Mathematical modelling to investigate the impact of awareness programs on the spread of HIV/AIDS amongst people who inject drugs (PWIDs)

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Abstract

Injecting drug use is a growing risk factor for the transmission of the human immunodeficiency virus (HIV) and acquired immunodeficiency syndrome (AIDS)in the majority of countries, and the high prevalence of HIV among many populations of persons who inject drugs (PWIDs) presents a huge global health issue (Mathers et al. 2008). Approximately 11.3% of the world's population uses injection medicines in relation to drugs and crime on Drugs and Crime (2020). The risk of drug overdose and blood-borne infection, especially HIV and Hepatitis B and C, which are transmitted through the sharing of contaminated needles and syringes and risky sexual behaviours of individuals who have been infected, makes injection drug use a major public health problem and a leading cause of morbidity and mortality on Drugs and Crime (2020).

The spread of HIV has seen the widespread application of mathematical modelling approaches. In most nations around the world, the injection of drugs is a significant contributor to the spread of HIV/AIDS. The media plays a significant role in raising health consciousness and influencing behaviour change. The existing literature illustrates how differential equation models can be used to describe the effects of media awareness initiatives on the spread and containment of disease (Greenhalgh et al. 2015). In this thesis, we consider the effect of an awareness program on the dynamic behaviour of the spread of HIV/AIDS amongst PWIDs. The HIV/AIDS model can be modelled using the SIS and SIR models with time-varying parameter values. We develop the mathematical differential equation model that extends the research by Greenhalgh and Hay (1997),

Liang et al. (2016) andLewis and Greenhalgh (2001) to illustrate the impact of disease awareness campaigns on the rate of HIV transmission among PWIDs. The new assumption of the model is that PWIDs clean their needles before use.

For each of these different epidemic models, we have developed a mathematical model to represent the new, more effective model that curbs the spread of the diseases by decreasing the prevalence of needle and syringe sharing among PWIDs. We have primarily discussed two approaches for examining how awareness of infection levels affects epidemic modelling. First, we perform an analysis of stability and provide both local and global results. The fundamental reproduction number R_0, an essential factor in our work, has a formula that we determine. If R_0 is greater than one, there are two steady states: one without disease and one with it. Additionally, we demonstrated that the disease-free equilibrium point is locally asymptotically stable when R_0 is less than one and neutrally stable when $R_0 = 1$, and unstable when $R_0 > 1$.

These analytical results are confirmed and investigated numerically by simulating the equations with the SOLVER computer simulation software. The realistic parameters for these simulations were derived from data and the infectious disease literature. To conclude the thesis, a brief discussion and summary section are provided.

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Chapter 1

Introduction and Literature Review

1.1 Motivation

In the early 1980s, human immunodeficiency virus (HIV) was found. It is an immunodeficiency virus that weakens the immune system, increases susceptibility to infection, and causes acquired immunodeficiency syndrome (AIDS) in the long run. HIV is transferred through a variety of routes, including homosexual and heterosexual sex. However, the injection of drugs is one of the most significant ways HIV is transmitted; persons who inject drugs (PWIDs) are often uninformed of their infection and this contributes to the spread of HIV. The coexistence of additional opportunistic infections and diseases with HIV among people who inject drugs (PWIDs) is associated with high morbidity and mortality rates and significant healthcare costs. To prevent and limit the transmission of the disease, the early identification of HIV in PWIDs is crucial.

According to the National Health Service (NHS 2022), the vast majority of infected people develop a mild illness that is comparable to flu and lasts for around two weeks. After that, the person may not have any symptoms for a considerable amount of time. Individuals who are not treated progress from being acutely infected to being asymptomatic, experiencing signs of pre-AIDS, and finally de-

veloping full-blown AIDS. Some tests can determine whether or not someone has HIV. Since HIV/AIDS is still a significant issue, it is of the utmost importance to create mathematical models that can assist us in this endeavour.

Since infectious diseases pose a serious threat to humankind and result in death, disfigurement, as well as social and economic costs as governments and health organizations take action to stop the spread of infection. The logical course of action is to immediately inform the public about the disease and its preventive measures through the media in the absence of effective vaccines and therapies.

The media, which serves as the primary information source, has the power not only to affect how people behave but also to boost their engagement in healthcare provided by the government and healthcare providers, which helps to prevent the spread of disease. People are made aware of the illnesses through the media, as well as the needed precautions to help prevent transmission, such as immunization, social isolation, and wearing protective masks.

In recent years, there has been an increase in the number of papers that use mathematical models to estimate the impact of media awareness campaigns on the spread of infectious diseases. Additionally, there are several authors who have studied mathematical models in epidemiology and have a better understanding of the predictions that these models can make. These authors use mathematical models to comprehend disease dynamics and the transmission of infections and to determine the potential effectiveness of influence approaches in limiting HIV/AIDS infections amongst PWIDs.

Thus, this thesis will aim to create mathematical models which explore the impact of the awareness programs on the HIV/AIDS model transmission amongst PWIDs, an area where no study has been done to make the models more realistic. This thesis aims to close that gap. To do so, we primarily use a more basic form of disease awareness program applied to a modified version of the models by (Greenhalgh and Hay 1997), (Liang et al. 2016) and(Lewis and Greenhalgh

2001). This can be accomplished by assuming the assumption that PWIDs clean their needles prior to use, which would result in this change and the validity of this research contribution. The next section provides a brief overview of the research presented in this thesis.

1.2 Overview and Outline of the Thesis

In this thesis, we create a mathematical model to explain the improved model that prevents the transmission of HIV/AIDS diseases through the impact of awareness programs of disease on sharing needles and syringes amongst the PWID population. The model assumes that PWIDs clean their needles prior to usage rather than after. There are two different approaches to modelling this. The first and easiest method, which we will apply in this study, is to reduce the disease transmission term by a factor ϕ $(0 \leq \phi < 1)$ to account for the behavioural changes people make as a result of knowing the prevalence of the disease in their environment. In the second, individuals (typically the susceptible class) are divided into aware and unaware individuals, and where the level of media awareness is modelled as a separate variable(Misra et al. (2011), Greenhalgh et al. (2015)).

In Chapter Two, we present a deterministic mathematical model of the spread of HIV/AIDS amongst people who inject drugs (PWIDs) with an awareness program that is based on the mathematical work of (Greenhalgh and Hay 1997) and the work of (Liang et al. 2016). We then examine the One-dimensional Model for the Spread of HIV/AIDS amongst PWIDs with awareness programs and demonstrate that there is also a unique non-negative solution. Next, we investigate the existence of equilibrium points. We perform stability and equilibrium analyses, showing that the fundamental reproductive number R_0 controls the behaviour of the model. To support our analytical findings for HIV/AIDS models with disease awareness campaigns, numerical simulations are also generated. These simula-

tions use both theoretical and realistic parameter values. Then, we use our model to address the HCV transmission among PWIDs and run additional simulations to confirm their analytical findings.

In Chapter Three, based on the model constructed in Chapter Two, we discuss the impact of awareness programs in the two-dimensional model of HIV transmission amongst PWIDs. We investigate the existence of a unique positive solution to the differential equations. The existence of a solution at equilibrium is then investigated. Next, we assess the stability of equilibrium at the local and global stability. Then, additional simulations are conducted to verify the analytical results.

In Chapter Four, we modified a three-stage infectivity model for the transmission of HIV/AIDS among intravenous PWIDs by (Lewis and Greenhalgh 2001) to make it more realistic by applying for awareness programs as described in Chapter Two. This model allows PWIDs to pass through three stages of infection before the development of AIDS. We analyse the equation model system to gain insights into its dynamical characteristics, which will help us better understand how the awareness program impacts the three-stage infection model for the transmission of HIV/AIDS amongst PWIDs. Additionally, we compute an expression for R_0. We consider and analyse the system's likely endemic equilibrium, as well as the disease-free equilibrium, are taken into consideration and analysed. We also examine the stability of these equilibria locally and globally, as well as the persistence of the disease. Simulation and a numerical analysis are also provided.

In Chapter Five, motivated by the work that has been done in the previous chapter, we extend and develop the mathematical model of the effect of awareness programs on the HIV/AIDS models with successful antiviral treatment in Chapter Two. We extended the model to include two groups within our PWID population: those PWIDs who are infected but unaware that they are infected, and

those PWIDs who are on successful highly active antiretroviral therapy (HAART). Driven by the fact that the model includes two groups of PWID population, those PWIDs who are infected but unaware that they are infected and those PWIDs who are on successful HAART. we extend our model to consider this more realistic assumption that allows PWIDs to move through the phases of HIV/AIDS infection. The model is then theoretically analysed, a formula for the fundamental reproductive number R_0 is derived, and simulations based on parameter values for our model are run to confirm our theoretical results.

The final chapter, Chapter Six, provides a summary of the work presented in this thesis and discusses some proposals for future research. Our summary of the work presented in this thesis is now complete.The rest of this chapter will continue with give a literature survey and present an introduction to the HIV/AIDS virus, its discovery and its transmission routes. We then provide a review of awareness programs in the epidemic model as well as the use of mathematical models in epidemiology is provided. Finally, we shall present a review of some previous mathematical modelling of HIV/AIDS amongst PWIDs.

1.3 Background on HIV/AIDS Virus Infection

Since the first case of HIV infection was identified from a 1959 sample collected from a man in the Belgian Congo, scientists have studied the virus long enough to know it is an immune deficiency virus that not only weakens the cells but also attacks the immune system and makes the cells deficient in fighting other infections. In other words, the virus weakens the immune system and its ability to defend the body from other infections that could attack it. The transmission of the HIV virus through body fluids means that its methods of transmission are as diverse as the people it attacks. Some known ways HIV can be transmitted include semen, a blood transfusion, breast milk, vaginal fluids, and rectal fluids.

Since the discovery of HIV about 40 years ago, it has become a serious public health issue, and many screening centres have been established to ease the counselling and testing of people for the virus (Opeodu and Ogunrinde 2015). The global community has witnessed momentous innovations that have significantly changed the landscape of HIV care. In particular, advancements in antiretroviral therapy (ART) over the last twenty years have transformed HIV/AIDS from a rapidly progressing ailment to what most consider a chronic disease (Ivy et al., 2017). HIV is an infective organism that usually targets the immune system of the victims, making them more susceptible to a wide range of infections and certain types of cancers (Ndibuagu et al. 2017). The disease was given various names in the past, including gay-related immune deficiency (GRID), but in the year 1982, the Centers for Disease Control named it Acquired Immune Deficiency Syndrome (AIDS). which it is still being called this present day (Ndibuagu et al. 2017).

The first two cases of AIDS in Nigeria were diagnosed in Lagos in the year 1985 and reported at the International AIDS Conference in 1986 (Ndibuagu et al. 2017). HIV attacks immune cells called CD4 cells. These T cells (white blood cells) circulate, detecting infections throughout the body, along with faults and anomalies in other cells. HIV targets and infiltrates the CD4 cells, using them to create more of the virus. This act consumes the cells and reduces the body's ability to combat other infections and diseases. It increases the risk and influence of opportunistic infections and some forms of cancer. It is worth noting that some people have HIV for long periods without experiencing any symptoms. It is a life-long condition, but treatments and specific techniques can prevent the virus from transmitting and the infection from further infiltrating. Medical personnel identify AIDS as having a CD4 count of fewer than 200 cells per cubic millimetre. Also, they may diagnose AIDS if a person has attributes of opportunistic infections that are associated with any form of cancer or both. When a person with HIV does not receive treatment, AIDS likely develops as the immune system procedurally

wears down. However, advances in antiretroviral treatments have made this progression to AIDS increasingly less common (Felman (2020)).

HIV has become a pandemic and a significant cause of global mortality. An estimated 33.2 million adults and children are living with HIV. And in 2007 alone, around 2.7 million people were newly infected, out of which 2.0 million died. Global adult (age 15–49 years) prevalence in 2007 was estimated at 0.8%. And that same year, 67% of all people and 90% of the estimated 2.0 million children living with HIV lived in sub-Saharan Africa (SSA). Adult prevalence in sub-Saharan Africa was estimated to be 5%. Acquired Immune Deficiency Syndrome (AIDS), and its causative agent, HIV, are now known globally and no longer some strange to the universe (Hoskins 2014).

The HIV/AIDS epidemic is one of the world's mysterious public health and social problems. Promoting knowledge and a positive attitude towards HIV/AIDS are central to controlling the prevalence of this epidemic(Yaya et al. 2019). The virus is the cause of one of the most destructive diseases in human history, having killed over 25 million people in less than 30 years. Globally, an estimated 33 million people were living with the virus at the end of 2008. This might have risen sporadically. The greatest burden of the virus is experienced in developing nations, specifically in sub-Saharan Africa (SSA), which is home to more than two-thirds of the infected persons worldwide. As HIV treatment becomes more accessible and patient life expectancy increases, the number of patients requiring long-term management in care and treatment programs increases meaningfully (Hoskins 2014).

In 2016, an estimated 36.7 million people were living with HIV, and 1.8 million new HIV infections were documented. Women aged 15–24 years are particularly at risk of HIV infection, and they accounted for 26% of new HIV infections among adults globally. A closer look at regional data reveals that the vast majority of people living with HIV are located in low- and middle-income countries. In

the Sub-Saharan Africa (SSA) region, where 71% of new infections occur, West African women aged between 15 and 24 years accounted for 22% of new HIV infections (Awofala and Ogundele 2018, Yaya et al. 2019).

The eastern and southern parts of Africa have the highest number of cases of HIV in the world. These parts of Africa are home to the majority of the recorded cases of the HIV virus. Even when the HIV virus is generalized, young women, homosexuals, transgender people, sex workers, and people who inject drugs into their bodies are most prone to contracting the HIV virus. Statistics drawn from avert.org show that East and Southern Africa are the worst hit by the HIV virus. These parts of Africa are only home to 6.2% of the world's population, but they sheltered 54% of the > 20 million infected people in the world in 2018. In research conducted in 2018, South Africa had more than one-quarter (240, 000) of Southern Africa's infections. Moreover, Mozambique, Tanzania, Uganda, Zambia, Kenya, Malawi, and Zimbabwe accounted for more than 50% (445, 000) of the new infections in 2018.

Presently, around 38 million people are currently living with HIV, and tens of millions of people have died of AIDS-related causes since the beginning of the epidemic. Globally, around 76 million people have become infected with HIV since its inception. Overall, African continent accounts for 67.5% (25,720,000) of the total population of infected persons globally. Asia has 17%, Europe 8%, North America 3% and South America 6% (Avert, 2019; Kaiser Family Foundation, 2021). Meanwhile, more than 2 million people are infected in the European nations, particularly in the region's eastern part. Nearly 137,000 people were diagnosed with HIV in European countries in 2019 alone (European Centre for Disease Prevention and Control, 2020).

HIV is the virus that orchestrated AIDS, one of the universe's most deadly public health challenges. But there is a global commitment to stopping the spread of the virus and ensuring that everyone with it has access to HIV treatment. There

were approximately 37.6 million people across the world with HIV in 2020. Of these, 35.9 million were adults, and 1.7 million were children. In 2020, 1.5 million individuals worldwide acquired the virus, marking a 30% decline in new HIV infections since 2010 (Global Health, 2020).

In the absence of treatment, life expectancy with HIV is severely reduced. Untreated, it is estimated that the median survival time after HIV infection for adults in developing nations is 11 years. For untreated infants in developing countries, disease progression is rapid, the risk of dying in the first two to six months of life is essentially high, and one-third of HIV-infected infants are estimated to die before their first birthday, with 30%–50% dying before the age of two. Vertical, horizontal and community-acquired transmission are some of the ways in which children can be infected with the virus. HIV-related immune suppression increases the opportunity for microbes and pathogens in the environment to find a host, resulting in so-called 'opportunistic infections' (OIs) and malignancies developing in HIV-positive individuals (Hoskins 2014).

The virus and its affiliated agent continue to be a major public health issue with a rise in campaigns to improve public general knowledge of the virus and its transmission. Despite interventions and breakthroughs in our scientific understanding of HIV and its prevention, many people continue to be infected by the virus (Awofala and Ogundele 2018,Yaya et al. 2019)

1.3.1 Transmission Routes

HIV infections have been mainly restricted to intravenous drug users, the core transmission route of the virus being sharing contaminated needles. Most of the adults who became infected were men who were injecting drugs. However, the types of people being infected by the virus and the main transmission routes are quickly changing. Young people account for most of the new infections. The proportion of women (who are less likely to be intravenous drug users) infected with

the virus suggests that the number of virus infections spread by sexual contact is increasing. The increased infection rates of HIV among new units are fuelled by the growth of drug injections, increased sexual activity among young people, and the growing number of public sex workers. The virus is likely to reach a wider population from these subgroups.

In its report, UNICEF warns that the HIV virus is the greatest threat to human health, as it spreads virtually unchecked into the midst of initially uninfected countries. According to the Joint United Nations Program on HIV and AIDS (UNAIDS), despite the significant progress in the prevention of HIV, about 36.9 million people are still living with the virus. A significant portion of these people are unaware of their infection status, even when it develops into advanced HIV disease (AHD), due to prolonged asymptomatic phases after HIV infection. In some countries, the infected persons are being stigmatized in their various communities. Such stigmatization prevents them from seeking medical advice, which results in a higher risk of developing (Chen et al. 2019). Approximately 84% of people with the virus globally knew their status in 2020. The remaining 16% (around 6.0 million people) still need access to HIV testing services, as this will inform them about whether they have been infected or not. Testing is essential to HIV prevention, treatment, care and support services.

According to (Yaya et al. 2019), inadequate knowledge of HIV and its transmission has been identified as a major factor contributing to the spread of the epidemic among Nigerian youths. When the academic levels of the respondents in their study were compared with their knowledge of the possible routes of transmission of HIV, it was discovered that the higher the level of their academic qualifications, the higher the percentage that agreed that the use of non-sterile dental instruments is a possible means of transmitting HIV. This was a statistically significant result ($p < .002$) (Opeodu and Ogunrinde 2015).

Specifically, the most common transmission routes of HIV are the following:

1.3.2 Mother to Child.

In 2020, 84% of pregnant women with HIV received ART to prevent the transmission of HIV to their babies during pregnancy and childbirth and to protect their health (GlobalHealth (2020)). This is the mother-child route. This can be further delineated into three periods: during pregnancy, delivery and breastfeeding. Vertical transmission is the predominant route for the acquisition of HIV infection by children, either in utero, intrapartum or postnatally through breastfeeding. Less frequently, children may acquire HIV by horizontal transmission. Community-acquired HIV transmission to children may occur following surrogate breastfeeding, pre-mastication of food, and sexual abuse. In most instances of horizontal HIV acquisition in children, the exact transmission route is difficult to determine due to the time elapsed between the HIV-exposure event/s and confirmation of HIV in the child (Myburgh et al. 2020).

1.3.3 Sexual Intercourse without a Condom.

Some researchers have identified the organism that causes AIDS and named it variously, but the International Committee on the Taxonomy of Viruses officially named it the Human Immunodeficiency Virus (HIV) in 1986. HIV is found in the following bodily fluids of infected persons; blood, vaginal fluids, rectal fluids, breast milk, semen and pre-seminal fluid (Ndibuagu et al. 2017). Correct and consistent use of latex condoms during sexual intercourse (vaginal, anal, and oral) can greatly reduce the chances of acquiring or transmitting HIV and other sexually transmitted infections (STIs). Natural-membrane condoms, often made from sheep gut, are not recommended because they have tiny pores through which HIV can pass.

The probability that a person has acquired an STI is generally proportional to the number of sexual partners that person has had in recent years. However, in areas where the prevalence of HIV is high, people may become infected who

have had only one partner. Sexual intercourse refers to the penetration of the penis into an orifice: vagina, rectum, or mouth. Sexual behaviour is any act of sexual gratification between two or more individuals or by oneself. Sexual intercourse is a risk behaviour for acquiring HIV and other STIs, but not all sexual behaviours promote risk (Ferris et al. 2010) Shared Intravenous Fluids/Equipment: Rarely children born to women uninfected by HIV acquire HIV by horizontal transmission. This may occur through healthcare-associated transmission by infusion of HIV-contaminated blood or blood products, the re-use of contaminated needles/syringes or other medical equipment, and the ingestion of HIV in expressed breast milk in neonatal units (Myburgh et al. 2020).

AIDS was first noticed among women who had sex with infected men in 1983, suggesting that the disease could also be transmitted through the heterosexual route. Heterosexual intercourse with an infected person is now the main route of HIV transmission, accounting for about 80% of cases (Ndibuagu et al. 2017).

1.3.4 Blood Transfusion.

Previous studies have reported the presence of some misconceptions concerning the possible transmission of HIV among different study populations. Bassey et al. reported that about 15% of the antenatal women studied believed that a mosquito bite could transmit HIV/AIDS and 13.7% stated that HIV/AIDS could be transmitted by sharing a meal with an infected person. There was the misconception that once somebody was infected with HIV, he or she already had AIDS. Another study among army personnel reported that 9.1% of the participants believed that HIV could be contracted through a mosquito bite, and 2.1% stated that it could be contracted through body contact such as huggings (Opeodu and Ogunrinde 2015). Others were shared intravenous material, men-men sexual relationships, and drug paraphernalia Gilroy (2020).

It is essential to be familiar with the correlation that exists between AHD and HIV transmission routes in other to prioritize prevention techniques. As part of efforts to prevent the pandemic, it has been suggested that the criminalization of commercial sex workers, compulsory drug treatment and the prohibition of homosexuality should be adopted.

1.3.5 Prevalence of HIV/AIDS Virus Among People Who Inject Drugs (PWIDs)

It is estimated that 15.6 million people inject drugs globally, and 30% of this population are women. A national consensus size estimate from 2014 indicated that the total number of people who injected drugs in the Eastern African Nation (Tanzania) was 30,000. Generally, it is opined that injecting drugs poses health challenges (Likindikoki et al. 2020). This is the primary area of this study

PWIDs are at increased risk of acquiring and transmitting HIV and Hepatitis C (HCV) as they share injection paraphernalia and have unprotected sex. In the recruited sample of mostly current injectors with a long duration of injecting drugs, seroprevalence for HIV and HCV varied greatly between the city samples. HCV was endemic among the participants in all the city samples. The authors' results demonstrate the necessity of intensifying prevention approaches for blood-borne infections among PWIDs in Germany. To tackle the risk of blood-borne and sexually transmitted infections among PWIDs, it is essential to combine behavioural, socio-demographic and serological data to inform the planning and implementation of effective prevention and intervention techniques (Wenz et al. 2016).

Lack of correct information about possible modes of HIV transmission may hinder people's willingness to receive voluntary counselling and testing. It also increases the likelihood of the stigmatization and isolation of people living with HIV, among other adverse psychosocial influences (Opeodu and Ogunrinde 2015). This has led to the widespread of the virus among drug injectors.

In 1981, there were 100,000 injectors infected with HIV. In recent years, a reduction in newly diagnosed cases of HIV among PWIDs has been observed. In 2010, 110,000 new cases were reported; in 2013, only 98,000 were reported. It is estimated that the injection of drugs exists in 148 nations, and HIV infection exists among PWIDs in 61 countries. There are wide ranges in the estimates of PWIDs and the number of PWIDs who are HIV-infected globally. More recent reports estimate 8.9-22.4 million PWIDs in nations of the world, and approximately 0.9 to 4.8 million PWIDs are HIV positive. The same factors that have led to the globalization of trade in illicit goods (improved communications, improved transportation, reduced restrictions on the flow of capital) have led to the worldwide diffusion of drug injecting, with HIV infection frequently following drug distribution routes (Des Jarlais et al. 2016). HIV is thus prevalent among women, men, boys and girls who inject drugs across the globe – it has been recorded in 91% of independent nations. The Mashriq (eastern) part of the region is more affected by this public health challenge than the Mghrib (western) part (Mumtaz et al. 2014). The high levels of injecting drugs in the Mashriq (eastern) part appear to be related to the increased availability and purity of heroin at lower prices. This is not surprising since 83% of the global supply of heroin is produced in Afghanistan (Mumtaz et al. 2021). Among PWIDs, the prevalence of HIV is 5–15%, and at least half of all countries have such epidemics among men who have sex with men (HIV prevalence 3–10%). Some of these epidemics have the potential for further growth, a potential that is facilitated by the high levels of injected drug use and risky sexual behaviours and by the overlap of high-risk behaviour between PWIDs, men who have sex with men, and female sex workers. This high-risk environment is exacerbated by overall high levels of stigma towards key populations, which increases the vulnerability for further spread. With the exception of two countries that experienced large epidemics among commercial heterosexual sex networks involving female sex workers and their clients, HIV prevalence

among female sex workers continues to be overall well below 5%, and even at vanishing levels, in most countries. Yet, the contribution of commercial heterosexual sex networks to HIV transmission in this region remains sizeable due to the relatively large size of these networks, compared with PWIDs and networks of men who have sex with men (Mumtaz et al. 2021).

The prevalence of drug injection is up to 0.46% in Iran and 0.5% in Pakistan, while it is reported to be 0.14% in Lebanon, 0.10% in Morocco, and 0.07% in Syria (Mumtaz et al. 2014). However, overall regional prevalence is comparable with global figures, ranging from 0.09% in South Asia to 1.30% in Eastern Europe (Degenhardt et al. 2017).

In Tanzania, the prevalence of HIV infection among PWIDs has been filed as more than the entire population of drug users. In 2020, it was reported that there had been a decline in the prevalence of HIV infections among PWID: 8.7% compared to a previous empirical study where it was 15.5%. Despite this, HIV prevalence is still high among PWIDs (Likindikoki et al. 2020).

1.4 Review of Mathematical Models

1.4.1 Epidemic Models

In human history, communicable diseases have played an integral part. It is well known that epidemics have infected populations since the dawn of recorded history, resulting in many deaths before disappearing, possibly recurring. As populations develop immunity, the severity may decrease. For example, the 1918-19 "Spanish" flu pandemic killed over 50,000,000 people globally, and annual influenza seasonal epidemics kill up to 35,000 people worldwide.

Between 1346 and 1350, the Black Death (possibly bubonic plague) moved over Europe in multiple waves, originating in Asia. It is believed that one-third of Europe's population died. For more than 300 years, the disease recurred in

various regions of Europe, most memorably as the Great Plague of London in 1665-1666. It then gradually pulled out of Europe.

Other illnesses have become endemic (constantly prevalent) in some communities and are responsible for a large number of fatalities. This is especially prevalent in impoverished nations with underdeveloped healthcare systems. Every year, millions of people die from illnesses like measles, lung infections, diarrhoea, and other ailments that are easily treatable and not considered deadly in the West. Malaria, typhus, cholera, schistosomiasis, and sleeping sickness are all prevalent in various places of the globe. The economic consequences of high illness mortality on mean life duration, disease debilitation, and mortality in affected nations are significant. According to the World Health Organization, there were 1,400,000 tuberculosis deaths in 2011 and 1,200,000 HIV/AIDS death in. The purpose of epidemiologists is first to understand the origins of a disease, then anticipate its development, and ultimately devise methods for controlling it, which includes comparing alternative techniques.

In order to predict the behaviour of diseases and help control particular epidemics, mathematical models have been constructed. Compartmental models of epidemics, such as the SIS and SIR models, can be used to understand how an epidemic spreads by assigning each person to a subgroup representing a specific disease stage (Kermack and McKendrick 1927) developed the Susceptible-Infected-Removed SIR model in 1927. The Susceptible-Infected-SusceptibleSIS epidemic model is another sort of epidemic model that provides a scenario different from the SIR model. In the following, we'll describe the three types of epidemic models used throughout this thesis. Let S(t) stand for the number of susceptible at time t, I(t) for the number of infected people, and R(t) for the number of people who have recovered from their infection at time t.

1.4.1.1 SIS Epidemic Model

Some infections do not produce immunity. Such infections do not recover, and individuals become susceptible again following infection as in many infectious diseases transmitted by bacterial agents (e.g., tuberculosis) or sexually transmitted diseases (e.g., gonorrhoea) can be researched using SIS epidemiology.

The Susceptible–Infective Susceptible SIS type can be used to model this type of disease. The overall population is divided into two distinct divisions based on epidemiological state; people are classed as either susceptible or infected. The sizes of these groupings are denoted by $S(t)$ and $I(t)$, respectively. Because one usual path goes through susceptible, then infected, and then back to susceptible, the SIS model is used. The differential equations that describe the transmission of the disease are as follows:

$$\frac{dS}{dt} = \mu N - \beta S((t) + \gamma I(t) - \mu S((t),$$
$$\frac{dI}{dt} = \beta S(t) I(t) - (\mu + \gamma) I(t),$$

given proper initial values $S(t) = S(0)$ and $I(t) = I(0)$ with $S(0) + I(0) = N$.

In these equations μ represents the death rate per capita for a single individual.

γ represents the per capita recovery rate of an individual. Consequently, assuming the infectious time follows an exponential distribution, the mean infectious period is $1/\gamma$. The transmission rate, denoted by β, is the frequency with which an infected person comes into touch with and infects a susceptible person. Therefore, $\beta = \lambda/N$, where λ is a single infected person's per capita disease contact rate.

The fundamental reproduction number R_0 is a central topic in mathematical epidemiology. This is the expected number of secondary cases created by a single newly infected individual entering a disease-free community at equilibrium It is defined as the expected number of secondary cases produced by a single

newly infected individual (Brauer et al. 2008). The findings show that

$$R_0 = \frac{\beta N}{\mu + \gamma}.$$

A single newly infected person will die at a rate of μ, become susceptible at a rate of γ, and hence remain in this state for a period of time equal to $1/(\mu + \gamma)$ if they enter a disease-free population. During this period, he or she interacts with the susceptible people who are there at rates of β each, and if N is large, there are roughly N of them. Therefore, the average number of infections created throughout the infectious time is $\beta N/(\mu + \gamma)$, which equals R_0 as previously mentioned.

1.4.1.2 SIR Epidemic Model

SIR model was initially proposed by(Kermack and McKendrick 1927) and is considered one of the most prominent mathematical models of epidemics. It also holds a significant amount of historical significance. A SIR model is comparable to a SIS model, with the exception that in a SIR model, once an individual has completed their infectious phase, they are placed in the permanently removed class.

The primary premise of such a model is that the population in which a pathogenic agent is active is divided into three distinct subgroups. These subgroups are as follows: the healthy individuals who are susceptible(*S*) to infection; the already infected individuals (*I*) who are able to spread the disease to the healthy individuals; and the individuals removed (*R*) who are no longer part of the infection cycle, either through immunisation and recovery or through natural attrition. The model is appropriate for describing a well-localized epidemic outburst since it only looks at the temporal dynamics of the infection cycle. Kermack and McKendrick initially

proposed the SIR model as a differential system.

$$\begin{aligned}\frac{dS}{dt} &= -\beta I(t)S(t),\\ \frac{dI}{dt} &= \beta I(t)S(t) - \gamma I(t),\\ \frac{dR}{dt} &= \gamma I(t).\end{aligned}$$

Given that the right-hand sides of these equations add up to zero, the sum of S, I, and R is a constant that is equal to the total number of individuals in a population $S + I + R = N$. β is the rate at which a disease spreads, and γ is the rate at which a person with an infection is cured and transfers to the recovery group. For the SIR model, the most critical parameter is the ratio by $\beta N/\gamma$, also known as the Basic Reproduction Number R_0.

1.4.2 Awareness Programs in Epidemic Models

The modelling, investigation, and data analysis of infectious disease propagation are extremely valuable in assessing measures for controlling such illnesses in communities. Classic models of infectious disease transmission depend primarily on interactions between susceptibles and infectives. Other factors, such as media attention, immunisation, population movement etc., have an impact on the spread of the disease

The media significantly impacts people's attitudes regarding illnesses and government health-care actions aimed at preventing disease transmission. It is a media-driven public awareness campaign that educates people about the disease and encourages them to adopt measures such as social isolation and wearing protective masks.

Greenhalgh et al. (2015) presents a review of a study on the effects of media awareness campaigns on infectious disease epidemics in their article. These studies are divided into two categories. The influence of media coverage on the spread and control of infectious illness is investigated using mathematical models

in the first class. In the next section, we would like to introduce some of the epidemic models with the awareness program that we work with in this thesis.

1.4.2.1 SEI Model with Media Impact

Cui et al. (2008) created and tested an SEI model that took into account the media effect on the transmission of an infectious illness in a specific area. They came to the conclusion that if the basic reproduction number is more than one and the media impact is significant, the model would display various endemic equilibria, posing a danger to disease control.

Consider the transmission of certain infectious diseases (such as SARS) in a given region/area. We classify the population into the following categories:

- S(t) , the number of susceptible individuals;
- E(t), the number of individuals exposed to the infected but not infectious;
- I(t) , the infected who are infectious.

We assume that infectious individuals receive medical treatment in hospital settings as soon as they are identified from the category of exposed. Once they are recovered, they no longer impose risk on the susceptible individuals. In most of the studies, the compartmental models were built by either assuming the total population to be a constant or satisfy exponential growth (Brauer et al. (2012), Busenberg and Cooke (1993), Diekmann and Heesterbeek (2000) and Hethcote (2000)). It is more reasonable to assume that the population of a given region obey the Logistic growth. Then we have the model

$$\begin{aligned}
\frac{dS}{dt} &= bS\Big(1 - \frac{S}{K}\Big) - \beta e^{-mI} SI, \\
\frac{dE}{dt} &= \beta e^{-mI} SI - (c + d)E, \\
\frac{dI}{dt} &= cE - \gamma I,
\end{aligned}$$

where all the parameters are positive, and

• *b*, the intrinsic growth rate of the human population, *k* is the carrying capacity for the human population of a given region/area.

• βe^{-mI} is the contact and transmission term (β) together with the disease awareness function e^{-mI}. It measures the spread of the virus from the infected individuals to the susceptible individuals. If $m = 0$ then the transmission rate is constant. In Cui et al.'s paper, this was μe^{-mI} but we have changed the μ to β so as to unify the interpretation of parameters in this section.

• *c* is the rate per unit time (day) that infected exposed become infectious.

• *d* is the natural death rate for the exposed population .

• γ is the removal rate from the infected compartment, which includes the recovery rate of the hospitalized infectious individuals and natural death. Hence we have $\gamma > d$.

1.4.2.2 Model Emphasizing the Psychological Impact

Liu et al. (2007b) constructed an EIH compartmental model to investigate the role of the media and its psychological impact on multiple disease outbreaks. Their model analysis reveals that this impact leads to differences in the transmission pattern here, we simply assume that this impact is described by an exponential decreasing factor, resulting in the transmission coefficient as $\beta_0 = \beta e^{-a_1 E - a_2 I - a_3 H}$. Here β is the basic transmission rate if the impact of the reported numbers of exposed, infectious and hospitalized were ignored, and a_1, a_2, a_3 are non-negative parameters to measure the effect of the psychological impact of media reported numbers of exposed, infectious and hospitalized individuals. The modified model then becomes

$$\begin{aligned} \frac{dE}{dt} &= \beta e^{-a_1 E - a_2 I - a_3 H} IS - cE \\ \frac{dI}{dt} &= cE - dI - hI \\ \frac{dH}{dt} &= hI - \gamma H \end{aligned} \tag{1.1}$$

where $E = E(t)$ is the number of individuals who are exposed to the infected but not yet infectious, $I = I(t)$ is the number of infectious individuals, and $H = H(t)$ is the number of infectious individuals who are receiving medical treatment in hospital settings.

We also assume that the hospitalized individuals no longer impose risk on the susceptible individuals. In model (1.1), the parameters involved, which are positive, are

β: We assume that the exposed population is increased following infection via contact between a susceptible and an infectious individual with a transmission coefficient β. This parameter measures the effect of both the infectiousness of the disease and the transmission rates;

S: as mentioned above, we assume that the total number of susceptible individuals remains unchanged, and thus S will be regarded as a parameter;

c: the transmission rate per unit of time (day, in case of SARS) that exposed individuals become infectious;

d: the disease-induced death rate of infectious individuals before entering the health care settings;

h: the rate at which infectious individuals enter the health care settings seeking treatment;

γ: the combined per capita disease recovery rate and death rate of hospitalised individuals. In the paper of Liu et al. (2007b) this was represented as two separate terms but we have chosen to represent them as a combined term to achieve a unified parameter notation between this model and the previous one.

In general, the first available information is the reported number of hospitalized patients when the infectious disease is at the emerging stage. Hence we will focus more on the impact of the number of reported hospitalized cases.

1.4.3 Modelling Disease Awareness Programs in HIV/AIDS Models

So we have established that the spread of HIV and AIDS amongst PWIDs is an important problem that needs attention. However, recently, there has also been increased interest in the effect of disease awareness programs. Infected individuals may adjust their behaviour to reduce potential contacts in the presence of high levels of disease. There are two ways to model this. The first and simplest way, and the one which we shall adopt in this thesis, is to reduce the disease transmission term by a factor ϕ $(0 \leq \phi < 1)$ to take account of the behavioural modifications individuals make because of their knowledge of current disease levels. In the second, the amount of media awareness is modelled as a separate variable, and the individuals (usually the susceptible class) are split into aware and unaware individuals (Misra et al. (2011), Greenhalgh et al. (2015)).

1.4.3.1 Disease Awareness Programs Using a Multiplicative Factor

We shall first look at disease awareness models that reduce the disease awareness function by a factor $\phi(I)$ between 0 and 1. Xiao and Ruan (2007) study a SIR model where the disease transmission function in the absence of an awareness program is βSI, where S is the number of uninfected individuals, I is the infected individuals, and β is a constant. This is multiplied by a disease-awareness function

$$\phi(I) = \frac{1}{1 + \alpha I^2}.$$

Here α is a positive constant. We see clearly that $\phi(I)$ is a strictly positive monotone decreasing function between zero and one. This is illustrated in Figure 1.1 with $\alpha = 0.00002$.

Li et al. (2008b) look at an SIS epidemic model with constant and impulsive vaccination and where there is media awareness and the disease transmission

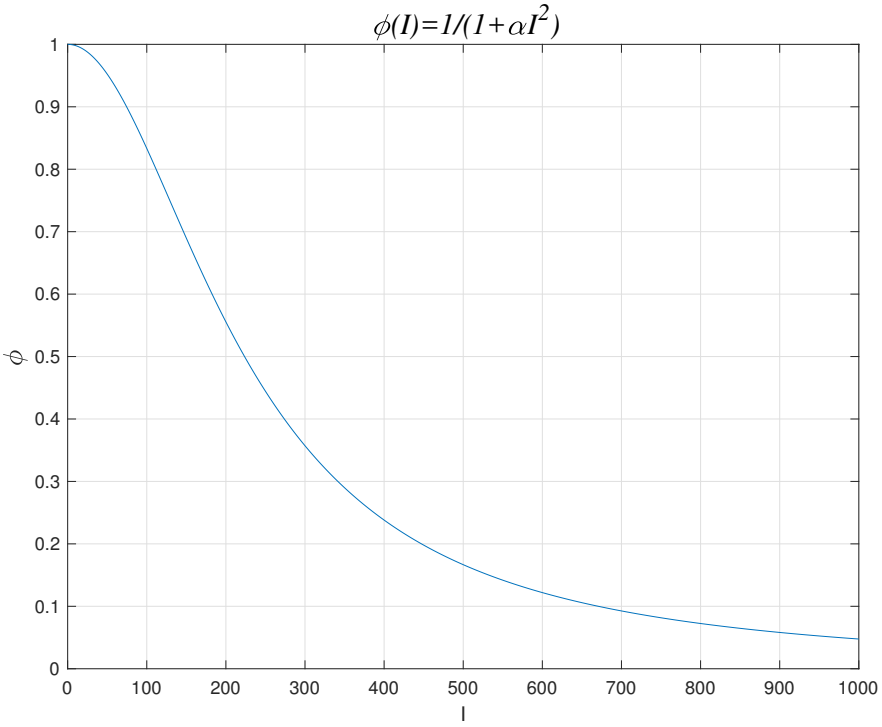


Figure 1.1: Awareness Program function $\phi(I) = \frac{1}{1+\alpha I^2}$.

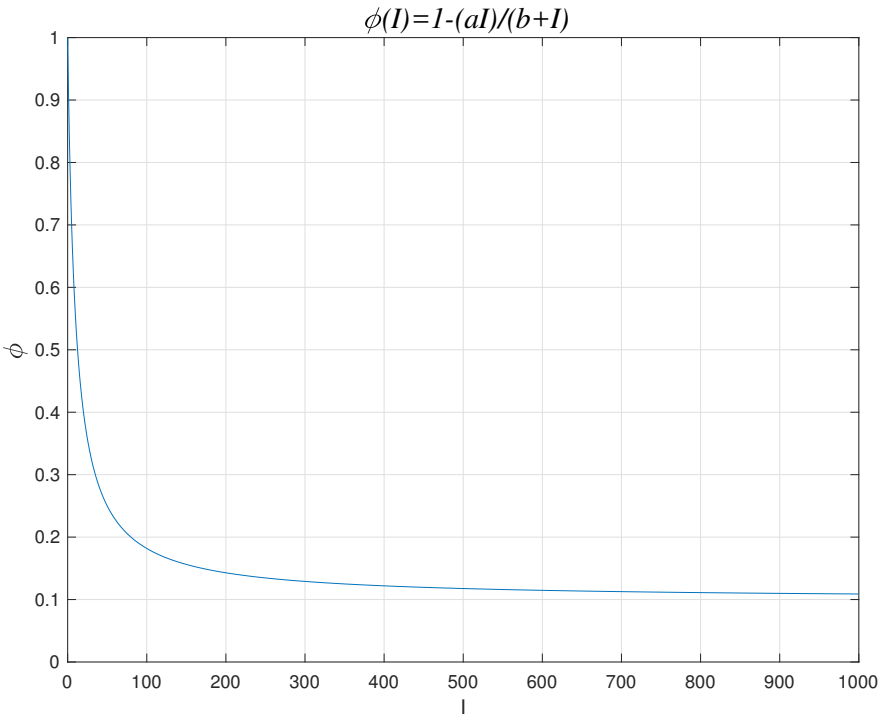


Figure 1.2: Awareness Program function $\phi(I) = 1 - \frac{aI}{b+I}$.

term, again fundamentally βSI, is reduced by a factor

$$1 - \frac{aI}{b+I}.$$

Here a and b are positive constants and $a \leq 1$. Again, $\phi(I)$ is a monotone decreasing function of I. This approach and the same form of media awareness function is also used by Tuenche et al. (2011) in a mathematical model of the spread of influenza. Liu (2013) investigates the spread of disease in a SIRS model using a similar awareness function, although stochasticity is introduced. Salman (2021) uses the same disease awareness function in his model. The key thing to note for our purposes is that the disease transmission term is reduced by the same disease awareness program factor as in Li et al. (2008b) because of awareness of infected individuals. The same function will be used to model the reduction in disease transmission in some of our numerical examples. This is illustrated in Figure 1.2 with a $a = 0.9$ and $b = 10$.

Cui, Sun and Zhao(Cui et al. (2008)) consider an SEI model where the disease transmission function (again effectively βSI with no disease awareness) is reduced by a multiplicative factor

$$\phi(I) = e^{-mI}$$

due to the effect of disease awareness. Again we will use a similar function in our numerical examples. A graph of this function is illustrated in Figure 1.3 with $m = 0.005$. Liu et al. (2007a) consider a model for an EIH (exposed-infectious-hospitalized) epidemic. The fundamental disease transmission term is again βSI, which is reduced by a factor $e^{-a_1 E - a_2 I - a_3 H}$, where a_1, a_2 and a_3 are constants and E and H are the number of exposed and hospitalized people. Strictly speaking, this is not the same type of disease awareness function we have been discussing as it depends on both the exposed and hospitalised individuals in addition to infections, but it is based on the same idea.

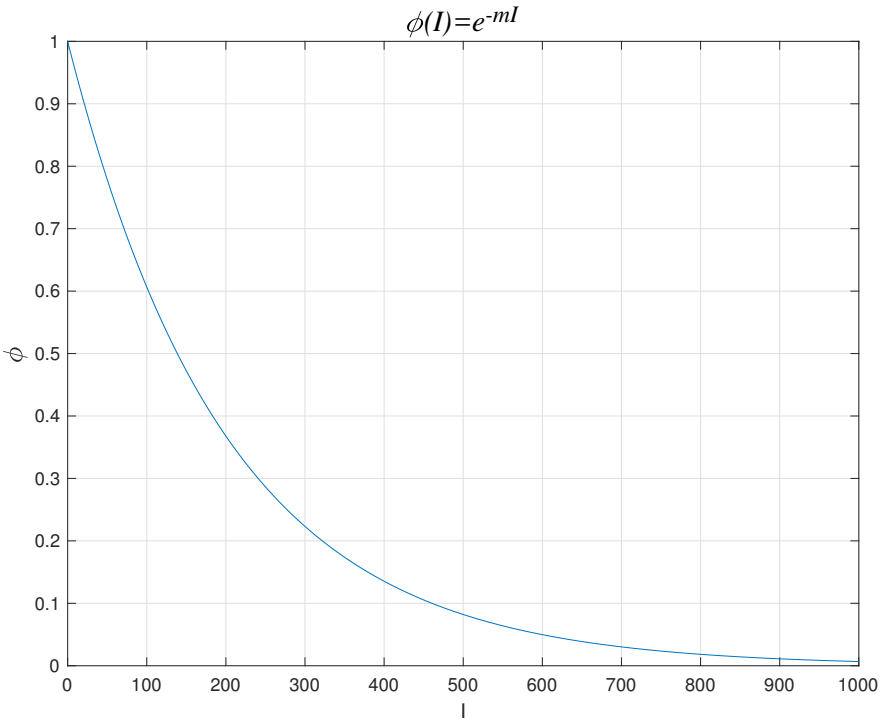


Figure 1.3: Awareness Program function $\phi(I) = e^{-mI}$.

In a different paper Cui, Tao and Xu (Cui et al. (2008)) look at an SIS model in which the basic disease transmission function with no awareness is

$$\frac{\beta SI}{S+I}.$$

With behavioural modification due to knowledge of disease levels, this is decreased by a multiplicative term $\phi(I) = 1 - kf(I)$ where $k < 1$. Here $f(I)$ is a positive monotone increasing function with $f(0) = 0$ and $\lim_{I\to\infty} f(I) = 1$. This disease awareness program function is a generalisation of the one used by Li et al. (2008b). Sun et al. (2011) study the effect of media-induced social distancing on how disease spreads in a setting with two patches using a similar modification of the disease awareness function.

We shall compare four types of multiplicative awareness functions, first an exponentially decreasing factor,

$$\phi_1(I) = e^{-mI},$$

as used by Cui, Sun and Zhao(Cui et al. (2008)), Secondly

$$\phi_2(I) = 1 - \frac{aI}{b+I},$$

as discussed by Li et al. (2008a).

$$\phi_3(I) = \frac{1}{1+\alpha I^2},$$

as discussed by Xiao and Ruan (2007), and

$$\phi_4(I) = 1 - kf(I),$$

as discussed by Cui, Tao and Xu (Cui et al. (2008)).

For all of these awareness functions $\phi(I)$, $1-\phi(I)$ represents the proportion by which susceptible individuals reduce their potentially infectious contacts when there are I infected individuals in the population. All of these functions start off at 1 when $I = 0$ (the baseline level) and decrease as I increases and the susceptibles make less contacts. We can see that if I is very large ϕ_1 and ϕ_3 both tend to zero, ϕ_2 tends to $1-a$, and ϕ_4 tends to $1-k$. So if there are a large number of infectious individuals, with ϕ_1 and ϕ_3 the susceptibles completely cut off their potentially infectious contacts, whereas with ϕ_2 and ϕ_4, even in the presence of a large number of infectious individuals, the susceptibles still make a basic level of infectious contacts. Moreover as for I very large,

$$e^{-mI} < \frac{1}{(1+\alpha I^2)}$$

. So if I is very large the relative ordering of these functions is

$$\phi_1 < \phi_3 < min(\phi_2, \phi_4),$$

and the relative sizes of ϕ_2 and ϕ_4 are determined by the relative sizes of a and k.

We can also look at the behaviour of the four functions ϕ_1, ϕ_2, ϕ_3 and ϕ_4 near $I = 0$, with a small number of infectious individuals, by looking at $\phi'(0)$.

$$\phi_1'(0) = -m, \quad \phi_2'(0) = -\frac{a}{b}, \quad \phi_3'(0) = 0 \quad \text{and} \quad \phi_4'(0) = -kf'(0).$$

So ϕ_3 is initially a very flat function and ϕ_1, ϕ_2 and ϕ_4 all initially decrease faster with the initial rates of decrease determined by the relative sizes of $m, \frac{a}{b}$ and

$kf'(0)$.

1.4.3.2 Disease Awareness Programs Modelling Unaware and Aware Individuals

Now we turn to models which use the other approach, that is they divide the population into aware and unaware individuals and model the amount of disease awareness as a separate variable in some way. These models are necessarily more complex as they have more classes but nowadays are used more often to model disease awareness programs. Many models use this type of disease awareness function, and we can give only a small selection here.

Misra et al. (2011) consider a simple SIS model with aware and unaware individuals. X denotes the unaware susceptible classes, X_m the aware susceptibles, Y the number of infected individuals, and M the cumulative density of media programs. Unaware uninfected individuals catch the disease at rate βXY and aware susceptibles at rate λXM, where β and λ are constants. The cumulative density of media awareness is modelled as

$$\frac{dM}{dt} = \mu Y - \mu_0 M,$$

where μ and μ_0 are constants. At the end of their infectious period infected individuals return to the aware susceptible class.

Samanta et al. (2013) consider a more complex SIS model. It is built on the model of Misra et al. (2011) but allows unaware susceptibles to become infected and also, at the end of their infectious period, infected individuals may become either unaware susceptibles a fraction $1 - p$ of the time or aware susceptibles, a fraction p of the time. Moreover, individuals can move out of the aware uninfected group to the aware infected group. Greenhalgh et al. (2015) further build on the model of Samanta et al. (2013). Instead of the unaware susceptibles X_- becoming aware at rate $\lambda X_- M$, they become aware at rate

$$\frac{\lambda X\_M}{k+M},$$

where k is a constant. Similarly aware susceptibles $X_+(t)$ become infected at rate

$$\frac{\beta}{1+\beta_1 M} X_+ Y,$$

where β_1 is a constant.

Disease awareness programs can have applications in other areas too. For example, Ma et al. (2015) modelled alcoholism using a mathematical model with a time delay and awareness, using two types of individuals, aware and unaware and modelling the media awareness as a separate variable. Lastly, the advent of COVID-19 has focused our attention on how people modify their behaviour when there is a threat from infectious disease Musa et al. (2021) suggest an epidemic model using disease awareness programs which split the population into aware and unaware individuals for COVID-19 transmission in Nigeria. They fit the model to Nigerian COVID-19 data and assess the impact of disease awareness programs on disease transmission. They explain the effect of awareness programs with regard to the basic reproduction number.

Most modern papers tend to use the more sophisticated approach of dividing the population into aware and unaware individuals. However, the more straightforward approach is historically significant and is still a crude way to model media awareness.

1.5 Mathematical Modelling of HIV/AIDS Amongst PWIDs.

Mathematical models have been effectively employed to analyse and predict the dynamic behaviour of biological systems in recent years. Kaplan and O'Keefe (1993) devised the first mathematical model for the transmission of HIV and AIDS among PWIDs in shooting galleries, where a shooting gallery is a venue where PWIDs buy and inject narcotics. In order to better understand how HIV is spread within this sort of group, Kaplan integrated numerous aspects into his model, such as the rate of injection equipment sharing and the effect of cleaning injection equipment.

1.5.1 The Needle Sharing Model

One of the earliest mathematical models explicitly created to explain how sharing needles and syringes in shooting galleries might transmit HIV and AIDS among IDUs was given by Kaplan (1989). This research offered helpful insights into how HIV spreads in shooting ranges and made suggestions for the types of data that would help researchers better understand how HIV spreads (like rates of sharing needles and syringes, the likelihood that needles and syringes are cleaned, and the average length of risky behaviour involving sharing needles and syringes). The author adopted the following assumptions in order to simulate the percentage of the populace who was HIV-positive at time t, represented in the model by the symbol (t):

1. Injecting drug usage exclusively takes place in the m shooting galleries (places where PWIDs hire the same needles and syringes). A user of illicit drugs injects once each time they attend a shooting range.

2. Independent of what the other PWIDs do, each PWID randomly chooses to attend shooting galleries at rate λ. This assumption suggests that, for all PWIDs,

is the per capita needle and syringe sharing rate λ since PWIDs only inject once every shooting gallery visit.

3. After being used by an infected PWID, all injecting equipment will become infectious. Additionally, a needle and syringe used by a PWID who is not contagious may be flushed (with probability θ), which will make it contagious-free. (That is, throughout the injection operation, the infected contents of the needle and syringe are entirely re-located). According to this presumption, although uninfected PWIDs who use infectious needles and syringes run the risk of contracting HIV, they may also reduce the risk for the PWIDs who use the needle and syringe the following time.

4. The likelihood of HIV transmission through the use of shared needles and syringes is per injection α. Additionally, the only way for PWIDs to get HIV is via sharing needles and syringes. This presumption suggests that HIV infection is always contagious and that other known transmission mechanisms, such as sexual contact, have no bearing on the prevalence of the illness in this community.

5. The size of the PWID population is n, where n is a significant and stable number. As a result, any PWIDs who depart the population (for example, owing to death, admission to treatment programmes, or jail) are quickly replaced by PWIDs who are vulnerable. The per capita rate at which IDUs depart or enter the population is represented by μ. Kaplan constructed the following differential equations that control the spread of the illness by taking into account the population's PWID prevalence at time t+t and the quantity of contaminated needles at time $t + \Delta$, where Δt is a brief interval of time.

$$\frac{d\pi(t)}{dt} = [1 - \pi(t)]\lambda\beta(t)\alpha - \pi(t)\mu,$$
$$\frac{d\beta(t)}{dt} = \lambda\gamma\pi(t) - \lambda\gamma\beta(t)[1 - [1 - \pi(t)](1 - \theta)].$$

The percentage of contaminated needles and syringes at time t is indicated by the symbol $\beta(t)$, while the gallery ratio, or the number of PWIDs per shooting gallery, is shown by the symbol γ. Along with these equations, the author also

deduced an expression for the fundamental reproductive number, R_0, which is represented by the ratio $\lambda\alpha/\mu\theta$, and demonstrated that this expression must be greater than one in order for an endemic equilibrium solution to be possible.

Kaplan looked at the impact that various gallery ratios have on the transmission of HIV in this group in his first set of numerical simulation findings. The findings demonstrated that whereas low values of γ lead to a significantly slower initial disease spread, big values of γ cause the transmission of HIV among this PWIDs group to achieve equilibrium extremely fast.

The model is then modified one more to support the cleaning of syringes and needles. The author assumes that every PWID, whether or not they are infected, cleans their needle and syringe after use with a probability of ξ and that this cleaning successfully removes the viral load from the needle and syringe. The simulation findings shown that, even if the cleaning procedure is imperfect or if PWIDs don't always clean their needles and syringes, washing can still have an influence on and even abolish HIV prevalence in the community.

1.5.2 Greenhalgh and Hay Model

In order to include more plausible hypotheses about the spread of HIV among IDUs, Greenhalgh and Hay (1997) modified the Kaplan (1989) model. These suppositions included:

(i) adjustments to enable IDUs with and without HIV to access shooting ranges at various rates (previously assumed the same),

(ii) Assuming different transmission probabilities for flushed and unflushed needles (In the Greenhalgh and Hay model, it is possible for an infectious needle used once by a susceptible IDU to be flushed or cleaned of infectious blood during the injection procedure and therefore become uninfectious,)

(iii) adjustments to account for the risk that IDUs who are HIV-positive would not always leave a dirty needle uncleaned. The authors discovered the model be-

haviour by R_0 after thorough mathematical and numerical study of their work. When $R_0 < 1$, they were able to demonstrate that their system would arrive at the DFE. They demonstrated the existence of a specific, locally stable positive endemic equilibrium if $R_0 > 1$.

1.5.3 Lewis and Greenhalgh Model

The Kaplan and O'Keefe (1993) model was expanded by Lewis and Greenhalgh (2001) to incorporate three stages of changing infectivity prior to the onset of AIDS. Contrary to the findings the research of Lewis and Greenhalgh (2001), the authors assumed that the most infectious IDU who most recently used the needle and syringe determined the infectivity of a needle and syringe.

This indicates that as they progress toward the peak infectivity stage, needles and syringes become progressively more infectious. The model's predictions were negative as a result of this assumption, and they established upper limits for the prevalence of HIV among IDUs and needle users.

The authors' mathematical research discovered similar findings to those of Lewis and Greenhalgh (2001). This model's HIV prevalence was compared to the model developed by Kaplan and O'Keefe (1993) using numerical simulations. Both of the models in these simulations, according to the results, attained an endemic equilibrium solution. However, the three-stage infectivity model did so earlier than theKaplan and O'Keefe (1993) model. Additionally, compared to Kaplan and O'Keefe (1993), the three-stage infectivity model predicted a higher long-term HIV prevalence.

1.6 Conclusion

This chapter presents the findings of a literature study that focused on the epidemiology and modelling of HIV/AIDs. A more fundamental description of the

ideas utilised when modelling infectious diseases has also been included. To begin, it is patently apparent that HIV infection is a significant issue all over the world.

Public health officials continue to face a major problem due to the high rates of HIV/AIDS infection, especially in underdeveloped regions of the world (Campbell et al. (2017), Paraskevis et al. (2011), Bonovas and Nikolopoulos (2012)). In 2017, there were roughly 36,900,000 persons living with HIV, 940,000 deaths attributable to AIDS, and 1,800,000 new infections globally, as reported by the Joint United Nations Programme on AIDS (UNAIDS) (UNAIDS, 2018). AIDS is a major threat to global public health since it is a chronic disease. And the mortality caused by AIDS is much higher than another sexually transmitted disease (STD) Li et al. (2014).

In fact, we investigate the risk of injecting drugs and the prevalence of HIV among those who inject drugs. Outbreaks of HIV among PWID occurred in south-eastern Saskatchewan, Canada; Athens, Greece; Dublin, Ireland; Tel Aviv, Israel; Luxembourg; Bucharest, Romania; Glasgow, Scotland; and the United States (Scott County, Indiana) between 2011 and 2016. Community economic issues, homelessness, and alterations in drug injecting behaviours were common to a number of these outbreaks. The outbreaks were different in size (from less than 100 to more than 1,000 new HIV cases reported among PWID) and varied in level of prevention before, during, and after the outbreaks Des Jarlais et al. (2020).

The mathematical epidemic models established in this thesis can be used to evaluate the efficacy of intervention strategies and bring attention to the needs that must be met in order to eradicate infectious diseases such as HIV, which are complex in nature and developing at an alarming rate. Additionally, these models are used to comprehend the spread of illnesses and assess the possible effectiveness of control initiatives in lowering morbidity and death. Here we discuss some of the more prevalent model structures, such as deterministic, SIS, and

SIR models, but there are many others. In order to demonstrate the use of these models, examples of their application were provided.

Also, in this chapter, we have provided a review of the impact of media outreach programs on the epidemiological model that will be examined in this thesis and described some of the previous work on these models.

Previously, we have covered a number of articles that deal with the transmission modelling of HIV/AIDS amongst PWID through the sharing of infected injection equipment or when this is a component of the model. Several significant heterogeneities are involved in the modelling of HIV transmission by needle sharing. For instance, there is a wide variety of needle-sharing rates among addicts, and the efficacy of needle cleansing is highly variable. This is a characteristic that has been covered in numerous publications earlier.

In the next chapter, we shall develop accurate models of shall develop HIV/AIDS models with awareness programs and also develop the model of Greenhalgh and Hay (1997)to suppose more realistically that PWIDs clean their needles before, not after use. It will examine the model governing system of differential equations that reflect the impact of HIV/AIDS awareness programmes on preventing the progression of the disease's spread.

Chapter 2

Incorporation of Awareness Programs into a One-dimensional Model of the spread of HIV/AIDS Amongst People who Inject Drugs

2.1 Introduction

In this chapter, we shall study and set up a deterministic mathematical model of the spread of HIV/AIDS amongst People who Inject Drugs(PWIDs). This chapter is organised as follows. In the next section, we shall develop an HIV/AIDS model with awareness programs and also develop the model of Greenhalgh and Hay (1997) to suppose more realistically that PWIDs clean their needles before not after use. Section 2.3 is divided into five subsections. Firstly we show the existence of unique non-negative solution, then we shall explore the existence of equilibrium points and analyse their stability both locally and globally. At the end of the subsection, we perform some simulations with realistic parameter values to verify the analytical results for HIV/AIDS models with disease awareness pro-

grams. In section 2.4 we shall adopt our model to deal with the spread of HCV amongst PWIDs and perform further simulations to verify the analytical results. The chapter concludes with a brief summary and discussion.

There are a huge number of deaths recorded each year around the world due to infectious diseases such as Pneumonia, Tuberculosis (TB), Diarrhoeal diseases, Cholera, Malaria and HIV/AIDS. The outbreaks of diseases are a strong cause for researchers to find a solution that reduces spread of these diseases. The media plays an important role in spreading health awareness by changing mixing behaviour. The published studies show some of the mathematical models which have been used to explore the effect of media awareness programs on the spread and control of infectious disease (Greenhalgh et al. 2015). In my current research, I incorporate awareness programs in a model of the spread of HIV/AIDS amongst people who inject drugs (PWIDs) in a population Greenhalgh and Hay (1997). One of our aims is to incorporate the effect of awareness of disease on sharing needles and syringes amongst the PWID population. We also modify the model of Greenhalgh and Hay (1997) to include more realistic cleaning of needles before use rather than after when visiting shooting galleries. There are two ways to include the effect of awareness programs into disease transmission models. We have chosen to focus on the first and simplest which is to reduce the disease transmission by a factor $\phi(\pi)$ between zero and one. The more complicated alternative is to model the amount of awareness as a separate differential equation. We developed a mathematical model of spread of HIV amongst PWIDs with an awareness program.

Our basic mathematical model for spread of HIV amongst PWIDs was studied by Greenhalgh and Hay (1997) and Liang et al. (2016), based on a previously described original model by Kaplan (Kaplan 1989). Our model analyses the spread of HIV/AIDS amongst a population of PWIDs. We introduced a function $\phi(\pi)$ which defines the fraction by which PWIDs reduce their needle sharing because

they are aware of the level of HIV infection in PWIDs. This is a positive monotone decreasing function of π, the fraction of PWIDs infected by HIV/AIDS. The model has also been applied to the spread of HCV amongst PWIDs. Now, we are going to derive the model equations for the spread of HIV/AIDS amongst PWIDs including awareness programs.

The majority of the research discussed in Chapter 2 can be found in a paper that was published in Alsharari and Greenhalgh (2023).

2.2 Formulation of HIV/AIDS Models with Awareness Programs

We modify the differential equation model for the spread of HIV has been described by Liang et al. (2016), multiplying the disease transmission term by the factor $\phi(\pi)$ to represent the reduction in the spread of HIV due to awareness programs. The biological parameters of the model are as described in Table 1 adapted from Greenhalgh and Hay (1997).

Note that P_1, P_2, P_3 and $P_4 \geq 0$ are positive and $P_1 + P_2 + P_3 + P_4 = 1$. Define

$$\begin{aligned}
\sigma &= \left[\lambda_1(1-p) + \lambda_2 p\right]\gamma(1-\xi)(1-\phi_1), \\
\tau &= \left[\lambda_1(1-p) + \lambda_2 p\right]\gamma\left[1-\phi_1(1-\xi) + \theta_1(1-\xi)\right], \\
\rho &= \lambda_1\gamma\left[1-(1-\xi)(1-P_1-P_2)\right], \\
\nu &= \lambda_1(P_1+P_3).
\end{aligned} \tag{2.1}$$

Let $\pi(t)$ be the fraction of HIV-infected PWIDs at time t and let $\beta(t)$ be the fraction of needles infected at time t. So we introduce the model as follows:

$$\frac{d\pi}{dt} = \phi(\pi)(1-\pi)\nu\beta - \mu\pi, \tag{2.2}$$

$$\frac{d\beta}{dt} = \phi(\pi)\pi(\sigma - \tau\beta) - \phi(\pi)(1-\pi)\rho\beta. \tag{2.3}$$

In general, we shall assume that ϕ is a positive monotone decreasing function with $\phi(0) = 1$.we reduced the dimensions of the model in (2.2) and (2.3), by

Table 2.1: Description of Parameters

Parameter	Definition
λ_1	How fast PWIDs who are susceptible and PWIDs who have disease but are not aware of this fact visit locations where PWIDs share needles.
λ_2	How fast infected PWIDs who are aware that they have the disease visit places where PWIDs share needles.
P_1	Chance that the PWID catches disease but the syringe remains uninfected when an initially susceptible PWID injects with an initially uninfected needle.
P_2	Chance that the PWID does not catch the disease and the needle becomes uninfected when an initially susceptible PWID injects with an initially infected needle.
P_3	Chance that the PWID catches the disease and the needle remains infected when an initially susceptible PWID injects with an initially infected needle.
P_4	The chance that the PWID does not catch the disease and the needle stays infected when an initially susceptible PWID injects with an initially infected needle.
ϕ_1	Chance that an infected PWID leaves uninfected an initially uninfected syringe.
θ_1	Chance that a PWID with disease leaves uninfected a needle that contained the virus before injection.
ξ	Proportion of PWIDs who that clean syringes after using them.
γ	Gallery ratio, where $\gamma = \frac{n}{m}$, and n is the total number of PWIDs and m is the total number of shared needles.
p	The chance that PWIDs with disease are aware of being infected.
μ	Rate per PWID at which PWIDS either stop sharing needles or develop full-blown AIDS.

supposing that the equation (2.3) is at steady state as a similar technique is used in models for HIV amongst PWIDs as discussed by Liang et al. (2016). So we do that and then give the basic analytical results and simulations. We got that

$$\frac{d\pi(t)}{dt} = \frac{\phi(\pi(t))(1-\pi(t))\nu\sigma\pi(t)}{\pi(t)\tau + \rho - \pi(t)\rho} - \mu\pi(t). \tag{2.4}$$

Next, we are going to make the model more realistic by modifying Greenhalgh and Hay's model.

2.2.1 Development of Greenhalgh and Hay's model

Greenhalgh and Hay's model was based on Kaplan's basic model which assumed that PWIDs cleaned their needles after use. In practice, PWIDs are more likely to disinfect their syringes before injecting. So we modify the model of Greenhalgh and Hay to make it more realistic so PWIDs clean their needles before use and we also introduce a disease awareness function Let $I(t)$ denote the number of infected PWIDs at time t and $i(t)$ the number of infected needles at time t. For a small time interval $[t, t + \Delta t]$:

$$\begin{aligned} I(t+\Delta t) &= \text{number of infected PWIDs at time } t \\ &\quad + \text{number of new PWIDs infected in } [t, t+\Delta t) \\ &\quad - \text{number of PWIDs who stop sharing needles or} \\ &\quad \text{develop full-blown AIDS } [t, t+\Delta t). \\ I(t+\Delta t) &= I(t) + (n-I)\lambda_1\phi(\pi)(P_1+P_3)(1-\xi)\beta\Delta t - \mu I\Delta t + o(\Delta t). \end{aligned}$$

We use the notation that if $f(x)$ and $g(x)$ are two functions, then $f(x) = o(g(x))$ means that $\frac{f(x)}{g(x)} \to 0$ as $x \to 0$. The term $(n-I)\lambda_1\phi(\pi)(P_1+P_3)(1-\xi)\beta\Delta t$ is because there are $n - I$ uninfected PWIDs each of whom injects at rate $\lambda_1\phi(\pi)$, chooses an infected needle with probability β, does not clean the needle before use with probability $1 - \xi$ and is infected at each injection with probability $P_1 + P_3$. Rearranging

$$\frac{I(t+\Delta t) - I(t)}{\Delta t} = (n-I)\lambda_1\phi(\pi)(P_1+P_3)(1-\xi)\beta - \mu I + o(1). \tag{2.5}$$

Letting $\Delta t \to 0$

$$\frac{dI}{dt} = (n - I)\lambda_1\phi(\pi)(P_1 + P_3)(1 - \xi)\beta - \mu I. \tag{2.6}$$

Dividing by n, the number of PWIDs,

$$\frac{d\pi(t)}{dt} = (1 - \pi)\lambda_1\phi(\pi)\beta(P_1 + P_3)(1 - \xi) - \mu\pi. \tag{2.7}$$

Now we turn to the needle equations. We are going to construct and examine the differential equations for $\pi(t)$ the proportion of PWIDs with disease and $\beta(t)$, the proportion of needles with the disease. We construct and examine the differential equations for these quantities. Consider the number of infected needles at time $t + \Delta t$.

$i(t + \Delta t)$ = number of syringes infectious at time $t + \Delta t$,

= number of syringes infectious at time t and not visited by PWIDs in $[t, t + \Delta t)$

+ number of syringes left infectious at time $t + \Delta t$ after being visited PWIDs in by infected $[t, t + \Delta t)$

+ number of syringes left infectious at time $t + \Delta t$ after being visited PWIDs in by susceptible $[t, t + \Delta t)$.

(i) $n(1 - p\pi)$ PWIDs arrive at shooting galleries at rate $\lambda_1\phi(\pi)$. Also $np\pi$ PWIDs visit at a rate $\lambda_2\phi(\pi)$. Each PWID chooses one of the m shooting galleries randomly. So at one given shooting gallery PWIDs arrive at rate $[\lambda_1(1 - p\pi) + \lambda_2 p\pi]\,\gamma\phi(\pi)$. Here $\gamma = n/m$ is the number of PWIDs divided by the number of needles. So

$$\left\{1 - [\lambda_1(1 - p\pi) + \lambda_2 p\pi]\,\gamma\phi(\pi)\Delta t\right\} i + o(\Delta t) \tag{2.8}$$

syringes are infectious at time t and no PWIDs use them in $[t, t + \Delta t)$.
(ii) For a given shooting gallery infected PWIDs enter it at rate $[\lambda_1(1 - p) + \lambda_2 p]\,\gamma\phi(\pi)$. If a PWID who is infected uses a syringe, this syringe will be infectious after being used and cleaned with probability $(1 - \beta + \beta\xi)(1 - \phi_1) + \beta(1 - \xi)(1 - \theta_1)$ and in a

small interval $[t, t+\Delta t]$

$$m\left[\lambda_1(1-p)+\lambda_2 p\right]\pi\gamma\phi(\pi)\Big[(1-\beta+\beta\xi)(1-\phi_1)+\beta(1-\xi)(1-\theta_1)\Big]\Delta t+o(\Delta t), \tag{2.9}$$

units of injection equipment will be left infectious subsequent to use by a PWID with the disease.

(*iii*) If we take a given syringe uninfected PWIDs come to it at a rate $\lambda_1\gamma\phi(\pi)(1-\pi)$. If a susceptible PWID uses an infectious syringe, afterwards, the equipment will be capable of transmitting infection with probability $(1-P_1-P_2)(1-\xi)$. So the number of syringes infectious after use by an uninfected PWID in $[t, t+\Delta t]$ is

$$\lambda_1\gamma\phi(\pi)(1-\pi)i(1-P_1-P_2)(1-\xi)\Delta t+o(\Delta t). \tag{2.10}$$

Hence

$$\begin{aligned} i(t+\Delta t) = i\Big\{1-\left[\lambda_1(1-p\pi)+\lambda_2 p\pi\right]\gamma\phi(\pi)\Delta t\Big\} \\ +m\left[\lambda_1(1-p)+\lambda_2 p\right]\phi(\pi)\pi\gamma\Big[(1-\beta+\beta\xi)(1-\phi_1)+\beta(1-\xi)(1-\theta_1)\Big]\Delta t \\ +\lambda_1\gamma(1-\pi)i(1-P_1-P_2)(1-\xi)\phi(\pi)\Delta t+o(\Delta t). \end{aligned} \tag{2.11}$$

Subtracting $i(t)$ from both sides and dividing by Δt

$$\begin{aligned} \frac{i(t+\Delta t)-i(t)}{\Delta t} = -\left[\lambda_1(1-p\pi)+\lambda_2 p\pi\right]\gamma\phi(\pi)i \\ +m\left[\lambda_1(1-p)+\lambda_2 p\right]\phi(\pi)\pi\gamma\Big[(1-\beta+\beta\xi)(1-\phi_1)+\beta(1-\xi)(1-\theta_1)\Big] \\ +\lambda_1\gamma(1-\pi)i(1-P_1-P_2)(1-\xi)\phi(\pi)+o(1). \end{aligned} \tag{2.12}$$

Letting $\Delta t\to 0$

$$\begin{aligned} \frac{di}{dt} = -\left[\lambda_1(1-p\pi)+\lambda_2 p\pi\right]\gamma\phi(\pi)i \\ +m\left[\lambda_1(1-p)+\lambda_2 p\right]\phi(\pi)\pi\gamma\Big[(1-\beta+\beta\xi)(1-\phi_1)+\beta(1-\xi)(1-\theta_1)\Big] \\ +\lambda_1\gamma(1-\pi)i(1-P_1-P_2)(1-\xi)\phi(\pi). \end{aligned} \tag{2.13}$$

Dividing by m

$$\begin{aligned}\frac{d\beta}{dt} =& -\left[\lambda_1(1-p\pi)+\lambda_2 p\pi\right]\gamma\phi(\pi)\beta \\ &+\left[\lambda_1(1-p)+\lambda_2 p\right]\phi(\pi)\pi\gamma\left[(1-\beta+\beta\xi)(1-\phi_1)+\beta(1-\xi)(1-\theta_1)\right] \\ &+\lambda_1\gamma(1-\pi)\beta(1-P_1-P_2)(1-\xi)\phi(\pi).\end{aligned}$$

Hence we deduce that equations (2.2) and (2.3) hold where σ, τ, ρ and ν are redefined as

$$\begin{aligned}\sigma &= \left[\lambda_1(1-p)+\lambda_2 p\right]\gamma(1-\phi_1), \\ \tau &= \left[\lambda_1(1-p)+\lambda_2 p\right]\gamma\left[1-(1-\xi)(1-\theta_1)+(1-\xi)(1-\phi_1)\right], \\ \rho &= \lambda_1\gamma\left[1-(1-\xi)(1-P_1-P_2)\right], \\ \nu &= \lambda_1(P_1+P_3)(1-\xi).\end{aligned} \tag{2.14}$$

Note also that using numbers not fractions of needles and PWIDs with disease equations (2.2) and (2.3) become

$$\frac{dI}{dt} = \phi(\pi)(n-I)\nu\frac{i}{m} - \mu I, \tag{2.15}$$

$$\frac{di}{dt} = \phi(\pi)\frac{I\sigma}{n}(m-i) - \phi(\pi)\frac{(n-I)}{n}\rho i - \phi(\pi)\frac{I}{n}(\tau-\sigma)i. \tag{2.16}$$

Note that $\tau > \sigma$. These equations are explained by the flow diagram in Figure 2.1. In equation (2.15)

$$\phi(\pi)(n-I)\frac{\nu i}{m},$$

is the rate at which susceptible PWIDs arrive at infected needles and become infectious. On the other hand, μI is how fast infected PWIDs stop sharing needles and are replaced by uninfected PWIDs.

Equation (2.16) is more complicated. The term

$$\phi(\pi)\frac{I\sigma}{n}(m-i),$$

is the rate at which infected PWIDs arrive at uninfected needles, do not clean the needle before use and infect the needle, i.e. the rate at which new infected needles occur. Of the two terms on the right hand side

$$\phi(\pi)\frac{(n-I)}{n}\rho i,$$

is the rate at which uninfected PWIDs visit infected needles and either clean the needle before use or flush the needle, in other words the rate at which uninfected PWIDs visit infected needles and leave the needle uninfected. The other term on the right hand side is

$$\phi(\pi)\frac{Ii}{n}(\tau-\sigma), \tag{2.17}$$

and note $\tau - \sigma = [\lambda_1(1-p) + \lambda_2 p]\gamma[\xi\phi_1 + \theta_1(1-\xi)]$ so (2.17) is the rate at which infected PWIDs visit infected needles and leave the needles uninfected. This completes our interpretation of equations (2.15) and (2.16).

Now we move on to compute the basic reproductive number of equation model (2.4).

2.2.2 The Basic Reproductive Number R_0

R_0 is important in epidemic models. Usually, the disease becomes extinct if $R_0 < 1$ and takes off if $R_0 > 1$, so to derive R_0, we consider a new infected PWID coming into a steady state population with no disease. The basic reproduction number is the average number of PWIDs who catch the disease via only one infectious syringe. The definition of R_0 used here is similar to the definition used in Macdonald (1952) , Massad et al. (2001), Sanches and Massad (2016) and Van den Driessche (2017). It takes the basic infectious unit to be an infectious human. This method and the corresponding definition of R_0 are different than the Next Generation Matrix approach as discussed by Diekmann et al. (1990b),

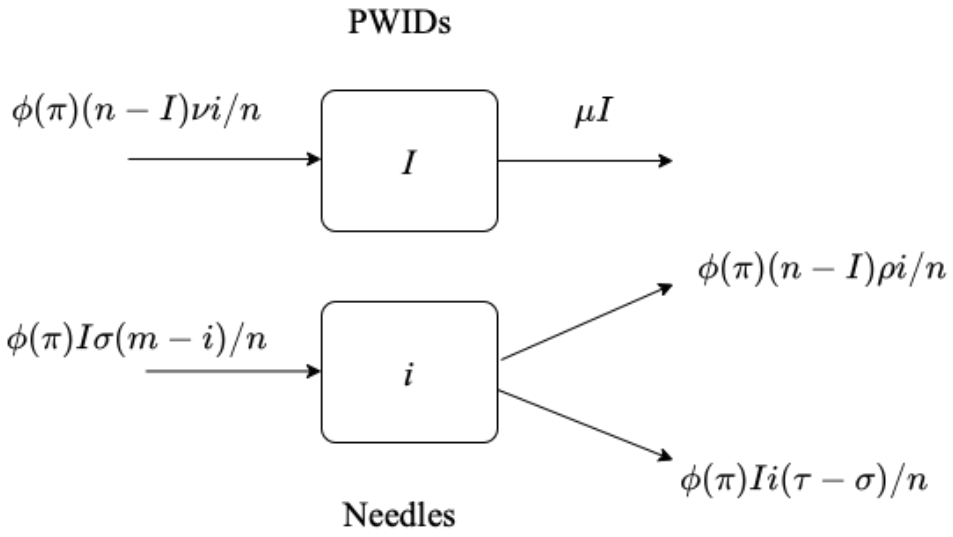


Figure 2.1: Flow diagram of equations (2.15) and (2.16).

Van den Driessche (2017), Van den Driessche and Watmough (2002, 2008) and Roberts and Heesterbeek (2003). The Next Generation Matrix Method treats PWIDs and syringes as separate infectious entities. Consequently, the value of R_0 derived by this method is the square root of the one we obtained. However, as each passes through one at the same time they give equivalent qualitative results if used as a threshold value.

R_0 is given as

$$R_0 = \frac{\nu\sigma}{\rho\mu}. \tag{2.18}$$

This expression for R_0 can be derived by considering a single PWID who is infected with HIV in a completely disease-free environment when no other PWIDs

have HIV, and all needles are clean. From equations (2.2) and (2.3) we have

$$\begin{aligned}\frac{dI}{dt} &= n\phi(\pi)(1-\pi)\nu\beta - \mu I,\\ &= \gamma\nu\phi(\pi)(1-\pi)i - \mu I,\\ \frac{di}{dt} &= \frac{m}{n}\phi(\pi)I(\sigma-\tau\beta) - \phi(\pi)(1-\pi)\rho i,\\ &= \Big(\frac{\sigma-\tau\beta}{\gamma}\Big)\phi(\pi)I - \phi(\pi)(1-\pi)\rho i.\end{aligned} \tag{2.19}$$

As we are near the disease-free equilibrium (DFE) we neglect second order terms in small quantities to obtain

$$\begin{aligned}\frac{dI}{dt} &= \gamma\nu i - \mu I,\\ \frac{di}{dt} &= \frac{\sigma}{\gamma}I - \rho i.\end{aligned} \tag{2.20}$$

A newly infected PWID remains in the sharing injecting population for time $\frac{1}{\mu}$. During that time he or she contaminates the number of needles denoted by $\frac{\sigma}{\mu\gamma}$. Each needle remains infectious for time $\frac{1}{\rho}$ and during that time it infects $\frac{\gamma\nu}{\rho}$ PWIDs. So each PWID causes $\frac{\sigma}{\mu\gamma}\cdot\frac{\gamma\nu}{\rho} = \frac{\nu\sigma}{\rho\mu}$ secondary infections in PWIDs, so $R_0 = \frac{\nu\sigma}{\rho\mu}$. R_0 can also be derived by considering the expected number of syringes infected via only one infected PWID caused by a single syringe which has just been infected and entering a steady-state population with no disease. This syringe causes $\frac{\gamma\nu}{\rho}$ infectious PWIDs who each in turn infect $\frac{\sigma}{\mu\gamma}$ infectious needles. So again $R_0 = \left(\frac{\gamma\nu}{\rho}\right)\cdot\left(\frac{\sigma}{\mu\gamma}\right) = \frac{\nu\sigma}{\rho\mu}$. We will see that R_0 is a critical parameter which will determine if the disease can sustain itself.

This concludes our analysis of the basic reproduction number. In the next section, we shall analytically study the one-dimensional model equation (2.4).

2.3 Analysis of the One Dimensional Model

We are going to determine the dynamical behaviour of the model in (2.4) depending on the basic reproductive number we shall start off by showing the existence and uniqueness of a non-negative solutions to (2.4) then discuss the

existence of equilibrium solutions. Next we discuss the local and global stability of these solutions. Finally our analytical results will be illustrated by using simulation with realistic parameter values.

2.3.1 Existence of Unique Non-negative Solution

To show that there is one and only one non-negative model solution of the model (2.4), we require to apply the concept of a Lipschitz continuous functions and the Picard-Lindelöf theorem.

(i) Lipschitz continuous functions: (Wikipedia (2022), Searcóid (2006))

Definition: Let (X, dX) and (Y, dY) be two metric spaces as described in Choudhary (2011) where dX denotes the metric on the set X and dY is the metric on set Y, a function $f : X \to Y$ is called a Lipschitz continuous function if there exists a real constant $K \geq 0$ such that for all x_1 and $x_2 \in X$.

$$\left|f(x_1) - f(x_2)\right| < K\left|x_1 - x_2\right|.$$

K is called a Lipschitz constant for the function f. In particular, for a real-valued function define Y as the set of real numbers of R with the metric $dY(y_1, y_2) = |y_1 - y_2|$, and X might be a subset of R with the same metric.

(ii) The Picard-Lindelöf Theorem: the Picard-Lindelöf existence theorem is an important theorem in the study of differential equations, which indicates existence and uniqueness of solutions to first-order equations with given initial conditions. Consider the initial value problem

$$\frac{dy}{dt} = f(t, y(t)), \qquad y(t_0) = y_0.$$

Suppose that $f : R \times R \to R$ is uniformly continuous in y. This means that the Lipschitz constant K is independent of t. Then for some $\xi > 0$ there exists a unique solution for $y(t)$ to the initial value problem in the interval [t_0-ξ , t_0+ξ].

Theorem 2.3.1. *Suppose that ϕ is a Lipschitz continuous function of π for any particular starting value $\pi(0) = \pi_0 \in [0,1]$ there is one and only one non negative solution for the PWID equation model (2.4).*

Proof: Define $f(\pi)$ to be the right-hand side of equation (2.4) where σ, τ, ρ and ν are defined by equations (2.14). First we going to show that the function f is Lipschitz continuous. So for all $\pi_1, \pi_2 \in$ [0,1].

$$\begin{aligned} \left|f(\pi_1) - f(\pi_2)\right| = \Bigg|&\left(\frac{\phi(\pi_1(1-\pi_1)\nu\sigma\pi_1}{\pi_1\tau + \rho - \pi_1\rho} - \mu\pi_1\right) \\ &- \left(\frac{\phi(\pi_2(1-\pi_2)\nu\sigma\pi_2}{\pi_2\tau + \rho - \pi_2\rho} - \mu\pi_2\right)\Bigg|. \end{aligned} \tag{2.21}$$

So now we going to split it by using the triangle inequality.

$$\begin{aligned} \left|f(\pi_1) - f(\pi_2)\right| &\leq \left|\left(\frac{\phi(\pi_1(1-\pi_1)\nu\sigma\pi_1}{\pi_1\tau + \rho - \pi_1\rho}\right) - \left(\frac{\phi(\pi_2)(1-\pi_2)\nu\sigma\pi_2}{\pi_2\tau + \rho - \pi_2\rho}\right)\right| \\ &\quad + \mu\left|\pi_1 - \pi_2\right|, \\ &\leq \left|\left(\frac{\phi(\pi_1)(1-\pi_1)\nu\sigma\pi_1}{\pi_1\tau + \rho - \pi_1\rho}\right) - \left(\frac{\phi(\pi_2)(1-\pi_1)\nu\sigma\pi_1}{\pi_1\tau + \rho - \pi_1\rho}\right)\right| \\ &\quad + \left|\left(\frac{\phi(\pi_2)(1-\pi_1)\nu\sigma\pi_1}{\pi_1\tau + \rho - \pi_1\rho}\right) - \left(\frac{\phi(\pi_2)(1-\pi_2)\nu\sigma\pi_2(t)}{\pi_2\tau + \rho - \pi_2\rho}\right)\right| \\ &\quad + \mu\left|\pi_1 - \pi_2\right|, \\ &\leq \left|\phi(\pi_1) - \phi(\pi_2)\right| \frac{(1-\pi_1)\nu\sigma\pi_1}{\pi_1\tau + \rho - \pi_1\rho} \\ &\quad + \left|\phi(\pi_2)\right| \left|\frac{(1-\pi_1)\nu\sigma\pi_1}{\pi_1\tau + \rho - \pi_1(t)\rho} - \frac{(1-\pi_2)\nu\sigma\pi_2}{\pi_2\tau + \rho - \pi_2\rho}\right| \\ &\quad + \mu\left|\pi_1 - \pi_2\right|, \\ &\leq K\frac{(\pi_1 - \pi_2)\nu\sigma}{min(\rho,\tau)} + K_1\left|\pi_1 - \pi_2\right| + \mu\left|\pi_1 - \pi_2\right|. \end{aligned} \tag{2.22}$$

Here K is the Lipschitz constant for ϕ and for the first term we are using the fact

that ϕ is Lipschitz continuous. For the second term we use the fact that

$$g(\pi) = \frac{\nu\sigma(1-\pi)\pi}{\pi\tau + \rho - \pi\rho}.$$

is continuously differentiable for $\pi \in [0,1]$ with derivative bounded above by a constant K_1. Then by the Intermediate Value Theorem

$$|g(\pi_1) - g(\pi_2)| \leq K_1|\pi_1 - \pi_2|.$$

From inequality (2.22) we deduce that f is Lipschitz continuous for $\pi \in [0,1]$.

We now continue with the proof of Theorem 2.3.1. We shall split the rest of the proof into three different cases. The first one is $\pi(0) \in (0,1)$, the second one is $\pi(0) = 1$ and the third one is $\pi(0) = 0$.

First, suppose that $\pi(0) \in (0,1)$. By applying the Picard–Lindelöf Theorem there exists a unique local solution.

Let us define $[0, \tau_e)$ to be the maximum interval where a solution exists and $\pi \in (0,1)$ for all ξ in $[0, \tau_e)$. We shall show that $\tau_e = \infty$ by using an argument by contradiction.

(i) We suppose that $\tau_e < \infty$. By using the Picard-Lindelöf Theorem, $\exists\ \Delta t > 0$ such that the solution exists in $[0, \Delta t]$. As $\pi(0) \in (0,1)$ we must have $\pi(s) \in (0,1)$ for $s \in [0, \Delta t]$, if Δt is small enough. Hence we have shown that $\tau_e > 0$. Now by integrating the expression given in equation (2.4)

$$\frac{1}{\pi}\frac{d\pi(t)}{dt} = \frac{\phi(\pi(t))(1-\pi(t))\nu\sigma}{\pi(t)\tau + \rho - \pi(t)\rho} - \mu, \tag{2.23}$$

for $t < \tau_e$,

$$\pi(t) = \pi(0)\exp\left(\int_0^t \left(\frac{\phi(\pi(t))(1-\pi(t))}{\pi(t)\tau + \rho - \pi(t)\rho} - \mu\right)dt\right). \tag{2.24}$$

Letting $t \to \tau_e$

$$\lim_{t\to\tau_e} \pi(t) = \pi(\tau_e) = \pi(0)\exp\left(\int_0^{\tau_e} \left(\frac{\phi(\pi(t))(1-\pi(t))}{\pi(t)\tau + \rho - \pi(t)\rho} - \mu\right)dt\right) > 0. \tag{2.25}$$

Let $f(\pi)$ denote the right hand side of the equation (2.23) as $\pi \to 1$, then $f(\pi) \to -\mu$. So there exists $\alpha < 1$, as such that for $\pi \geq \alpha$, $f(\pi) < 0$. $\pi(t)$ can never exceed α as if it does it must increase from α to its new value

contradicting $\frac{d\pi(t)}{dt} < 0$, for $\pi \geq \alpha$. By using the Picard́-Lindelöf Theorem there exists a unique local solution to the equation in $[\tau_e - \eta, \tau_e + \eta]$ for some $\eta > 0$. As the unique solution is continuous at τ_e,

$$\lim_{t \to \tau_e} \pi(t) \leq \alpha < 1.$$

So $\pi(\tau_e) \in (0, 1)$, moreover a similar argument shows that $\pi(t) \in (0, 1)$ for $t \in [0, \tau_e + \epsilon]$, where ϵ is small and positive. This is a contradiction to the definition of τ_e, so $\tau_e = \infty$. So this completes the proof of Theorem 2.3.1 in this case.

(ii) Suppose that $\pi(0) = 1$. Then by using the Picard́-Lindelöf Theorem, there exists $\Delta t > 0$ such that the solution exists in $[0, \Delta t]$. If Δt is small enough $\pi(\eta) > 0$ for $\eta \in [0, \Delta t)$ as,

$$\pi(\eta) = \pi(0) + f(0)\eta + o(\eta),$$

$$= 1 - \mu\eta + o(\eta).$$

If Δt is small enough then $\pi(\eta) < 1$ on $(0, \Delta t]$, so $0 < \pi(\Delta t) < 1$. The result of Theorem 2.3.1 follows by Case 1.

(iii) Suppose that $\pi(0) = 0$. By using the Picard́-Lindelöf Theorem, there exists $\Delta t > 0$ such that the equation has a unique local solution in $[0, \Delta t]$. We can see that $\pi(t) = 0$ is the solution for all time. Let τ_e be the maximum interval where a unique solution exists with $\pi(t) = 0$ for ξ in $[0, \tau_e)$. By the same argument as in Case 1 we have that $\tau_e > 0$. Suppose that $\tau_e < \infty$ and $\pi(t) = 0$ for all $t < \tau_e$, again by using the Picard́-Lindelöf Theorem there exists a unique local solution in $(\tau_e - \eta, \tau_e + \eta)$ for some $\eta > 0$, and $\pi(t) = 0$ in $[0, \tau_e + \eta)$, this is a contradiction. So again we deduce that $\tau_e = \infty$. This completes the proof of Theorem 2.3.1 in Case 3 and the proof of Theorem 2.3.1 at together. To briefly summaries from the proof of Theorem 2.3.1 we have that

- If $\pi(0) \in (0,1)$, then $\pi(t) \in (0,1) \forall t \geq 0$,
- If $\pi(0) = 1$, then $\pi(t) \in (0,1) \forall t > 0$,
- If $\pi(0) = 0$, then $\pi(t) = 0 \forall t \geq 0$.

We have now finished the proof of Theorem 2.3.1. Next, we shall look at the existence of equilibria for the above model (2.4).

2.3.2 Existence of Equilibria

We shall show that if R_0 is less than one then there is only the steady state with no disease whereas if R_0 exceeds one, there is one and only one steady state with disease present.

if $R_0 \leq 1$ then there is only the disease-free equilibrium whereas if $R_0 > 1$ then there is a unique endemic equilibrium as well as the DFE. We shall first look at the case where ϕ is strictly decreases with π, and then the case where it is just (possibly not strictly) monotone decreasing.

Theorem 2.3.2. *Suppose that ϕ is strictly monotone decreasing and $R_0 \leq 1$ then the equation (2.4) will have one and only one steady-state solution where eventually there is no disease present where the disease dies out in PWIDs, $\pi^* = 0$. This is the only equilibrium. If $R_0 > 1$ there exists exactly one non-zero steady-state solution $\pi^* > 0$ in (0,1] as well as the DFE.*

Proof: The trivial equilibrium is $\pi^* = 0$, and any other equilibrium must satisfy the equation

$$\frac{\phi(\pi)(1-\pi)\nu\sigma}{\pi\tau + \rho - \pi\rho} - \mu = 0. \tag{2.26}$$

Re-arranging (2.26) we deduce that

$$\frac{1}{\pi} = \frac{1}{\frac{\nu\sigma}{\mu\tau}\phi(\pi) - \frac{\rho}{\tau}} + 1. \tag{2.27}$$

Define

$$g_1(\pi) = \frac{1}{\pi}, \text{ and } g_2(\pi) = \frac{1}{\frac{\nu\sigma}{\mu\tau}\phi(\pi) - \frac{\rho}{\tau}} + 1.$$

There are several situations to consider.

(i) Suppose that $R_0 = \frac{\nu\sigma}{\rho\mu} < 1$. In this case we have that

$$\frac{\nu\sigma}{\mu\tau}\phi(\pi) - \frac{\rho}{\tau} \leq \frac{\nu\sigma}{\mu\tau} - \frac{\rho}{\tau} < 0, \ \forall \pi(t).$$

Hence $g_2(\pi) < 1$ and $g_1(\pi) \geq 1$ in the equation (2.27), for $\pi \in (0, 1]$. Therefore, there is no non-zero solution in this case.

(ii) If $R_0 = \frac{\nu\sigma}{\rho\mu} = 1$, then we have the same thing that

$$\frac{\nu\sigma}{\mu\tau}\phi(\pi) - \frac{\rho}{\tau} \leq \frac{\nu\sigma}{\mu\tau} - \frac{\rho}{\tau} = 0, \ \forall \pi > 0.$$

So again $g_2(\pi) < 1$ and $g_1(\pi) \geq 1$ in $(0, 1]$. Thus again there is no strictly positive solution.

(iii) If $R_0 = \frac{\nu\sigma}{\rho\mu} > 1$, we know that $\phi(\pi)$ is strictly monotone decreasing so we consider the equation given by

$$\phi(\pi) = \frac{\rho\mu}{\nu\sigma} < 1. \tag{2.28}$$

We consider three cases.

(a) If $\phi(1) > \frac{\rho\mu}{\nu\sigma}$, there are no roots of the equation (2.28) in $[0, 1]$. In this case as $\pi \to 0$, then $g_1(\pi) \to \infty$ and

$$g_2(\pi) \to 1 + \frac{1}{\frac{\nu\sigma}{\mu\tau} - \frac{\rho}{\tau}} < \infty, \text{ because } \frac{\nu\sigma}{\mu\rho} > 1.$$

At $\pi = 1$, $g_1(\pi) = 1$ and

$$g_2(\pi) = 1 + \frac{1}{\frac{\nu\sigma}{\mu\tau}\phi(1) - \frac{\rho}{\tau}} > 1, \text{ as } \frac{\nu\sigma}{\mu\tau}\phi(1) > \frac{\rho}{\tau}.$$

So the equation (2.27) has a non-zero root π^* in $(0, 1)$. Moreover $g_1(\pi)$ is strictly monotone decreasing in π and $g_2(\pi)$ is strictly monotone increasing in π, so this root is unique in $(0, 1]$.

(b) If $\phi(1)=\frac{\rho\mu}{\nu\sigma}$, then the equation (2.28) has a unique root at $\pi = 1$. Again as $\pi \to 0$ then $g_1(\pi) \to \infty$ and $\lim_{\pi\to 0} g_2(\pi) < \infty$.

For $\pi < 1$, $g_1(\pi) > 1$ and $g_2(\pi) < \infty$ arguing as above.

For $\pi = 1$, $g_1(\pi) = 1$ and $g_2(\pi) = \infty$. We have

$$\lim_{\pi\to 0} g_1(\pi) > \lim_{\pi\to 0} g_2(\pi),$$

$$\lim_{\pi\to 1} g_1(\pi) = 1 < \lim_{\pi\to 1} g_2(\pi) = \infty.$$

So the equation (2.28) has a root in $(0, 1)$ and similarly to case (a) this root is unique in $[0, 1]$.

(c) If $\phi(1) < \frac{\rho\mu}{\nu\sigma}$, then we know that **(1)** $\phi(0) = 1 > \frac{\rho\mu}{\nu\sigma}$ and **(2)** $\phi(1) < \frac{\rho\mu}{\nu\sigma}$, so equation (2.28) has a unique root π^{**} in $[0, 1]$. This case is illustrated by Figure (2.2). As $\pi \to 0$ then $g_1(\pi) \to \infty$ and $g_2(\pi) \to 1+\frac{1}{\frac{\nu\sigma}{\mu\tau} - \frac{\rho}{\tau}} < \infty$.

As $\pi \to \pi^{**}$ then $g_1(\pi) \to \frac{1}{\pi^{**}} < \infty$ and $g_2(\pi) \to \infty$ so the equation (2.27) has a unique root in $(0, \pi^{**})$, uniqueness follows as previously. At $\pi = \pi^{**}$, $g_1(\pi^{**}) < 1$ and $g_2(\pi^{**}) = \infty$. For $\pi \in (\pi^{**}, 1]$, $g_1 \geq 1$ and $\phi(\pi) < \frac{\rho\mu}{\nu\sigma}$ so $g_2(\pi) < 1$. So there are no roots of the equation (2.27) in $[\pi^{**}, 1]$. So equation (2.27) has a unique root in $[0, 1]$. The proof of Theorem 2.3.2 is thus finished.

Corollary 2.3.1. *Suppose that ϕ is monotone decreasing. Then the conclusion of Theorem 2.3.2 regarding the existence and uniqueness of equilibria for $R_0 \leq 1$ and $R_0 > 1$ still holds.*

Proof: Any non- trivial solution must satisfy the equation (2.27). Again there are three situations to consider, the first is $R_0 < 1$, the second $R_0 = 1$ and the third $R_0 > 1$.

(i) $R_0 = \frac{\nu\sigma}{\rho\mu} < 1$. The proof given in Theorem (2.3.2) is still valid in this case.

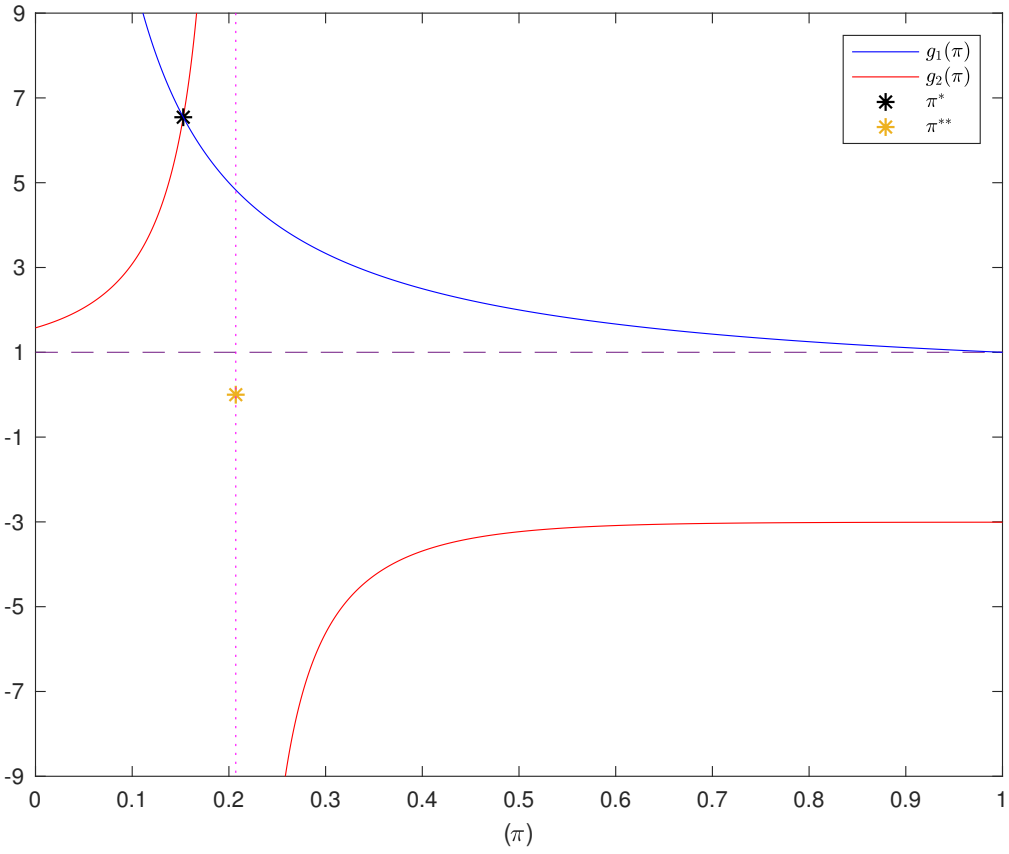


Figure 2.2: Illustration of Theorem 2.3.2 Case 3(c).

(ii) If $R_0 = \dfrac{\nu\sigma}{\rho\mu} = 1$,then we have the same thing that

$$\frac{\nu\sigma}{\mu\tau}\phi(\pi) - \frac{\rho}{\tau} \leq \frac{\nu\sigma}{\mu\tau} - \frac{\rho}{\tau} = 0.$$

for$\pi > 0$.

So we have $g_1(\pi) \geq 1$ and either $g_2(\pi) < 1$

if $\phi(\pi) < 1$ or $g_2(\pi)$=∞ if $\phi(\pi) = 1$ in (0,1]. Thus again there is no strictly positive solution.

(iii) If R_0= $\dfrac{\nu\sigma}{\rho\mu} > 1$, we know $\phi(\pi)$ is monotone decreasing so we consider the equation given by

$$\phi(\pi) = \frac{\rho\mu}{\nu\sigma} < 1. \tag{2.29}$$

We consider three cases.

(a) If $\phi(1) > \frac{\rho\mu}{\nu\sigma}$, there are no roots of the equation (2.29). In this case as $\pi \to 0$, then $g_1(\pi) \to \infty$ and

$$g_2(\pi) \to 1 + \frac{1}{\frac{\nu\sigma}{\mu\tau} - \frac{\rho}{\tau}} < \infty, \text{ because } \frac{\nu\sigma}{\mu\rho} > 1.$$

At $\pi = 1$, $g_1(\pi) = 1$ and

$$g_2(\pi) = 1 + \frac{1}{\frac{\nu\sigma}{\mu\tau}\phi(1) - \frac{\rho}{\tau}} > 1, \text{ as } \quad \frac{\nu\sigma}{\mu\tau}\phi(1) > \frac{\rho}{\tau}.$$

So the equation (2.27) has a non-zero root π^* in $(0, 1]$. Moreover $g_1(\pi)$ is strictly monotone decreasing in π and $g_2(\pi)$ is monotone increasing in π, so this root is unique in (0,1].

(b) If $\phi(1) = \frac{\rho\mu}{\nu\sigma}$, then the equation (2.29) has as root any value in the closed interval $[\pi^+, 1]$, given by $\phi(\pi) = \phi(1)$ with right limit 1.

For $\pi \in [0, \pi^+)$, $\phi(\pi) > \phi(1)$. As $\pi \to 0$ then $g_1(\pi) \to \infty$ and

$$g_2(\pi) \to 1 + \frac{1}{\frac{\nu\sigma}{\mu\tau} - \frac{\rho}{\tau}} < \infty.$$

As $\pi \to \pi^+$ then $g_1(\pi) \to \frac{1}{\pi^+}$ and $g_2(\pi) \to \infty$, hence there is a root in $(0, \pi^+)$ and arguing as in case (a) it is unique. For $[\pi^+, 1]$, $g_1(\pi) < \infty$ and $g_2(\pi) = \infty$ so there are no roots in this region. So the equation (2.27) has a root in $[0, \pi^+)$ and similarly to case (a) this root is unique in $[0, \pi^+)$, hence unique in $[0, 1]$ as there are no roots in $[\pi^+, 1]$.

(c) If $\phi(1) < \frac{\rho\mu}{\nu\sigma}$, then we know that $\phi(0) = 1 > \frac{\rho\mu}{\nu\sigma}$, so equation (2.29) has roots in a closed interval $[\pi_1^{**}, \pi_2^{**}] \subset [0, 1]$. As $\pi \to 0$ then $g_1(\pi) \to \infty$ and

$$g_2(\pi) \to 1 + \frac{1}{\frac{\nu\sigma}{\mu\tau} - \frac{\rho}{\tau}} < \infty.$$

As $\pi \to \pi_1^{**}$ then $g_1(\pi) \to \frac{1}{\pi_1^{**}} < \infty$ and $g_2(\pi) \to \infty$. So the equation (2.27) has a unique root in $(0, \pi_1^{**})$. Uniqueness follows as previously.

For $\pi \in [\pi_1^{**}, \pi_2^{**}]$, $\phi(\pi) = \phi(\pi_1^{**})$, $g_1(\pi) < \infty$ and $g_2(\pi) = g_2(\pi^{**}) = \infty$. On the other hand for $\pi \in (\pi_2^{**}, 1]$, $g_1 \geq 1$ and $\phi(\pi) < \frac{\rho\mu}{\nu\sigma}$, so $g_2(\pi) < 1$. So there are no roots of the equation (2.27) in $[\pi_1^{**}, 1]$. Hence equation (2.27) has a unique root in [0,1]. This completes the proof of Corollary (2.3.1).

We have shown that if R_0 is less than or equal to one then there is only the steady state with no disease present whereas if R_0 exceeds one then there is the DFE and a unique steady state with disease present (denoted the endemic equilibrium (EE)). We shall now explore the local stability of the equilibrium.

2.3.3 Local Stability of Equilibrium

To study the local stability of the equilibrium we consider whether if π is slightly displaced from the equilibrium pointπ^*it will return to it or move away. We can write

$$\frac{d\pi(t)}{dt} = \frac{\phi(\pi(t))(1-\pi(t))\nu\sigma\pi(t)}{\pi(t)\tau + \rho - \pi(t)\rho} - \mu\pi(t) = f(\pi). \tag{2.30}$$

Theorem 2.3.3. *Assume that ϕ is a differentiable function of π. We have shown that*

(i) If $R_0 < 1$ then the solution with no disease to equation (2.4) is locally asymptotically stable.

(ii) If $R_0 = 1$ then the solution with no disease is neutrally stable.

(iii) If $R_0 > 1$ then the solution with no disease is unstable and the unique EE is locally asymptotically stable.

Proof: We can write

$$f(\pi) = \pi\left[\frac{\phi(\pi(t))(1-\pi(t))\nu\sigma}{\pi(t)\tau + \rho - \pi(t)\rho} - \mu\right]. \tag{2.31}$$

$$\begin{aligned}\frac{df}{d\pi} &= \frac{\phi(\pi)(1-\pi)\nu\sigma}{\pi\tau + \rho - \pi\rho} - \mu \\ &+ \pi\left[\frac{\phi'(\pi)(1-\pi)\nu\sigma}{\pi\tau + \rho - \pi\rho} - \frac{\phi(\pi)\nu\sigma}{\pi\tau + \rho - \pi\rho} - \frac{\phi(\pi)(1-\pi)\nu\sigma(\tau-\rho)}{(\pi\tau + \rho - \pi\rho)^2}\right].\end{aligned} \tag{2.32}$$

When $\pi = 0$ we have

$$\begin{aligned} \left.\frac{df}{d\pi}\right|_{\pi=0} &= \frac{\nu\sigma}{\rho} - \mu, \\ &= \mu(R_0 - 1). \end{aligned} \tag{2.33}$$

Hence if $R_0 < 1$ the DFE is locally asymptotically stable. If $R_0 = 1$ then the DFE is neutrally stable. if $R_0 > 1$ then the DFE is unstable.

If $R_0 > 1$ and $\pi = \pi^*$ then

$$\left.\frac{df}{d\pi}\right|_{\pi=\pi^*} = \frac{\phi'(\pi^*)(1-\pi^*)\nu\sigma\pi^*}{\pi^*\tau + \rho - \pi^*\rho} - \frac{\phi(\pi^*)\nu\sigma\pi^*}{\pi^*\tau + \rho - \pi^*\rho} - \frac{\phi(\pi^*)\nu\sigma(\tau-\rho)(1-\pi^*)\pi^*}{(\pi^*\tau + \rho - \pi^*\rho)^2}, \tag{2.34}$$

$$= \left[\frac{\phi'(\pi^*)(1-\pi^*)\nu\sigma\pi^*}{\pi^*\tau + \rho - \pi^*\rho} - \frac{\phi(\pi^*)\nu\sigma\tau\pi^*}{(\pi^*\tau + \rho - \pi^*\rho)^2}\right].$$

As both terms are negative. $\left.\frac{df}{d\pi}\right|_{\pi^*} < 0$ and the unique EE is locally asymptotically stable when it exists. This completes the proof of Theorem 2.3.3. We shall now proceed to look at the global behaviour of the system.

2.3.4 Global Stability of Equilibria

We have shown that there is always a DFE possible which is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$. In the latter case there is a unique the EE which is locally asymptotically stable.

Theorem 2.3.4. *Suppose that $\phi(\pi)$ is monotone decreasing in π. If $\pi(0) = 0$ then $\pi(t) = 0$ for all time. If $R_0 \leq 1$ then the disease will always die out whatever the initial fraction of PWIDs infected, so we have global stability of the DFE. If $R_0 > 1$ and disease is initially present then over a long time the solution to equation (2.4) approaches the unique endemic equilibrium.*

Proof: It is clear that $\pi(0) = 0$ implies that $\pi(t) = 0$ for all time.

(i) Suppose first that R_0 <1 and $\pi(0) > 0$. We will show that $\pi(t) \to 0$ as $t \to \infty$. By using the equation (2.25) $\pi(0) > 0$ implies that $\pi(t) > 0 \ \forall \ t$. Rewrite the

equation (2.23) as

$$\frac{1}{\pi}\frac{d\pi(t)}{dt} = \frac{\phi(\pi(t))(1-\pi(t))\nu\sigma}{\pi(t)\tau + \rho - \pi(t)\rho} - \mu = \frac{\phi(\pi)\nu\sigma}{\frac{\tau\pi}{1-\pi} + \rho} - \mu. \tag{2.35}$$

In the last fraction the numerator is $\phi(\pi)\nu\sigma$ which is decreasing in π and the denominator is monotone increasing in π for $\pi \geq 0$. So writing

$$\begin{aligned} g_3(\pi) &= \frac{\phi(\pi)\nu\sigma}{\frac{\tau\pi}{1-\pi} + \rho} - \mu. \\ g_3(\pi) &\leq g_3(0) = \frac{\nu\sigma}{\rho} - \mu = -\varepsilon < 0. \end{aligned} \tag{2.36}$$

where $\varepsilon > 0$. Hence from equation (2.35)

$$\begin{aligned} \int_0^t \frac{1}{\pi}\frac{d\pi}{dt}dt &\leq \int_0^t (-\varepsilon)dt, \\ \Big[\log \pi\Big]_0^t &\leq -\varepsilon t, \\ \log\left(\frac{\pi(t)}{\pi(0)}\right) &\leq -\varepsilon t. \end{aligned} \tag{2.37}$$

Hence $0 \leq \pi(t) \leq \pi(0)e^{-\varepsilon t}$. Now as $t \to \infty$ then $\pi(0)e^{-\epsilon t} \to 0$, so $\pi \to 0$. Hence the DFE is globally stable for R_0 <1.

(ii) Now we shall consider the case where $R_0 = 1$. Without loss of generality suppose that $\pi(0) > 0$. With the same notation as above note that $g_3(\pi) \leq g_3(0) = 0$. If ϕ is monotone decreasing in π then we assert that $\pi(t) \to 0$ as $t \to \infty$. If $\rho < \tau$ pick $\varepsilon > 0$ such that $\varepsilon \leq \min\left(1, \frac{\rho}{\tau - \rho}\right)$. If τ<ρ pick $\varepsilon < 1$. For

$\pi \geq \varepsilon$, we have

$$\begin{aligned} g_3(\pi) \leq g_3(\varepsilon) &\leq \frac{\nu\sigma}{\frac{\tau\varepsilon}{1-\varepsilon} + \rho} - \mu, \\ &= \frac{\nu\sigma(1-\varepsilon)}{\tau\varepsilon + \rho(1-\varepsilon)} - \mu, \\ &= \frac{\nu\sigma(1-\varepsilon)}{\rho + (\tau-\rho)\varepsilon} - \frac{\nu\sigma}{\rho}, \\ &= \frac{\nu\sigma}{\rho}\frac{[\rho(1-\varepsilon) - [\rho + (\tau-\rho)\varepsilon]]}{(\rho + (\tau-\rho)\varepsilon)}, \\ &= -\frac{\nu\sigma\tau\varepsilon}{\rho(\rho + (\tau-\rho)\varepsilon)}, \\ &\leq -\frac{\nu\sigma\tau\varepsilon}{2\rho^2}, \\ &= -a\varepsilon. \end{aligned} \tag{2.38}$$

Here $a = \frac{\nu\sigma^2}{2\rho^2}$, as $2\rho \geq \rho + (\tau - \rho)\varepsilon$. Hence for $\pi \geq \varepsilon$, $\frac{1}{\pi}\frac{d\pi}{dt} \leq -a\varepsilon$.
So π is monotone decreases and $0 \leq \pi \leq \pi(0)e^{-a\varepsilon t}$. Eventually π decreasing below 2ε, at time t_0 , and as it is monotone decreases for $\pi \in [\varepsilon, 1]$ it cannot rise above 2ε again so $0 \leq \pi(t) \leq 2\varepsilon$ for $t \geq t_0$. But ε can be made arbitrarily small so $\pi(t) \to 0$ as $t \to \infty$.

(iii) Suppose that $R_0 > 1$ and $1 \geq \pi(0) > 0$. We shall consider three cases (*a*) $\pi(0) = \pi^*$, (*b*) $\pi(0) < \pi^*$ and (*c*) $\pi(0) > \pi^*$.

Now we are going to prove Theorem 2.3.4 in these cases, the first one is

(*I*) $\pi(0) = \pi^*$ then it is clear that $\pi(t) \to \pi^*$ as $t \to \infty$.

(*II*) $\pi(0) < \pi^*$ then by the proof of Corollary 2.3.1 case (iii) (all sub-cases) for $0 < \pi < \pi^*$

$$1 + \frac{1}{\frac{\nu\sigma}{\mu\tau}\phi(\pi) - \frac{\rho}{\tau}} < \frac{1}{\pi}.$$

Re-arranging

$$\begin{aligned}\left(\frac{\nu\sigma}{\mu\tau}\phi(\pi)-\frac{\rho}{\tau}\right)(\pi-1) &< -\pi,\\ (\nu\sigma\phi(\pi)-\mu\rho)(1-\pi) &> \mu\tau\pi,\\ \nu\sigma\phi(\pi)(1-\pi) &> \mu[\tau\pi+\rho(1-\pi)],\\ \frac{\nu\sigma\phi(\pi)(1-\pi)}{\tau\pi+\rho(1-\pi)} &> \mu.\end{aligned} \tag{2.39}$$

Hence

$$\frac{\mathrm{d}\pi}{\mathrm{d}t} = \frac{\nu\sigma\phi(\pi)\pi(1-\pi)}{\tau\pi+\rho(1-\pi)} - \mu\pi > 0.$$

Therefore $\pi(t)$ is monotone increasing in t.

If $\pi(t_0) = \pi^*$ for some t_0 then $\pi(t) = \pi^*$ for all $t \geq t_0$ and the result follows.

If $\pi(t) < \pi^*\ \forall\ t$, then $\pi(t)$ is monotone increasing and bounded above, so tends to a limit $\pi_l > \pi(0) > 0$. If $\pi_l = \pi^*$ then we are done.

Suppose that π_l<π^*. Arguing as above

$$\varepsilon = \frac{\nu\sigma(1-\pi_l)\phi(\pi_l)}{\pi_l\tau+\rho(1-\pi_l)} - \mu > 0. \tag{2.40}$$

Recall from earlier that

$$g_3(\pi) = \frac{\nu\sigma\phi(\pi_l)}{\frac{\tau\pi}{1-\pi}+\rho} - \mu.$$

is monotone decreasing in π. Hence for $\pi < \pi_l, g_3(\pi) \geq g_3(\pi_l) = \varepsilon > 0$, so

$$\frac{1}{\pi}\frac{\mathrm{d}\pi}{\mathrm{d}t} \geq \varepsilon > 0.$$

Hence integrating

$$\frac{1}{\pi}\frac{dloge(\pi)}{dt} \geq \varepsilon > 0.$$

So $\pi(t) \geq \pi(0)\ e^{\varepsilon t} \to \infty$ as $t \to \infty$. But that is a contradiction and we are done.

(*III*) The other case is $\pi(0) > \pi^*$. We shall first deal with the case where ϕ

is strictly monotone decreasing (Theorem 2.3.2) and then the case where ϕ is only monotone decreasing (Corollary 2.3.1). For the first case where ϕ is strictly monotone decreasing recall the proof of Theorem 2.3.2 that π^{**} is the unique root of

$$\phi(\pi) = \frac{\rho\mu}{\nu\sigma} \quad in \quad [0,1].$$

Either (*a*) $\pi^* < \pi < \pi^{**}$, (*b*) $\pi = \pi^{**}$ or (*c*) $\pi > \pi^{**}$. By the proof of Theorem 2.3.2 case (3) (all three cases).

If (*a*) or (*b*) is true, then by rearranging and arguing as above we have

$$\begin{aligned} &1 + \frac{1}{\frac{\nu\sigma}{\mu\tau}\phi(\pi) - \frac{\rho}{\tau}} > \frac{1}{\pi}. \\ &\frac{\mathrm{d}\pi}{\mathrm{d}t} = \frac{\nu\sigma\phi(\pi))\pi(1-\pi)}{\pi\tau + \rho(1-\pi)} - \mu\pi < 0. \end{aligned} \tag{2.41}$$

In Case (*c*) we have

$$\frac{1}{\pi} > 1 + \frac{1}{\frac{\nu\sigma}{\mu\tau}\phi(\pi) - \frac{\rho}{\tau}}. \tag{2.42}$$

Arguing as above

$$\begin{aligned} &-\frac{1}{\frac{\nu\sigma}{\mu\tau}\phi(\pi) - \frac{\rho}{\tau}} > 0 > 1 - \frac{1}{\pi} = \frac{\pi - 1}{\pi}. \\ &\left(\frac{\nu\sigma}{\mu\tau}\phi(\pi) - \frac{\rho}{\tau}\right)(\pi - 1) > 0 > -\pi. \\ &(\nu\sigma\phi(\pi) - \mu\rho)(1-\pi) < \mu\tau\pi. \end{aligned} \tag{2.43}$$

So again, arguing as above

$$\frac{\mathrm{d}\pi}{\mathrm{d}t} = \frac{\nu\sigma\phi(\pi)\pi(1-\pi)}{\pi\tau + \rho(1-\pi)} - \mu\pi < 0. \tag{2.44}$$

Hence $\pi(t)$ is monotone decreasing in *t*.

If $\pi(t) = \pi^*$ for some t_0 then $\pi(t) = \pi^*$ for all $t \geq t_0$ so we are done.

If $\pi(t) < \pi^*(t)$ $\forall$ *t*, then $\pi(t)$ is monotone decreasing and bounded below so tends to a limit π_l where $\pi^* \leq \pi_l < \pi(0) \leq 1$. If $\pi_l = \pi^*$ then we are done.

Suppose that $\pi_l > \pi^*$ then arguing as above

$$\varepsilon = \frac{\nu\sigma(1-\pi_l)\phi(\pi_l)}{\pi_l\tau + \rho(1-\pi_l)} - \mu < 0. \tag{2.45}$$

So for $\pi \geq \pi_l$, $g_3(\pi) \leq g_3(\pi_l) < 0$. Then for $\pi > \pi_l$, we have

$$\frac{1}{\pi}\frac{\mathrm{d}\pi}{\mathrm{d}t} \leq \varepsilon < 0.$$

So $0 \leq \pi(t) \leq \pi(0)e^{\varepsilon t}$, hence $\pi(t) \to 0$ as $t \to \infty$, but that is a contradiction as $\pi_l \geq \pi^* > 0$. Hence $\pi_l = \pi*$ and we are done.

We now return to Case (*III*) $\pi(0) > \pi^*$ of the Theorem 2.3.4 where ϕ is just monotone decreasing. In this case from the proof of Corollary 2.3.1, the equation (2.29) has roots in an interval $[\pi_1^{**}, \pi_2^{**}] \subset [0,1]$. Either (*a*) $\pi^* < \pi < \pi_1^{**}$, (*b*) $\pi \in [\pi_1^{**}, \pi_2^{**}]$ or (*c*) $\pi > \pi_2^{**}$ If (*a*) or (*b*) is true, then again we have

$$1 + \frac{1}{\frac{\nu\sigma}{\mu\tau}\phi(\pi) - \frac{\rho}{\tau}} > \frac{1}{\pi}, \tag{2.46}$$

and the proof proceeds as in case (*a*) and (*b*) above. If (*c*) is true then again

$$\frac{1}{\pi} > 1 + \frac{1}{\frac{\nu\sigma}{\mu\tau}\phi(\pi) - \frac{\rho}{\tau}}, \tag{2.47}$$

and the proof proceeds as in cases (c) above, so Theorem 2.3.4 is still true if ϕ is just monotone decreasing. This completes the proof of Theorem 2.3.4.

Hence we have shown that if R_0 is less than or equal to one disease will become extinct whatever the starting value. If there is no disease initially then there will never be any disease. If there is initially disease and $R_0 > 1$, then the solutions will tend to the unique steady state with disease present for a large time. So in particular, limit cycle solutions cannot exist.

Next, we are going to show some numerical simulations and confirm our theoretical analysis results.

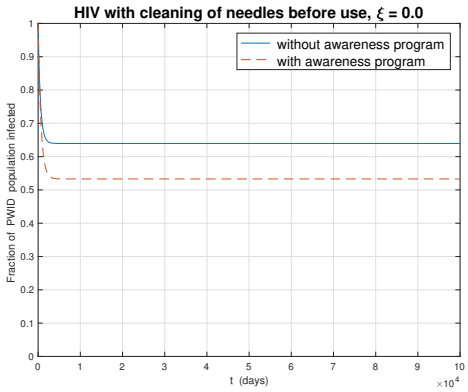
2.3.5 Simulations

We support our analytical results given in Theorems 2.3.1 - 2.3.4 and Corollary 2.3.1 by numerical simulations. Our simulations were performed using MATLAB and the numerical ordinary differential equation solver (ode45). Our computer program was tested using comprehensive output from a large number of runs. Throughout various simulations, we have used realistic parameter values for HIV and HCV amongst PWIDs but our main objective is to verify the analytic results which estimate the spread of HIV amongst PWIDs for model (2.4) with two disease awareness programs. We showed that if $R_0 \leq 1$ then the disease will die out, and if the disease is initially present and $R_0 > 1$ then the disease will tend to the unique endemic equilibrium.

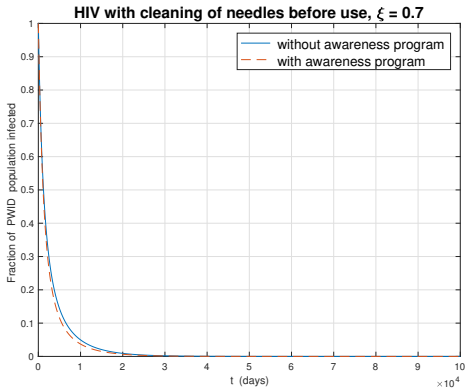
Motivated by the literature (Greenhalgh et al. (2015), Misra et al. (2011), Samanta et al. (2013)) , we take two functional forms for $\phi(\pi)$. The first one is $\phi(\pi) = \left(1 - \frac{a\pi}{b+\pi}\right)$ where a and b are positive constants with $0 \leq a \leq 1$, and the second is $\phi(\pi) = e^{-m_0 n\pi}$ where m_0 is constant and n represents the number of the PWIDs population. An alternative form is

$$\phi(\pi) = e^{-M(t)}, \text{where } M(t) = max\left[0, c\pi + d\frac{\mathrm{d}\pi}{\mathrm{d}t}\right], \tag{2.48}$$

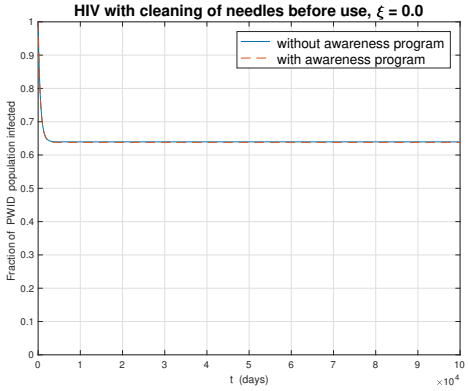
where *c* and *d* are strictly positive constants Misra et al. (2011), but we have not used this. We shall make similar assumptions as in Liang et al. (2016). We shall take $p = 0$ and assume that $\lambda_1 = \lambda_2$ so that all PWIDs visit shooting galleries at the same rate whether or not they are infected. Also, we take ϕ_1, the probability that after a single injection an initially infected PWID leaves uninfected a syringe that was initially infected, and θ_1, the probability that after a single injection an initially infected PWID leaves uninfected a syringe that was initially infected, to be zero as these probabilities are very small and in simple models of the spread of HIV amongst PWIDs these probabilities are normally taken as zero. We choose realistic values for μ, the per capital rate at which addicts leave the sharing injecting population of μ= 0.258/year = 7.0637 $\times 10^{-4}$/day, $P_1 = 0.0$, $P_2 = 0.25$,



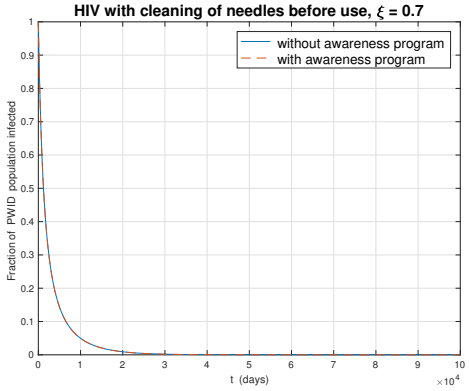
(a) With values of awareness program function parameters $a = 0.9, b = 1$.



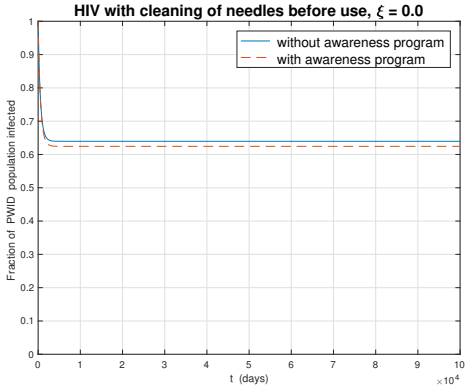
(b) With values of awareness program function parameters $a = 0.9, b = 1$.



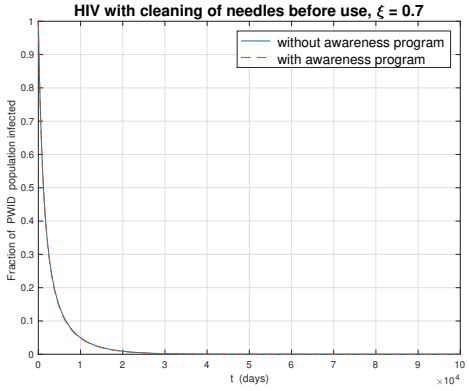
(c) With values of awareness program function parameters $a = 0.1, b = 10$.



(d) With values of awareness program function parameters $a = 0.1, b = 10$.

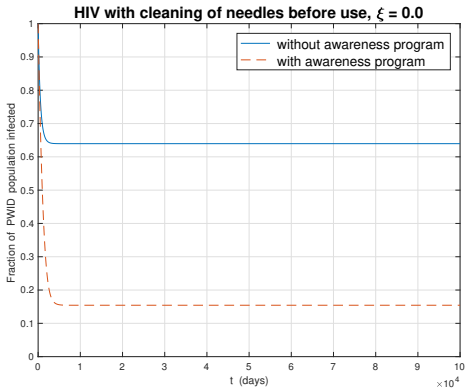


(e) With values of awareness program function parameters $a = 0.5, b = 5$.

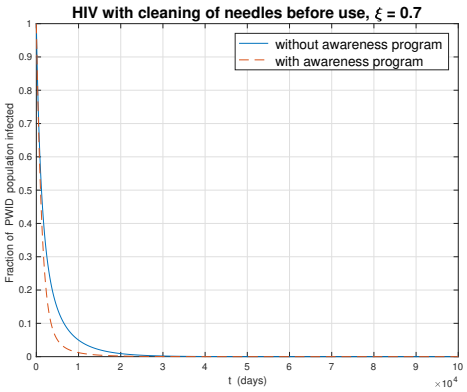


(f) With values of awareness program function parameters $a = 0.5, b = 5$.

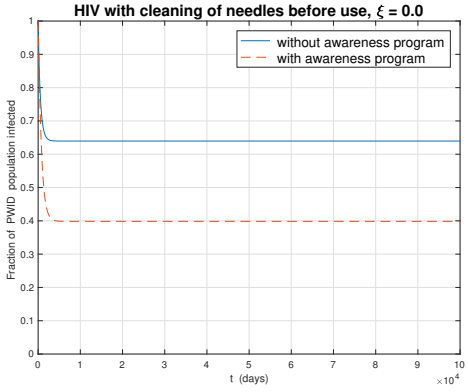
Figure 2.3: The plots of simulations for the solution of model (2.4) with awareness program function $\phi(\pi)=1-\frac{a\pi}{b+\pi}$ and so $\xi = 0.0$ when $R_0 > 1$ and so $\xi = 0.7$ when $R_0 < 1$.



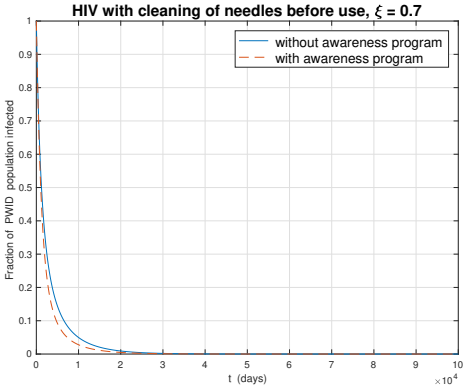
(a) With values of awareness program function parameters $m_0 = 10.0/n$.



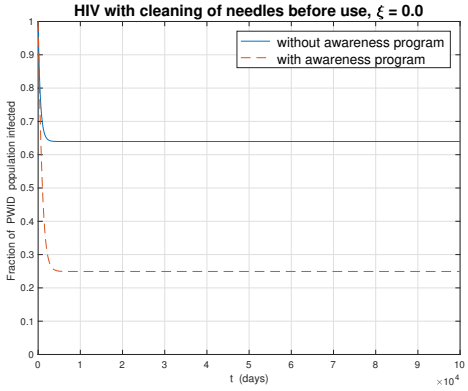
(b) With values of awareness program function parameters $m_0 = 10.0/n$.



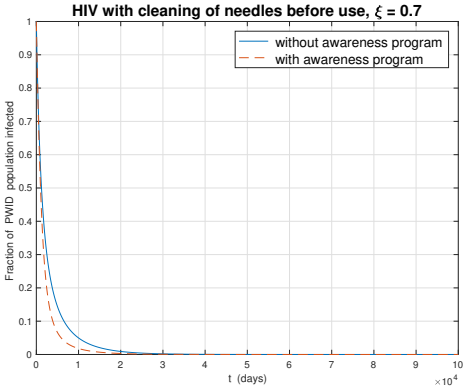
(c) With values of awareness program function parameters $m_0 = 2.0/n$.



(d) With values of awareness program function parameters $m_0 = 2.0/n$.



(e) With values of awareness program function parameters $m_0 = 5.0/n$.



(f) With values of awareness program function parameters $m_0 = 5.0/n$.

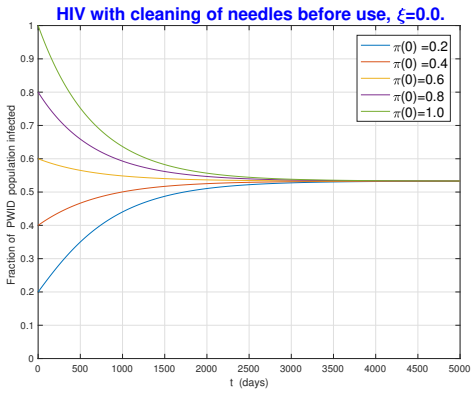
Figure 2.4: The plots of simulations for the solution of model (2.4) with awareness program function $\phi(\pi) = e^{-m_0 n \pi}$, where $n = 1000$ and when $\xi = 0.0$ so $R_0 > 1$ and so $\xi = 0.7$ then $R_0 < 1$.

$P_3 = 0.01$, $P_4 = 0.74$, $\lambda_1 = \lambda_2$= 0.143/day and $\gamma = 1$ (based on Liang et al. (2016)) and varying values of the needle cleaning probability ξ with $0 \leq \xi \leq 1$.

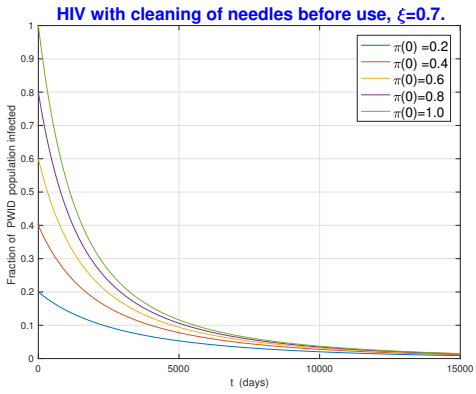
We studied the behaviour of the model of equation (2.4) through altering R_0 by choosing different values of ξ. In all cases, the starting value was initially $\pi(0) = 1$. Figure 2.3 shows plots of six simulations with disease awareness program $\phi(\pi)$=$1 - \frac{a\pi}{b+\pi}$ (taken from Li et al. (2008a)), with different values of the constants a and b constant are shown in Figure 2.3. In the sub-figures 2.3a , 2.3c and 2.3e of Figure 2.3 where $R_0 > 1$ we choose $\xi = 0.0$, then from equations (2.14) and (2.18) we have σ= 0.143/day, τ= 0.143/day, ρ = 0.0358/day and ν= 0.0014/day giving R_0 = 8.0977 . For the other sub-figures 2.3b, 2.3d and 2.3f of Figure 2.3 where $R_0 < 1$ we choose ξ= 0.7 then from equations (2.14) we have σ =0.143/day, τ= 0.143/day, ρ= 0.1108/day and ν = 0.000429/day giving R_0 =0.7838 . Figure 2.4 shows plots of six simulations with the disease awareness program $\phi(\pi)$= $e^{-m_0 n\pi}$ (taken from Cui et al. (2008)), with different values of m_0. Similarly to the results of Figure 2.3 we have that $R_0 > 1$ for sub-figures 2.4a, 2.4c and 2.4e of Figure 2.4 and $R_0 < 1$ for sub-figures 2.4b 2.4d and 2.4f of Figure 2.4.

In Figure 2.5 by using both the disease awareness program functions with the same parameters in the sub-figures 2.3a, 2.3b, 2.4a and 2.4b of Figures 2.3 and 2.4, we considered five different initial values $\pi(0) \in$[0,1] of the infected PWID population who do not clean their needles before use. The cases with $\xi = 0.0$ are given in the sub-figure in 2.5a and 2.5c with the same results as previously, we observed that if the PWID population do not clean their needles before use ($\xi = 0.0$) then $R_0 > 1$ and this means that over a long time the fraction of PWID population which was HIV infected tended to the unique endemic equilibrium. For the other two cases (given in the sub-figures 2.5b and 2.5d) with $\xi = 0.7$, the PWIDs often cleaned their needles successfully before use, so $R_0 < 1$, then the HIV virus died out after a long period of time in both PWIDs and needles.

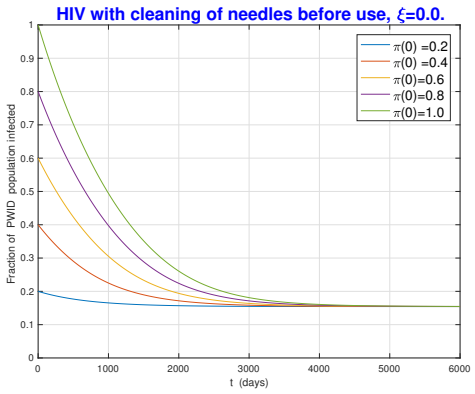
We did other simulations with a variety of other starting values and a variety of



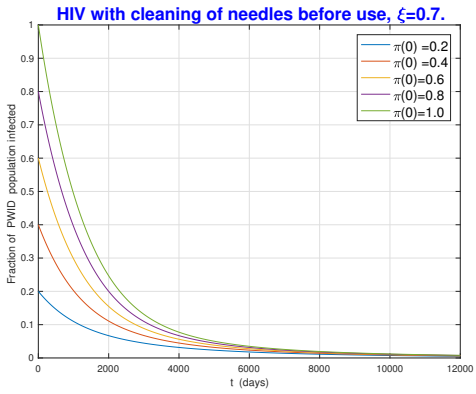
(a) With values of awareness program function parameters $a = 0.9, b = 1$.



(b) With values of awareness program function parameters $a = 0.9, b = 1$.



(c) With values of awareness program function parameters $m_0 = 10.0/n$.



(d) With values of awareness program function parameters $m_0 = 10.0/n$.

Figure 2.5: The plots of simulations for the solution of model (2.4) with disease awareness program function $\phi(\pi)=1-\frac{a\pi}{b+\pi}$ for sub-figures (*a*) and (*b*), for sub-figures (*c*) and (*d*) with several different starting values of $\pi(0)$ the disease awareness program function $\phi(\pi)= e^{-m_0 n\pi}$, where $n = 1000$.

other model parameters. In each case, the results of Theorems 2.3.1-2.3.4 and Corollary 2.3.1 were verified. For $R_0 \leq 1$ the disease always dies out whatever the starting values, whereas for $R_0 > 1$ and disease initially presents the disease tends to a unique endemic equilibrium.

We have performed an equilibrium and stability analysis for this model. Our discussion has been focused on two ways of studying the effect of awareness programs in disease transmission models. The key biological parameter of our model is the primary reproductive number R_0. We find that there is a critical threshold parameter $R_0 = 1$, which determines the behaviour of the model. We have shown that the system has a unique equilibrium solution. We have shown that if $R_0 \leq 1$,

then the disease-free equilibrium is globally asymptotically stable, so whatever the initial fraction of infected individuals, the disease will die out as time becomes large. If no disease is initially present, there will never be any disease. If $R_0 > 1$, there is the disease-free equilibrium and, additionally, a unique endemic equilibrium. If there is disease initially present and $R_0 > 1$, then the system tends to the unique endemic equilibrium. We also showed that the disease-free equilibrium is locally asymptotically stable if $R_0 < 1$, neutrally stable if $R_0 = 1$ and unstable if $R_0 > 1$. In the case that $R_0 > 1$ we showed that the endemic equilibrium was locally asymptotically stable too. Our analytical results are confirmed by using simulation with realistic parameter values.

2.4 Adapting Our Model to Deal with HCV Amongst PWIDs

2.4.1 Description of the Model Dealing with HCV

Another disease spread by sharing infected needles is the Hepatitis C virus (HCV) amongst PWIDs. The model is almost the same as for HIV except that for HCV there is now treatment so infected individuals can recover from HCV. If δ denotes the per capita rate at which an HCV-infected individual is treated and recovers, then using the same notation as for HIV, let $\pi(t)$ denote the fraction of PWIDs infected with HCV and $\beta(t)$ denote the fraction of needles infected with HCV. Then equations (2.2) and (2.3) become

$$\frac{d\pi}{dt} = \phi(\pi)(1-\pi)\nu\beta - (\mu+\delta)\pi, \tag{2.49}$$

$$\frac{d\beta}{dt} = \phi(\pi)\pi(\sigma - \tau\beta) - \phi(\pi)(1-\pi)\rho\beta, \tag{2.50}$$

and the corresponding version of equation (2.4)

$$\frac{d\pi(t)}{dt} = \frac{\phi(\pi(t))(1-\pi(t))\nu\sigma\pi(t)}{\pi(t)\tau + \rho - \pi(t)\rho} - (\mu+\delta)\pi(t). \tag{2.51}$$

So the model is the same as equation (2.4) with μ replaced by $(\mu + \delta)$. So the basic reproduction number is

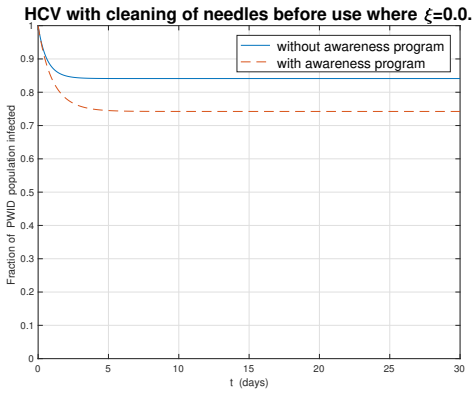
$$R_0 = \frac{\nu\sigma}{\rho(\mu + \delta)}. \tag{2.52}$$

and with this value of R_0 Theorems 2.3.1 - 2.3.4 and Corollary 2.3.1 (all of the theorems) shill hold. This is a very simplified approximate model of HCV as it assumes that infected individuals do not spontaneously recover and become susceptible again apart from treatment and also ignores the short, highly infectious acute phase Corson et al. (2012) but nonetheless, it can still be regarded as a very simple approximation of the spread of HCV amongst PWIDs.

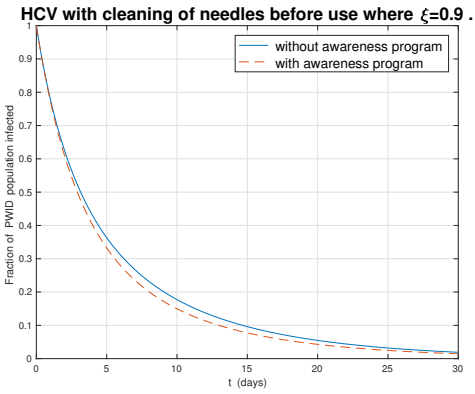
2.4.2 Numerical Simulations

We have also performed simulations for the model (2.50) for the spread of HCV amongst PWIDs. We used parameter values taken from Corson et al. (2012). Recall that $P_1 + P_2$, the probability that when an initially susceptible PWID injects with an initially infected needle, the needle is flushed (i.e. flushed of infectious blood and left uninfectious after use). In Corson et al. (2012), the probability that a needle is flushed in this situation is taken as one similarly in the model of Corson et al. (2012) $P_1 + P_3 = \alpha$, the probability that when an initially susceptible PWID injects with an initially infected needle, then α, the average probability that the PWID becomes infected is $\alpha = P_1 + P_3 = 0.0165$. α is calculated as a weighted average of the corresponding probabilities for acutely infected and chronically infected PWIDs α_h and α_y in Corson et al. (2012) by weighting each of these probabilities by the average time that a newly infected PWID spends in each of these states at the endemic equilibrium.

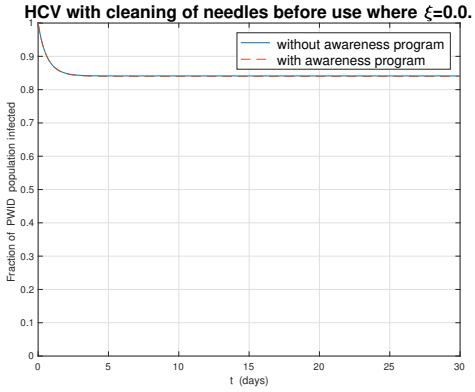
These probabilities are are $\alpha_h = 0.0432$, α_y=0.016. Also as $P_1 + P_2 + P_3 + P_4 = 1$ and P_1+$P_2 = 1$, we must have $P_3 = P_4 = 0$ and then as P_1+$P_3 = 0.0165$, $P_1 = 0.0165$ and $P_2 = 0.9835$. And also λ_1 =λ_2=103/year, μ = 0.17/year and δ=0.1/year. As in



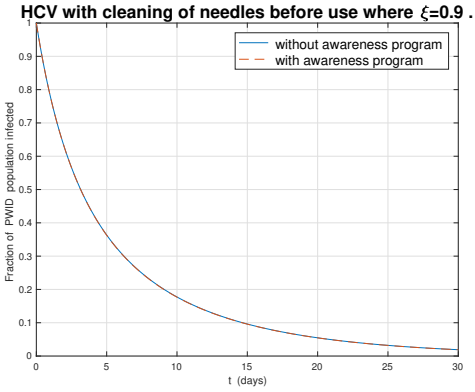
(a) With values of awareness program function parameters $a = 0.9, b = 1$.



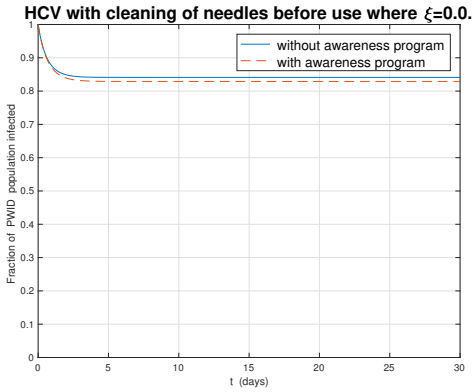
(b) With values of awareness program function parameters $a = 0.9, b = 1$.



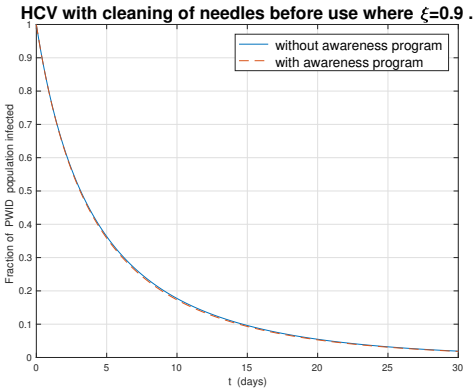
(c) With values of awareness program function parameters $a = 0.1, b = 10$.



(d) With values of awareness program function parameters $a = 0.1, b = 10$.

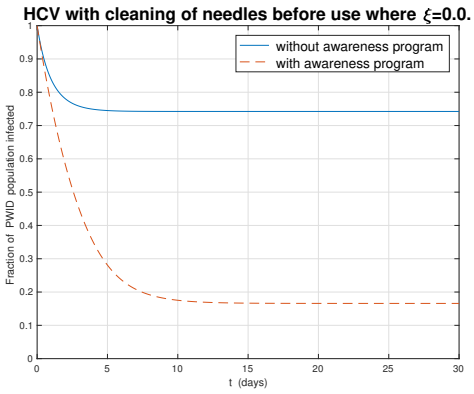


(e) With values of awareness program function parameters $a = 0.5, b = 5$.

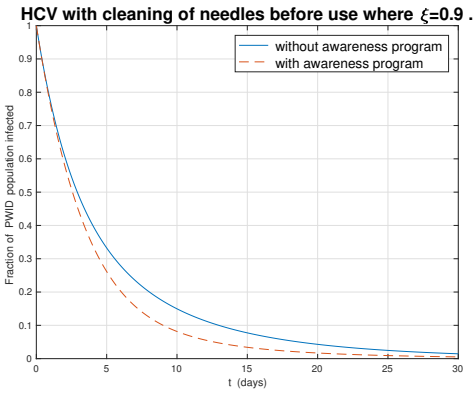


(f) With values of awareness program function parameters $a = 0.5, b = 5$.

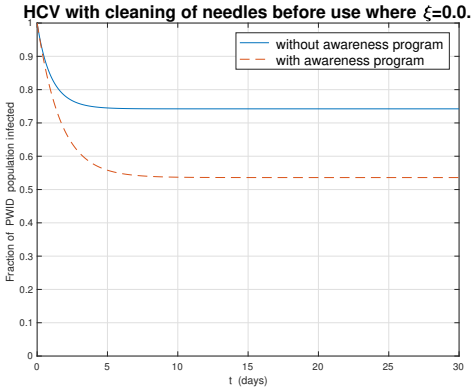
Figure 2.6: The plots of simulations for the solution of model (2.50) with awareness program function $\phi(\pi)$=$1 - \frac{a\pi}{b+\pi}$ and when $\xi = 0.0$ so $R_0 > 1$ and when $\xi = 0.9$ so $R_0 < 1$.



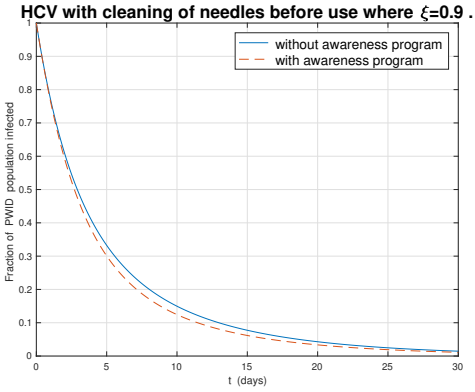
(a) With values of awareness program function parameters $m_0 = 10.0/n$.



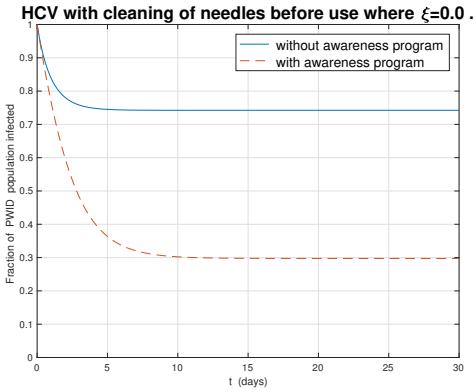
(b) With values of awareness program function parameters $m_0 = 10.0/n$.



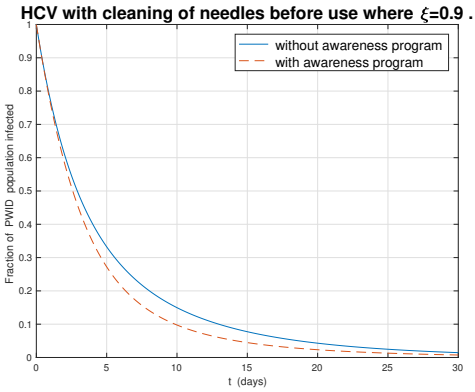
(c) With values of awareness program function parameters $m_0 = 2.0/n$.



(d) With values of awareness program function parameters $m_0 = 2.0/n$.

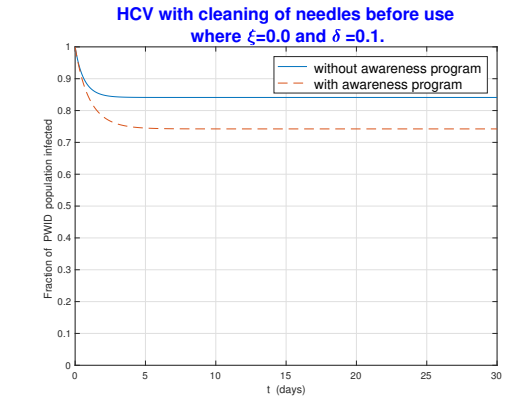


(e) With values of awareness program function parameters $m_0 = 5.0/n$.

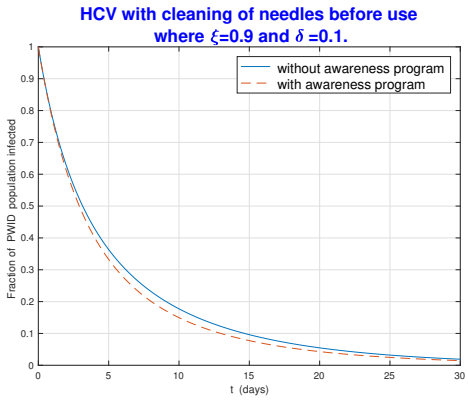


(f) With values of awareness program function parameters $m_0 = 5.0/n$.

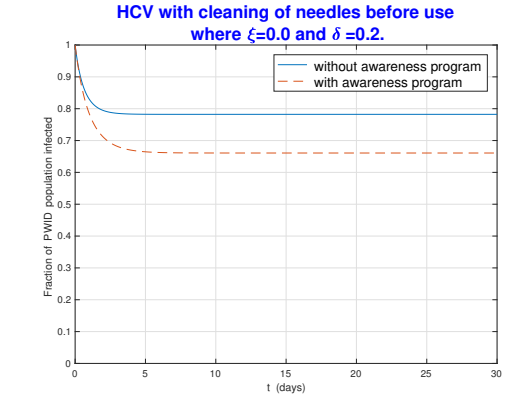
Figure 2.7: The plots of simulations for the solution of model (2.50) with awareness program function $\phi(\pi) = e^{-m_0 n \pi}$ and when $\xi = 0.0$ so $R_0 > 1$ and when $\xi = 0.9$ so $R_0 < 1$.



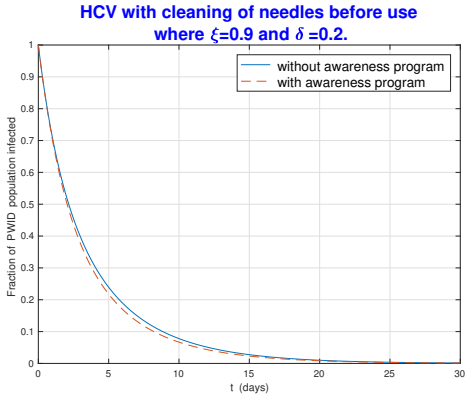
(a)



(b)



(c)



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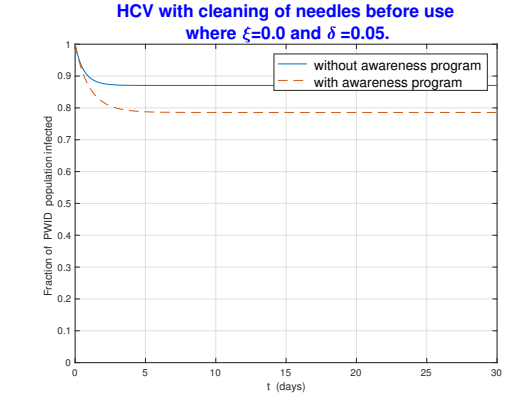

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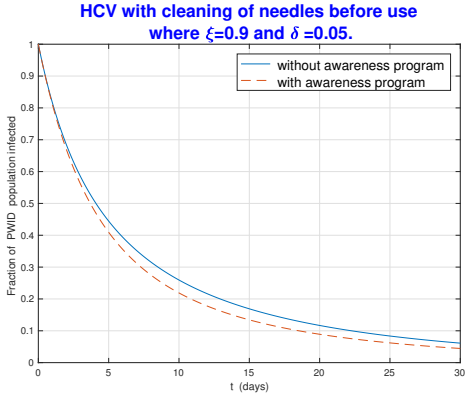
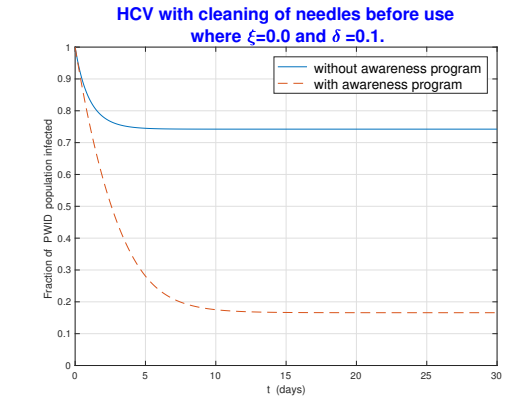
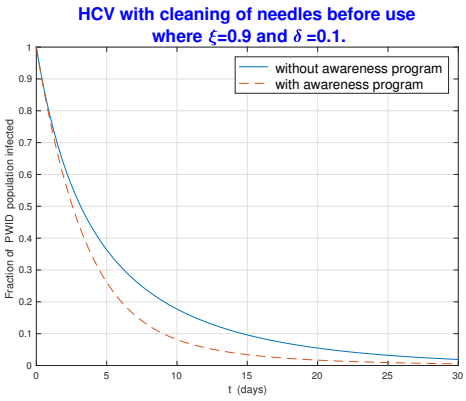


(f)

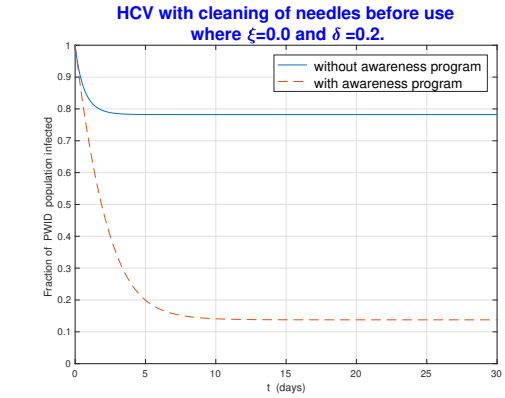
Figure 2.8: The plots of simulations for the solution of model (2.50) with values of awareness program function parameters $a = 0.9, b = 1$. $\phi(\pi)$=1 $- \frac{a\pi}{b+\pi}$ and when $\xi = 0.0$ then $R_0 > 1$ and when $\xi = 0.9$ then $R_0 < 1$.



(a)



(b)



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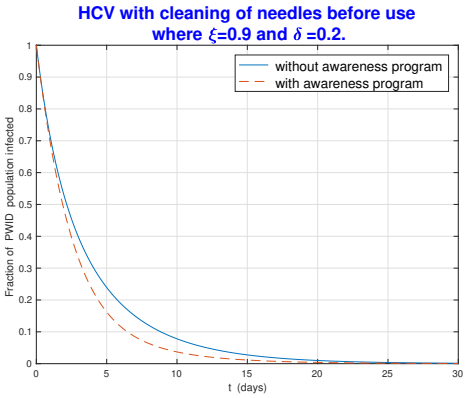

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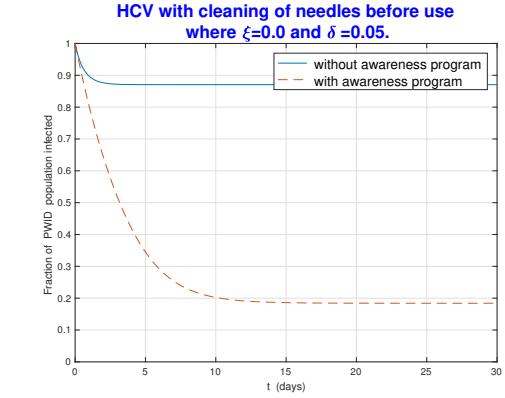
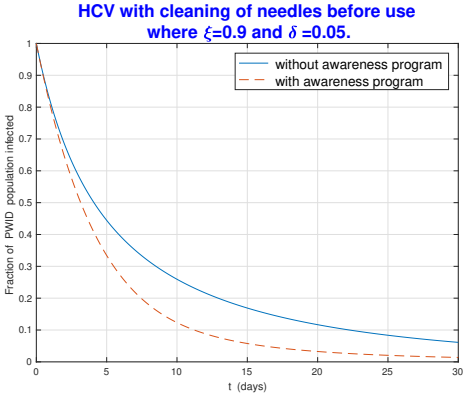


(e)



(f)

Figure 2.9: The plots of simulations for the solution of model (2.50) with values of awareness program function parameters $m_0 = 10.0/n$. $\phi(\pi)$= $e^{-m_0 n\pi}$ where $n = 1000$ and when $\xi = 0.0$ so $R_0 > 1$ and when $\xi = 0.9$ then $R_0 < 1$.

the HIV transmission model, we take $\phi_1 = \theta_1 = 0$ for our simulation and again we take $\gamma = 1$. Again we altered the values of R_0 by choosing different values of ξ. Again in the displayed simulation, the starting value was again $\pi(0) = 1$.

Figure 2.6 shows plots of six simulations with disease awareness program $\phi(\pi)$=$1 - \frac{a\pi}{b+\pi}$ with different values of parameters a and b. For sub-figures 2.6a ,2.6c and 2.6e of Figure 2.6 we choose $\xi = 0.0$ then we have σ =103/year, τ =103/year, ρ =103/year and ν=1.6995/year giving R_0 =6.2944. For the other sub-figures 2.6b, 2.6d and 2.6f we choose $\xi = 0.9$ Then we have σ =103/year, τ=103/year, ρ =103/year and ν =0.16995/year giving R_0 =0.6294. Figure 2.7 shows plots of six simulations with the disease awareness program $\phi(\pi)$= $e^{-m_0 n\pi}$ with three different values of m_0 where $n = 1000$. Similar results were obtained as are shown in Figure 2.6 where $R_0 > 1$ for sub-figures 2.7a, 2.7c and 2.7e of Figure 2.7 and $R_0 < 1$ for sub-figures 2.7b,2.7d and 2.7f of Figure 2.7.

In Figures 2.8 and 2.9 we repeated the simulations in Figures 2.6 and 2.7, respectively, with different values of δ and with the same values of the constants of the awareness program functions, for all sub-figures. In the Figure 2.8 we use the values $a = 0.9$ and $b = 1$ with disease awareness program $\phi(\pi)$=$1 - \frac{a\pi}{b+\pi}$. we choose $\xi = 0.0$ for sub-figures 2.8a ,2.8c and 2.8e of Figure 2.8 then we have σ =103/year, τ =103/year, ρ =103/year and ν =1.6995/year. We have different values of R_0 in each time that δ is changed. So we had $R_0 = 6.2944$ when δ=0.1/year in the sub-figure 2.8a, $R_0 = 4.5932$ when δ=0.2/year in 2.8c and for the sub-figure 2.8e we have R_0 =7.7249 when δ=0.05/year. For the rest of the sub-figures 2.8b, 2.8d and 2.8f in Figure 2.8 we we choose $\xi = 0.9$ Then we have σ =103/year, τ =103/year, ρ =103/year and ν =0.1699/year. Then we obtain values of R_0 as in the sub-figure 2.8b R_0 =0.6294 when δ=0.1000/year, in the sub-figure 2.8d, R_0 =0.4593 when δ=0.2/year and for the sub-figure 2.8f we have R_0 =0.7725 when δ=0.05/year.

In Figure 2.9 shows the plots of six simulations with the disease awareness

program $\phi(\pi)= e^{-m_0 n\pi}$ with the fixed values of $m_0 = 10.0/n$. Again here we obtained the same values of the parameters σ,τ,ρ and ν were discussed in Figure 2.8 for all the sub-figures in Figure 2.9. In both figures $R_0 > 1$ for sub-figures 2.8a, 2.8c and 2.8e (2.9a,2.9c and 2.9e) and $R_0 < 1$ for sub-figures 2.8b,2.8d and 2.8f (2.9b, 2.9d and 2.9f).

Again the simulations show that the disease died out if $R_0 \leq 1$ and that the disease tended to the unique endemic equilibrium if $R_0 > 1$ and disease is initially present. This supports the analytical results.

2.5 Conclusion

In this chapter, we have developed a mathematical model of the effect of disease awareness programs on the prevalence of HIV amongst PWIDs, building on the models developed by Greenhalgh and Hay (1997) and Liang et al. (2016). A system of differential equations has been deduced to describe the improved model that reduces the spread of the diseases through the effect of awareness of the disease on sharing needles and syringes amongst the PWID population.

The model differs from the original model that it is based on as it is a one-dimensional model that includes the factor of the awareness program that has the effect of reducing the spread of HIV amongst the population. A system of differential equations has been deduced to describe the improved model that reduces the spread of the diseases through the effect of awareness of the disease on sharing needles and syringes amongst the PWID population, as a result, we obtained a new definition for the parameter of the basic reproduction number R_0 that gives us new results for the analytical and numerical solutions.

We performed numerical simulation on the equation (2.4), describing the effect of awareness programs on reducing the spread of HIV amongst PWIDs. We started off with realistic parameters taken from a literature review, and we as-

sumed that the visiting rate of the shooting gallery is the same ($\lambda_1 = \lambda_2$) for both susceptible PWIDs and the PWIDs infected PWIDs whether or not they know that they are infected. The simulations were divided to simulate two disease awareness programs by changing the constants in these awareness programs for each one. Also, we calculated the result of the basic reproductive number we simulated the total proportion of the PWID population infected over time. The was calculated for different values of the basic reproduction number R_0, which was changed by altering (ξ), the fraction of PWIDs (susceptible or not) who successfully clean their needles before use. We repeated this simulation for both awareness program equations with different initial values of the fraction of PWIDs.

At the end of this chapter, we modified our model to deal with the spread of HCV amongst PWIDs where an HCV-infected individual is treated and recovered. The resulting model was described in differential equation (2.51). This model was used to describe the spread of HCV amongst PWIDs. We kept the same parameter values used in the previous simulation with fixed values of the per capita treatment and recovery rate (δ) for both awareness program functions. We also repeated the simulation with different values of (δ). The basic reproduction number R_0 decreased as the per capita HCV treatment, and recovery rate (δ) increased. Again the simulations confirmed our analytical results if $R_0 \leq 1$ HCV will eventually die out in both PWIDs and needles and if $R_0 > 1$ and disease is initially present. The system will tend to the unique endemic equilibrium.

This concludes our analysis of the one-dimensional system given by (2.4). However, recall that equation (2.4) was obtained as an approximation of a more realistic two-dimensional model (2.2) and (2.3) by realistically assuming that the timescale on which PWIDs inject is short compared with the timescale for epidemiological changes. In the next section, we shall analyse the behaviour of the more realistic two- dimensional model (2.2) and (2.3) (with σ, τ, ρ and ν) given by (2.14).

Chapter 3

Incorporation of Awareness Programs into a Two-dimensional model of the spread of HIV/AIDS amongst People who Inject Drugs

3.1 Analysis of the Two-Dimensional Model

3.1.1 Introduction

Inspired by the model constructed in the previous chapter, in this chapter, we will study the effect of awareness programs in the full two-dimensional model of the spread of HIV amongst PWIDs (2.2) and (2.3) discussed in the last chapter. The results we will obtain and the techniques used to follow the structure of the previous chapter. First of all, we study the existence of a unique non-negative solution to differential equations. Then we look at the existence of an equilibrium solution. After that, we examine the local and global stability of equilibrium. Finally, we do some simulations to confirm the analytical results.

3.1.2 Existence of Unique Non-negative Solution

Recall equations (2.2) and (2.3). $\beta(t)$ represents the fraction of needles infected with HIV at time t and $\pi(t)$ represents the fraction of PWIDs infected with HIV at time t . The biologically feasible region of solutions to this system is $D = \{(\pi, \beta) \in [0,1] \times [0,1]\}$ in R^2. Equations (2.2) and (2.3) are

$$\frac{d\pi}{dt} = \phi(\pi)(1-\pi)\nu\beta - \mu\pi, \tag{3.1}$$

$$\frac{d\beta}{dt} = \phi(\pi)\pi(\sigma - \tau\beta) - \phi(\pi)(1-\pi)\rho\beta. \tag{3.2}$$

We apply similar techniques as have been used to show the existence of a unique non-negative solution in Subsection (2.3.1) in Chapter 2. We show firstly that the right-hand sides of equations (3.1) and (3.2) are Lipschitz continuous and then use the Picard–Lindelöf Theorem to show the existence of a unique continuous solution.

Theorem 3.1.1. *Suppose that ϕ is Lipschitz continuous in π for $0 \leq \pi \leq 1$. For any given initial value $(\pi(0), \beta(0)) = (\pi_0, \beta_0) \in [0,1] \times [0,1]$ the two-dimensional model for the spread of HIV amongst PWIDs given by (3.1) and (3.2) has a unique non-negative solution $(\pi(t), \beta(t)) \in [0,1] \times [0,1]$ moreover.*
(i) If (π_0, β_0) = (0,0) then $(\pi(t), \beta(t))$ = (0,0) for all $t \geq 0$.
(ii) If either $\pi_0 > 0$ or $\beta_0 > 0$ then $(\pi(t), \beta(t)) \in (0,1) \times (0,1)$ for all $t \geq 0$.

Proof: We have already stated the definition of Lipschitz continuity and the Picard–Lindelöf Theorem in Subsection (2.3.1) of the previous chapter, Write $\underline{\mathbf{x}} = (\pi_1, \beta_1)$ We write equations (3.1) and (3.2) as

$$\frac{d\pi}{dt} = f_1(\underline{\mathbf{x}})$$

$$\frac{d\beta}{dt} = f_2(\underline{\mathbf{x}})$$

Write

$$f(\mathbf{x}) = \begin{pmatrix} f_1(\mathbf{x}) \\ \\ f_2(\mathbf{x}) \end{pmatrix}. \tag{3.3}$$

The first stage is to show Lipschitz continuity of the right-hand side of equations (3.1) and (3.2). We need to show that for any $\mathbf{x} = (\pi_x, \beta_x) \in D$, $\mathbf{y} = (\pi_y, \beta_y) \in D$

$$\Big\|f(x) - f(y)\Big\| \leq L\Big\|x - y\Big\|. \tag{3.4}$$

For some constant L where $\|.\|$ denotes the Euclidean norm in R^2. We split the proof into two parts, firstly

1. $\Big|f_1(x) - f_1(y)\Big| \leq L_1\Big|x - y\Big|,$
2. $\Big|f_2(x) - f_2(y)\Big| \leq L_2\Big|x - y\Big|.$

where L_1 and L_2 are Lipschitz constants for f_1 and f_2 respectively. Note that as ϕ is Lipschitz continuous on $[0, 1]$.

$$\Big|\phi(\pi_x) - \phi(\pi_y)\Big| \leq K_1\Big|\pi_x - \pi_y\Big|.$$

For some constant $K_1 \geq 0$ for any $\pi_x \in [0, 1], \pi_y \in [0, 1]$. We start off with the first part

$$\begin{aligned} \Big|f_1(\pi_x, \beta_x) - f_1(\pi_y, \beta_y)\Big| =& \Big|[\phi(\pi_x)(1 - \pi_x)\nu\beta_x - \mu\pi_x] - [\phi(\pi_y)(1 - \pi_y)\nu\beta_y - \mu\pi_y]\Big|. \\ \leq& \Big|\phi(\pi_x)(1 - \pi_x)\nu\beta_x - \phi(\pi_y)(1 - \pi_y)\nu\beta_y\Big| + \mu\Big|\pi_x - \pi_y\Big|. \end{aligned} \tag{3.5}$$

By using the triangle inequality

$$\begin{aligned} \leq& |\phi(\pi_x)(1 - \pi_x)\nu\beta_x - \phi(\pi_y)(1 - \pi_x)\nu\beta_x \\ &+ \phi(\pi_y)(1 - \pi_x)\nu\beta_x - \phi(\pi_y)(1 - \pi_y)\nu\beta_x \\ &+ \phi(\pi_y)(1 - \pi_y)\nu\beta_x - \phi(\pi_y)(1 - \pi_y)\nu\beta_y| + \mu|\pi_x - \pi_y|. \\ \leq& |\phi(\pi_x)(1 - \pi_x)\nu\beta_x - \phi(\pi_y)(1 - \pi_x)\nu\beta_x| \\ &+ |\phi(\pi_y)(1 - \pi_x)\nu\beta_x - \phi(\pi_y)(1 - \pi_y)\nu\beta_x| \\ &+ |\phi(\pi_y)(1 - \pi_y)\nu\beta_x - \phi(\pi_y)(1 - \pi_y)\nu\beta_y| + \mu|\pi_x - \pi_y|. \end{aligned} \tag{3.6}$$

By using the triangle inequality again

$$\begin{aligned} &\leq |\phi(\pi_x) - \phi(\pi_y)|\nu(1-\pi_x)\beta_x + \phi(\pi_y)|\pi_x - \pi_y|\nu\beta_x \\ &\quad + |\phi(\pi_y)|(1-\pi_y)\nu|\beta_x - \beta_y| + \mu|\pi_x - \pi_y|. \end{aligned} \tag{3.7}$$

this implies that

$$\leq K_1\nu|\pi_x - \pi_y| + \nu|\pi_x - \pi_y| + \nu|\beta_x - \beta_y| + \mu|\pi_x - \pi_y|. \tag{3.8}$$

as ϕ is Lipschitz continuous function, then

$$\leq L_1\Big\|x_1 - y_1\Big\|.$$

Here $L_1 = (K_1 + 2)\nu + \mu$. This completes the proof of the first part.

Similarly, we prove the second part using the definition (3.4). We have

$$\begin{aligned} &\Big|f_2(\pi_x, \beta_x) - f_2(\pi_y, \beta_y)\Big| \\ &\qquad = \Big|\Big[\phi(\pi_x)(\sigma - \tau\beta_x) - \phi(\pi_x)(1-\pi_x)\rho\beta_x] \\ &\qquad\quad - [\phi(\pi_y)(\sigma - \tau\beta_y) - \phi(\pi_y)(1-\pi_y)\rho\beta_y\Big]\Big|. \\ &\qquad \leq \Big|\phi(\pi_x)(\sigma - \tau\beta_x) - \phi(\pi_y)(\sigma - \tau\beta_y)\Big| \\ &\qquad\quad + \rho\Big|\phi(\pi_x)(1-\pi_x)\beta_x - \phi(\pi_y)(1-\pi_y)\beta_y\Big|. \end{aligned} \tag{3.9}$$

After applying the triangle inequality, we got

$$\begin{aligned} &\leq \sigma\Big|\phi(\pi_x) - \phi(\pi_y)\Big| + \tau\Big|\phi(\pi_x)\beta_x - \phi(\pi_y)\beta_y\Big| \\ &\quad + \rho\Big|\phi(\pi_x)(1-\pi_x)\beta_x - \phi(\pi_y)(1-\pi_x)\beta_x \\ &\quad + \phi(\pi_y)(1-\pi_x)\beta_x - \phi(\pi_y)(1-\pi_y)\beta_x \\ &\quad + \phi(\pi_y)(1-\pi_y)\beta_x - \phi(\pi_y)(1-\pi_y)\beta_y\Big| \\ &\leq \sigma K_1\Big|\pi_x - \pi_y\Big| \\ &\quad + \tau\Big|\phi(\pi_x)\beta_x - \phi(\pi_y)\beta_x + \phi(\pi_y)\beta_x - \phi(\pi_y)\beta_y\Big| \\ &\quad + \rho\Big|\phi(\pi_x) - \phi(\pi_y)\Big|(1-\pi_x)\beta_x \\ &\quad + \rho\phi(\pi_y)\beta_x\Big|\pi_x - \pi_y\Big| + \rho\phi(\pi_y)(1-\pi_y)\Big|\beta_x - \beta_y\Big| \end{aligned} \tag{3.10}$$

Using the triangle inequality and Lipschitz continuity of ϕ

$$\begin{aligned} &\leq \sigma K_1\Big|\pi_x - \pi_y\Big| + \tau\beta_x\Big|\phi(\pi_x) - \phi(\pi_y)\Big| + \tau\phi(\pi_y)\Big|\beta_x - \beta_y\Big| \\ &\quad + \rho K_1\Big|\pi_x - \pi_y\Big| + \rho\Big|\pi_x - \pi_y\Big| + \rho\Big|\beta_x - \beta_y\Big| \end{aligned} \tag{3.11}$$

Similarity

$$\begin{aligned} &\leq \Big((\sigma + \rho + \tau)k_1 + 2\rho\Big)\Big\|x - y\Big\| + \tau\Big|\beta_x - \beta_y\Big|. \\ &\leq L_2\Big\|x - y\Big\|. \end{aligned} \tag{3.12}$$

Here $L_2 = (\sigma + \rho + \tau)k_1 + 2\rho + \tau$.

This completes the proof of the Lipschitz continuity of the right-hand sides of the system (3.1) and (3.2). We now complete the existence and uniqueness proof.

Case One: $\pi(0) = \beta(0) = 0$.

In this case, we can see that $\pi(t) = \beta(t) = 0$ is a solution for all time. By using the Picard–Lindelöf Theorem, there exists $\Delta t > 0$ such that the equation has a unique local solution in $[0, \Delta t]$. Let $[0, \tau_e)$ be the maximum interval where a unique solution exists with $\pi(\xi) = \beta(\xi) = 0$ for all ξ in $[0, \tau_e)$. We must have $\tau_e \geq \Delta t > 0$. Suppose that $\tau_e < \infty$. Then $\pi(t) = \beta(t) = 0$ for all $t < \tau_e$. Again by using the Picard–Lindelöf Theorem, there exists a unique local solution in $[\tau_e - \eta, \tau_e + \eta]$ for some $\eta > 0$. Hence a solution exists with $\pi(\xi) = \beta(\xi) = 0$ in $[0, \tau_e + \eta]$. This contradicts the definition of τ_e. So $\tau_e = \infty$ and there is a unique solution $\pi(t) = \beta(t) = 0$ for all $t \leq 0$. This completes the proof of the first part of Theorem (3.1.1).

Case Two: $\pi(0) > 0$ or $\beta(0) > 0$.

We shall divide the proof into three cases. First of all, we assume that $\phi > 0$, $\forall\ \pi \in [0, 1]$ and $\sigma > 0$. We can write $\psi = 1 - \pi$ and $\chi = 1 - \beta$. Then the equations (3.1) and (3.2) become as

$$\frac{d\psi}{dt} = \mu(1 - \psi) - \phi(\pi)\psi\nu\beta, \tag{3.13}$$

$$\frac{d\chi}{dt} = \phi(\pi)\psi\rho\beta + \phi(\pi)\pi(\tau - \sigma) - \pi\phi(\pi)\tau\chi. \tag{3.14}$$

Define $(0, \tau_e)$ to be the maximum interval where a unique $(\pi(t), \beta(t))$ to the system equations(3.1) and (3.2) exists for $t \in [0, \tau_e)$ with $(\pi(t), \beta(t)) \in (0,1) \times (0,1)$ for $t \in (0, \tau_e)$.

Lemma 3.1.1. *There exists* $k_1 > 0, k_2 > 0$ *and* $\Delta t > 0$ *such that for* $s \in [0, \Delta t]$.

$$min(\pi(s), \beta(s), \psi(s), \chi(s)) \geq k_1 s^{k_2} \tag{3.15}$$

Proof. By using the Picard–Lindelöf Theorem the equations (3.1) and (3.2) have a unique continuous solution in $[0, \Delta t)$ for some $\Delta t > 0$.

If $1 > \pi(0) > 0$ and $1 > \beta(0) > 0$, then the Lemma (3.1.1) follows by continuity of $(\pi(t), \beta(t)) \in [0, \Delta t)$.

(i) First, If $\pi(0) = 0$ but $1 > \beta(0) > 0$, then for Δt small and strictly positive, then the equation (3.1) becomes

$$\pi(\Delta t) = \nu\beta(0)\Delta t + o(\Delta t)$$

For Δt small enough

$$\pi(\Delta t) \geq \frac{1}{2}\nu\beta(0)\Delta t.$$
$$\pi(s) \geq \frac{1}{2}\nu\beta(0)s.$$

So for $s \in [0, \Delta t]$ if Δt is small enough. So the Lemma (3.1.1) follows.

- If $\pi(0) = 1$ but $1 > \beta(0) > 0$, then for Δt small and strictly positive, then from equation (3.13)

$$\psi(\Delta t) = \mu\Delta t + o(\Delta t). \tag{3.16}$$
$$\psi(s) \geq \frac{1}{2}\mu s. \tag{3.17}$$

So for $s \in [0, \Delta t]$, if Δt is small enough. So the Lemma (3.1.1) follows.

(ii) Second, suppose that $\beta(0) = 0$ and $1 > \pi(0) > 0$. Then

$$\beta(\Delta t) = \sigma\phi(\pi(0))\pi(0)\Delta t + o(\Delta t).$$
$$\beta(s) \geq \frac{1}{2}\sigma\phi(\pi(0))\pi(0)s.$$

for $s \in [0, \Delta t]$, if Δt is small enough,so the Lemma (3.1.1) is then true.

- If $\beta(0) = 0$ and $\pi(0) = 1$, then by the argument for $\beta(0) = 0$ and $1 > \pi(0) > 0$ for $\beta(s)$ and the argument for $\pi(0) = 1$ and $1 > \beta(0) > 0$ for $\psi(s)$, we have

$$min(\beta(s), \psi(s)) \geq k_1 s^{k_2}, \tag{3.18}$$

for $s \in [0, \Delta t]$, for some k_1, k_2 and $\Delta t > 0$. As a result, the Lemma (3.1.1) is true.

- If $\beta(0) = 1$ and $1 > \pi(0) > 0$, in this case from the equation (3.14)

$$\chi(\Delta t) = \phi(\pi(0))\Big[\psi(0)\rho + (\tau - \sigma)\pi(0)\Big]\Delta t + 0(\Delta t).$$

and

$$\begin{aligned}\tau - \sigma &= \Big[\lambda_1(1-p) + \lambda_2 p\Big]\gamma\Big[1 - (1-\xi) + \theta_1(1-\xi)\Big]. \\ &= \Big[\lambda_1(1-p) + \lambda_2 p\Big]\gamma\Big[1 - (1-\xi)(1-\theta_1)\Big] \geq 0.\end{aligned}$$

Hence $\chi(s) \geq k_1, s^{k_2}$, for $s \in [0, \Delta t]$ for some $k_1 > 0, k_2 > 0$ and $\Delta t > 0$. The result of the Lemma (3.1.1) follows.

- If $\beta(0) = 1$ and $\pi(0) = 0$, then using the argument for $\beta(0) = 1$ and $1 > \pi(0) > 0$ for $\chi(s)$ and the argument for $\pi(0) = 0$ and $1 > \beta(0) > 0$ for $\pi(s)$ we have

$$min(\pi(s), \chi(s)) \geq k_1 s^{k_2},$$

for some $k_1 > 0, k_2 > 0$ and $\Delta t > 0$, then the Lemma (3.1.1) is true.

(iii) Finally, if $\beta(0) = 1$ and $\pi(0) = 1$, then by using the argument for $\pi(0) = 1$ and $1 > \beta(0) > 0$ for $\psi(s)$ we have

$$\psi(s) \geq k_1 s^{k_2} \quad \textit{for} \quad s \in [0, \Delta t].$$

for some $k_1 > 0$, $k_2 > 0$ and $\Delta t > 0$.

If $\phi_1 \neq 0$ or $\theta_1 \neq 0$ then $\tau > \sigma$ and the argument for $\beta(0) = 1$ and $1 > \pi(0) > 0$ shows the same result for $\chi(s)$.

If $\phi_1 = \theta_1 = 0$,then $\tau = \sigma$ and

$$\frac{d\chi}{dt}\Big|_{t=0} = 0.$$

However assuming that ϕ is differentiable with respect to t

$$\begin{aligned}\frac{d^2\chi}{dt^2}\Big|_{t=0} &= \phi(\pi(0))\frac{d\psi}{dt}\Big|_{t=0}\rho\beta(0),\\ &= \phi(\pi(0))\mu\rho > 0.\end{aligned}$$

So again $\chi(s) \geq k_1 s^{k_2}$ for $s \in [0, \Delta t]$ for some $k_1 > 0$, $k_2 > 0$ and $\Delta t > 0$. Then the Lemma (3.1.1) holds.

Hence the Lemma (3.1.1) holds for all $\beta(0)$ and $\pi(0)$. So $\tau_e > \Delta t > 0$ for some $\Delta t > 0$.

Now suppose that $\tau_e < \Delta t$. Then for $t \in (\Delta t, \tau_e)$, then the equations 3.1, 3.2, 3.13 and 3.14 imply that

$$\begin{aligned}\frac{d\pi}{dt} &\geq -\mu\pi,\\ \frac{d\beta}{dt} &\geq -\phi(\pi)\pi\tau\beta - \phi(\pi)(1-\pi)\rho\beta,\\ &\geq -(\tau+\rho)\beta,\\ \frac{d\psi}{dt} &\geq -\phi(\pi)\nu\beta\psi,\\ &\geq -\nu\psi,\\ \frac{d\chi}{dt} &\geq -\phi(\pi)\pi\tau\chi,\\ &\geq -\tau\chi.\end{aligned}$$

Note that $\sigma > 0$ implies that $\phi_1 < 1$ and $\lambda_1(1-p) + \lambda_2 p > 0$ which implies that $\tau > 0$. Hence in $(\Delta t, \tau_e)$, $\pi(t) > 0$ and

$$\begin{aligned}\frac{1}{\pi}\frac{d\pi}{dt} &\geq -\mu,\\ \text{So} \qquad \pi(t) &\geq \pi(\Delta t)e^{-\mu(t-\Delta t)}.\end{aligned}$$

By the Picard́–Lindelöf Theorem, there is a unique continuous solution of the

equations 3.1 and 3.2 in $(\tau_e - \eta, \tau_e + \eta)$ for some $\eta > 0$, moreover

$$\pi(\tau_e) = \lim_{t \to \tau_e} \pi(t),$$
$$\geq \pi(\Delta t) e^{-\mu(\tau_e - \Delta t)} > 0.$$

Similarly

$$\beta(\tau_e) \geq \beta(\Delta t) e^{-(\tau+\rho)(\tau_e - \Delta t)} > 0,$$
$$\psi(\tau_e) \geq \psi(\Delta t) e^{-\nu(\tau_e - \Delta t)} > 0,$$
$$\chi(\tau_e) \geq \chi(\Delta t) e^{-\tau(\tau_e - \Delta t)} > 0.$$

Hence by continuity the unique continuous solution to the equations 3.1 and 3.2 is in $(0,1)\times(0,1)$ for $t \in (0, \tau_e + \eta)$ for some $\eta > 0$. This contradicts the definition of τ_e. Hence $\tau_e = \infty$.

This completes the proof of Case Two under the assumptions that $\phi(\pi) > 0$ $\forall$ $\pi \in [0,1]$ and $\sigma > 0$.

Now we shall look at the proof of Theorem 3.1.1 under the assumptions that $\sigma > 0$ and $\exists$ π^* with $1 \geq \pi^* > 0$ such that $\phi(\pi) = 0$ for $\pi \geq \pi^*$ and $\phi(\pi) > 0$ for $\pi < \pi^*$.

Lemma 3.1.2. *Assume that* $\sigma > 0$ *,but there* $\exists$ π^* *with* $1 \geq \pi^* > 0$ *such that* $\phi(\pi) = 0$ *for* $\pi \geq \pi^*$ *and* $\phi(\pi) > 0$ *for* $\pi < \pi^*$

Proof. We shall consider the following three cases

(i) If $\pi(0) < \pi^*$, then by the argument as above $\exists \Delta t > 0$ with $\tau_e \geq \Delta t > 0$. Now as $\pi \to \pi^{*-}$

$$\frac{d\pi}{dt} \to -\mu\pi^*.$$

So $\exists$ $\pi^+ < \pi^*$ such that for $\pi \in [\pi^+, \pi^*]$ π is strictly monotone decreasing, so if π starts strictly beneath π^* it can never reach it and $\pi \leq max\,(\pi(0), \pi^+) < \pi^*$. Hence the proof proceeds as in the previous case.

(ii) If $\pi(0) = \pi^*$, then for Δt small and strictly positive

$$\pi(\Delta t) = \pi^* - \mu\pi^*\Delta t + o(\Delta t).$$

So if Δt is sufficiently small and strictly positive

$$\pi(s) \leq \pi^* - \frac{1}{2}\mu\pi^* s \quad \textit{for} \quad s \in (0, \Delta t].$$

We assume that there is some strictly positive integer $k \geq 1$ with

$$(-1)^k \frac{d^k\phi(\pi)}{d\pi^k}\Big|_{\pi^{*-}} > 0,$$

and

$$(-1)^l \frac{d^l\phi(\pi)}{d\pi^l}\Big|_{\pi^{*-}} = 0 \quad \textit{for } 1 \leq l < k.$$

Let us define $(0, \tau_e)$ to be the maximal interval where a solution exists and $1 > \beta(s) > 0$ and $1 \geq \pi^* > \pi(s)$ for $s \in (0, \tau_e)$.

The argument proceeds as in the case where $\phi(\pi) > 0 \; \forall \pi \in [0, 1]$ and $\sigma > 0$ until we reach the case $\beta(0) = 0$ and $1 > \pi^* = \pi(0) > 0$. Then

$$\frac{d^l\beta}{dt^l}\Big|_{t=0^+} = 0, \; for \; l = 1, 2, \ldots k-1.$$

and

$$\begin{aligned} \frac{d^k\beta}{dt^k}\Big|_{t=0^+} &= \frac{d^k\phi(\pi)}{dt^k}\Big|_{t=0^+}\sigma\pi^* \\ &= (-1)^k(\mu\pi^*)^k\sigma\pi^*\frac{d^k\phi(\pi)}{d\pi^k}\Big|_{\pi^{*-}} > 0. \end{aligned}$$

So if Δt is small enough

$$\pi(s) \leq \pi^* - k_1 s, \quad \textit{and}$$

$$\beta(s) > k_2 s^{k_3} s, \quad \textit{for} \quad s \in [0, \Delta t].$$

for some $k_1, k_2, k_3 > 0$. So again the Lemma (3.1.1) holds. and $\pi(\Delta t) < \pi^*$ for Δt small and sufficiently positive.

If suppose that $\beta(0) = 1$ and $1 > \pi(0) > 0$. Then

$$\begin{aligned}
\frac{d\chi}{dt} &= \phi(\pi)\psi\rho\beta + \phi(\pi)\pi(\tau - \sigma)\pi - \pi\phi(\pi)\tau\chi. \\
\frac{d^l\chi}{dt^l}\Big|_{t=0^+} &= 0, \qquad \text{for} \qquad 1 \le l < k. \\
\frac{d^k\chi}{dt^k}|_{t=0^+} &= \frac{d^k\phi(\pi)}{dt^k}\Big|_{t=0^+}((1-\pi^*)\rho + (\tau-\sigma)\pi^*), \\
&= (-1)^k(\mu\pi^*)^k\frac{d^k\phi(\pi)}{d\pi^k}\Big|_{\pi^*=\pi^{*-}}[(1-\pi^*)\rho + (\tau-\sigma)\pi^*], \\
&> 0.
\end{aligned}$$

So again the Lemma (3.1.1) holds.

If $\beta(0) = 1$ and $\pi(0) = 1$, then the previous argument for $\pi(0) = 1$ and $1 > \beta(0) > 0$ shill still holds for $\psi(s)$.

- For $\chi(s)$ first consider the case $\phi_1 \neq 0$ or $\theta_1 \neq 0$. Then

$$\begin{aligned}
\frac{d^l\chi}{dt^l}\Big|_{t=0^+} &= 0, \qquad \textit{for} \qquad 1 \le l < k. \\
\frac{d^k\chi}{dt^k}\Big|_{t=0^+} &= \frac{d^k\phi(\pi)}{dt^k}\Big|_{t=0^+}(\tau-\sigma), \\
&= (-1)^k(\mu\pi^*)^k\frac{d^k\phi(\pi)}{d\pi^k}\Big|_{\pi^{*-}}(\tau-\sigma), \\
&> 0.
\end{aligned}$$

  So the Lemma (3.1.1) holds.

- For $\psi(s)$ and $\phi_1 = \theta_1 = 0$, then $\tau = \sigma$ and

$$\begin{aligned}
\frac{d^l\chi}{dt^l}\Big|_{t=0^+} &= 0, \qquad \text{for} \qquad 1 \le l \le k. \\
\frac{d^{k+1}\chi}{dt^{k+1}}\Big|_{t=0^+} &= \frac{d^k\phi(\pi)}{dt^k}\Big|_{t=0^+}\frac{d\psi}{dt}\Big|_{t=0^+}\rho \\
&= (-1)^k(\mu\pi^*)^k\mu\rho\frac{d^k\phi(\pi)}{d\pi^k}\Big|_{\pi^{*-}} \\
&> 0.
\end{aligned}$$

So the Lemma (3.1.1) holds.

Hence again the Lemma (3.1.1) holds for all $\beta(0)$ and $\pi(0)$ unless $\beta(0) = \pi(0) = \pi^* > 0$.

The proof proceeds again as in the case where $\phi(\pi) > 0$ all $\pi \in [0,1]$ and $\sigma > 0$.

(iii) If $\pi(0) > \pi^*$.

For $\pi(0) > \pi^*$ then for Δt small and strictly positive

$$\pi(\Delta t) = \pi(0) - \mu\pi(0)\Delta t + o(\Delta t)$$

So provided that $\pi(0) \geq \pi \geq \phi^*$

$$\frac{d\pi}{dt} = -\mu\pi.$$
$$\frac{d\beta}{dt} = 0.$$

So

$$\pi = \pi(0)e^{-\mu t}.$$

and

$$\beta = \beta(0).$$

This is the unique solution provided that

$$\pi \geq \pi^*.$$

I.e. $\pi(0)e^{-\mu t} \geq \pi \cdot *$

$$t \leq t_1 = \frac{-1}{\mu} \log_e\left(\frac{\pi^*}{\pi(0)}\right).$$

Define $(0, \tau_e')$ to be the maximal interval where a solution exists and $1 > \beta(s) \geq 0$ and $\pi(s) \geq \pi* > 0$.

Hence $\tau_e' = t_1$.

$\lim_{t \to t_1^-} \beta$ and $\lim_{t \to t_1^-} \pi$ exist and satisfy,

$$1 \geq \lim_{t \to t_1^-} \beta \geq 0 \quad \textit{and} \quad 1 > \lim_{t \to t_1^-} \pi > 0,$$

moreover, both limits cannot be zero. For $t \geq t_1$, the result follows by the case where $\pi(0) = \pi^*$ discussed earlier.

For $\sigma = 0$, the equations are

$$\frac{d\pi}{dt} = \phi(\pi)(1-\pi)\nu\beta - \mu\pi.$$
$$\frac{d\beta}{dt} = -\phi(\pi)(\tau\pi + \rho(1-\pi))\beta.$$

- If $\beta(0) = 0$ and $1 \geq \pi(0) \geq 0$,then the unique solution is

  $\beta(t) = 0 \quad \pi(t) = \pi(0)e^{-\mu t}$ for all time.

- If $\beta(0) > 0$, then the equations for ψ and χ are

$$\frac{d\psi}{dt} = \mu(1-\psi) - \phi(\pi)\psi\beta.$$
$$\frac{d\chi}{dt} = \phi(\pi)\Big(\tau\pi + \rho(1-\pi)\Big)\beta.$$

We proceed as in the case where $\sigma > 0$ and $\exists\ \pi^*$ such that $1 \geq \pi^* > 0$ such that $\phi(\pi) = 0$ for $\pi \geq \pi^*$ and $\phi(\pi) > 0$ for $\pi < \pi^*$.

A. For $\pi(0) < \pi^*$ again

$$\pi \leq \max(\pi(0), \pi^+) \quad \text{for all time.}$$

Define $(0, \tau_e)$ to be the maximal interval where a solution exists and

$$1 > \beta(s) > 0 \quad \textit{and} \quad 1 > \pi(s) > 0, \quad \text{in} \quad (0, \tau_e).$$

The proof follows by the same argument as used in the case where $\sigma > 0$ and either $\phi(\pi) > 0\ \forall\ \pi \in [0,1]$, or $\exists\ \pi^*$ with $\phi(\pi) = 0$ for $1 \geq \pi \geq \pi^* > 0$

For $\beta(0) > 0$ and either

B. $\pi(0) = \pi^*$ or

C. $\pi(0) > \pi^*$, the argument proceeds as in the case where $\sigma > 0$.

3.1.2.1 Summary of Results of Existence and Uniqueness Theorem

Here we summarised the results of Theorem 3.1.1 in points:

If $\beta(0) = \pi(0) = 0$, then $\beta(t) = \pi(t) = 0 \quad \forall t > 0$.

(i) If $\sigma > 0$ and $\phi(\pi) > 0 \; \forall \; \pi$ and $\beta(0) > 0$ or $\pi(0) > 0$ then $1 > \beta(t) > 0$, $1 > \pi(t) > 0$ for all $t > 0$.

(ii) If $\sigma > 0$ and $\exists \; \pi^*$ with $\phi(\pi) = 0$ for $1 \geq \pi \geq \pi^* > 0$.

(a). $\pi(0) \leq \pi^*$ then $1 > \beta(t) > 0, \pi^* > \pi(t) > 0$ for all $t > 0$.

(b). $\pi(0) > \pi*$ .For

$$t \leq t_1 = \frac{-1}{\mu} \log e\left(\frac{\pi^*}{\pi(0)}\right), \quad \beta(t) = \beta(0).$$

$$\pi(t) = \pi(0)e^{-\mu t} \quad (\text{ so } \beta(t_1) = \beta(0), \quad \pi(t_1) = \pi^*).$$

for $t > t_1 \quad 1 > \beta(t) > 0, \quad \pi^* > \pi(t) > 0$ for all $t > 0$.

(iii) If $\sigma = 0$ and $\exists \; \pi^*$ with $\phi(\pi) = 0$ for $1 \geq \pi \geq \pi^* > 0$.
If $\beta(0) = 0$, then $\beta(t) = 0, \pi(t) = \pi(0)e^{-\mu t}$ for all t.
If $\beta(0) > 0$ then the solutions have the same properties as in case (i) or (ii) appropriate.

This completes the proof of Theorem 3.1.1. Next, we look at the existence of equilibrium values in the system.

3.1.3 Existence of Equilibrium

Consider the differential equations (3.1)and (3.2) which describe the effect of the spread of the disease with an awareness program. We show that if $R_0 \leq 1$ then there is only the disease-free equilibrium, whereas if $R_0 > 1$ then there is a unique endemic equilibrium as well as the disease-free equilibrium.

Theorem 3.1.2. *Suppose that ϕ is monotone decreasing and $R_0 \leq 1$ then the equations (3.1)and (3.2)have a unique equilibrium solution where the disease dies out in PWIDs and needles, (π^*, β^*)=$(0, 0)$. This is the only equilibrium. For $R_0 > 1$ there is a unique nonzero equilibrium $(\pi^*,\beta^*) > 0$ in (0,1]×(0,1] as well as the disease-free equilibrium.*

Proof: See the proof of Theorem 2.3.2 is mentioned in the previous chapter. The model 2.4 as discussed in Chapter 2 I was obtained from the model 2.2 and 2.3 (equivalently 3.1and 3.2 by setting equation (1.3) to any equilibrium. Hence

Next, we going to study the stability analysis of the equilibrium of the system.

3.1.4 Local Stability Analysis of Equilibrium

We determine the local asymptotic stability of the DFE and EE values by the same techniques as were used by Greenhalgh and Hay (1997) and Agaba et al. (2017). This was to using the Routh-Hurwitz criterion (May (2001),DeJesus and Kaufman (1987)). We look at the eigenvalues of variational matrix of the linearized system about the DFE and EE to see if a small perturbation from these equilibria stays near the equilibrium or moves away.

Theorem 3.1.3. *One can verify that the basic reproduction number is the same as the reproduction number* $R_0 = \dfrac{\nu\sigma}{\rho\mu}$ *in one-dimensional model in the previous chapter. Assume that* ϕ *is a differentiable function of* π *in [0,1], we have shown that if* $R_0 < 1$ *then DFE of equations (2.2) and (2.3) is locally asymptotically stable, and if* $R_0 = 1$ *then the disease-free solution is neutrally stable. If* $R_0 > 1$ *then the DFE is unstable, whereas the unique EE is locally asymptotically stable.*

Proof: As the derivation of the basic reproduction number is the same as the reproduction number R_0 in the previous chapter used two-dimensional version of model it is straightforward to show that R_0 is the same for both models.

By using the variational matrix method around the equilibrium points, we recall the equations (2.2) and (2.3) as follows

$$\frac{d\pi}{dt} = \phi(\pi)(1-\pi)\nu\beta - \mu\pi = f(\pi,\beta). \tag{3.19}$$

$$\frac{d\beta}{dt} = \phi(\pi)\pi(\sigma - \tau\beta) - \phi(\pi)(1-\pi)\rho\beta = g(\pi,\beta). \tag{3.20}$$

Then the Jacobian matrix of the above model at (π^*, β^*) is an equilbrium

$$\boldsymbol{J} = \begin{pmatrix} \frac{\partial f}{\partial \pi} & \frac{\partial f}{\partial \beta} \\ \frac{\partial g}{\partial \pi} & \frac{\partial g}{\partial \beta} \end{pmatrix}.$$

$$= \begin{pmatrix} \phi'(\pi)(1-\pi)\nu\beta - \phi(\pi)\nu\beta - \mu & \phi(\pi)(1-\pi)\nu \\ \phi(\pi)(\sigma - \tau\beta) + \phi(\pi)\rho\beta + \phi'(\pi)[\pi(\sigma - \tau\beta) - (1-\pi)\rho\beta] & -\tau\phi(\pi)\pi - \phi(\pi)(1-\pi)\rho \end{pmatrix}.$$

First, we examined the local stability about the DFE point $\boldsymbol{E}_0 = (\pi_0, \beta_0) = (0, 0)$, then the Jacobian matrix $\boldsymbol{J}_1$ of $\boldsymbol{E}_0$ is obtained as

$$J_1|_{(\pi_0,\beta_0)} = \begin{pmatrix} -\mu & \nu \\ \sigma & -\rho \end{pmatrix}.$$

Then the eigenvalues λ of the the Jacobian matrix $\boldsymbol{J}_1$ are the roots of the characteristic equation

$$\lambda^2 + (\mu + \rho)\lambda + (\rho\mu - \nu\sigma) = 0.$$

This can written as

$$\lambda^2 + a_0\lambda + b_0 = 0, \tag{3.21}$$

where $a_0 = (\mu + \rho)$ and $b_0 = (\rho\mu - \nu\sigma)$.

According to the Routh-Hurwitz conditions (May (2001), DeJesus and Kaufman (1987)), which tell us that the equations have two roots with strictly negative real parts if and only if $a > 0$ and $b > 0$. We have $a = (\mu + \rho) > 0$ and $b = \rho\mu - \nu\sigma$.

Then, we see that according to the value of the reproduction number R_0, at DFE we have three different situations of stability depending on the satisfying of the Routh-Hurwitz conditions.

(1) If $R_0 = \frac{\nu\sigma}{\rho\mu} < 1$ it is straightforward that $\nu\sigma < \rho\mu$ that is $b_0 > 0$, thus the DFE is locally stable because the Routh-Hurwitz conditions here are satisfied. (2) If

$R_0 = \dfrac{\nu\sigma}{\rho\mu} > 1$,then $b_0 > 0$ and thus the FDE is unstable.
(3) If $R_0 = 1$, then it is straightforward to show $b = 0$ and the eigenvalues of (3.21) are $\lambda = 0$ and $\lambda = -(\mu + \rho)$. Hence the (DFE) is neutrally locally stable.

Similarly, for the endemic equilibrium point $\boldsymbol{E}_1 = (\pi^*, \beta^*)$ of the above system then the Jacobian matrix $\boldsymbol{J}_2$ of system corresponding to $\boldsymbol{E}_1$ is obtained as

$$\boldsymbol{J}_2|_{(\pi^*,\beta^*)} = \begin{pmatrix} \phi'(\pi^*)(1-\pi^*)\nu\beta^* - \phi(\pi^*)\nu\beta^* - \mu & \phi(\pi^*)(1-\pi^*)\nu \\ \phi(\pi^*)(\sigma - \tau\beta^*) + \phi(\pi^*)\rho\beta^* & -\tau\phi(\pi^*)\pi^* - \phi(\pi^*)(1-\pi^*)\rho \end{pmatrix}.$$

Then, the characteristic equation is

$$\lambda^2 + a_1\lambda + b_1 = 0.$$

Hence

$$a_1 = \tau\phi(\pi^*)\pi^* + \phi(\pi^*)(1-\pi^*)\rho + \mu + \phi(\pi^*)\nu\beta^* - \phi'(\pi^*)(1-\pi^*)\nu\beta^* > 0.$$

Note that $\phi'(\pi^*) \leq 0$, since $\phi(\pi)$ is monotone decreasing. Also

$$\begin{aligned} b_1 &= [\tau\phi(\pi^*)\pi^* + \phi(\pi^*)(1-\pi^*)\rho][\mu + \phi(\pi^*)\nu\beta^* - \phi'(\pi^*)(1-\pi^*)\nu\beta^*] \\ &\quad - \phi(\pi^*)^2(1-\pi^*)\nu[\rho\beta^* + (\sigma - \tau\beta^*)]. \\ b_1 &\geq [\tau\phi(\pi^*)\pi^* + \phi(\pi^*)(1-\pi^*)\rho][\mu + \phi(\pi^*)\nu\beta^*] - \phi(\pi^*)^2(1-\pi^*)\nu[\rho\beta^* + (\sigma - \tau\beta^*)], \end{aligned}$$

Using the fact that $\phi'(\pi^*) \leq 0$. Note that from the equilibrium of e we must have $\phi(\pi^*) > 0$ at the endemic equilibrium. From the equilibrium version of we using it to simplify b_1:

$$\mu + \phi(\pi^*)\nu\beta^* = \frac{\phi(\pi^*)\nu\beta^*}{\pi^*}.$$

$$\rho\beta^*(1-\pi^*) = (\sigma - \tau\beta^*)\pi^*.$$

Thus

$$b_1 \geq [\tau\phi(\pi^*)\pi^* + \phi(\pi^*)(1-\pi^*)\rho]\frac{\phi(\pi)\nu\beta}{\pi} - \phi(\pi^*)^2(1-\pi^*)\nu\Big[\rho\beta^* + \rho\beta\frac{(1-\pi^*)}{\pi^*}\Big].$$
$$= \phi(\pi^*)^2\left[\Big[\tau\pi^* + \rho(1-\pi^*)\Big]\frac{\nu\beta^*}{\pi^*} - (1-\pi^*)\nu\Big[\rho\beta^* + \rho\beta(1-\frac{\rho\beta}{\pi^*}\Big]\right].$$
$$= \tau\phi(\pi^*)^2\nu\beta^* > 0.$$

Thus the EE is locally asymptotically stable if $R_0 > 1$, since the Routh-Hurwitz conditions $a_1 > 0$ and $b_1 > 0$ are satisfied. The proof is completed.

In the next section, we will continue to study the stability analysis of DFE and EE by investigating the global stability of these equilibria.

3.1.5 Global Stability of Equilibrium

We investigate global stability of equilibrium by using the construction of Dulacś criterion and the Poincaré-Bendixson Theorem (Strogatz (2018), May (2001), DeJesus and Kaufman (1987)).

- **Dulacś criterion**

  Let D be a simply connected region of the plane. If there exists a continuously differentiable function $\Phi(x, y)$ such that

  $$\frac{\partial}{\partial x}\Big[\Phi(x,y)f(x,y)\Big] + \frac{\partial}{\partial y}\Big[\Phi(x,y)g(x,y)\Big].$$

  is of constant sign in D then the dynamical system

  $$\dot{x} = f(x, y).$$
  $$\dot{y} = g(x, y).$$

  has no closed orbits wholly contained in D.

- **Poincaré-Bendixson Theorem**

  a differentiable real dynamical system defined on an open subset of the plane then every non-empty compact ω-limit set of an orbit which contains only finitely many fixed points is either

– a fixed point,

– a periodic orbit, or

– a connected set consisting of a finite number of fixed points together with homoclinic and heteroclinic orbits connecting them.

Lemma 3.1.3. *Suppose that* $\pi(0) > 0$ *or* $\beta(0) > 0$*, we assert that there exists* $\alpha > 0$ *and* $t_1 > 0$ *such that* $\phi(\pi) \geq \alpha > 0$ *for* $t \geq t_1$

Proof: As ϕ is a monotone decreasing function with $\phi(\pi) \to 1$ as $\pi \to 0$ and π^2 is a strictly monotone increasing function for $\pi \in [0,1]$ as the Figure 3.1 shows there exists $\epsilon_0 \in (0,1]$, such that if $\phi(\pi) \leq \epsilon_0^2$, then $\pi \geq \epsilon_0$. So if $\epsilon < \epsilon_0$ then for $\phi(\pi) \leq \epsilon^2$

$$\begin{aligned}\frac{d\pi}{dt} &= \phi(\pi)(1-\pi)\nu\beta - \mu\pi, \\ &\leq \epsilon^2\nu - \mu\pi. \\ &\leq \epsilon\pi\nu - \mu\pi.\end{aligned}$$

As $\pi(0) > 0$ or $\beta(0) > 0$, from the results of Theorem 3.1.1 we must have $\pi(t) > 0$ for all t. Choose $\epsilon = \frac{1}{2}$ min $(\epsilon_0, \frac{\mu}{\nu})$, then

$$\frac{d\pi}{dt} \leq -\frac{1}{2}\mu\pi < 0.$$

So π is decreasing and $\phi(\pi)$ is increasing , so $\phi(\pi)$ cannot go beneath $\frac{1}{8}\Big[min(\epsilon_0, \frac{\mu}{\nu})\Big]^2 = \alpha$. So if $\phi(\pi)$ starts below α it must rise until it reaches to 2α. If $\phi(\pi)$ ever rises above 2α it can never fall beneath it. So there exists t_1 such that $\phi(\pi) \geq \alpha$ for $t \geq t_1 \geq 0$.

Theorem 3.1.4. *We have shown that*

(i) The disease-free equilibrium (DFE) E_0 *of equations (3.1) and (3.2) is globally stable when* $R_0 \leq 1$*, where the disease dies out and both* $\pi(t)$ *and* $\beta(t)$ *will tend to zero, whatever the initial conditions*

(ii) If $R_0 > 1$ *then the system has a unique endemic equilibrium (EE)* E_1 *which is globally stable, whenever the disease is present and either* $\pi(0) > 0$ *or* $\beta(0) > 0$.

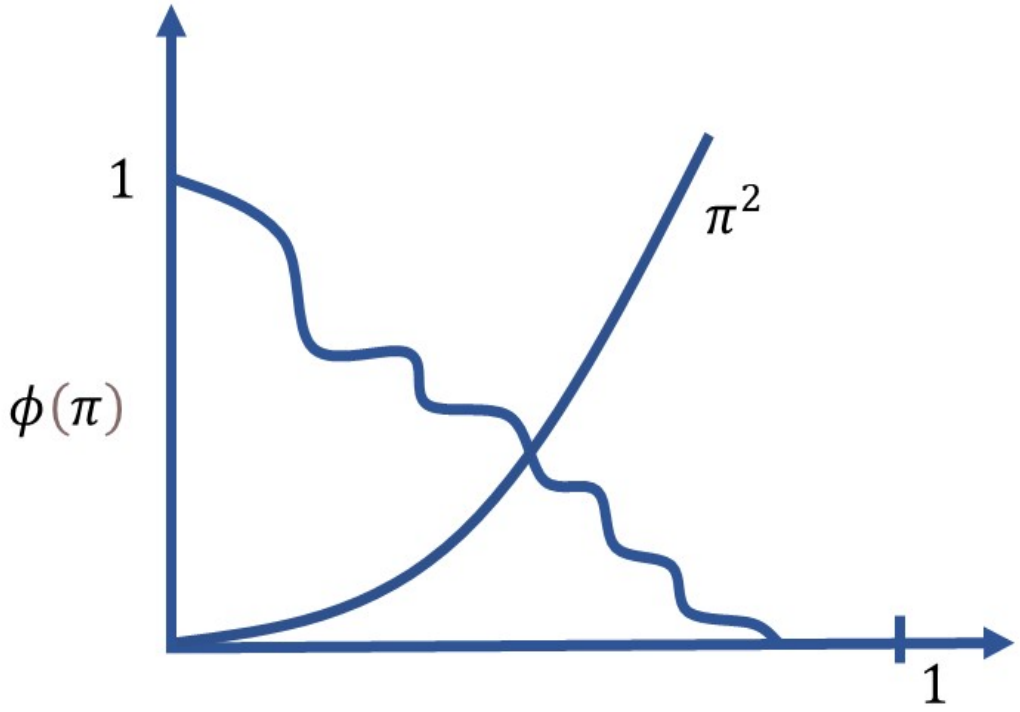


Figure 3.1: Illustration of Lemma 3.1.3.

So if σ>0 either and $\pi(0) > 0$ or $\beta(0) > 0$ the system tends to the unique endemic equilibrium E_1.

Proof: If $\pi(0) = \beta(0) = 0$ then $\pi(t) = \beta(t) = 0$ for all t and the results of Theorem 3.1.4 are obvious. Hence we shall assume that either $\pi(0) > 0$ or $\beta(0) > 0$. By following the mathematical techniques which are used in (Greenhalgh and Hay 1997), we are now going to prove global stability of the DFE first.

For $R_0 < 1$, we define $u = \beta + k\pi$, for $k \geq 0$ to show that $\pi \to 0$ and $\beta \to 0$ as $t \to \infty$. Then from (3.1) and (3.2)

$$\frac{du}{dt} = (\sigma\phi(\pi) - \mu k)\pi + \phi(\pi)(\nu k - \rho)\beta - \phi(\pi)\pi\beta(k\nu - \rho + \tau). \tag{3.22}$$

We choose $k = \frac{\rho}{\nu} - \epsilon$, where $\epsilon < \frac{\rho}{\nu}$ is small and positive. Recall that $R_0 = \frac{\sigma\nu}{\rho\mu}$. Then we can express the equation (3.22) as

$$\frac{du}{dt} = \Big(\sigma\phi(\pi) - \frac{\sigma}{R_0} + \epsilon\mu\Big)\pi - \epsilon\nu\phi(\pi)\beta - \phi(\pi)\pi\beta(\tau - \epsilon\nu). \tag{3.23}$$

Choose $\epsilon > 0$ sufficiently small so that $k > 0$, $\tau - \epsilon\nu > 0$, and

$$\sigma\Big(\phi(\pi) - \frac{1}{R_0}\Big) - \epsilon\mu \leq \sigma\Big(1 - \frac{1}{R_0}\Big) - \epsilon\mu = -\eta < 0.$$

Then

$$\frac{du}{dt} \leq -\eta\pi - \epsilon\nu\phi(\pi)\beta.$$

For $t \geq t_1$.

$$\begin{aligned}\frac{du}{dt} &\leq -\eta\pi - \alpha\epsilon\nu\beta,\\ &=\leq -\psi\beta - \psi k\pi,\\ &= \psi u,\end{aligned}$$

where

$$\psi = min\Big(\alpha\nu\epsilon, \frac{\eta}{k}\Big) > 0.$$

As $\psi k \leq \eta$ and $\psi \leq \alpha\nu\epsilon$. Hence $0 \leq u \leq u(t_1)e^{-\psi(t-t_1)}$ and $u \to 0$ as $t \to \infty$. So both $\pi(t)$ and $\beta(t)$ tend to zero as $t \to \infty$. This complete the proof of the global stability of the DFE in case $R_0 < 1$ or $\pi(0) = \beta(0) = 0$. We shall deal with case $R_0 = 1$ and $\pi(0) > 0$ or $\beta(0) > 0$ later.

Next, we shall prove the global stability of the EE for $R_0 > 1$ and $\pi(0) > 0$ or $\beta(0) > 0$. By Theorem 3.1.1 the solution to the differential equation system (3.19) and (3.20) remains with the simple connected region

$$D_0 = \Big\{(\pi, \beta) \in [0,1] \times [0,1]\Big\}.$$

To apply Dulacś criterion we need to find a continuously differentiable function

$\Phi(\pi,\beta)$ such that

$$\frac{\partial}{\partial\pi}[\Phi(\pi,\beta)f(\pi,\beta)] + \frac{\partial}{\partial\beta}[\Phi(\pi,\beta)g(\pi,\beta)]. \tag{3.24}$$

does not change sign in D_0. We take $\phi \equiv 1$, by applying the equation (3.24) for the equations (3.19) and (3.20) then we get

$$\frac{\partial}{\partial\pi}[\phi(\pi)(1-\pi)\nu\beta - \mu\pi] + \frac{\partial}{\partial\beta}[\phi(\pi)\pi(\sigma-\tau\beta) - \phi(\pi)(1-\pi)\rho\beta] = \phi'(\pi)(1-\pi)\nu\beta$$
$$-\phi(\pi)\nu\beta - \mu - \tau\phi(\pi)\pi - \phi(\pi)(1-\pi)\rho < 0.$$

So the set of equations (3.19) and (3.20) have no closed orbits wholly contained in D. In the Figure 3.2 shows the $\pi\beta$-plane which define $A = (0,1), B = (1,1)$

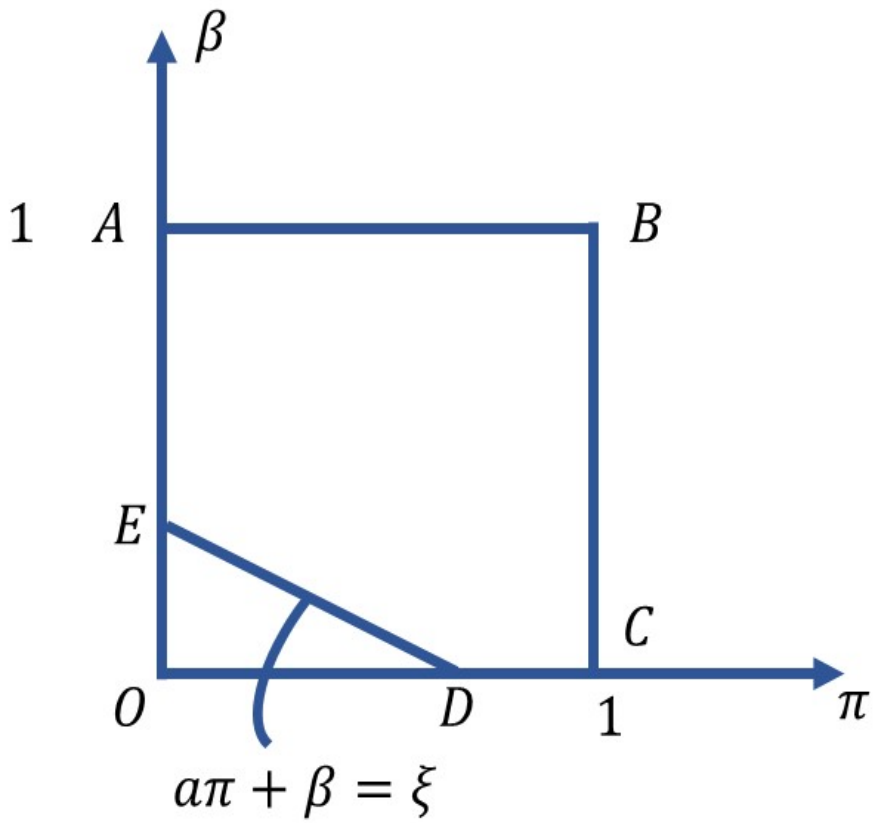


Figure 3.2: Illustration of Theorem 3.1.4 .

and $C = (1, 0)$. Let the points of intersection of the line $a\pi + \beta + \xi$ with the π and β axes be respectively. $D = (\xi/a, 0)$ and $E = (0, \xi)$. In the case where there exists $\pi^* > 0$ such that $\phi(\pi) = 0$ for $\pi \geq \pi^*$,we assume that ξ is small enough so that $\xi/a < \pi^*$.

We shall next prove that the closed polygon $ABCDE$is a closed attracting region for (3.19) and (3.20). Note that as $R_0 > 1, \sigma > 0$. We have already shown in Theorem 3.1.1 that if we start on the boundaries AB, BC, CD of EA we will move into The interior of $ABCDE$ in a finite time. This competes the proof of Theorem 3.1.4 in the case $R_0 > 1$.

The line DE the normal to DE is $(a, 1)$, then we have

$$(a,1).\left(\frac{d\pi}{dt}, \frac{d\beta}{dt}\right) = a[\phi(\pi)(1-\pi)\nu\beta - \mu\pi] + [\phi(\pi)\pi(\sigma - \tau\beta) - \phi(\pi)(1-\pi)\rho\beta],$$
$$= a(\phi(0)\nu\beta - \mu\pi) + \phi(0)(\sigma\pi - \rho\beta) + o(\epsilon),$$

as ϕ is continuous at $\pi = 0$.

So

$$(a,1).\left(\frac{d\pi}{dt}, \frac{d\beta}{dt}\right) = a(\nu\beta - \mu\pi) + \sigma\pi - \rho\beta + f(\epsilon).$$

Hence $f(\epsilon)$ is $0(\epsilon)$, in other words $\dfrac{|f(\epsilon)|}{\epsilon} \to 0$ as $\epsilon \to 0$. Then

$$\begin{aligned}(a,1).\left(\frac{d\pi}{dt}, \frac{d\beta}{dt}\right) &= (\sigma - a\mu)\pi + (a\nu - \rho)\beta + f(\epsilon),\\ &= \frac{(\sigma - a\mu)}{a} a\pi + (a\nu - \rho)\beta + f(\epsilon),\\ &\geq min(\frac{(\sigma - a\mu)}{a}, a\nu - \rho)(a\pi + \beta) + f(\epsilon),\\ &= min\left(\frac{(\sigma - a\mu)}{a}, a\nu - \rho\right)\epsilon + f(\epsilon).\end{aligned}$$

So choose ϵ small enough so that

$$\frac{|f|}{\epsilon} \leq \frac{1}{2} min\left(\frac{(\sigma - a\mu)}{a}, a\nu - \rho\right).$$

Then on the line DE $(a,1).\left(\frac{d\pi}{dt},\frac{d\beta}{dt}\right) > 0$, so if θ is the angle between these two vectors

$\cos\theta > 0 \Rightarrow$ this implies that $-\frac{-\pi}{2} < \theta < \frac{\pi}{2}$.

So $ABCDE$ is a closed invariant region for (3.19) and (3.20) containing no limit cycles and only one equilibrium point. Given a starting point $(\pi(0),\beta(0))$ with either $\pi(0) > 0$ or $\beta(0) > 0$ by choosing ϵ small enough we can ensure that the starting point is in $ABCDE$. As the unique endemic equilibrium is locally asymptotically stable there is a small neighbourhood of it such that any trajectory starting in this neighbourhood tends to it. So there cannot be any homoclinic loops , also there cannot be any homoclinic loops because they are closed orbits.

By the Poincaré-Bendixson Theorem the trajectory either tends to the unique fixed point in $ABCDE$ or a limit cycle. But we have already shown that there are no limit cycles in $ABCDE$. Hence the trajectory must tends to the unique EE.

In case where $R_0 = 1$ and $\pi(0) > 0$ or $\beta(0) > 0$ consider the closed square $OABC$ we must have $\alpha > 0$ as $R_0 = 1$. We have already shown in Theorem 3.1.1 that if we start on the boundaries OA, AB, BC or CO we must move into the interior of $OABC$ in at most finite time $OABC$ is a closed attracting region for (3.19) and (3.20) containing only one fixed point E_0 and no closed orbits. Thus there cannot be any homoclinic loops as they are closed orbits. So the trajectory approaches the unique fixed point which is the DFE E_0. This completes the proof of Theorem 3.1.4.

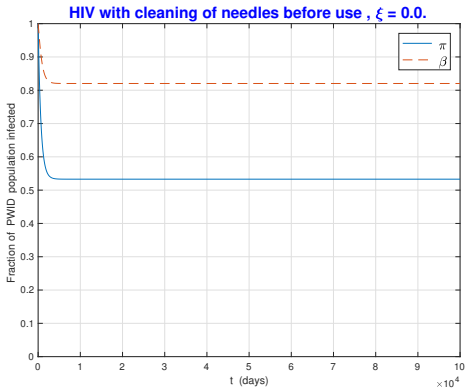
3.1.6 Simulations

In this section we shall demonstrate numerically the analytical results on existence of a unique non-negative solution to the equations (3.1) and (3.2) and their stability. We have used similar parameter values to those which were used in the numerical simulations which we performed in the previous Chapter . The simulations were performed using the computer package MATLAB and our simulations

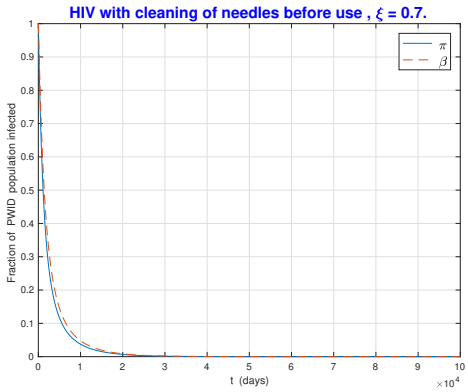
were integrated using the numerical integration package SOLVER which was the numerical ordinary differential equation (ode45). Our computer program was verified using comprehensive output from a large number of runs.We have aimed to use realistic parameter values for the spread of HIV amongst PWIDs but our main objective is to prove the analytic results obtained in Theorems 3.1.1 - 3.1.4 which estimate the spread of HIV amongst PWIDs for model (3.1) and (3.2) with two disease awareness programs. In these Theorems we showed that if $R_0 \leq 1$ then the disease will die out and that if the disease is initially present and $R_0 > 1$ then the disease will tend to the unique endemic equilibrium.

The realistic value we chose previously for μ, the per capital rate at which PWIDs leave the sharing injecting population was $\mu = 0.258$/year =7.0637×10^{-4}/day, $P_1 = 0.0$, $P_2 = 0.25$, $P_3 = 0.01$, $P_4 = 0.74$, $\lambda_1 = \lambda_2 = 0.143$/day and $\gamma = 1$ (based on Liang et al. (2016)) and varying values of the needle cleaning probability ξ with $0 \leq \xi \leq 1$.

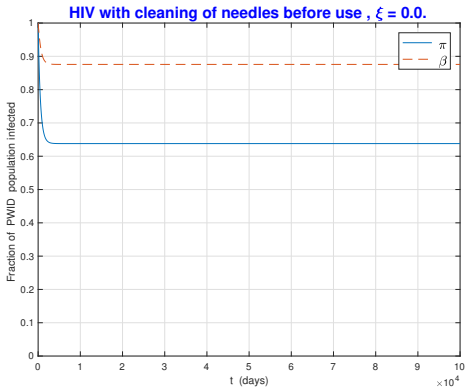
We studied the behaviour of the model of the equations (3.1) and (3.2) through altering R_0 by choosing different values of ξ. In Figures (3.3) and (3.4) starting value was initially $\pi(0) = 1$, $\beta(0) = 1$ in all cases. The plots of six simulations in Figure (3.3) shows the disease with awareness program $\phi(\pi)$=$1 - \frac{a\pi}{b+\pi}$ with various values of the constants a and b are shown in Figure 3.3. In the sub-Figures a, c and e of Figure 3.3 where $R_0 > 1$ we choose $\xi = 0.0$, then from equations (2.14) and (2.18) we have $\sigma = 0.143$/day, $\tau = 0.143$/day, $\rho = 0.0357$/day and $\nu = 0.0014$/day giving $R_0 = 8.0978$. For the other sub-Figures b, d and f of Figure 3.3 where $R_0 < 1$ we choose $\xi = 0.7$ then from equations (2.14) and (2.18) we have $\sigma = 0.143/$ day, $\tau = 0.143$/day, $\rho = 0.1108$/day and $\nu = 0.000429$/day giving $R_0 = 0.7837$. Similarly, Figure 3.4 shows plots of six simulations with the disease awareness program $\phi(\pi)$= $e^{-m_0 n \pi}$ (taken from Cui et al. (2008)), with different values of m_0. Similarly to the results of figure 3.3 we have that $R_0 > 1$ for sub-Figures a, c and e of Figure 3.4 and $R_0 < 1$ for sub-Figures b, d and f of Figure 3.4. In all



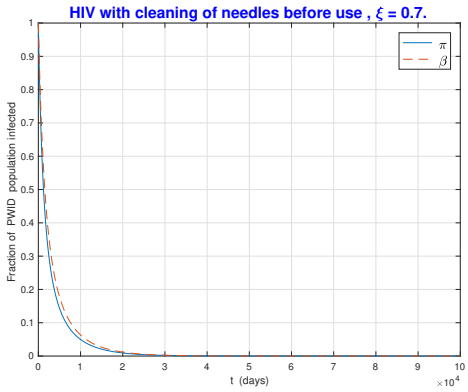
(a) With values of awareness program function parameters $a = 0.9, b = 1$.



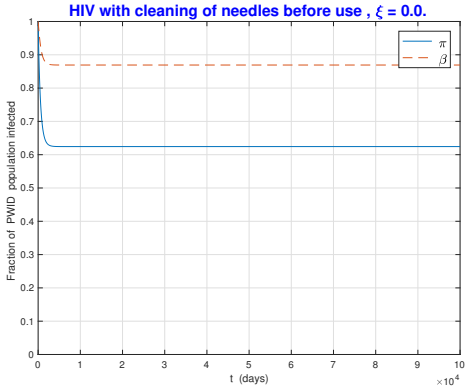
(b) With values of awareness program function parameters $a = 0.9, b = 1$.



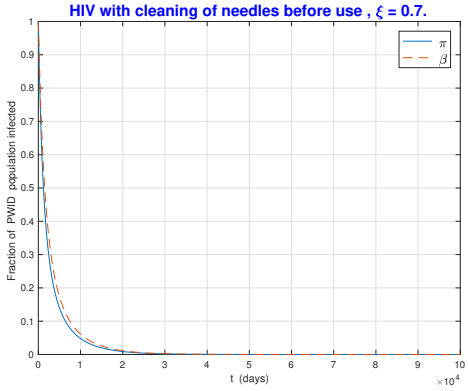
(c) With values of awareness program function parameters $a = 0.1, b = 10$.



(d) With values of awareness program function parameters $a = 0.1, b = 10$.

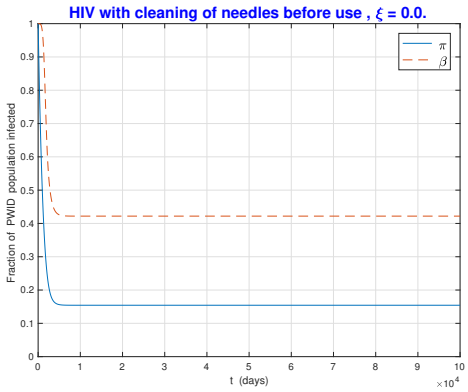


(e) With values of awareness program function parameters $a = 0.5, b = 5$.

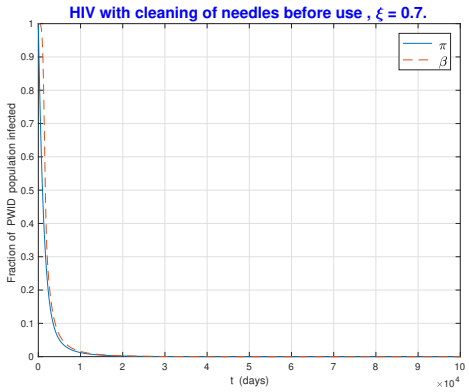


(f) With values of awareness program function parameters $a = 0.5, b = 5$.

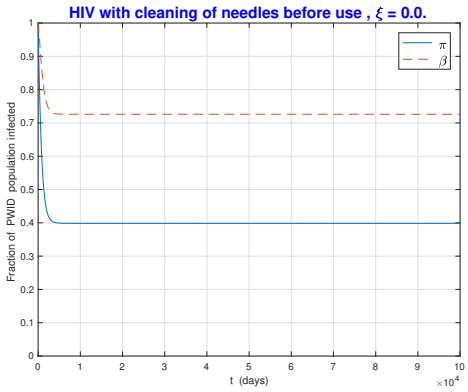
Figure 3.3: The plots of simulations for the solution of model (3.1) and (3.2) with awareness program function $\phi(\pi)=1-\frac{a\pi}{b+\pi}$ and when $\xi = 0.0$ when $R_0 > 1$ and when $\xi = 0.7$ when $R_0 < 1$.



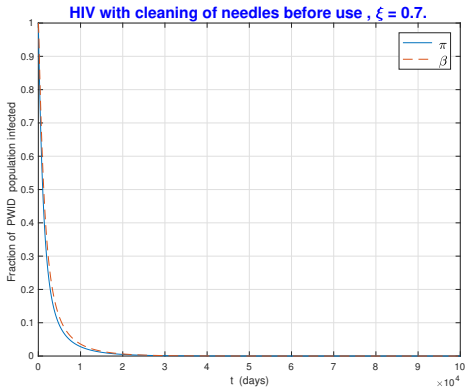
(a) With values of awareness program function parameters $m_0 = 10.0/n$.



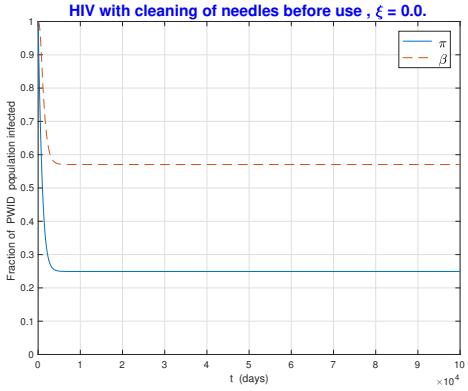
(b) With values of awareness program function parameters $m_0 = 10.0/n$.



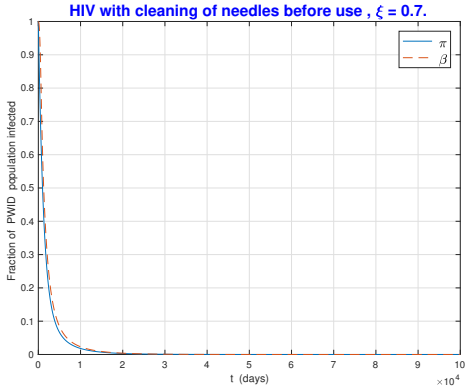
(c) With values of awareness program function parameters $m_0 = 2.0/n$.



(d) With values of awareness program function parameters $m_0 = 2.0/n$.



(e) With values of awareness program function parameters $m_0 = 5.0/n$.



(f) With values of awareness program function parameters $m_0 = 5.0/n$.

Figure 3.4: The plots of simulations for the solution of model (3.1) and (3.2) with awareness program function $\phi(\pi)$= $e^{-m_0 n\pi}$, where $n = 1000$ and when $\xi = 0.0$ when $R_0 > 1$ and when $\xi = 0.7$ then $R_0 < 1$.

cases the disease died out if $R_0 \leq 1$ and took off if $R_0 > 1$ and disease was initially present, confirming our analytical results. Again the simulations were repeated with other parameter values and other initial values for π and β and in each case the disease died out if $R_0 \leq 1$ and took off if $R_0 > 1$ and disease was initially present again confirming our analytical results.

3.2 Conclusion

We began this chapter by expanding the model under study to be a two-dimensional model. We derived a system of differential equations for the spread of HIV amongst PWIDs, incorporating a disease awareness program. The expression for the biological parameter R_0 was the same as in the previous chapter. The model under study in this chapter improves the model in the previous chapter as the two-dimensional model with PWIDs and needles is more accurate. It again improves previous work in the literature as it adds a disease awareness program into the model. Because the model is two-dimensional different techniques have to be used to prove the results.

We showed the equilibrium solutions analytically if ϕ is strictly monotone decreasing or monotone decreasing. Then we have shown that if $R_0 \leq 1$ is the only condition for the disease to die out in all PWIDs and needles. Whereas if the disease is initially present and $R_0 > 1$ the disease will present among the population of PWIDs for all time. Furthermore, we proved that the free disease equilibrium of the model (3.1) and (3.2) is locally and globally stable if $R_0 < 1$, whereas it is unstable if $R_0 > 1$. Also, we showed that if $R_0 > 1$ the system has a unique endemic solution which is locally and globally stable, wherever the disease is present and either $\pi(0) > 0$ or $\beta(0) > 0$. So if either $\pi(0) > 0$ or $\beta(0) > 0$ and $R_0 > 1$the system tends to the unique endemic equilibrium.

Finally, we illustrated the dynamic behaviour of this model graphically using numerical simulations. The analytic results which we obtained by simulations confirmed our analytical results in Theorems (3.1.1) - (3.1.4) which allowed us to estimate the spread of HIV amongst PWIDs for model (3.1) and (3.2)with two disease awareness programs. We have used similar parameter values which were used in the numerical simulations which have been done in the previous chapter.

Chapter 4

The Impact of Awareness Programs in a Three-Stage Infectivity Model of HIV/AIDS

4.1 Introduction

Motivated by the work that has been done in the previous chapters, in this chapter we shall study the impact of awareness programs in a Three-Stage Infectivity Model of HIV/AIDS to assume more realistically by applying for awareness programs as described in previous chapters, under the assumption that a population of intravenous drug users cleans their needles before not after use. As a result, the chapter is structured as follows: We initially describe the standard assumptions of the three-stage HIV/AIDS infection model by Lewis (2000) we are going to show the existence of a unique non-negative solution and explore the existence of equilibrium points. Then we move on to investigate the stability analysis procedure to study the behaviour of our model over time, in particular, we shall pay attention to the conditions necessary for HIV/AIDS to die out or persist in the IDU population. Then, we perform some simulations with realistic parameter

values to verify the analytical results for our models. Eventually, a brief summary and discussion conclude.

4.1.1 Three-Stage Infectivity Model of HIV/AIDS

This deterministic model for the transmission of HIV among intravenous PWIDs, known as the "Simple model," is investigated byLewis and Greenhalgh (2001) model, It allows PWIDs to pass through three stages of infectivity before the development of AIDS. In this part, we revise Lewis and Greenhalgh's model to incorporate more realistic cleaning of needles prior to usage as opposed to after visiting shooting ranges.

Therefore in this section, we will briefly explain the definition of the modelling assumptions that Lewis and Greenhalgh (2001) model presented in detail. These assumptions were first proposed by Kaplan Kaplan (1989), and it was further discussed by Greenhalgh and Hay (1997). The following is assumed in the model for a size n susceptible population of PWIDs, where n is a massive number:

Table 4.1: Description of Parameters

Parameter	Definition
m	is defined to be a location where PWIDs share injecting equipment, PWIDs choose at random among various shooting ranges and inject once each visit (Equivalently, this can be thought of as m drug injection equipment 'kits' being in circulation).
λ	is the rate at which each PWID, who visits shooting ranges independently of other PWIDs, according to a Poisson process.
θ	is the probability of the needle is the flushed rate by replacing the infectious blood by non-infectious blood When an uninfected PWID uses the infected injecting equipment.
α	is the probability of the infectivity of HIV through shared injecting equipment if a PWID is exposed to HIV. Where the PWIDs can become infected with HIV only through shared injecting equipment.
δ_1	is the initial infection rate. PWID is defined as highly infectious and enters an asymptomatic stage according to the Poisson process.
δ_2	Asymptomatic PWIDs rate enter the Pre-AIDS stage according to a Poisson process .
δ_3	Pre-AIDS PWIDs rate enter the full-blown AIDS stage according to a Poisson process, and at this point PWIDs leave the sharing, injecting population.
μ	is the rate number of Infectious PWIDs who leave the population for other reasons (for example death, treatment or relocation) and are replaced by susceptible PWIDs.
ξ	Proportion of PWIDs (susceptible or not) who successfully bleach their injection equipment after use.
τ	It is the exchange rate of each needle for an uninfected needle according to the Poisson process.

4.2 Formulation of Three-Stage Infectivity Model of HIV/AIDS with Awareness Programs

In this section, we will develop the differential equations that determine the three-stage infectivity model of HIV/AIDS amongst PWIDs, as discussed by Lewis and Greenhalgh (2001) model by reflecting the reduction in the spread of HIV/AIDS

amongst PWIDs due to the awareness programs, multiply the term transmission by a factor $\phi(\pi)$. So we modify the model of Lewis and Greenhalgh (2001) to make it more realistic so PWIDs clean their needles before use, this modify will apply just into the first stage of infected PWIDs equation $\pi_1(t)$ and $\beta(t)$ the infected needles equation to include the awareness program function $\phi(\pi)$ with keeping the same form of the rest of the differential equations of the model as Lewis and Greenhalgh (2001) paper described.

The four equations are as follows: one for each stage of infectious PWIDs $\pi_1(t), \pi_2(t), \pi_3(t)$, and the one for infected needles $\beta(t)$.

First- stage equation $\pi_1(t)$:

The number of first-stage infected PWIDs at the time $t + \Delta t$

$n\pi_1(t + \Delta t)$= number of first- stage of PWIDs at time t .

+ number of uninfected PWIDs each of whom injects at a rate $\lambda\phi(\pi)$ at time t.

$\times \Big($ the proportion of addicts who inject in $[t, t + \Delta t)$ with an infectious needle that has not been cleansed before usage and where HIV is transmitted in a single injection $\Big)$.

- the number of first-stage infected PWIDs who develop to second-stage infectivity or depart the shooting galleries the population in $[t, t + \Delta t)$.

This equation can be written as follows:

$$n\pi_1(t + \Delta t) = n\pi_1(t) + n\left(1 - \pi_1(t) - \pi_2(t) - \pi_3(t)\right)\lambda\phi(\pi)\Delta t\beta(t)\alpha(1 - \xi) - n\pi_1(t)\Delta t\left(\mu + \delta_1\right) + o(\Delta t).$$

Next, we subtract $n\pi(t)$ from both sides. we deduce that

$$n\pi_1(t + \Delta t) - n\pi_1(t) = + n\left(1 - \pi_1(t) - \pi_2(t) - \pi_3(t)\right)\lambda\phi(\pi)\Delta t\beta(t)\alpha(1 - \xi) - n\pi_1(t)\Delta t\left(\mu + \delta_1\right) + o(\Delta t).$$

Then we divide by $n\Delta t$ and let $\Delta t \to 0$, gives the following

$$\frac{d\pi_1}{dt} = \left(1 - \sum_{i=1}^{3}\pi_i\right)\lambda\phi(\pi)\beta\alpha(1 - \xi) - \left(\mu + \delta_1\right)\pi_1.$$

Second -Stage equation $\pi_2(t)$:

In this equation, we keep the same method have been used in Lewis and Greenhalgh Lewis (2000) paper to derive the second-stage equation $\phi_2(t)$ it is shown as

$$\frac{d\pi_2}{dt} = \delta_1\pi_1 - (\mu + \delta_2)\,\pi_2.$$

Third -Stage equation $\pi_3(t)$:

Similarly, from Lewis and Greenhalgh Lewis (2000) paper we have that

$$\frac{d\pi_3}{dt} = \delta_2\pi_2 - (\mu + \delta_3)\,\pi_3.$$

Infected needles equation $\beta(t)$:

We use the same argument above to calculate the number of infected needles at time $t + \Delta t$.

$= \left\{\text{number of infected needles at time } [t, t+\Delta t)\ \right\}$

$+ \left\{\ \text{number of uninfected needles at time } t\right\}$

$\times \left\{\text{fraction of needles used by infected PWIDs who inject at rate } \lambda\phi(\pi) \text{ in } [t, t+\Delta t)\right\}$

$- \left\{\text{number of infected needles at time } t\ \right\}$

$\times \left\{\ \text{fraction of infected needles used by uninfectious PWIDs who inject at rate } \lambda\phi(\pi) \text{ in } [t, t+\Delta t) \text{ and left in an uninfected state}\right\}.$

We adopt a similar procedure to compute the rate of change in the number of infected needles at time$t$ to get at the following findings.

$$\begin{aligned} m\beta(t+\Delta t) =& m\beta(t) + m(1-\beta(t))\lambda\phi(\pi)\Delta t\gamma\,(\pi_1(t) + \pi_2(t) + \pi_3(t)) \\ &- m\beta(t)\lambda\phi(\pi)\Delta t\gamma\,(1 - \pi_1(t) - \pi_2(t) - \pi_3(t))\,(1 - (1-\xi)(1-\theta)) \\ &- m\beta(t)\tau\Delta t + o(\Delta t). \end{aligned}$$

We can conclude that by subtracting $m\beta(t)$ from both sides, dividing by $m\Delta t$ and letting $\Delta t \to 0$,we get

$$\frac{d\beta}{dt} = (1-\beta)\lambda\phi(\pi)\gamma\left(\sum_{i=1}^{3}\pi_i\right) - \beta\lambda\phi(\pi)\gamma\left(1 - \sum_{i=1}^{3}\pi_i\right)(1 - (1-\theta)(1-\xi)) - \beta\tau.$$

As a result, the system of differential equations that characterises the preva-

lence of HIV/AIDS amongst PWIDs with awareness programs is:

$$\frac{d\pi_1}{dt} = \left(1 - \sum_{i=1}^{3} \pi_i\right) \lambda\beta\alpha\phi(\pi)(1-\xi) - (\mu + \delta_1)\pi_1, \tag{4.1}$$

$$\frac{d\pi_2}{dt} = \delta_1\pi_1 - (\mu + \delta_2)\pi_2, \tag{4.2}$$

$$\frac{d\pi_3}{dt} = \delta_2\pi_2 - (\mu + \delta_3)\pi_3, \tag{4.3}$$

$$\frac{d\beta}{dt} = (1-\beta)\lambda\gamma\phi(\pi)\left(\sum_{i=1}^{3} \pi_i\right) - \beta\lambda\gamma\phi(\pi)\left(\sum_{i=1}^{3} \pi_i\right)(1-(1-\theta)(1-\xi)) - \beta\tau. \tag{4.4}$$

Let $\pi_i(t)$ denote the number of three-stage infected PWIDs at time $[t, t+\Delta t]$ and $\beta(t)$ the number of infected needles at time $[t, t+\Delta t]$. The model's biological parameters are as given in the previous section, with proper initial conditions:

(1) $0 \leq \pi_i(0), \beta(0)$, where $i = 1, 2, 3$ and

(2) $\pi_1(0) + \pi_2(0) + \pi_3(0), \beta(0) \leq 1$.

We can rewrite the equations

$$\frac{d\pi_1}{dt} = (1-\pi)\lambda\beta\alpha\phi(\pi)(1-\xi) - (\mu + \delta_1)\pi_1, \tag{4.5}$$

$$\frac{d\pi_2}{dt} = \delta_1\pi_1 - (\mu + \delta_2)\pi_2, \tag{4.6}$$

$$\frac{d\pi_3}{dt} = \delta_2\pi_2 - (\mu + \delta_3)\pi_3, \tag{4.7}$$

$$\frac{d\beta}{dt} = (1-\beta)\lambda\gamma\phi(\pi)\pi - \beta\lambda\gamma\phi(\pi)(1-\pi)(1-(1-\theta)(1-\xi)) - \beta\tau. \tag{4.8}$$

where π=$\pi_1 + \pi_2 + \pi_3$.

Next, we move on to calculate the fundamental reproduction numberR_0 for the model.

4.2.1 The Basic Reproductive Number R_0

The expected number of secondary infections caused by a single newly infected person (or needle) entering a completely disease-free population at equilibrium Diekmann et al. (1990a) is known as the basic reproductive number R_0. Secondary infection occurs when a person becomes infected after using an infectious needle and syringe that has been contaminated by the original infected

PWIDs. In epidemiological models, R_0 is a significant parameter as it determines the overall behaviour of our model over time, where the disease generally dies out when $R_0 \leq 1$ and an epidemic usually arises when $R_0 > 1$.

In this section, we going to use a similar a framework similar to that found in Chapter 2 of Lewis (2000) to derive an expression for R_0 for model equations (4.5)-(4.8).

In our case, the number of secondary infection cases is described in a disease-free equilibrium, where a PWID enters one newly infected into a group containing only susceptible PWIDs and non-infectious needles. The scenario for finding the structure infection process can be as follows:

- The virus passes from a single infected PWID to a non-infectious needle.
- The virus passes from a newly infected needle(at any stage of infectivity) to a susceptible PWID.

As a result, to calculate reproduction numberR_0, we want to know the expected number of infectious needles produced by a single infectious PWID during the period of their infectious lifetime, as well as the number of PWIDs that each of these needles is likely to infect. By assuming that all rates are constant the development of PWIDs can be described in three infectious stages as follows: We recall that F is the rate of new infections, so we have

$$\begin{aligned} F_1 =& (1-\pi)\lambda\beta\alpha\phi(\pi)(1-\xi), \\ F_2 =& \delta_1\pi_1, \\ F_3 =& \delta_2\pi_2, \\ F_4 =& \lambda\gamma\phi(\pi)\pi + \beta\lambda\gamma\phi(\pi)(1-(1-\theta)(1-\xi). \end{aligned}$$

We have also V is the net rate of transfer of disease into other classes.

$$V_1 = (\mu + \delta_1)\pi_1,$$

$$V_2 = (\mu + \delta_2)\pi_2,$$

$$V_3 = (\mu + \delta_3)\pi_3,$$

$$V_4 = \beta\lambda\gamma\phi(\pi) + \beta\lambda\gamma\phi(\pi)(1 - (1 - \theta)(1 - \xi)) + \beta\tau.$$

Here $\hat{\theta} = (1 - (1 - \theta)(1 - \xi))$ and $\hat{\tau} = \dfrac{\tau}{\lambda\gamma}$, around the disease-free equilibrium (DFE), F defined by $\partial F/\partial x$ and $V = \partial V/\partial x$ are given by

$$\boldsymbol{F} = \begin{bmatrix} 0 & 0 & 0 & \lambda\alpha(1-\xi) \\ \delta_1 & 0 & 0 & 0 \\ 0 & \delta_2 & 0 & 0 \\ \lambda\gamma & \lambda\gamma & \lambda\gamma & 0 \end{bmatrix}.$$

And

$$\boldsymbol{V} = \begin{bmatrix} (\mu + \delta_1) & 0 & 0 & 0 \\ 0 & (\mu + \delta_2) & 0 & 0 \\ 0 & 0 & (\mu + \delta_3) & 0 \\ 0 & 0 & 0 & \lambda\gamma(\hat{\theta} + \hat{\tau}) \end{bmatrix}.$$

we have that

$$\boldsymbol{V}^{-1} = \begin{bmatrix} \frac{1}{(\mu+\delta_1)} & 0 & 0 & 0 \\ 0 & \frac{1}{(\mu+\delta_2)} & 0 & 0 \\ 0 & 0 & \frac{1}{(\mu+\delta_3)} & 0 \\ 0 & 0 & 0 & \frac{1}{\lambda\gamma(\hat{\theta}+\hat{\tau})} \end{bmatrix}.$$

Then we get

$$\boldsymbol{G} = \boldsymbol{F}\boldsymbol{V}^{-1} = \begin{bmatrix} 0 & 0 & 0 & \frac{\lambda\alpha(1-\xi)}{\lambda\gamma(\hat{\theta}+\hat{\tau})} \\ \frac{\delta_1}{(\mu+\delta_1)} & 0 & 0 & 0 \\ 0 & \frac{\delta_2}{(\mu+\delta_2)} & 0 & 0 \\ \frac{\lambda\gamma}{(\mu+\delta_1)} & \frac{\lambda\gamma}{(\mu+\delta_2)} & \frac{\lambda\gamma}{(\mu+\delta_3)} & 0 \end{bmatrix}.$$

R_0 is the spectral radius of matrix given by $det(FV^{-1} - \omega I) = 0$, so

$$= \begin{bmatrix} 0-\omega & 0 & 0 & \frac{\lambda\alpha(1-\xi)}{\lambda\gamma(\hat{\theta}+\hat{\tau})} \\ \frac{\delta_1}{(\mu+\delta_1)} & 0-\omega & 0 & 0 \\ 0 & \frac{\delta_2}{(\mu+\delta_2)} & 0-\omega & 0 \\ \frac{\lambda\gamma}{(\mu+\delta_1)} & \frac{\lambda\gamma}{(\mu+\delta_2)} & \frac{\lambda\gamma}{(\mu+\delta_3)} & 0-\omega \end{bmatrix} = 0.$$

$$f(\omega) = \omega^4 - \frac{\lambda\alpha(1-\xi)}{\lambda\gamma(\hat{\theta}+\hat{\tau})}\left[\frac{\delta_1}{(\mu+\delta_1)}\left[\left(\frac{\delta_2}{(\mu+\delta_2)}\right)\left(\frac{\lambda\gamma}{(\mu+\delta_3)}\right) + \frac{\lambda\gamma\omega}{(\mu+\delta_2)}\right] + \frac{\lambda\gamma\omega^2}{(\mu+\delta_1)}\right] = 0$$

$$f(\omega) = \omega^4 - \frac{\lambda\alpha(1-\xi)}{(\hat{\theta}+\hat{\tau})}\left[\frac{\delta_1\delta_2}{(\mu+\delta_1)(\mu+\delta_2)(\mu+\delta_3)} + \frac{\delta_1\omega}{(\mu+\delta_1)(\mu+\delta_2)}\right] + \frac{\omega^2}{(\mu+\delta_1)}\Big] = 0$$

So we define

$$R_0 = \frac{L\lambda\alpha(1-\xi)}{(\mu+\delta_1)(\hat{\tau}+\hat{\theta})}. \tag{4.9}$$

$$L = 1 + \frac{\delta_1}{(\mu+\delta_2)} + \frac{\delta_1\delta_2}{(\mu+\delta_2)(\mu+\delta_3)}.$$

This is the value of R_0 obtained by Lewis and Greenhalgh (2001) using their definition of R_0. We shall show that it has the same threshold value as R_0^* the value defined by the next generation matrix method.

We argue as in Greenhalgh and Al-Rashidi (2022). As $f(0)$ is negative and $f(\omega)$ becomes large and positive as ω becomes large, the equation $f(\omega) = 0$ has either one or three roots on the positive axis. As there is only one change of sign in the coefficients Descartes' rule of signs says that there is just one positive real root. Additionally, because the Next Generation matrix (NGM) is positive and irreducible its largest absolute eigenvalue is R_0^*, the NGM basic reproduction number. Additionally $f(1) = 1 - R_0$. So R_0 exceeds one exactly when R_0^* does. Hence $R_0$and R_0^* pass through one together as the parameters vary.

4.3 Analysis of Three-Stage Infectivity Model

4.3.1 Existence of Unique Non-negative Solution

To study the existence of a unique non-negative solution of the model (4.5)-(4.8), we require to apply the concept of a Lipschitz continuous functions and the

Picard–Lindelöf theorem.

Theorem 4.3.1. *Assume that ϕ is Lipschitz continuous and differentiable in $\sum_{i=1}^{3} \pi_i$ for $0 \leq \pi_i \leq 1$. For any given initial value condition in in the region D=$[0,1]^4$ in R^4,the system of the model has a unique non-negative solution that remains in D for all time, moreover.*

(A) The first case is that we assume that $\xi < 1$ and $\phi(\pi) > 0$, for $1 \geq \pi \geq 0$.

(B) The second case is that we assume that $\xi < 1$ and $\exists\ \pi^*$ with $1 \geq \pi^* \geq 0$ such that $\phi(\pi^*) = 0$.

(C) The third case is that we assume that $\xi = 1$.

1. $\beta(0) = 0,\ \pi(0) > 0.$

2. $\beta(0) > 0\ ,\ \pi(0) = 0.$

3. $\beta(0) > 0\ ,\ \pi(0) > 0\ ,\ 1 - \pi(0) > 0.$

4.$\beta(0) > 0,\ \pi(0) > 0,\ 1 - \pi(0) = 0.$

Proof: According to the definition of Lipschitz continuous functions and The Picard Lindelof Theorem, we defined

$$\frac{d\pi_1}{dt} = f_1(\mathbf{x}x, \mathbf{y}) \qquad \frac{d\pi_2}{dt} = f_2(\mathbf{x}, \mathbf{y}) \tag{4.10}$$

$$\frac{d\pi_3}{dt} = f_3(\mathbf{x}, \mathbf{y}) \qquad \frac{d\pi_4}{dt} = f_4(\mathbf{x}, \mathbf{y}) \tag{4.11}$$

So if $\pi = \pi_1 + \pi_2 + \pi_3$, we can write $\mathbf{x} = (\pi_1^x, \pi_2^x, \pi_3^x, \beta^x)$ and $\mathbf{y} = (\pi_1^y, \pi_2^y, \pi_3^y, \beta^y)$. So we have

$$||\mathbf{f}(\mathbf{x}) - \mathbf{f}(\mathbf{y})|| \leq L||\mathbf{x} - \mathbf{y}||. \tag{4.12}$$

Then we now split the definition to four parts to prove that the function f is Lips-

chitz continuous

$$\begin{aligned}
|\mathbf{f}_1(\mathbf{x}) - \mathbf{f}_1(\mathbf{y})| &\leq L_1 |\mathbf{x} - \mathbf{y}|, \\
|\mathbf{f}_2(\mathbf{x}) - \mathbf{f}_2(\mathbf{y})| &\leq L_2 |\mathbf{x} - \mathbf{y}|, \\
|\mathbf{f}_3(\mathbf{x}) - \mathbf{f}_3(\mathbf{y})| &\leq L_3 |\mathbf{x} - \mathbf{y}|, \\
|\mathbf{f}_4(\mathbf{x}) - \mathbf{f}_4(\mathbf{y})| &\leq L_4 |\mathbf{x} - \mathbf{y}|,
\end{aligned} \tag{4.13}$$

where $L_1 + L_2 + L_3 + L_4 = L \in L \geq 0$ for L, here L_1, L_2, L_3 and L_4 represent Lipschitz constant for the function $\mathbf{f}_1, \mathbf{f}_2, \mathbf{f}_3$ and $\mathbf{f}_4$ respectively. To obtain these inequalities we have assumed that the function **f** is Lipschitz continuous. We start with the first part

$$|\mathbf{f}_1(\mathbf{x}) - \mathbf{f}_1(\mathbf{y})| \leq L_1 |\mathbf{x} - \mathbf{y}|. \tag{4.14}$$

Consider the first term on the right hand side of the equation (4.14)

$$\left|[(1-\pi^x)\lambda\alpha\beta^x\phi(\pi^x)(1-\xi) - (\mu+\delta_1)\pi_1^x] - [(1-\pi^y)\lambda\alpha\beta^y\phi(\pi^y)(1-\xi) - (\mu+\delta_1)\pi_1^y]\right|,$$

by using the triangle inequality

$$\begin{aligned}
&\leq \left|[(1-\pi^x)\lambda\alpha\beta^x\phi(\pi^x)(1-\xi) - (1-\pi^y)\lambda\alpha\beta^y\phi(\pi^y)(1-\xi)]\right| \\
&\quad + (\mu+\delta_1)\left|\pi_1^x - \pi_1^y\right|, \\
&\leq \Big|[(1-\pi^x)\lambda\alpha\beta^x\phi(\pi^x)(1-\xi) - (1-\pi^y)\lambda\alpha\beta^x\phi(\pi^x)(1-\xi) \\
&\quad + (1-\pi^y)\lambda\alpha\beta^x\phi(\pi^x)(1-\xi) - (1-\pi^y)\lambda\alpha\beta^y\phi(\pi^x)(1-\xi) \\
&\quad + (1-\pi^y)\lambda\alpha\beta^y\phi(\pi^x)(1-\xi) - (1-\pi^y)\lambda\alpha\beta^y\phi(\pi^y)(1-\xi)]\Big| \\
&\quad + (\mu+\delta_1)\left|\pi_1^x - \pi_1^y\right|, \\
&\leq \left|(1-\pi^x)\lambda\alpha\beta^x\phi(\pi^x)(1-\xi) - (1-\pi^y)\lambda\alpha\beta^x\phi(\pi^x)(1-\xi)\right| \\
&\quad + \left|(1-\pi^y)\lambda\alpha\beta^x\phi(\pi^x)(1-\xi) - (1-\pi^y)\lambda\alpha\beta^y\phi(\pi^x)(1-\xi)\right| \\
&\quad + \left|(1-\pi^y)\lambda\alpha\beta^y\phi(\pi^x)(1-\xi) - (1-\pi^y)\lambda\alpha\beta^y\phi(\pi^y)(1-\xi)\right| \\
&\quad + (\mu+\delta_1)\left|\pi_1^x - \pi_1^y\right|,
\end{aligned}$$

$$
\begin{aligned}
&\leq \left|(\pi^x - \pi^y)\right|\left|\lambda\alpha\beta^x\phi(\pi^x)(1-\xi)\right| \\
&\quad + \left|(\beta^x - \beta^y)\right|\left|(1-\pi^y)\lambda\alpha\phi(\pi^x)(1-\xi)\right| \\
&\quad + \left|\phi(\pi^x) - \phi(\pi^y)\right|\left| - (1-\pi^y)\lambda\alpha\beta^y\phi(\pi^y)(1-\xi)\right| \\
&\quad + (\mu+\delta_1)\left|\pi_1^x - \pi_1^y\right|.
\end{aligned}
$$

That implies that

$$
\begin{aligned}
&\leq K_1\left|(\pi^x - \pi^y)\right| + K_2\left|(\beta^x - \beta^y)\right| + K_3\left|\phi(\pi^x) - \phi(\pi^y)\right| + K_4\left|\pi_1^x - \pi_1^y\right|. \\
&\leq K\left|\mathbf{x} - \mathbf{y}\right|.
\end{aligned}
$$

Where $K = K_1 + K_2 + K_3 + K_4$.

Next, we going to prove the second part

$$
\begin{aligned}
&\left|f_2(\mathbf{x}) - f_2(\mathbf{y})\right| \leq L_2\left|\mathbf{x} - \mathbf{y}\right|. \\
&\left|[(\delta_1\pi_1^x - (\mu+\delta_2)\pi_2^x] - [\delta_1\pi_1^y - (\mu+\delta_2)\pi_2^y]\right|.
\end{aligned}
\tag{4.15}
$$

By using the triangle inequality, we get

$$
\begin{aligned}
&\leq \left|\pi_1^x - \pi_1^y\right|\delta_1 + \left|\pi_2^x - \pi_2^y\right|(\mu+\delta_2). \\
&\leq K_5\left|\mathbf{x} - \mathbf{y}\right|.
\end{aligned}
$$

Similarly, for the third part

$$
\begin{aligned}
&\left|f_3(\mathbf{x}) - f_3(\mathbf{y})\right| \leq L_3\left|\mathbf{x} - \mathbf{y}\right|. \\
&\left|[\delta_2\pi_2^x - (\mu+\delta_3)\pi_3^x] - [\delta_2\pi_2^y - (\mu+\delta_3)\pi_3^y]\right|.
\end{aligned}
\tag{4.16}
$$

Then we have

$$
\begin{aligned}
&\leq \left|\pi_2^x - \pi_2^y\right|\delta_2 + \left|\pi_3^x - \pi_3^y\right|(\mu+\delta_3). \\
&\leq K_6\left|\mathbf{x} - \mathbf{y}\right|.
\end{aligned}
$$

Similarly for the fourth part

$$
\left|f_4(\mathbf{x}) - f_4(\mathbf{y})\right| \leq L_4\left|\mathbf{x} - \mathbf{y}\right|. \tag{4.17}
$$

To see this consider the right-hand side of the equation (4.17)

$$\begin{aligned}
&\Big|[(1-\beta^x)\lambda\gamma\phi(\pi^x)(\pi^x) - \beta^x\lambda\gamma\phi(\pi^x)(1-\pi^x)(1-(1-\theta)(1-\xi)) - \beta^x\tau] \\
&-[(1-\beta^y)\lambda\gamma\phi(\pi^y)(\pi^y) - \beta^y\lambda\gamma\phi(\pi^y)(1-\pi^y)(1-(1-\theta)(1-\xi)) - \beta^y\tau]\Big|, \\
&=\Big|[(1-\beta^x)\lambda\gamma\phi(\pi^x)(\pi^x) - (1-\beta^y)\lambda\gamma\phi(\pi^y)(\pi^y) + \beta^x\lambda\gamma\phi(\pi^x)(1-\pi^x)(1-(1-\theta)(1-\xi)) \\
&-\beta^y\lambda\gamma\phi(\pi^y)(1-\pi^y)(1-(1-\theta)(1-\xi)) + \beta^x\tau - \beta^y\tau]\Big|.
\end{aligned}$$

After applying the triangle inequality, we have that this quantity is bounded above by

$$\begin{aligned}
&\leq\Big|(1-\beta^x)\lambda\gamma\phi(\pi^x)(\pi^x) - (1-\beta^y)\lambda\gamma\phi(\pi^x)(\pi^x)\Big| \\
&+\Big|(1-\beta^y)\lambda\gamma\phi(\pi^x)(\pi^x) - (1-\beta^y)\lambda\gamma\phi(\pi^y)(\pi^x)\Big| \\
&+\Big|(1-\beta^y)\lambda\gamma\phi(\pi^y)(\pi^x) - (1-\beta^y)\lambda\gamma\phi(\pi^y)(\pi^y)\Big| \\
&+\Big|\beta^x\lambda\gamma\phi(\pi^x)(1-\pi^x)(1-(1-\theta)(1-\xi)) - \beta^y\alpha\gamma\phi(\pi^x)(1-\pi^x)(1-(1-\theta)(1-\xi))\Big| \\
&+\Big|\beta^y\lambda\gamma\phi(\pi^x)(1-\pi^y)(1-(1-\theta)(1-\xi)) - \beta^y\alpha\gamma\phi(\pi^y)(1-\pi^x)(1-(1-\theta)(1-\xi))\Big| \\
&+\Big|\beta^y\lambda\gamma\phi(\pi^y)(1-\pi^x)(1-(1-\theta)(1-\xi)) - \beta^y\alpha\gamma\phi(\pi^y)(1-\pi^y)(1-(1-\theta)(1-\xi))\Big| \\
&+\Big|\beta^x-\beta^y\Big|\tau \\
&\leq\Big|\beta^x-\beta^y\Big|\lambda\gamma\phi(\pi^x)\pi^x \\
&+\Big|\phi(\pi^x)-\phi(\pi^y)\Big|(1-\beta^y)\lambda\gamma\pi^x \\
&+\Big|\pi^x-\pi^y\Big|(1-\beta^y)\lambda\gamma\phi(\pi^y) \\
&+\Big|\beta^x-\beta^y\Big|\lambda\gamma\phi(\pi^x)(1-\pi^x)(1-(1-\theta)(1-\xi) \\
&+\Big|\phi(\pi^x)-\phi(\pi^y)\Big|\beta^y\lambda\gamma(1-\pi^y)(1-(1-\theta)(1-\xi)) \\
&+\Big|\pi^x-\pi^y\Big|\beta^y\gamma\phi(\pi^y)(1-(1-\theta)(1-\xi) \\
&+\Big|\beta^x-\beta^y\Big|\tau \\
&\quad\leq K_7\Big|\mathbf{x}-\mathbf{y}\Big|,
\end{aligned}$$

For some constant K_7. This finishes the proof of Lipschitz continuity.

Now, we will apply the definition of the Picard–Lindelöf Theorem to prove the cases below, using the same technique we used in earlier chapters to finish the

proof.

We define $\psi = 1 - \pi$, and $\chi = 1 - \beta$. We will divide the proof into three cases.

(A) The first case is that we assume that $\xi < 1$ and $\phi(\pi) > 0$, for $1 \geq \pi \geq 0$.

(B) The second case is that we assume that $\xi < 1$ and $\exists\ \pi^*$ with $1 \geq \pi^* \geq 0$ such that $\phi(\pi^*) = 0$.

(C) The third case is that we assume that $\xi = 1$.

Now we will consider four initial conditions.

1. $\beta(0) = 0,\ \ \pi(0) > 0.$

2. $\beta(0) > 0$, $\pi(0) = 0.$

3. $\beta(0) > 0$, $\pi(0) > 0$, $\ 1 - \pi(0) > 0.$

4.$\beta(0) > 0$, $\pi(0) > 0$, $1 - \pi(0) = 0.$

Case A: $\xi < 1$ and $\phi(\pi) > 0$, for $1 \geq \pi \geq 0$.

Case A One: $\beta(0) = 0,\ \ \pi(0) > 0.$

First suppose that $\beta(0) = 0$ and $1 \geq \pi(0) > 0$. Let us define $(0, \tau_e)$ to be the maximal interval where a solution exists and

$1 > \beta(s) > 0,\ \pi_1(s) > 0,\ \pi_2(s) > 0,\ \pi_3(s) > 0$, and $1 > \pi(s)$ for $s \in (0, \tau_e)$.

We suppose first that $\tau_e < \infty$. By using the Picard–Lindelöf Theorem $\exists\ \Delta t > 0$ such that the solution exists in $[0, \Delta t]$.

By using Taylor series expansions about $t = 0$ and the appropriate model equations gives

$$\pi(\Delta t) = \pi(0) - \mu\pi(0)\Delta t - \delta_3\pi_3(0)\Delta t + o(\Delta t).$$

$$\beta(\Delta t) = \pi(0)\lambda\gamma\phi(\pi(0))\Delta t + o(\Delta t).$$

If Δt is small enough then $\pi(\Delta t) \geq k_1\Delta t$ and $\beta(\Delta t) \geq k_2\Delta t$, for some constants k_1 and k_2 with $min(k_1, k_2) > 0$.

If $\psi(0) > 0$, then clearly $\psi(\Delta t) \geq k_3 \Delta t$ for some $k_3 > 0$, for Δt small and strictly positive. If $\psi(0) = 0$ then as

$$\frac{d\psi}{dt} = -\lambda(1-\xi)\psi\alpha\beta\phi(\pi) + \mu\pi + \delta_3\pi_3.$$

We have $\psi(\Delta t) \geq \mu\Delta t + o(\Delta t)$ and the same result is true.

If $\pi_1(0) = 0$ then $\frac{d\pi_1}{dt}\Big|_{t=0} = 0$. As ϕ is twice differentiable with respect to π

$$\frac{d^2\pi_1}{dt^2}\Big|_{t=0} = (1-\pi)\lambda\alpha\frac{d\beta}{dt}\phi(\pi)(1-\xi)\Big|_{t=0}.$$

So if $\psi(0) > 0$ then $\frac{d^2\pi_1}{dt^2}\Big|_{t=0} > 0$ and

$$\pi_1(t) \geq k_4\Delta t^2 + o(\Delta t^2) \qquad \textit{for some} \quad k_4 > 0.$$

If $\psi(0) = 0$ then $\frac{d^2\pi_1}{dt^2}\Big|_{t=0} = 0$, but as ϕ is three times differentiable with respect to π

$$\frac{d^3\pi_1}{dt^3} = \frac{d\psi}{dt}\lambda\alpha\frac{d\beta}{dt}\phi(\pi)(1-\xi)\Big|_{t=0} > 0.$$

So in this case, we have

$$\pi_1(t) \geq k_4\Delta t^3 \qquad \text{for some } k_4 > 0,$$

($\psi(0) > 0$ or $\psi(0) = 0$), so in either case if Δt is sufficiently small $\pi_1(t) \geq k_4\ \Delta t^3$ for some $K_4 > 0$. A similar argument shows that if Δt is small enough.

$$\pi_2(t) \geq k_5\Delta t^4 \qquad \textit{for some } k_5 > 0.$$

$$\textit{and} \qquad \pi_3(t) \geq k_6\Delta t^2 \qquad \textit{for some } k_6 > 0.$$

Note that $\pi(0) > 0$, implies that $\pi_1(0) > 0, \pi_2(0) > 0$ or $\pi_3(0) > 0$. Hence if Δt is sufficiently small and strictly positive, $1 > \beta(s) > 0, \pi_1(s) > 0, \pi_2(s) > 0, \pi_3(s) > 0$ and $1 > \pi(s)$ for s $\in (0, \Delta t]$ so $\tau_e \geq \Delta t > 0$.

Now for Δt small and strictly positive $\pi_1(\Delta t) > 0$ and

$$\frac{d\pi_1}{dt} \geq -(\mu + \delta_1)\pi_1 \qquad \in [\Delta t, \tau_e).$$

Hence, for $t \in [\Delta t, \tau_e)$

$$\pi_1(t) \geq \pi_1(\Delta t)e^{-(\mu+\delta_1)(t-\Delta t)}.$$

Similarly for $t \in [\Delta t, \tau_e)$, we have

$$\begin{aligned}
\pi_2(t) &\geq \pi_2(\Delta t)e^{-(\mu+\delta_2)(t-\Delta t)}, \\
\pi_3(t) &\geq \pi_3(\Delta t)e^{-(\mu+\delta_3)(t-\Delta t)}, \\
\beta(t) &\geq \beta(\Delta t)e^{-(\lambda\gamma+\tau)(t-\Delta t)}, \\
\psi(t) &\geq \psi(\Delta t)e^{-\int_{\Delta t}^{t} \lambda\alpha\beta\phi(\pi(s))(1-\xi)ds}, \\
&\geq \psi(\Delta t)e^{-\lambda\alpha(t-\Delta t)(1-\xi)}, \\
\chi(t) &\geq \psi(\Delta t)e^{-\int_{\Delta t}^{t} \pi(s)\lambda\gamma\phi(\pi(s))ds}, \\
&\geq \chi(t)(\Delta t)e^{-\lambda\gamma(t-\Delta t)}.
\end{aligned}$$

Now by the Picard–Lindelöf Theorem there exists a unique local solution to the equation in $[\tau_e - \xi, \tau_e + \xi]$ for some $\xi > 0$. As the unique solution is continuous at τ_e.

$$\begin{aligned}
\pi_1(\tau_e) &\geq \lim_{t\to\tau_e} \pi_1(t), \\
&= \pi_1(\Delta t)e^{-(\mu+\delta t)(\tau_e-\Delta t)} > 0.
\end{aligned}$$

Similarly $\pi_2(\tau_e)$, $\pi_3(\tau_e), \beta(\tau_e), \psi(\tau_e)$, and $\chi(\tau_e)$ are all strictly positive. So by continuity the solution can be extended past τ_e with $1 > \beta(t) > 0, \pi_1(t) > 0, \pi_2(t) > 0, \pi_3(t) > 0$ and $1 > \pi(t)$ for $t \in (0, \tau_e + \xi)$ some $\xi > 0$.

This contradicts the definition of τ_e so $\tau_e = \infty$. This completes the proof of Case (A) one.

Case A Two: $\beta(0) > 0$, $\pi(0) = 0$.

Arguing as in Case A one as $\pi_1(0) = 0$.

$$\pi_1(\Delta t) = \lambda\alpha\beta(0)(1 - \xi)\Delta t + o(\Delta t).$$

So $\pi_1\ (\Delta t) \geq k_1\ \Delta t$ where $k_1 > 0$ for all Δt sufficiently small. Arguing as previously. $\pi_2(\Delta t) \geq k_2\ \Delta t^2$ where $k_2 > 0$, and $\pi_3(\Delta t) \geq k_3\Delta t^3$ where $k_3 > 0$ for all Δt sufficiently small.

$$\begin{aligned}
\beta(\Delta t) &= \beta(0)\Big[1 - \Big\{\tau + \lambda\gamma(1 - (1 - \theta)(1 - \xi))\Big\}\Big]\Delta t + o(\Delta t), \\
\psi(\Delta t) &= 1 - \lambda\alpha\beta(0)(1 - \xi)\Delta t > 0.
\end{aligned}$$

So $\beta(\Delta t) \geq k_4 \Delta t$ and $\psi(\Delta t) \geq k_5 \Delta t$, for some k_4, $k_5 > 0$ and Δt sufficiently small. If $\chi(0) = 0$, then $\chi(\Delta t) \geq \tau \Delta t + o(\Delta t)$, so if $\Delta t > 0$ is sufficiently small. $\chi(s) \geq k_6 s$ for $s \in (0, \Delta t]$ for some $k_6 > 0$. This inequality is clearly true if $\chi(0) > 0$ so is true whatever the value of χ.

Hence arguing as in Case (A) one if Δt is sufficiently small and positive $\tau_e \geq \Delta t$. The proof proceeds as in Case (A) one.

Case A Three: $\beta(0) > 0,\ \pi(0) > 0,\ 1 - \pi(0) > 0.$

Suppose that $\beta(0) > 0, \pi(0) > 0$ and $\psi(0) > 0$. It is straightforward to show that the result holds in this case, using the previous argument

Case A Four: $\beta(0) > 0,\ \pi(0) > 0,\ 1 - \pi(0) = 0.$

Suppose that $\beta(0) > 0$, $\pi(0) > 0$ and $\psi(0) = 0$ so $\pi(0) = 1$. The proof proceeds as above using arguments from Case (A) one and Case (A) two.This completes the proof of Theorem (4.3.1) in Case (A) where $\xi < 1$ and $\phi(\pi) > 0$, for $1 \geq \pi \geq 0$. We now move on to Case (B) where there is $\xi < 1$ and $\exists\ \pi^*$ with $1 \geq \pi^* \geq 0$ such that $\phi(\pi^*) = 0$.

Case B: $\xi < 1$ and $\exists\ \pi^*$ with $1 \geq \pi^* \geq 0$ such that $\phi(\pi^*) = 0$.

We shall consider three Cases for this case, the first one (I) is $\pi(0) < \pi^*$ and the second one (II) is $\pi(0) = \pi^*$ and the third one (III) is $\pi(0) > \pi^*$.

Case B(I) : If $\pi(0) < \pi^*$, then arguing as above $\exists \Delta t > 0$ with $\tau_e \geq \Delta t > 0$. Now as $\pi \to \pi^{*-}$

$$\frac{d\pi}{dt} \to -\mu\pi^* - \delta_3\pi_3 \leq -\mu\pi^*.$$

So $\exists\ \pi^+ < \pi^*$ such that for $\pi \in [\pi^+, \pi^*]$ π is strictly monotone decreasing. So if π starts beneath π^* it can never reach it and $\pi \leq max(\pi(0), \pi^+) < \pi^*\ \forall t$. Hence the proof proceeds as in the previous case.

Case B(II) : If $\pi(0) = \pi^*$, then for Δt small and strictly positive

$$\pi(\Delta t) = \pi^* - (\mu\pi^* + \delta_3\pi_3(0))\Delta t + o(\Delta t).$$

So if Δt is sufficiently small and strictly positive, then we have

$$\pi(s) \leq \pi^* - \frac{1}{2}\mu\pi^* s \qquad \textit{for } s \in (0, \Delta t].$$

Since we know that the ϕ is differentiable, we assume that there is some strictly positive integer $k \geq 1$ with

$$(-1)^k \frac{d^k \phi'}{d\pi^k}\bigg|_{\pi^{*-}} > 0. \qquad \textit{and}$$
$$(-1)^l \frac{d^l \phi'}{d\pi^l}\bigg|_{\pi^{*-}} = 0 \qquad \textit{for } 0 \leq l < k.$$

Case B(II) One: First suppose that $\beta(0) = 0$ and $\pi(0) = \pi^* > 0$. Let us define $(0, \tau_e)$ to be the maximal interval where a solution exists and

$$1 > \beta(s) > 0, \pi_1(s) > 0, \pi_2(s) > 0, \pi_3(s) > 0 \quad \textit{and } \pi^* > \pi(s) \quad \textit{for } s \in (0, \tau_e).$$

We first suppose that $\tau_e < \infty$. By using the Picard–Lindelöf Theorem $\exists$ $\Delta t > 0$ such that the solution exists in $[0, \Delta t)$.

$$\frac{d\beta^+}{dt}\bigg|_{t=0^+} = 0.$$
$$\frac{d^2\beta^+}{dt^2}\bigg|_{t=0^+} = \pi^* \lambda\gamma \frac{d\phi(\pi)}{dt}\bigg|_{t=0^+}$$

As $\frac{d\beta^+}{dt}\Big|_{t=0} = 0$ is the right hand side derivative,now

$$\begin{aligned}
\frac{d\phi(\pi)}{dt}\bigg|_{t=0^+} &= \lim_{\Delta t \to 0^+} \frac{\phi(\pi(\Delta t)) - \phi(\pi(0))}{\Delta t}, \\
&= \lim_{\Delta t \to 0^+, \Delta\pi \to 0} \frac{\phi(\pi^* + \Delta\pi) - \phi(\pi^*)}{\Delta\pi} \frac{\Delta\pi}{\Delta t}, \\
&= \frac{d\phi}{d\pi}\bigg|_{\pi=\pi^{*-}} \frac{d\pi}{dt}\bigg|_{t=0+}, \\
&= -(\mu\pi^* + \delta_3\pi_3(0)) \frac{d\phi}{d\pi}\bigg|_{\pi=\pi^{*-}} > 0.
\end{aligned}$$

So if $k = 1$ then $\frac{d^2\beta}{dt^2}\Big|_{t=0^+} > 0$. On the other hand if $\frac{d\phi}{d\pi}\Big|_{\pi=\pi^{*-}} = 0$, but

$$\frac{d^2\phi(\pi)}{d\pi^2}\bigg|_{\pi=\pi^{*-}} > 0.$$

then

$$\frac{d^2\beta^+}{dt^2}\bigg|_{t=0^+} = 0.$$

Now note that

$$\frac{d\phi}{dt} = \frac{d\phi}{d\pi}\frac{d\pi}{dt}.$$

Then

$$\begin{aligned}
\frac{d^2\phi}{dt^2} =& \frac{d^2\phi}{d\pi^2}\left(\frac{d\pi}{dt}\right)^2 + \textit{terms involving } \frac{d\phi}{d\pi},\\
=& \frac{d^2\phi}{d\pi^2}\left(\frac{d\pi}{dt}\right)^2 \qquad \textit{at } t = 0.\\
\left.\frac{d^3\beta^+}{dt^3}\right|_{t=0} =& \pi^*\lambda\gamma \left.\frac{d^2\phi(\pi)}{dt^2}\right|_{t=0+},\\
=& \pi^*\lambda\gamma \left.\frac{d^2\phi(\pi)}{dt^2}\right|_{\pi=\pi^{*-}} \left(\left.\frac{d\pi}{dt}\right|_{t=0+}\right)^2,\\
=& \pi^*\lambda\gamma\Big(\mu\pi^* + \delta_3\pi(0)\Big)^2 \left.\frac{d^2\phi(\pi)}{d\pi^2}\right|_{\pi=\pi^{*-}} > 0.
\end{aligned}$$

Similarly in general

$$\left.\frac{d^l\beta}{dt^l}\right|_{t=0^+} = 0 \quad \textit{for} \quad l = 0, 1, ..., k \quad \textit{and} \quad \left.\frac{d^{k+1}\beta}{dt^{k+1}}\right|_{t=0^+} > 0.$$

So if Δt is small enough, then $\pi(s) \le \pi^* - k_1 s$ and $\beta(s) \ge k_2 s^{k+1}$ for $s \in (0, \Delta t]$ for some $k_1, k_2 > 0$. Arguing as in the previous case

$$\psi(\Delta t) \ge k_3 \Delta t + o(\Delta t) \qquad \textit{for some } k_3 > 0.$$

A similar argument to above shows that if $\pi_1(0) = 0$ and $\psi(0) > 0$ then for $0 \le m \le 2k + 1$ then

$$\left.\frac{d^m\pi_1}{dt^m}\right|_{t=0^+} = 0,$$

and

$$\begin{aligned}
\left.\frac{d^{2k+2}\pi_1}{dt^{2k+2}}\right|_{t=0^+} =& (1-\pi^*)\lambda\alpha(1-\xi)\left.\frac{d^{k+1}}{dt^{k+1}}\beta\right|_{t=0^+}\left.\frac{d^k\phi}{d\pi^k}\right|_{t=0^+},\\
=& (1-\pi^*)\lambda\alpha(1-\xi)\left.\frac{d^{k+1}}{dt^{k+1}}\beta\right|_{t=0^+}\left.\frac{d^k\phi}{d\pi^k}\right|_{\pi=\pi^{*-}}\left(\left.\frac{d\pi}{dt}\right|_{t=0^+}\right)^k > 0.
\end{aligned}$$

On the other hand if $\psi(0) = 0$ then for $0 \le m \le 2k + 2$.

$$\left.\frac{d^m\pi_1}{dt^m}\right|_{t=0^+} = 0.$$

and

$$\left.\frac{d^{2k+3}\pi_1}{dt^{2k+3}}\right|_{t=0^+} = \lambda\alpha(1-\xi)\left.\frac{d\psi}{dt}\right|_{t=0^+}\left.\frac{d^{k+1}}{dt^{k+1}}\beta\right|_{t=0^+}\left.\frac{d^{k+1}}{dt^{k+1}}\beta\right|_{t=0^+}\left.\frac{d^k\phi}{d\pi^k}\right|_{\pi=\pi^{*-}}\left(\left.\frac{d\pi}{dt}\right|_{t=0^+}\right)^k > 0.$$

So in all cases $\pi_1(s) \geq k_4 s^{2k+3}$ for $s \in (0, \Delta t]$ if Δt is small enough for some constant $k_4 > 0$ also obviously true if $\pi_1(0) > 0$.

If $\pi_2(0) > 0$ then $\pi_2(s) \geq k_5\ s$ for $s \in (0, \Delta t]$ for Δt sufficiently small for some $k_5 > 0$. On the other hand, if $\pi_2(0) = 0$, but $\pi_1(0) > 0$ then $\pi_2(s) \geq k_5 s$ for $s \in (0, \Delta t]$ for Δt sufficiently small for some $k_5 > 0$. If $\pi_1(0) = \pi_2(0) = 0$ then a similar argument to above shows that $\pi_2(s) \geq k_5 s^{2k+4}$ for $s \in (0, \Delta t]$ for some $k_5 > 0$. So in all cases $\pi_2(s) \geq k_5\ s^{2k+4}$ for $s \in (0, \Delta t]$ for some $k_5 > 0$. If $\pi_3(0) > 0$ then, $\pi_3(0) \geq K_6 s$ for $s \in (0, \Delta t]$ for Δt sufficiently small for some $k_6 > 0$. If $\pi_3(0) = 0$, but $\pi_2(0) > 0$ then $\pi_3(s) \geq k_6 s$ for $s \in (0, \Delta t]$ for Δt sufficiently small for some $k_6 > 0$. If $\pi_2(0) = \pi_3(0) = 0$, then $\pi_1(0) > 0$ and $\pi_3(s) \geq k_6 s^2$ for $s \in (0, \Delta t]$ for Δt sufficiently small for some $k_6 > 0$. So in all cases $\pi_3(s) \geq k_6 s^2$ for $s \in (0, \Delta t]$ for some $k_6 > 0$. Hence for Δt sufficiently small and strictly positive

$$1 > \beta(s) > 0, \pi_1(s) > 0, \pi_2(s) > 0, \pi_3(s) > 0 \quad \textit{and}$$

$$1 \geq \pi^* > \pi(s) \quad \textit{for } s \in (0, \Delta t] \quad \textit{so} \quad \tau_e \geq \Delta t > 0.$$

By the argument for $\pi(0) < \pi^*$ we see that

$$\pi(t) < max(\pi(\Delta t), \pi^+) < \pi^* \ in\ [\Delta t, \tau_e).$$

Arguing as in the Case(A) where $\phi(\pi) > 0 \forall \pi$ and $\xi < 1$ we deduce that $\tau_e = \infty$.

Case B(II) Tow: $\beta(0), \pi(0) = 0$ is not applicable here as $\pi(0) = \pi^* > 0$.

Case B(II) Three: $\beta(0) > 0, \pi(0) = \pi^* > 0$ and $\psi(0) > 0$ it is straight forward to show that the result holds in this case.

Case B(II) Four: $\beta(0) > 0, \pi(0) = \pi^* > 0$ and $\psi(0) = 0$ it is straight forward to show that the result holds in this case too. Hence the result of Theorem (4.3.1) holds in Case B(II)

Case B(III) : If $\pi(0) > \pi^*$, then for Δt small and strictly positive

$$\pi(\Delta t) = \pi(0) - (\mu\pi(0) + \delta_3\pi(0))\Delta t + o(\Delta t).$$

So provided that $\pi(0) \geq \pi \geq \pi^*$

$$\begin{aligned}
\frac{d\pi_1}{dt} &= -(\mu + \delta_1)\pi_1, \\
\frac{d\pi_2}{dt} &= \delta_1\pi_1 - (\mu + \delta_2)\pi_2, \\
\frac{d\pi_3}{dt} &= \delta_2\pi_2 - (\mu + \delta_3)\pi_3, \\
\frac{d\beta}{dt} &= -\beta\tau.
\end{aligned}$$

Define $(0, \tau_{e'})$ to be the maximal interval where a solution exists and

$$1 > \beta(s) \geq 0, \pi_1(s) \geq 0, \pi_2(s) \geq 0, \pi_3(s) \geq 0 \quad \textit{and} \quad \pi \geq \pi^* > 0.$$

It is straightforward to show that if Δt is sufficiently small and positive $\tau_{e'} \geq \Delta t > 0$, moreover for $t \in (0, \tau_{e'})$,

$$\frac{d\pi}{dt} = -\mu\pi - \delta_3\pi_3 \leq -\mu\pi.$$

Hence

$$\pi^* \leq \pi \leq \pi(0)e^{-\mu t} \leq e^{-\mu t}.$$

So

$$\tau_{e'} \leq -\frac{1}{\mu}\log\pi^* < \infty.$$

The proof shows that $\lim_{t\to\tau_{e'}} \beta$, π_1, π_2 and π_3 exist and satisfy

$$1 > \lim_{t\to\tau_{e'}} \beta, \lim_{t\to\tau_{e'}} \pi_1, \lim_{t\to\tau_{e'}} \pi_2, \lim_{t\to\tau_{e'}} \pi_3 \geq 0.$$

If $\pi(\tau_{e'}) > \pi^*$ then the solution can be continued past $\tau_{e'}$ using the Picard-Lindelöf Theorem which is a contradiction. Hence $\pi(\tau_{e'}) = \pi^*$. For $t \geq \tau_{e'}$ the result follows by the case where $\pi(0) = \pi^*$ discussed previously. This completes the proof of Theorem (4.3.1) in Case B(III) and hence in Case B. We now move on to the proof of the Theorem in Case C where $\xi = 1$.

Case C: For $\xi = 1$ the equations are

$$\begin{aligned}
\frac{d\pi_1}{dt} &= -(\mu + \delta_1)\pi_1. \\
\frac{d\pi_2}{dt} &= \delta_1\pi_1 - (\mu + \delta_2)\pi_2. \\
\frac{d\pi_3}{dt} &= \delta_2\pi_2 - (\mu + \delta_3)\pi_3. \\
\frac{d\beta}{dt} &= \pi(1-\beta)\lambda\gamma\phi(\pi) - \beta\lambda\gamma\phi(\pi)(1-\pi) - \beta\tau.
\end{aligned}$$

As in Case B, we need to discuss three conditions, the first (I) is $\pi(0) < \pi^*$, the second (II) is $\pi(0) = \pi^*$ and the third (III) is $1 > \pi(0) > \pi^*$.

Case C(I) : $\pi(0) < \pi^*$.

Again $\pi \leq max(\pi(0), \pi^+)$ for all time.

Case C(I) One: $\beta(0) = 0$, $1 \geq \pi(0) > 0$.

Define $(0, \tau_e)$ to be the maximal interval where a solution exists and

If $\pi_1(0) = \pi_2(0) = 0$, then

$1 > \beta(s) > 0, \pi_1(s) = \pi_2(s) = 0, \pi_3(s) > 0$ *and* $1 > \pi(s)$ in $(0, \tau_e)$.

If $\pi_1(0) = 0, \pi_2(0) > 0$, then

$1 > \beta(s) > 0, \pi_1(s) = 0, \pi_2(s) > 0, \pi_3(s) > 0$ *and* $1 > \pi(s)$ in $(0, \tau_e)$.

If $\pi_1(0) > 0$, then

$1 > \beta(s) > 0, \pi_1(s) > 0, \pi_2(s) > 0, \pi_3(s) > 0$ *and* $1 > \pi(s)$ in $(0, \tau_e)$.

The proof proceeds as in the Case A where $\xi < 1$ and $\phi(\pi) > 0$ for $1 \geq \pi \geq 0$.

Case C(I)Two: $\beta(0) > 0, \pi(0) = 0$.

Define $(0, \tau_e)$ as in Case C (I) One to be the maximal interval where a solution exists and

$$1 > \beta(s) > 0, \pi_1(s) = \pi_2(s) = \pi_3(s) = 0 \in (0, \tau_e).$$

The unique solution is $\pi_1(t) = \pi_2(t) + \pi_3(t) = 0$ and $\beta(t) = \beta(0)e^{-(\lambda\gamma+\tau)t}$. So clearly $\tau_e = \infty$ and the Theorem (4.3.1) holds in this case.

Case C(I) Three: $\beta(0) > 0 \quad \pi(0) > 0$ and $\psi(0) > 0$.

Define $(0, \tau_e)$ as in Case C (I) One.

As in Case A where $\xi < 1$ and $\phi(\pi) > 0$for $1 \geq \pi \geq 0$, the proof of this case follows from the proof of Case C (I) One.

Case C(I) Four: Suppose that $\beta(0) > 0, \pi(0) > 0$ and $\psi(0) = 0$.

Define τ_e as in Case C (I) One.

As in Case A where $\xi < 1$ and $\phi(\pi) > 0$ for $1 \geq \pi \geq 0$, the proof of this case follows from the proof of Case C (I) One and Case C (I) Two. This completes the proof of Case C (I).

Case C(II) : $\pi(0) = \pi^*$.

Case C(II) One: $\beta(0) = 0, \pi(0) = \pi^* > 0$.

Define τ_e as in Case C (I) One with $\xi = 1$ and $\pi(0) < \pi^*$. The proof is straightforward using the ideas discussed previously.

Case C(II) Two: is not possible here as $\pi(0) > 0$.

Case C(II)Three: $\beta(0) > 0, \pi(0) > 0$ and $\psi(0) > 0$.

Define τ_e as in Case C (I) One with $\xi = 1$ and $\pi(0) < \pi^*$. The proof is straightforward using the ideas discussed above.

Case C(II) Four: $\beta(0) > 0, \pi(0) > 0$ and $\psi(0) = 0$.

Define τ_e as in Case C (I) One. The proof is straightforward using the ideas discussed above.

Case C(III) : $1 \geq \pi(0) > \pi^*$.

Notethat for $1 \geq \pi \geq \pi^*$ the differential equations are exactly the same as in Case B(III). The result then follows by the arguments in Case B(III) and the argument in Case C(II) above.

This completes the proof of Theorem (4.3.1) in Case C(III) hence in Case C, hence the overall proof of Theorem (4.3.1). Next we are going to find the existence of equilibrium solution for our model.

4.3.2 Existence of equilibrium

Theorem 4.3.2. *If ϕ is strictly monotonic decreasing and $R_0 \leq 1$, the system of equations* (4.5)-(4.8) *has a unique equilibrium solution* (DFE) *where the HIV/AIDS virus has died out in both PWIDs and needles. For $R_0 > 1$ there exists a unique positive endemic equilibrium solution* (EE) *as well as a disease-free equilibrium solution* (DFE)*.*

Proof. Let π_i^*, β^* represent the equilibrium values of PWIDs and needles respectively, where $i = 1,2,3$ and $\pi = \pi_1 + \pi_2 + \pi_3$.From the equilibrium versions of equations (4.6) and (4.7),we have the following

$$\pi_2^* = \frac{\delta_1 \pi_1^*}{\mu + \delta_2}. \tag{4.18}$$

$$\pi_3^* = \frac{\delta_2 \pi_2^*}{(\mu + \delta_3)}. \tag{4.19}$$

We have

$$\pi^* = \pi_1^* + \pi_2^* + \pi_3^* = \pi_1^* L,$$

where

$$L = 1 + \frac{\delta_1}{(\mu + \delta_2)} + \frac{\delta_1 \delta_2}{(\mu + \delta_2)(\mu + \delta_3)}.$$

The equation (4.5) gives

$$\beta^* = \frac{(\mu + \delta_1)\pi^*}{L(1 - \pi^*)\lambda\alpha\phi(\pi^*)(1 - \xi)}. \tag{4.20}$$

by using $\pi^* = \pi_1^* L$. The equation (4.8) becomes

$$\beta^* = \frac{\pi^* \phi(\pi^*)}{\pi^* \phi(\pi^*) + (1 - \pi^*)\phi(\pi^*)\hat{\theta} + \hat{\tau}}. \tag{4.21}$$

Recall $\hat{\tau} = \frac{\tau}{\lambda\gamma}$ and $\hat{\theta} = 1 - (1 - \xi)(1 - \theta)$. Assume that $\pi^* \neq 0$ and is increasing ,as $\phi(\pi^*)$ is monotone decreasing function on π^*.

Dividing the equations(4.20) and (4.21) by π^* we deduce

$$\frac{\mu + \delta_1}{L(1 - \pi^*)\lambda\alpha\phi(\pi^*)(1 - \xi)} = \frac{1}{\hat{\theta} + \pi^*\left(1 - \hat{\theta}\right) + \left(\frac{\hat{\tau}}{\phi(\pi^*)}\right)}. \tag{4.22}$$

The trivial equilibrium is $E_0 = (\pi_0, \beta_0) = (0, 0)$, and any other non- zero equilibrium solution $E_1 = (\pi^*, \beta^*)$ must satisfy the equation (4.22).

Next,we define the left-hand side of the equation (4.22) to be $f_1(\pi)$ and the right-hand to be $f_2(\pi)$.

Recall that $\phi(\pi)$ is strictly monotone decreasing function in π, moreover at $\pi^* = 0$ and $\phi(0) = 1$,so we have

$$f_1(0) = \frac{\mu + \delta_1}{L\lambda\alpha(1-\xi)}, \qquad \textit{and} \tag{4.23}$$

$$f_2(0) = \frac{1}{\hat{\theta} + \hat{\tau}}. \tag{4.24}$$

The expression of the basic reproductive number is given by the equation (4.9) can be rearrange by substituting the value of L, as follows

$$R_0 = \frac{L\lambda\alpha(1-\xi)}{(\mu + \delta_1)(\hat{\tau} + \hat{\theta})}. \tag{4.25}$$

There are three cases to consider.

(a) Suppose that $R_0 < 1$

$$\frac{L\lambda\alpha(1-\xi)}{(\mu + \delta_1)(\hat{\tau} + \hat{\theta})} < 1.$$

In this case we have that

$$L\lambda\alpha(1-\xi) < (\mu + \delta_1)(\hat{\tau} + \hat{\theta}).$$

We get

$$f_1(0) = \frac{\mu + \delta_1}{L\lambda\alpha(1-\xi)} > \frac{1}{\hat{\theta} + \hat{\tau}} = f_2(0). \tag{4.26}$$

Moreover, we know that $f_1(\pi)$ is a strictly monotone increasing function of π and and $f_2(\pi)$ a strictly decreasing function of π. Thus $f_1(\pi) > f_2(\pi)$ $\forall \pi \in (0, 1]$ using the inequality (4.26).

Therefore, there is no non-zero solution in this case for $R_0 < 1$.

(b) If $R_0 = 1$, then we have the same thing that

$$L\lambda\alpha(1-\xi) = (\mu + \delta_1) + (\hat{\tau} + \hat{\theta}).$$

We get

$$\frac{1}{L\lambda\alpha\,(1-\xi)} = \frac{1}{(\hat{\theta} + \hat{\tau})(\mu + \delta_1)}. \tag{4.27}$$

Hence by the equation (4.27), we have $f_1(\pi) > f_2(\pi)\ \forall \pi > 0$. Hence if $R_0 = 1$, the equation (4.22) has non-zero solution.

(c) If $R_0 > 1$, then we have

$$L\lambda\alpha(1-\xi) > (\mu + \delta_1)\,(\hat{\tau} + \hat{\theta}).$$

So by the equation (4.26), we have

$$\frac{1}{L\lambda\alpha\,(1-\xi)} < \frac{1}{(\hat{\theta} + \hat{\tau})(\mu + \delta_1)}.$$

Therefore, using the monotonicity of $f_1(\pi)$ and $f_2(\pi)$ we deduce that equation (4.22) has a unique non-zero solution in $(0, 1]$, and we define the non-zero solution to be the endemic equilibrium of the system. This concludes the proof of the Theorem.

Now, we move on to study the local Stability Analysis of Equilibrium points of our model.

4.3.3 The Local Stability Analysis of Equilibrium

It is important to identify the behaviour of our model's local stability equilibrium from a mathematical and biological aspect.

We used the same techniques as Greenhalgh and Hay (1997) and Agaba et al. (2017) to determine the local asymptotic stability of the equilibrium value, which was by using the Routh-Hurwitz criterion (May (2001),DeJesus and Kaufman (1987), which was sufficient to look to the eigenvalues of the variational matrix of the system about the disease-free (DFE) and endemic (EE) equilibrium points.

Theorem 4.3.3. *Consider that $\phi(\pi)$ is a differentiable function. The system of equations* (4.5)-(4.8) *has a locally stable solution for the disease-free equilibrium DFE as well as for endemic equilibrium EE in the three following cases.*

i. The disease-free solution to the system is locally asymptotically stable if $R_0 < 1$.

ii. The disease-free solution is neutrally stable if $R_0 = 1$.

iii. The disease-free solution is unstable if $R_0 > 1$, but the unique EE is locally asymptotically stable.

Proof. To determine the local stability by linearising the system of equations (4.5)-(4.8) around the equilibrium point. This system can be represented in matrix form as
$\frac{d\boldsymbol{y}}{dt} = \boldsymbol{Jy}$, where $\boldsymbol{y}^T = (\pi_1, \pi_2, \pi_3, \beta)$.

$$\boldsymbol{J} = \begin{bmatrix} \frac{\partial f_1}{\partial \pi_1} & \frac{\partial f_1}{\partial \pi_2} & \frac{\partial f_1}{\partial \pi_3} & \frac{\partial f_1}{\partial \beta} \\ \frac{\partial f_2}{\partial \pi_1} & \frac{\partial f_2}{\partial \pi_2} & \frac{\partial f_2}{\partial \pi_3} & \frac{\partial f_2}{\partial \beta} \\ \frac{\partial f_3}{\partial \pi_1} & \frac{\partial f_3}{\partial \pi_2} & \frac{\partial f3}{\partial \pi_3} & \frac{\partial f_3}{\partial \beta} \\ \frac{\partial f_4}{\partial \pi_1} & \frac{\partial f_4}{\partial \pi_2} & \frac{\partial f_4}{\partial \pi_3} & \frac{\partial f_4}{\partial \beta} \end{bmatrix}.$$

Here

$\hat{\theta} = (1 - (1 - \theta)(1 - \xi))$, and $\hat{\tau} = \frac{\tau}{\lambda\gamma}$.

So, the Jacobian matrix for our system is

$$\boldsymbol{J} = \begin{bmatrix} A-(\mu+\delta_1) & A & A & B \\ \delta_1 & -(\mu+\delta_2) & 0 & 0 \\ 0 & \delta_2 & -(\mu+\delta_3) & 0 \\ C & C & C & -D \end{bmatrix}.$$

Here

$$A = \phi'(\pi^*)(1-\pi^*)\lambda\alpha\beta^*(1-\xi) - \lambda\alpha\beta^*\phi(\pi^*)(1-\xi),$$

$$B = (1-\pi^*)\lambda\alpha\phi(\pi^*)(1-\xi),$$

$$C = [(1-\beta^*(1-\hat{\theta}))\phi(\pi^*) + (1-\beta^*)\phi'(\pi^*)\pi^* - \beta^*\hat{\theta}(1-\pi^*)\phi'(\pi^*)]\lambda\gamma,$$

and $D = -((\hat{\theta} + (1-\hat{\theta})\pi^*)\phi(\pi^*) + \hat{\tau})\lambda\gamma.$

(4.28)

4.3.3.1 The Disease-Free Equilibrium.

The Jacobian matrix for our system evaluated at DFE $(\pi_1^*, \pi_2^*, \pi_3^*, \beta^*) = (0,0,0,0)$ is

$$\boldsymbol{J} = \begin{bmatrix} -(\mu+\delta_1) & 0 & 0 & \lambda\alpha(1-\xi) \\ \delta_1 & -(\mu+\delta_2) & 0 & 0 \\ 0 & \delta_2 & -(\mu+\delta_3) & 0 \\ \lambda\gamma & \lambda\gamma & \lambda\gamma & -\lambda\gamma(\hat{\theta}+\hat{\tau}) \end{bmatrix}.$$

In the disease-free equilibrium (DFE), the characteristic equation of the Jacobian of our model, by calculating its determinant

$$det(\boldsymbol{J} - \omega\boldsymbol{I}) = 0.$$

$$\begin{vmatrix} -(\mu+\delta_1+\omega) & 0 & 0 & \lambda\alpha(1-\xi) \\ \delta_1 & -(\mu+\delta_2+\omega) & 0 & 0 \\ 0 & \delta_2 & -(\mu+\delta_3+\omega) & 0 \\ \lambda\gamma & \lambda\gamma & \lambda\gamma & -(\lambda\gamma(\hat{\theta}+\hat{\tau})+\omega) \end{vmatrix} = 0.$$

Expanding along the top row this equation is

$$(\mu+\delta_1+\omega)(\mu+\delta_2+\omega)(\mu+\delta_3+\omega)(\lambda\gamma\hat{\theta}+\hat{\tau}+\omega)$$
$$-\lambda\alpha(1-\xi))\begin{vmatrix} \delta_1 & -(\mu+\delta_2+\omega) & 0 \\ 0 & \delta_2 & -(\mu+\delta_3+\omega) \\ \lambda\gamma & \lambda\gamma & \lambda\gamma \end{vmatrix} = 0$$

We would like to demonstrate that at least one of the eigenvalues of ***J*** has a strictly positive real part. So the eigenvalues of this 4×4 matrix will now be investigated. The equation can be rewritten.

$$(\mu+\delta_1+\omega)(\mu+\delta_2+\omega)(\mu+\delta_3+\omega)(\lambda\gamma(\hat{\theta}+\hat{\tau}))+\omega-\lambda^2\alpha\gamma(1-\xi)[\delta_1\delta_2+$$
$$(\mu+\delta_2+\omega)(\mu+\delta_3+\omega)+\delta_1(\mu+\delta_3+\omega)]=0.$$

To make the notation simple we write

$$x_1=\mu+\delta_1, x_2=\mu+\delta_2, x_3=\mu+\delta_3 \textit{and}\ x_4=\lambda\gamma(\hat{\theta}+\hat{\tau}).$$

As a result, the characteristic equation of the Jacobiani is written as $\omega^4+a_1\omega^3+a_2\omega^2+a_3\omega+a_4=0$. So by applying the Routh-Hurwitz criteria it is sufficient for local stability to show that $a_i>0$ for $i=1,2,3,4$ and $a_1a_2>a_3$ and $(a_1a_2-a_3)a_3>a_1^2a_4$.

Then, we have

$$a_1 = x_1 + x_2 + x_3 + x_4 > 0,$$

$$a_2 = x_1x_2 + x_1x_3 + x_1x_4 + x_2x_3 + x_2x_4 + x_3x_4 - \lambda^2\alpha\gamma(1-\xi),$$

$$a_3 = x_1x_2x_3 + x_1x_2x_4 + x_1x_3x_4 + x_2x_3x_4 - \lambda^2\alpha\gamma(1-\xi)[\delta_1 + (\mu+\delta_2) + (\mu+\delta_3)],$$

and

$$a_4 = x_1x_2x_3x_4 - \lambda^2\alpha\gamma(1-\xi)[\delta_1\delta_2 + (\mu+\delta_2)(\mu+\delta_3) + \delta_1(\mu+\delta_3)],$$

$$= x_1x_2x_3x_4(1-R_0) \quad > 0.$$

Note that $R_0 < 1$ if and only if $a_4 > 0$, $R_0 = 1$ if and only if $a_4 = 0$ and $R_0 > 1$ if and only if $a_4 < 0$. Hence for $R_0 > 1$, the disease-free equilibrium is unstable.

Hence the statements follow. Now

$$1 > R_0 = \frac{\lambda\alpha(1-\xi)}{(\mu+\delta_1)(\hat{\tau}+\hat{\theta})}\left[1 + \frac{\delta_1}{\mu+\delta_2} + \frac{\delta_1\delta_2}{(\mu+\delta_2)(\mu+\delta_3)}\right].$$

Next, we will show the Routh-Hurwitz conditions, Thus

$$\begin{aligned} x_1x_4 &= \lambda\gamma(\mu+\delta_1)(\hat{\theta}+\hat{\tau}) \\ &> \lambda^2\alpha(1-\xi)\gamma\left[1 + \frac{\delta_1}{\mu+\delta_2} + \frac{\delta_1\delta_2}{(\mu+\delta_2)(\mu+\delta_3)}\right]. \end{aligned}$$

So

$$a_2 = x_1x_2 + x_1x_3 + x_2x_3 + x_2x_4 + x_3x_4 + (x_1x_4 - \lambda^2\alpha(1-\xi)\gamma) > 0.$$

Now note that

$$\begin{aligned} x_1x_2x_4 &= \lambda\gamma(\mu+\delta_1)(\mu+\delta_2)(\hat{\theta}+\hat{\tau}) \\ &> \lambda^2\alpha\gamma(1-\xi)(\mu+\delta_2)\left[1 + \frac{\delta_1}{\mu+\delta_2} + \frac{\delta_1\delta_2}{(\mu+\delta_2)(\mu+\delta_3)}\right], \\ &> \lambda^2\alpha\gamma(1-\xi)(\mu+\delta_1+\delta_2). \end{aligned}$$

moreover

$$\begin{aligned} x_1x_3x_4 &= \lambda\gamma(\mu+\delta_1)(\mu+\delta_3)(\hat{\theta}+\hat{\tau}) \\ &> \lambda^2\alpha\gamma(1-\xi)(\mu+\delta_3)\Big[1+\frac{\delta_1}{\mu+\delta_2}+\frac{\delta_1\delta_2}{(\mu+\delta_2)(\mu+\delta_3)}\Big], \\ &> \lambda^2\alpha\gamma(1-\xi)(\mu+\delta_3). \end{aligned}$$

So

$$\begin{aligned} a_3 &= x_1x_2x_3 + x_2x_3x_4 + \Big(x_1x_3x_4 + x_1x_2x_4 - \lambda^2\alpha\gamma(1-\xi)\big[\delta_1+(\mu+\delta_2)+(\mu+\delta_3)\big]\Big), \\ &> x_1x_2x_3 + x_2x_3x_4, \\ &> 0. \end{aligned}$$

So, $a_3 > x_1x_2x_3 + x_2x_3x_4$. Hence

$$\begin{aligned} a_1a_2 &= (x_1+x_2+x_3+x_4)\times\Big[x_1x_2+x_1x_3+x_2x_3+x_2x_4+x_3x_4+\Big(x_1x_4-\lambda^2\alpha\gamma(1-\xi)\Big)\Big]. \\ &> x_1(x_2x_3)+x_1(x_2x_4)+x_1(x_3x_4)+x_3(x_2x_4) \\ &\quad + x_1\Big(x_1x_2+x_1x_3+(x_1x_4-\lambda^2\alpha\gamma(1-\xi))\Big) \qquad +x \\ &\quad + x_3(x_1x_2+x_1x_3+x_2x_4+x_3x_4) \\ &\quad + x_4\Big(x_1x_2+x_1x_3+x_2x_3+x_2x_4+x_3x_4)+(x_1x_4-\lambda^2\alpha\gamma(1-\xi))\Big), \\ &> x_1(x_2x_3)+x_1(x_2x_4)+x_1(x_3x_4)+x_3(x_2x_4) \\ &= a_3+\lambda^2\alpha\gamma(1-\xi)\big[\delta_1+(\mu+\delta_2)+(\mu+\delta_3)\big]. \end{aligned}$$

$a_1a_2 > a_3$.

So $a_1a_2 > a_3$. In fact, the proof shows that

$$\begin{aligned}
a_1a_2 &> a_3 + x_1(x_1x_2 + x_1x_3 + x_1x_4 - \lambda^2\alpha\gamma(1-\xi)) \\
&\quad + x_2(x_1x_2 + x_1x_3 + x_2x_3 + x_2x_4 + x_3x_4) \\
&\quad + x_3(x_1x_2 + x_1x_3 + x_2x_4 + x_3x_4) \\
&\quad + x_4(x_1x_2 + x_1x_3 + x_2x_3 + x_2x_4 + x_3x_4) + (x_1x_4 - \lambda^2\alpha\gamma(1-\xi))) \\
&\quad + \lambda^2\alpha\gamma(1-\xi)[\delta_1 + (\mu + \delta_2) + (\mu + \delta_3)] \\
&> a_3 + x_1(x_1x_2 + x_1x_3 + x_1x_4) + x_2(x_1x_2 + x_1x_3 + x_2x_3 + x_2x_4 + x_3x_4) \\
&\quad + x_3(x_1x_2 + x_1x_3 + x_2x_4 + x_3x_4) \\
&\quad + x_4(x_1x_2 + x_1x_3 + x_2x_3 + x_2x_4 + x_3x_4) + (x_1x_4 - \lambda^2\alpha\gamma(1-\xi)))
\end{aligned}$$

Hence

$$\begin{aligned}
(a_1a_2 - a_3)a_3 &> \Big[x_1(x_1x_2 + x_1x_3 + x_1x_4) + x_2(x_1x_2 + x_1x_3 + x_2x_3 + x_2x_4 + x_3x_4) \\
&\quad + x_3(x_1x_2 + x_1x_3 + x_2x_4 + x_3x_4) + x_4(x_1x_2 + x_1x_3 + x_2x_3 + x_2x_4 \\
&\quad + x_3x_4 + (x_1x_4 - \lambda^2\alpha\gamma(1-\xi)))\Big](x_1x_2x_3 + x_2x_3x_4) \\
&= \Big[x_1^2x_2 + x_1^2x_3 + x_1^2x_4 + x_1x_2^2 + x_1x_2x_3 + x_2^2x_3 + x_2^2x_4 + x_2x_3x_4 + x_1x_2x_3 \\
&\quad + x_1x_3^2 + x_2x_3x_4 + x_3^2x_4 + x_1x_2x_4 + x_1x_3x_4 + x_2x_3x_4 + x_2x_4^2 + x_3x_4^2 \\
&\quad + (x_1x_4 - \lambda^2\alpha\gamma(1-\xi))x_4\Big](x_1x_2x_3 + x_2x_3x_4) \\
&> x_1^2x_2(x_2x_3x_4) + x_1^2x_3(x_2x_3x_4) + x_1x_2^2(x_2x_3x_4) + x_1^2x_4(x_1x_2x_3) \\
&\quad + x_1^2x_4(x_2x_3x_4) + x_1x_2x_3(x_2x_3x_4) + x_2x_3x_4(x_1x_2x_3) + x_1x_3^2(x_2x_3x_4) \\
&\quad + x_1x_2x_4(x_1x_2x_3 + x_2x_3x_4) + x_1x_3x_4(x_1x_2x_3 + x_2x_3x_4) \\
&\quad + x_2x_4^2(x_1x_2x_3) + x_3x_4^2(x_1x_2x_3)
\end{aligned}$$

$$
\begin{aligned}
&+ x_1x_4^2(x_1x_2x_3 + x_2x_3x_4) + x_1x_4^2(x_1x_2x_3 + x_2x_3x_4) \\
&- \lambda^2\alpha\gamma(1-\xi)x_4(x_1x_2x_3 + x_2x_3x_4) \\
&> x_1^2x_2^2x_3x_4 + x_1^2x_2x_3^2x_4 + x_1x_2^3x_3x_4 + x_1^3x_2x_3x_4 + x_1^2x_2x_3^2x_4^2 + x_1x_2x_3^3x_4 \\
&+ x_1x_2^2x_3^2x_4 + x_1x_2x_3^2x_4 + x_1^2x_2^2x_3x_4 + x_1x_2^2x_3x_4^2 + x_1^2x_2x_3^2x_4 + x_1x_2x_3^2x_4^2 \\
&+ x_1x_2^2x_3x_4^2 + x_1x_2x_3^2x_4^2 + x_1^3x_2x_3x_4 + x_1^2x_2x_3x_4^2 \\
&- \lambda^2\alpha\gamma(1-\xi)(x_1x_2x_3x_4 + x_2x_3x_4^2) \\
&= (x_1^2 + x_2^2 + x_3^2 + x_4^2 + 2x_1x_2 + 2x_1x_3 + 2x_1x_4 + 2x_2x_3 + 2x_2x_4 + 2x_3x_4)x_1x_2x_3x_4 \\
&- \lambda^2\alpha\gamma(1-\xi)(x_1x_2x_3x_4 + x_2x_3x_4^2) \\
&= (x_1 + x_2 + x_3 + x_4)^2x_1x_2x_3x_4 - \lambda^2\alpha\gamma(1-\xi)(x_1x_2x_3x_4 + x_2x_3x_4^2) \\
&= (x_1 + x_2 + x_3 + x_4)^2(a_4 + \lambda^2\alpha\gamma(1-\xi)[\delta_1\delta_2 + (\mu+\delta_2)(\mu+\delta_3) + \delta_1(\mu+\delta_3)] \\
&- \lambda^2\alpha\gamma(1-\xi)(x_1x_4 + x_4^2)x_2x_3 \\
&> a_1^2a_4
\end{aligned}
$$

as required.

Now for $R_0 = 1$, we know that 0 is an eigenvalue and for $R_0 < 1$ all eigenvalues have strictly negative real parts. Choose a sequence of parameters so that $R_0 \to 1^-$ then as the eigenvalues are continuous functions of the parameters (Harris and Martin (1987)) we see that for $R_0 = 1$, no eigenvalue can have a strictly positive real part so therefore the disease-free equilibrium is neutrally stable.

4.3.3.2 The Endemic equilibrium.

Similarly, we explore local stability at the endemic equilibrium point using the same argument we used to investigate free disease equilibrium.

The Jacobian matrix for our system evaluated at EE $(\pi_i^*, \beta^*) = (\pi_i^*, \beta^*)$

$$\boldsymbol{J} = \begin{bmatrix} A - x_1 & A & A & B) \\ \delta_1 & -x_2 & 0 & 0 \\ 0 & \delta_2 & -x_3 & 0 \\ C & C & C & D \end{bmatrix}.$$

Then the characteristic equation is $\omega^4 + a_1\omega^3 + a_2\omega^2 + a_3\omega + a_4 = 0$, we must prove that $a_1 > 0$, $a_2 > 0$, $a_3 > 0$ $a_4 > 0$ and $(a_1a_2 - a_3)a_3 > a_1^2a_4$ using the Routh-Hurwitz requirements for a quartic polynomial.

In the same way, we use the same technical method to collect the constant term of $\det(J - \omega I) = 0$.

$$\begin{vmatrix} A - (\mu + \delta_1 + \omega) & A & A & B \\ \delta_1 & -(\mu + \delta_2 + \omega) & 0 & 0 \\ 0 & \delta_2 & -(\mu + \delta_3 + \omega) & 0 \\ C & C & C & D - \omega \end{vmatrix} = 0.$$

To easily compute the terms in ω, ω^2,ω^3 and ω^4 by using the equation definition of (4.28) with $x_1 = \mu + \delta_1, x_2 = \mu + \delta_2, x_3 = \mu + \delta_3, x_4 = \lambda\gamma(\hat{\theta} + \hat{\tau})$.

The term in ω^3 gives

$$\begin{aligned} a_1 &= -A - D + x_1 + x_2 + x_3. \\ &= (\mu + \delta_1) + \lambda\alpha\beta^*\phi(\pi^*)(1 - \xi) - \phi'(\pi^*)(1 - \pi^*)\lambda\alpha\beta^*(1 - \xi) \\ &+ \lambda\gamma[\hat{\tau} + (\hat{\theta} + (1 - \hat{\theta})\pi^*)\phi(\pi^*)] + (\mu + \delta_2) + (\mu + \delta_3). \\ &= \mu + \delta_1 + \mu + \delta_2 + \mu + \delta_3 + \lambda\gamma\frac{\pi^*\phi(\pi^*)}{\beta^*} + (\lambda\alpha\beta^*\phi(\pi^*)(1 - \xi)) \\ &- \phi'(\pi^*)(1 - \pi^*)\lambda\alpha\beta^*(1 - \xi) > 0. \end{aligned} \tag{4.29}$$

Here we have used the equation (4.21), to replace $\beta^*(\hat{\tau} + (\hat{\theta} + (1 -$

$\hat{\theta})\pi^*)\phi(\pi^*))$ with $\pi^*\phi(\pi^*)$, and also used the fact that ϕ is monotone decreasing in π. For ω^2, we have

$$
\begin{aligned}
a_2 &= -BC + AD - Dx_1 - Dx_2 - Dx_3 - \delta_1 A - x_2 A - x_3 A + x_1 x_2 + x_1 x_3 + x_2 x_3, \\
&= [\lambda\alpha\beta^*\phi(\pi^*)(1-\xi) - \phi^{'}(\pi^*)(1-\pi^*)\lambda\alpha\beta^*](1-\xi)[\delta_1 + \mu + \delta_2 + (\mu + \delta_3] \\
&+ \lambda\gamma[\hat{\tau} + (\hat{\theta} + (1-\hat{\theta})\pi^*)\phi(\pi^*)][\mu + \delta_1 + \mu + \delta_2 + \mu + \delta_3] \\
&+ (\mu + \delta_1)(\mu + \delta_2) + (\mu + \delta_2)(\mu + \delta_3) + (\mu + \delta_3)(\mu + \delta_1) \\
&+ [\lambda\alpha\beta^*\phi(\pi^*)(1-\xi) - \phi'(\pi^*)(1-\pi^*)\lambda\alpha\beta^*(1-\xi)][\hat{\tau} + (\hat{\theta} + (1-\hat{\theta})\pi^*)\phi(\pi^*)]\lambda\gamma \\
&- (1-\pi^*)\lambda\alpha\phi(\pi^*)(1-\xi)[(1-\beta^*(1-\hat{\theta}))\phi(\pi^*) + (1-\beta^*)\phi'(\pi^*)\pi^* \\
&- \beta^*\hat{\theta}(1-\pi^*)\phi'(\pi^*)]\lambda\gamma.
\end{aligned}
\tag{4.30}
$$

Again from equation (4.21) we can replace $(\hat{\tau} + (\hat{\theta} + (1-\hat{\theta})\pi^*)\phi(\pi^*))$ with $\pi^*\phi(\pi^*)/\beta^*$ in the same way that we replace $\phi(\pi^*)(1-\pi^*)(1-\beta^*(1-\hat{\theta}))$ with $\phi(\pi^*)(1-\beta) - \beta\hat{\tau}$.

By using the equation (4.21) and equation (4.20)together, we replace $(\mu + \delta_1)(\hat{\tau} + \hat{\theta} + (1-\hat{\theta})\pi^*)$ with $L\,(1-\pi^*)\,\phi(\pi^*)^2\lambda\alpha(1-\xi)$.

We find that by substituting and simplifying these different terms, we get that

$$
a_2 = [\lambda\alpha\beta^*\phi(\pi^*)(1-\xi) - \phi^{'}(\pi^*)(1-\pi^*)\lambda\alpha\beta^*(1-\xi)][\delta_1 + \mu + \delta_2 + \mu + \delta_3]
\tag{4.31}
$$

$$
+ \lambda\gamma\frac{\pi^*\phi(\pi^*)}{\beta^*}[\mu + \delta_2 + \mu + \delta_3]
\tag{4.32}
$$

$$
\begin{aligned}
&+ \lambda\gamma\lambda\alpha(1-\xi)[\beta^*(\phi(\pi^*) + \hat{\tau})\phi(\pi^*) - (1-\pi^*)\phi(\pi^*)(1-\beta^*)\phi'(\pi^*)\pi^* \\
&+ (1-\pi^*)^2\phi(\pi^*)\beta^*\hat{\theta}\phi'(\pi^*) - \pi^*(1-\pi^*)\phi'(\pi^*)\phi(\pi^*)]
\end{aligned}
\tag{4.33}
$$

$$
+ (\mu + \delta_1)(\mu + \delta_2) + (\mu + \delta_2)(\mu + \delta_3) + (\mu + \delta_3)(\mu + \delta_1)
\tag{4.34}
$$

$$
+ \left[\frac{\delta_1}{\mu + \delta_2} + \frac{\delta_1\delta_2}{(\mu + \delta_2)(\mu + \delta_3)}\right](1-\pi^*)\phi^2(\pi^*)\lambda\alpha(1-\xi)\lambda\gamma.
\tag{4.35}
$$

Note that, from equation (4.21) we have that

$$\begin{aligned}(1-\beta^*)\pi^* - \beta^*\hat{\theta}(1-\pi^*) &= \frac{\pi^*[\phi(\pi^*)(1-\pi^*)\hat{\theta}+\hat{\tau}]}{\pi^*\phi(\pi^*+\phi(\pi^*)(1-\pi^*)\hat{\theta}+\hat{\tau}} \\ &- \frac{\pi^*\phi(\pi^*)\hat{\theta}(1-\pi^*)}{\pi^*\phi(\pi^*+\phi(\pi^*)(1-\pi^*)\hat{\theta}+\hat{\tau}} \\ &= \frac{\tau\pi^*}{\pi^*\phi(\pi^*+\phi(\pi^*)(1-\pi^*)\hat{\theta}+\hat{\tau}} \\ &> 0.\end{aligned} \tag{4.36}$$

So clearly $a_2 > 0$.

Similarly, we collect the terms in ω

$$\begin{aligned}a_3 &= -BC(\delta_1+x_2+x_3) - (\delta_1\delta_2+\delta_1x_3+x_2x_3)A \\ &+ x_1x_2x_3 - x_2x_3D + \delta_1AD - D[x_1-A][x_2+x_3]. \\ &= -[([(1-\beta^*(1-\hat{\theta})\phi(\pi^*)+(1-\beta^*)\phi'(\pi^*)\pi^* - \beta^*\hat{\theta}(1-\pi^*)\phi'(\pi^*)]\lambda\gamma) \\ &\lambda\alpha(1-\pi^*)\phi(\pi^*)(1-\xi)][\delta_1+\mu+\delta_2+\mu+\delta_3] \\ &- [\delta_1\delta_2+\delta_1(\mu+\delta_3)+(\mu+\delta_2)(\mu+\delta_3)][\lambda\alpha\beta^*(1-\xi)(\phi'(\pi)(1-\pi^*)-\phi(\pi^*))] \\ &+ (\mu+\delta_1)(\mu+\delta_2)(\mu+\delta_3)+(\mu+\delta_2)(\mu+\delta_3)[((\hat{\theta}+(1-\hat{\theta})\pi^*)\phi(\pi^*)+\hat{\tau})\lambda\gamma)] \\ &+ \delta_1\lambda\alpha\beta^*\phi(\pi^*)(1-\xi)[((\hat{\theta}+(1-\hat{\theta})\pi^*)\phi(\pi^*)+\hat{\tau})\lambda\gamma] \\ &- \delta_1\phi'(\pi^*)(1-\pi^*)\lambda\alpha\beta^*(1-\xi)[((\hat{\theta}+(1-\hat{\theta})\pi^*)\phi(\pi^*)+\hat{\tau})\lambda\gamma] + [\mu+\delta_1 \\ &+ \lambda\alpha\beta^*(1-\xi)(\phi(\pi^*)-\phi'(\pi^*)(1-\pi^*))][\lambda\gamma((\hat{\theta}+(1-\hat{\theta})\pi^*)+\hat{\tau}][(\mu+\delta_2+\mu+\delta_3)].\end{aligned} \tag{4.37}$$

In a similar fashion to the term a_2 above, we have a_3 as follows

$$\begin{aligned}
a_3 &= \Big[\lambda\alpha\beta^*\phi(\pi^*)(1-\xi) - \phi'(\pi^*)(1-\pi^*)\lambda\alpha\beta^*(1-\xi)\Big]L(\mu+\delta_2)(\mu+\delta_3) \\
&+ (\mu+\delta_1)(\mu+\delta_2)(\mu+\delta_3) + (\mu+\delta_2)(\mu+\delta_3)\lambda\gamma\frac{\pi^*\phi(\pi^*)}{\beta^*} \\
&+ \lambda\gamma\lambda\alpha(1-\xi)[\beta^*(\phi(\pi^*)+\hat{\tau})\phi(\pi^*) - (1-\pi^*)\phi(\pi^*)(1-\beta^*)\phi'(\pi^*)\pi^* \\
&+ (1-\pi^*)^2\phi(\pi^*)\beta^*\hat{\theta}\phi'(\pi^*) - \pi^*(1-\pi^*)\phi'(\pi^*)\phi(\pi^*)][\delta_1+\mu+\delta_2+\mu+\delta_3] \\
&- \lambda\gamma\lambda\alpha\phi'(\pi^*)(1-\pi^*)\phi(\pi^*)\pi^*(1-\xi)[\delta_1+\mu+\delta_2+\mu+\delta_3] \\
&+ \lambda\gamma\lambda\alpha(1-\pi^*)\phi^2(\pi^*)(1-\xi)\left[\frac{\delta_1\delta_2}{\mu+\delta_3} + \frac{\delta_1\delta_2}{(\mu+\delta_2)} + \frac{\delta_1(\mu+\delta_3)}{(\mu+\delta_2)}\right]
\end{aligned}$$

$a_3 > 0.$

(4.38)

Now collecting the constant term a_4

$$\begin{aligned}
a_4 &= -BC\delta_1\delta_2 - x_3BC\delta_1 - x_2x_3BC + A\delta_1\delta_2D + x_3\delta_1AD - x_1x_2x_3D + x_2x_3AD. \\
&= -\Big[(1-\beta^*(1-\hat{\theta}))\phi(\pi^*) + (1-\beta^*)\phi'(\pi^*)\pi^* - \beta^*\hat{\theta}(1-\pi^*)\phi'(\pi^*)\Big]\lambda\gamma \\
&(1-\pi^*)\lambda\alpha\phi(\pi^*)(1-\xi)\left[(\mu+\delta_2)(\mu+\delta_3)\right]\left[1 + \frac{\delta_1}{(\mu+\delta_2)} + \frac{\delta_1\delta_2}{(\mu+\delta_2)(\mu+\delta_3)}\right] \\
&+ [\lambda\alpha\beta^*\phi(\pi^*)(1-\xi) - \phi'(\pi^*)(1-\pi^*)\lambda\alpha\beta^*(1-\xi)] \\
&\lambda\gamma[(\hat{\theta} + (1-\hat{\theta})\pi^*)\phi(\pi^*) + \hat{\tau}][(\mu+\delta_2)(\mu+\delta_3)]L \\
&+ [(\mu+\delta_1)(\mu+\delta_2)(\mu+\delta_3)][(\hat{\theta} + (1-\hat{\theta})\pi^*)\phi(\pi^*) + \hat{\tau}]\lambda\gamma.
\end{aligned}$$

(4.39)

Here

$$L = \left[1 + \frac{\delta_1}{(\mu+\delta_2)} + \frac{\delta_1\delta_2}{(\mu+\delta_2)(\mu+\delta_3)}\right].$$

By using the same replacement as the previous one used for the terms a_2, a_3 we get that

$$\begin{aligned}
a_4 &= \lambda\gamma\lambda\alpha(1-\xi)[\beta^*(\phi(\pi^*)+\hat{\tau})\phi(\pi^*) - (1-\pi^*)\phi(\pi^*)(1-\beta^*)\phi'(\pi^*)\pi^* \\
&+ (1-\pi^*)^2\phi(\pi^*)\beta^*\hat{\theta}\phi'(\pi^*) - \pi^*(1-\pi^*)\phi'(\pi^*)\phi(\pi^*)]\left[(\mu+\delta_2)(\mu+\delta_3)\right]L.
\end{aligned}$$

(4.40)

By using the fact expression on the equation(4.36), we deduce that

$a_4 > 0$ if $R_0 > 1$. We know that $\phi'(\pi^*)$ is a monotone decreasing function so $\phi'(\pi^*) > 0$, therefore, it is clear that for all terms of a_i to be strictly positive it is sufficient that the equilibrium points (π_i^*, β^*) is strictly positive where $R_0 > 1$.

Now we require to show that $a_1a_2 - a_3 > 0$. We see that a_1 can be written as a sum of three positive terms $a_1^1 = \mu + \delta_1 + \mu + \delta_2 + \mu + \delta_3$, $a_1^2 = \frac{\lambda\gamma\pi^*\phi(\pi^*)}{\beta^*}$ and $a_1^3 = \lambda\alpha\beta^*\phi(\pi^*)(1-\xi) - \phi'(\pi^*)(1-\pi^*)\lambda\alpha\beta^*(1-\xi)$, and these terms are involved in the expansion of a_3 given in equation 4.48, we write the positive components summing to a_2 as a_2^1 (4.31), a_2^2 (4.32) ,a_2^3 (4.33),a_2^4 (4.34) and a_2^5 (4.35). So

$$a_1a_2 - a_3 = [a_1^1 + a_1^2 + a_1^3][a_2^1 + a_2^2 + a_2^3 + a_2^4 + a_2^5]$$

Then considering each one of a_1^1, a_1^2 and a_1^3 separately and cancelling similar terms. We deduce that

$$\begin{aligned}
a_1a_2 - a_3 =& [\lambda\alpha\beta^*\phi(\pi^*)(1-\xi) - \phi'(\pi^*)(1-\pi^*)\lambda\alpha\beta^*(1-\xi)] \\
& [a_2^1 + a_2^2 + a_2^3 + a_2^5 + \mu(\mu+\delta_3) + \mu(\mu+\delta_1+\delta_2)] \\
& + (\mu+\delta_1+\mu+\delta_2+\mu+\delta_3)\Big[a_2^1 + a_2^2 \\
& + (\mu+\delta_1)(\mu+\delta_2) + (\mu+\delta_1)(\mu+\delta_3)] \\
& + \mu a_2^3 + (\mu+\delta_2)(\mu+\delta_3)(\mu+\delta_2+\mu+\delta_3) \\
& + [(\mu+\delta_1)\left[\frac{\delta_1}{\mu+\delta_2} + \frac{\delta_1\delta_2}{(\mu+\delta_2)(\mu+\delta_3)}\right] + \delta_1] \\
& (1-\pi^*)\phi(\pi^*)^2\lambda^2\alpha\gamma(1-\xi)) \\
& \frac{\lambda\gamma\pi^*\phi(\pi^*)}{\beta^*}[a_2^1 + a_2^2 + a_2^3 + a_2^5 + (\mu+\delta_1)(\mu+\delta_2) + (\mu+\delta_1)(\mu+\delta_3)]
\end{aligned} \tag{4.41}$$

Thus $a_1a_2 - a_3 > 0$.

Next, we going to show that $(a_1a_2 - a_3)a_3 > a_1^2a_4$. Since

$$\begin{aligned}
\psi = & [\beta^*(\phi(\pi^*) + \hat{\tau})\phi(\pi^*) - (1-\pi^*)\phi(\pi^*)(1-\beta^*)\phi'(\pi^*)\pi^* \\
& + (1-\pi^*)^2\phi(\pi^*)\beta^*\hat{\theta}\phi'(\pi^*) - \pi^*(1-\pi^*)\phi'(\pi^*)\phi(\pi^*)]
\end{aligned}$$

is a factor of $a_1^2 a_4$ we shall base our argument around showing that $(a_1 a_2 - a_3)a_3$ has sufficient terms containing ψ and extra terms such that

$$(a_1 a_2 - a_3)a_3 > a_1^2 a_4.$$

It is sufficient to show that

$$\begin{aligned}
&\Bigg\{\Bigg[\delta_1(1-\pi^*)\phi(\pi^*)^2\lambda\alpha(1-\xi)\lambda\gamma + \frac{\lambda\gamma\pi^*\phi(\pi^*)}{\beta^*}(Eq.(4.31)) + (\mu+\delta_1)\\
&\qquad (\mu+\delta_2+\mu+\delta_3)\lambda\gamma\frac{\pi^*\phi(\pi^*)}{\beta^*}\Bigg]\times(\mu+\delta_1)(\mu+\delta_2)(\mu+\delta_3)\Bigg\}\\
&\qquad + \Bigg\{\lambda\gamma\frac{\pi^*\phi(\pi^*)}{\beta^*}(\mu+\delta_1)(\mu+\delta_2+\mu+\delta_3)\\
&\qquad \times\Big[\lambda\alpha\beta^*\phi(\pi^*)(1-\xi) - \phi'(\pi^*)(1-\pi^*)\lambda\alpha\beta^*(1-\xi)\Big]\\
&\qquad L(\mu+\delta_2)(\mu+\delta_3)\Bigg\}\\
&+\Bigg\{\Big[\lambda\alpha\beta^*\phi(\pi^*)(1-\xi) - \phi'(\pi^*)(1-\pi^*)\lambda\alpha\beta^*(1-\xi) + \mu + \lambda\gamma\pi^*\frac{\phi(\pi^*)}{\beta^*}\Big]\\
&\qquad \psi\lambda\alpha\lambda\gamma(1-\xi)\\
&\qquad \times\Big(a_3 - \lambda\gamma\lambda\alpha(1-\xi)\psi[\delta_1+\mu+\delta_2+\mu+\delta_3]\Big)\Bigg\}\\
&\qquad + (a_1a_2 - a_3)(\delta_1+\mu+\delta_2+\mu+\delta_3)\psi\lambda\alpha\lambda\gamma(1-\xi).\\
&> a_1^2\Big[(\mu+\delta_2)(\mu+\delta_3) + \delta_1(\mu+\delta_3) + \delta_1\delta_2\Big]\psi\lambda\alpha\lambda\gamma(1-\xi).
\end{aligned} \tag{4.42}$$

Consider the term in the first square bracket in the inequality (4.42)

above

$$\delta_1(1-\pi^*)\lambda\alpha(1-\xi)\lambda\gamma\phi(\pi^*)^2 + \lambda\gamma\lambda\alpha(1-\xi)\phi(\pi^*)^2(\delta_1+\mu+\delta_2+\mu+\delta_3)\pi^*$$
$$-\phi'(\pi^*)(1-\pi^*)(\delta_1+\mu+\delta_2+\mu+\delta_3)\pi^*\lambda\alpha\lambda\gamma\phi(\pi^*)(1-\xi)$$
$$+(\mu+\delta_1)(\mu+\delta_2+\mu+\delta_3)\lambda\gamma\frac{\pi^*\phi(\pi^*)}{\beta^*},$$
$$=\lambda\alpha(1-\xi)\lambda\gamma\phi(\pi^*)\Big[\delta_1(1-\pi^*)\phi(\pi^*)+(\delta_1+\mu+\delta_2+\mu+\delta_3)\pi^*\phi(\pi^*)$$
$$-\phi'(\pi^*)(1-\pi^*)(\delta_1+\mu+\delta_2+\mu+\delta_3)\pi^*+(\mu+\delta_2+\mu+\delta_3)L(1-\pi^*)\phi(\pi^*)\Big]$$
$$>\lambda\alpha(1-\xi)\lambda\gamma\phi(\pi^*)(\delta_1+\mu+\delta_2+\mu+\delta_3)(\phi(\pi^*)-\phi'(\pi^*)\pi^*(1-\pi^*)).$$
$$>\lambda\alpha(1-\xi)\lambda\gamma\phi(\pi^*)(\delta_1+\mu+\delta_2+\mu+\delta_3)(\beta^*(\phi(\pi^*)+\hat{\tau})-\phi'(\pi^*)\pi^*(1-\pi^*)).$$

This is because

$$\phi(\pi^*)-\beta^*(\phi(\pi^*)+\hat{\tau})=\phi(\pi^*)(1-\pi^*)(1-\beta^*(1-\hat{\theta}))>0.$$

So $\phi(\pi^*)>\beta^*(\phi(\pi^*)+\hat{\tau})$.

Now consider the second term on the right-hand side in the inequality

(4.42)

$$\frac{\lambda\gamma\pi^*\phi(\pi^*)}{\beta^*}(\mu+\delta_1)(\mu+\delta_2+\mu+\delta_3)[\lambda\alpha\beta^*\phi(\pi^*)(1-\xi)-\phi'(\pi^*)(1-\pi^*)\lambda\alpha\beta^*(1-\xi)]$$
$$L(\mu+\delta_2)(\mu+\delta_3)$$
$$>-\lambda\alpha(1-\xi)\lambda\gamma\phi(\pi^*)\phi'(\pi^*)\pi^*(1-\pi^*)(\mu+\delta_1)(\mu+\delta_2)(\mu+\delta_3)(\delta_1+\mu+\delta_2+\mu+\delta_3)$$
$$>-\lambda\alpha(1-\xi)\lambda\gamma\phi(\pi^*)\phi'(\pi^*)(1-\pi^*)[\pi^*(1-\beta^*)-(1-\pi^*)\beta^*\hat{\theta}](\mu+\delta_1)(\mu+\delta_2)(\mu+\delta_3)$$
$$(\delta_1+\mu+\delta_2+\mu+\delta_3).$$

Hence the sum of the first two terms in the inequality (4.42) exceeds

$$\lambda\alpha\lambda\gamma(1-\xi)(\delta_1+\mu+\delta_2+\mu+\delta_3)\psi.$$

i.e.

$$\Big[\delta_1(1-\pi^*)\phi(\pi^*)^2\lambda\alpha(1-\xi)\lambda\gamma + \frac{\lambda\gamma\pi^*\phi(\pi^*)}{\beta^*}a_2^{'} + (\mu+\delta_1)(\mu+\delta_2+\mu+\delta_3)\frac{\lambda\gamma\pi^*\phi(\pi^*)}{\beta^*}\Big]$$

$$\times(\mu+\delta_1)(\mu+\delta_2)(\mu+\delta_3),$$

$$+\frac{\lambda\gamma\pi^*\phi(\pi^*)}{\beta^*}(\mu+\delta_1)(\mu+\delta_2+\mu+\delta_3)$$

$$\times\Big[\lambda\alpha\beta^*\phi(\pi^*)(1-\xi)-\phi'(\pi^*)(1-\pi^*)\lambda\alpha\beta^*(1-\xi)\Big]L(\mu+\delta_2)(\mu+\delta_3),$$

$$>\lambda\alpha\lambda\gamma(1-\xi)(\delta_1+\mu+\delta_2+\mu+\delta_3)(\mu+\delta_1)(\mu+\delta_2)(\mu+\delta_3)\psi. \tag{4.43}$$

Hence using inequality (4.43) it is sufficient to show that

$$(\delta_1+\mu+\delta_2+\mu+\delta_3)(\mu+\delta_1)(\mu+\delta_2)(\mu+\delta_3)+\Big(\lambda\alpha\beta^*(1-\xi)\phi(\pi^*)$$

$$-\phi'(\pi^*)(1-\pi^*)\lambda\alpha\beta^*(1-\xi)+\mu+\frac{\lambda\gamma\pi^*\phi(\pi^*)}{\beta^*}\Big)\Big(a_3-\lambda\alpha\lambda\gamma(1-\xi)$$

$$\psi\Big[\delta_1+\mu+\delta_2+\mu+\delta_3\Big]\Big)+(a_1a_2-a_3)(\delta_1+\mu+\delta_2+\mu+\delta_3) \tag{4.44}$$

$$>\Big[(\mu+\delta_2)(\mu+\delta_3)+\delta_1(\mu+\delta_3)+\delta_1\delta_2\Big] \tag{4.45}$$

$$\times\Big[\Big(\mu+\delta_1+\mu+\delta_2+\mu+\delta_3\Big)^2\Big]$$

$$+\Big(\frac{\lambda\gamma\pi^*\phi(\pi^*)}{\beta^*}\Big)^2 \tag{4.46}$$

$$+\Big[\lambda\alpha\beta^*\phi(\pi^*)(1-\xi)-\phi'(\pi^*)(1-\pi^*)\lambda\alpha\beta^*(1-\xi)\Big]^2 \tag{4.47}$$

$$+2\Big(\mu+\delta_1+\mu+\delta_2+\mu+\delta_3\Big)\frac{\lambda\gamma\pi^*\phi(\pi^*)}{\beta^*} \tag{4.48}$$

$$+2\Big(\mu+\delta_1+\mu+\delta_2+\mu+\delta_3\Big) \tag{4.49}$$

$$\times\Big[\lambda\alpha\beta^*\phi(\pi^*)(1-\xi)-\phi'(\pi^*)(1-\pi^*)\lambda\alpha\beta^*(1-\xi)\Big]$$

$$+2\lambda\gamma\pi^*\phi(\pi^*)\lambda\alpha(1-\xi)\Big[\phi(\pi^*)-(1-\pi^*)\phi'(\pi^*)\Big]. \tag{4.50}$$

It is straightforward to show that

$$
\begin{aligned}
&(\delta_1+\mu+\delta_2+\mu+\delta_3)(\mu+\delta_1)(\mu+\delta_2)(\mu+\delta_3)+\mu(\mu+\delta_1)(\mu+\delta_2)(\mu+\delta_3)\\
&\quad+(\mu+\delta_1+\mu+\delta_2+\mu+\delta_3)(\mu+\delta_1)(\mu+\delta_2+\mu+\delta_3)\times(\delta_1+\mu+\delta_2+\mu+\delta_3)\\
&\quad+(\mu+\delta_2)(\mu+\delta_3)(\mu+\delta_2+\mu+\delta_3)(\delta_1+\mu+\delta_2+\mu+\delta_3)\\
&>\Big(\mu+\delta_1+\mu+\delta_2+\mu+\delta_3\Big)^2\Big[(\mu+\delta_2)(\mu+\delta_3)+\delta_1(\mu+\delta_3)+\delta_1\delta_2\Big].
\end{aligned}
$$

Hence the term in (4.45) can be cancelled by the terms in (4.44) containing only μ, δ_1, δ_2 and δ_3 and similarly

$$
\begin{aligned}
&(\mu+\delta_2)(\mu+\delta_3)+(\mu+\delta_2+\mu+\delta_3)(\delta_1+\mu+\delta_2+\mu+\delta_3).\\
&>\Big[(\mu+\delta_2)(\mu+\delta_3)+\delta_1(\mu+\delta_3)+\delta_1\delta_2\Big].
\end{aligned}
$$

and (4.46) can be cancelled by the terms in (4.44) containing $\left(\frac{\lambda\gamma\pi^*\phi(\pi^*)}{\beta^*}\right)^2$. The term (4.47)can be found explicitly in (4.44), moreover (4.48) can be cancelled by the terms in (4.44) containing $\lambda\gamma\dfrac{\pi^*\phi(\pi^*)}{\beta^*}$ as

$$
\begin{aligned}
&(\mu+\delta_1)(\mu+\delta_2)(\mu+\delta_3)+\mu(\mu+\delta_2)(\mu+\delta_3)\\
&\quad+(\delta_1+\mu+\delta_2+\mu+\delta_3)(\mu+\delta_1+\mu+\delta_2+\mu+\delta_3)\\
&\quad(\mu+\delta_2+\mu+\delta_3)+(\delta_1+\mu+\delta_2+\mu+\delta_3)(\mu+\delta_1)(\mu+\delta_2+\mu+\delta_3)\\
&>2(\mu+\delta_1+\mu+\delta_2+\mu+\delta_3)\Big[(\mu+\delta_2)(\mu+\delta_3)+\delta_1(\mu+\delta_2+\delta_3)\Big]
\end{aligned}
$$

In a similar fashion, we have

$$
\begin{aligned}
&(\mu+\delta_1+\mu+\delta_2+\mu+\delta_3)(\delta_1+\mu+\delta_2+\mu+\delta_3)^2\\
&\quad>2(\mu+\delta_1+\mu+\delta_2+\mu+\delta_3)\Big[(\mu+\delta_2)(\mu+\delta_3)+\delta_1(\mu+\delta_2+\delta_3)\Big].
\end{aligned}
$$

Hence (4.49) can be cancelled by the terms in (4.44) containing

$$[\lambda\alpha\beta^*\phi(\pi^*)(1-\xi)-\phi'(\pi^*)\lambda\alpha\beta^*(1-\xi)].$$

Finally the terms in (4.44) containing

$$\lambda\gamma\pi^*\phi(\pi^*)\lambda\alpha(1-\xi)[\phi(\pi^*)-(1-\pi^*)\phi'(\pi^*)]$$

will cancel (4.50) as

$$(\mu+\delta_2)(\mu+\delta_3)L+(\mu+\delta_2+\mu+\delta_3)(\delta_1+\mu+\delta_2+\mu+\delta_3)+(\delta_1+\mu+\delta_2+\mu+\delta_3)^2$$
$$>2\Big[(\mu+\delta_2)(\mu+\delta_3)+\delta_1(\mu+\delta_3)+\delta_1\delta_2\Big].$$

Hence $a_1a_2a_3 > a_3^2 + a_1^2a_4$ and all the Routh-Hurwitz conditions are satisfied for $R_0 > 1$. This completes the proof of Theorem 4.3.3

In this following section, we shall show that if $R_0 \leq 1$ the disease-free equilibrium is globally stable.

4.3.4 Global Stability of Equilibrium

In this section, we concentrate on the global stability of disease-free equilibrium DFE. The next theorem is proved using a method similar to Lewis (2000).

4.3.4.1 The Disease-Free Equilibrium (DFE).

Theorem 4.3.4. *The disease-free solution for the system* (4.5) -(4.8) *is globally stable if* $R_0 \leq 1$*, and HIV/AIDS will eventually be eradicated from all PWIDs, as well as needles and syringes.*

Proof: Suppose that $\phi(\pi)$ is monotones decreasing in π. If $\pi(0) = 0$ then $\pi(t) = 0$ for all time. Our proof requires several arguments to demonstrate that $\lim_{t\to\infty}\pi_1(t) = 0$. as well as from this directly that the limsup of $\pi_2(t)$, $\pi_3(t)$ and $\beta(t)$ must ultimately reach to zero

Let $\tilde{\pi}_1(t) = \sup_{\xi\geq t}\pi_1(\xi)$, this is monotone decreasing in t.

Thus, for given $\epsilon > 0$ there exists $t_1(\epsilon)$ such that $\pi_1(t) \leq \pi_1^\infty + \epsilon$ for all $t \geq t_1(\epsilon)$ where $\pi_1^\infty = \limsup_{t\to\infty}\pi_1(t) = \lim_{t\to\infty}\bar{\pi}_1(t)$.

We have the following lemma

Lemma 4.3.1. *If* $\pi_2^{\infty} = \limsup_{t\to\infty} \pi_2(t)$, *then*

$$\pi_2^{\infty} \leq \frac{\delta_1 \pi_1^{\infty}}{\mu + \delta_2}.$$

Proof: From equation (4.6) we have

$$\frac{d\pi_2}{dt} + (\mu_1 + \delta_2)\,\pi_2$$

$$= \delta_1 \pi_1 \times \exp^{(\mu+\delta_2)t},$$

$$\frac{d\pi_2}{dt}\left[\exp^{(\mu+\delta_2)t}\right] + \exp^{(\mu+\delta_2)t}\,\dot{(}\mu + \delta_2)\pi_2$$

$$= \delta_1 \pi_1 \exp^{(\mu+\delta_2)t},$$

$$\frac{d}{dt}\left[\pi_2 \exp^{(\mu+\delta_2)t}\right]$$

$$= \delta_1 \pi_1 \exp^{(\mu+\delta_2)t} \quad \forall t \geq t_1 \quad \textit{and}$$

$$\pi \leqslant \pi_1^{\infty} + \epsilon > 0, \textit{ for any } \quad \epsilon > 0.$$

$$\frac{d}{dt}\left[\pi_2 \exp^{(\mu+\delta_2)t}\right]$$

$$\leq \delta_1 \left(\pi_1^{\infty} + \epsilon\right) \exp^{(\mu+\delta_2)t}.$$

similar to Lewis and Greenhalgh (2001) work, integrating over $[t_1(\epsilon), t]$ provides

$$\pi_2 \exp^{(\mu+\delta_2)t} - \pi_2\left(t_1(\epsilon)\right) \exp^{(\mu+\delta_2)t_1(\epsilon)} \leq \frac{\delta_1(\pi_1^{\infty} + \epsilon)}{\mu + \delta_2.} \left[\exp^{(\mu+\delta_2)t} - \exp^{(\mu+\delta_2)t_1(\epsilon)}\right].$$

$$\pi_2 \exp^{(\mu+\delta_2)t} \leq \pi_2\left(t_1(\epsilon)\right) \exp^{(\mu+\delta_2)t_1(\epsilon)} + \frac{\delta_1\left(\pi_1^{\infty} + \epsilon\right)}{\mu + \delta_2} \left[\exp^{(\mu+\delta_2)t} - \exp^{(\mu+\delta_2)t_1(\epsilon)}\right].$$

By dividing both sides by $\exp^{(\mu+\delta_2)t}$, we get

$$\pi_2 \leq \pi_2\left(t_1(\epsilon)\right) \exp^{(\mu+\delta_2)(t-t_1(\epsilon))} + \frac{\delta_1\left(\pi_1^{\infty} + \epsilon\right)}{\mu + \delta_2} \left[1 - \exp^{-(\mu+\delta_2)(t-t_1(\epsilon))}\right].$$

By choosing $t_2(\epsilon) > t_1(\epsilon)$ $\forall t \geq t_2(\epsilon)$ large enough, so that we have $e^{(\mu+\delta_2)(t-t_1(\epsilon))} \leq \epsilon$. Thus

$$\bar{\pi}_2(t) \leq \pi_2\left(t_1(\epsilon)\right) \cdot \epsilon + \frac{\delta_1\left(\pi_1^{\infty} + \epsilon\right)}{\mu + \delta_2}.$$

$$\bar{\pi}_2(t) \leq \epsilon + \frac{\delta_1\left(\pi_1^{\infty} + \epsilon\right)}{\mu + \delta_2}, \qquad \forall t \geq t_2(\epsilon).$$

Taking $\bar{\pi}_2(t) = \sup_{\xi \geq t} \pi_1(\xi)$, and letting $t \to \infty$ then

$$\pi_2^\infty \leq \epsilon + \frac{\delta_1 \pi_1^\infty + \epsilon}{\mu + \delta_2}.$$

$$\pi_2^\infty \leq \frac{\delta_1 \pi_1^\infty}{\mu + \delta_2} + \epsilon_1.$$

where $\epsilon_1 = \epsilon\left(1 + \frac{\delta_1}{\mu + \delta_2}\right)$. But ϵ is arbitrarily small. So the result follows.

Corollary 4.3.1. *If* $\pi_3^\infty = \limsup_{t\to\infty} \pi_3(t)$ *then*

$$\pi_3^\infty \leq \frac{\delta_1 \delta_2 \pi_1^\infty}{(\mu + \delta_2)(\mu + \delta_3)}.$$

Proof: Using equation (4.7) and following the technique of Lemma 4.3.1. We determine that

$$\pi_3^\infty \leq \frac{\delta_2 \pi_2^\infty}{\mu + \delta_3}.$$

The result follows.

Lemma 4.3.2. *If* $\beta^\infty = \limsup_{t\to\infty} \beta(t)$ *then*

$$\beta^\infty \leq \frac{\pi_1^\infty + \pi_2^\infty + \pi_3^\infty}{\hat{\theta} + \hat{\tau}}.$$

Proof: From the equation (4.8)

$$\frac{d\beta}{dt} \leq \lambda\gamma\phi\left(\pi_1 + \pi_2 + \pi_3\right) - \beta\left(\lambda\gamma\hat{\theta} + \tau\right)\phi,$$

multiplying by the integrating factor $e^{\int_0^t (\lambda\gamma\hat{\theta}+\tau)\phi d\xi}$

$$\frac{d}{dt}\left[\beta e^{\int_0^t (\lambda\gamma\hat{\theta}+\tau)\phi d\xi}\right] \leq \lambda\gamma\phi\left(\pi_1 + \pi_2 + \pi_3\right) e^{\int_0^t (\lambda\gamma\hat{\theta}+\tau)\phi d\xi},$$

$$\beta(t) e^{\int_0^t (\lambda\gamma\hat{\theta}+\tau)\phi d\xi} - \beta\left(t_0\right) e^{\int_0^{t_0} (\lambda\gamma\hat{\theta}+\tau)\phi d\xi} \leq \frac{\lambda\gamma}{\lambda\gamma\hat{\theta} + \tau}\left(\pi_1^\infty + \pi_2^\infty + \pi_3^\infty + \xi\right)\left[e^{\int_0^t (\lambda\gamma\hat{\theta}+\tau)\phi d\xi}\right]_{t_0}^t$$

$$\beta(t) \leq \beta(t_0) e^{-\int_{t_0}^t (\lambda\gamma\hat{\theta}+\tau)\phi d\xi} + \frac{\lambda\gamma}{\lambda\gamma\hat{\theta} + \tau}\left(\pi_1^\infty + \pi_2^\infty + \pi_3^\infty + \xi\right)\left[1 - e^{-\int_{t_0}^t \left(\lambda\gamma\hat{\theta}+\tau\right)\phi d\xi}\right].$$

Given $\xi > 0$ there exists to such that for t_0 $\pi_1 + \pi_2 + \pi_3 \leq \pi_1^\infty + \pi_2^\infty + \pi_3^\infty + \xi$

If $\phi(\pi) \geq \epsilon$ and we use the same idea as in Lemma 4.3.1, then there are two cases

(*i*) If $\int_{t_0}^{t} \phi(\xi)d\xi \to \infty$, as $t \to \infty$,then the result follows.

(*ii*) If $\int_{t_0}^{t} \phi(\xi)d\xi$ dose not approach infinity ,as $t \to \infty$ then we have $\int_{0}^{\infty} \phi(\xi)d\xi \leq M$ for some $M < \infty$.

In this case given $\xi > 0$ $\exists$ t_0 such that $\int_{t_0}^{\infty} \phi(\xi)d\xi \leq \xi$ for all $t \geq t_0$. From the equation (4.8) we find that

$$\frac{d\beta}{dt} \leqslant (1-\beta)\lambda\gamma\phi\pi - \beta\tau \leqslant \lambda\gamma\phi - \beta\tau.$$

Moving $\beta\tau$ over the other side, multiplying by an integrating factor and integrating.

$$\frac{d}{dt}\left(\beta e^{\tau t}\right) \leqslant \lambda\gamma\phi e^{\tau t}.$$

Then integrating between t and t_0

$$\begin{aligned} 0 \leq \beta(t) &\leq \beta\left(t_0\right) e^{-\tau(t-t_0)} + \int_{t_0}^{t} \lambda\gamma\phi e^{-\tau(u-t)}du, \\ &\leq e^{-\tau(t-t_0)} + \int_{t_0}^{\infty} \lambda\gamma\phi du \\ &= e^{-\tau(t-t_0)} + \lambda\gamma\epsilon. \end{aligned}$$

If t is large enough then

$$0 \leq \beta(t) \leq \epsilon + \lambda\gamma\epsilon.$$

$$0 \leqslant \beta^{\infty} \leqslant (\lambda\gamma + 1)\epsilon.$$

Hence we deuce that $\beta^{\infty} = 0$, as ϵ is arbitrary in other words $\beta(t) \to 0$ as $t \to \infty$. Thus the result of Lemma (4.3.2) follows.

Next, we assume that $\pi_1^{\infty} > 0$. We note that by equation (4.5)

$$\frac{d\pi_1}{dt} \leq \left(1-\pi_1\right)\lambda\beta\alpha\phi(\pi)(1-\xi) - \left(\mu+\delta_1\right)\pi_1,$$

By Lemma 4.3.2 there exists to such that for $t \geq t_0$

$$\frac{d\pi_1}{dt} \leq \left(1-\pi_1\right)\lambda\alpha(1-\xi)\left(\frac{\pi_1^{\infty}+\pi_2^{\infty}+\pi_3^{\infty}}{\hat{\theta}+\hat{\tau}} + \epsilon\right) - \left(\mu+\delta_1\right)\pi_1.$$

Now using the definition of the basic reproductive number (4.9) and

Lemma 4.3.1 and Corollary 4.3.1 we get that

$$\frac{d\pi_1}{dt} \leq (1-\pi_1)\,\lambda\alpha(1-\xi)\,(\mu+\delta_1)\,(R_0+\epsilon_1)\,\pi_1^{\infty} - (\mu+\delta_1)\,\pi_1,$$

where $\epsilon_1 = \dfrac{\lambda\alpha(1-\xi)\epsilon}{(\hat{\theta}+\hat{\tau})\,(\mu+\delta_1)\,\pi_1^{\infty}}$,

$$\leq (\mu+\delta_1)\Big[(R_0+\epsilon_1)\,\pi_1^{\infty} - \pi_1\left(1+R_0\pi_1^{\infty}\right)\Big].$$

Hence we have

$$\frac{d}{dt}\Big[\pi_1(t)\exp\Big[\,(\mu+\delta_1)\left(1+R_0\pi_1^{\infty}\right)t\Big]\Big]$$

$$\leq (\mu+\delta_1)\,(R_0+\epsilon_1)\,\pi_1^{\infty}\exp\Big[(\mu+\delta_1)\left(1+R_0\pi_1^{\infty}\right)t\Big].$$

Using the same procedure as in Lemma 4.3.1, we obtain that

$$\pi_1(t)e^{(\mu+\delta_1)(1+R_0\pi_1^{\infty})t} - \pi_1(t_0)e^{(\mu+\delta_1)(1+R_0\pi_1^{\infty})t_0}$$

$$\leq \frac{(R_0+\epsilon_1)}{1+R_0\pi_1^{\infty}}\pi_1^{\infty}\Big[e^{((\mu+\delta_1)(1+R_0\pi_1^{\infty})t} - e^{((\mu+\delta_1)(1+R_0\pi_1^{\infty})t_0}\Big],$$

and hence that there exists $t_1 \geq t_0$ such that for $t_1 \geq t_1$

$$\pi_1 \leq \frac{R_0\pi_1^{\infty}}{1+R_0\pi_1^{\infty}} + \epsilon_1\Big(1+\frac{\pi_1^{\infty}}{1+R_0\pi_1^{\infty}}\Big),$$

and thus as ϵ_1 is arbitrarily small

$$\pi_1^{\infty} \leq \frac{R_0\pi_1^{\infty}}{1+R_0\pi_1^{\infty}}.$$

Therefore as $\pi_1^{\infty} > 0, 1 \leq \dfrac{R_0}{1+R_0\pi_1^{\infty}}$. Hence $1 \geq R_0 \geq 1+R_0\pi_1^{\infty}$.

So this is a contradiction and hence $\pi_1^{\infty} = 0$. So Lemma 4.3.1,Corollary 4.3.1 and Lemma 4.3.2 then imply $\pi_1^{\infty} = \pi_2^{\infty} = \pi_3^{\infty} = \beta^{\infty} = 0$ and hence π_1, π_2, π_3 and β all approach zero as $t \to \infty$ completing the proof of Theorem (4.3.4).

Next ,applying condition of the second case of 4.3.2 , that $\beta^{\infty} = 0$, if $\beta(t) \to 0$ as $t \to \infty$. the equation (4.5) becomes

Suppose the definition of $\liminf_{t\to\infty}\pi_i(t) = \pi_{i,\infty}$ and note that $\pi_i^{\infty} \geq \pi_{i,\infty} \geq 0$ and $\beta^{\infty} \geq \beta_{\infty} \geq 0$. This implies that $\pi_i^{\infty} = \pi_{i,\infty} = 0$ and $\beta^{\infty} = \beta_{\infty} = 0$, and hence $\lim_{t\to\infty}\pi_1(t) = 0$.It is straightforward to show

that

$$\lim_{t\to\infty} \pi_2(t) = \lim_{t\to\infty} \pi_3(t) = \lim_{t\to\infty} \beta(t) = 0.$$

This completes the proof of the global stability of DFE when $1 \geq R_0 \geq 0$. Following that, we will look into the persistence of the disease in the system.

4.3.5 Persistence of the Disease When $R_0 > 1$

In this section, we will demonstrate that if $R_0 > 1$ and HIV/AIDS are present in the community at the outset, whether in addicts groups or shooting galleries, the disease will remain in both PWIDs and needles. Note that the analytical results for our model show exactly the results of Theorem 2.4 of Lewis (2000) for equations 4.6 and 4.7 and different argument results, thus including awareness program function for equations 4.5 and 4.8

Theorem 4.3.5. *Suppose that $R_0 > 1$ and either $\pi_i(0) > 0$ for some $i = 1,2,3$ or $\beta(0) > 0$. Then there exists a fixed $\varepsilon > 0$, which depends only on the model parameters, not the initial conditions, such that for some $\eta > 0$ and $i = 1,2,3$*

$$\pi_i \geq \varepsilon\pi_i^* \text{ and } \beta \geq \varepsilon\beta^*, \quad \forall t \geq \eta. \tag{4.51}$$

Proof: We prove this by following a similar argument used to prove Theorem 2.4 ofLewis (2000). We need to follow several steps to prove this result. The common sense argument is π_1 is the dominant component of $(\pi_1, \pi_2, \pi_3, \beta)$ If π_1 is small, then all of the other components will be small as well. From the local stability of the equilibria of the system, we know that the disease-free (DFE) equilibrium is unstable if $R_0 > 1$. We are going to show that π_1 cannot become arbitrarily close to zero.

Let $\bar{\pi}_1(t) = \inf_{\xi \geq t} \pi_1(\xi)$, this is monotone increasing in t. For given $\epsilon > 0$, there exists $t_5(\varepsilon)$ such that $\pi_1(t) \geq \pi_{1,\infty} - \varepsilon$ for all $t \geq t_5(\varepsilon)$ where $\pi_{1,\infty} = \liminf_{t\to\infty} \pi_1(t)$.

Lemma 4.3.3. *Let* $\pi_{2,\infty} = \liminf_{t\to\infty} \pi_2(t)$*, then*

$$\pi_{2,\infty} \geq \frac{\delta_1 \pi_{1,\infty}}{\mu + \delta_2}.$$

Proof. From equation (4.6), and the result was proved in the (Lewis and Greenhalgh 2001) paper, that was

$$\pi_2(t) \geq \frac{\delta_1 \pi_{1,\infty}}{(\mu + \delta_2)} - \varepsilon_1.$$

Lemma 4.3.4. *There exists a time* $T > 0$ *and a small quantity* $\eta > 0$ *such that* $\phi(\pi) \geq \eta > 0$ *for* $t \geq T$*.*

Proof. If $\phi(\pi) > 0$ in $[0, 1]$ this is straightforward. Otherwise, $\exists\ \pi^*$ in $[0, 1]$ with $\phi(\pi^*) = 0$ and the arguments used in the existence uniqueness proof show that this result is true.

Corollary 4.3.2. *Let* $\pi_{3,\infty} = \liminf_{t\to\infty} \pi_3(t)$*, then*

$$\pi_{3,\infty} \geq \frac{\delta_1 \delta_2 \pi_{1,\infty}}{(\mu + \delta_2)(\mu + \delta_3)}.$$

Proof. By using equation(4.7) and the result was proved in the (Lewis and Greenhalgh 2001) paper, that was

$$\pi_{3,\infty} \geq \frac{\delta_2 \pi_{2,\infty}}{\mu + \delta_3}.$$

Corollary 4.3.3. *Let* $\beta_\infty = \liminf_{t\to\infty} \beta(t)$*, then*

$$\beta_\infty \geq \frac{(\pi_{1,\infty} + \pi_{2,\infty} + \pi_{3,\infty})\eta}{(1 + \hat{\theta} + \hat{\tau})}.$$

Proof. Equation (4.8) can be written in the form

$$\frac{d\beta}{dt} = \lambda\gamma\pi_i\phi - \beta\lambda\gamma\Big((1+\hat{\theta})\phi + \hat{\tau}\Big),$$
$$\geq \lambda\gamma\pi_i\phi - \beta\lambda\gamma\Big(1+\hat{\theta}+\hat{\tau}\Big),$$

So given $\varepsilon > 0$, there exists $t_7\ (\varepsilon)$ such that

$$\frac{d\beta}{dt} \geq \lambda\gamma(\pi_{1,\infty} + \pi_{2,\infty} + \pi_{3,\infty} - \epsilon)\eta - \beta\lambda\gamma\Big((1+\hat{\theta}) + \hat{\tau}\Big),$$

for $t \leq t_7(\varepsilon)$. A similar argument to Lemma (4.3.3) then shows that Corollary (4.3.3) holds. We use a similar argument as in the previous equations to obtain the following

$$\frac{d}{dt}\beta e^{[\lambda\gamma(1+\hat{\theta})+\hat{\tau}]d\xi} \geq \lambda\gamma(\pi_{1,\infty} + \pi_{2,\infty} + \pi_{3,\infty} - \epsilon)\eta e^{\int_0^t[\lambda\gamma(1+\hat{\theta})+\hat{\tau}]d\xi}.$$
$$\beta \geq \frac{\lambda\gamma}{\lambda\gamma(1+\hat{\theta})+\hat{\tau}}(\pi_{1,\infty} + \pi_{2,\infty} + \pi_{3,\infty} - \epsilon)\eta[1 - e^{-\int_0^t[\lambda\gamma(1+\hat{\theta})+\hat{\tau}]du}]$$

If $\int_0^t \phi(\xi)d\xi = \infty$, then we have that

$$\beta_\infty \geq \frac{(\pi_{1,\infty} + \pi_{2,\infty} + \pi_{3,\infty})\eta}{(1+\hat{\theta}+\hat{\tau})}.$$

Note that either $\pi_{1,\infty} \geq \frac{1}{2}\epsilon\pi_1^*$, or $\pi_{1,\infty} < \frac{1}{2}\epsilon\pi_1^*$.

First, we assume that $\pi_{1,\infty} \geq \frac{1}{2}\epsilon\pi_1^*$. So there $\exists\ T_1$ such that for $t \geq T_1, \pi_1 \geq \frac{1}{4}\epsilon\pi_1^*$. By using the result of Lemma 4.3.3, we get that

$$\pi_{2,\infty} \geq \frac{\delta_1\pi_{1,\infty}}{\mu+\delta_2},$$
$$\textit{So} \qquad \pi_{2,\infty} \geq \frac{1}{2}\frac{\epsilon\delta_1}{\mu+\delta_2}\pi_1^* = \frac{1}{2}\epsilon_2\pi_2^*.$$

Here $\epsilon_2 = \frac{1}{2}\frac{\epsilon\delta_1\pi_1^*}{(\mu+\delta_2)\pi_2^*}$.

Assuming in the same way as before, there $\exists\ T_2$ such that for $t \geq T_2, \pi_2 \geq \frac{1}{4}\epsilon\pi_2^*$. In a similar way, by using the Corollary 4.3.2 there exists T_3 such that for $t \geq T_3, \pi_3 \geq$

$\frac{1}{4}\epsilon\pi_3^*$. Similarly, using the result of Corollary 4.3.3, we have that

$$\beta_\infty \geq \frac{\pi_{1,\infty}\eta}{1+\hat{\theta}+\hat{\tau}}.$$

$$\beta_\infty \geq \frac{\frac{1}{2}\epsilon\pi_1^*\eta}{1+\hat{\theta}+\hat{\tau}} = \frac{1}{2}\epsilon_4\beta^*.$$

where $\epsilon_1 = \frac{\epsilon}{1+\hat{\theta}+\hat{\tau}}\ \frac{\pi_1^*\eta}{\beta^*}$. So there exists T_4 such that for $t \geq T_4$, $\beta \geq \frac{1}{4}\epsilon_1\beta^*$. Hence if $T = \max\{T_1T_2, T_3, T_4\}$ and $\bar{\epsilon}$=min $\left\{\frac{1}{4}\epsilon, \frac{1}{4}\epsilon_1\right\}$ the results of (4.51) hold with ϵ replaced by $\bar{\epsilon}$.

Next, assume that $\pi_{1,\infty} < \frac{1}{2}\epsilon\pi_1^*$, so here we want know in which case there exists $\kappa \geq \Delta t$, where $\pi_1(\kappa) < \frac{1}{2}\epsilon\pi_1^*$.

Now, letting

$$t_0 = \inf\left\{\kappa \geq \Delta t, \pi_1(\kappa) < \frac{1}{2}\epsilon\pi_1^*\right\}, \qquad \textit{and}$$
$$t_1 = \inf\left\{\kappa \geq t_0, \pi_1(\kappa) > \frac{1}{2}\epsilon\pi_1^*\right\}.$$

Here ϵ is a constant value that is both positive and fixed. According to the definition of t_0 we have that $\pi_1(t_0+\nu) < \frac{1}{2} \in \pi_1^*$.If ν is small and positive, then $t_1 > t_0$. By continuity $\pi_1(t_0) = \pi_1(t_1) = \frac{1}{2}\epsilon\pi_1^*$, and therefore π_1 is less than $\frac{1}{2}\epsilon\pi_1^*$in (t_0, t_1) and greater than $\frac{1}{2}\epsilon\pi_1^*$ just after t_1. We now show that if π_1. becomes small then π_3, π_3 and β must become small also.

Lemma 4.3.5. *Suppose that Δ is small. Then there exists a time $\bar{T}_1 > 0$ dependent only on the model parameters, Δ and ϵ. With $0 < \pi_2 < \left(\frac{1}{2}+\Delta\right)$ for t between $t_0+\bar{T}_1$ and t_1,*

Proof. We have that $\pi_1 \leq (1/2)\ \epsilon\ \pi_1^* \in [t_0, t_1]$. Thus we write the equation (4.6) as

$$\frac{d\pi_2}{dt} \leq \frac{1}{2}\epsilon\pi_1^*\delta_1 - (\mu+\delta_2)\pi_2,$$
$$\frac{d}{dt}\left[\pi_2 e^{(\mu+\delta_2)t}\right] \leq \frac{1}{2}\epsilon\pi_1^*\delta_1 e^{(\mu+\delta_2)t}.$$

By integrating over $[t_0, t]$ this gives

$$\pi_2 e^{(\mu+\delta_2)t} - \pi_2(t_0)e^{(\mu+\delta_2)t_0} \leq \frac{1}{2}\frac{\epsilon\pi_1^*\delta_1}{\mu+\delta_2}\left(e^{(\mu+\delta_2)t} - e^{(\mu+\delta_2)t_0}\right),$$

$$\pi_2 \leq \left(e^{-(\mu+\delta_2)(t-t_0)}\right) + \frac{1}{2}\epsilon\pi_2^*.$$

So the result here was proved in the (Lewis and Greenhalgh 2001) and paper. We have proved that if π_1 is small, it causes π_2 to become small as well, Next, we will prove similar results for π_3 and β.

Corollary 4.3.4. *Suppose that $\Delta > 0$. Then there exists $\bar{T}_2 > 0$ depending only on the model parameters, Δ and ϵ such that for $t \in [t_0 + \bar{T}_1 + \bar{T}_2, t_1]$ π_3 is at most $\left(\frac{1}{2} + 2\Delta\right)\pi_3^*\epsilon$, assuming that $t_0 + \bar{T}_1 + \bar{T}_2 < t_1$*

Proof.

To prove that we follow the similar proof of Lemma 4.3.5, so from the equation (4.7) has the form as

$$\frac{d\pi_3}{dt} \leq \left(\frac{1}{2} + \Delta\right)\epsilon\pi_2^*\delta_2 - (\mu + \delta_3)\,\pi_3.$$

And then integrating the equation over $[t_0 + \bar{T}_1, t]$, then the result follows.

Corollary 4.3.5. *Suppose that Δ is small. Then there exists $\bar{T}_3 > 0$ depending only on the model parameters, Δ and ϵ such that for $t \in [t_0 + \bar{T}_1 + \bar{T}_2 + \bar{T}_3, t_1]$ β is at most $\left(\frac{1}{2} + 3\Delta\right)\beta^*\epsilon_1$, where*

$$\epsilon_1 = max\left(1, \frac{\pi^*\phi(\pi^*) + (1-\pi^*)\phi(\pi*)\hat{\theta} + \hat{\tau}}{\hat{\theta} + \hat{\tau}}\right)\epsilon.$$

We supposing that $t_0 + \bar{T}_1 + \bar{T}_2 + \bar{T}_3 < t_1$

Proof. We use a similar argument with equation (4.8), to obtain the following

$$\frac{d\beta}{dt} = \lambda\gamma\phi(\pi)\pi - \beta\lambda\gamma(\hat{\theta}\phi(\pi) + \hat{\tau}).$$

So for $t \geq \bar{T}_1 + \bar{T}_2$,

$$\frac{d\beta}{dt} \leq \lambda\gamma(\frac{1}{2} + 2\Delta)\phi(\pi) - \beta\lambda\gamma(\hat{\theta} + \tau)\phi(\pi).$$

By integrating over $[t_0 + \bar{T}_1 + \bar{T}_2, t]$, we get that

$$\frac{d}{dt}\left[\beta e^{\int_{t_0+\bar{T}_1+\bar{T}_2}^{t}\left[\lambda\gamma(\hat{\theta}+\hat{\tau})\phi(\pi)\right]d\xi}\right] \leq \lambda\gamma\Big(\frac{1}{2}+2\Delta\Big)\epsilon\pi^*\phi(\pi)$$

$$exp\left[\int_{t_0+\bar{T}_1+\bar{T}_2}^{t}\lambda\gamma\Big(\hat{\theta}+\hat{\tau}\Big)\phi(\pi)d\xi\right].$$

$$\beta \leq \beta(t_0+\bar{T}_1+\bar{T}_2)\; e^{-\int_{t_0+\bar{T}_1+\bar{T}_2}^{t}\lambda\gamma(\hat{\theta}+\hat{\tau})\phi(\pi)d\xi}$$

$$+\frac{\Big(\frac{1}{2}+2\Delta\Big)}{\hat{\theta}+\hat{\tau}}\epsilon\pi^*\left[1-e^{-\int_{t_0+\bar{T}_1+\bar{T}_2}^{t}\lambda\gamma(\hat{\theta}+\hat{\tau})\phi(\pi)d\xi}\right]$$

By Lemma 4.3.5 there exists a time $T > 0$ and a small quantity $\eta > 0$ such that $\phi(\pi) \geq \eta > 0$ for $t \geq T$. So $t \geq max\ (T, t_0 + \bar{T}_1 + \bar{T}_2)$ is sufficiently large, then $t \geq t_0 + \bar{T}_1 + \bar{T}_2 + \bar{T}_3$. So there we have that

$$\beta \leq \frac{\frac{1}{2}+3\Delta}{\hat{\theta}+\hat{\tau}}\epsilon\pi^*.$$

Note that it is not always obvious that $\beta(t) \leq \Big(\frac{1}{2}+3\Delta\Big)\epsilon\beta^*$ since we have

$$\beta^* = \frac{\pi^*\phi(\pi^*)}{\pi^*\phi(\pi^*)+(1-\pi^*)\phi(\pi^*)\hat{\theta}+\hat{\tau}} \leq \frac{\pi^*}{\hat{\theta}+\hat{\tau}},$$

and it may be that

$$\beta^* \leq \frac{\pi^*}{\hat{\theta}+\hat{\tau}}.$$

However if we define

$$\epsilon_1 = max\left(1, \frac{\Big[\pi^*\phi(\pi^*)+(1-\pi^*)\phi(\pi*)\hat{\theta}+\hat{\tau}\Big]}{\hat{\theta}+\hat{\tau}}\right)\epsilon,$$

(note that $\epsilon_1 > \epsilon$), then the result of Corollary 4.3.5 follows.

We have proved that if π_1 approaches zero, then all components must approach zero as well. Next, we going to show that π_1 cannot become arbitrarily small. This is obtained by showing that t_1 may be bounded above by a fixed finite value that is only affected by the model parameters ϵ and Δ. Thus π_1 does not go below $\frac{1}{2}\epsilon\pi_1^*$ for a long enough time to approach zero arbitrarily. Then there are two possible cases. The first one is

i. π_1 is remains beneath $\frac{1}{2}\epsilon\pi_1^*$,long enough for other components to become small.

ii. π_1 increases above $\frac{1}{2}\epsilon\pi_1^*$, before other components become small.

As a result, we have that either

A. $t_1 \geq t_0 + \max\left[\bar{T}_1, \bar{T}_1 + \bar{T}_2, \bar{T}_1 + \bar{T}_2 + \bar{T}_3\right]$, or

B. $t_1 < t_0 + \max\left[\bar{T}_1, \bar{T}_1 + \bar{T}_2, \bar{T}_1 + \bar{T}_2 + \bar{T}_3\right]$.

Therefore , we will demonstrate that $t_1 < T$ where T is a fixed finite value that is only affected by the model parameters, ϵ and Δ. Then the result is completed, if case (2) is true then π_1 increases above $\frac{1}{2}\epsilon\pi_1^*$, before other components become small. Now, we are dealing with the first case where t_1 occurs at a time greater than or equal to the time that other components become small. we are going to prove that π_1 cannot stay small constantly, by using the result of the disease-free equilibrium is unstable when $R_0 > 1$.

Corollary 4.3.6. *Suppose that $G(\omega, \epsilon)$ be a polynomial of degree n^{th} in ω and ϵ. Indicate the roots (can be complex)$G(\omega, \epsilon) = 0$ by $\omega_i(\epsilon)$ for $j = 1, 2, ..., n$. Then in a neighbourhood of $\epsilon = 0$, each $\omega_i(\epsilon)$ is defined and continuous in ϵ.*

Proof. Although $G(\omega, \epsilon)$ is a polynomial, it is analytic in the $(0, 0)$ neighbourhood,Corollary 6.6 in Chow and Hale (1982) yields the result.

Our following Lemma is an important part of the argument to show π_1 cannot stay small constantly.

Lemma 4.3.6. *If $\pi_1(t)$ decreases below $\frac{1}{2}\epsilon\pi_1^*$ at time T_0, then $\pi_1(t)$ returns to $\frac{1}{2}\epsilon\pi_1^*$ by of least time $t_1^+ = t_0 + \max\left[\bar{T}_1, \bar{T}_1 + \bar{T}_2, \bar{T}_1 + \bar{T}_2 + T_3, t_2 + \bar{T}_4\right]$ where $t_1^+ - t_0$ is finite are depends only on the parameters model Δ, ϵ.*

Proof. We are going to follow a similar technique to the one used in Lemma 2.5 in Lewis (2000). Assume that ϵ_2 is real and positive and $1 \geq \epsilon_2 \geq 0$, then we define the matrix $\mathbf{J}(\epsilon_2)$ as

$$\mathbf{J}(\epsilon_2) = \begin{bmatrix} -(\mu+\delta_1) & 0 & 0 & \lambda\alpha(1-\xi)\phi(\epsilon_2)(1-\epsilon_2) \\ \delta_1 & -(\mu+\delta_2) & 0 & 0 \\ 0 & \delta_2 & -(\mu+\delta_3) & 0 \\ \lambda\gamma\phi(\epsilon_2) & \lambda\gamma\phi(\epsilon_2) & \lambda\gamma\phi(\epsilon_2) & -(\lambda\gamma(\epsilon_2+\hat{\theta})+\tau) \end{bmatrix}.$$

We evaluated the system at the the disease-free equilibrium points as in the proof of Theorem 4.3.3, when $\epsilon_2 = 0$, $\mathbf{J}(0) = \mathbf{J}$, Then we denote the eigenvalues of $\mathbf{J}(\epsilon_2)$ by ω_i for $i = 1, ..4$. Since $\mathbf{J}(\epsilon_2) + M\mathbf{I}$ is a non-negative irreducible matrix where M is big and positive, then the characteristic equation of $J\mathbf{J}(\epsilon_2) + M\mathbf{I}$ has a simple root equal to its spectral radius, according to Lemma 2.5 in Lewis (2000) and Lemma 2.1 from Nold (1980).

Then the eigenvalues of $\mathbf{J}(\epsilon_2) + M\mathbf{I}$ are

$$\mathbf{J}(\epsilon_2) + M\mathbf{I} = M + \omega_1(\epsilon_2), M + \omega_2(\epsilon_2), M + \omega_3(\epsilon_2) \textit{ and } M + \omega_4(\epsilon_2).$$

If the root $M + \omega_1(\epsilon_2)$ is real, then all other eigenvalues of $J(\epsilon_2) + MI$ have strictly smaller real parts. As a result, the other eigenvalues of $J(\epsilon_2)$ have strictly smaller real parts if $\omega_1(\epsilon_2)$ is real too. This is especially true for $\epsilon_2 = 0$. Furthermore, according to the definition of Corollary4.3.6

Thus, as $\epsilon_2 \to 0$ $\omega_1(\epsilon_2) \to \omega_1(0)$ and we know that $\omega_1(0) > 0$ if $R_0 > 1$ as Theorem 4.3.3 shown that about the disease-free equilibrium points. As a result, we may verify that $\omega_1(\epsilon_2) > 0$, by picking ϵ_2 small enough.

We can assume that $1 > \epsilon_2 > 0$ without any loss of generality. We can choose ϵ_2 that is small enough that

$$\frac{1}{2}\epsilon\pi_1^* + \left(\frac{1}{2} + \Delta\right)\epsilon\pi_2^* + \left(\frac{1}{2} + 2\Delta\right)\epsilon\pi_3^* < \epsilon_2.$$

By using the definition of Lemma 4.3.5 and Corollary 4.3.4, we find that $\pi_1 + \pi_2 + \pi_3 < \epsilon_2$ for $t_1 > t \geq t_0 + \bar{T}_1 + \bar{T}_2$. Define $t_2 = \inf\{\zeta : \text{for } t_1 > t \geq t_0 + \zeta, \pi(t) < \epsilon_2,$

Thus we have two cases either $t_2 = 0$ or $\pi(t_0 + t_2) = \epsilon_2$ where $t_0 + t_2$ is the last time before t_1 that $\pi(t) \geq \epsilon_2$. Note that $t_2 \leq \bar{T}_1 + T_2$. If $t_1 < t_0 + \bar{T}_1 + \bar{T}_2$ then we go the result we want to prove. Now, we consider the case where $t_1 \geq t \geq t_0 + \bar{T}_t + \bar{T}_2$. The system of the equations (4.5)-(4.8) can be written as following for $t_1 \geq t \geq t_0 + \bar{T}_1 + \bar{T}_2$

$$\begin{aligned}
\frac{d\pi_1}{dt} &\geq (1-\epsilon_2)\lambda\beta\phi(\epsilon_2)(1-\xi) - (\mu+\delta_1)\pi_1, \\
\frac{d\pi_2}{dt} &= \delta_1\pi_1 - (\mu+\delta_2)\pi_2, \\
\frac{d\pi_3}{dt} &= \delta_2\pi_2 - (\mu+\delta_3)\pi_3, \\
\frac{d\beta}{dt} &\geq \lambda\gamma\pi_i\phi(\epsilon_2) - \left(\lambda\gamma\left(\epsilon_2 + \hat{\theta}\right) + \hat{\tau}\right)\beta.
\end{aligned}$$

We have that

$$\frac{d\mathbf{x}}{dt} \geq \mathbf{J}(\epsilon_2)\,\mathbf{x}.$$

where $\mathbf{x} = (\pi_1, \pi_2, \pi_3, \beta)^T$. We know that the left eigenvector of $\mathbf{J}(\epsilon 2)$ is strictly positive $e = (e_1, e_2, e_3, e_4)$ which corresponds to the spectral radius $\omega_1(\epsilon_2)$, as Lemma 2.1 in Nold (1980). Thus

$$e\frac{d\mathbf{x}}{dt} \geq eJ(\epsilon_2)\,\mathbf{x} = \omega_1(\epsilon_2)\,e \cdot \mathbf{x}.$$

Integrating over $[t_0 + t_2, t]$

$$\begin{aligned}
e \cdot \mathbf{x}(t) &\geq e \cdot \mathbf{x}(t_0 + t_2)\exp\left[\omega_1(\epsilon_2)(t - t_0 - t_2)\right], \\
&\geq (e_1\pi_1(t_0 + t_2) + e_2\pi_2(t_0 + t_2) + e_3\pi_3(t_0 + t_2))\exp\left[\omega_1(\epsilon_2)(t - t_0 - t_2)\right], \\
&\geq \pi(t_0 + t_2)\,m\,\mathrm{in}(e_1, e_2, e_3)\exp\left[\omega_1(\epsilon_2)(t - t_0 - t_2)\right],
\end{aligned}$$

So we have the following conditions. If $t_2 > 0_2$, then

$$e \cdot \mathbf{x}(t) = \epsilon_2 \min(e_1, e_2, \epsilon_3)\exp\left[\omega_1(\epsilon_2)(t - t_0 - t_2)\right].$$

On the other hand if $t_2 = 0$, then

$$e \cdot \mathbf{x}(t) \geq \frac{1}{2}\epsilon\pi_1^* \min(e_1, e_2, e_3)\exp\left[\omega_1(\epsilon_2)(t - t_0 - t_2)\right].$$

As we provide that $t_1 \geq t_0+t_2+\bar{T}_4$ where $\bar{T}_4$ depends only on the model parameters ϵ_1, ϵ and Δ, then after a time $t_0 + t_2 + \bar{T}_4$ we have that

$$e \cdot \mathbf{x}(t) > e \cdot \left(\frac{1}{2}\epsilon\pi_1^*, \left(\frac{1}{2}+\Delta\right)\epsilon\pi_2^*, \left(\frac{1}{2}+2\Delta\right)\epsilon\pi_3^\star \left(\frac{1}{2}+3\Delta\right)\epsilon_1\beta^*\right), \tag{4.52}$$

Also, proven that if $t_0 \leq t \leq t_1$ then after a time $t_0 + \max\left[\bar{T}_1\bar{T}_1 + \bar{T}_2, \bar{T}_1 + \bar{T}_2 + \bar{T}_3\right]$ we know that

$$\begin{aligned}
\pi_1(t) &\leq \frac{1}{2}\epsilon\pi_1^*, \\
\pi_2(t) &\leq \left(\frac{1}{2}+\Delta\right)\epsilon\pi_2^*, \\
\pi_3(t) &\leq \left(\frac{1}{2}+2\Delta\right)\epsilon\pi_3^*, \\
\beta(t) &\leq \left(\frac{1}{2}+3\Delta\right)\epsilon_1\beta^*.
\end{aligned}$$

Hence

$$e \cdot x(t) \leq e \cdot \left(\frac{1}{2}\epsilon\pi_1^*, \left(\frac{1}{2}+\Delta\right)\epsilon\pi_2^*, \left(\frac{1}{2}+2\Delta\right)\epsilon\pi_3^*, \left(\frac{1}{2}+3\Delta\right)\epsilon_1\beta^*\right). \tag{4.53}$$

From equation (4.52), we have a contradiction if

$$t_1 \geq t_0 + \max\left[\bar{T}_1, \bar{T}_1 + \bar{T}_2, \bar{T}_1 + \bar{T}_2 + \bar{T}_3, t_2 + \bar{T}_4\right].$$

Therefore

$$t_1 < t_0 + \max\left[T_1, \bar{T}_1 + \bar{T}_2, \bar{T}_1 + \bar{T}_2 + \bar{T}_3, t_2 + \bar{T}_4\right].$$

The proof of Lemma 4.3.6 is now complete.

As a result, we have proven that π_1 decreases below $\frac{1}{2}\epsilon\pi_1^*$, then at least one $\pi_1(t)$ returns to this level after $T = \max\left[\bar{T}_1, \bar{T}_1 + \bar{T}_2, \bar{T}_1 + \bar{T}_2 + \bar{T}_3, t_2 + \bar{T}_4\right]$. This argument may be extended to any moment when $\pi_1(t)$ falls below $\frac{1}{2}\epsilon\pi_1^*$. As a result, if $\pi_1(t)$ falls below this level at time $\tilde{t}_0$ then for $t \in [\tilde{t}_0, \tilde{t}_0 + T]$

$$\frac{d\pi_1}{dt} \geq -\left(\mu + \delta_1\right)\pi_1.$$

Using integration over $[\tilde{t}_0, \tilde{t}_0 + T]$, we can derive that

$$\frac{d\pi_1}{dt} \geq \frac{1}{2}\epsilon\pi_1^* \exp\left[-\left(\mu + \delta_1\right)T\right],$$

where T is a fixed duration dependent only on $\epsilon, \epsilon_2, \Delta$ and the model parameters.

we got that $\pi_{1,\infty} > 0$, if the last term of $\frac{d\pi_1}{dt}$ is strictly positive. As a result, the argument given at the start of Theorem 4.3.5 indicates that the result of Theorem 4.3.5 is true. So, by reducing ϵ there exists a fixed lower bound $\epsilon > 0$ and $\eta > 0$ such that for all $t \geq \eta, \pi_1(t) \geq \epsilon, \pi_2(t) \geq \epsilon, \pi_3(t) \geq \epsilon$ and $\beta(t) \geq \epsilon$. The proof of Theorem 4.3.5 is finished.

In the next section, we going to show some numerical simulations for our analytical results for the models.

4.4 Simulations

Mathematical experiments were carried out using MATLAB to observe the dynamical system. Consequently, we attempt to numerically describe some of these theoretical results that showed the behaviour of HIV/AIDS with awareness programs indicating population death or epidemic, one of the important findings that we previously demonstrated if $R_0 \leq 1$ or $R_0 > 1$.

To create estimates of disease prevalence over a long time, we now employ the SOLVER(ode45) numerical ordinary differential equation package. As part of various simulations, we employed realistic parameter settings for the model (4.5)-(4.8) with two disease awareness programs function $\phi(\pi)$ (Misra et al. (2011), Samanta et al. (2013) and Greenhalgh et al. (2015)). In this simulation, we test two functional forms of awareness programs $\phi(\pi)$ that have been used in the previous chapters. The two functions are as follows

i. $\phi(\pi) = \left(1 - \frac{a\pi}{b+\pi}\right)$ where a and b are positive constants with $0 \leq a \leq 1$.

ii. $\phi(\pi) = e^{-m_0 n\pi}$ where m_0 is constant and n represents the number of the PWIDs population.

Early in this chapter, we discussed the various values of the parameters and some of them are estimated by Lewis (2000). However, we do not use all these

original estimates. So we set up the following parameter values as the Table 4.2 shows

Table 4.2: Estimates of parameter values for the model.

Parameters	Estimate Values	Parameters	Estimate Values
λ	246.22 /year	δ_3	0.1920/year
α	0.00601 per shared equipment	γ	0.90797
θ	0.0	μ	0.1333/year
τ	15.531/year	$\hat{\tau}$ =$\tau/\lambda\gamma$	0.0695/year
δ_1	4.6154/year	δ_2	0.2281/year

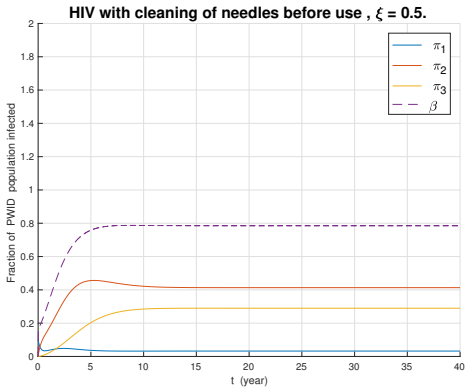
In our simulation, we use the realistic parameter that is shown in the Table 4.2 with varying values of the needle cleaning before using probability ξ according to $0 \leq \xi \leq 1$. As a result, we examined the dynamics of the model equations (4.5)- (4.8) by altering R_0 by selecting different values of ξ, so we picked $\xi = 0.5$ and $\xi = 0.9$. As initial starting values $\pi_1^*(0), \pi_2^*(0), \pi_3^*(0)$ and $\beta^*(0)$ were equal to $0.0, 0.0, 0.0$ and (0.1) in all cases. Now we are going to show three examples of the simulations

- **Example 1: The simulation without using the disease awareness program functions.**

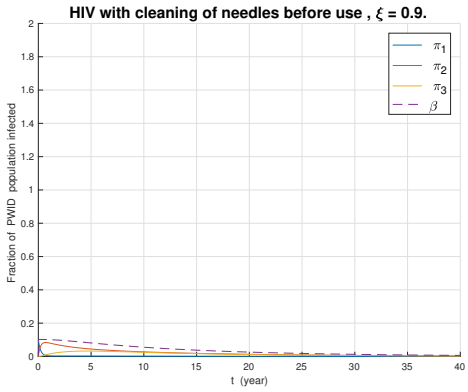
  Figure 4.1 shows the plot of two simulations without using the disease awareness program function over 40 years. we use the current set of parameter values shown in Table 4.2. So the sub-Figures 4.1a show the fraction of infected PWIDs and infected needles where the disease is present in both addicts and needles if $R_0 > 1$ when choosing $\xi = 0.5$. Then the equations model (4.5)-(4.8) have $\hat{\theta}$ = 0.5000/day giving $R_0 = 6.2179$.

  On the other hand, the sub-Figures 4.1b show the fraction of infected PWIDs and infected needles where the disease dies out

in both addicts and needles if $R_0 < 1$ when choosing $\xi = 0.9$. Then the equations model (4.5)- (4.8) have $\hat{\theta}$ = 0.9000/day giving $R_0 = 0.7305$.



(a) $\xi = 0.5$, then $R_0 > 1$.



(b) $\xi = 0.9$, then $R_0 < 1$.

Figure 4.1: The plots of simulations for the solution of model (4.5)- (4.8) without values of awareness program function where $n = 1000$.

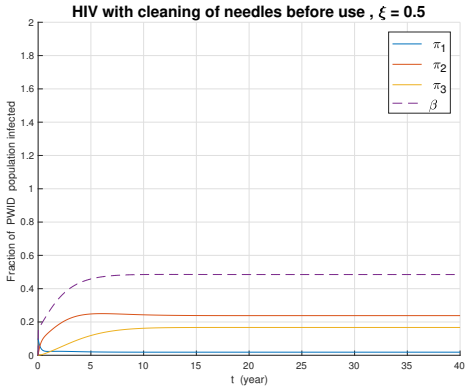
- **Example 2: The simulation with using the disease awareness program function $\phi(\pi) = e^{-m_0 n\pi}$.**

  The Figure 4.2 shows plots of six simulations with the disease awareness program over 40 years, and we use the function $\phi(\pi)$= $e^{-m_0 n\pi}$ (provided from Cui et al. (2008)) with choosing different values of m_0 where is constant and n represents the number of the PWIDs population. We picked $m_0 = 2.0/n, 5.0/n$ and $10.0/n$, and keep using the same current set of parameter values shown in table 4.2.

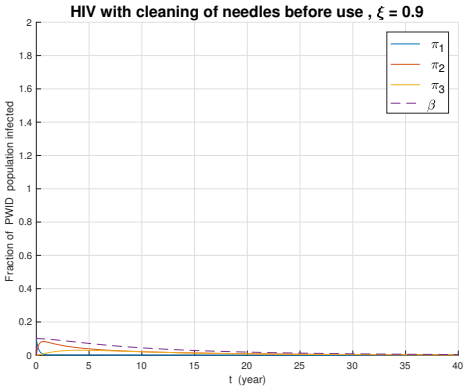
  The sub-Figures 4.2a, 4.2c and 4.2e with the choice $m_0 = 2.0, 5.0$ and 10 respectively, illustrate the fraction of infected PWIDs population who do not clean their needles before use with choosing $\xi = 0.5$ so that $R_0 > 1$, moreover the model equations (4.5)- (4.8) which have $\hat{\theta}$ = 0.5000/day giving $R_0 = 6.2179$.

  However, the sub-Figures 4.2b, 4.2d and 4.2f with the choice $m_0 = 2.0, 5.0$ and 10 respectively, illustrate the fraction of the infected

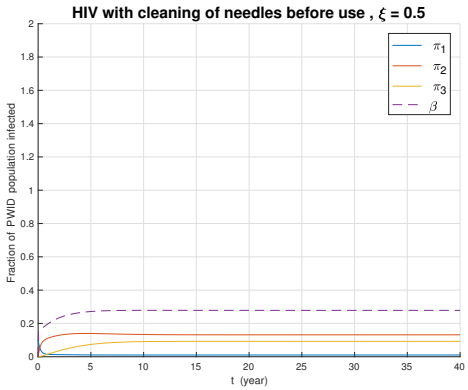
PWIDs population where the PWIDs often cleaned their needles successfully before use (modelled by choosing $\xi = 0.9$) then $R_0 < 1$, moreover the model equations (4.5)- (4.8) which have $\hat{\theta} = 0.9000$/day giving $R_0 = 0.7305$.



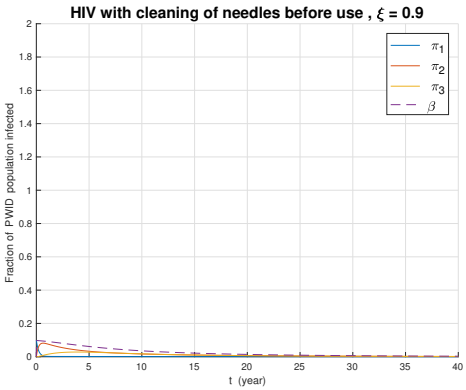
(a) With values of awareness program function parameters $m_0 = 2.0/n$.



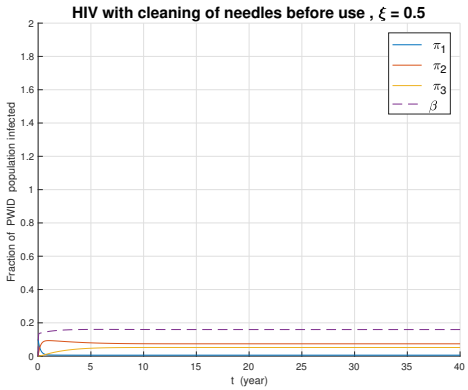
(b) With values of awareness program function parameters $m_0 = 2.0/n$.



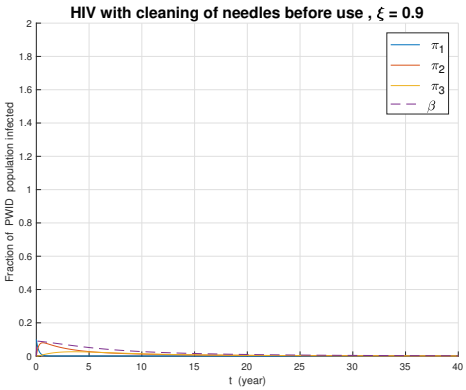
(c) With values of awareness program function parameters $m_0 = 5.0/n$.



(d) With values of awareness program function parameters $m_0 = 5.0/n$.



(e) With values of awareness program function parameters $m_0 = 10.0/n$.



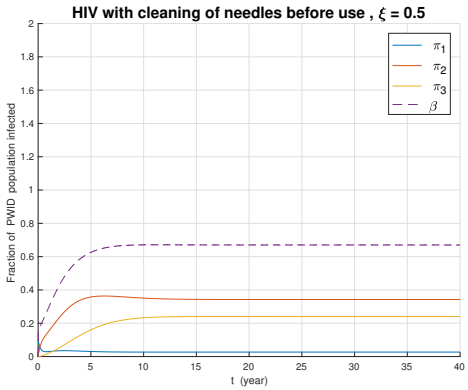
(f) With values of awareness program function parameters $m_0 = 10.0/n$.

Figure 4.2: The plots of simulations for the solution of model (4.5)- (4.8) with awareness program function $\phi(\pi) = e^{-m_0 n \pi}$, where $n = 1000$ and when $\xi = 0.5$ so $R_0 > 1$ and so $\xi = 0.9$ then $R_0 < 1$.

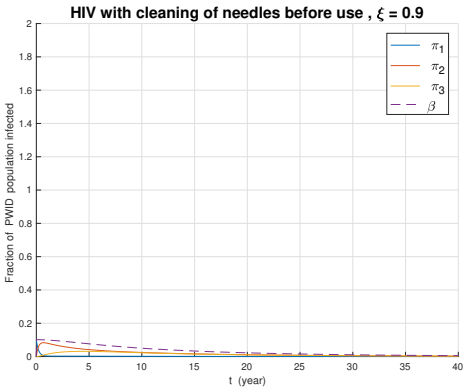
- **Example 3:The simulation with using the disease awareness program function $\phi(\pi)$ = ($1 - \frac{a\pi}{b+\pi}$).**

  The Figure 4.3 shows plots of six simulations with the disease awareness program over 40 years, and we use the function $\phi(\pi)$ = $\left(1 - \frac{a\pi}{b+\pi}\right)$ where a and b are positive constants with $0 \leq a \leq 1$ (provided from Dubey et al. (2016)), with different values of the constants a and b as shown in Figure 4.3 and using the same current set of parameters values shown in table 4.2.

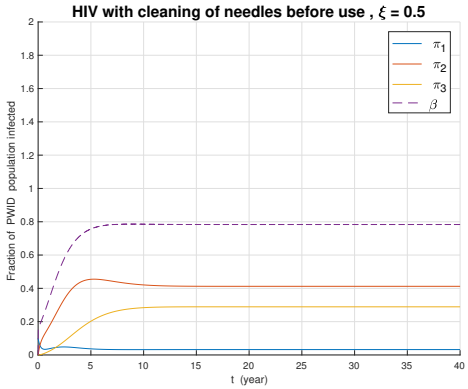
  The sub-Figures 4.3a, 4.3c and 4.3e with the same results as previously with $\xi = 0.5$ then $R_0 > 1$. Similarly, for the sub-figures 4.3b, 4.3d and 4.3f $\xi = 0.9$ then $R_0 < 1$.



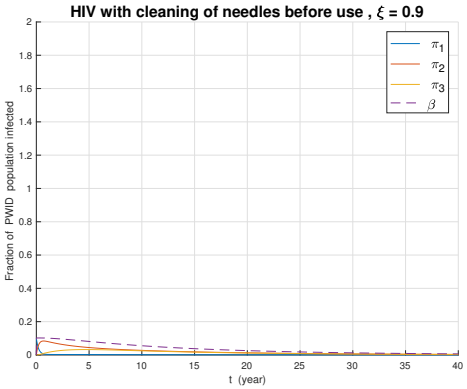
(a) With values of awareness program function parameters $a = 0.9, b = 1$.



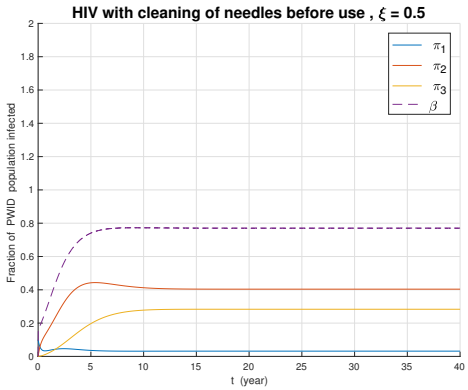
(b) With values of awareness program function parameters $a = 0.9, b = 1$.



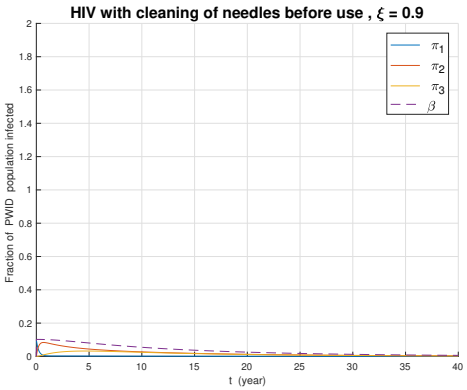
(c) With values of awareness program function parameters $a = 0.1, b = 10$.



(d) With values of awareness program function parameters $a = 0.1, b = 10$.



(e) With values of awareness program function parameters $a = 0.5, b = 5$.



(f) With values of awareness program function parameters $a = 0.5, b = 5$.

Figure 4.3: The plots of simulations for the solution of model (4.5)- (4.8) with awareness program function $\phi(\pi)$=1 $- \frac{a\pi}{b+\pi}$ and so $\xi = 0.5$ when $R_0 > 1$ and so $\xi = 0.9$ when $R_0 < 1$.

In our simulation, we can summarise two results by using the disease awareness program functions as shown in Figures 4.2and 4.3.

First, if the PWID population do not clean their needles before use with ($\xi = 0.5$),

and the disease is initially present $R_0 > 1$ after a long time the fraction of the infected PWID population will tend to the unique endemic equilibrium.

The second result is that if the PWIDs often cleaned their needles successfully before use with $\xi = 0.9$, then $R_0 < 1$ and the disease will die out after a long time in both PWIDs and needles and the fraction of the PWID population which is infected will approach to the disease-free equilibrium. We also observed the long-term endemic equilibrium level of disease prevalence for the system described by the model (4.5)- (4.8) is reduced when compared to the system with no disease awareness program functions in 4.2 and 4.3 as compared to the disease behaviour 4.1.

4.5 Conclusion

In this chapter, we have considered the effect of awareness programs in a three-stage infectivity model of HIV/AIDS. We developed the three-stage HIV/AIDS infection model studied by Lewis (2000) by applying awareness program functions. Then we derived the system of differential equations for the spread of HIV amongst PWIDs with disease awareness programs.

The model studied in this chapter is more realistic than the models studied in Chapters 2 and 3 because it takes account of the fact that the infectivity of HIV differs throughout the course of the infection. This substantially alters the analysis. It differs from the work of Lewis (2000)as an awareness program has been introduced. The value of R_0 is the same as Lewis (2000) and the results are qualitatively similar but the analysis is more complex.

We calculated an expression for the basic reproduction number R_0 that allowed us to figure out the analysis of the model, we have shown that for any given initial value condition in the region D=$[0, 1]^4$ in R^4, the system of the model has a unique non-negative solution that remains in D for all time, and also the conditions

required for persistence for the infected fractions of PWIDs and needles π_1, π_2, π_3 and β. In general, the disease is persistent when $R_0 > 1$ as shown in Theorem 4.3.5. Analytically, we determined the equilibrium solution where the disease dies out or persists in both PWIDs and needles for our model and evaluated its local and global stability.

We showed that if $R_0 < 1$ there is only the disease-free equilibrium where if $R_0 \leq 1$ disease free DFE equilibrium is locally asymptotically stable, as well as globally stable. Otherwise, the disease-free solution DFE is unstable if $R_0 > 1$. Also there is endemic equilibrium solution EE which is locally asymptotically stable if $R_0 < 1$. Finally, we performed some numerical simulations that showed the dynamic behaviour of this model graphically. The numerical simulations confirmed the analytical results for the models.This concludes the analysis of this model. We will develop a mathematical model for HIV/AIDS awareness programs with successful antiretroviral treatments and study the analytical behavior of the model in the next chapter.

Chapter 5

The Effect of Awareness Programs on the HIV/AIDS Models with Successful Antiretroviral Treatment

5.1 Introduction

Human immunodeficiency virus type 1 (HIV-1) can be managed and treated with highly active antiretroviral therapy (HAART). A treatment plan containing three or more antiretroviral medications is known as highly active antiretroviral therapy (HAART). Antiretroviral therapy (ART) or combined antiretroviral therapy (CART) are other terms for HAART. HAART aims to curb the development of HIV into other diseases such as AIDS-related and non-AIDS related cause deaths (such as reducing morbidity and mortality) and aims to prevent transmission to others such as sex partners, needle-sharing partners, mothers to infants, etc (Eggleton and Nagalli 2020).

In recent years, HIV/AIDS has no longer been viewed as a fatal condition due to the development of antiretroviral drugs, and antiretroviral therapy ART has dramatically reduced HIV/AIDS-related mortality and morbidity as shown by (Mocroft

et al. 2003). (Li and Wang 2014) and (Granich et al. 2009) used a mathematical model that shows how antiretroviral treatments (ART) such as HAART control or reduce the transmission of the HIV/AIDS virus.

In this chapter, we extend and develop the mathematical model of the spread of HIV amongst PWIDs with an awareness program that was analysed in Chapter 2. This was based on a model originally described by Kaplan (1989). We extended the model to include two groups within our PWID population: those PWIDs who are infected but unaware that they are infected and those PWIDs who are on successful HAART. First, we will describe the model and the assumptions that allow IDUs to move through the phases of HIV/AIDS infection. The set of governing equations is deduced using these assumptions. Then, before analysing the model mathematically, we develop a formula for the fundamental reproductive number R_0,and then perform simulations based on parameter values for our model to verify our theoretical results.

5.2 HIV/AIDS Models with Successful HAART

In this part, we modify the differential equation model introduced in Chapter 2 equations ((2.2) and (2.3)) where we analyse the effectiveness of the awareness program $\phi(\pi)$. We do this by considering two cases: one where the infected PWID has had successful HAART, and one where the PWID is unaware of their infection. We divide the infected PWIDs $\pi(t)$ with HIV/AIDS into two groups: $\pi_I(t)$ is the PWIDs who are infected but unaware of it and the second group $\pi_v(t)$ it is aware infected PWIDs who are on successful HAART.

We use the same biological parameters as described in Table 4.1 in Chapter 2 with the following changes to the definition of parameters λ_1, λ_2, μ_1, μ_2, δ, shown in Table 5.1

Table 5.1: Description of Parameters

Parameter	Definition
λ_1	Shooting gallery visiting rate for susceptible PWIDs and the PWIDs who are infected but unaware that they are infected.
λ_2	Shooting gallery visiting rate for aware infected PWIDs who are on successful HAART so that they are do not transmit HIV virus.
μ_1	Per capita rate at which PWIDs on successful HAART leave the sharing injecting population.
μ_2	Per capita rate at which infected but unaware PWIDs leave the sharing, injecting population (due to either ceasing sharing injection equipment, treatment for HIV/AIDS or death).
δ	Per capita rate at which PWIDs infected but unaware transfer to successful HAART.

Unaware infected PWIDs visit shooting galleries at rate λ_1 and aware infected PWIDs on HAART visit shooting galleries at rate λ_2, those PWIDs who are aware that they are infected inject at a lower rate. We assume that aware individuals reduce their shooting gallery visiting rate so that $\lambda_1 > \lambda_2$. As being on HAART will reduce the death rate we assume that $\mu_2 > \mu_1$.

5.2.1 Formulation of the Effect of Awareness Programs on HIV/AIDS Models with Successful HAART

We now show how we restructure the differential equation model for the spread of HIV/AIDS amongst PWIDs with an awareness program $\phi(\pi)$ that has been analysed in Chapter 2 (equations (2.2) and (2.3)). Let $\pi_I(t)$ denote the fraction of the susceptible PWIDs and the PWIDs infected but unaware of it at time t, $\pi_v(t)$ denote the fraction of the aware infected PWIDs and on successful HAART time t and $\beta(t)$ denote the fraction of needles infected at time t.

Assume that i is the number of infectious needles at time $t + \Delta t$, so we have $\beta = \dfrac{i}{m}$, $\pi_I = \dfrac{I}{n}$ and $\pi_V = \dfrac{V}{n}$ where n is the number of the PWIDs population and m

represents the number of shared needles. We know that $\gamma = \frac{n}{m}$ is the gallery ratio that represents the number of PWIDs per shared needles in the population, as given in Table 4.1. So we use a similar technique that has been used in Chapter 2 to derive a three-equation model. Two of these equations represent the infection of PWIDs, and the other one represents the infectious needle. The model can be described by the following differential equations:

The $\pi_I(t)$ equation:

The number of unaware and infected PWIDs at time $t + \Delta t$

= number of unaware and infected PWIDs at time t

+ number of new PWIDs who are infected in time $[t, t + \Delta t)$.

– the number of the unaware infected class who progress on to successful infected HAART treatment or leave the sharing, injecting population $[t, t + \Delta t)$.

Note that the number of new PWIDs who are infected in the time interval $[t, t + \Delta t)$ is the number of susceptible PWIDs who inject in $[t, t + \Delta t)$ $\Big(\lambda_1\phi(\pi_V(t))\Delta t + o(\Delta t)\Big)$ multiplied by the probability $(P_1 + P_3)(1 - \xi)\ \beta(t)$ that they inject with an infectious needle that has not been cleaned before use $(1-\xi)\beta(t)$ and the infection is transmitted $(P_1 + P_3)$. Hence

$$n\pi_I(t + \Delta t) = n\pi_I(t) + \Big(n - n\pi_I(t) - n\pi_V(t)\Big)\lambda_1\phi(\pi_V(t))(P_1 + P_3)(1 - \xi)\beta(t)\Delta t - (\mu_1 + \delta)\pi_I(t)\Delta t + o(\Delta t).$$

Next, subtracting both sides by $n\pi_I$, and then dividing by $n\Delta t$ and letting $\Delta t \to 0$ gives the following:

$$\frac{d\pi_I}{dt} = (1 - \pi_I - \pi_V)\lambda_1\phi(\pi_V)(P_1 + P_3)(1 - \xi)\beta - (\mu_1 + \delta)\pi_I. \tag{5.1}$$

The $\pi_V(t)$ equation:

Similarly, the number of $\pi_V(t)$- infected PWIDs who are on successful HAART at

time $t + \Delta t$

= the number of infected PWIDs on successful HAART at time t

+ the number of PWIDs who are infected but unaware and transfer to successful HAART in $[t, t + \Delta t)$

– the number of infected PWIDs on successful HAART who leave the sharing injecting population in $[t, t + \Delta t)$.

Thus

$$\frac{d\pi_V}{dt} = \delta\pi_I - \mu_2\pi_V. \tag{5.2}$$

The $\beta(t)$ equation :

Similarly, for the infectious needles $\beta(t)$, we assume that aware PWIDs on HAART interact with needles in the same way as susceptible PWIDs.

The number of infectious needles at time $t + \Delta t$.

= the number of infectious needles at time t not shared by PWIDs in $[t, t + \Delta t)$

+ the number of needles left infectious at time $t + \Delta t$ after being shared by unaware infected PWIDs in $[t, t + \Delta t)$

+ the number of needles left infectious at time $t + \Delta t$ after being shared by susceptible PWIDs in $[t, t + \Delta t)$

+ the number of needles left infectious at time $t + \Delta t$ after being shared by aware PWIDs on successful HAART in $[t, t + \Delta t)$.

Thus

$$\begin{aligned} i(t + \Delta t) = i\Big[1 - [\lambda_1 (1 - \pi_V) + \lambda_2\pi_V]\,\gamma\phi(\pi_V)\Delta t\Big] \\ + m\lambda_1\pi_I\phi(\pi_V)\gamma\Delta t\Big[(1 - \beta + \beta\xi)(1 - \phi_1) + \beta(1 - \xi)\,(1 - \theta_1)\Big] \\ + \lambda_1\gamma\,(1 - \pi_I - \pi_V)\,i\,(1 - P_1 - P_2)\,(1 - \xi)\phi(\pi_V)\Delta t. \\ + \lambda_2\gamma\pi_V i\,(1 - P_1 - P_2)\,(1 - \xi)\phi(\pi_V)\Delta t + o(\Delta t). \end{aligned}$$

Subtracting $i(t)$ from both sides, dividing by m and letting Δt go to zero we deduce

that

$$\begin{aligned}\frac{d\beta}{dt} = &-\left[\lambda_1\left(1-\pi_V\right)+\lambda_2\pi_V\right]\gamma\phi(\pi_V)\beta \\ &+\lambda_1\pi_I\phi(\pi_V)\gamma\left[\Big(1-\beta+\beta\xi\Big)(1-\phi_1)+\beta(1-\xi)\left(1-\theta_1\right)\right] \\ &+\lambda_1\gamma\left(1-\pi_I-\pi_V\right)\beta\left(1-P_1-P_2\right)(1-\xi)\phi(\pi_V) \\ &+\lambda_2\gamma\pi_V\phi(\pi_V)\beta\left(1-P_1-P_2\right)(1-\xi).\end{aligned} \tag{5.3}$$

Hence the system of differential equations which describe the model can be given as follows

$$\frac{d\pi_I}{dt} = (1-\pi_I-\pi_V)\lambda_1\phi(\pi_V)\nu\beta-(\mu_1+\delta)\pi_I. \tag{5.4}$$

$$\frac{d\pi_V}{dt} = \delta\pi_I-\mu_2\pi_V. \tag{5.5}$$

$$\frac{d\beta}{dt} = \phi(\pi_V)\pi_I(\bar{\sigma}-\bar{\tau}\beta)-\phi(\pi_V)\bar{\lambda}_2\gamma\pi_V\beta-\phi(\pi_V)\left(1-\pi_I-\pi_V\right)\bar{\rho}\beta. \tag{5.6}$$

We define the new composite parameters as the follows

$$\begin{aligned}\nu &= \lambda_1(P_1+P_3)(1-\xi), \\ \bar{\sigma} &= \lambda_1\gamma(1-\phi_1), \\ \bar{\tau} &= \lambda_1\gamma\Big[1-(1-\xi)(1-\theta_1)+(1-\xi)(1-\phi_1)\Big], \\ \bar{\rho} &= \lambda_1\gamma\left[1-(1-\xi)(1-P_1-P_2)\right], \\ \bar{\lambda}_2 &= \lambda_2\left[1-(1-\xi)(1-P_1-P_2)\right].\end{aligned} \tag{5.7}$$

We suppose that $\phi(\pi_V)$ is a monotonically decreasing function with $\phi(0)=1$. We previously minimized the model's dimensions in (5.4) and (5.6) by assuming that the needle equation (5.6) is at equilibrium. A similar method is employed in the analytical part of Chapter 2 that is based on models for HIV/AIDs among PWIDs, as explained by Liang et al. (2016) in models for HIV among PWIDs. Hence the system can be written as

$$\frac{d\pi_I}{dt} = \frac{\phi(\pi_V)\nu\pi_I\bar{\sigma}(1-\pi_I-\pi_V)}{\pi_I\bar{\tau}+\bar{\lambda}_2\gamma\pi_V+\bar{\rho}\left(1-\pi_I-\pi_V\right)}-(\mu_1+\delta)\pi_I, \tag{5.8}$$

$$\frac{d\pi_V}{dt} = \delta\pi_I-\mu_2\pi_V. \tag{5.9}$$

Each of these parameters ν, $\bar{\sigma}$, $\bar{\tau}$, $\bar{\rho}$ and $\bar{\lambda}_2$ are positive.

The differential equations in (5.4)- (5.6) and (5.8) -(5.9) describe the effect of Awareness Programs on HIV/AIDS models with successful HAART. Now the model has been formulated. In the next section, we shall look at the existence of the unique non-negative solution.

5.3 Existence of Unique Non-Negative Solution

In this section, we perform an analysis of the existence and uniqueness of a non-negative solution and then analyze whether any equilibrium solutions exist.

Liang et al. (2016) used the Picard–Lindelöf theory and the concept of Lipschitz continuous functions Choudhary (2011) to prove the existence of a unique non-negative solution, as discussed in earlier chapters (2,3 and 4) in this thesis. In this part, we are going to analyze the existence of the unique non-negative solution using the Picard–Lindelöf theory. The following theorem proves the existence of the unique non-negative solution.

Theorem 5.3.1. *For any given initial value* $\pi_I(t), \pi_V(t)) = (\pi_I(0), \pi_V(0)) \in [0,1] \times [0,1]$ *in the* R^2 *region, we assume that the function* ϕ *is Lipschitz continuous in* π_V *for* $0 \leq \pi_V \leq 1$. *Then the model equations* (5.8) *and* (5.9) *have a unique non negative solution* $\in [0,1] \times [0,1]$, *moreover they are determined by the following three cases*

i. $\pi_I(0) = 0$, $\pi_V(0) = 0$. *In this case* $\pi_I(t) = \pi_V(t) = 0$ *for all time.*

ii. $\pi_I(0) > 0$, $\pi_V(0) \geq 0$ *and* $1 \geq \pi_I(0) + \pi_V(0)$. *In this case* $\pi_I(t) > 0$, $\pi_V(t) > 0$ *and* $1 > \pi_I(t) + \pi_V(t)$ *for all time* $t > 0$.

iii. $\pi_I(0) = 0$, $\pi_V(0) > 0$ *and* $1 \geq \pi_I(0) + \pi_V(0)$. *In this case* $\pi_I(0) = 0$, $\pi_V(t) > 0$ *and* $1 > \pi_V(t)$ *for all time.*

Proof: To establish this theorem, we need to show first the right-hand sides of the equations (5.8) and (5.9) are Lipschitz continuous, and then we apply the

Picard–Lindelöf theory. We also must verify that for any $\mathbf{x} = (I_x, V_x) \in D$, $\mathbf{y} = (I_y, V_y) \in D$

$$\|f(\mathbf{x}) - f(\mathbf{y})\| \leq L\|\mathbf{x} - \mathbf{y}\|,$$

for some constant L.

Writing $I = \pi_I$ and $V = \pi_V$, the equations (5.8) and (5.9) can be written in the form

$$\frac{d\pi_I}{dt} = f_1(I, V),$$
$$\frac{d\pi_V}{dt} = f_2(I, V).$$

We divide the proof into two parts. Write $\mathbf{x}(I_x, V_x)$ and $\mathbf{y}(I_y, V_y)$. We shall show that

$$\left|f_1(I_x, V_x) - f_1(I_y, V_y)\right| \leq L_1\|x - y\|. \tag{5.10}$$

$$\left|f_2(I_x, V_x) - f_2(I_y, V_y)\right| \leq L_2\|x - y\|. \tag{5.11}$$

In the other words the Lipschitz constants for f_1 and f_2 are L_1 and L_2 respectively. First, let us look at part one

$$\left|f_1(I_x, V_x) - f_1(I_y, V_y)\right| \leq L_1\|x - y\|.$$

for some constant $L_1 \geq 0$, and for any $I_x \in [0, 1]$,$I_y \in [0, 1]$. Consider

$$\begin{aligned}
\left|f_1(I_x, V_x) - f_1(I_y, V_y)\right| &= \left|\left(\frac{\phi(V_x)\nu I_x\bar{\sigma}(1 - I_x - V_x)}{I_x\bar{\tau} + \bar{\lambda}_2\gamma V_x + \bar{\rho}\,(1 - I_x - V_x)} - (\mu_1 + \delta)I_x\right)\right. \\
&\quad \left. - \left(\frac{\phi(V_y)\nu I_y\bar{\sigma}(1 - I_y - V_y)}{I_y\bar{\tau} + \bar{\lambda}_2\gamma V_y + \bar{\rho}\left(1 - I_y - V_y\right)} - (\mu_1 + \delta)I_y\right)\right|. \\
&\leq \left|\left(\frac{\phi(V_x)\nu I_x\bar{\sigma}(1 - I_x - V_x)}{I_x\bar{\tau} + \bar{\lambda}_2\gamma V_x + \bar{\rho}\,(1 - I_x - V_x)}\right) - \left(\frac{\phi(V_y)\nu I_y\bar{\sigma}(1 - I_y - V_y)}{I_y\bar{\tau} + \bar{\lambda}_2\gamma V_y + \bar{\rho}\left(1 - I_y - V_y\right)}\right)\right| \\
&\quad + (\mu_1 + \delta)\left|I_x - I_y\right|.
\end{aligned} \tag{5.12}$$

By the triangle inequality, we have that

$$\begin{aligned}\left|f_1(I_x,V_x)-f_1(I_y,V_y)\right| \leq & \left|\left(\frac{\phi(V_x)\nu I_x\bar{\sigma}(1-I_x-V_x)}{I_x\bar{\tau}+\bar{\lambda}_2\gamma V_x+\bar{\rho}\,(1-I_x-V_x)}\right)-\left(\frac{\phi(V_y)\nu I_x\bar{\sigma}(1-I_x-V_x)}{I_x\bar{\tau}+\bar{\lambda}_2\gamma V_x+\bar{\rho}\,(1-I_x-V_x)}\right)\right| \\ & + \left|\left(\frac{\phi(V_y)\nu I_x\bar{\sigma}(1-I_x-V_x)}{I_x\bar{\tau}+\bar{\lambda}_2\gamma V_x+\bar{\rho}\,(1-I_x-V_x)}\right)-\left(\frac{\phi(V_y)\nu I_y\bar{\sigma}(1-I_y-V_x)}{I_y\bar{\tau}+\bar{\lambda}_2\gamma V_x+\bar{\rho}\left(1-I_y-V_x\right)}\right)\right| \\ & + \left|\left(\frac{\phi(V_y)\nu I_y\bar{\sigma}(1-I_y-V_x)}{I_y\bar{\tau}+\bar{\lambda}_2\gamma V_x+\bar{\rho}\left(1-I_y-V_x\right)}\right)-\left(\frac{\phi(V_y)\nu I_y\bar{\sigma}(1-I_y-V_y)}{I_y\bar{\tau}+\bar{\lambda}_2\gamma V_y+\bar{\rho}\left(1-I_y-V_y\right)}\right)\right| \\ & + (\mu_1+\delta)\left|I_x-I_y\right|.\end{aligned} \tag{5.13}$$

Once again, by the triangle inequality

$$\begin{aligned}\left|f_1(I_x,V_x)-f_1(I_y,V_y)\right| \leq & \left|\phi(V_x)-\phi(V_y)\right|\left|\frac{\nu\bar{\sigma}I_x(1-I_x-V_x)}{I_x\bar{\tau}+\bar{\lambda}_2\gamma V_x+\bar{\rho}\,(1-I_x-V_x)}\right| \\ & + \nu\bar{\sigma}\phi(V_y)\left|\frac{I_x(1-I_x-V_x)}{I_x\bar{\tau}+\bar{\lambda}_2\gamma V_x+\bar{\rho}\,(1-I_x-V_x)}\right. \\ & \qquad \left. - \frac{I_y(1-I_y-V_x)}{I_y\bar{\tau}+\bar{\lambda}_2\gamma V_x+\bar{\rho}(1-I_y-V_x)}\right| \\ & + \phi(V_y)\nu\bar{\sigma}I_y\left|\frac{(1-I_y-V_x)}{I_y\bar{\tau}+\bar{\lambda}_2\gamma V_x+\bar{\rho}(1-I_y-V_x)}\right. \\ & \qquad \left. - \frac{(1-I_y-V_y)}{I_y\bar{\tau}+\bar{\lambda}_2\gamma V_y+\bar{\rho}(1-I_y-V_y)}\right| \\ & + (\mu_1+\delta)\left|I_x-I_y\right|.\end{aligned} \tag{5.14}$$

Since ϕ is Lipschitz continuous on $[0,1]$ we have

$$\left|\phi(V_x)-\phi(V_y)\right| \leq L_{1a}\left|V_x-V_y\right|$$

for some Lipschitz constant L_{1a} and it is straightforward to show that

$$\begin{aligned}\left|f_1(I_x,V_x)-f_1(I_y,V_y)\right| \leq & L_{1a}\left|V_x-V_y\right|+\nu\bar{\sigma}L_{1b}\left|I_x-I_y\right|+\nu\bar{\sigma}L_{1c}\left|V_x-V_y\right| \\ & + (\mu_1+\delta)\left|I_x-I_y\right|.\end{aligned}$$

where $L_1 = L_{1a} + L_{1b} + L_{1c}$.

This completes the proof of inequality (5.10).

Using similar arguments, we prove the second part as well

$$\begin{aligned}
\left|f_2(I_x, V_x) - f_2(I_y, V_y)\right| &\leq L_2 \left|x - y\right|. \\
\left|f_2(I_x, V_x) - f_2(I_y, V_y)\right| &= \left|(\delta I_x - \mu_2 V_x) - \left(\delta I_y - \mu_2 V_y\right)\right|. \\
&\leq \left|(\delta I_x - \mu_2 V_x) - \left(\delta I_y - \mu_2 V_x\right)\right| \\
&\quad + \left|\left(\delta I_y - \mu_2 V_x\right) - \left(\delta I_y - \mu_2 V_y\right)\right|.
\end{aligned} \tag{5.15}$$

It follows that

$$\left|f_2(I_x, V_x) - f_2(I_y, V_y)\right| \leq \delta \left|I_x - I_y\right| + \mu_2 \left|V_x - V_y\right|. \tag{5.16}$$

This completes the proof of inequality (5.11)

To finish the proof, we look at the following conditions, We first suppose that $\pi_V(0) > 0$ for all $\pi_V \in [0, 1]$. So there exists $\epsilon > 0$ with $\phi(\pi_V) > \epsilon$ for all $\pi_V \in [0, 1]$. We consider three sets of initial conditions

i. $\pi_I(0) = 0$, $\pi_V(0) = 0$. In this case $\pi_I(t) = \pi_V(t) = 0$ for all time.

ii. $\pi_I(0) > 0$, $\pi_V(0) \geq 0$ and $1 \geq \pi_I(0) + \pi_V(0)$. In this case $\pi_I(t) > 0$, $\pi_V(t) > 0$ and $1 > \pi_I(t) + \pi_V(t)$ for all time $t > 0$.

iii. $\pi_I(0) = 0$, $\pi_V(0) > 0$ and $1 \geq \pi_I(0) + \pi_V(0)$. In this case $\pi_I(0) = 0$, $\pi_V(t) > 0$ and $1 > \pi_V(t)$ for all time $t > 0$.

We set $\psi = (1 - \pi_I - \pi_V)$, so we can rewrite (5.8) and (5.9) in the form

$$\begin{aligned}
\frac{d\psi}{dt} &= -\left(\frac{d\pi_I}{dt} + \frac{d\pi_V}{dt}\right) \\
&= -\left(\frac{(1 - \pi_I - \pi_V)\phi(\pi_V)\nu\pi_I\bar{\sigma}}{\pi_I\bar{\tau} + \bar{\lambda}_2\gamma\pi_V + \bar{\rho}(1 - \pi_I - \pi_V)}\right) + \mu_1\pi_I + \mu_2\pi_V.
\end{aligned} \tag{5.17}$$

We start with the first condition

i. $\pi_I(0) = 0$, $\pi_V(0) = 0$

Using the Picard–Lindelöf Theorem, we can see that $\pi_I = \pi_V = 0$ is a solution for all time. As long as Δt is greater than 0, the system has an unique local solution in $[0, \Delta t]$. For all ξ in $[0, \tau_e)$, let $[0, \tau_e)$ be the maximum interval where a solution exists with $\pi_I(\xi) = \pi_V(\xi) = 0$. It is necessary to have $\tau_e \geq \Delta t > 0$, so we assume that $\tau_e < \infty$

and $\pi_I(0) = \pi_V(0) = 0$ for all $t < \tau_e$. By using the Picard–Lindelöf Theorem the solution exists with $\pi_I(\xi) = \pi_V(\xi) = 0$ in $[0, \tau_e + \xi]$. This contradicts the definition of τ_e. Therefore, there is unique solution on $\pi_I(\xi) = \pi_V(\xi) = 0$ for all time $t \geq 0$.

ii. $\pi_I(0) > 0$, $\pi_V(0) \geq 0$ and $1 \geq \pi_I(0) + \pi_V(0)$.

Suppose that $(0, \tau_e)$ is the maximum interval where a solution exists and for

$$\Big\{ For \quad \xi \in (0, \tau_e) : \pi_I(\xi) > 0, \pi_V(\xi) > 0 \quad and \quad 1 > \pi_I(\xi) + \pi_V(\xi) \Big\}.$$

By the Picard–Lindelöf Theorem there exists $\Delta t > 0$ so that the equations (5.8) and (5.9) have a unique solution in $\xi \in [0, \Delta t]$. It is straightforward to show that if Δt is small enough $\pi_I(\Delta t) > 0$, $\pi_V(\Delta t) > 0$ and $\psi(\Delta t) > 0$.

We assume that $\tau_e < \infty$, and by integrating equation (5.8) we have that

$$\frac{d\pi_I}{dt} \geq -(\mu_1 + \delta)\pi_I,$$

so

$$\pi_I(\xi) \geq \pi_I(0) exp[-(\mu_1 + \delta)\xi].$$

Hence by letting $\xi \to \tau_e$

$$\lim_{\xi \to \tau_e} \pi_I(\xi) \geq \pi_I(0) exp[-(\mu_1 + \delta)\tau_e].$$

$$> 0$$

for $\xi \in (0, \tau_e]$.

Similarity, for equation (5.9) and (5.17) we have

$$\frac{d\pi_V}{dt} \geq -\mu_2\pi_V.$$

So

$$\pi_V \geq \pi_V(\Delta t)exp[-\mu_2(t-\Delta t)].$$

$$\pi_V(\tau_e) \geq \pi_V(\Delta t)exp[-\mu_2(\tau_e-\Delta t)].$$

$$> 0.$$

$$\begin{aligned}\frac{d\psi}{dt} &\geq \frac{-\psi\phi(\pi_V)\nu\pi_I\bar{\sigma}}{\pi_I\bar{\tau}+\bar{\lambda}_2\gamma\pi_V+\bar{\rho}(1-\pi_I+\pi_V)}.\\ &\geq \frac{-\epsilon\psi\nu\pi_I,[\Delta t,\tau_e]\bar{\sigma}}{min[\bar{\tau},\bar{\lambda}_2\gamma,\bar{\rho}]},\\ &= -k_1\psi.\end{aligned}$$

where $\pi_I,[\Delta t,\tau_e]$ denotes the strictly positive lower bound of π_I in $[\Delta t,\tau_e]$ and $k_1 > 0$. Hence

$$\psi(t) \geq \psi(\Delta t)exp[-k_1(t-\Delta t)].$$

Letting $t \to \tau_e$

$$\psi(\tau_e) > \psi(\Delta t)exp[-k_1(\tau_e-\Delta t)] > 0.$$

So we can extend the solution a little beyond τ_e. This contradicts the definition of τ_e so $\tau_e = \infty$.

iii. $\pi_I(0) = 0$, $\pi_V(0) > 0$ and $1 \geq \pi_I(0)+\pi_V(0)$. In this case it is clear that $\pi_I(t) = 0$ for all t, $\pi_V(t) = \pi_V(0)\ e^{-\mu_2 t}$ is a solution to equations (5.8) and (5.9) so by the Picard–Lindelöf Theorem it is unique solution there. This completes the proof of the theorem 5.3.1 in the case where $\phi(\pi_v) > 0$ for all $\pi_v \in [0,1]$.

Now suppose that there exists $\pi_V^* > \frac{\delta}{(\mu_2+\delta)}$ with $\phi(\pi_V) = 0$ for $\pi_V > \pi_V^*$. Then the above proof and theorem is not valid but the results and proof can be modified as follows:

The case (i) and (iii) are the same. For case (ii) we have two possibilities

a $\pi_V(0) \leq \pi_V^*$. Then it is straightforward to show
$\pi_I(t) \geq 0$, $\pi_V^* \geq \pi_V(t) \geq 0$ and $1 \geq \pi_I(t) + \pi_V(t)$ for all $t \geq 0$ and
$\pi_I(t) > 0$, $\pi_V^* > \pi_V(t) > 0$ and $1 > \pi_I(t) + \pi_V(t)$ for all $t > 0$.

b $\pi_V(0) > \pi_V^*$. Then it is straightforward to show that π_V must decrease to π_V^* in a finite time T^* and during this time π_I^* is also exponentially decreasing. After this finite time for $t \geq T^*$
$\pi_I(t) > 0$, $\pi_V^* > \pi_V(t) > 0$ and $1 > \pi_I(t) + \pi_V(t)$.

By this augment we have completed the proof of the existence of a unique non-negative solution. Next, we will calculate the basic reproduction number, which is an important factor in our model.

5.4 The Basic Reproductive Number R_0

The basic reproductive number R_0 plays an important role when studying the behavior of analytical mathematical models of disease. For our model we use a similar definition of the basic reproductive number R_0 that has been used in Chapter 2. The basic reproductive number R_0 is calculated by considering a single newly PWID who is infected with HIV virus entering a disease-free population, when all needles are clean. The infection can occur in two ways during a visit to the shooting gallery

- The infected PWIDs who are unaware and have not had successful HAART passes HIV virus to an uninfected needle.
- The newly infected needle passes the virus to a susceptible PWID
.

If we write $\bar{I} = n\pi_I$ to be the total number of unaware infectious PWIDs, $\bar{V} = n\pi_V$ to be the total number of aware infectious PWIDs, and $i = m\beta$ to be the total number

of infectious needles at time t then from equations (5.4) and (5.6), we have

$$\frac{d\bar{I}}{dt} = (n - \bar{I} - \bar{V})\phi(\pi_V)\nu\frac{i}{m} - (\mu_1 + \delta)\bar{I},$$
$$\frac{d\bar{V}}{dt} = \delta\bar{I} - \mu_2\bar{V},$$

and

$$\frac{di}{dt} = \frac{m}{n}\phi(\pi_V)\bar{I}(\bar{\sigma} - \bar{\tau}\beta) - \bar{\lambda}_2\bar{V}\phi(\pi_V)i - \phi(\pi_V)\left(\frac{n - \bar{I} - \bar{V}}{n}\right)\bar{\rho}i.$$

Linearising about the disease-free equilibrium we have

$$\frac{d\bar{I}}{dt} = n\nu\frac{i}{m} - (\mu_1 + \delta)\bar{I},$$
$$\frac{d\bar{V}}{dt} = \delta\bar{I} - \mu_2\bar{V},$$

and

$$\frac{di}{dt} = \frac{m}{n}\bar{\sigma}\bar{I} - \bar{\rho}i.$$

To calculate R_0 we proceed as follows: Each unaware newly infected PWIDs remains in the sharing, injecting population for an average time $\frac{1}{\mu_1 + \delta}$ time units. During that time he or she visits the shooting galleries at rate λ_1 and they infect $\frac{\bar{\sigma}}{\gamma(\mu_1 + \delta)}$ new needles during the time that they are infected. Once the needle is infected with HIV/AIDs it remains infected for time $\frac{1}{\bar{\rho}}$, and during that time it infects PWIDs at an average rate $\frac{n\nu}{m}$, so altogether it infects $\frac{n\nu}{m\bar{\rho}}$. Aware infected PWIDs (who are on HAART) do not infect any needles. So the basic reproduction number can be written as

$$R_0 = \frac{\bar{\sigma}}{\gamma(\mu_1 + \delta)} \cdot \frac{n\nu}{m\bar{\rho}} = \frac{\bar{\sigma}\nu}{(\mu_1 + \delta)\bar{\rho}}. \tag{5.18}$$

This concludes our analysis of the basic reproduction number R_0. In the next section, we are going to show the behaviour of our model analytically.

5.5 Analysis of The Model

We study the behaviour of our transmission model in this section, concentrating on the key of biological parameter R_0 since that will show whether the HIV/AIDS disease persists or whether it disappears. In order to determine the nature of any equilibrium solution, We shall prove that there are two types of equilibrium solutions: a zero solution (disease-free equilibrium) and unique non-zero (endemic equilibrium). Then we will perform stability analysis, and furthermore, we will also do numerical simulations of our analytical comes system to verify the implementation of disease awareness programs is indeed effective. Next we are going to perform a detailed analysis to investigate the existence of equilibrium.

5.5.1 Existence of equilibrium

In this section, we are going to explore the possibility of the existence of an equilibrium for our model. We shall show the model has two non-negative equilibria, the first one being the disease-free equilibrium DFE $E_0 = (\pi_{I0}, \pi_{V0}) = (0, 0)$ and the second one being the endemic equilibrium EE $E_1 = (\pi_I^*, \pi_V^*) > 0$ in $(0, 1]\times(0, 1]$. This is shown in the following:

Theorem 5.5.1. *The system* (5.8) *and* (5.9) *has a unique disease-free equilibrium solution where the disease dies out in bothPWIDs and needles if* $R_0 \leq 1$, *whereas if* $R_0 > 1$ *then there is a unique endemic equilibrium where the disease is present.*

Proof: Suppose that ϕ is strictly monotone decreasing in π_V. From the equilibrium solution of (5.8) and (5.9) one solution is clearly $\pi_I^* = \pi_V^* = 0$.
For a non-zero solution

$$\pi_I^* = \frac{\mu_2}{\delta}\pi_V^*, \tag{5.19}$$

$$\text{and} \qquad \frac{1}{\pi_V^*} = 1 + \frac{\mu_2}{\delta} + \frac{(\mu_1+\delta)\Big[\frac{\mu_2}{\delta}\bar{\tau} + \bar{\lambda}_2\gamma\Big]}{\nu\bar{\sigma}\phi(\pi_V^*) - (\mu_1+\delta)\bar{\rho}}. \tag{5.20}$$

Re-arranging (5.20) we deduce that

$$\frac{1}{\pi_V^*} = 1 + \frac{\mu_2}{\delta} + \frac{1}{\frac{\nu\bar{\sigma}}{(\mu_1+\delta)\tau^*}\phi(\pi_V^*) - \frac{\bar{\rho}}{\tau^*}}, \tag{5.21}$$

where $\tau^* = \frac{\mu_2}{\delta}\bar{\tau} + \bar{\lambda}_2\gamma$.

Define

$$g_1(\pi_V^*) = \frac{1}{\pi_V^*}, \qquad \textit{and} \tag{5.22}$$

$$g_2(\pi_V^*) = 1 + \frac{\mu_2}{\delta} + \frac{1}{\frac{\nu\bar{\sigma}}{(\mu_1+\delta)\tau^*}\phi(\pi_V^*) - \frac{\bar{\rho}}{\tau^*}}. \tag{5.23}$$

By using the reproduction number equation

$$R_0 = \frac{\bar{\sigma}\nu}{(\mu_1+\delta)\bar{\rho}},$$

we take into account several situations.

(I) Suppose that $R_0 = \frac{\bar{\sigma}\nu}{(\mu_1+\delta)\bar{\rho}} < 1$. In this case, we have that from the equilibrium equation $\pi_I^* = \frac{\mu_2}{\delta}\pi_V^*$,

$$\begin{aligned}
\pi_I^* + \pi_V^* &< 1,\\
\pi_V^*\Big(1 + \frac{\mu_2}{\delta}\Big) &< 1,\\
\pi_V^* &< \frac{1}{1 + \frac{\mu_2}{\delta}}.
\end{aligned}$$

Thus $g_1(\pi_V^*) = \frac{1}{\pi_V^*} > 1 + \frac{\mu_2}{\delta}$. We know that $\phi(\pi_V^*)$ is strictly monotone decreasing in π_V^*. Therefore, the the denominator of the equation $g_2(\pi_V^*)$ in (5.23) gives

$$\frac{\nu\bar{\sigma}}{(\mu_1+\delta)\tau^*}\phi(\pi_V^*) - \frac{\bar{\rho}}{\tau^*} \le \frac{\nu\bar{\sigma}}{(\mu_1+\delta)\tau^*} - \frac{\bar{\rho}}{\tau^*} < 0, \qquad \forall \pi_V,$$

so we have $g_2(\pi_V^*) < 1 + \frac{\mu_2}{\delta}$.

Hence $g_2(\pi_V^*) < 1 + \frac{\mu_2}{\delta}$ and $g_1(\pi_V^*) > 1 + \frac{\mu_2}{\delta}$for $\pi_V^* \in \Big(0, \delta/(\delta + \mu_2)\Big)$. Therefore, there is no feasible solution with $R_0 < 1$.

(II) If $R_0 = \dfrac{\bar{\sigma}\nu}{(\mu_1 + \delta)\bar{\rho}} = 1$, this is similar to we had at the first case

$$\frac{\nu\bar{\sigma}}{(\mu_1 + \delta)\tau^*}\phi(\pi_V^*) - \frac{\bar{\rho}}{\tau^*} \leq \frac{\nu\bar{\sigma}}{(\mu_1 + \delta)\tau^*} - \frac{\bar{\rho}}{\tau^*} = 0, \qquad \forall \pi_V^*.$$

Hence $g_2(\pi_V^*) < 1 + \dfrac{\mu_2}{\delta}$ or $g_2(\pi_V^*) = \infty$ and $g_1(\pi_V^*) > 1 + \dfrac{\mu_2}{\delta}$ for $\pi_V^* \in \Big(0, \delta/(\delta + \mu_2)\Big)$. In this case also, there is no feasible solution with $R_0 = 1$.

(III) If $R_0 = \dfrac{\bar{\sigma}\nu}{(\mu_1 + \delta)\bar{\rho}} > 1$, since $\phi(\pi_V^*)$ is strictly monotonically decreasing, we consider the equation given by

$$\phi(\pi_V^*) = \frac{\bar{\rho}(\mu_1 + \delta)}{\nu\bar{\sigma}} < 1. \tag{5.24}$$

Our considerations are based on three cases.

(a) If $\phi\left(\dfrac{\delta}{\delta + \mu_2}\right) > \dfrac{\bar{\rho}(\mu_1 + \delta)}{\nu\bar{\sigma}}$, the equation (5.24) has no roots in $\left(0, \dfrac{\delta}{\delta + \mu_2}\right)$. In this case as $\pi_V \to 0$, then $g_1(\pi_V) \to \infty$ and

$$g_2(\pi_V^*) \to 1 + \frac{\mu_2}{\delta} + \frac{1}{\dfrac{\nu\bar{\sigma}}{(\mu_1 + \delta)\tau^*} - \dfrac{\bar{\rho}}{\tau^*}} > 1 + \frac{\mu_2}{\delta}.$$

At $\pi_V^* = \dfrac{\delta}{\delta + \mu_2}$, then we have $g_1(\pi_V^*) = 1 + \dfrac{\mu_2}{\delta}$. Therefore

$$g_2(\pi_V^*) = 1 + \frac{\mu_2}{\delta} + \frac{1}{\dfrac{\nu\bar{\sigma}}{(\mu_1 + \delta)\tau^*}\phi\left(\dfrac{\delta}{\delta + \mu_2}\right) - \dfrac{\bar{\rho}}{\tau^*}} > 1 + \frac{\mu_2}{\delta},$$

since

$$\frac{\nu\bar{\sigma}}{(\mu_1 + \delta)\tau^*}\phi\left(\frac{\delta}{\delta + \mu_2}\right) > \frac{\bar{\rho}}{\tau^*}.$$

As a result, the equation (5.21) has a non-zero root π_V^* in $\left(0, \dfrac{\delta}{\delta + \mu_2}\right)$. In addition, because $g_1(\pi_V^*)$ is strictly monotone decreasing in π_V^* and $g_2(\pi_V^*)$ is strictly monotone increasing in π_V^*, this root is unique in $\left(0, \dfrac{\delta}{\delta + \mu_2}\right)$.

(b) If $\phi\left(\frac{\delta}{\delta+\mu_2}\right) = \frac{\bar{\rho}(\mu_1+\delta)}{\nu\bar{\sigma}}$, the equation (5.24) has a unique root $\pi_V^* = \frac{\delta}{\delta+\mu_2}$. Again, we use the same argument as in Case (a), so in the same way as $\pi_V^* \to 0$, then we have that $g_1(\pi_V^*) \to \infty$ and

$$g_2(\pi_V^*) = 1 + \frac{\mu_2}{\delta} + \frac{1}{\frac{\nu\bar{\sigma}}{(\mu_1+\delta)\tau^*} - \frac{\bar{\rho}}{\tau^*}} < \infty.$$

because $R_0 = \frac{\bar{\sigma}\nu}{(\mu_1+\delta)\bar{\rho}} > 1$, and

$$\frac{dg(\pi_V)}{d\pi_V} = \frac{-\phi'(\pi_V)\frac{\nu\bar{\sigma}}{(\mu_1+\delta)\tau^*}}{\left(\frac{\nu\bar{\sigma}}{(\mu_1+\delta)\tau^*}\phi(\pi_V) - \frac{\bar{\rho}}{\tau^*}\right)^2} > 0.$$

So the right hand side of (5.21) is increasing.

Again at $\pi_V^* = \frac{1}{1+\frac{\mu_2}{\delta}}$, then we have that $g_1(\pi_V^*) = 1 + \frac{\mu_2}{\delta}$. In a similar way,

$$g_2\left(\frac{\delta}{\delta+\mu_2}\right) = 1 + \frac{\mu_2}{\delta} + \frac{1}{\frac{\nu\bar{\sigma}}{(\mu_1+\delta)\tau^*}\phi\left(\frac{\delta}{\delta+\mu_2}\right) - \frac{\bar{\rho}}{\tau^*}},$$

and therefore because

$$\frac{\nu\bar{\sigma}}{(\mu_1+\delta)\tau^*}\phi\left(\frac{\delta}{\delta+\mu_2}\right) = \frac{\bar{\rho}}{\tau^*},$$

$$g_2\left(\frac{\delta}{\delta+\mu_2}\right) = 1 + \frac{\mu_2}{\delta} + \frac{1}{\frac{\bar{\rho}}{\tau^*} - \frac{\bar{\rho}}{\tau^*}} = \infty,$$

so $g_1(\pi_V^*) < g_2(\pi_V^*)$. It follows then the equation (5.21) has a unique root π_V^* in $\left(0, \frac{\delta}{\delta+\mu_2}\right)$, and similarly to case (a), this root is unique in $\left[0, \frac{\delta}{\delta+\mu_2}\right]$.

(c) If $\phi\left(\frac{\delta}{\delta+\mu_2}\right) < \frac{\bar{\rho}(\mu_1+\delta)}{\nu\bar{\sigma}}$, then we know that

(i) $\phi(0) = 1 > \frac{\bar{\rho}(\mu_1+\delta)}{\nu\bar{\sigma}}$ and,

(ii) $\phi\left(\frac{\delta}{\delta+\mu_2}\right) < \frac{\bar{\rho}(\mu_1+\delta)}{\nu\bar{\sigma}}$.

As result, the equation (5.24) has a unique root π_V^{**} in $\left(0, \frac{\delta}{\delta+\mu_2}\right)$. Similarly, in this case as $\pi_V \to 0$, we have $g_1(\pi_V) \to \infty$ and

$$g_2(\pi_V) \to 1 + \frac{\mu_2}{\delta} + \frac{1}{\frac{\nu\bar{\sigma}}{(\mu_1+\delta)\tau^*} - \frac{\bar{\rho}}{\tau^*}} < \infty.$$

As $\pi_V \to \pi_V^{**}$ then $g_1(\pi_V^{**}) \to \frac{1}{\pi_V^{**}} < \infty$ and $g_2(\pi_V^{**}) \to \infty$. As a result, the equation (5.21) has a root in $(0, \pi_V^{**})$, and uniqueness follows as before.

At $\pi_V = \pi_V^{**}$, $g_1(\pi_V^{**}) < \infty$ and $g_2(\pi_V^{**}) = \infty$. For $\pi_V \in \left(\pi_V^{**}, \frac{\delta}{\delta+\mu_2}\right)$ we have that $g_1(\pi_V) > 1 + \frac{\mu_2}{\delta}$ and $g_2(\pi_V) < 1 + \frac{\mu_2}{\delta}$. So there are no roots of the equation (5.21) in $\left[\pi_V^{**}, \frac{\delta}{\delta+\mu_2}\right]$. Therefore the equation (5.21) has a unique root in $\left(0, \frac{\delta}{\delta+\mu_2}\right)$.

The proof of Theorem (5.5.1) is now completed. In the next section, we are going to show the local stability analysis of the equilibria.

5.5.2 Local Stability Analysis of Equilibrium

To do the local stability analysis we use the Routh–Hurwitz criterion (May (2001) and DeJesus and Kaufman (1987)). We examine the stability of the system using the coefficients of polynomial in the characteristic equation. It is the technique used to investigate the local stability of the equilibrium, as done in previous chapters. We are going to look first at the disease-free equilibrium DFE and then the endemic equilibrium points EE.

Theorem 5.5.2. *The disease-free equilibrium of the system of equations* (5.8) *and* (5.9) *is locally asymptotically stable if* $R_0 < 1$ *and unstable if* $R_0 > 1$. *If* $R_0 = 1$ *the disease-free equilibrium is neutrally stable. If* $R_0 > 1$ *there is a unique endemic equilibrium which is locally asymptotically stable.*

Proof. We assume that $\phi(\pi)$ is a differentiable function. By linearizing the system of equations (5.8) and (5.9), and using the variational matrix method around the equilibrium point we can identify the local stability.

We write equations (5.8) and (5.9) as

$$f_1(\pi_I, \pi_V) = \frac{\phi(\pi_V)\nu\pi_I\bar{\sigma}(1-\pi_I-\pi_V)}{\pi_I\bar{\tau}+\bar{\lambda}_2\gamma\pi_V+\bar{\rho}\,(1-\pi_I-\pi_v)} - (\mu_1+\delta)\pi_I = 0. \tag{5.25}$$

$$f_2(\pi_I, \pi_V) = \delta\pi_I - \mu_2\pi_V = 0. \tag{5.26}$$

We define the matrix representation of this system as follows This system can be represented in matrix form as $\frac{d\boldsymbol{y}}{dt} = \boldsymbol{J}$, where $\boldsymbol{y}^T = (\pi_I, \pi_V)$. Our Jacobian matrix is

$$\boldsymbol{J}_|(\pi_I^*, \pi_V^*) = \begin{bmatrix} \frac{\partial f_1}{\partial \pi_I} & \frac{\partial f_1}{\partial \pi_V} \\ \frac{\partial f_2}{\partial \pi_I} & \frac{\partial f_2}{\partial \pi_V} \end{bmatrix} = \begin{pmatrix} A-(\mu_1+\delta) & B \\ \delta & -\mu_2 \end{pmatrix}. \tag{5.27}$$

Here

$$A = \frac{\Big(1-2\pi_I^*-\pi_V^*\Big)\phi(\pi_V^*)\nu\bar{\sigma}\Big[\pi_I^*\bar{\tau}+\bar{\lambda}_2\gamma\pi_V^*+\bar{\rho}(1-\pi_I^*-\pi_V^*)\Big]}{\Big(\pi_I^*\bar{\tau}+\bar{\lambda}_2\gamma\pi_V^*+\bar{\rho}(1-\pi_I^*-\pi_V^*)\Big)^2} - \frac{\Big(\bar{\tau}-\bar{\rho}\Big)\Big[(1-\pi_I^*-\pi_V^*)\phi(\pi_V^*)\nu\pi_I^*\bar{\sigma}\Big]}{\Big(\pi_I^*\bar{\tau}+\bar{\lambda}_2\gamma\pi_V^*+\bar{\rho}(1-\pi_I^*-\pi_V^*)\Big)^2},$$

and

$$B = \frac{\phi(\pi_V^*)\pi_I^*\nu\bar{\sigma}\Big[-\Big(\pi_I^*\bar{\tau}+\bar{\lambda}_2\gamma\pi_V^*+\bar{\rho}(1-\pi_I^*-\pi_V^*)\Big)-(\bar{\lambda}_2\gamma-\bar{\rho})(1-\pi_I^*-\pi_V^*)\Big]}{\Big(\pi_I^*\bar{\tau}+\bar{\lambda}_2\gamma\pi_V^*+\bar{\rho}(1-\pi_I^*-\pi_V^*)\Big)^2} + \frac{(1-\pi_I^*-\pi_V^*)\phi'(\pi_V^*)\pi_I\nu\bar{\sigma}\Big(\pi_I^*\bar{\tau}+\bar{\lambda}_2\gamma\pi_V^*+\bar{\rho}(1-\pi_I^*-\pi_V^*)\Big)}{\Big(\pi_I^*\bar{\tau}+\bar{\lambda}_2\gamma\pi_V^*+\bar{\rho}(1-\pi_I^*-\pi_V^*)\Big)^2}. \tag{5.28}$$

5.5.2.1 The Disease-Free Equilibrium.

At the disease-free equilibrium DFE $\boldsymbol{E}_1 = (\pi_I^*, \pi_V^*) = (0, 0)$.

$$det(\boldsymbol{J}_1 - \omega I) = 0,$$

In other words

$$det\boldsymbol{J}_1 = \begin{pmatrix} \frac{\nu\bar{\sigma}\bar{\rho}}{\bar{\rho}^2} - (\mu_1 + \delta) - \omega & 0 \\ \delta & -\mu_2 - \omega \end{pmatrix} = 0. \tag{5.29}$$

We define the eigenvalues ω of the Jacobian matrix $\boldsymbol{J}_1$ as the roots of the characteristic equation

$$\left(\omega + \mu_1 + \delta - \frac{\nu\bar{\sigma}}{\bar{\rho}}\right)\left(\omega + \mu_2\right) = 0$$

with roots $\omega_1 = -(\mu_1 + \delta) + \frac{\nu\bar{\sigma}}{\bar{\rho}}$ and $\omega_2 = -\mu_2$. ω_2 is clearly always negative. ω_1 is positive if $R_0 > 1$, zero if $R_0 = 1$ and negative if $R_0 < 1$.

As a result, the local asymptotic stability of the DFE equilibrium point depends on the value of the basic reproduction number $R_0 = \frac{\bar{\sigma}\nu}{(\mu_1 + \delta)\bar{\rho}}$. Hence there are three possible stability scenarios.

- If $R_0 = \frac{\bar{\sigma}\nu}{(\mu_1 + \delta)\bar{\rho}} < 1$, then the DFE is locally asymptotically stable.
- If $R_0 = \frac{\bar{\sigma}\nu}{(\mu_1 + \delta)\bar{\rho}} = 1$, then the DFE is neutrally stable.
- If $R_0 = \frac{\bar{\sigma}\nu}{(\mu_1 + \delta)\bar{\rho}} > 1$, then the DFE is unstable.

5.5.2.2 The Endemic equilibrium.

Similarly, we use a similar argument to examine local stability at the endemic equilibrium point as we used to investigate the disease-free equilibrium.

Recall the Jacobian matrix around the endemic equilibrium point EE. $\boldsymbol{E}_2 = (\pi_I^*, \pi_V^*)$ of the above system then the Jacobian matrix $\boldsymbol{J}_2(\pi_I^*, \pi_V^*)$ of system corresponding to $\boldsymbol{E}_2$ is obtained as

$$\mathbf{J}_2|_{(\pi_I^*,\pi_V^*)} = \begin{pmatrix} A-(\mu_1+\delta)-\omega & B \\ \\ \delta & -\mu_2-\omega \end{pmatrix} \tag{5.30}$$

For $\boldsymbol{J}_2$ the characteristic equation is

$$\omega^2 + a\omega + b = 0, \quad \text{and}$$

where $a = \mu_1 + \mu_2 + \delta - A$ and $b = \mu_2(\mu_1+\delta) - A\mu_2 - \delta B$.

Using the equilibrium equations, we now have

$$\begin{aligned} A-(\mu_1+\delta) &= \frac{-\phi(\pi_V^*)\pi_I^*\nu\bar{\sigma}}{\Big(\pi_I\bar{\tau}+\bar{\lambda}_2\gamma\pi_V^*+\bar{\rho}(1-\pi_I^*-\pi_V^*)\Big)^2} - \frac{\Big(\bar{\tau}-\bar{\rho}\Big)(1-\pi_I^*-\pi_V^*)\phi(\pi_V^*)\nu\pi_I^*\bar{\sigma}}{\Big(\pi_I^*\bar{\tau}+\bar{\lambda}_2\gamma\pi_V^*+\bar{\rho}(1-\pi_I^*-\pi_V^*)\Big)^2}. \\ &= \frac{-\phi(\pi_V^*)\pi_I^*\nu\bar{\sigma}\Big[\pi_I^*\bar{\tau}+\bar{\lambda}_2\gamma\pi_V^*+\bar{\tau}(1-\pi_I^*-\pi_V^*)\Big]}{\Big(\pi_I^*\bar{\tau}+\bar{\lambda}_2\gamma\pi_V^*+\bar{\rho}(1-\pi_I^*-\pi_V^*)\Big)^2} < 0. \end{aligned}$$

Note that $\phi'(\pi_V^*) \leq 0$ since $\phi(\pi_V)$ is monotone decreasing. So

$$B \leq -\phi(\pi_V^*)\pi_I^*\nu\bar{\sigma}\frac{\Big[\pi_I^*\bar{\tau}+\bar{\lambda}_2\gamma(1-\pi_I^*)\Big]}{\Big[\pi_I^*\bar{\tau}+\bar{\lambda}_2\gamma\pi_V^*+\bar{\rho}(1-\pi_I^*-\pi_V^*\Big]^2} < 0.$$

So we have

$$\begin{aligned} a &= \mu_1+\mu_2+\delta-A > 0, \quad \text{and} \\ b &= \mu_2\Big((\mu_1+\delta)-A\Big) - \delta B > 0. \end{aligned}$$

Since $a > 0$ and $b > 0$ satisfy the Routh-Hurwitz conditions, the endemic equilibrium EE is locally asymptotically stable if $R_0 > 1$. This completes the proof of the Theorem 5.5.2.

5.5.3 Global Stability of Equilibrium

In this section we are going to show the global stability of disease free equilibrium DFE and discuss the global stability of the endemic equilibrium.

5.5.3.1 The Disease-Free Equilibrium .

Theorem 5.5.3. *If $R_0 \leq 1$, then the disease-free solution for the system* (5.8) *and* (5.9) *is globally stable, and HIV/AIDS will be eliminated from all PWIDs, as well as needles and syringes.*

Proof: To prove this theorem we use a mathematical technique. First, we pick the equation (5.8)

$$\frac{d\pi_I}{dt} = \frac{\phi(\pi_V)\nu\bar{\sigma}(1-\pi_I-\pi_V)\pi_I}{\pi_I\bar{\tau}+\bar{\lambda}_2\gamma\pi_V+\bar{\rho}\,(1-\pi_I-\pi_V)} - (\mu_1+\delta)\pi_I,$$

We are going to show that $\pi_I(t) \to 0$ as $t \to \infty$. We have that $\pi_I(0) = 0$ implies that $\pi_I(t) = 0$ for all time. So we can suppose that $\pi_I(0) > 0$ and by the results of the existence and uniqueness theorem $\pi_I(0) > 0$ for all t. We can rewrite the equation (5.8) as

$$\frac{1}{\pi_I}\frac{d\pi_I}{dt} = g(\pi_I), \tag{5.31}$$

where

$$\begin{aligned} g_1(\pi_I) &= \frac{\phi(\pi_V)\nu\bar{\sigma}}{\dfrac{\pi_I\bar{\tau}}{(1-\pi_I-\pi_V)}+\dfrac{\bar{\lambda}_2\gamma\pi_V}{(1-\pi_I-\pi_V)}+\bar{\rho}} - (\mu_1+\delta), \\ &\leq \frac{\nu\bar{\sigma}}{\bar{\rho}} - (\mu_1+\delta). \end{aligned} \tag{5.32}$$

as $\phi(\pi_V)$ is monotone decreasing in π_V. Hence $g_1(\pi_I) \leq -(\mu_1+\delta)(1-R_0) = -\varepsilon$ where $\varepsilon > 0$, so from equation (5.31) we have

$$\begin{aligned} \int_0^t \frac{1}{\pi_I}\frac{d\pi_I}{dt}dt &\leq \int_0^t (-\varepsilon)dt, \\ \Big[\log \pi_I\Big]_0^t &\leq -\varepsilon t, \\ \log\left(\frac{\pi_I(t)}{\pi_I(0)}\right) &\leq -\varepsilon t. \end{aligned} \tag{5.33}$$

Hence $0 \leq \pi_I(t) \leq \pi_I(0)e^{-\varepsilon t}$. Now as $t \to \infty$ then $\pi_I(0)e^{-\varepsilon t} \to 0$, so $\pi_I(t) \to 0$ as $t \to \infty$.

It is then straightforward to show that $\pi_V(t) \to 0$ as $t \to \infty$. Given $\varepsilon > 0$ there

exists to such that $\pi_I \leq \varepsilon$ for $t \geq t_0$. Hence for $t \geq t_0$

$$\frac{d}{dt}(\pi_V e^{\mu_2 t}) \leq \delta \pi_I e^{\mu_2 t}.$$

$$\leq \delta \varepsilon e^{\mu_2 t},$$

$$\pi_V e^{\mu_2 t} - \pi_V(0) \leq \delta \varepsilon e^{\mu_2 t},$$

$$0 \leq \pi_V(t) \leq \pi_V(0) e^{-\mu_2 t} + \frac{\delta \varepsilon}{\mu_2}(1 - e^{-\mu_2 t}).$$

so

$$limsup \pi_V(t) \leq \frac{\delta \varepsilon}{\mu_2}.$$

As ε can be made arbitrarily small it is straightforward to show that $\pi_V \to 0$.

This completes the proof of Theorem (5.5.3). So the DFE is globally stable for R_0 <1.

However, to investigate the global stability of the endemic equilibrium (EE) we attempted several mathematical methods such as the construction of Dulacś criterion and the Poincaré-Bendixson Theorem (Strogatz (2018), May (2001) and DeJesus and Kaufman (1987)) to analyse the global stability of endemic equilibrium EE. Unfortunately, this proved difficult, and no results were obtained.

As we move forward in the following section, we will present some numerical simulations of our analytical results for the models.

5.5.4 Simulations

We employ numerical simulations to verify our theoretical results, which showed the behaviour of our HIV/AIDS model with a disease awareness program showing that the disease will die out if $R_0 \leq 1$ or become endemic if $R_0 > 1$. Similarly to previous chapters, in section simulations,we used MATLAB and a numerical ODE solver (ode45) to estimate the behaviour of HIV/AIDS disease prevalence over time. We used realistic parameter settings for the model (5.8) and (5.9) with two disease awareness programs function $\phi(\pi)$ ((Greenhalgh et al. 2015), (Misra

et al. 2011) and (Samanta et al. 2013)). In this simulation, we test two functional forms of awareness programs $\phi(\pi)$ that have been used in the previous chapters. The two functions are as follows

(i) $\phi(\pi) = \left(1 - \frac{a\pi}{b+\pi}\right)$ where a and b are positive constants with $0 \leq a \leq 1$.

(ii) $\phi(\pi) = e^{-m_0 n\pi}$ where m_0 is constant and n represents the number of the PWIDs population.

We shall make similar assumptions as in Chapter 2. We assume that $\lambda_1 > \lambda_2$ so that susceptible and infected but unaware PWIDs visit shooting galleries at a higher rate λ_1 than aware infected PWIDs who are on HAART treatment.

We kept the same values for the probabilities parameters as shown in Chapter 2 namely $p = 0$, ϕ_1 and θ_1 are zero as these probabilities are very small. We assume that the realistic values for $\mu_2 > \mu_1$, which are the per capita rates at which infected PWIDs leave the sharing injecting population, and we have $\gamma = n/m = 1$ (based on Liang et al. (2016)). We assume the value of δ=1/six months ,which is the per capita rate at which infected but unaware PWIDs transfer to successful HAART. These values are as fallows:

$$\mu_1 = 7.0637\times10^{-4}/\text{ day}, \quad \mu_2 = 7.9398 \times10^{-4}/\text{ day}, \qquad P_1 = 0.74,$$

$$P_2 = 0.25, \qquad P_3 = 0.01, \qquad P_4 = 0.0,$$

$$\lambda_1 = 0.190/\text{day}, \qquad \lambda_2 = 0.143/\text{day}, \qquad \delta = 1/180 = 0.0056\ /\text{day}.$$

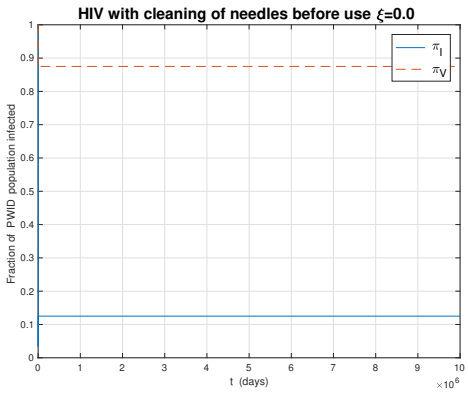
We choose to vary the needle cleaning probability ξ with $0 \leq \xi \leq 1$,

We examined the behaviour of the model equations (5.8) and (5.9) by altering R_0 by choosing two different values of ξ. In the following examples, we chose two values of ξ one where the disease is die out when $R_0 \leq 1$ and one where the disease is endemic when $R_0 > 1$. The starting value was initially $\pi(0) = 1$ $\beta(0) = 1$ in all cases.

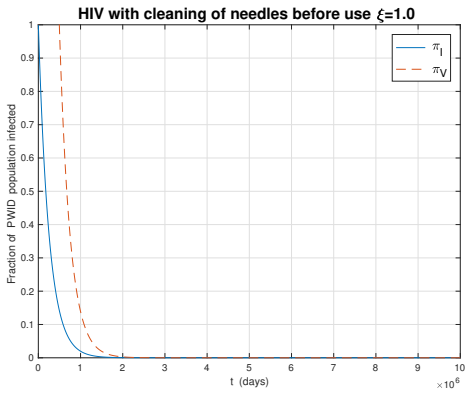
- **Example 5.1:**

  Figure 5.1 shows the plot of two simulations without using disease awareness program function over time. We use the the current set of parameter values shown above .So the sub-Figures 5.1a show π_I and π_V the fraction of PWIDs who are unaware infected and aware infected respectively, where the disease is present in both PWIDs and needles if $R_0 > 1$ when choosing $\xi = 0.0$. Then from the equations model (5.8) - (5.9) and the composite parameters in equation (5.7), we have $\nu = 0.1425$/day, $\bar{\sigma} = 0.1900$/day, $\bar{\tau} = 0.1900$/day and $\bar{\rho} = 0.0475$/day,$_2 = 0.1416$/day, giving $R_0 = 22.9865$.

  On the other hand, the sub-Figures 5.1b show π_I and π_V the fraction of PWIDs who are unaware infected and aware infected respectively, where the disease is die out in both PWIDs and needles if $R_0 < 1$ when choosing $\xi = 1.0$, then from the model equations (5.8) - (5.9) and the composite parameters in equations (5.7), we have $\nu = 0.0$/day, $\bar{\sigma} = 0.1900$/day, $\bar{\tau} = 0.1900$/day and $\bar{\rho} = 0.1900$/day, $_2 = 0.1430$/day, giving $R_0 = 0.0$.



(a) $\xi = 0.0$, then $R_0 > 1$.



(b) $\xi = 1.0$, then $R_0 < 1$.

Figure 5.1: The plots of simulations for the solution of model (5.8) and (5.9) without values of awareness program function where $n = 1000$.

- **Example 5.2:** $\phi(\pi) = e^{-m_0 n \pi}$.

We now simulate our model (5.8) and (5.9) using the first disease awareness program function, the function we use is $\phi(\pi)$= $e^{-m_0 n\pi}$ (taken from Cui et al. (2008)) with choosing different values of m_0 where is constant and n represents the number of the PWIDs population. In the Figure 5.2 shows plots of six simulations with the disease awareness program over time, we picked $m_0 = 2.0/n, 3.7/n$ and $10.0/n$, and use the same set of parameters values shown above.

Again what seems to be illustrated in sub-Figures 5.2a, 5.2c and 5.2e is the fraction of PWIDs who are unaware infected an aware infected respectively. These PWIDs do not clean their needles before use As previous an example if $\xi = 0.0$ then $R_0 = 22.9865$. This indicates that over a considerable period of time, the proportion of the PWID population that was HIV-positive tended to the particular endemic equilibrium.

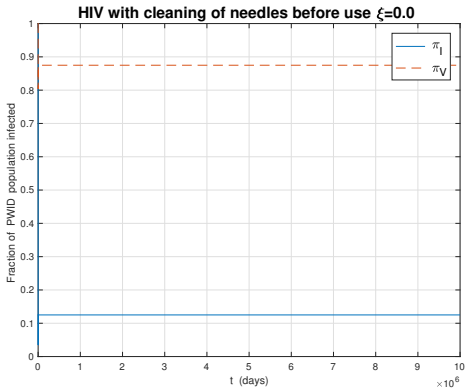
One other case, the sub-Figures 5.2b, 5.2d and 5.2f choosing $m_0 = 2.0/n, 3.7/n$ and $10.0/n$ show the fraction of PWIDs who are unaware infected an aware infected respectively. These PWIDs often cleaned their needles successfully before use. if $\xi = 1.0$ then we have $R_0 = 0.0$ as case $R_0 < 1$ in example 5.1, then After a long period of time thee HIV virus eliminated from both PWIDs and needles.

- **Example 5.3:** $\phi(\pi)$=$1 - \frac{a\pi}{b+\pi}$ We now simulate our model (5.8) and (5.9) using the second disease awareness program function $\phi(\pi) = 1 - \frac{a\pi}{b+\pi}$ (taken from Li et al. (2008a)), with different values of the constants a and b constant are shown in the Figure 5.3. Apply the same values to the parameters as were previously shown.

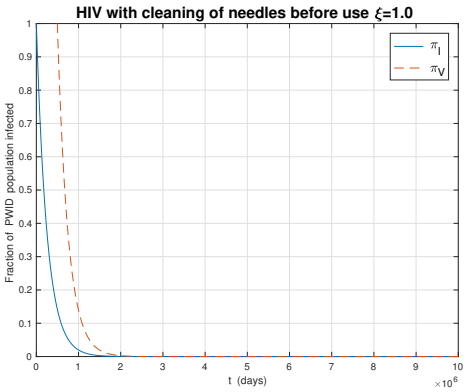
  The plots of six simulations in Figure 5.3 are shown the fraction of

PWIDs who are unaware infected an aware infected respectively. So aging we have a similarly results to the previous Figures 5.2 and 5.1. If $\xi = 0.0$, so we have that $R_0 > 1$ for the sub-figures 5.3a, 5.3c and 5.3e of Figure 5.3 when both PWIDs and needles are infected with the virus. The composite parameters in equation (5.7), and the equations model (5.8) - (5.9), thus give us $\nu = 0.1425$/day, $\bar{\sigma} = 0.1900$/day, $\bar{\tau} = 0.1900$/day and $\bar{\rho} = 0.0475$/day,$_2 = 0.1416$/day, giving $R_0 = 22.9865$.

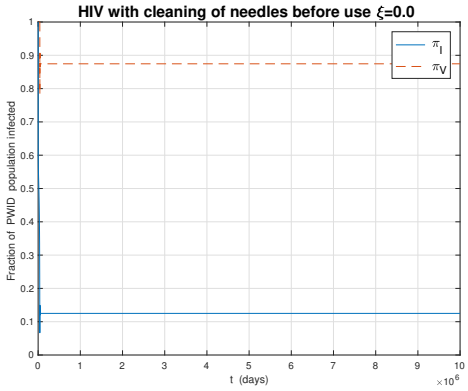
On other hand, if $\xi = 1.0$ then we have $R_0 < 1$ for sub-figures 5.3b 5.3d and 5.3f of Figure 5.3. So in this case We can observe that the proportion of infected PWIDs and needles will eventually reach zero. So from the equations model (5.8) - (5.9)and the composite parameters in equation (5.7) we have that $\nu = 0.0$/day, $\bar{\sigma} = 0.1900$/day, $\bar{\tau} = 0.1900$/day and $\bar{\rho} = 0.1900$/day, $_2 = 0.1430$/day, giving $R_0 = 0.0$.



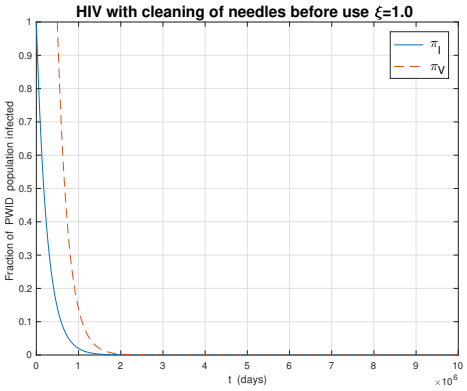
(a) With values of awareness program function parameters $m_0 = 2.0/n$.



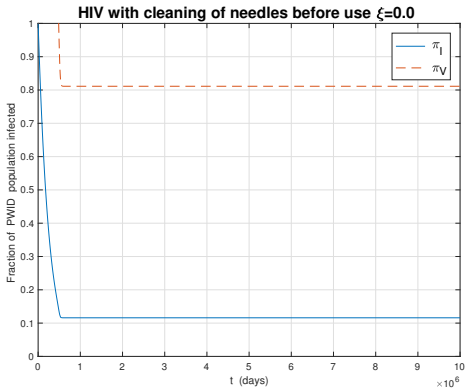
(b) With values of awareness program function parameters $m_0 = 2.0/n$.



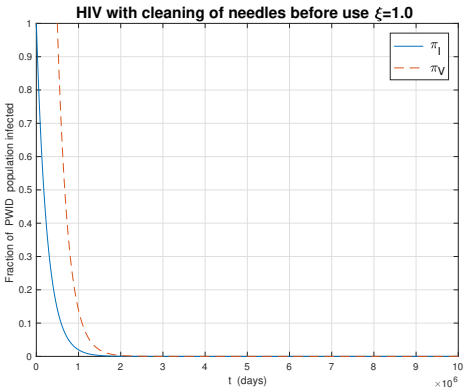
(c) With values of awareness program function parameters $m_0 = 3.7/n$.



(d) With values of awareness program function parameters $m_0 = 3.7/n$.

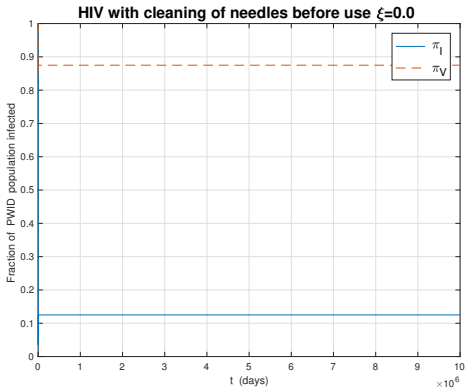


(e) With values of awareness program function parameters $m_0 = 10.0/n$.

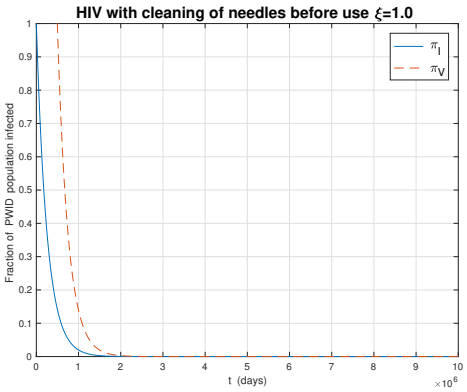


(f) With values of awareness program function parameters $m_0 = 10.0/n$.

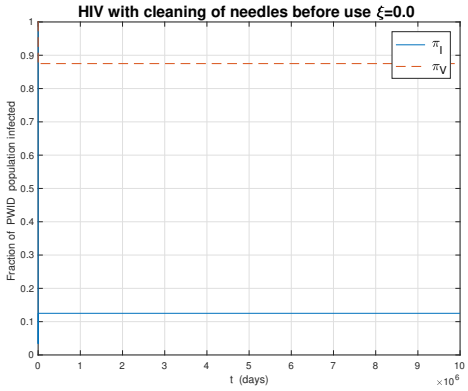
Figure 5.2: The plots of simulations for the solution of model (5.8) and (5.9) with awareness program function $\phi(\pi)$= $e^{-m_0 n\pi}$, where $n = 1000$ and when $\xi = 0.0$ so $R_0 > 1$ and so $\xi = 1.0$ then $R_0 < 1$.



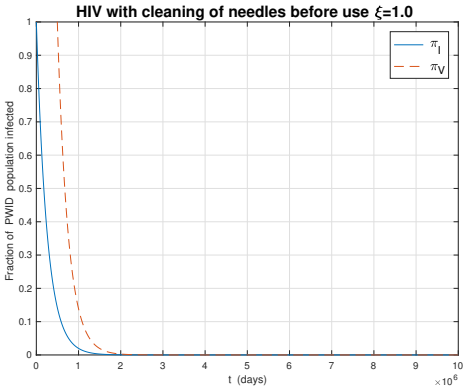
(a) With values of awareness program function parameters $a = 0.9, b = 1$.



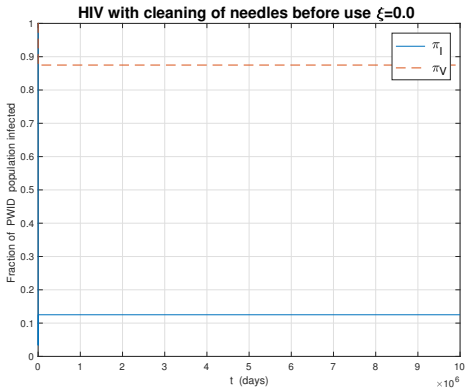
(b) With values of awareness program function parameters $a = 0.9, b = 1$.



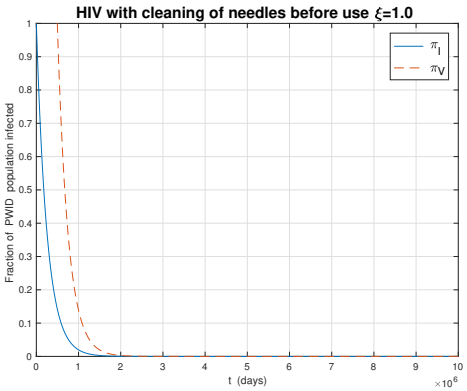
(c) With values of awareness program function parameters $a = 0.5, b = 5$.



(d) With values of awareness program function parameters $a = 0.5, b = 5$



(e) With values of awareness program function parameters $a = 0.1, b = 10$.



(f) With values of awareness program function parameters $a = 0.1, b = 10$.

Figure 5.3: The plots of simulations for the solution of model (5.8) and (5.9) with awareness program function $\phi(\pi)$=1 − $\frac{a\pi}{b+\pi}$ and so $\xi = 0.0$ when $R_0 > 1$ and so $\xi = 1.0$ when $R_0 < 1$.

5.6 Conclusion

In this chapter, we have discussed the effects of awareness programs on the transmission dynamics of HIV among PWIDS on successful HAART. We have developed and investigated a deterministic model that focussed on the impact of awareness programs on the behaviour of HIV/AIDS transmission in two groups of infected PWIDS where the first group is unaware and the second group is successfully treated with HAART. This model is an original model although its derivation is based on the models discussed in Chapters 2 and 3. As far as we are aware this is the first model in the literature that takes account of the effect of HAART on the spread of HIV amongst PWIDs.

The model studied in this chapter differs from the models discussed in the previous chapters in that the susceptibles are split into successfully treated (with HAART) and unaware susceptibles.

A formula for the basic reproduction number R_0 was derived that allowed us to perform the analysis of the model. Our analysis indicates that this is a critical threshold parameter $R_0 = 1$. Based on our analytical results, the system has two equilibria: the disease-free equilibrium solution and the endemic equilibrium solution. Our analysis is assuming that ϕ is monotone decreasing shows that the disease-free equilibrium is locally asymptotically stable if $R_0 < 1$, neutrally stable if $R_0 = 1$ and unstable if $R_0 > 1$. In the case when $R_0 > 1$, we demonstrated that the endemic equilibrium existed and is also locally asymptotically stable. Also, we showed that the disease-free equilibrium solution was globally asymptotically stable when If $R_0 \leq 1$. Lastly, we carried out numerical simulations to illustrate the dynamic behaviour of this model.

Chapter 6

Conclusions and Further Work

The spread of HIV and AIDS among communities that use intravenous drugs poses a significant threat to their health. Sharing needles and other injectable supplies makes it possible for this virus to spread more quickly and efficiently within these populations than it would within the general population. The transmission of HIV through the sharing of needles is influenced by a diverse set of factors, each of which has been demonstrated to be significant in its own right. Social networks that facilitate the sharing of injections amongst addicts have also been identified as potentially key parts of the problem. Greenhalgh and Hay (1997)is one author who has proven the significance of variation in needle-sharing rates and needle-cleaning efficacy.

In Chapter 2, we have developed a mathematical model of the effect of disease awareness programs on the prevalence of HIV amongst PWIDs, building on the models developed by Greenhalgh and Hay (1997) and Liang et al. (2016). A system of differential equations has been deduced to describe the improved model that reduces the spread of the diseases through the effect of awareness of the disease on sharing needles and syringes amongst the PWID population.

Our discussion has been focused on two ways of studying the effect of awareness programs into disease transmission models. The key biological parameter of our

model is the basic reproductive number R_0. We have shown that the system has a unique equilibrium solution. If ϕ is monotone decreasing, then we have shown that if $R_0 \leq 1$ then the DFE is globally asymptotically stable, so whatever the initial fraction of infected individuals, the disease will die out as time becomes large. If there is no disease initially present, then there will never be any disease. If there is disease initially present and $R_0 > 1$ then the system tends to the unique EE. We also showed that the DFE is locally asymptotically stable if $R_0 < 1$, neutrally stable if $R_0 = 1$ and unstable if $R_0 > 1$. In the case if $R_0 > 1$, we showed that the EE was locally asymptotically stable too.

Chapter 3, We began chapter 3 by expanding our study model to be a two-dimensional model. We derived a system of differential equations of HIV amongst PWIDs with the disease awareness programs which kept the expression of the biological parameter for R_0 as same as in the previous chapter. We showed the equilibrium solutions analytically if ϕ is strictly monotone decreasing or monotone decreasing. Then we have shown that if $R_0 \leq 1$ is the only condition for the disease to die out in all PWIDs and needles. Whereas if the disease is initially present and $R_0 > 1$ the disease will present among the population of PWIDs for all time. Furthermore, we proved that the free disease equilibrium of the model (3.1) and (3.2) is locally and globally stable if $R_0 < 1$, whereas it is unstable if $R_0 > 1$. Also, we showed that if $R_0 > 1$ the system has a unique endemic solution which is locally and globally stable, wherever the disease is present and either $\pi(0) > 0$ or $\beta(0) > 0$. So if either $\pi(0) > 0$ or $\beta(0) > 0$ and $R_0 > 1$the system tends to the unique endemic equilibrium.

In Chapter 4 we have considered the effect of awareness programs in a three-stage infectivity model of HIV/AIDS. We developed the three-stage HIV/AIDS infection model studied by Lewis (2000) by applying awareness programs functions. And then we derived the system of differential equations for the spread of HIV amongst PWIDs with disease awareness programs. We calculated an ex-

pression for the basic reproduction number R_0 that allowed us to figure out the analysis of the model, we have shown that for any given initial value condition in the region D=$[0,1]^4$ in R^4, the system of the model has a unique non-negative solution that remains in D for all time, and also the conditions required for persistence for the infected fractions of PWIDs and needles π_1, π_2, π_3 and β. In general, the disease is persistent when $R_0 > 1$ as shown in Theorem 4.3.5. Analytically, we determined the equilibrium solution where the disease dies out or persists in both PWIDs and needles for our model and evaluated its local and global stability. We showed that if $R_0 < 1$ there is only the disease-free equilibrium where if $R_0 \leq 1$ disease free DFE equilibrium is locally asymptotically stable, as well as globally stable. Otherwise, the disease-free solution DFE is unstable if $R_0 > 1$. Also, there is endemic equilibrium solution EE which is locally asymptotically stable if $R_0 < 1$.

In the last chapter, we have discussed the effects of awareness programs on the transmission dynamics of HIV among people who inject drugs (PWIDs) on successful HAART. We have developed and investigated a deterministic model, that focused on the impact of awareness programs on the behaviour of HIV/AIDS transmission in two groups of infected PWIDs where the first group is unaware and the second group is successfully treated with HAART.

A formula for the basic reproduction number R_0 was derived that allowed us to perform the analysis of the model. Our analysis indicates that this is a critical threshold parameter $R_0 = 1$. Based on our analytical results, the system has two equilibria: the disease-free equilibrium solution and the endemic equilibrium solution. Our analysis assumes that ϕ is monotone decreasing shows that the disease-free equilibrium is locally asymptotically stable if $R_0 < 1$, neutrally stable if $R_0 = 1$ and unstable if $R_0 > 1$. In the case when $R_0 > 1$, we demonstrated that the endemic equilibrium existed and is also locally asymptotically stable. Also, we showed that the disease-free equilibrium solution was globally asymptotically stable when If $R_0 \leq 1$. Lastly, we carried out numerical simulations to illustrate the

dynamic behaviour of this model.

To the greatest extent of our knowledge, the study we are doing presently fills a research gap as it is the first time disease awareness programs have been applied to a mathematical model of HIV among PWIDs.

For further study, we believe that the awareness programs technique employed in this thesis can be applied to additional diseases such as COVID and measles, To reduce the global spread of epidemic diseases.

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