mu l n s\omega}{k} + \mu mn + Y(ln + lm + mn) \right)$$

$$= \mu \left((l + m + n)Y + \frac{ls\omega}{k}\mu + \mu(n + m) \right)$$

$$+ m \left(mY + \frac{ls\omega}{k}(\mu + n) + \mu(n + m) + mn \right)$$

$$+ n \left((m + n)Y + \frac{ls\omega}{k}(\mu + n) + \mu(n + m) + mn \right)$$

$$+ \frac{ls\omega}{k} \left((l + m + n)Y + \frac{ls\omega}{k}(\mu + n) + \mu(n + m) + mn \right)$$

$$+ Y \left((l + m + n)Y + \frac{ls\omega}{k}(\mu + n) + \mu(n + m) + mn \right)$$

With the use of MAPLE, we look next at the determinant condition and proceed to simplify. A program listing and results are provided in figure F.18 and the following pages. The symbols Y0, Y1, Y2, and Y3 represent the resultant powers of Y. Other symbols besides those previously mentioned in this section, that are used in the programming, include,

$$w = \omega,$$

$$u = \mu, \text{ and}$$

Figure F.18: Program listing for Routh Hurwitz condition: SIQPA

```
a1:=Y+l*s*w/k+u+m+n:
a2:=(l+m+n)*Y+l*s*w/k*(u+n)+u*(n+m)+m*n:
a3:=u*l*n*s*w/k+u*m*n+Y*(l*n+l*m+m*n):
a4:=u*X*Y+v*d*w*Y*G:
det:=expand(a1*a2-u*l*s*w*n/k-m*(l+n)*Y-n*l*Y-u*m*n):
rhc:=collect(expand(a3*det-a1^2*a4),Y):
Y0:=coeff(rhc,Y,0);
Y1:=coeff(rhc,Y,1);
Y2:=coeff(rhc,Y,2);
Y3:=coeff(rhc,Y,3);
quit
```

$$G \;=\; \frac{\beta ck - lm}{\beta ck}$$

```
               2      2      2   3                 3
       u l n   s w m      u  l n  s w       u l n  s w m
Y0 := 3 --------------- + ------------- + 2 --------------
               k               k                   k


         2 2 2     2   3      2 3     3 2
       +2 u   m   n  + u m n  + u m  n  + u m  n


         2 2   2 2                   2    2
       u   l n   w  m       3 2    u l n  s w m
     + 3 ---------------- + u m  n + 4 --------------
             k^2                          k
```

```
                          2           2
    2 3      3   2      u l n s w m
+ u  m  n + u  m n  + 3 ---------------
                             k


         2 2 2 2         3   2          3 2   2 2
     u l n s w m       u l n s w      u l n s  w
+ 3 ----------------- + ------------- + ---------------
             2                k              2
            k                               k


       3 2 3 3         2 2 2 2         2 3 2 2
    u l n s w        u l n s w       u l n s w
+ --------------- + 2 --------------- + ---------------
         3                2                2
        k                k                k


    2 3   3 3          3
   u l n s w        u l n s w m
+ --------------- + 2 ---------------
       k^3                k


            3       2 2       2   2   2 3   3 2
Y1 := 2 u m n + 4 u m  n + 3 u  m n + m n + m n


** 1 **   ** 1 **
                              2
    3       2          u l n s w m         3
- u  X + 3 u  m n l + 4 --------------- + 2 u m  n
                              k
```

$$+n\,l\,u \;+\; 2\,\frac{u\,l\,n\,s\,w\,m}{k^2}^{\,2\;2} \;+\; 3\,u\,m\,n\,n^{\,2\;2}$$

$$+\; 6\,\frac{u\,l\,n\,s\,w\,m}{k}^{\,2} \;+\; 4\,\frac{u\,l\,n\,s\,w\,m}{k}^{\,2} \;-\; 2\,m\,n\,u\,X^{\;**\,2\,**}$$

$$+\; 5\,\frac{u\,m\,n\,l\,s\,w}{k}^{\;**\,7\,**\;2} \;+\; 2\,n\,l\,m^{\;**\,2\,**\;2\;2} \;+\; n\,l\,u^{\;3} \;+\; n\,l\,m^{\;**\,6\,**\;3}$$

$$+\; \frac{u\,l\,n\,s\,w}{k}^{\,3} \;+\; 2\,\frac{u\,l\,n\,s\,w}{k}^{\,2\;2} \;+\; 2\,\frac{u\,l\,n\,s\,w}{k}^{\,2\;2}$$

$$+\; 2\,\frac{u\,l\,n\,s\,w}{k^2}^{\,3\;2\;2} \;+\; \frac{u\,l\,n\,s\,w}{k^2}^{\,2\;2\;2\;2} \;+\; 2\,\frac{u\,l\,n\,s\,w}{k^2}^{\,2\;2\;2\;2}$$

$$+\; 3\,n\,l\,u\,m^{**\,4\,**\;2} \;+\; 3\,n\,l\,u\,m^{**\,3\,**\;2} \;+\; m\,l\,u^{\,2\;2} \;+\; \frac{n\,l\,s\,w}{k^2}^{\,2\;3\;2\;2}$$

$$
** 8 ** \qquad\qquad\qquad ** 5 **
$$

$$
+3\,\frac{n\ l\ s\overset{2}{w}\overset{2}{m}}{k} + \frac{n\ l\ s\ \overset{3}{w}\ \overset{2}{3}}{k} + m\ l\ u\ \overset{3}{+}m\ l\ n
$$

$$
** 2 ** \qquad ** 4 ** \quad ** 5 ** \quad ** 6 **
$$

$$
-2\,\overset{2}{u}\ n\ X - 2\,\overset{2}{u}\ m\ X - m\ \overset{2}{u}\ X - n\ \overset{2}{u}\ X + 2\,\frac{n\ l\ u\ s\ w}{k}^{2\ 2}
$$

$$
** 9 **
$$

$$
+\frac{m\ l\ \overset{3}{s}\ \overset{2}{w}\ \overset{2}{u}}{\overset{2}{k}} + \frac{m\ l\ \overset{3}{s}\ \overset{2}{w}\ \overset{2}{n}}{\overset{2}{k}} + 2\,\frac{m\ l\ s\ \overset{2}{w}\ \overset{2}{u}}{k}
$$

$$
** 10 **
$$

$$
+2\,\frac{m\ l\ s\overset{2}{w}\overset{2}{n}}{k} + \frac{m\ l\ \overset{2}{s}\ w\ \overset{2}{u}}{k} + \frac{m\ n\ \overset{2}{l}\ \overset{2}{s}\ \overset{2}{w}}{\overset{2}{k}}
$$

$$
** 7 **
$$

$$
+2\,\frac{m\ n\ l\overset{2}{s}\overset{2}{w}}{k} + \frac{m\ n\ l\ \overset{3}{s}\ w}{k} - 2\,\frac{l\ s\ w\ u\ \overset{2}{X}}{k}
$$

** 7 **

$$-2\,\frac{l\,s\,w^{2}\,u\,v\,d\,G}{k}$$

** 8 **

$$-2\,\frac{l\,s\,w\,n\,u\,X}{k}$$

** 8 **

$$-2\,\frac{l\,s\,w^{2}\,n\,v\,d\,G}{k}$$

** 9 **

$$-\,\frac{l^{2}\,s^{2}\,w^{2}\,u\,X}{k^{2}}$$

** 9 **

$$-\,\frac{l^{2}\,s^{2}\,w^{3}\,v\,d\,G}{k^{2}}$$

** 1 **   ** 5 **   ** 6 **   ** 10 **

$$-\,u^{2}\,v\,d\,w\,G\;-\;m^{2}\,v\,d\,w\,G\;-\;n^{2}\,v\,d\,w\,G\;-\;2\,\frac{l\,s\,w\,m\,u\,X}{k}$$

** 10 **   ** 2 **   ** 3 **

$$-2\,\frac{l\,s\,w^{2}\,m\,v\,d\,G}{k}\;-\;2\,m\,n\,v\,d\,w\,G\;-\;2\,u\,n\,v\,d\,w\,G$$

** 4 **

$$-2\,u\,m\,v\,d\,w\,G$$

$$Y2 := 3\,u\,m^{2}\,n + 2\,m\,n^{2} + m^{2}\,l^{3} + n^{2}\,l^{3} + m\,n^{3} + m^{3}\,n$$

$$\mathbf{**\ 11\ **} \quad\quad \mathbf{**\ 12\ **}$$

$$-\,2u\overset{2}{X} +\,3\,u\,m\,n \,+\,6\,u\,m\,n\,l\,+\,2\,\frac{u\,l\,n\,s\,w\,m}{k}$$

$$\mathbf{**\ 12\ **} \quad \mathbf{**\ 12\ **} \quad\quad \mathbf{**\ 13\ **}$$

$$-\,2\,m\,u\overset{2}{X}+\,3\,n\,l\,m+\,2\,n\overset{2}{}\,l\,u+\,3\,n\overset{2}{}\,l\,m+\,n\,l\overset{2}{}\,u$$

$$+\,2\,\frac{u\,l\,n\,s\,w}{k}^{2} + \frac{u\,l\,n\,s\,w}{k}^{2} + 2\,\frac{n\,l\,s\,w}{k}^{2\,2}$$

$$\mathbf{**\ 14\ **}$$

$$+\,2\,m\,l\,u+n\,l\overset{2}{}\,u+\,\frac{n\,l\,s\,w}{k}^{3}+\,4\,\frac{n\,l\,s\,w\,m}{k}^{2}$$

$$+\,\frac{m\,l\,s\,w}{k}^{3} + \frac{m\,l\,s\,w}{k}^{2\,2} + \frac{m\,l\,s\,w\,u}{k}^{2} + \frac{m\,n\,l\,s\,w}{k}^{2}$$

$$\mathbf{**\ 13\ **} \quad \mathbf{**\ 14\ **} \quad\quad \mathbf{**\ 14\ **}$$

$$-\,2\,n\,u\,X -\,2\,\frac{l\,s\,w\,u\,X}{k} -\,2\,\frac{l\,s\,w\,v\,d\,G}{k}^{2}$$

```
                    ** 12 **     ** 13 **
            2
        m n  l s w
    + 2 ------------ - 2 m v d w G - 2 n v d w G
            k


        ** 11 **
    - 2 u v d w G


                    ** 15 **      ** 15 ** 15 **
        2      2               2                       2
Y3 := m  l + m  n + 3 n l m + n  l - u X - v d w G + m n


        2      2
    + n l  + m l            .
```

The simplifications required to determine positivity follow, and are matched with the numbering system used in the computer results.

$$
\begin{aligned}
1 &= 2\mu^2 lmn + \mu^2(lmn - \mu mn - \mu n\omega - \mu v\omega) - \mu^2 vd\omega G \\
&= 2\mu^2 lmn - \mu^2 vd\omega + \frac{\mu^2 vd\omega lm}{\beta ck} + \mu^2 vd\omega \\
&= 2\mu^2 lmn + \frac{\mu^2 lmvd\omega}{\beta ck}
\end{aligned}
$$

$$
\begin{aligned}
2 &= 2n^2 lm^2 - 2mn\mu X - 2mnvd\omega G \\
&= 2nm(lmn - \mu X) - 2mnvd\omega + 2\frac{m^2 lnvd\omega}{\beta ck}
\end{aligned}
$$

$$
\begin{aligned}
3 &= \mu lmn^2 + 2\mu lmn^2 - 2\mu^2 nX - 2\mu nvd\omega G \\
&= \mu lmn^2 + 2\mu n(lmn - \mu X) - 2\mu nvd\omega + 2\frac{\mu nlmvd\omega}{\beta ck}
\end{aligned}
$$

$$= \mu l m n^2 + 2\frac{\mu n l m v d\omega}{\beta c k}$$

$$** 4 ** \quad = \quad \mu l m^2 n + 2\mu l m^2 n - 2\mu^2 m X - 2\mu m v d\omega G$$

$$= \quad \mu l m^2 n + 2\mu m(lmn - \mu X) - 2\mu m v d\omega + 2\frac{\mu l m^2 v d\omega}{\beta c k}$$

$$= \quad \mu l m^2 n + 2\frac{\mu l m^2 v d\omega}{\beta c k}$$

$$** 5 ** \quad = \quad m^3 l n - m^2 \mu X - m^2 v d\omega G$$

$$= \quad m^2(lmn - \mu X) - m^2 v d\omega + \frac{m^3 l v d\omega}{\beta c k}$$

$$= \quad \frac{m^3 l v d\omega}{\beta c k}$$

$$** 6 ** \quad = \quad n^3 l m - n^2 \mu X - n^2 v d\omega G$$

$$= \quad \frac{n^2 l m v d\omega}{\beta c k}$$

$$** 7 ** \quad = \quad 5\frac{\mu m n l^2 s\omega}{k} - 2\frac{l s\omega\mu^2 X}{k} - 2\frac{l s\omega^2 \mu v d}{k} G$$

$$= \quad 3\frac{\mu m n l^2 s\omega}{k} + 2\frac{s\omega^2 l^2 m \mu v d}{\beta c k^2}$$

$$** 8 ** \quad = \quad 3\frac{l^2 s\omega m n^2}{k} - 2\frac{l s\omega n\mu X}{k} - 2\frac{l s\omega^2 n v d}{k} G$$

$$= \quad \frac{l^2 s\omega m n^2}{k} + 2\frac{l^2 s\omega^2 m n v d}{\beta c k^2}$$

$$** 9 ** \quad = \quad \frac{l^3 s^2 \omega^2 m n}{k^2} - \frac{l^2 s^2 \omega^2 \mu X}{k^2} - \frac{l^2 s^2 \omega^3 v d}{k^2} G$$

$$= \quad \frac{l^3 m s^2 \omega^3 v d}{\beta c k^3}$$

$$** 10 ** \quad = \quad 2\frac{l^2 m^2 s\omega n}{k} - 2\frac{l s\omega\mu m X}{k} - 2\frac{l s\omega^2 m v d}{k} G$$

$$= 2\frac{l^2 m^2 s\omega}{\beta c k^2}$$

$$** 11 ** \quad = \quad 6\mu lmn - 2\mu^X - 2\mu v d\omega G$$
$$= \quad 4\mu lmn + 2\frac{\mu lmvd\omega}{\beta ck}$$

$$** 12 ** \quad = \quad 3lm^2 n - 2\mu m X - 2mvd\omega G$$
$$= \quad lm^2 n + 2\frac{lm^2 vd\omega}{\beta ck}$$

$$** 13 ** \quad = \quad 3lmn^2 - 2\mu n X - 2nvd\omega G$$
$$= \quad lmn^2 + 2\frac{lmnvd\omega}{\beta ck}$$

$$** 14 ** \quad = \quad 4\frac{nl^2 s\omega m}{k} - 2\frac{ls\omega \mu X}{k} - 2\frac{ls\omega^2 vd}{k}G$$
$$= \quad 2\frac{nl^2 s\omega m}{k} + 2\frac{l^2 s\omega^2 mvd}{\beta ck^2}$$

$$** 15 ** \quad = \quad 3lmn - \mu X - vd\omega G$$
$$= \quad 2lmn + \frac{lmvd\omega}{\beta ck}$$

Thus, since all negative terms have been accounted for, $a_3(a_1 a_2 - a_3) - a_1^2 a_4 > 0$. Therefore, we have proved the Routh Hurwitz criteria hold for the endemic equilibrium of the SIQPA model, and so this equilibrium is locally asymptotically stable whenever $\beta c > \frac{lm}{m+(1-\rho)\omega}$.

Appendix G

The SIQ/PA Model

G.1   Equilibria

The model under consideration is listed in equations 4.7. The SIQ/PA system given in equation 4.8, has two equilibrium points. They have a similar form as for the SIQPA model, however, with the new definition for $N(t)$ there are some minor variations.

G.1.1   Disease Free Equilibrium

When $I = 0$, in equation 4.8

$$
\begin{pmatrix} \hat{S} \\ \hat{I} \\ \hat{Q} \\ \hat{P} \\ \hat{A} \end{pmatrix} = \begin{pmatrix} \frac{\Lambda}{\mu} \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}
$$

G.1.2   The Endemic Equilibrium

We use the substitutions found in E.1 as well as

$$k = \mu + v + (1 - \rho)\omega.$$

When $I \neq 0$, then $\bar{S} = \frac{lm\bar{N}}{\beta ck}$. Setting $S' = 0$ in 4.8 we solve for $\bar{I}$:

$$\Lambda - \mu\bar{S} \;=\; \frac{\beta ck\bar{S}}{m\bar{N}}\bar{I}$$

$$\bar{I} \;=\; \frac{\Lambda}{l} - \frac{\mu m\bar{N}}{\beta ck}.$$

Further,

$$\bar{Q} \;=\; \frac{(1-\rho)\omega}{m}\left(\frac{\Lambda}{l} - \frac{\mu m\bar{N}}{\beta ck}\right)$$

$$\bar{P} \;=\; \frac{\rho\omega}{m}\left(\frac{\Lambda}{l} - \frac{\mu m\bar{N}}{\beta ck}\right)$$

$$\bar{A} \;=\; \frac{v\omega}{mn}\left(\frac{\Lambda}{l} - \frac{\mu m\bar{N}}{\beta ck}\right)$$

The endemic equilibrium as a function of $\bar{N}$ is:

$$\begin{pmatrix} \bar{S} \\[6pt] \bar{I} \\[6pt] \bar{Q} \\[6pt] \bar{P} \\[6pt] \bar{A} \end{pmatrix} = \begin{pmatrix} \frac{lm\bar{N}}{\beta ck} \\[6pt] \frac{\Lambda}{l} - \frac{\mu m\bar{N}}{\beta ck} \\[6pt] \frac{(1-\rho)\omega}{m}\left(\frac{\Lambda}{l} - \frac{\mu m\bar{N}}{\beta ck}\right) \\[6pt] \frac{\rho\omega}{m}\left(\frac{\Lambda}{l} - \frac{\mu m\bar{N}}{\beta ck}\right) \\[6pt] \frac{v\omega}{mn}\left(\frac{\Lambda}{l} - \frac{\mu m\bar{N}}{\beta ck}\right) \end{pmatrix} \qquad (G.1)$$

Using equation G.1 and $\bar{N} = \bar{S} + \bar{I} + \bar{Q}$ we derive an explicit expression for $\bar{N}$ in terms of the parameters of the model:

$$
\begin{aligned}
\bar{N} &= \frac{lm\bar{N}}{\beta ck} + \frac{\Lambda}{l} - \frac{\mu m\bar{N}}{\beta ck} \\
&\quad + \frac{((1-\rho)\omega)}{m}\left(\frac{\Lambda}{l} - \frac{\mu m\bar{N}}{\beta ck}\right) \\
&= \frac{lm\bar{N}}{\beta ck} + \frac{k}{m}\bar{I} \\
&= \bar{N}\left(\frac{lm - \mu k}{\beta ck}\right) + \frac{\Lambda k}{lm}
\end{aligned}
$$

so that we obtain

$$
\bar{N} = \frac{\Lambda k}{lm}\left(\frac{\beta ck}{\beta ck - lm + \mu k}\right).
$$

Substituting $\bar{N}$ into equation G.1, we derive expressions for the endemic equilibrium in terms of the parameters of the system.

$$
\begin{aligned}
\bar{S} &= \frac{lm}{\beta ck}\left(\frac{\beta ck}{lm}\right)\left(\frac{\Lambda k}{\beta ck - lm + \mu k}\right) \\
&= \frac{\Lambda k}{\beta ck - lm + \mu k} \\
\bar{I} &= \frac{\Lambda}{l} - \frac{\mu m}{\beta ck}\left(\frac{\beta ck}{lm}\right)\left(\frac{\Lambda k}{\beta ck - lm + \mu k}\right) \\
&= \frac{\Lambda}{l}\left(\frac{\beta ck - lm}{\beta ck - lm + \mu k}\right) \\
\bar{Q} &= \frac{\Lambda(1-\rho)\omega}{lm}\left(\frac{\beta ck - lm}{\beta ck - lm + \mu k}\right) \\
\bar{P} &= \frac{\Lambda\rho\omega}{lm}\left(\frac{\beta ck - lm}{\beta ck - lm + \mu k}\right)
\end{aligned}
$$

and

$$\bar{A} = \frac{\Lambda v \omega}{lmn} \left(\frac{\beta ck - lm}{\beta ck - lm + \mu k} \right)$$

We summarize these below.

$$\begin{pmatrix} \bar{S} \\ \bar{I} \\ \bar{Q} \\ \bar{P} \\ \bar{A} \end{pmatrix} = \begin{pmatrix} \frac{\Lambda k}{\beta ck - lm + \mu k} \\ \frac{\Lambda}{l} \left(\frac{\beta ck - lm}{\beta ck - lm + \mu k} \right) \\ \frac{\Lambda(1-\rho)\omega}{lm} \left(\frac{\beta ck - lm}{\beta ck - lm + \mu k} \right) \\ \frac{\Lambda \rho \omega}{lm} \left(\frac{\beta ck - lm}{\beta ck - lm + \mu k} \right) \\ \frac{\Lambda v \omega}{lmn} \left(\frac{\beta ck - lm}{\beta ck - lm + \mu k} \right) \end{pmatrix}$$

Note that the endemic equilibrium exists if and only if $\beta ck > lm$, i.e. $\beta c > \frac{(\mu+\omega)(\mu+v)}{\mu+v+(1-\rho)\omega}$.

G.2 Local Stability Properties

We now proceed to look at the local analysis of these two fixed points. The Jacobian is:

$$\begin{pmatrix} -\mu - Z_1 & -Z_2 & -Z_2 & 0 & 0 \\ Z_1 & Z_2 - l & Z_2 & 0 & 0 \\ 0 & (1-\rho)\omega & -m & 0 & 0 \\ 0 & \rho\omega & 0 & -m & 0 \\ 0 & 0 & v & v & -n \end{pmatrix}$$

where,

$$Z_1 = \frac{\beta c (I + Q)(N - S)}{N^2}, \text{ and}$$
$$Z_2 = \frac{\beta c (N - (I + Q))S}{N^2}.$$

G.2.1 Disease Free Equilibrium

The Jacobian at the disease free equilibrium, $(\frac{\Lambda}{\mu}, 0, 0, 0, 0)$, is identical to that for the SIQPA model. Recall the eigenvalues,

$$\lambda_1 = -\mu$$

$$\lambda_2 = \frac{\beta c - l - m}{2} + \frac{1}{2}\sqrt{(\beta c - l + m)^2 + 4\beta c(1 - \rho)\omega}$$

$$\lambda_3 = \frac{\beta c - l - m}{2} - \frac{1}{2}\sqrt{(\beta c - l + m)^2 + 4\beta c(1 - \rho)\omega}$$

$$\lambda_4 = -n$$

$$\lambda_5 = -m$$

As in previous sections, note that the eigenvalues are real and all negative, provided $\beta c k < l m$. Therefore the disease free equilibrium is locally asymptotically provided $\beta c < \frac{l m}{m + (1 - \rho)\omega}$.

G.2.2   Endemic Equilibrium

The characteristic equation is derived first.

$$|J_{(S,I,\bar{Q},P,\bar{A})} - \lambda I| =$$

$$
\begin{vmatrix}
-(\mu + \lambda) - Z_1 & -Z_2 & -Z_2 & 0 & 0 \\
Z_1 & Z_2 - (l + \lambda) & Z_2 & 0 & 0 \\
0 & (1 - \rho)\omega & -(m + \lambda) & 0 & 0 \\
0 & \rho\omega & 0 & -(m + \lambda) & 0 \\
0 & 0 & v & v & -(n + \lambda)
\end{vmatrix}
$$

$(R1 \rightarrow R1 + R2)$

$$
= \begin{vmatrix}
-(\mu + \lambda) & -(l + \lambda) & 0 & 0 & 0 \\
Z_1 & Z_2 - (l + \lambda) & Z_2 & 0 & 0 \\
0 & (1 - \rho)\omega & -(m + \lambda) & 0 & 0 \\
0 & \rho\omega & 0 & -(m + \lambda) & 0 \\
0 & 0 & v & v & -(n + \lambda)
\end{vmatrix}
$$

$$
= (n + \lambda)(m + \lambda) \begin{vmatrix}
-(\mu + \lambda) & -(l + \lambda) & 0 \\
Z_1 & Z_2 - (l + \lambda) & Z_2 \\
0 & (1 - \rho)\omega & -(m + \lambda)
\end{vmatrix}
$$

$$
= -(n + \lambda)(m + \lambda)\left(Z_1(l + \lambda)(m + \lambda) - (\mu + \lambda)((Z_2 - l - \lambda)(m + \lambda))\right)
$$
$$
+ (\mu + \lambda)(n + \lambda)(m + \lambda)Z_2(1 - \rho)\omega
$$

At this point we see that two of the eigenvalues,

$$\lambda_4 = -n$$

$$\lambda_5 = -m, \tag{G.2}$$

those corresponding to the P and A populations, are negative. Thus we need only examine the remaining cubic polynomial of the characteristic equation for local stability properties of the endemic equilibrium.

Collecting the cubic polynomial with respect to λ we have,

$$
\begin{aligned}
0 =\ & \lambda^3 + \lambda^2(\mu - Z_2 + l + m + Z_1) \\
& + \lambda((l+m)Z_1 + lm - Z_2 k + \mu(l+m) - \mu Z_2) \\
& + \mu lm + lm Z_1 - \mu k Z_2.
\end{aligned}
$$

We now simplify the terms Z_1 and Z_2.

$$
\begin{aligned}
\frac{I}{N} &= \frac{\Lambda}{lN} - \frac{\mu m}{\beta ck} \\[2mm]
&= \frac{\Lambda}{l}\left(\frac{lm}{\Lambda k \beta ck}\right)(\beta ck - lm + \mu k) - \frac{\mu m}{\beta ck} \\[2mm]
&= \frac{m}{k\beta ck}(\beta ck - lm)
\end{aligned}
$$

$$
\begin{aligned}
Z_1 &= \frac{\beta c(I+Q)(N-S)}{N^2} \\[2mm]
&= \frac{\beta ckI}{mN} - \left(\frac{\beta ckI}{mN}\right)\frac{S}{N} \\[2mm]
&= \frac{\beta ck}{m}\left(\frac{m}{k\beta ck}(\beta ck - lm)\right)\left(1 - \frac{lm}{\beta ck}\right) \\[2mm]
&= \frac{(\beta ck - lm)^2}{k\beta ck}
\end{aligned}
$$

$$
Z_2 = \frac{\beta c(N-(I+Q))S}{N^2}
$$

$$= \beta c \left(\frac{lm}{\beta ck} \right) \left(1 - \frac{kI}{mN} \right)$$

$$= \frac{lm}{k} \left(\frac{lm}{\beta ck} \right)$$

Substituting these values for Z_1 and Z_2 into the characteristic equation, we derive the coefficients a_i for $i = 0, 1, 2, 3$ corresponding to λ^{3-i}.

$$a_0 = 1$$

$$a_1 = \mu + l + m + \frac{(\beta ck - lm)^2}{k\beta ck} - \frac{lm}{k} \left(\frac{lm}{\beta ck} \right)$$

$$= \mu + l + m + \frac{\beta ck(\beta ck - 2lm) + l^2 m^2 - l^2 m^2}{k\beta ck}$$

$$= \mu + m + \frac{\beta ck - lm}{k} + l \left(\frac{k - m}{k} \right)$$

$$= \mu + m + \frac{\beta ck - lm}{k} + \frac{l}{k}(1 - \rho)\omega$$

$$a_2 = (l + m)Z_1 + lm + \mu(l + m) - (\mu + k)\frac{lm}{k} \left(\frac{lm}{\beta ck} \right)$$

$$= (l + m)Z_1 + \mu m + l \left(\frac{\beta ck^2 \mu + \beta ck^2 m - \mu lm^2 - klm^2}{k\beta ck} \right)$$

$$= (l + m)Z_1 + \mu m + \frac{\mu l(1 - \rho)\omega}{k}$$
$$+ l \left(\frac{\beta cl\mu m - \mu lm^2 + \beta ck^2 m - klm^2}{k\beta ck} \right)$$

$$= (l + m)Z_1 + \mu m + \frac{\mu l(1 - \rho)\omega}{k} + \frac{lm(\mu + k)(\beta ck - lm)}{k\beta ck}$$

$$a_3 \;\; = \;\; \mu lm + lmZ_1 - \mu k \frac{lm}{k} \left(\frac{lm}{\beta ck} \right)$$

$$= \;\; \mu lm \left(\frac{\beta ck - lm}{\beta ck} \right) + lmZ_1.$$

Thus, all coefficients of the characteristic equation are positive. To satisfy the Routh-Hurwitz requirements we have left to show, that the determinant condition $a_1 a_2 - a_3 > 0$.

$a_1 a_2 - a_3 =$

$$\frac{\beta ck - lm}{k} \left((l+m)Z_1 + \mu m + \frac{\mu l(1-\rho)\omega}{k} + \frac{lm(\mu+k)(\beta ck - lm)}{k\beta ck} \right)$$

$$+ \frac{l(1-\rho)\omega}{k} \left((l+m)Z_1 + \mu m + \frac{\mu l(1-\rho)\omega}{k} + \frac{lm(\mu+k)(\beta ck - lm)}{k\beta ck} \right)$$

$$+ \mu \left((l+m)Z_1 + \mu m + \frac{\mu l(1-\rho)\omega}{k} + \frac{lm(\mu+k)(\beta ck - lm)}{k\beta ck} \right)$$

$$+ m \left((l+m)Z_1 + \mu m + \frac{\mu l(1-\rho)\omega}{k} + \frac{lm(\mu+k)(\beta ck - lm)}{k\beta ck} \right)$$

$$- \left(\mu lm \left(\frac{\beta ck - lm}{\beta ck} \right) + lmZ_1 \right)$$

$$= \;\; \frac{\beta ck - lm}{k} \left((l+m)Z_1 + \mu m + \frac{\mu l(1-\rho)\omega}{k} + \frac{lm(\mu+k)(\beta ck - lm)}{k\beta ck} \right)$$

$$+ \frac{l(1-\rho)\omega}{k} \left((l+m)Z_1 + \mu m + \frac{\mu l(1-\rho)\omega}{k} + \frac{lm(\mu+k)(\beta ck - lm)}{k\beta ck} \right)$$

$$+ \mu \left((l+m)Z_1 + \mu m + \frac{\mu l(1-\rho)\omega}{k} + \frac{lm\mu(\beta ck - lm)}{k\beta ck} \right)$$

$$+ m \left(mZ_1 + \mu m + \frac{\mu l(1-\rho)\omega}{k} + \frac{lm(\mu+k)(\beta ck - lm)}{k\beta ck} \right)$$

$$> \;\; 0.$$

Thus, the Routh-Hurwitz criteria are satisfied for the cubic polynomial of the characteristic equation and since $\lambda_{1,2}$ in G.2 are negative, we can conclude that the endemic equilibrium for the SIQ/PA model is locally asymptotically stable provided $\beta c > \frac{lm}{m+(1-\rho)\omega}$.

Appendix H

Supporting Proofs and Background Theory

H.1 Boundedness

We require that our models be well-posed, that is, solutions must remain positive and bounded. The proofs are provided below.

Theorem H.1 *All solutions $S(t), I(t), Q(t), P(t), A(t)$ of 2.1 are (a) positive and (b) bounded for $t > 0$.*

Proof of (a): $S_o > 0$. By definition, $S(t)$ is continuous. Suppose there exists $\bar{t} > 0$ such that $S(t) > 0$ for $0 \leq t < \bar{t}$ and $S(\bar{t}) = 0$. Then $S'(\bar{t}) \leq 0$. However, by (2.1), $S(\bar{t}) = 0$ implies that $S'(\bar{t}) = \Lambda > 0$, a contradiction. Therefore $S(t) > 0$ for all $t \geq 0$.

$Q_o \geq 0$ and $I_o > 0$. By definition, $I(t)$ and $Q(t)$ are continuous. Suppose there exists $\bar{t} > 0$ such that $I(t) > 0$ and $Q(t) \geq 0$ for $0 \leq t < \bar{t}, Q(\bar{t}) > 0$ and $I(\bar{t}) = 0$. Then $I'(\bar{t}) \leq 0$. However, by (2.1), $I(\bar{t}) = 0$ and $Q(\bar{t}) > 0$ implies that $I'(\bar{t}) = \frac{\beta c Q(\bar{t}) S(\bar{t})}{N(\bar{t})} > 0$, a contradiction.

We obtain a contradiction similarly if we assume $Q(\bar{t}) = 0$ and $I(\bar{t}) > 0$, with $Q(t) \geq 0$ and $I(t) > 0$ for $0 \leq t < \bar{t}$.

Now suppose that $I(t)$ and $Q(t)$ are as above for $0 \leq t < \bar{t}$, but $I(\bar{t}) = Q(\bar{t}) = 0$. Then $I(t) = Q(t) \equiv 0$ for all $t > \bar{t}$. But, this contradicts uniqueness of solutions. $I(t) \equiv 0$ must be a solution for all time, and if $I(t) > 0$ for some time $0 \leq t < t_\epsilon \leq \bar{t}$, (since $I_o > 0$) then there are two solutions $I(t) \equiv 0$ and $I(t) > 0$ for $0 < t < t_\epsilon$, a contradiction to uniqueness of solution. Thus $I(t) > 0$ and $Q(t) \geq 0$ for all time $t \geq 0$.

Using positivity of $I(t)$ and $Q(t)$, the arguments for $P(t)$ and $A(t)$ follow similarly. Therefore all solutions $S(t), I(t), Q(t), P(t)$, and $A(t)$ are positive.

Proof of (b): Recall that

$$
\begin{aligned}
N'(t) &= \Lambda - \mu N(t) - dA(t) \\
&\leq \Lambda - \mu N(t)
\end{aligned}
$$

Therefore

$$
N(t) \leq \left[N(0) - \frac{\Lambda}{\mu} \right] e^{-\mu t} + \frac{\Lambda}{\mu}
$$

If $N(0) < \frac{\Lambda}{\mu}$ then $N(t) \leq \frac{\Lambda}{\mu}$, otherwise $N(t) \leq N(0)$. Since $N(t) = S(t) + I(t) + Q(t) + P(t) + A(t)$, then

$$
S(t) + I(t) + Q(t) + P(t) + A(t) \leq \left\{ \begin{array}{l} \frac{\Lambda}{\mu} \text{ if } N_o \leq \frac{\Lambda}{\mu} \\ N_o \text{ otherwise.} \end{array} \right\}
$$

Since all solutions are positive by (a), then all solutions of system 2.1 are bounded.

We can similarly show that all the models in this thesis have bounded and positive solutions.

H.2   Background Theory

H.2: Routh-Hurwitz Criteria [23] Consider

$$p(\alpha) = A_o\alpha^n + A_1\alpha^{n-1} + \ldots + A_{n-1}\alpha + A_n \text{ with } A_o > 0$$

Define

$$\triangle_1 = A_1, \quad \triangle_2 = \begin{vmatrix} A_1 & A_o \\ A_3 & A_2 \end{vmatrix}, \quad \triangle_3 = \begin{vmatrix} A_1 & A_o & 0 \\ A_3 & A_2 & A_1 \\ A_5 & A_4 & A_3 \end{vmatrix}.$$

In general, define

$$\triangle_n = \begin{vmatrix} A_1 & A_o & 0 & 0 & 0 & 0 & 0 & \ldots & 0 \\ A_3 & A_2 & A_1 & A_o & 0 & 0 & 0 & \ldots & 0 \\ A_5 & A_4 & A_3 & A_2 & A_1 & A_o & 0 & \ldots & 0 \\ \vdots & \vdots & \vdots & \vdots & \vdots & \vdots & \vdots & \ldots & \vdots \\ A_{2n-1} & A_{2n-2} & A_{2n-3} & A_{2n-4} & A_{2n-5} & A_{2n-6} & A_{2n-7} & \ldots & A_n \end{vmatrix},$$

where $A_j = 0$ for $j > n$. In particular, $\triangle_n \equiv A_n \triangle_{n-1}$.

A necessary and sufficient condition for all of the roots of $p(\alpha)$ to have their real parts negative is that all the determinants $\triangle_i$, $i = 1, \ldots, n$, be positive.

H.3: Persistence [22]

A population $p(t)$ is said to persist in $\Re^n$ if $p(0) > 0$ and $\liminf_{t\to\infty} p(t) > 0$. A system is said to be persistent if each component population persists. For ecological dynamical systems, a solution with initial conditions in the positive cone will persist if there are no Ω- limit points on the boundary of $\Re^n_+$.

H.4: The Butler-McGehee Lemma [22]

Let P be an isolated equilibrium with nonzero eigenvalues in the omega limit set $\Omega(R)$ of an orbit $\mathcal{O}(R)$ through the point R. Then either $\Omega(R) = \{\mathcal{P}\}$ or there exist points $\mathcal{P}^s$, $\mathcal{P}^u$ in $\Omega(R)$ with $\mathcal{P}^s \in W^s(\mathcal{P}) \setminus \{\mathcal{P}\}$ and $\mathcal{P}^u \in W^u(\mathcal{P}) \setminus \{\mathcal{P}\}$.

H.5: Lyapunov Function [40]

Consider the general system of differential equations

$$x' = f(x) \tag{H.1}$$

Here $f(x)$ is a vector-valued function, continuous in x for $x \in cl\mathcal{G}$ where $\mathcal{G}$ is an open subset of $\Re^n$. The function V mapping $\Re^n$ to $\Re$ is said to be a *Lyapunov Function* in $\mathcal{G}$ for H.1 if it satisfies the following properties:

1. $V(x)$ is continuous on $cl\mathcal{G}$.
2. $\dot{V} = (\nabla V) \cdot f \leq 0$ in $\mathcal{G}$.

H.6: La Salle's Extension Theorem [40]

Let V be a Lyapunov function in $\mathcal{G}$ for H.1. Then each bounded orbit of H.1 approaches $\mathcal{M}$ where $\mathcal{M}$ is the largest invariant subset of $\{x \in cl\mathcal{G} : \dot{V} = 0\}$.

H.3 Substitutions

Throughout the thesis we use many simplifying substitutions. These are provided below:

$$m = \mu + v$$

$$n = \mu + d$$

$$l = \mu + \omega$$

$$M = \beta c - (\mu + v)$$

$$T = \beta c(1 - \rho) - (\mu + v)$$

$$D = \beta c(\mu + v + d)$$

$$X = mn + n\omega + v\omega$$

$$f = m + \omega$$

$$U = \beta c - (\mu + \omega)$$

$$k = m + (1 - \rho)\omega$$

$$Y = \frac{n}{X}(\beta ck - lm)$$

$$R_1 = \frac{\beta cS}{N}$$

$$R_2 = \frac{\beta cIS}{N^2}$$

$$R_3 = \frac{\beta cI}{N}$$

$$Z_1 = \frac{\beta c(I + Q)(N - S)}{N^2}$$

$$Z_2 = \frac{\beta c(N - (I + Q))S}{N^2}$$

$$Z_3 = \frac{\beta c(I + Q)S}{N^2}$$

with appropriate values for N in models SIQPA and SIQ/PA

All other substitutions that are used in this thesis are listed in the appropriate sections.

Bibliography

[1] Allen, J.R., Curran, J.W., *Prevention of AIDS and HIV Infection: Needs and Priorities for Epidemiologic Research,* Amer. J. Public Health **78,** No. **4,** (1988), 381-386.

[2] Anderson, R.M., and May, R.M., *Epidemiological parameters of HIV transmission,* Nature **333,** (1988), 514-519.

[3] Anderson, R.M., and May, R.M., *Population biology of infectious diseases: Part I,* Nature, **280,** (1979), 361-367.

[4] Anderson, R.M., and May, R.M., *Population biology of infectious diseases: Part II,* Nature **280,** (1979), 455-461.

[5] Anderson, R.M. and May, R.M., *Transmission dynamics of HIV infection,* Nature **326,** (1987),137-142.

[6] Anderson, R.M., May, R.M., Blower, S.M., *The Epidemiology and Transmission Dynamics of HIV-AIDS,* Journal of the American Academy of Arts and Sciences, **118, No.2,** (1989), 163-201.

[7] Anderson, R.M., Medley, G.F., May, R.M., and Johnson, A.M., *A preliminary study of the transmission dynamics of the human immunodeficiency virus (HIV), the causative agent of AIDS,* IMA J. Math. Appl. Med. & Biol.**3**(1986),229-263.

[8] Bailey, N.T.J., *The Mathematical Theory of Epidemics,* 2nd ed., Hafner, New York, (1975).

[9] Blythe, S.P., and Castillo-Chavez, C., *Like-with-Like Preference and Sexual Mixing Models,* Mathematical Biosciences **96**, (1989), 221-238.

[10] Busenberg, S., and Castillo-Chavez, C., *Interaction, pair formation and force of infection terms in sexually transmitted disease,* Lecture Notes in Biomathematics **83**, Springer-Verlag, Berlin, Heidelberg, New York, Tokyo, (1989), 289-300.

[11] Castillo-Chavez, C., *Review of Recent Models of HIV/AIDS Transmission,* in Applied Mathematical Ecology. (Eds. S.A. Levin, T.G, Hallam, and L.J. Gross.) Biomathematics **18**,(1989), 253-262.

[12] Castillo-Chavez, C., Cooke, K., Huang, W., Levin, S., *On the role of long incubation periods in the dynamics of acquired immunodeficiency syndrome (AIDS), Part 1,* J. Math. Biol. **27**, (1989), 373-398.

[13] Castillo-Chavez, C., Cooke, K., Huang, W., and Levin, S., *On the Role of Long Incubation Periods in the Dynamics of Acquired Immunodeficiency Syndrome (AIDS),* Lecture Notes in Biomathematics **83**, Springer-Verlag, Berlin, Heidelberg, New York, Tokyo. (1989), 200-217.

[14] Castillo-Chavez, C., Cooke, K., Huang, W., and Levin, S., *The role of long periods of infectiousness in the dynamics of acquired immunodeficiency syndrome (AIDS),* in Mathematical approaches to problems in resource management and epidemiology. (Eds. C. Castillo-Chavez, S.A, Levin, and C. Shoemaker.) Lecture notes in Biomathematics **81**, Springer-Verlag, Berlin, Heidelberg, New York, Tokyo. (In press.)

[15] Castillo-Chavez, C., Hethcote, H., Andreasen, V., Levin, S., and Liu, W., *Epidemiological models with age structure, proportionate mixing, and cross-immunity,* J. Math. Biol. **27**, (1989), 233-258.

[16] Colgate, S.A., Stanley, E.A., Hyman, J.H., Layne, S.P., and Qualls, C., *Risk behavior-based model of the cubic growth of acquired immunodeficiency syndrome in the United States,* Proc. Natl. Acad. Sci. USA **86**, (1989), 4793- 4797.

[17] Cooke, K.L., and Yorke, J.A., *Equations Modelling Population Growth and Gonorrhea Epidemiology,* Math. Biosci. **16**, (1973) 75-101.

[18] Cooper, Necia Grant (Ed.), *Los Alamos Science,* **18**, Los Alamos National Laboratory, (1989).

[19] Dietz, K., and Hadeler, K.P., *Epidemiological models for sexually transmitted diseases,* J. Math. Biol. **26** (1988),1-25.

[20] Fredrickson, A.G., *A Mathematical Theory of Age Structure in Sexual Populations: Random Mating and Monogamous Marriage Models,* Mathematical Biosciences **10**, (1971), 117-143.

[21] Freedman, H.I., and Waltman, P., *Mathematical analysis of some three-species food-chain models,* Math. Biosci. **68**, (1984), 213-231.

[22] Freedman, H.I., and Waltman, P., *Persistence in models of three interacting predator-prey populations,* Mathematical Biosciences **68**, (1984), 213-231.

[23] Hacker, T., *Flight Stability and Control,* American Elsevier, (1970).

[24] Hadeler, K.P., Waldstätter, R., and Wörz- Busekros, A., *Models for pair formation in bisexual populations,* J. Math. Biol. **26**, (1988), 635-649.

[25] Hale, J.K., *Ordinary Differential Equations,* Wiley- Interscience, New York, London, Sydney, Toronto, (1969).

[26] Hethcote, Herbert W., *Qualitative analyses of communicable disease models,* Mathematical Biosciences **28**, (1976),335-356.

[27] Hethcote, H.W., van Ark, J.W, *Epidemiological methods for heterogeneous populations: proportional mixing, parameter estimation, and immunization programs,* Math. Biosci. **84**, (1987), 85-118.

[28] Hethcote, H.W. and Yorke, J.A., *Gonorrhea transmission dynamics and control,* Lecture Notes in Biomathematics **56**, Springer-Verlag, Berlin, Heidelberg, New York, Tokyo, (1984).

[29] Hoppensteadt, F., and Waltman, P., *A Problem in the Theory of Epidemics*, Math. Biosciences **9**, (1970), 71-91.

[30] Hoppensteadt, F., and Waltman, P., *A Problem in the Theory of Epidemics II*, Math. Biosci. **12**, (1971), 133-145.

[31] Hyman, J., and Stanley, E. *The Effect of Social Mixing Patterns on the Spread of AIDS.* Proceedings of Mathematical Approaches to Environmental and Ecological Problems. September 1988.

[32] Hyman, J.M., and Stanley, E.A., *Using Mathematical Models to Understand the AIDS Epidemic,* Mathematical Biosciences **volume** , (1988) pages.

[33] Jacquez, J., Simon, C., Koopman, J., *Structured mixing: heterogeneous mixing by the definition of activity groups,* Lecture Notes in Biomathematics **83**, Springer-Verlag, Berlin, Heidelberg, New York, Tokyo, (1989), 301-315.

[34] Jacquez, J., Simon, C., Koopman, J., Sattenspiel, L., and Perry, T., *Modeling and Analyzing HIV Transmission:The Effect of Contact Patterns,* Mathematical Biosciences **92**, (1988), 119-199.

[35] Judson, F.N., MD, and Vernon, T.M., Jr., MD, *The Impact of AIDS on State and Local Health Departments: Issues and a Few Answers,* Amer. J. Public Health, **78** No. **4**, (1988), 387-393.

[36] Kermack, W.O., and McKendrick, A.G., *Contributions to the mathematical theory of epidemics, part I,* Proc. R. Soc., Ser A, **115**, (1927), 700-721.

[37] Kermack, W.O., and McKendrick, A.G., *Contributions to the mathematical theory of epidemics, part II,* Proc. Roy. Soc., Ser. A, **138**, (1932), 55-83.

[38] Koopman, J., Simon, C., Jacquez, J., Joseph, J., Sattenspiel, L., and Park, T., *Sexual Partner Selectiveness Effects on Homosexual HIV Transmission Dynamics,* J. AIDS **1**, (1988), 486-504.

[39] Lajmanovich, A., and Yorke, J.A., *A deterministic model for gonorrhea in a nonhomogeneous population,* Math. Biosci. **28**m (1976), 221-236.

[40] LaSalle, J.P., and Lefschetz, S., *Stability by Liapunov's Direct Method with Applications,* Academic Press, New York, (1961).

[41] Lyter, D.W., Valdiserri, R.O., Kingsley, L.A., Amoroso, W.P., Rinaldo, C.R., *The HIV Antibody Test: Why Gay and Bisexual Men Want or Do Not Want To Know Their Results,* Public Health Rep **102**, (1987), 468-474.

[42] May, R.M, Anderson, R.M., and McLean, A., *Possible Demographic Consequences of HIV/AIDS Epidemics. 1. Assuming HIV Infection Always Leads to AIDS,* Mathematical Biosciences **90**, (1988), 475-505.

[43] McCusker, J., Stoddard, A.M., Mayer, K.H., Zapka, J., Morrison, C. and Saltzman, S.P., *Effects of HIV Antibody Test Knowledge on Subsequent Sexual Behaviours in a Cohort of Homosexually Active Men,* Amer. J. Public Health, **78**, No. **4**, (1988), 462-467.

[44] Medley, G., Anderson, R., Cox, D., and Billard, L., *Incubation period of AIDS in patients infected via blood transfusion,* Nature **328**, (1987), 719-721.

[45] Sattenspiel, L. Simon, C., *The Spread and Persistence of Infectious Diseases in Structured Populations,* Mathematical Biosciences **90**, (1988) 341-366.

[46] Schwager, S., Castillo-Chavez, C., and Hethcote, H., *Statistical and Mathematical Approaches in HIV/AIDS Modeling: A Review,* Lecture Notes in Biomathematics **83**, Springer-Verlag, Berlin, Heidelberg, New York, Tokyo (1989), 2-36.

[47] Simon, C., Jacquez, J., *Reproduction Numbers and the Stability of Equilibria of SI Models for Heterogeneous Populations,* Preprint (1990).

[48] Stanley, E.A., *Mathematical Models of the AIDS Epidemic: An Historical Perspective,* Lectures in Complexity, Santa Fe Institute Studies in the Science of Complexity. Addison-Wesley, Reading, MA., **VIII**, (1989).

[49] Thieme, H.R. and Castillo-Chavez, *On the Role of Variable Infectivity in the Dynamics of the Human Immunodeficiency Virus Epidemic,* Lecture Notes in Biomathematics **83**, Springer-Verlag, Berlin, Heidelberg, New York, Tokyo. (1989), 157-177.

[50] Waltman, Paul, **Deterministic Threshold Models in the Theory of Epidemics,** Lecture Notes in Biomathematics **1**, (ed. S. Levin), Springer-Verlag, Berlin, Heidelberg, New York, Tokyo, (1974).

[51] Winkelstein, W., Lyman, D., Padian, N., Grant, R., Samuel, M., Wiley, J., Anderson, R.E., Lang, W. Riggs, J., Levy, J., *Sexual Practices and Risk of Infection by the Human Immunodeficiency Virus. The San Francisco Men's Health Study,* JAMA, **257, No.3**, (1987), 321-325.

[52] Workshop **A national effort to model AIDS epidemiology,** Office of Science and Technology Policy, Executive Office of the President, Report of a workshop held at Leesburg, Virginia, July 25- 29, 1988.

[53] Yorke, J.A., Hethcote, H.W., and Nold, A., *Dynamics and control of the transmission of gonorrhea,* Sexually Transmitted Diseases **10**, (1978), 72-76.