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Mathematical transmission model of the spread of HIV through sexual contact and IV drug usage among heterosexuals in Western Australia

B. Glockner
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**MATHEMATICAL TRANSMISSION MODEL
OF THE SPREAD OF HIV THROUGH SEXUAL
CONTACT AND IV DRUG USAGE AMONG
HETEROSEXUALS IN WESTERN AUSTRALIA**

B. Glockner

BAppSc (Hons) Mathematics

1993

USE OF THESIS

The Use of Thesis statement is not included in this version of the thesis.

MATHEMATICAL TRANSMISSION MODEL OF THE SPREAD OF HIV THROUGH SEXUAL CONTACT AND IV DRUG USAGE AMONG HETEROSEXUALS IN WESTERN AUSTRALIA

BY

B. Glockner

**A Thesis Submitted in Partial Fulfilment of the Requirements for the award of
Bachelor of Applied Science Honours (Mathematics) at the Faculty of Science and
Technology, Edith Cowan University.**

December 1993

Abstract

The human immunodeficiency virus (HIV) and its latter stage, the acquired immune deficiency syndrome (AIDS), are still a major health problem in the world and the full extent of the epidemic is unknown. The HIV virus in Australia has been spread mainly through homosexual contact, but this mode of transmission is decreasing while transmission from heterosexual contact and from IV drug use is increasing. The proposed mathematical model concentrates on transmission of the HIV virus through heterosexual contact and IV drug use among heterosexuals in Western Australia. The model parameters will be estimated from surveys conducted by the Australian National Injecting Drug Use Study 1989-1991. The estimated parameters will be incorporated into the developed model and used in conjunction with computer simulations to study the spread of HIV and its progression to AIDS in Western Australian heterosexuals.

Preface

The figures found in Appendix 4 have been labelled using the following code :

I : The number of intravenous drug users which are infected with the HIV virus and have not developed AIDS.

N : The number of non-intravenous drug users which are infected with the HIV virus and have not developed AIDS.

C : The average percentage of time condoms are used.

P : The average number of needle sharing 'partners' which an intravenous drug user has in a year.

e / f : The results are provided for both 'e' and 'f' values for the purpose of a comparison between the two.

g (94) : The results are provided for when the original value for condom usage or needle sharing 'partners' was changed to 'g' in 1994.

Therefore Figure A4.22 : $I, C = 16.5, P = 1.6 / C = 35.5, P = 1$. is the number of living intravenous drug users that are infected with the HIV virus and have not progressed to AIDS given that in one instance condoms are used on average 16.5% of the time and IVDUs had on average 1.6 needle sharing 'partners' in a year and in the second instance condoms are used on average 35.5% of the time and IVDUs had on average 1 needle sharing 'partner' in a year.

Declaration

"I certify that this thesis does not incorporate without acknowledgment any material previously submitted for a degree or diploma in any institution of higher education; and that to the best of my knowledge and belief it does not contain any material previously published or written by another person except where due reference is made in the text."

Acknowledgments

I wish to acknowledge my supervisor, Dr Catherine Comiskey, for the support received throughout the year and The AIDS Bureau for providing me with up to date statistics.

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1. HIV and AIDS

1.1 Background to HIV and AIDS

The human immunodeficiency virus (HIV) was first identified in the United States of America in 1981 when the US Centers for Disease Control reported 5 cases of a young gay man in Los Angeles suffering from a rare pneumonia, *pneumocystis carinii* pneumonia. Another ten cases of the rare pneumonia were reported in Los Angeles, New York and San Francisco a month later as well as 26 cases of Kaposi's sarcoma. The first case definition of the acquired immune deficiency syndrome (AIDS) was published in September 1982 (Elford et al, 1991).

The HIV virus is contracted through contact with body fluids infected with the virus. The virus can be isolated from various body fluids such as blood, semen, cervical and vaginal secretions, tissues, saliva, breast milk and tears of an infected individual. Tears and saliva are the only body fluids that have not transmitted the HIV virus. Breast milk has not definitely been identified as being responsible in the transmission of the virus, but has been assumed to be possible of transmission. Non sexual social contact and insects do not transmit the virus (Nation Health and Medical Research Council, 1990).

The HIV virus affects the immune system of the infected individuals making them susceptible to other diseases. It is when the individuals show the symptoms of these 'opportunistic' infections that they are diagnosed as having the acquired immune deficiency syndrome (AIDS).

The United States Centers for Disease Control in January 1993 revised the case definition for AIDS. The revised case definition of AIDS provides a more accurate

description of severe cases of HIV-related immunodeficiency. Both Australia and Europe have decided to follow only the second point of the revised definition. In Australia this new case definition is effective from 1 January 1993 (National Centre in HIV Epidemiology and Clinical Research, 1993). Appendix 1 provides the case definition of AIDS developed by the Centers for Disease Control as of 1987.

The revised definition of AIDS includes the following two points as described by the National Centre in HIV Epidemiology and Clinical Research, page 8, 1993.

"(i) adults and adolescents with diagnosed HIV infection who have a CD4+T-lymphocyte count of less than 200ul of blood or a CD4+T-lymphocyte percentage of less than 14, with or without the diagnosis of disease(s) indicative of a defect in cell-mediated immunity, and (ii) pulmonary tuberculosis, recurrent pneumonia and invasive cervical cancer as AIDS-defining conditions. All AIDS-defining conditions included in the 1987 case definition are retained in the 1993 definition."

1.2 Global Patterns

There are three main global patterns of spread of the HIV virus. The first pattern, which fits western industrialised world countries, describes the epidemic as starting in the late 1970's to early 1980's. The virus has affected mainly homosexual / bisexual individuals as well as intravenous drug users. Small numbers of heterosexuals have been infected and the male to female ratio is approximately 10:1. Infection from blood products is under control through screening programs (Sato, et al, 1989).

Pattern Two, which fits countries in the Caribbean and sub-Saharan Africa, describes the epidemic as starting in the late 1970's to early 1980's. The main mode of infection is through heterosexual contact, thereby providing a male to female ratio of approximately 1:1 and also high numbers of perinatal cases. Infection from

contaminated blood products is still significant as there is no adequate screening program (Sato, et al, 1989).

Pattern Three fits communist bloc countries and those which up until the last few years were predominantly communist. The spread of the HIV virus started in the early to mid 1980's and occurred in all risk groups. The main mode of infection was in people travelling to high risk areas and contracting the disease (Sato, et al, 1989).

1.3 HIV and AIDS in Australia

The first recorded AIDS case in Australia occurred in December of 1982 in Sydney (Kaldor et al, 1993). The individual contracted the virus from homosexual contact while in the U.S.A. Since then the number of new AIDS cases in Australia has been increasing with 692 cases in 1992 and 231 cases in the first six months of 1993, making a total of 4258 reported AIDS cases from 1982 to the end of March 1993 (National Centre in HIV Epidemiology and Clinical Research, 1993).

By 30 June 1992, 36 AIDS cases were reported to be diagnosed in January 1992. By 31 March 1993, there were 50 reported cases of AIDS being diagnosed for the same month. Therefore 14 cases were reported at least 5 months after diagnosis. Another 2 cases diagnosed in January 1992 were reported by 30 June 1993, at least 14 months after first diagnosis (National Centre in HIV Epidemiology and Clinical Research, July 1992, July 1993, October 1993).

At least 206 AIDS cases reported by June 1993 for diagnosis in the first 6 months of 1992 were not reported in the month they were diagnosed. These delayed reported cases more than doubled the number, for those months, of diagnosed AIDS cases

reported to 30 June 1992 (National Centre in HIV Epidemiology and Clinical Research, July 1992, October 1993).

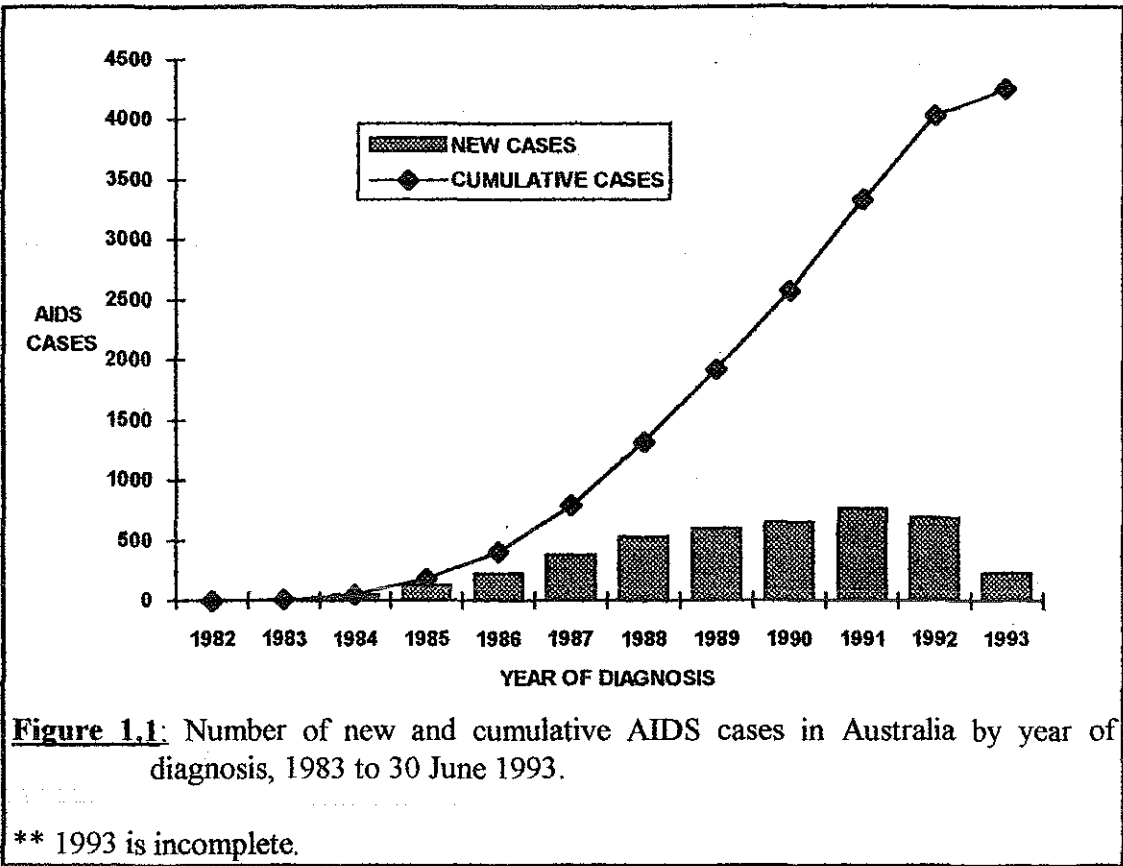


Figure 1.1 is based upon Table 1.1 below and shows the growth in the number of diagnosed AIDS cases in Australia each year up to the end of June 1993. Although it appears that the number of new AIDS cases diagnosed each year is starting to decline, delays in diagnosis can increase the statistics for some years. This is demonstrated in Table 1.1 where a comparison of the Australian HIV Surveillance Reports for July 1992 and October 1993 is provided. The difference in Table 1.1 is for delays in diagnosis only as the data is incomplete for each year of the reports being published. The difference shows that there are long reporting delays of diagnosis of AIDS for recent years and until they are reported they can give a false impression of a reduction in the number of new AIDS cases.

Table 1.1: Number of new and cumulative AIDS cases in Australia by year of diagnosis, 1983 to 30 June 1993.

YEAR	NUMBER OF AIDS CASES					
	To June 1992 *1		To June 1993 *2		DIFFERENCE *3	
	NEW	CUMULATIVE	NEW	CUMULATIVE	NEW	CUMULATIVE
1982	1	1	1	1	0	0
1983	6	7	6	7	0	0
1984	46	53	46	53	0	0
1985	126	179	127	180	1	1
1986	227	406	227	407	0	1
1987	376	782	381	788	5	6
1988	527	1309	531	1319	4	10
1989	582	1891	600	1919	18	28
1990	633	2524	653	2572	20	48
1991	670	3194	763	3335	93	141
1992#	174	3368	692	4027	206	347
1993#	231	4258				

*1 (National Centre in HIV Epidemiology and Clinical Research, July 1992, Table 1.8, page 12).

*2 (National Centre in HIV Epidemiology and Clinical Research, October 1993, Table 4.8, page 13).

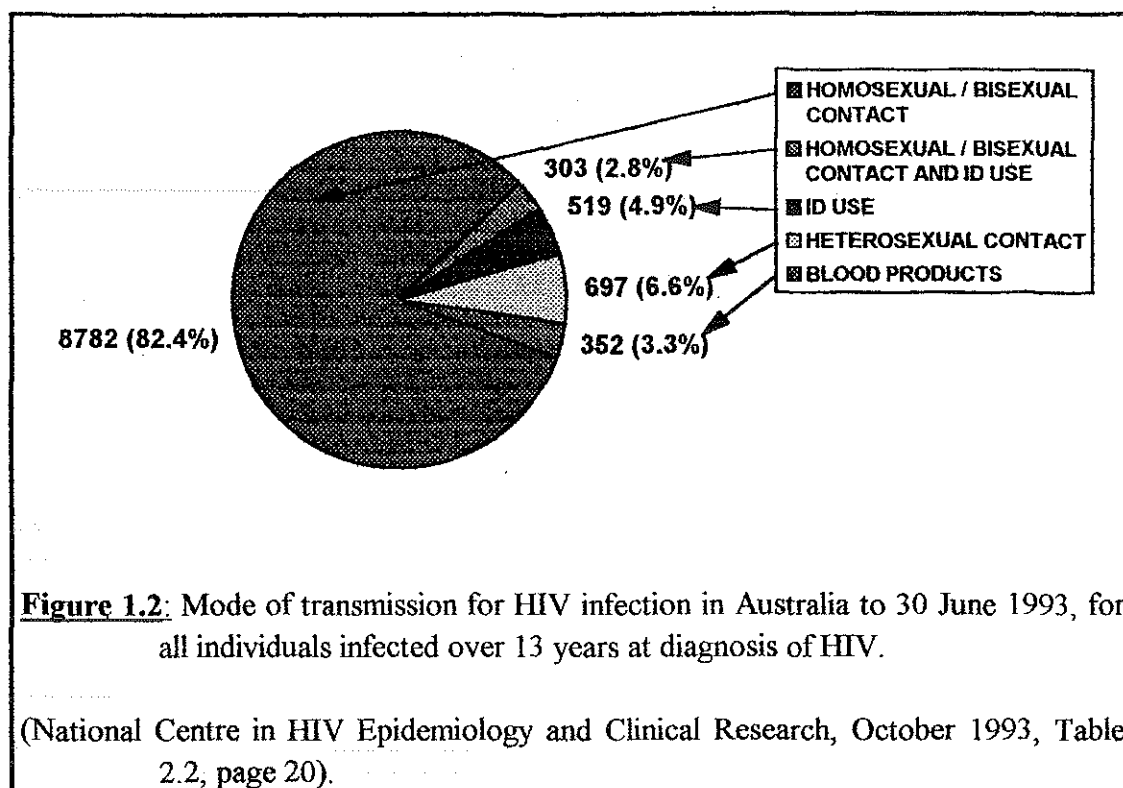
*3 Delays in diagnosis where the cases were reported after 30 June 1992 and before 30 June 1993, but occurred before 1 July 1992.

1992 and 1993 values are incomplete.

Between 30 June 1992 and 30 June 1993, a total of 347 AIDS cases were reported after delays ranging from a few months to nearly 8 years. Most of the delays between diagnosis and reporting of the case are 1 to 2 years (National Centre in HIV Epidemiology and Clinical Research, July 1992, July 1993, October 1993).

By the end of June 1993, there were 17475 reported cases of HIV infection in Australia. Homosexual/bisexual contact accounted for 82.4% of known transmission of infection of the HIV virus among adults 13 years or over at the time of diagnosis. IV drug use and heterosexual contact accounted for 4.9% and 6.5% of known modes of transmission of adults 13 years and over at the time of diagnosis, respectively

(National Centre in HIV Epidemiology and Clinical Research, October 1993). Figure 1.2 shows the number of adults infected via these modes of transmission of the HIV virus and the percentages.



By 30 June 1993 there were 10653 adolescents and adults 13 years and over diagnosed as being infected with the HIV virus and whose mode of transmission was known. Children under the age of 13 years whose mode of transmission was known, accounted for 94 cases of diagnosed HIV infection and there were 6728 cases where the mode of transmission was not determined or is classified as 'other'. 32 of these other / undetermined cases were of children and 86 cases of the total of 6728 other / undetermined cases were reported between 30 June 1992 and 30 June 1993 (National Centre in HIV Epidemiology and Clinical Research, July 1992, October 1993).

Table 1.2 shows the number and percentage of males and females that were diagnosed as being HIV positive to the 31 March 1993. The ratio of male to female shows that

even among children under the age of 13 years the number of diagnoses of HIV is much higher in males than females. The high male to female ratio is expected among the adult cases of HIV diagnosis due to the high number of male homosexuals and bisexuals infected, the male to female ratio of HIV diagnosis among children would be expected to be equal except for the high number of haemophiliacs infected. Over half of the reported diagnosed cases of HIV infection among children under the age of 13 years were through this disorder which affects mostly males.

Table 1.2: Number and percentage of cumulative diagnosis of HIV infection in Australia by sex, to 31 March 1993.

CATEGORY	MALE	FEMALE	OTHER ^{#1}	TOTAL	RATIO ^{#3}
ADOLESCENT / ADULT	10168 (58.18%)	457 (2.62%)	28 (0.16%)	10653 (60.96%)	95.4: 4.3: 0.3
CHILD <13 YEARS	76 (0.43%)	15 (0.09%)	3 (0.02%)	94 (0.54%)	80.8:16.0: 3.2
OTHER ^{#2}	4334 (24.80%)	311 (1.78%)	2083 (11.92%)	6728 (38.50%)	64.4:4.6: 31.0
TOTAL	14578 (83.41%)	783 (4.49%)	2114 (12.1%)	17475 (100%)	83.4:4.5: 12.1

(National Centre in HIV Epidemiology and Clinical Research, July 1993, Table 2.2, page 20).

^{#1} Sex was not reported or reported as transsexual.

^{#2} Other or undetermined mode of transmission.

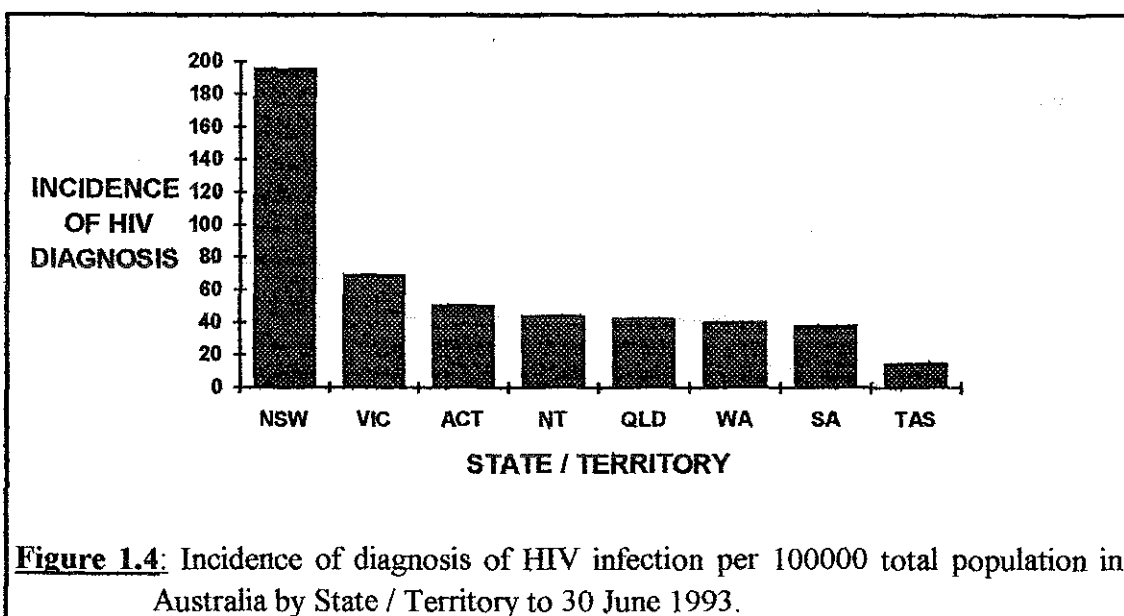
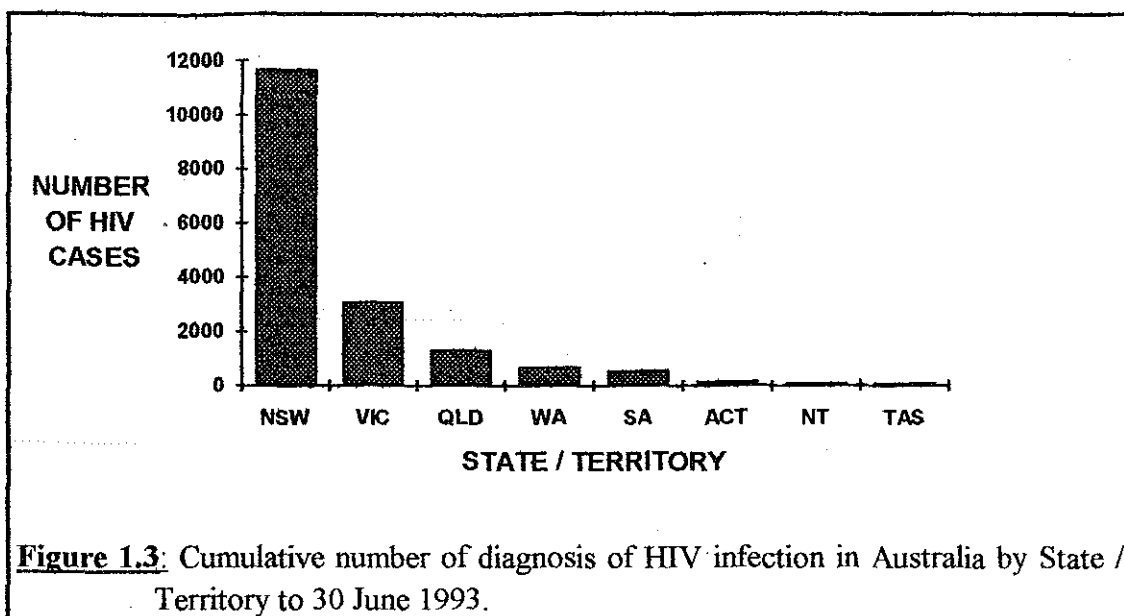
^{#3} Ratio of male to female to 'other ^{#1}' for the cumulative number of diagnosed HIV infection in Australia.

Table 1.3 shows the numbers and rates of diagnosis per 100000 total current population of HIV infection and AIDS cases for all States and Territories of Australia. The Table shows that the total number of diagnosed cases of HIV infection and AIDS is not reflective on the incidence of AIDS and HIV cases within the population. Figures 1.3 and 1.4 are based upon Table 1.3 and clearly show the differences between the cumulative number of HIV cases and rate of HIV diagnosis per 100000 total population among the States and territories of Australia.

Table 1.3: Cumulative number and rate of diagnosis of HIV infection and AIDS in Australia by State / Territory to 30 June 1993.						
STATE/ TERRITORY	HIV INFECTION			AIDS CASES		
	NUMBER	%#1	RATE#2	NUMBER	%#1	RATE#2
ACT	148	0.9%	50.3	54	1.3%	18.4
NSW	11649	66.7%	195.5	2502	58.8%	42.0
NT	73	0.4%	43.7	17	0.4%	10.2
QLD	1280	7.3%	42.2	375	8.8%	12.4
SA	547	3.1%	37.6	174	4.1%	12.0
TAS	68	0.4%	14.5	26	0.6%	5.5
VIC	3048	17.4%	68.5	918	21.5%	20.6
WA	662	3.8%	40.0	192	4.5%	11.6
TOTAL	17475	100%	100.0	4258	100%	24.4
(National Centre in HIV Epidemiology and Clinical Research, October 1993, Tables 4.1, 4.2 and 5.1, pages 7, 8 and 14).						
#1 Percentage is of the total number of AIDS / HIV cases in Australia.						
#2 Rate is the incidence of HIV infection or AIDS diagnosis per one hundred thousand current population.						

New South Wales has both the highest number and rate of AIDS and HIV diagnosed cases in all of the States and territories in Australia. Victoria is the State with the next highest incidence of HIV and AIDS diagnosis. New South Wales and Victoria account for 84.1% of the total number of diagnosed cases of HIV infection in Australia and 80.4% of the total number of diagnosed AIDS cases (National Centre in HIV Epidemiology and Clinical Research, October 1993).

Although the Australian Capital Territory has only the sixth highest number of diagnosed cases of HIV infection, the rate per 100000 total current population is third highest. The same is for the number of diagnosed AIDS cases. The State / Territory with the third highest number of diagnosed cases of HIV infection is Queensland. Queensland has also the third highest number of diagnosed AIDS cases among all the States and Territories of Australia.



Western Australia accounts for only 3.8% of the total number of diagnosed cases of HIV infection in Australia and 4.5% of the total number of diagnosed AIDS cases. Although Western Australia accounts for less than 5% of the total number of HIV and AIDS cases, it is the State / Territory with the fourth highest number of diagnosed cases of infection. Western Australia has an incidence of HIV infection that is only the sixth highest among the eight States/ Territories.

1.4 HIV and AIDS in Western Australia

The first AIDS case in Western Australia occurred in 1983 and again this was from homosexual contact (AIDS Bureau, 1993) (Health Department of Western Australia, 1993). The number of new diagnosis of AIDS cases has not been continuously increasing as is the case for Australia. There was a decrease in the number of newly diagnosed HIV and AIDS cases in 1987 and again in 1988. Figure 1.5 is based upon Table 1.4 and shows the changes in the number of newly diagnosed AIDS cases each year.

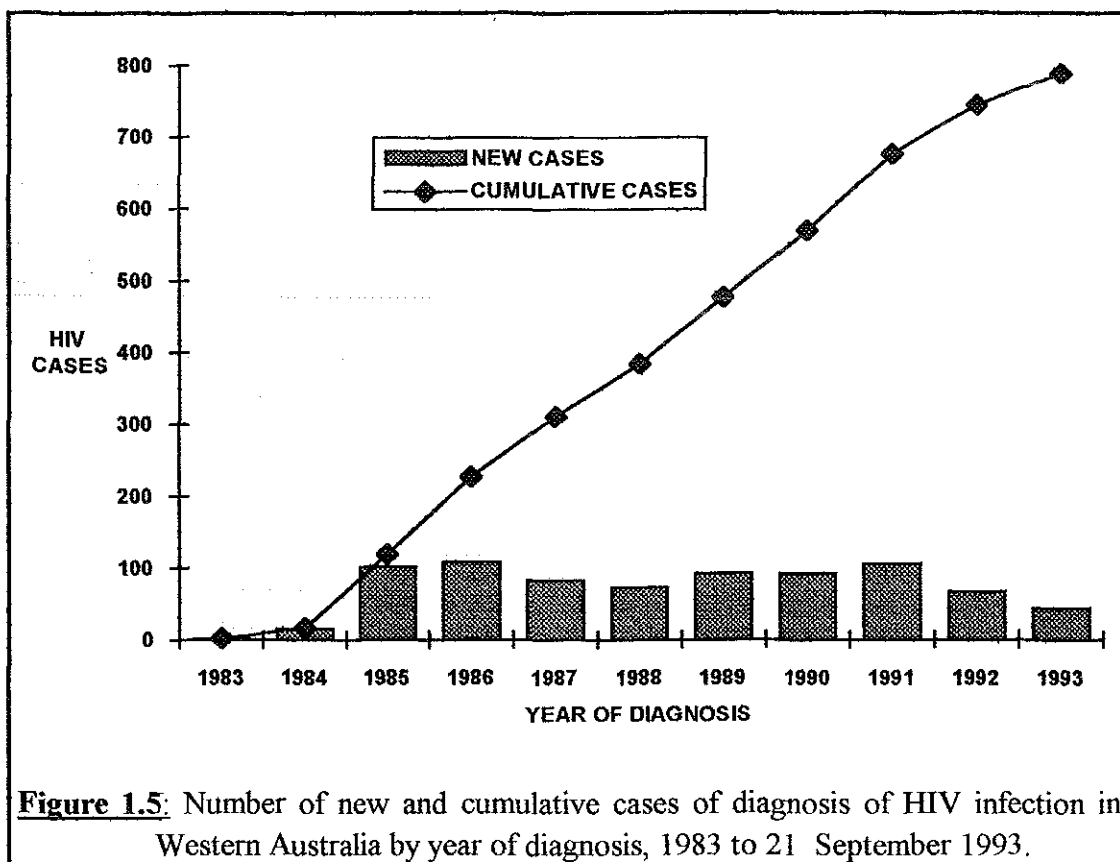


Figure 1.5: Number of new and cumulative cases of diagnosis of HIV infection in Western Australia by year of diagnosis, 1983 to 21 September 1993.

By 21 September 1993 there were a total of 788 diagnosed cases of HIV infection of which 238 had progressed to AIDS as shown in Table 1.4 (AIDS Bureau, 1993). Figure 1.6 shows that of the 238 diagnosed AIDS cases, 155 have died from AIDS.

68% of all diagnosed cases of HIV infection are living and have not progressed to AIDS and 2% have died from causes other than AIDS.

Table 1.4: Number of new and cumulative cases of diagnosis of HIV infection and AIDS in Western Australia by year of diagnosis, 1983 to 21 September 1993.

YEAR	HIV INFECTION		AIDS CASES	
	NEW	CUMULATIVE	NEW	CUMULATIVE
1983	2	2	2	2
1984	15	17	4	6
1985	102	119	35	41
1986	109	228	39	80
1987	83	311	38	118
1988	73	384	19	137
1989	93	477	26	163
1990	93	570	28	191
1991	106	676	25	216
1992	68	744	17	233
1993 [#]	44	788	5	238

(AIDS Bureau 1993, Table 4).

[#] Statistics for 1993 are incomplete.

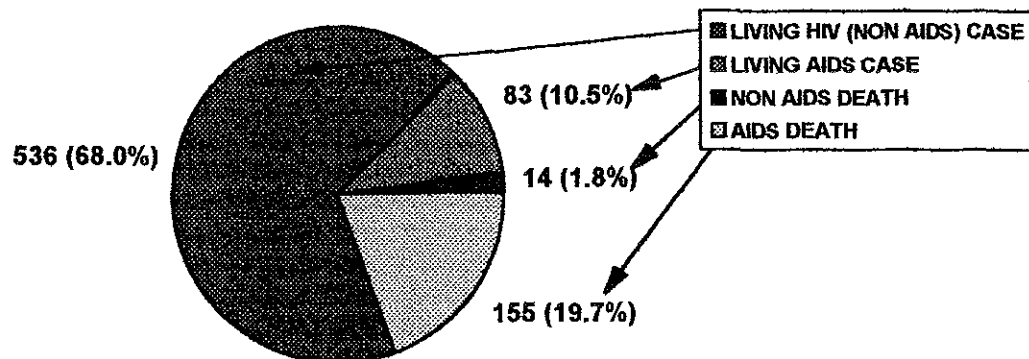


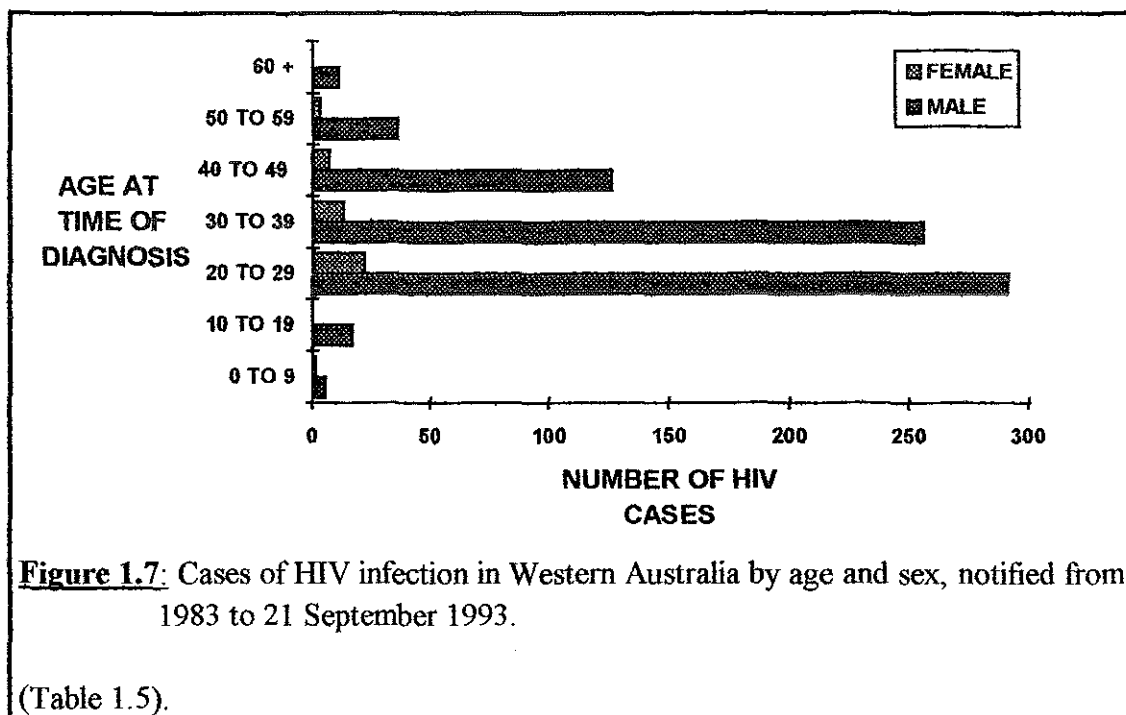
Figure 1.6: State of all HIV cases diagnosed in Western Australia, notified from 1983 to 21 September 1993.

(AIDS Bureau 1993, Table 1a)

Table 1.5: Cases of HIV infection in Western Australia by age and sex, notified from 1983 to 21 September 1993.

AGE	MALE		FEMALE		TOTAL	
	NUMBER OF CASES	PERCENTAGE	NUMBER OF CASES	PERCENTAGE	NUMBER OF CASES	PERCENTAGE
0-9	5	0.7 %	1	2.2 %	6	0.8 %
10-19	17	2.3 %	0	0.0 %	17	2.2 %
20-29	291	39.2 %	22	47.8 %	313	39.7 %
30-39	256	34.5 %	13	28.3 %	269	34.1 %
40-49	126	17.0 %	7	15.2 %	133	16.9 %
50-59	36	4.8 %	3	6.5 %	39	4.9 %
60+	11	1.5 %	0	0.0 %	11	1.4 %
ALL AGES	742	100 %	46	100 %	788	100 %

(AIDS Bureau 1993, Table 2).



Approximately 74% of diagnosed HIV infection in Western Australia occurred in individuals aged between 20 and 39, shown in Figure 1.7. Of the 46 females diagnosed with the HIV virus in Western Australia, 76.1% were aged between 20 and 39 and 15.2% were aged between 40 and 49. This is similar to the males of which 73.7% of the 742 diagnosed cases of HIV infection were between the ages of 20 and

39, and 17% were between the ages of 40 and 49. These percentages and numbers of diagnosed cases of HIV infection in Western Australia by age and sex are shown in Table 1.5. 94.2% of all diagnosed cases of HIV infection in Western Australia were males. Males aged between 20 and 29, the largest group by sex and age, accounted for 36.9% of all diagnosed cases.

Table 1.6: Number of new diagnosis of HIV infection in Western Australia by transmission category and year of diagnosis, 1983 to 21 September 1993.

YEAR	CATEGORY						
	H/BI	H/BI&ID	IVDU	HET.	BLOOD	PER.	UNKNOWN
1983	2	0	0	0	0	0	0
1984	8	0	0	0	7	0	0
1985	83	5	1	4	9	0	0
1986	87	6	6	3	5	0	2
1987	65	6	5	5	1	1	0
1988	54	8	3	4	3	0	1
1989	65	9	9	4	4	0	2
1990	65	6	6	10	3	0	3
1991	79	3	8	13	1	0	2
1992	48	3	6	11	0	0	0
1993#	32	1	0	7	0	2	2
TOTAL	588	47	44	61	33	3	12

(AIDS Bureau 1993, Table 4).

Statistics for 1993 are incomplete.

H/BI: Homosexual or bisexual contact.

H/BI&ID: Homosexual or bisexual contact and IV drug use.

IVDU: Intravenous drug user.

HETERO: Heterosexual contact.

BLOOD: Contaminated blood products.

PER: Perinatal transmission.

The percentage of known modes of transmission of the virus that occurred from homosexual/bisexual contact, at approximately 75%, is slightly less than the percentage for Australia. Infection from heterosexual contact and IV drug use, approximately 8% and 6% respectively, is slightly higher for Western Australia than

for Australia (Health Department of Western Australia, 1993) (AIDS Bureau, 1993). Figure 1.8 shows the numbers and percentages of the cumulative number of diagnosed cases of HIV infection accounted for by various modes of transmission of the virus in Western Australia.

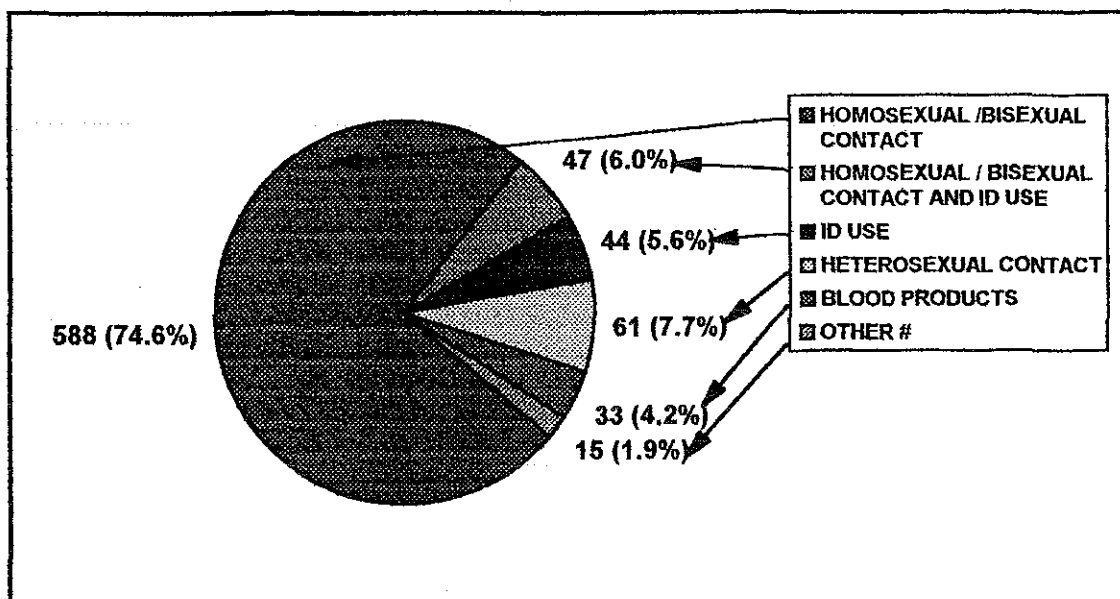


Figure 1.8: Mode of transmission for HIV infection for all cases in Western Australia to 21 September 1993.

(AIDS Bureau 1993, Table 4).

Perinatal transmission or transmission is unknown.

Table 1.7: Estimated number of living HIV and AIDS cases among heterosexuals in Western Australia in 1992 and 1993 # from sexual contact or intravenous drug use.

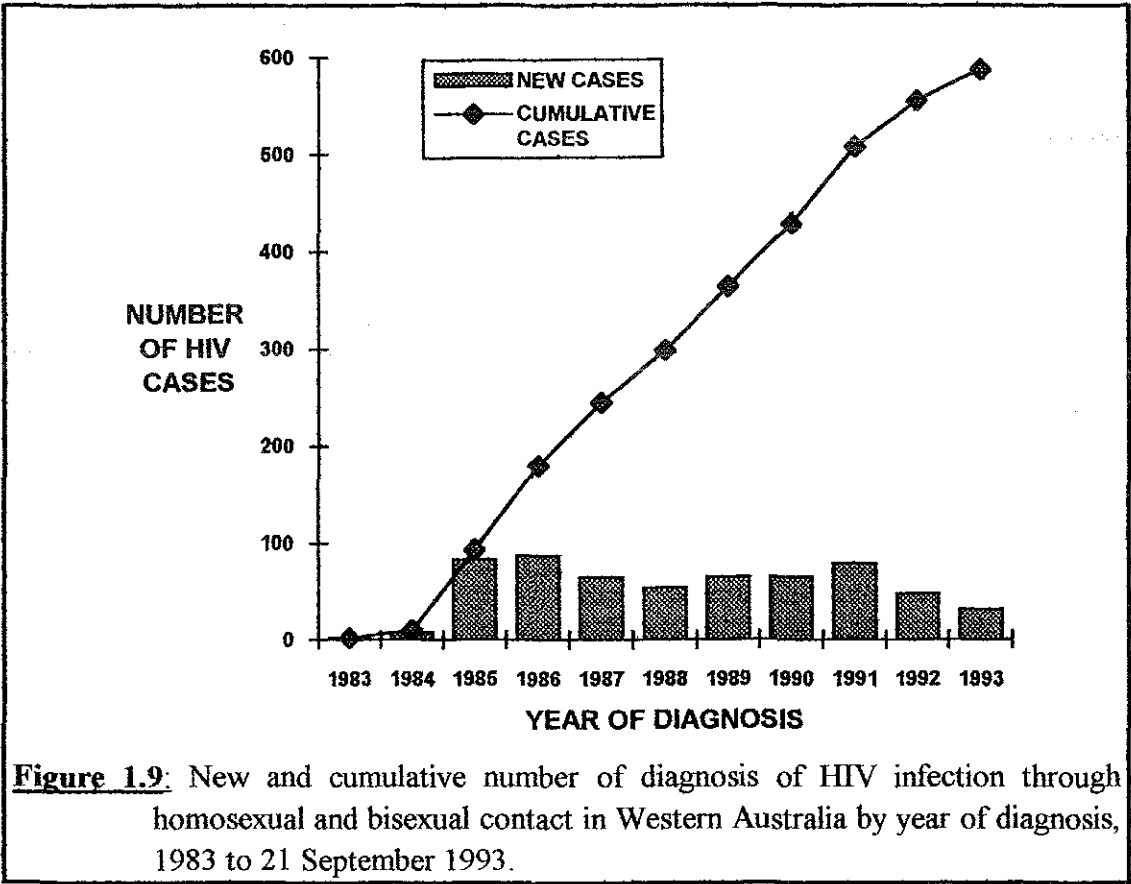
SEX	MODE OF TRANSMISSION	HIV INFECTION		AIDS CASE	
		1992	1993 [#]	1992	1993 [#]
MALE	IVDU	21	20	3	3
	SEXUAL	10	10	2	1
FEMALE	IVDU	20	23	3	4
	SEXUAL	17	18	3	3

(Health Department of Western Australia, 1993, Tables 12 and 15).

(AIDS Bureau, 1993, Tables 1a and 4).

#1993 data is only to 21 September 1993 and is therefore incomplete.

Figures 1.9, 1.10 and 1.11 show the number of newly diagnosed cases of HIV infection in Western Australia due to the main modes of infection of the HIV virus based upon Table 1.6. Homosexual and bisexual contact accounted for the largest number of diagnosed infections, but the number of new cases diagnosed each year due to this mode of transmission has been decreasing. Heterosexual contact has not accounted for a large number of infections, but the number of newly diagnosed cases has been increasing as has been the case for transmission through intravenous drug use.



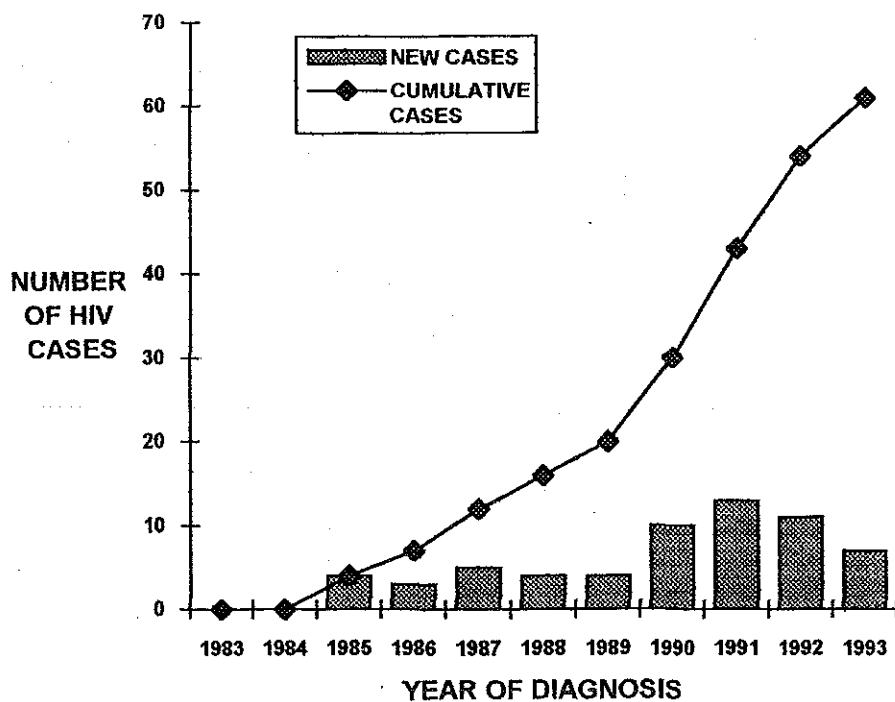


Figure 1.10: New and cumulative number of diagnosis of HIV infection through heterosexual contact in Western Australia by year of diagnosis, 1983 to 21 September 1993.

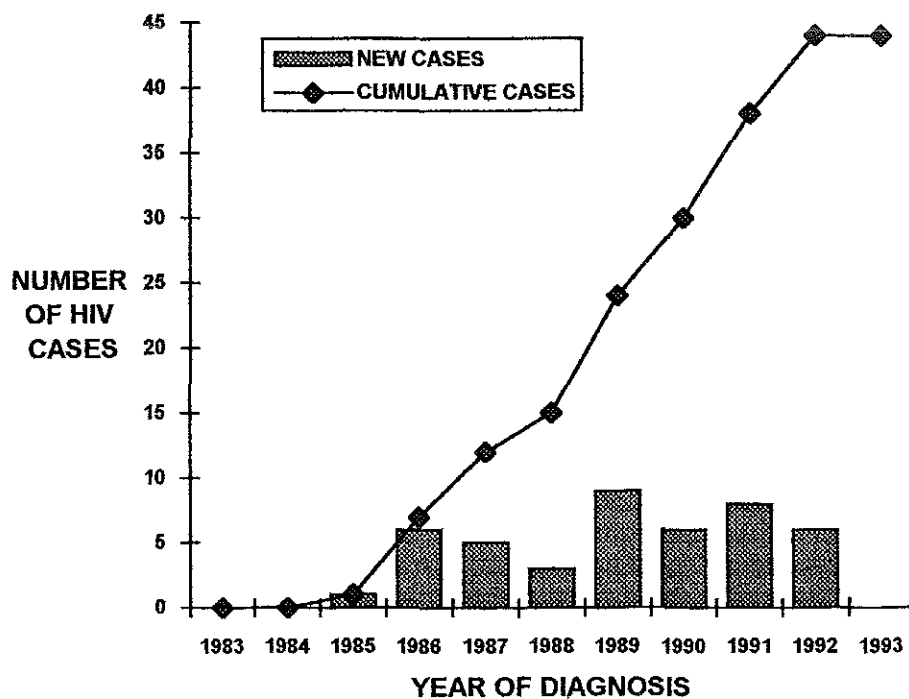


Figure 1.11: New and cumulative number of diagnosis of HIV infection through intravenous drug use in Western Australia by year of diagnosis, 1983 to 21 September 1993.

The pattern of spread for Western Australia can be summarised as follows:

- The initial cases of HIV and AIDS occurred in the early 1980's.
- Homosexual transmission was the main mode of transmission in the early stage of the epidemic.
- Transmission from homosexual contact is decreasing while transmission from heterosexual contact and from IV drug use is increasing.
- Transmission from blood products is under control through screening programs.
- The male to female ratio of HIV infection in Western Australia is approximately 16:1.
- Few perinatal cases have occurred.

2. Mathematical Models

2.1 Background to Epidemic Models

Bernoulli (1700-1782) was the first known individual to use an explicit model to describe an infectious disease, smallpox. His was the first instance where sensitivity analysis was applied to a mathematical model when he made comparisons of the life expectancy without variolation, with variolation being effective, and with death from variolation (Bailey, 1985).

Duvillard followed the same lines as Bernoulli, but using the vaccination instead of variolations for his work. Farr used empirical curve-fitting on data to extrapolate results. These results were inaccurate, but the theory applied provided the correct curve shape for the data (Bailey, 1985).

Hamer was the first to work on the theory that an epidemic depends upon the number of susceptibles at risk and the contact rate between susceptibles and infectious individuals. Ross, studying malaria, derived a simple threshold result which stated that malaria could be eliminated by reducing the mosquito population instead of eliminating the mosquito population altogether. Ross, using differential equations and data manipulation, noted that epidemics were probabilistic and followed a Poisson distribution (Bailey, 1985).

Kermack and Mckendrick produced the 'Threshold Theorem' which applies to an epidemic in a homogeneous population where complete immunity occurs after recovery from infection. The theorem states that there exists a population density threshold value which depends upon the transmission and recovery rates of the particular disease. The theorem also states that an epidemic would only occur if, at

the introduction of an infectious individual into a susceptible population, the density of the susceptibles was above the particular critical threshold level. The effect of the epidemic will be to reduce the density of the susceptibles, through infection, to a level as far below the critical threshold value as initially it was above it. The critical threshold value is the relative removal rate of the disease. The relative removal rate is given by the total removal rate divided by the total infection rate of the disease (Bailey, 1975, 1985).

Mckendrick was the first to use a stochastic model in continuous time with various different classes for susceptibles, infectives, those who die from the disease and those who recover and are immune to the disease. Work continued on the probabilistic nature of epidemics as well as deterministic and stochastic theory and application. Both of these processes were used to account for epidemics that were periodic due to temporary immunity (Bailey, 1985).

2.2 Simple Epidemic Models

The following section on epidemic models of direct contact transmission only was based upon Bailey (1975) and James & Steele (1990). One should first consider a simple model where individuals are either susceptible or infective and the population is a constant size. All infectives are equally infective and are not removed and do not recover from infection. All susceptibles are equally susceptible and the population mixes homogeneously. The number of susceptibles at time t is represented by $x(t)$ and the number of infectives at time t is represented by $y(t)$.

At the start of the epidemic, there are N susceptibles and 1 infective. The number of new infectives is dependent upon the number of susceptibles and the number of

infectives. The transmission rate β , is a combination of the proportion of susceptibles coming into contact with an infective and the proportion of these susceptibles which become infected through this contact. The number of new infectives, δy during the time interval $[t, t + \delta t]$ is :

$$\delta y = \beta x(t) y(t) \delta t .$$

As the limit $\delta t \rightarrow 0$, the number of infectives at time t is given by :

$$\frac{dy}{dt} = \beta xy$$

or $\frac{dy}{dt} = \beta y(N - y + 1)$ as $x + y = N + 1$.

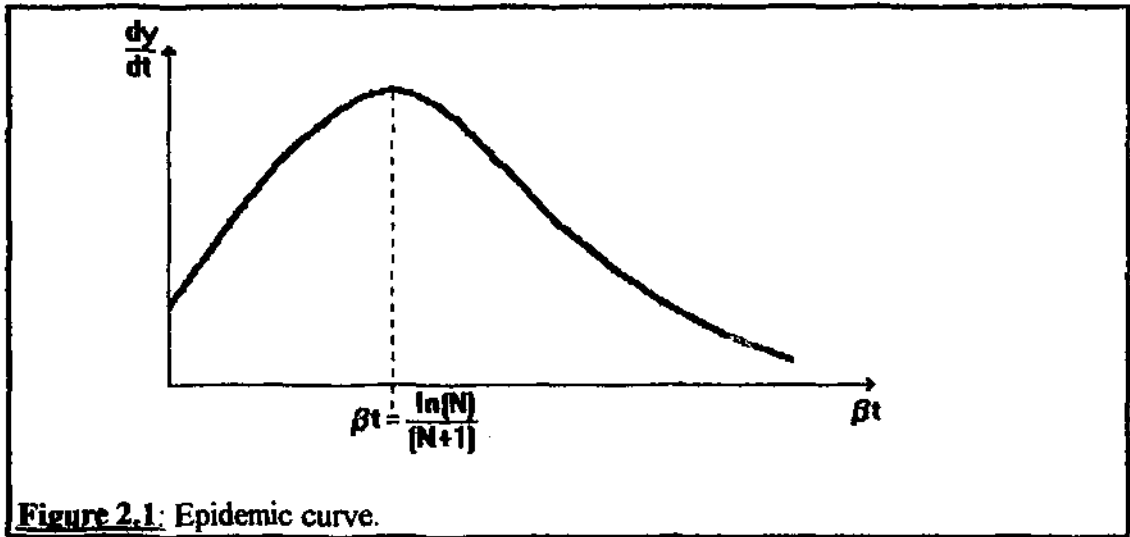


Figure 2.1: Epidemic curve.

The last equation can be rewritten as :

$$\int_{y_0}^y \frac{dy}{y(N - y + 1)} = \int_0^t \beta dt$$

and integrated to give :

$$y(t) = \frac{N + 1}{1 + ne^{-(n+1)\beta t}} ,$$

known as the logistic equation. A plot of the rate at which new infections occur is referred to as an epidemic curve, Figure 2.1. This curve has a maximum when :

$$\beta x = \frac{\ln(N)}{N+1}$$

and $x = y = \frac{1}{2}(N+1).$

The simple model is described by Figure 2.2, a flow diagram of individuals in the population and by equations 2.1 below.

$$\begin{aligned} \frac{dx}{dt} &= -\beta xy \\ \frac{dy}{dt} &= \beta xy \end{aligned} \quad (2.1)$$

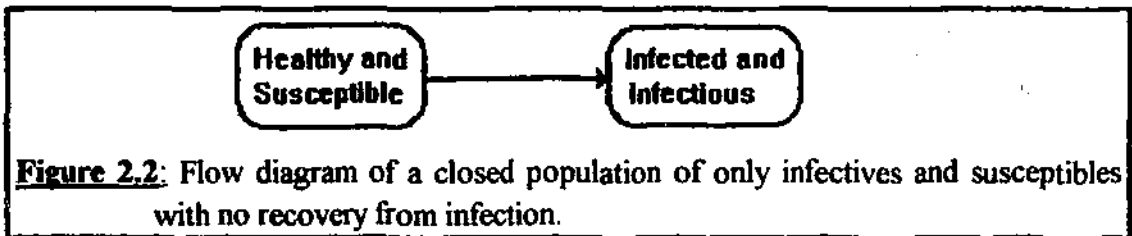


Figure 2.2: Flow diagram of a closed population of only infectives and susceptibles with no recovery from infection.

The next step in the model is to incorporate recovery from infection. Those individuals which recover are assumed to return to being susceptible. The model for a closed population of only susceptibles and infectives with recovery from infection is given below and a flow diagram of individuals in the population is given by Figure 2.3.

$$\begin{aligned} \frac{dx}{dt} &= -\beta xy + \gamma y \\ \frac{dy}{dt} &= \beta xy - \gamma y \end{aligned} \quad (2.2)$$

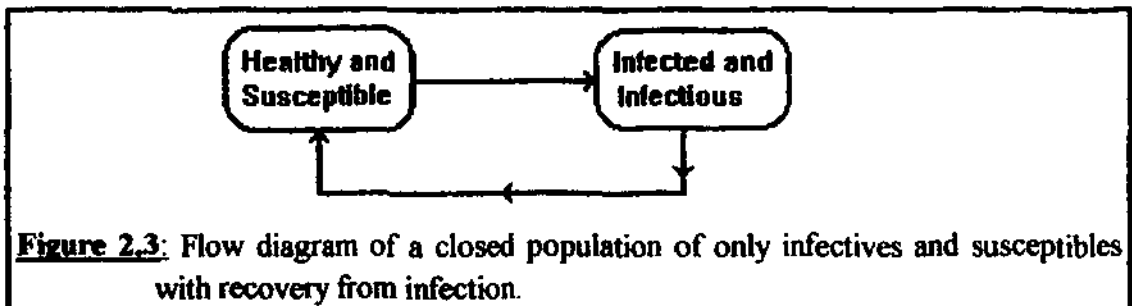
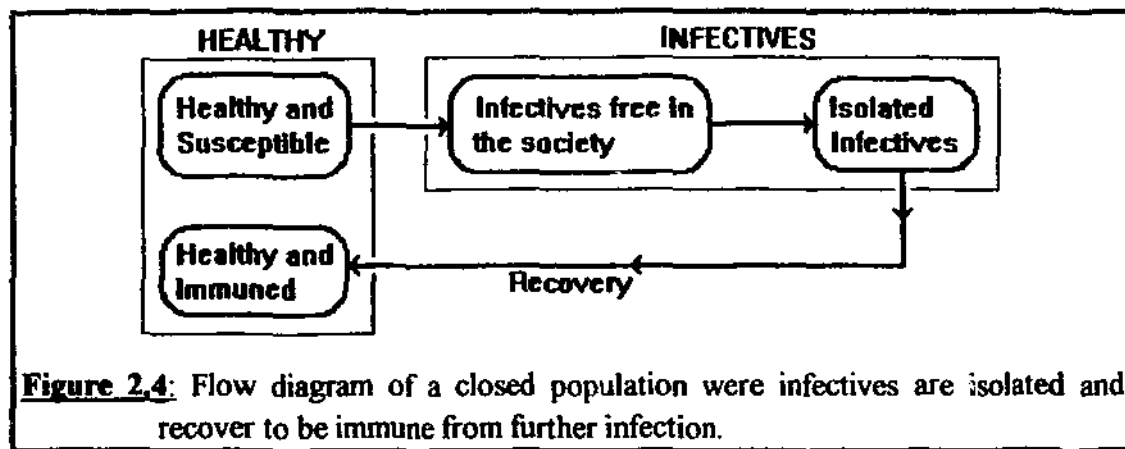


Figure 2.3: Flow diagram of a closed population of only infectives and susceptibles with recovery from infection.

The recovery rate is given by γ and the number of individuals which recover is proportional to the total number of infectives. Again the size of the susceptibles is N and 1 infective is introduced at the start of the epidemic. The disease spreads through the population and the number of infectives reaches an equilibrium of $N + 1 - \frac{\gamma}{\beta}$, and the number of susceptibles reaches the equilibrium point of $\frac{\gamma}{\beta}$ only if the number of susceptibles was greater than $\frac{\gamma}{\beta}$ when the first infective was introduced into the population. If the number of susceptibles was not greater than the relative removal rate, $\frac{\gamma}{\beta}$ for the particular disease, then the disease would not spread through the population. As the number of infectives and susceptibles reaches a stable equilibrium, the disease is referred to as endemic.

Rather than the infectives recovering and returning to the susceptible population to be re-infected, the following model allows for infectives to be immune after having recovered. The model for a closed population of only susceptibles and infectives with recovery and immunity from infection is given below and where the number of individuals which are immune at time t is given by $z(t)$.

$$\begin{aligned}\frac{dx}{dt} &= -\beta xy \\ \frac{dy}{dt} &= \beta xy - \alpha y \\ \frac{dz}{dt} &= \alpha y\end{aligned}\quad (2.3)$$



The removal rate is given by α and the number of infectives being immune is still proportional to the total number of infectives. At the start of the epidemic there are again N susceptibles and 1 infective. The epidemic will only occur if the number of susceptibles at the start of the epidemic is greater than the relative removal rate, $\frac{\alpha}{\beta}$ of the particular disease. The following AIDS models are based upon equations 2.3 with $z(t)$ being infectives which have progressed to AIDS and are considered no longer infectious.

2.3 Early HIV/AIDS Models

One of the earliest models described below, was developed by Anderson (1988a) and is a very simple homogeneous mixing model describing the epidemic in a closed population. This model does not allow migration or recruitment into the population and removal is only by death from AIDS. In a homogeneous model individuals choose on average the same number of partners. The rate of change in the number of susceptibles $dX(t)/dt$ is given by the number of susceptibles infected. This is determined by the proportion of the population infected Y/N , the average number of new sexual partners of an individual c , the probability of infection from a single sexual contact β , and the number of susceptibles in the population. The term 'single sexual

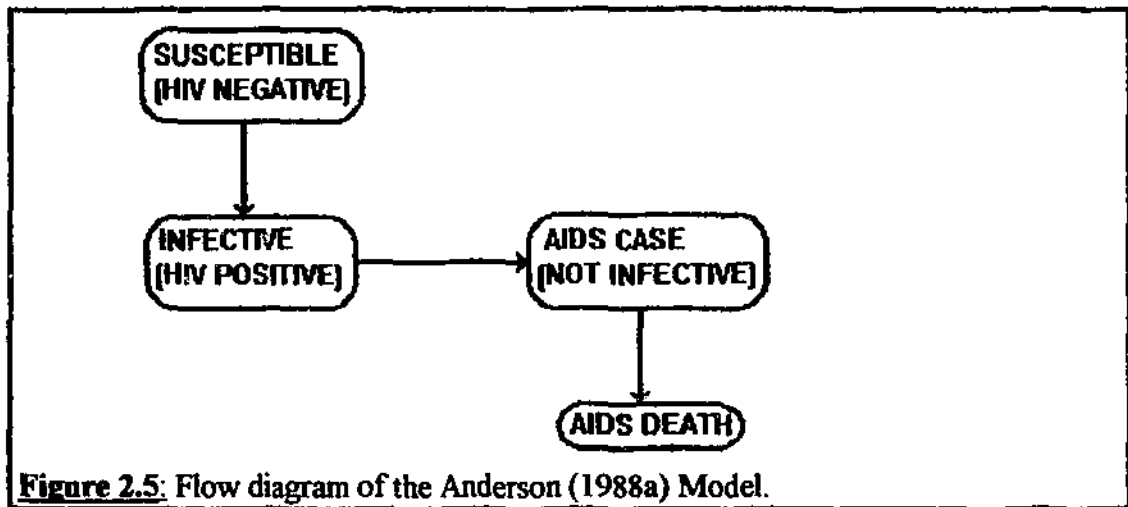
contact' is often used and generally refers to a single sexual partnership rather than a single act of sexual intercourse. If the term used refers to a single sexual act then another parameter is required which represents the average frequency of sexual intercourse. Studies have shown that the frequency of sexual contact has little effect on the probability of transmission of the HIV virus (Anderson, 1988b), (Anderson, and Medley, 1988).

The rate of change in the number of infectives, $dY(t)/dt$ is determined by the number of susceptibles infected minus the number of infectives which have progressed to AIDS. The number of infectives progressing to AIDS is determined by the removal rate ν , where $1/\nu$ is the incubation period for the HIV virus. The rate of change in the population $dN(t)/dt$ is determined by the deaths from AIDS, given by α , the mortality rate and the number of AIDS cases, A . The rate of change in the number of AIDS cases, $dA(t)/dt$ is determined by the number of new AIDS cases from infectives progressing to AIDS minus deaths from AIDS. The Anderson (1988a) model is given by:

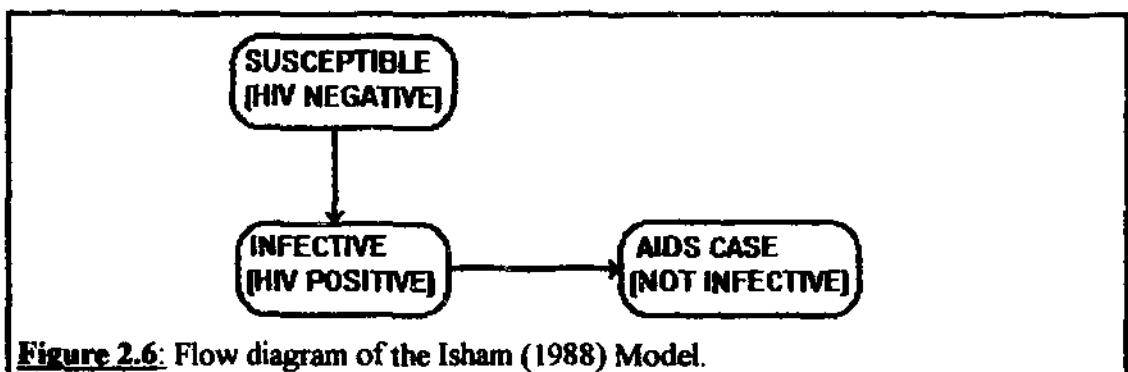
$$\begin{aligned}dX(t)/dt &= -\beta cXY/N \\dY(t)/dt &= \beta cXY/N - \nu Y \\dA(t)/dt &= \nu Y - \alpha A \\dN(t)/dt &= -\alpha A\end{aligned}\tag{2.4}$$

where

$$N(t) = X(t) + Y(t) + A(t)$$



A later model was developed by Isham (1988) and is again a closed population, but this model is a heterogeneous model as it allows for differences in choices of new sexual partners for males, c_m and females, c_f . This model also allows for differences in the probability of infection from sexual contact, β for males and females and different incubation periods for males, $1/\nu_m$ and females, $1/\nu_f$. The probability of a male being infected by a female is given by β_f and the probability of a female being infected by a male is given by β_m . The equations are very similar to the previous model except for the susceptibles and infectives being divided into male, X_m and Y_m respectively, and female, X_f and Y_f respectively, groups. The Isham (1988) model is given by:



$$\begin{aligned}
\frac{dX_m(t)}{dt} &= -\beta_f c_m X_m \frac{Y_f}{N_f} \\
\frac{dX_f(t)}{dt} &= -\beta_m c_f X_f \frac{Y_m}{N_m} \\
\frac{dY_m(t)}{dt} &= \beta_f c_m X_m \frac{Y_f}{N_f} - \nu_m Y_m \\
\frac{dY_f(t)}{dt} &= \beta_m c_f X_f \frac{Y_m}{N_m} - \nu_f Y_f \\
\frac{dA_m(t)}{dt} &= \nu_m Y_m \\
\frac{dA_f(t)}{dt} &= \nu_f Y_f \\
N_m(t) &= X_m(t) + Y_m(t) + A_m(t) \\
N_f(t) &= X_f(t) + Y_f(t) + A_f(t) \\
N(t) &= N_m(t) + N_f(t)
\end{aligned}
\tag{2.5}$$

This model is a heterogeneous model and therefore allows for differences between males and females in partner choices and probability of transmission. The model does not, however, allow for migration and recruitment into the population, something which occurs in reality. The above Isham model only deals with transmission among the heterosexual population through sexual contact. The model required needs to incorporate transmission of the HIV virus through intravenous drug use.

2.4 Proposed Model

The proposed model is based on a model developed by Comiskey (1992) where the virus is transmitted through heterosexual contact and IV drug use. This is not a closed population, and therefore includes migration and recruitment to and from the population. Recruitment into the population is through starting drugs or sexual activity. Migration from the population can be from death, either from AIDS or from

other causes, or from no longer using drugs or no longer being sexually active. The population consists of heterosexuals in Western Australia who either use intravenous drugs or are sexually active.

The population is divided into three classes, susceptible, $X(t)$, infective, $Y(t)$, and AIDS sufferers, $A(t)$. These classes are further divided into four groups, (1) male IV drug users, (2) female IV drug users, (3) male non-IV drug users and (4) female non-IV drug users. The following is a list of all the variables and parameters used in the model.

$X_i(t)$: Number of susceptibles at time t from group i .

$Y_i(t)$: Number of infectives at time t from group i .

$A_i(t)$: Number of AIDS cases at time t from group i .

$P_i(t)$: Total population of group i at time t .

β_{fm} : Probability of transmission of the HIV virus through sexual contact from a female infective to a male susceptible.

β_{mf} : Probability of transmission of the HIV virus through sexual contact from a male infective to a female susceptible.

$\hat{\beta}$: Probability of transmission of the HIV virus through the sharing of needles.

Λ_i : Recruitment rate into group i .

μ_i : Migration rate from group i .

η_i : Average number of needle sharing 'partners' that an individual from group i has in a year ($i = 1, 2$).

c_{ij} : Average number of sexual partners an individual from group i chooses from group j in a year.

α : Rate of progression of the HIV virus (Mean incubation period is $1/\alpha$).

δ : Death rate from AIDS.

The rate of change in the number of susceptible male IV drug users $dX_1(t)/dt$ is determined by the recruitment rate into the group Λ_1 , minus susceptibles being infected by needle sharing or from sexual contact and migration of individuals from this group μ_1 . The rate of change in the number of susceptible female IV drug users $dX_2(t)/dt$ is determined by the recruitment rate into the group Λ_2 , infection from needle sharing and sexual contact and migration from the group μ_2 .

$$\begin{aligned} \frac{dX_1(t)}{dt} = & \Lambda_1 - \eta_1 \hat{\beta} X_1(t) \left[\frac{Y_1(t) + Y_2(t)}{P_1(t) + P_2(t)} \right] - c_{12} \beta_{fm} X_1(t) \frac{Y_2(t)}{P_2(t)} - c_{14} \beta_{fm} X_1(t) \frac{Y_4(t)}{P_4(t)} \\ & - \mu_1 X_1(t) \end{aligned} \quad (2.6.1)$$

$$\begin{aligned} \frac{dX_2(t)}{dt} = & \Lambda_2 - \eta_2 \hat{\beta} X_2(t) \left[\frac{Y_1(t) + Y_2(t)}{P_1(t) + P_2(t)} \right] - c_{21} \beta_{mf} X_2(t) \frac{Y_1(t)}{P_1(t)} - c_{23} \beta_{mf} X_2(t) \frac{Y_3(t)}{P_3(t)} \\ & - \mu_2 X_2(t) \end{aligned} \quad (2.6.2)$$

Infection from sharing needles is given by $\hat{\beta}$, the probability of infection from a single needle sharing act, η_j , the average number of times an individual from group j shares a needle, the number of susceptibles in group j , $X_j(t)$ and the proportion of drug users which are infected, $\{Y_1(t) + Y_2(t)\} / \{P_1(t) + P_2(t)\}$.

The rate of change in the number of susceptible male non-IV drug users $dX_3(t)/dt$ is determined by the recruitment rate into the group Λ_3 , infection from sexual contact and the migration rate from the group μ_3 . The rate of change in the number of susceptible female non-IV drug users $dX_4(t)/dt$ is determined by the recruitment rate Λ_4 , infection from sexual contact and migration from the group μ_4 .

$$\frac{dX_3(t)}{dt} = \Lambda_3 - \rho_3 c_{32} \beta_{fm} X_3(t) \frac{Y_2(t)}{P_2(t)} - c_{34} \beta_{fm} X_3(t) \frac{Y_4(t)}{P_4(t)} - \mu_3 X_3(t) \quad (2.6.3)$$

$$\frac{dX_4(t)}{dt} = \Lambda_4 - \rho_4 c_{41} \beta_{mf} X_4(t) \frac{Y_1(t)}{P_1(t)} - c_{43} \beta_{mf} X_4(t) \frac{Y_3(t)}{P_3(t)} - \mu_4 X_4(t) \quad (2.6.4)$$

Infection from sexual contact is divided into contact with IV drug users or non IV drug users. Only a proportion of non IV drug users will mix with IV drug users and this proportion represented by ρ_j , for group j , where j is 3 or 4, male and female non-IV drug users respectively. The mean number of sexual partners an individual from group i chooses from group j is represented by c_{ij} . For example c_{14} is the mean number of female non-IVDU sexual partners which a male intravenous drug user has in a year. The probability of infection of a male susceptible from sexual contact with a female infective partner is given by β_{fm} and the probability of a female susceptible being infected by a male infective partner through a sexual contact is given by β_{mf} . The proposed model is given by:

$$\begin{aligned} \frac{dX_1(t)}{dt} = & \Lambda_1 - \eta_1 \hat{\beta} X_1(t) \left[\frac{Y_1(t) + Y_2(t)}{P_1(t) + P_2(t)} \right] - c_{12} \beta_{fm} X_1(t) \frac{Y_2(t)}{P_2(t)} - c_{14} \beta_{fm} X_1(t) \frac{Y_4(t)}{P_4(t)} \\ & - \mu_1 X_1(t) \end{aligned} \quad (2.6.1)$$

$$\begin{aligned} \frac{dX_2(t)}{dt} = & \Lambda_2 - \eta_2 \hat{\beta} X_2(t) \left[\frac{Y_1(t) + Y_2(t)}{P_1(t) + P_2(t)} \right] - c_{21} \beta_{mf} X_2(t) \frac{Y_1(t)}{P_1(t)} - c_{23} \beta_{mf} X_2(t) \frac{Y_3(t)}{P_3(t)} \\ & - \mu_2 X_2(t) \end{aligned} \quad (2.6.2)$$

$$\frac{dX_3(t)}{dt} = \Lambda_3 - \rho_3 c_{32} \beta_{fm} X_3(t) \frac{Y_2(t)}{P_2(t)} - c_{34} \beta_{fm} X_3(t) \frac{Y_4(t)}{P_4(t)} - \mu_3 X_3(t) \quad (2.6.3)$$

$$\frac{dX_4(t)}{dt} = \Lambda_4 - \rho_4 c_{41} \beta_{mf} X_4(t) \frac{Y_1(t)}{P_1(t)} - c_{43} \beta_{mf} X_4(t) \frac{Y_3(t)}{P_3(t)} - \mu_4 X_4(t) \quad (2.6.4)$$

$$\begin{aligned} \frac{dY_1(t)}{dt} = & \eta_1 \hat{\beta} X_1(t) \left[\frac{Y_1(t) + Y_2(t)}{P_1(t) + P_2(t)} \right] + c_{12} \beta_{fm} X_1(t) \frac{Y_2(t)}{P_2(t)} + c_{14} \beta_{fm} X_1(t) \frac{Y_4(t)}{P_4(t)} \\ & - (\alpha + \mu_1) Y_1(t) \end{aligned} \quad (2.6.5)$$

$$\begin{aligned} \frac{dY_2(t)}{dt} = & \eta_2 \hat{\beta} X_2(t) \left[\frac{Y_1(t) + Y_2(t)}{P_1(t) + P_2(t)} \right] + c_{21} \beta_{mf} X_2(t) \frac{Y_1(t)}{P_1(t)} + c_{23} \beta_{mf} X_2(t) \frac{Y_3(t)}{P_3(t)} \\ & - (\alpha + \mu_2) Y_2(t) \end{aligned} \quad (2.6.6)$$

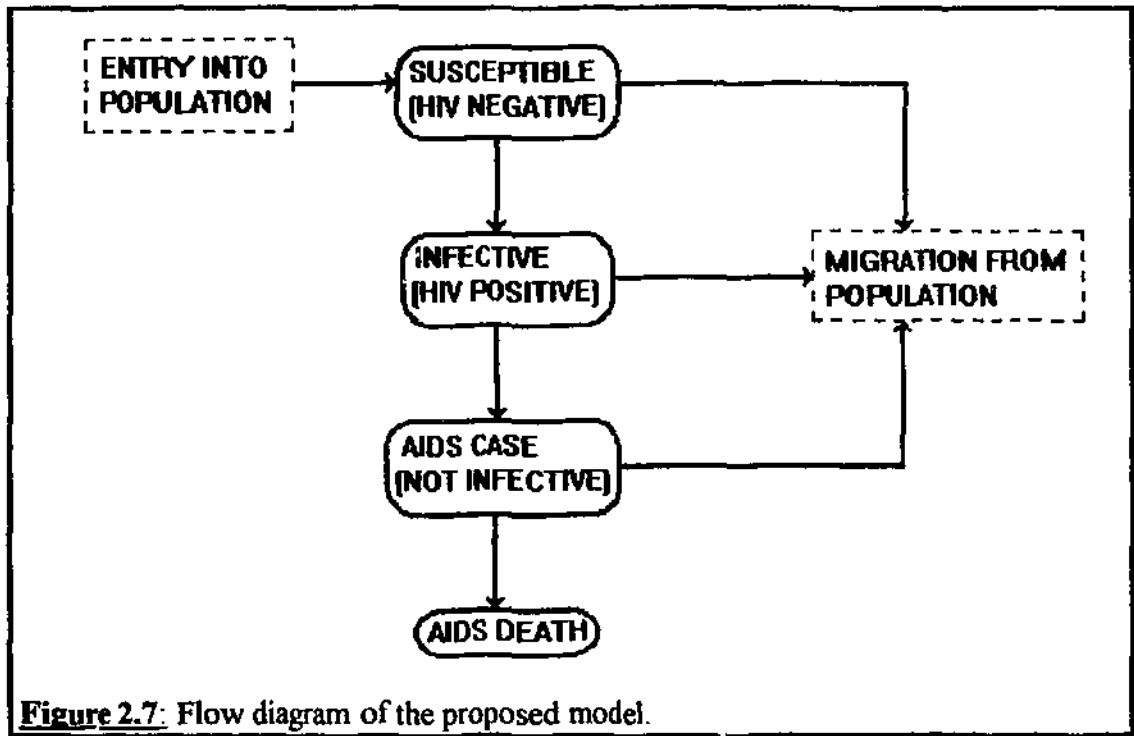
$$\frac{dY_3(t)}{dt} = \rho_3 c_{32} \beta_{fm} X_3(t) \frac{Y_2(t)}{P_2(t)} + c_{34} \beta_{fm} X_3(t) \frac{Y_4(t)}{P_4(t)} - (\alpha + \mu_3) Y_3(t) \quad (2.6.7)$$

$$\frac{dY_4(t)}{dt} = \rho_4 c_{41} \beta_{mf} X_4(t) \frac{Y_1(t)}{P_1(t)} + c_{43} \beta_{mf} X_4(t) \frac{Y_3(t)}{P_3(t)} - (\alpha + \mu_4) Y_4(t) \quad (2.6.8)$$

$$\frac{dA_i(t)}{dt} = \alpha Y_i(t) - (\delta + \mu_i) A_i(t) \quad (2.6.9)$$

$$P_i(t) = X_i(t) + Y_i(t) + A_i(t) \quad (2.6.10)$$

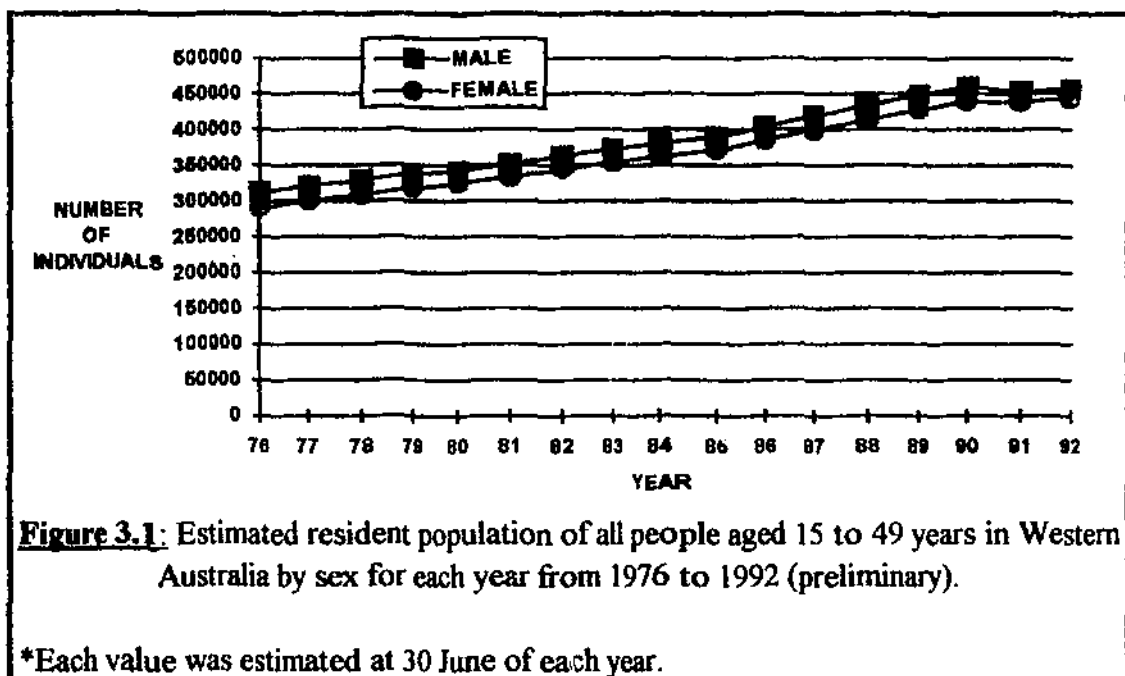
for $i=1,2,3,4$



3. Model Parameters

3.1 Population Sizes

The differential equations for the model of the spread of the HIV virus requires estimates of the population sizes of each risk group. The total number of males and females in Western Australia between the ages of 15 and 49, has been increasing each year, from 1976 to 1992, except for 1991 when there was a decrease of about 8000 people from the previous year, as shown in Figure 3.1 which is based upon estimated resident population figures (Australian Bureau of Statistics, 1982, 1987 and 1993). The increase in the size of the population needs to be included in the model.



The age range of 15 to 49 years is based upon Corniskey (1992) and Rollins (1989). These state that the mean age for first sexual intercourse for females is approximately 15 years. Most Australian studies have been targeted at the younger population,

which is why the duration of being sexually active for heterosexuals for 35 years is based upon Comiskey (1992).

Loxley, et al, 1991, page 363, state that "... there may be as many as 80000 IVUDs in Australia, ..." and " In one study it was found that 1 to 3% of people aged 16-24 had injected themselves with drugs within the last 12 months ...". The 1-3% of 16-24 year olds gives a Western Australian estimate for the number of IV drug users in 1985 to be between 2183 and 6548 people and in 1990 to be between 2377 and 7131 people. The male to female ratio of 63.3 : 36.7 of respondents in Perth to the ANAIDUS study is used for the ratio of male to female IVDUs in Western Australia.

The population of non-IVDUs is the total population aged 15-49 minus those using intravenous drugs. The population estimates for male and female IVDUs and non-IVDUs is given in Table 3.1. These estimates are for the total number of males and females including bisexuals and homosexuals.

Table 3.1: Estimated number of all male and female IVDUs and non-IVDUs in Western Australia in 1985 and 1992.				
% PEOPLE 16-24 YEARS USING IVDs	MALE IVDU	FEMALE IVDU	MALE NON-IVDU	FEMALE NON-IVDU
1985 (1%)	1382	801	388536	371213
1985 (2%)	2764	1602	387154	370412
1985 (3%)	4146	2402	385772	369612
1992 (1%)	1505	872	455920	443004
1992 (2%)	3009	1745	454416	442131
1992 (3%)	4514	2617	452911	441259

The sexual orientation of the respondents to the ANAIDUS study is given in Table 3.2, which is based on table 49 on page 62, Australian National AIDS and Injecting Drug Use Study, 1992 and table 22 on page 68, Loxley, et al, 1992 .

Table 3.2: Sexual orientation of Perth respondents to the ANAIDUS study.		
ORIENTATION OVER LAST 5 YEARS	MALE	FEMALE
HETEROSEXUAL	82 (89.1%)	40 (74.1%)
BISEXUAL	6 (6.5%)	13 (24.1%)
HOMOSEXUAL	4 (4.4%)	1 (1.8%)
TOTAL	92 (100%)	54 (100%)
CURRENT SEXUAL ORIENTATION	MALE	FEMALE
HETEROSEXUAL	81 (91.0%)	47 (85.5%)
BISEXUAL	1 (1.1%)	7 (12.7%)
HOMOSEXUAL	7 (7.9%)	1 (1.8%)
TOTAL	89 (100%)	55 (100%)

The percentage of heterosexual females appears too low compared to the percentage of heterosexual males in the same study. This low percentage could be due to a bias in the survey sample. The sexual orientation from the last 5 years will be used instead of current sexual orientation as it accounts for a reasonable length of time. Table 3.3 presents the population sizes for male and female IVDUs and non-IVDUs based upon the sexual orientation of the Perth respondents to the ANAIDUS survey. The population sizes for 1985 correspond to the initial populations of the risk groups at the start of the epidemic, $P_i(0)$ for $i = 1$ to 4.

Table 3.3: Population sizes for heterosexual male and female IVDUs and non-IVDUs in 1985.				
% OF PEOPLE 16-24 YEARS USING IVDs	MALE IVDU	FEMALE IVDU	MALE NON-IVDU	FEMALE NON-IVDU
	$P_1(0)$	$P_2(0)$	$P_3(0)$	$P_4(0)$
(1%)	1231	594	346186	275069
(2%)	2463	1187	344954	274475
(3%)	3694	1780	343723	273882

The annual increase in the size of the male heterosexual population from 1985 to 1992 is 8593 males per year and the annual increase in the size of the female population

from 1985 to 1992 is 7607 females per year when it is assumed that 2% of people aged 16 to 24 take up intravenous drugs. The annual increase for each population group is shown in Table 3.4.

Table 3.4: Annual increase in population sizes of heterosexual male and female IVDUs and non-IVDUs from 1985 to 1992.				
% OF PEOPLE 16-24 YEARS USING IVDs	MALE IVDU	FEMALE IVDU	MALE NON-IVDU	FEMALE NON-IVDU
(1%)	16	7	8577	7600
(2%)	31	15	8562	7592
(3%)	47	23	8546	7584

3.2 Recruitment and Migration Rates

Assuming the duration of being sexually active is 35 years for both male and female heterosexual non-IVDUs, age 15 to 49, the migration rate for both groups is 1/35 or 0.02857 per year. The migration rate for male and female IVDUs is based upon the reciprocal of the mean duration of their drug using habit as estimated by Comiskey (1992). The respondents to the ANAIDUS study were not asked how long they were using drugs, so the estimate is based upon the average age of the respondents at the time of the survey minus the average age with which they started taking intravenous drugs. The duration of the drug taking habit is used for IVDUs rather than the duration of being sexually active, as the probability of transmission of the HIV virus from sharing needles is greater than that for sexual contact among heterosexuals.

The mean age of first injecting drugs for the total Perth sample was 19.0 years with a 95% confidence interval of 18.4 - 19.6. The mean age of all the Perth respondents at the time of the survey was 28.6 years with a confidence interval 27.7 - 29.5 (Australian National AIDS and Injecting Drug Use Study, 1992) (Loxley, et al, 1992). At the time of the survey, some respondents were no longer taking drugs. The mean

age for respondents giving up drugs is therefore assumed to be 28.6 years and the duration of the drug taking habit for all respondents is estimated to be 9.6 years. The migration rate per year is therefore $1/9.6$ or 0.10417 for both male and female IVDUs.

Table 3.5: Migration rates for each risk group.		
RISK GROUP	PARAMETER	MIGRATION RATE PER YEAR
MALE IVDU	μ_1	0.10417
FEMALE IVDU	μ_2	0.10417
MALE NON-IVDU	μ_3	0.02857
FEMALE NON-IVDU	μ_4	0.02857

If it assumed that without the virus the population sizes of male and female IVDUs and non-IVDUs remains constant, then the recruitment rate equals the yearly migration rate. As the population sizes are not constant, the recruitment rate per year for each group is estimated using the migration rate multiplied by the initial population size in 1985 plus the annual increase shown in Table 3.4 plus the migration rate multiplied by the annual increase.

Table 3.6: Recruitment rates for each risk group.				
% OF PEOPLE 16-24 YEARS USING IVDs	MALE IVDU	FEMALE IVDU	MALE NON-IVDU	FEMALE NON-IVDU
	Λ_1	Λ_2	Λ_3	Λ_4
1%	146	70	18713	15676
2%	291	140	18662	15651
3%	437	211	18611	15626

In the case of male non-IVDUs aged 16 to 49 years, the population in 1985, the start of the epidemic, was 344954 individuals, assuming that 2% of people 16 to 24 years take up IV drugs. In 1986 this is reduced by 9856 through individuals migrating out of the population. In order for the model to be representative of the situation in Western Australia, the male non-IVDU population needs to have an increase in the number of individuals each year of about 8562, the annual increase. As migration will

occur in 1987 from those which were recruited through the annual increase, this migration is also added to the total recruitment rate. The recruitment rate for the male non-IVDU group is therefore 8562 (annual increase) plus 9856 (initial migration) plus 244 (migration of the annual increase). Table 3.6 represents the recruitment rate per year for each risk group.

3.3 Choice of Partners

The heterosexual female respondents of the ANAIDUS study had on average 8.3 male partners in a year with a 95% confidence interval of 4.7 - 11.8 partners. Of the regular partners in a year, 84.4% were intravenous drug users (Australian National AIDS and Injecting Drug Use Study, 1992). This percentage will be used for all partners, therefore of the 8.3 partners in a year, 7.0052 are IVDUs and 1.2948 are non-IVDUs.

The heterosexual male respondents of the ANAIDUS study had on average 4.4 female partners in a year. Of the regular partners of the male IVDU respondents, 62.2% were intravenous drug users (Australian National AIDS and Injecting Drug Use Study, 1992). This percentage is used for all partners, therefore of the 4.4 partners in a year, 2.7368 are IVDUs and 1.6632 are non-IVDUs. Table 3.7 represents the partner choices for a year for heterosexual IVDUs in Perth and Sydney combined.

Table 3.7: Partner choices for heterosexual male and female IVDUs for a year in Perth and Sydney.					
IVDU GROUP	NUMBER OF PARTNERS				
	MALE IVDU	FEMALE IVDU	MALE NON-IVDU	FEMALE NON-IVDU	TOTAL
MALE	-	2.7368	-	1.6632	4.4
FEMALE	7.0052	-	1.2948	-	8.3

The female respondents of the Perth study had on average 7.8 partners in a year. The respondents to the Perth study were not just heterosexuals, they also included bisexuals and homosexuals and the sexuality of the respondents was not taken into account in this question. Of the regular partners in a year, 92.7% were intravenous drug users (Marsh, et al, 1991). This percentage is used for all partners, therefore of the 7.8 partners in a year, 7.2306 are IVDUs and 0.5694 are non-IVDUs.

The male respondents of the Perth study had on average 4.6 partners in a year. Of the regular partners of the male IVDU respondents, 64.5% were intravenous drug users (Marsh, et al, 1991). This percentage is used for all partners, therefore of the 4.6 partners in a year, 2.967 are IVDUs and 1.633 are non-IVDUs. Table 3.8 represents the partner choices for a year for all male and female IVDUs in Perth which are not prostitutes.

Table 3.8: Partner choices for all male and female IVDUs for a year in Perth (excluding prostitutes).						
IVDU GROUP		NUMBER OF PARTNERS				TOTAL
		IVDU		NON-IVDU		
		<i>i</i> = 1	<i>i</i> = 2	<i>i</i> = 3	<i>i</i> = 4	
MALE	<i>c</i> _{1<i>i</i>}	-	2.967	-	1.633	4.6
FEMALE	<i>c</i> _{2<i>i</i>}	7.2306	-	0.5694	-	7.8

Tables 3.7 and 3.8 show that the total number of partners in a year for IVDU heterosexuals in Perth and Sydney combined is similar to the total number of partners in a year for all intravenous drug users in Perth. The number of partners in a year for heterosexual IVDUs in Perth should be lower than that for the total in Table 3.8, as homosexuals and bisexuals tend to have a higher number of partners in a year than heterosexuals (Australian National AIDS and Injecting Drug Use Study, 1991, 1992). As Table 3.8 incorporates the total number of partners in a year for Perth IVDUs and the percentage of regular partners being IVDU for both male and female respondents,

these partner choices will be used in the model. The percentage of regular partners being IVDUs does not depend upon the sexual orientation of the respondent, but is greater for female respondents than for male respondents.

Table 3.9: Proportion of the sexually active population that are intravenous drug users.

% OF PEOPLE 16 - 24 YEARS USING IVDs	MALE IVDUs	FEMALE IVDUs
1985 (1%)	0.003543	0.002155
1985 (2%)	0.007089	0.004306
1985 (3%)	0.010633	0.006457
1992 (1%)	0.003290	0.001964
1992 (2%)	0.006578	0.003931
1992 (3%)	0.009868	0.005895
PROPORTION TO BE USED (1%)	0.003	0.002
PROPORTION TO BE USED (2%)	0.007	0.004
PROPORTION TO BE USED (3%)	0.010	0.006

Table 3.10: Partner choices for male and female non-IVDUs for a year.

NON-IVDU GROUP		NUMBER OF PARTNERS				TOTAL
		MALE IVDU	FEMALE IVDU	MALE NON-IVDU	FEMALE NON-IVDU	
		$i = 1$	$i = 2$	$i = 3$	$i = 4$	
(*1) MALE	c_{3j}	-	0.0138	-	4.5862	4.6
FEMALE	c_{4j}	0.0156	-	7.7844	-	7.8
(*2) MALE	c_{3j}	-	0.0322	-	4.5678	4.6
FEMALE	c_{4j}	0.0312	-	7.7688	-	7.8
(*3) MALE	c_{3j}	-	0.0460	-	4.554	4.6
FEMALE	c_{4j}	0.0468	-	7.7532	-	7.8
(*1) 1% of people aged 16 to 24 years taking up intravenous drugs.						
(*2) 2% of people aged 16 to 24 years taking up intravenous drugs.						
(*3) 3% of people aged 16 to 24 years taking up intravenous drugs.						

Since the ANAIDUS study was only targeting drug users, the number of partners which a non-IVDU has in a year is assumed to be equal to that of the IVDUs. A heterosexual female is therefore assumed to have an average of 7.8 partners in a year and a heterosexual male is assumed to have an average of 4.6 partners in a year based

upon Table 3.8. The proportion of IVDUs in the sexually active population of people aged 15 to 49 years is used as the proportion of partners of non-IVDUs who are intravenous drug users. Table 3.9 represents the proportion of IVDUs in the population for males and females and Table 3.10 represents the partner choices for male and female non-IVDUs for the year based upon the proportions in Table 3.9.

3.4 Other Parameters

Only a proportion of non-IVDUs will have contact with intravenous drug users and this has been incorporated into the model using the estimated number of partners of the respondents to the ANAIDUS survey in Perth. The 82 heterosexual male respondents had contact with an estimated total of 377 females of which 35.5% or 134 were non-IVDUs (Tables 3.2 and 3.8). The 40 heterosexual female respondents to the ANAIDUS survey in Perth had contact with an estimated total of 312 males within the year of which 7.3% or 23 were non-IVDUs (Tables 3.2 and 3.8). The minimum proportion of non-IVDUs in contact with intravenous drug users is given in Table 3.11. The proportion for 1992 is used rather than that for 1985 as it is the most recent proportion estimate and it is closer to the time of the survey being conducted.

Table 3.11: Minimum proportion of non-IVDUs in contact with IVDUs.		
YEAR	MALE NON-IVDUs	FEMALE NON-IVDUs
	ρ_3	ρ_4
1985	0.00006	0.00037
1992	0.00005	0.00030
PARAMETER USED	0.00005	0.00030

The Perth respondents to the ANAIDUS study shared needles on average with 0.8 people during 6 months. It is therefore assumed that they share with 1.6 people within a year (Australian National AIDS and Injecting Drug Use Study, 1992). Table 1.12

represents the number of people IVDUs share needles with in a year and the 95% confidence intervals for the parameter.

Table 3.12: Number of people needles are shared with.		
MEAN NUMBER OF PEOPLE	PARAMETER	95% CONFIDENCE INTERVAL
1.6 PER YEAR	η_1, η_2	1.2 - 2.0

Transmission of the HIV virus through sexual contact is taken from published estimates (Anderson, and May, 1988), (Dietz, 1988). The probability of transmission of the HIV virus through sexual contact from a female infective to a male susceptible has been estimated to be 0.1 and the transmission of the virus from a male infective to a female susceptible is estimated to be 0.2.

The probability of transmission through sharing of needles is known to be higher than that for sexual contact. The probability is therefore taken to be 0.2, the highest probability of transmission through heterosexual contact. Transmission rates are shown in Table 3.13.

Table 3.13: Transmission parameters.	
PARAMETER ESTIMATED	VALUE
μ_{mf} : SEXUAL CONTACT (MALE TO FEMALE)	0.2
μ_{fm} : SEXUAL CONTACT (FEMALE TO MALE)	0.1
β : SHARING NEEDLES	0.2

The respondents to the ANAIDUS survey were using condoms some of the time. This can be taken into account either through the transmission parameters or through the number of partners. Table 3.14 is based upon tables 52 and 53 on page 66 of the third report of the Australian National AIDS and Injecting Drug Use Study, 1992. Table 3.15 represents the sexual transmission rate of infection if condom use is considered, using the mean for the whole sample surveyed given in Table 3.14. The

transmission rate for sexual contact from male to female is normally estimated to be 0.2, Table 3.13. If condom use for all partners is considered, the sexual transmission rate from male infective to female susceptible is estimated to be 0.2 multiplied by 0.645, the percentage of the time condoms are not used giving an estimated transmission rate of 0.129. The same applies for transmission from female infective to male susceptible and for condom use for regular partners.

Table 3.14: Condom use for vaginal sex among heterosexuals.		
GROUP	ALL PARTNERS (95% C.I.)	REGULAR PARTNERS (95% C.I.)
MALE	37.6% (33.5% - 41.7%)	18.4% (14.0% - 22.8%)
FEMALE	29.5% (23.1% - 35.9%)	12.5% (7.3% - 17.7%)
MEAN	35.5%	16.5%

Table 3.15: Sexual transmission rates considering current condom use.		
TRANSMISSION	ALL PARTNERS	REGULAR PARTNERS
β_{mf} : MALE TO FEMALE	0.1290	0.1670
β_{fm} : FEMALE TO MALE	0.0645	0.0835

The death rate from AIDS, δ , has been estimated to be 1.000, the reciprocal of the survival period of 1 year (Blythe, and Anderson, 1988),(May, and Anderson, 1987). The incubation period for the HIV virus has been estimated to be 8 years (Anderson, et al, 1986),(Medley, et al, 1987) and the removal rate due to the progression of the disease, α , is therefore 1/8 or 0.125.

4. Model Results

4.1 Simulations

The computer simulations on the program for the model of the spread of the HIV virus among heterosexuals in Western Australia, in Appendix 1 were performed using the NAG FORTRAN Library, D02EBF to solve the differential equations. The D02EBF Library uses the variable order, variable step Gear Method, Appendix 3.

Simulations were conducted using 1%, 2% and 3% of people aged 16 to 24 taking up intravenous drugs. The initial year of the simulation has been taken to be 1985, the first year in which an individual was diagnosed as being infected through IV drug use in Western Australia. In 1985 the first case of infection from heterosexual contact was also diagnosed. The simulations were set to forecast the total number of HIV and AIDS cases alive each year to the year 2025.

The probability of transmission through sexual contact was varied due to the difference in condom use between regular partners and all partners and to investigate the effect of the differences on the epidemic. The average number of people which an IV drug using individual had shared a needle with during a year was reduced from 1.6 to 1 to also investigate its effect on the epidemic. Reducing the needle sharing 'partners' from 1.6 to 1 was decided by choosing a number which was less than the lower bound of the 95% confidence interval for the mean number of partners in a year and above half of the mean number of partners in a year.

Some simulations were conducted where behaviour modifications were introduced in 1994. These were then compared to the original simulation for each model to investigate whether it would be worthwhile proposing such intervention policies be

introduced for these changes in behaviour. These comparisons will also aid in deciding on what type of behaviour, needle sharing or condom usage, to target in these intervention policies.

The results are divided into three parts for each 'model'. The first 'model', model A, is based upon 1 infected male IVDU being introduced into the population in 1985. This was to determine the size of the spread of the HIV virus from intravenous drug users to non-IVDUs. The choice of 1 male IVDU instead of 1 female IVDU was due to the actual reported situation in 1985 where the only case of infection occurring through intravenous drugs was in a male.

The second 'model', model B, is based upon 1 infected male non-IVDU being introduced into the population in 1985. This was to determine the spread of the HIV virus from non-IVDUs to IVDUs. As there were 3 reported cases of HIV infection through sexual contact among males and only 1 reported case among females, the introduction of 1 infected male non-IVDU into the population model is the most appropriate choice.

Model C, the third 'model', is based upon a total of 5 infected people being introduced into the population in 1985. Of the infected individuals, 1 is a male IVDU, 1 is a female non-IVDU and the rest are male non-IVDUs. These were the actual reported cases of infection through heterosexual contact and intravenous drug use in 1985.

The results from these three models have been displayed as graphs of the number of living HIV cases each year from 1985 to 2025 and are found in Appendix 4. Tables A4.1 and A4.2 at the start of Appendix 4 provide the legend used for all of the figures. All of the figures have been constructed using information based only upon 1% of people aged between 16 and 24 years using intravenous drugs. These figures provide

a comparison of different results given various simulations and should not be used for prediction purposes.

4.2 Results for Model A

The results for model A, when 1 infected male IVDU is introduced into the population in 1985 are in Tables 4.1 to 4.4 and are the estimated number of living HIV and AIDS cases for male and female IVDUs and non-IVDUs in the years 1992 and 2000 when 1%, 2% and 3% of people aged 16 to 24 take up drugs respectively. The Figures A4.1 to A4.15 in Appendix 4 are the results of simulations performed on model A.

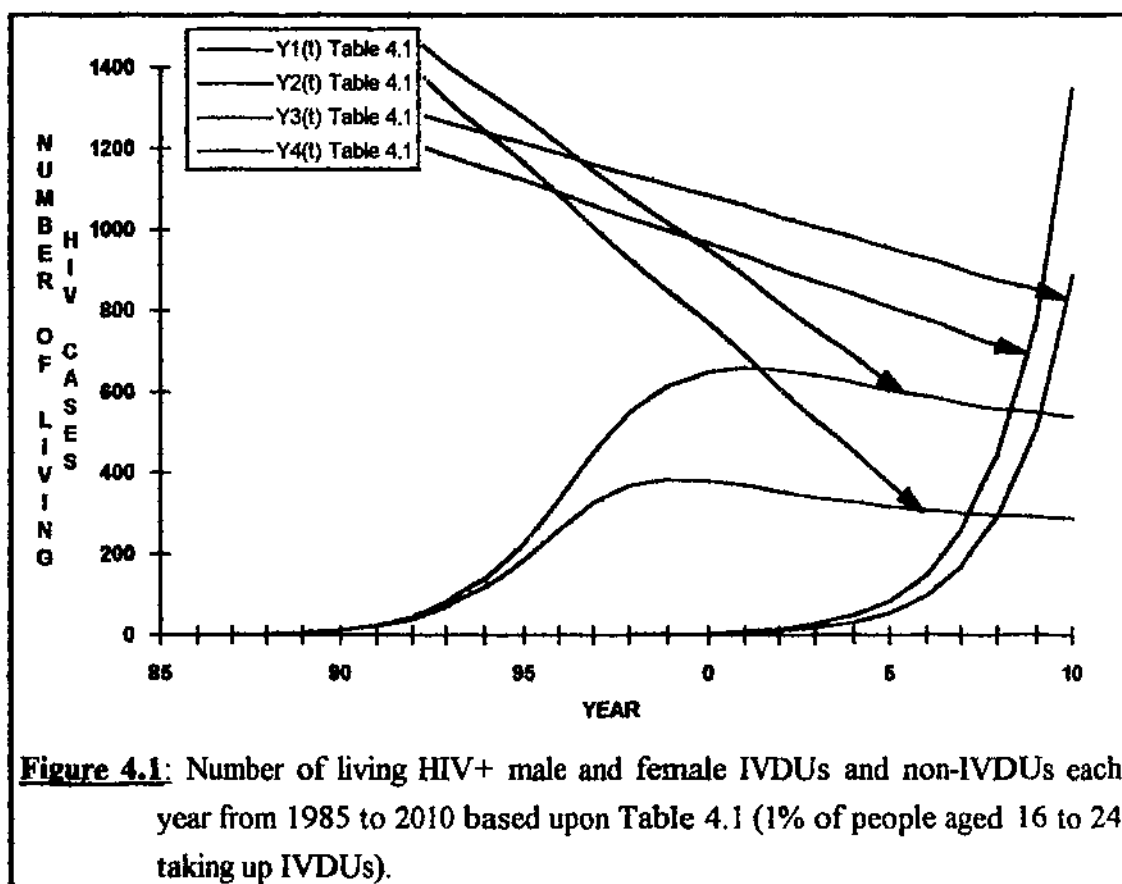


Table 4.1 shows that when condoms were used 16.5% of the time and intravenous drug users shared needles on average with 1.6 people in a year, an infected male

IVDU will start an epidemic which will cause 45 to 47 male IVDUs, and 40 to 42 female IVDUs to be infected with the HIV virus, but not have AIDS by 1992. In addition, 3 male IVDUs and 3 female IVDUs will be not only infected with the HIV virus, but they will have progressed to AIDS in 1992. No cases of HIV infection among non-IVDUs will have resulted in 1992 from this initial infection of a male IVDU.

From the initial infection of 1 male IVDU, between 650 and 1718 male IVDUs (depending upon the IVDU population size) will be living with the HIV virus in 2000. The number of AIDS cases for the same year is between 69 and 163 living cases among male IVDUs. Among female IVDUs, the number of living HIV cases is between 380 and 1134, and the number of living AIDS cases is between 42 and 116. Infection among non-IVDUs has occurred at this stage with 3 to 4 males being infected and living with the HIV virus and 5 to 6 females being infected and also living. No AIDS cases have occurred among non-IVDU heterosexuals in the year 2000 when 1 infected male IVDU was introduced into the population in 1985.

Table 4.1: Number of AIDS and HIV cases in 1992 and 2000 when condoms have been used 16.5% of the time and IVDUs share needles on average with 1.6 people in a year (Original Model A).									
% OF PEOPLE 16-24 YEARS USING IVDs		Y ₁ (t)	Y ₂ (t)	Y ₃ (t)	Y ₄ (t)	A ₁ (t)	A ₂ (t)	A ₃ (t)	A ₄ (t)
1992	1%	45	40	0	0	3	3	0	0
	2%	46	41	0	0	3	3	0	0
	3%	47	42	0	0	3	3	0	0
2000	1%	650	380	3	5	69	42	0	0
	2%	1223	768	4	6	122	81	0	0
	3%	1718	1134	4	6	163	116	0	0

In 1992 there is little difference in the number of infected male and female IVDUs and non-IVDUs between the different estimates of the size of the drug using population. In 2000, the model shows that although there was little difference in the number of

infection between the different population sizes of IVDUs in 1992, a difference does exist. The higher the size of the intravenous drug using population, the larger the number of individuals infected in both the IVDU group and the non-IVDU group. This is due to the larger pool of susceptibles at the start of the epidemic of the intravenous drug users.

Figure 4.1 shows that by about the year 2000, both of the IVDU groups have reached their peak in the number of living HIV cases. The number of living HIV cases among non-IVDU heterosexuals, while low in the year 2000, increases dramatically once the epidemic has entered the population, while the number of living HIV positive IVDUs appears to be decreasing to a stable level.

Table 4.2 shows that in 1992 when condoms are used 16.5% of the time and IVDUs share needles on average with 1 person in a year, the number of living HIV cases is 19 to 20 for male IVDUs and 18 to 19 for female IVDUs. Although the average number of people which an IVDU shares a needle with is reduced by less than 50%, the number of living HIV cases in 1992 is reduced by more than half. In the year 2000 the reduction in the number of needle sharing partners from 1.6 to 1 has a greater effect on the number of living HIV cases among the larger population sizes of IVDUs.

Table 4.2: Number of AIDS and HIV cases in 1992 and 2000 when condoms have been used 16.5% of the time and IVDUs share needles on average with 1 person in a year (Model A).									
% OF PEOPLE 16-24 YEARS USING IVDs		$Y_1(t)$	$Y_2(t)$	$Y_3(t)$	$Y_4(t)$	$A_1(t)$	$A_2(t)$	$A_3(t)$	$A_4(t)$
1992	1%	19	18	0	0	2	1	0	0
	2%	20	19	0	0	2	1	0	0
	3%	20	19	0	0	2	1	0	0
2000	1%	436	324	2	3	41	32	0	0
	2%	653	526	2	3	58	48	0	0
	3%	781	657	2	3	67	58	0	0

Table 4.2 shows that again in 1992 there is little difference among the living number of infectives due to the population sizes of intravenous drug users. However, in the year 2000 a difference does appear in the number of individuals infected. In 1992 the range for the total number of infected IVDUs was between 37 and 39 with the number of infectives based upon the 3% estimate, being 1.05 times the number of infectives based upon the 1% estimate. In the year 2000 this range is between 760 and 1438 with the number of infectives based upon the 3% estimate, being 1.89 times the number of infectives based upon the 1% estimate.

Figures A4.1 and A4.2 are based upon Tables 4.1 and 4.2. They show that although the reduction in needle sharing 'partners' does reduce the size of the peak as well as delaying it, the number of infectives among IVDUs is very similar after the year 2010. It is about the year 2010 when the number of infected intravenous drug users seems to reach a stable level. Among non-IVDUs the difference in reducing the needle sharing 'partners' appears to be simply a delay of when the peak occurs, as by the year 2025 only the number of infected female non-IVDUs based upon Table 4.1 has peaked.

Figure A4.3 in Appendix 4 shows that if in 1994 the general behaviour of heterosexuals was modified and needle sharing 'partners' were reduced to 1 per year, there is still a visible difference in the number of living infected IVDUs each year. The difference is, however, slight as the change in behaviour occurs about 5 years before the peak in the number of infected female IVDUs and about 10 years before the peak in the number of infected male IVDUs. As the peaks occur about 25 years after the first infected was introduced into the population, the modifications occur when there are already many infected individuals.

A graph for the comparison of the extension of Table 4.1 and the simulation of the behaviour modifications in 1994 of non-IVDUs was not provided as there wasn't any

visible difference between the two situations. This is due to the modifications being aimed at intravenous drug users and the spread of the HIV virus among non-IVDUs is mainly from within the group through sexual contact.

Table 4.3 shows that increasing condom usage from 16.5% to 35.5% of the time has a similar effect on the number of living cases of HIV infection among intravenous drug users, as does reducing the number of needle sharing 'partners' from 1.6 to 1 each year. There is a difference, however, in the reduction among male and female infected IVDUs. Reducing the number of needle sharing partners has reduced the number of infectives among male IVDUs to less than that which has resulted from increasing condom usage. Increasing condom usage has reduced the number of infectives among female IVDUs to less than the number of infectives resulting from a reduction in needle sharing partners.

Table 4.3: Number of AIDS and HIV cases in 1992 and 2000 when condoms have been used 35.5% of the time and IVDUs share needles on average with 1.6 people in a year (Model A).									
% OF PEOPLE 16-24 YEARS USING IVDs		$Y_1(t)$	$Y_2(t)$	$Y_3(t)$	$Y_4(t)$	$A_1(t)$	$A_2(t)$	$A_3(t)$	$A_4(t)$
1992	1%	20	17	0	0	2	1	0	0
	2%	20	17	0	0	2	1	0	0
	3%	20	17	0	0	2	1	0	0
2000	1%	448	312	0	1	42	30	0	0
	2%	663	498	0	1	58	45	0	0
	3%	788	615	0	1	67	54	0	0

Figures A4.4 and A4.5 are based upon the difference between Tables 4.1 and 4.3. They show that the increase in condom usage has a similar effect on the number of infectives among intravenous drug users as does reducing the needle sharing 'partners'. There is a slight difference, however, in the number of male IVDU infectives after the year 2015 where the numbers from increase in condom usage continue to decrease while the number of infectives from the lower condom usage is starting to slightly

increase. Figure A4.5 shows that the increase in condom usage reduces the number of infectives among non-IVDUs such, that it appears to be delayed by 10 years. The number of infectives in the year 2025 from the increased condom usage is very low compared to that of the higher condom usage.

Figure A4.6 show that increases in condom usage in 1994 has again a similar effect on the number of infected IVDUs as does reducing the needle sharing 'partners'. There is a slight change in the size and occurrence of the peak. Figure A4.7 shows that among non-IVDUs there is a significant difference in the number of infected individuals each year. This is due to the modifications occurring when there are only a few infected non-IVDUs compared to the possibility of over 400000 infected individuals, the peaks about the year 2020 when no modifications in behaviour occurred.

Comparing Tables 4.2 and 4.3 shows that naturally increasing condom usage has resulted in less infection among non-intravenous drug users than that from reducing needle sharing partners as needle sharing does not affect the spread of the virus among non-IVDUs. Reducing the needle sharing partners did reduce the number of living cases of HIV infection among non-IVDUs by nearly half in the year 2000, by reducing the infection among intravenous drug users. Increasing condom usage had a greater effect by reducing the total number of infected non-IVDUs in the year 2000 of at least 8 (Table 4.1) to only 1 infected female non-IVDU.

Table 4.4 show the results from the simulation where condoms were used 35.5% of the time and an intravenous drug user had on average 1 needle sharing 'partner' in a year. The results naturally show that there is a reduction in the number of infectives in both intravenous drug using groups and non-IVDUs.

A comparison of Tables 4.1 and 4.4 shows that reducing the number of needle sharing partners to 1 and increasing condom usage to 35.5% of the time, reduces the number of living HIV cases among IVDUs by about 80% in both the 1992 and year 2000 estimates. Table 4.4 shows reductions in the number of infections of over 50% from Tables 4.2 and 4.3 of using just one method of reducing the number of new infections. Even though the number of infections is low compared to those in Table 4.1, the rate of infection is high with an increase in the number of infected male IVDUs from 8 cases in 1992 to over 150 within 8 years.

Table 4.4: Number of AIDS and HIV cases in 1992 and 2000 when condoms have been used 35.5% of the time and IVDUs share needles on average with 1 person in a year (Model A).									
% OF PEOPLE 16-24 YEARS USING IVDs		$Y_1(t)$	$Y_2(t)$	$Y_3(t)$	$Y_4(t)$	$A_1(t)$	$A_2(t)$	$A_3(t)$	$A_4(t)$
1992	1%	8	7	0	0	1	1	0	0
	2%	8	8	0	0	1	1	0	0
	3%	8	8	0	0	1	1	0	0
2000	1%	151	130	0	0	13	12	0	0
	2%	172	153	0	0	15	13	0	0
	3%	181	162	0	0	15	14	0	0

Figures A4.8 and A4.9 are based upon Tables 4.1 and 4.4. These tables also show that the reduction of needle sharing 'partners' from 1.6 to 1 a year and the increase in condom usage from 16.5% to 35.5% of the time greatly affects the number of infectives each year. The peaks of the number of infected IVDUs is reduced by over 100 cases among males and over 50 cases for females. The lower peaks also occur about 10 years later than those of the higher number of needle sharing 'partners' and lower condom usage. The number of infected non-IVDUs is very low when condom usage is increased and needle sharing 'partners' are reduced to less than 10000 in the year 2025.

Figure A4.10 shows that reducing the needle sharing partners and increasing condom usage in 1994 still has a significant effect on the number of infectives among intravenous drug users. The peak in the number of male IVDUs infectives with the virus is reduced by at least 100 cases and is delayed by less than 5 years. The number of living infectives decreases continuously after the peak while in the original situation, the number of living HIV cases started to increase slightly after the year 2015. Among female IVDUs the number of living infectives after the peaks have occurred follow a similar pattern of decreasing slightly before reaching what appears to be a stable level. The two simulations do not reach the same stable level, with the modifications creating a level that is slightly lower than that of the original simulation.

Figure A4.11 shows that the modifications of the behaviour in 1994 of reducing needle sharing 'partners' and increasing condom usage naturally reduces the number of non-IVDUs infected given the results in Figure A4.7. There is little difference, if any, between these two figures as a reduction in needle sharing 'partners' in 1994 does not have a visible effect on the number of infectives among non-IVDUs after an initial infection has occurred in this group.

A further modification in 1994 was simulated. This was to increase condom usage to 50% as well as the reduction of needle sharing 'partners' to 1 per year. Figure A4.12 shows that among IVDUs the peak in the number of infectives is much lower and occurs later than without any modifications in 1994. The peaks occur at a level that is slightly higher than that of the eventual stable level of infectives. Figure A4.13 shows that among non-IVDUs the additional modifications reduce the number of infectives such that they do not show up on the graph.

A comparison of Figures A4.10 to A4.12 is displayed in Figure A4.14 and shows that the increase in condom usage from 35.5% to 50% among IVDUs reduces the peaks by

less than 100 cases for infected males and less than 50 cases for females. Figure A4.15 is a comparison between Figures A4.11 and A4.13 and shows that in the year 2025 there is a significant difference between the number of infected non-IVDUs. With condom usage increased to 35.5% in 1994 the total number of non-IVDU infectives in 2025 is over 50000 cases. The increase in condom usage to 50% in 1994 results in less than a total of 2000 cases of living non-IVDU infectives.

4.3 Results for Model B

The results for model B where 1 infected non-IVDU was introduced into the population in 1985, are in Tables 4.5 to 4.8 and are the estimated number of living HIV and AIDS cases for male and female IVDUs and non-IVDUs in 1992 and the year 2000 when 1%, 2% and 3% of people aged 16 to 24 take up drugs respectively. The Figures A4.16 to A4.29 in Appendix 4 are the results of simulations performed on model B.

Table 4.5: Number of AIDS and HIV cases in 1992 and 2000 when condoms have been used 16.5% of the time and IVDUs share needles on average with 1.6 people in a year (Original Model B).									
% OF PEOPLE 16-24 YEARS USING IVDs		$Y_1(t)$	$Y_2(t)$	$Y_3(t)$	$Y_4(t)$	$A_1(t)$	$A_2(t)$	$A_3(t)$	$A_4(t)$
1992	1%	0	0	24	35	0	0	2	3
	2%	0	0	23	35	0	0	2	3
	3%	0	0	23	34	0	0	2	3
2000	1%	30	26	1934	2900	2	2	153	230
	2%	59	51	1873	2817	4	4	149	224
	3%	88	76	1825	2749	6	5	145	218

Table 4.5 shows that when condoms are used 16.5% of the time and IVDUs share needles on average with 1.6 people in a year, introducing 1 infected male non-IVDU into the population in 1985 does not cause any infections among IVDUs in 1992.

Among non-IVDUs, there are between 23 and 24 infected males and between 34 and 35 infected females, depending upon the size of the intravenous drug using population.

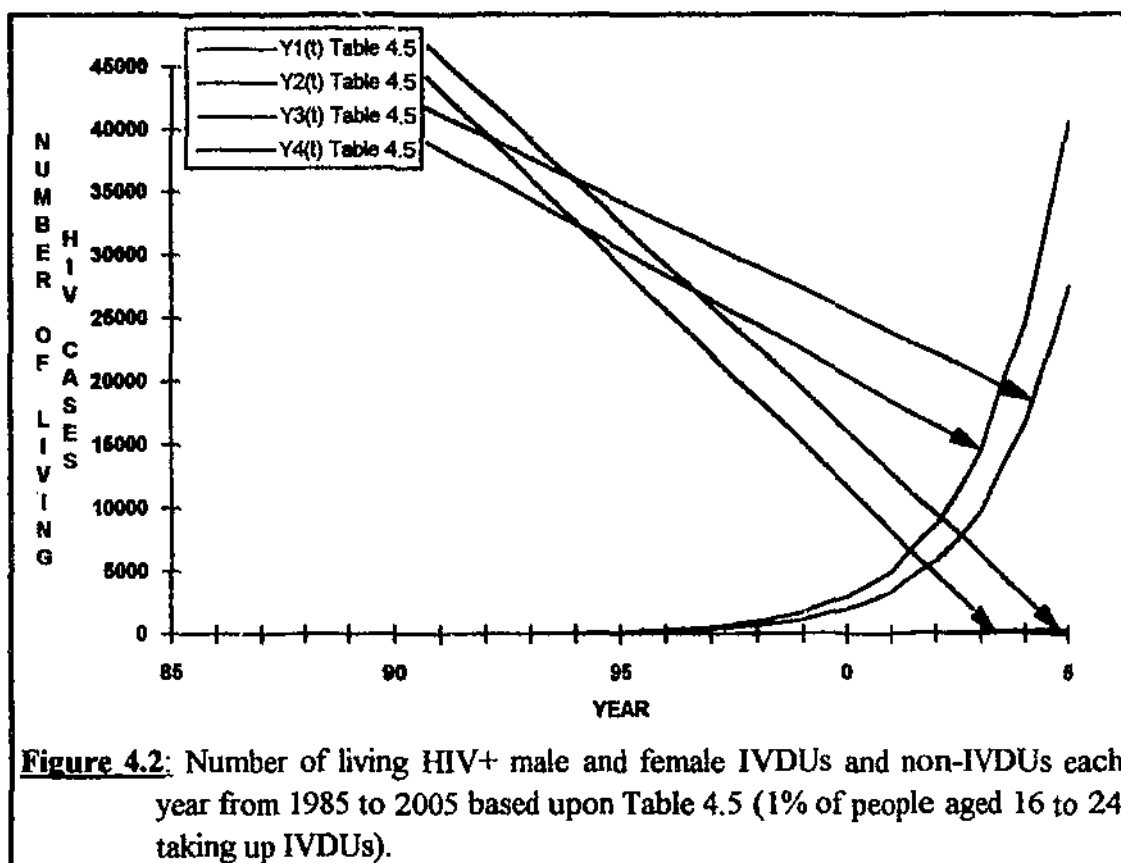


Figure 4.2 shows that when condom usage is 16.5% of the time and there are on average 1.6 needle sharing 'partners' in a year, that the number of non-IVDUs infected is much higher than the number of IVDUs infected. This is expected as the first infected was a non-IVDU.

In the year 2000, the number of infected male and female non-IVDUs has increased dramatically to nearly 2000 living cases among males and nearly 3000 cases among females. There are also infections occurring in the intravenous drug using population with between 30 and 88 cases of male living with HIV and between 26 and 76 cases of females living with the HIV virus in 2000, again depending upon the size of the intravenous drug using population.

Table 4.6: Number of AIDS and HIV cases in 1992 and 2000 when condoms have been used 16.5% of the time and IVDUs share needles on average with 1 person in a year (Model B).									
% OF PEOPLE 16-24 YEARS USING IVDs		$Y_1(t)$	$Y_2(t)$	$Y_3(t)$	$Y_4(t)$	$A_1(t)$	$A_2(t)$	$A_3(t)$	$A_4(t)$
1992	1%	0	0	24	35	0	0	2	3
	2%	0	0	23	35	0	0	2	3
	3%	0	0	23	34	0	0	2	3
2000	1%	12	10	1934	2900	1	1	153	230
	2%	23	20	1873	2817	2	2	149	224
	3%	34	30	1825	2749	3	2	145	218

A comparison between Tables 4.6 and 4.5 shows that naturally, reducing the number of needle sharing partners does not reduce the number of infected non-IVDUs in the early stages of the epidemic. The only effect in the year 2000 is on the number of infected IVDUs. The number of infected IVDUs is reduced by more than 50% by reducing the number of needle sharing partners to 1 per year.

Figure A4.16 is based upon Tables 4.5 and 4.6 and shows that the reduction in the number of IVDU infectives does not remain at 50% but is actually less. The difference is in a slightly lower peak that occurs less than 5 years later. The number of infectives for both simulations appear to be reaching the same level of stability. A graph is not provide for non-IVDUs as the difference in needle sharing partners does not create a visible difference. Naturally there is no visible difference for the number of IVDU infectives when needle sharing partners are reduced in 1994. Figure A4.17 shows the difference in the number of IVDU infectives when needle sharing 'partners' are reduced from 1.6 to 1 per year. The results are very similar to those in Figure A4.16 as the modifications occurred when there were only a few cases of infection among intravenous drug users.

Table 4.7: Number of AIDS and HIV cases in 1992 and 2000 when condoms have been used 35.5% of the time and IVDUs share needles on average with 1.6 people in a year (Model B).									
% OF PEOPLE 16-24 YEARS USING IVDs		$Y_1(t)$	$Y_2(t)$	$Y_3(t)$	$Y_4(t)$	$A_1(t)$	$A_2(t)$	$A_3(t)$	$A_4(t)$
1992	1%	0	0	8	11	0	0	1	1
	2%	0	0	8	11	0	0	1	1
	3%	0	0	8	11	0	0	1	1
2000	1%	3	2	175	263	0	0	15	23
	2%	6	5	171	257	0	0	15	23
	3%	9	7	168	253	1	1	15	22

Increasing condom usage from 16.5% to 35.5% has reduced the number of infections among non-intravenous drug users by more than 60% in 1992 and by more than 90% in the year 2000. The reduction among IVDUs is close to 90% in the year 2000. The number of infections among IVDUs and non-IVDUs is reduced more by increasing condom usage than from reducing the average number of needle sharing partners.

Increasing condom usage from 16.5% to 35.5% has a significant effect on the number of infectives. The delay in the peak in the number of IVDU infectives, Figure A4.18, is less 10 years while among non-IVDUs, Figure A4.19, the delay in the peak in the number of infectives is more than 10 years. There is a reduction in the height of the peaks in both groups, but the difference is not large. Increasing condom usage in 1994, Figures A4.20 and A4.21, produces similar effects. The main difference being that the delay in the peak in the number of infectives is not as significant, especially among intravenous drug users.

Comparing Tables 4.7 and 4.8 again shows that reducing the mean number of needle sharing partners when 1 infected male non-IVDU was introduced into the population, does not affect the number of infected non-IVDUs. The number of infected intravenous drug users in 2000 has been reduced by more than 50%.

Table 4.8: Number of AIDS and HIV cases in 1992 and 2000 when condoms have been used 35.5% of the time and IVDUs share needles on average with 1 person in a year (Model B).									
% OF PEOPLE 16-24 YEARS USING IVDs		$Y_1(t)$	$Y_2(t)$	$Y_3(t)$	$Y_4(t)$	$A_1(t)$	$A_2(t)$	$A_3(t)$	$A_4(t)$
1992	1%	0	0	8	11	0	0	1	1
	2%	0	0	8	11	0	0	1	1
	3%	0	0	8	11	0	0	1	1
2000	1%	1	1	175	263	0	0	15	23
	2%	2	2	171	257	0	0	15	23
	3%	3	3	168	253	0	0	15	22

Reducing needle sharing 'partners' and increasing condom usage has similar effects to increasing condom usage alone, though naturally more significant. This is especially noticeable among intravenous drug users, Figure A4.22, with the peak occurring at least 10 years later than for the original situation in Table 4.5. The difference in the number of infectives among non-IVDUs, Figure A4.23 is very similar to Figure A4.19 were the only change was that condom usage was increased. This is again due to the fact that changes in needle sharing 'partners' has little if any affect on the number of non -IVDUs infected.

Figures A4.24 and A4.25 are for the simulation where condom usage was increased to 35.5% and needle sharing partners were reduced to 1 per year in 1994. The figures are similar to those where the changes were made at the start of the epidemic. They still show a significant difference in the number of infectives but it is naturally not as great as those shown in Figures A4.22 and A4.23.

Increasing condom usage to 50% increases the delay in the peak of the number of IVDU infectives to nearly 20 years and again to a height that is close to the level of stability in the number of living HIV cases, Figure A4.26. Figure A4.27 shows that the delay in the number of non-IVDU infectives is such that by the year 2025 the

peaks have yet to occur. This is nearly 15 years after the original simulation results had peaked.

Figures A4.28 and A4.29 show that there is a significant difference in the occurrence of the peaks between increasing condom usage to 35.5% to increasing it to 50% of the time in 1994. There is also a difference in the size of the peaks of IVDU infectives. The same cannot be said for non-IVDUs as the number of non-IVDU infectives when condom usage was increased to 50% have not peaked by the year 2025.

4.4 Results for Model C

The results for model C, when 1 infected male IVDU, 3 infected male non-IVDUs and 1 infected female non-IVDU were introduced into the population in 1985 are in Tables 4.9 to 4.12. These tables provide the estimated number of living HIV and AIDS cases for male and female IVDUs and non-IVDUs in 1992 and the year 2000 when 1%, 2% and 3% of people aged 16 to 24 take up drugs respectively. The Figures A4.30 to A4.43 in Appendix 4 are the results of simulations performed on model C.

Table 4.9 shows that the virus can spread quickly throughout the population. In 1992 there were between 85 and 91 intravenous drug users infected with the HIV virus which had not progressed to AIDS and 6 IVDUs which were infected and progressed to AIDS. In the year 2000 the number of living HIV cases only had increased to between 1032 and 2880 living cases and the number of AIDS cases was between 112 and 285 cases. Among the non-IVDUs there were between 216 and 211 cases of HIV infection only and 17 living AIDS cases. In the year 2000 these had increased to between 17387 and 16467 cases of HIV infection and between 1387 and 1315 living AIDS cases.

Table 4.9: Number of AIDS and HIV cases in 1992 and 2000 when condoms have been used 16.5% of the time and IVDUs share needles on average with 1.6 people in a year (Original Model C).

% OF PEOPLE 16-24 YEARS USING IVDUs		$Y_1(t)$	$Y_2(t)$	$Y_3(t)$	$Y_4(t)$	$A_1(t)$	$A_2(t)$	$A_3(t)$	$A_4(t)$
1992	1%	45	40	87	129	3	3	7	10
	2%	47	42	86	128	3	3	7	10
	3%	48	43	85	126	3	3	7	10
2000	1%	652	380	6978	10419	69	43	556	831
	2%	1232	769	6762	10122	124	82	539	808
	3%	1740	1140	6588	9879	168	117	526	789

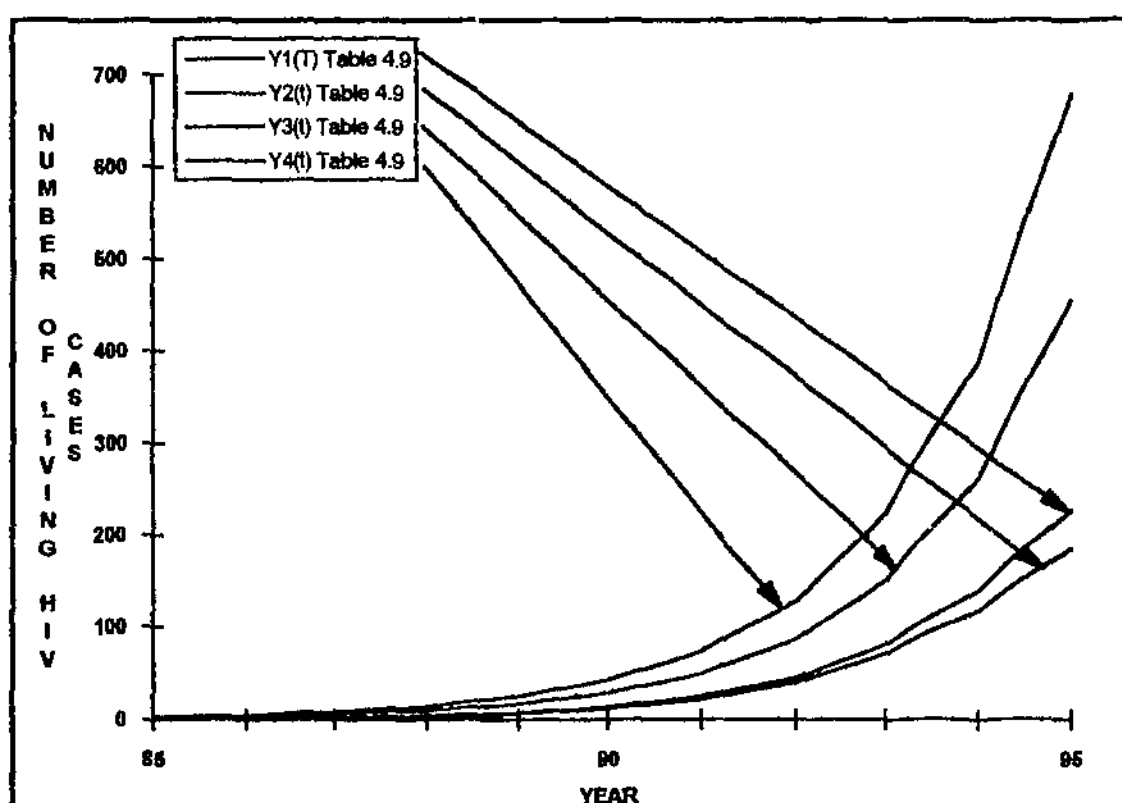


Figure 4.3: Number of living male and female IVDUs and non-IVDUs each year up from 1985 to 1995 based upon Table 4.9 (1% of people aged 16 to 24 taking IVDUs).

The differences in the size of the IVDU population had a significant effect on the number of infectives among the intravenous drug using population. The differences in the size of the population also affected the number of non-IVDU infectives, but not as significantly as that of the IVDUs. This is due to the fact that the size of the

intravenous drug using population affects the size of the pool of the susceptibles in both IVDU and non-IVDU groups, more so in the IVDU as the non-IVDU population is very large compared to that of the IVDUs.

The high numbers of infection among intravenous drug users is mainly attributed to the high probability of infection through the combination of sexual contact and needles sharing. The high numbers of infection in the non-IVDU group is mainly due to the high number of susceptibles. Figure 4.3 shows that the number of living HIV cases among both IVDUs and non-IVDUs is increasing dramatically between the years 1990 to 1995.

Table 4.10 again shows that reducing the mean number of needle sharing partners can reduce the number of infections significantly. Although the reduction in the number of infections among IVDUs is at least 50% in both 1992 and 2000, the number of infections among non-IVDUs is reduced by less than 0.1%.

Table 4.10: Number of AIDS and HIV cases in 1992 and 2000 when condoms have been used 16.5% of the time and IVDUs share needles on average with 1 person in a year (Model C).									
% OF PEOPLE 16-24 YEARS USING IVDs		$Y_1(t)$	$Y_2(t)$	$Y_3(t)$	$Y_4(t)$	$A_1(t)$	$A_2(t)$	$A_3(t)$	$A_4(t)$
1992	1%	20	18	87	129	2	1	7	10
	2%	20	19	86	128	2	1	7	10
	3%	21	19	85	126	2	1	7	10
2000	1%	445	327	6976	10417	42	32	556	831
	2%	681	542	6760	10119	60	50	539	808
	3%	836	691	6585	9876	72	61	526	789

Figure A4.30 is based upon the comparison in the number of IVDU infectives in Tables 4.9 and 4.10. Again the main difference is in the occurrence of the peak in the number of living HIV cases. The peaks occur less than 5 years after those for the number of infectives when there were 1.6 needle sharing 'partners' per year. The

difference in the height of the peak of male IVDUs is less than 100 cases and for females, a difference of less than 50 cases between the two simulations.

Figure A4.31 shows that reducing needle sharing 'partners' in 1994 creates peaks in the number of IVDU infectives that are simply reduced in height and not so much in being delayed. Among female IVDU infectives, both simulations are at the same level after the year 2002 and reach the same level of stability of around 300 cases after the year 2010. Among male IVDU infectives, the modified behaviour produces levels of infectives that is constantly slightly lower than that for the simulation of no behaviour modification. A graph was not provided for the differences in the simulations for the number of non-IVDU infectives as there wasn't any visible difference.

Table 4.11: Number of AIDS and HIV cases in 1992 and 2000 when condoms have been used 35.5% of the time and IVDUs share needles on average with 1.6 people in a year (Model C).									
% OF PEOPLE 16-24 YEARS USING IVDs		Y ₁ (t)	Y ₂ (t)	Y ₃ (t)	Y ₄ (t)	A ₁ (t)	A ₂ (t)	A ₃ (t)	A ₄ (t)
1992	1%	20	17	28	42	2	1	2	4
	2%	20	17	28	42	2	1	2	4
	3%	20	18	28	41	2	1	2	4
2000	1%	449	313	644	967	42	31	57	85
	2%	669	502	628	946	59	46	55	83
	3%	802	624	616	928	69	55	54	82

Increasing condom usage from 16.5% to 35.5% reduces the number of infections in 1992 by more than 50% among IVDUs and by more than 60% among non-IVDUs. The reduction in the year 2000 among IVDUs is not as significant as in 1992. Among intravenous drug users the reduction in the number of infections ranges from less than 10% to less than 50%, depending upon the size of the drug using population. The reduction in the number of non-IVDUs in the year 2000 is more than 90% of the cases in Table 4.9.

Figures A4.32 and A4.33, based upon Tables 4.10 and 4.11, shows that the increase in condom usage creates peaks that occur later and are slightly smaller to the original peaks. The delays in the peaks for IVDUs is less than 5 years and the height of the male infectives is reduced by less than 100 and the height of the female infectives is reduced by less than 50 cases. Among non-IVDUs the reduction in height is less than 25000 cases and the delays in the occurrence of the peaks is nearly 10 years. Changes in condom usage in 1994 still produces changes in the height of the peaks, though not as much for IVDUs, but the delays in the peak of the number of infectives is about 2 years for IVDUs and 5 years for non-IVDUs.

Table 4.12: Number of AIDS and HIV cases in 1992 and 2000 when condoms have been used 16.5% of the time and IVDUs share needles on average with 1 person in a year (Model C).									
% OF PEOPLE 16-24 YEARS USING IVDs		Y ₁ (t)	Y ₂ (t)	Y ₃ (t)	Y ₄ (t)	A ₁ (t)	A ₂ (t)	A ₃ (t)	A ₄ (t)
1992	1%	8	8	28	42	1	1	2	4
	2%	9	8	28	42	1	1	2	4
	3%	9	8	28	41	1	1	2	4
2000	1%	154	131	644	967	14	12	57	85
	2%	178	157	628	945	15	14	55	83
	3%	190	170	615	928	16	15	54	82

A comparison between Tables 4.9 and 4.12 shows that again a reduction in needle sharing 'partners' combined with an increase in condom usage has the greatest effect on the number of infectives and AIDS cases. The number of IVDU infectives in 1992 is reduced to about 1/5 of that of the original simulation and the number of non-IVDU infectives is reduced to about 1/3 of the original number. In the year 2000 these reductions range from about 66% to about 90% for IVDUs and for non-IVDUs the reduction in the number of infectives is more than 90%.

Figures A4.36 and A4.37 show that there is a significant difference in the number of infectives for both intravenous drug users and non-IVDUs. The new peaks of IVDU

infectives occur after the year 2005, and at a level slightly higher than that of the level of stability which the numbers later reach. The new peaks for non-IVDUs occurs at about the year 2020, 10 years after the peaks of the original simulation.

Reducing needle sharing 'partners' to 1 per year and increasing condom usage to 35.5% in 1994, Figures A4.38 and A4.39, have a significant effect on the size and occurrence of the peaks in the number of infectives for both IVDU and non-IVDU groups. The effects are naturally not as great as those for changes in behaviour at the early stages of infection. If the condom usage is increased to 50% as well as reducing needle sharing 'partners' in 1994, Figures A4.40 and A4.41, the changes in the peaks of the number of infectives is very significant for both IVDUs and non-IVDUs. A comparison between changes in condom usage to 35.5% and to 50% in 1994, Figures A4.42 and A4.43, show that the increase of 14.5% creates a visible difference in the number of IVDU infectives and non-IVDU infectives.

4.5 Discussion

The model for the spread of the HIV virus among heterosexuals through sexual contact and IV drug use shows that infection from drug users to non drug users does eventually occur but is not significant. The same applies to the spread of the virus from non-IVDUs to intravenous drug users. The main significance is from infection within the groups. Once an individual is infected, although there may be a small start to the number of newly infected cases, the number of cases of new infections increases dramatically soon afterwards. Figure 4.1 shows clearly that the virus has spread from IVDUs to non drug users and that once this transfer of infection has occurred, the virus spreads rapidly through the non-IVDU population.

The difference between condom usage of 16.5% and 35.5% can be a delay of 10 years among non drug users and 4 years among IVDUs in the peak of the number of living HIV cases (Figures A4.4, A4.5, A4.18, A4.1, A4.32 and A4.33). The difference in condom usage also affects the size of the peak among both non-IVDUs and intravenous drug users. The difference between 1.6 and 1 needle sharing 'partners' in a year is only visible among the number of IVDUs infected, or when the first case of infection occurred in an IVDU and the virus has spread from the IVDU group to non-IVDUs (Figures A4.1, A4.2, A4.1 and A4.30). This indicates that accurate information is required if the model is to be used to predict the spread of the virus through the population and to estimate the number of individuals infected with the HIV virus.

Simply increasing condom usage from 16.5% to 35.5% in 1994 has a substantial effect on delaying and reducing the size of the epidemic among both intravenous drug users and non-IVDUs (Figures A4.6, A4.7, A4.20, A4.21, A4.34 and A4.35). This is due to the change in behaviour occurring at an early stage of the epidemic and the behaviour modification is occurring in both groups. Reducing the mean number of people needles are shared with in a year from 1.6 to 1 will also reduce the size of the epidemic as well as delay it, but this is only among IVDUs (Figures A4.3, A4.17 and A4.31). This is due to the behaviour modification only occurring in the IVDU group and the spread of the HIV virus between groups is minimal.

The model has limitations in that it does not allow for individuals to transfer from one group to another. When an individual stops taking IV drugs they would normally transfer from the IVDU group to the non-IVDU group. The model assumed that all individuals would be either IVDUs or non-IVDUs for life. This ensures that the estimated number of infected non-IVDUs were actually infected through heterosexual

contact. By removing the intravenous drug users when they stop taking drugs, the number of cases of infection is under estimated.

The model has other limitations in that it only concentrates on the spread of the virus among heterosexuals through heterosexuals, yet the virus is constantly entering the heterosexual population through bisexual partners and homosexual and bisexual needle sharing 'partners' of heterosexuals. Bisexuals provide a path for the spread of the virus from the homosexual population to the heterosexual population. To include these 'partners' into the model might require the model to include the spread of the virus through these populations, as well as through the heterosexual population. An example of a model for the spread of the HIV virus throughout the entire sexually active population is given in Appendix 5.

Apart from bisexuals, there are other factors in the spread of the virus through the heterosexual population which have not been taken into account in the model. People travelling to high risk countries have a higher probability of infection, as many countries have higher numbers and incidences of infection than Western Australia and, therefore, probability of contact with infected individuals is greater. One method for introducing this factor into the model would be to consider the proportion of heterosexuals which travel to these high risk countries and an estimate of the proportion of infected individuals in that country.

Another factor in the spread of the HIV virus among heterosexuals is the presence of another sexually transmitted disease. The presence of S.T.D.s increases the probability of transmission of the HIV virus from the infected individual to the susceptible partner. To model this factor, an estimate of the proportion of susceptibles with S.T.D.s is required as they would have a different probability of transmission of the virus. Mathematical models are generally simplified in order to be easily adapted and

analysed. The more parameters or groups added to the model, the more complicated it becomes as shown when comparing the model used in Chapter 2 and the models in Appendix 5.

In spite of these limitations in the model used, the model and results are a significant first step towards the understanding of the transmission dynamics of the spread of the HIV virus among heterosexuals in Western Australia. The model has been useful as it has shown that simple changes can affect the results in the immediate future greatly. Some of the figures in Appendix 4 show that changes in behaviour in 1994, after the first infected has entered the population, still has a significant effect on the numbers of infected in the near future. This is due to the epidemic being in its early stages. Increasing condom usage had a greater effect on future numbers of infectives than reducing needle sharing 'partners' as it applies to both intravenous drug users and non-IVDUs.

The model has also shown that without any changes, such as a cure or vaccine, the numbers of infected individuals will remain at a high level and the situation will be that of an endemic rather than an epidemic. Modifications to behaviour patterns have little effect on this level of stability, if anything, a slight reduction in the number of living HIV cases (Figures A4.30, A4.40 and A4.41).

4.6 Recommendations

This study into the spread of the HIV virus among heterosexuals in Western Australia through intravenous drug use and sexual contact has shown that although studies have been conducted on drug users, they do not present the required information necessary for the execution of the proposed model. Many assumptions regarding the non-drug

using population were made based almost entirely on studies conducted on the drug using population, which accounts for less than 1% of the total sexually active population concerned.

If further information on the spread of the virus among heterosexuals is required, then studies need to be conducted not only on drug users, but also on the non-drug using majority of the Western Australian population. It is important that the information from the study be separated into male and female respondent groups, as well as heterosexual, bisexual and homosexual groups.

Information on all or most of the partners would be more useful in making some assumptions on the characteristics of the partners of the respondents, rather than relying on information on regular partners only, as not all respondents to the ANAIDUS study had a regular partner and naturally the mean number of regular partners in a year was less than the mean number of total partners in a year.

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Appendix 1

1987 Case Definition of AIDS

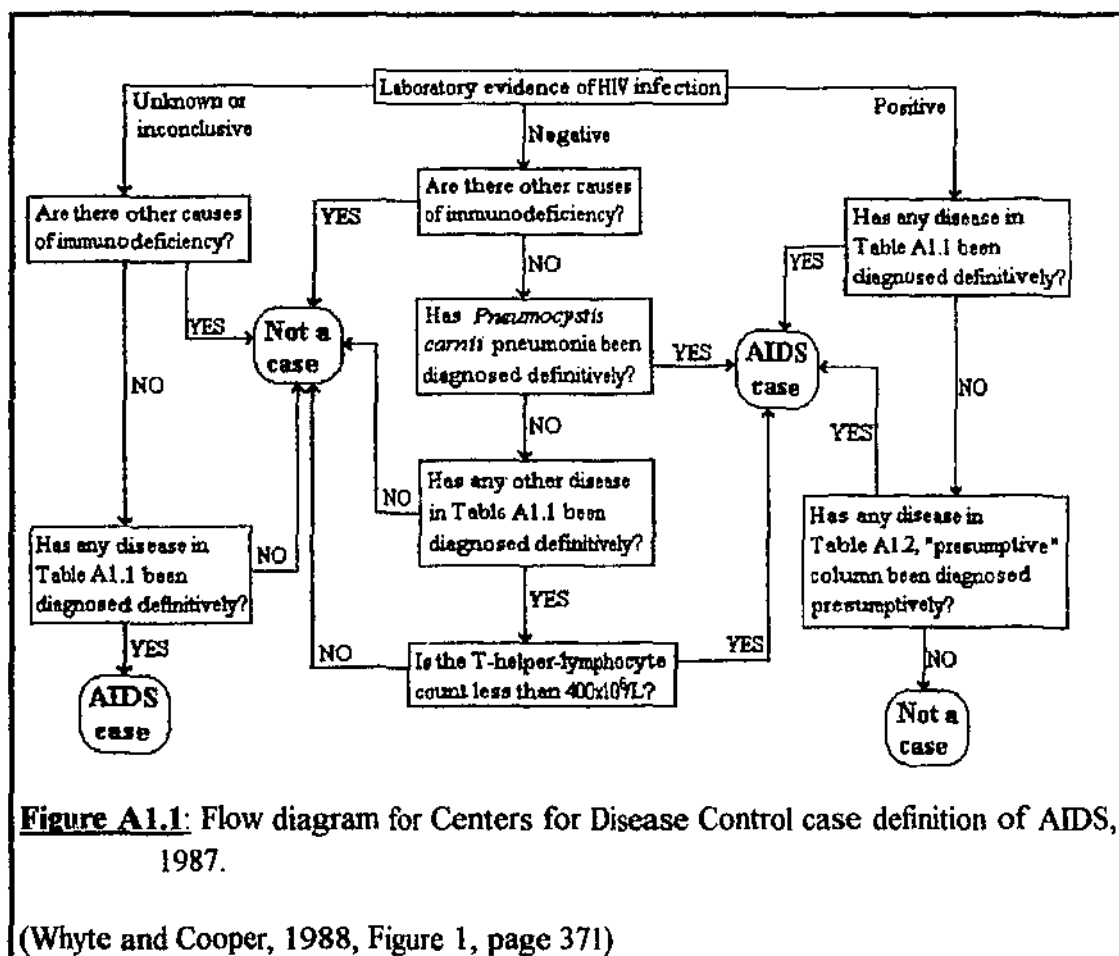


Figure A1.1: Flow diagram for Centers for Disease Control case definition of AIDS, 1987.

(Whyte and Cooper, 1988, Figure 1, page 371)

Table A1.1: Surveillance definition of AIDS.

INFECTION/CANCER	SITE(S)	METHOD OF DEFINITE DIAGNOSIS
<i>1982</i>		
Candidiasis	Oesophagus	Histology, microscopy, clinical
Cryptococcosis	Central nervous system or disseminated	Histology, culture
Cryptosporidiosis	Stools (diarrhoea for more than one month)	Histology, microscopy
Cytomegalovirus	Other than liver, spleen, lymph nodes	Histology, cytology
Herpes simplex virus	Mucocutaneous (chronic), lungs, gastrointestinal tract	Histology, cytology, culture
Kaposi's sarcoma	All (in persons of less than 60 years of age)	Histology
Lymphoma	Brain	Histology
<i>Mycobacterium avium</i>	Other than lungs, lymph nodes	Culture
<i>Mycobacterium kansasii</i>	Other than lungs, lymph nodes	Culture
<i>Pneumocystis carinii</i>	Lungs	Histology, microscopy
Progressive multifocal leukoencephalopathy	Brain	Histology
Strongyloidosis	Other than gastrointestinal tract	Histology
Toxoplasmosis	Other than liver, spleen, lymph nodes	Histology, microscopy
<i>1985: The above diseases plus the following with laboratory evidence of HIV infection</i>		
Candidiasis	Bronchi, lungs	Microscopy
Histoplasmosis	Disseminated	Histology, cytology, culture
Isoporiasis	Gastrointestinal tract	Histology, microscopy
Kaposi's sarcoma	All (in persons of 60 years of age and older)	Histology
Lymphoid interstitial pneumonitis	lungs (in children of less than 13 years of age)	Histology
Non-Hodgkin's lymphoma	All	Histology
<i>1987: All of the above diseases plus the following with laboratory evidence of HIV infection</i>		
Coccidioidomycosis	All	Histology, microscopy, cytology
HIV encephalopathy	Brain	Clinical
HIV wasting syndrome	Not applicable	Clinical
Multiple pyogenic bacteria	All (in children of less than 13 years of age)	Culture
<i>Mycobacterium tuberculosis</i>	Extrapulmonary	Culture
Recurrent salmonella bacteraemia	Blood	Culture
<i>Presumptive diagnoses of:</i>		
Candidiasis	Oesophagus	Not applicable
Cytomegalovirus	Eyes	Not applicable
Kaposi's sarcoma	All	Not applicable
Mycobacteriosis	Disseminated	Not applicable
<i>Pneumocystis carinii</i>	Lungs	Not applicable
Toxoplasmosis	Brain	Not applicable
Lymphoid interstitial pneumonitis	Lungs	Not applicable
<i>(Whyte and Cooper, 1988, Table 1, page 369).</i>		

Table A1.2: Diseases that are indicative of AIDS*.

DISEASE	DEFINITIVE	PRESUMPTIVE
Candidiasis	x	x
Coccidioidomycosis	x	-
Cryptococcosis	x	-
Cryptosporidiosis	x	-
Cytomegalovirus	x	x
HIV encephalopathy	-	x
HIV wasting syndrome	-	x
Herpes simplex virus	x	-
Histoplasmosis	x	-
Isosporiasis	x	-
Kaposi's sarcoma	x	x
Lymphoid interstitial pneumonitis	x	x
Lymphoma	x	-
Mycobacteriosis	x	x
<i>Pneumocystis carinii</i> (pneumonitis)	x	x
Progressive multifocal leukoencephalopathy	x	-
Salmonellosis	x	-
Strongyloidosis	x	-
Toxoplasmosis	x	x

*definitive diagnoses do not always require laboratory evidence of HIV infection. All the presumptive diagnoses require laboratory evidence of HIV infection.
x = require diagnostic method.

(Whyte and Cooper, 1988, Table 2, page 370).

Appendix 2

Program

```
C  WAFEX.FOR PROGRAM TEXT USING D02EBF (NAGD2 AND NAGX)
C  FORTRAN PC50 RELEASE 2. NAG COPYRIGHT 1983.
C    WESTERN AUSTRALIA
C  .. Parameters ..
  INTEGER    NOUT
  PARAMETER  (NOUT=6)
C  .. Scalars in Common ..
  DOUBLE PRECISION H, XEND
  INTEGER     I
C  .. Local Scalars ..
  DOUBLE PRECISION TOL, X
  INTEGER       IFAIL, IR, IW, J, MPED, N
C  .. Local Arrays ..
  DOUBLE PRECISION W(16,34), Y(16)
C  .. External Subroutines ..
  EXTERNAL     D02EBF, FCN, OUT, PEDERV
C  .. Common blocks ..
  COMMON       XEND, H, I
C  .. Executable Statements ..
  WRITE (NOUT,FMT=99996)
  WRITE (NOUT,FMT=99994)
  N = 16
  IW = 34
  MPED = 0
  IR = 2
  DO 20 J = 10,10
    TOL = 0.1D0*10.D0**(-J)
    WRITE (NOUT,FMT=99999) TOL
    WRITE (NOUT,FMT=99998)
    X = 0.0D0
    XEND = 25.0D0
C  Y(1) is  $X_1(0)$ : the number of male intravenous drug using susceptibles at the start of the
C  simulation.
    Y(1) = 2462.0D0
C  Y(2) is  $X_2(0)$ : the number of female intravenous drug using susceptibles at the start of the
C  simulation.
    Y(2) = 1187.0D0
C  Y(3) is  $X_3(0)$ : the number of male non-intravenous drug using susceptibles at the start of
C  the simulation.
```



```

      Y(3) = 344954.0D0
C  Y(4) is X4(0): the number of female non-intravenous drug using susceptibles at the start of
C  the simulation.
      Y(4) = 274475.0D0
C  Y(5) is Y1(0): the number of male intravenous drug using infectives at the start of the
C  simulation.
      Y(5) = 1.0D0
C  Y(6) is Y2(0): the number of female intravenous drug using infectives at the start of the
C  simulation.
      Y(6) = 0.0D0
C  Y(7) is Y3(0): the number of male non-intravenous drug using infectives at the start of the
C  simulation.
      Y(7) = 0.0D0
C  Y(8) is Y4(0): the number of female non-intravenous drug using infectives at the start of
C  the simulation.
      Y(8) = 0.0D0
C  Y(9) is A1(0): the number of male intravenous drug using AIDS cases at the start of the
C  simulation.
      Y(9) = 0.0D0
C  Y(10) is A2(0): the number of female intravenous drug using AIDS cases at the start of the
C  simulation.
      Y(10) = 0.0D0
C  Y(11) is A3(0): the number of male non-intravenous drug using AIDS cases at the start of
C  the simulation.
      Y(11) = 0.0D0
C  Y(12) is A4(0): the number of female non-intravenous drug using AIDS cases at the start
C  of the simulation.
      Y(12) = 0.0D0
C  Y(13) is P1(0): the total number of male intravenous drug users in the population at the
C  start of the simulation.
      Y(13) = 2463.0D0
C  Y(14) is P2(0): the total number of female intravenous drug users in the population at the
C  start of the simulation.
      Y(14) = 1187.0D0
C  Y(15) is P3(0): the total number of male non-intravenous drug users in the population at
C  the start of the simulation.
      Y(15) = 344954.0D0
C  Y(16) is P4(0): the total number of female non-intravenous drug users in the population at
C  the start of the simulation.
      Y(16) = 274475.0D0
      H = 1.0D0
      I = 24
      IFAIL = 1
      CALL D02EBF(X,XEND,N,Y,TOL,IR,FCN,MPED,PEDERV,OUT,W,IW,IFAIL)
      WRITE (NOUT,FMT=99997) IFAIL

```

```

      IF (TOL.LT.0.D0) WRITE (NOUT,FMT=99995)
20  CONTINUE
      STOP
C
99999 FORMAT (/, ' CALCULATION WITH TOL= ',D8.1)
99998 FORMAT ( ' X AND SOLUTION AT EQUALLY SPACED POINTS')
99997 FORMAT ( ' IFAIL= ',I1)
99996 FORMAT (/,/,/,/, ' D02EBF EXAMPLE PROGRAM RESULTS',/,1X)
99995 FORMAT ( ' RANGE TOO SHORT FOR TOL')
99994 FORMAT (/, ' CALCULATING JACOBIAN INTERNALLY')
99993 FORMAT (/, ' CALCULATING JACOBIAN BY PEDERV')
      END
      SUBROUTINE FCN(T,Y,F)
C      .. Scalar Arguments ..
      DOUBLE PRECISION T
C      .. Array Arguments ..
      DOUBLE PRECISION F(16), Y(16)
C
C      LAMDMU is  $\Lambda_1$ : the recruitment rate for the male intravenous drug using population.
      REAL*8 LAMDMU /291/
C      LAMDFU is  $\Lambda_2$ : the recruitment rate for the female intravenous drug using
C      population.
      REAL*8 LAMDFU /140/
C      LAMDMN is  $\Lambda_3$ : the recruitment rate for the male non-intravenous drug using
C      population.
      REAL*8 LAMDMN /18662/
C      LAMDFN is  $\Lambda_4$ : the recruitment rate for the female non-intravenous drug using
C      population.
      REAL*8 LAMDFN /15651/
C      NMU is  $\eta_1$ : the average number of needle sharing 'partners' of a male intravenous
C      drug user in a year.
      REAL*8 NMU /1.6/
C      NFU is  $\eta_2$ : the average number of needle sharing 'partners' of a female intravenous
C      drug user in a year.
      REAL*8 NFU /1.6/
C      BETAHT is  $\beta$ : the probability of transmission of the virus through the sharing of
C      needles.
      REAL*8 BETAHT /0.2/
C      RHOMN is  $p_3$ : the minimum proportion of male non-IVDUs that have come into
C      sexual contact with intravenous drug users.
      REAL*8 RHOMN /5.0D-5/
C      RHOFN is  $p_4$ : the minimum proportion of female non-IVDUs that have come into
C      sexual contact with intravenous drug users.
      REAL*8 RHOFN /3.0D-4/

```

C C12 : the average number of female IVDU partners that a male intravenous drug user
 C has in a year.
 REAL*8 C12 /2.9670/
 C C14 : the average number of female non-IVDU partners that a male intravenous drug
 C user has in a year.
 REAL*8 C14 /1.6330/
 C C21 : the average number of male IVDU partners that a female intravenous drug user
 C has in a year.
 REAL*8 C21 /7.2306/
 C C23 : the average number of male non-IVDU partners that a female intravenous drug
 C user has in a year.
 REAL*8 C23 /0.5694/
 C C32 : the average number of female IVDU partners that a male non-intravenous drug
 C user has in a year.
 REAL*8 C32 /0.0322/
 C C34 : the average number of female non-IVDU partners that a male non-intravenous
 C drug user has in a year.
 REAL*8 C34 /4.5678/
 C C41 : the average number of male IVDU partners that a female non-intravenous drug
 C user has in a year.
 REAL*8 C41 /0.0312/
 C C43 : the average number of male non-IVDU partners that a female non-intravenous
 C drug user has in a year.
 REAL*8 C43 /7.7532/
 C BETA_{FM} is β_{FM} : the probability of transmission of the HIV virus from a female
 C infective to a male susceptible through sexual contact.
 REAL*8 BETA_{FM} /0.0645/
 C BETA_{MF} is β_{MF} : the probability of transmission of the HIV virus from a male
 C infective to a female susceptible through sexual contact.
 REAL*8 BETA_{MF} /0.1290/
 C MUMU is μ_1 : the migration rate from the male intravenous drug using population.
 REAL*8 MUMU /0.10417/
 C MUFU is μ_2 : the migration rate from the female intravenous drug using population.
 REAL*8 MUFU /0.10417/
 C MUMN is μ_4 : the migration rate from the male non-intravenous drug using
 C population.
 REAL*8 MUMN /0.02857/
 C MUFN is μ_4 : the migration rate from the female non-intravenous drug using
 C population.
 REAL*8 MUFN /0.02857/
 C ALPHA is $1/\alpha$: the mean incubation period for the HIV virus.
 REAL*8 ALPHA /0.125/
 C DELTA is δ : the death rate from AIDS.
 REAL*8 DELTA /1.000/
 C .. Executable Statements ..

C $F(1) = \frac{dX_1(t)}{dt}, F(2) = \frac{dX_2(t)}{dt}, F(3) = \frac{dX_3(t)}{dt}, F(4) = \frac{dX_4(t)}{dt}$
 $F(1) = \text{LAMDMU} - \text{NMU} * \text{BETAHT} * Y(1) * (Y(5) + Y(6)) / (Y(13) + Y(14)) - \text{MUMU} * Y(1)$
 $+ -C12 * \text{BETAFM} * Y(1) * Y(6) / Y(14) - C14 * \text{BETAFM} * Y(1) * Y(8) / Y(16)$
 $F(2) = \text{LAMDFU} - \text{NFU} * \text{BETAHT} * Y(2) * (Y(5) + Y(6)) / (Y(13) + Y(14)) - \text{MUFU} * Y(2)$
 $+ -C21 * \text{BETAMF} * Y(2) * Y(5) / Y(13) - C23 * \text{BETAMF} * Y(2) * Y(7) / Y(15)$
 $F(3) = \text{LAMDMN} - \text{MUMN} * Y(3) - \text{RHOMN} * C32 * \text{BETAFM} * Y(3) * Y(6) / Y(14)$
 $+ -C34 * \text{BETAFM} * Y(3) * Y(8) / Y(16)$
 $F(4) = \text{LAMDFN} - \text{MUFN} * Y(4) - \text{RHOFN} * C41 * \text{BETAMF} * Y(4) * Y(5) / Y(13)$
 $+ -C43 * \text{BETAMF} * Y(4) * Y(7) / Y(15)$

C $F(5) = \frac{dY_1(t)}{dt}, F(6) = \frac{dY_2(t)}{dt}, F(7) = \frac{dY_3(t)}{dt}, F(8) = \frac{dY_4(t)}{dt}$
 $F(5) = \text{NMU} * \text{BETAHT} * Y(1) * (Y(5) + Y(6)) / (Y(13) + Y(14)) - (\text{ALPHA} + \text{MUMU}) * Y(5)$
 $+ +C12 * \text{BETAFM} * Y(1) * Y(6) / Y(14) + C14 * \text{BETAFM} * Y(1) * Y(8) / Y(16)$
 $F(6) = \text{NFU} * \text{BETAHT} * Y(2) * (Y(5) + Y(6)) / (Y(13) + Y(14)) - (\text{ALPHA} + \text{MUFU}) * Y(6)$
 $+ +C21 * \text{BETAMF} * Y(2) * Y(5) / Y(13) + C23 * \text{BETAMF} * Y(2) * Y(7) / Y(15)$
 $F(7) = -(\text{ALPHA} + \text{MUMN}) * Y(7) + \text{RHOMN} * C32 * \text{BETAFM} * Y(3) * Y(6) / Y(14)$
 $+ +C34 * \text{BETAFM} * Y(3) * Y(8) / Y(16)$
 $F(8) = -(\text{ALPHA} + \text{MUFN}) * Y(8) + \text{RHOFN} * C41 * \text{BETAMF} * Y(4) * Y(5) / Y(13)$
 $+ +C43 * \text{BETAMF} * Y(4) * Y(7) / Y(15)$

C $F(9) = \frac{dA_1(t)}{dt}, F(10) = \frac{dA_2(t)}{dt}, F(11) = \frac{dA_3(t)}{dt}, F(12) = \frac{dA_4(t)}{dt}$
 $F(9) = \text{ALPHA} * Y(5) - (\text{DELTA} + \text{MUMU}) * Y(9)$
 $F(10) = \text{ALPHA} * Y(6) - (\text{DELTA} + \text{MUFU}) * Y(10)$
 $F(11) = \text{ALPHA} * Y(7) - (\text{DELTA} + \text{MUMN}) * Y(11)$
 $F(12) = \text{ALPHA} * Y(8) - (\text{DELTA} + \text{MUFN}) * Y(12)$

C $F(13) = \frac{dP_1(t)}{dt}, F(14) = \frac{dP_2(t)}{dt}, F(15) = \frac{dP_3(t)}{dt}, F(16) = \frac{dP_4(t)}{dt}$
 $F(13) = F(1) + F(5) + F(9)$
 $F(14) = F(2) + F(6) + F(10)$
 $F(15) = F(3) + F(7) + F(11)$
 $F(16) = F(4) + F(8) + F(12)$

RETURN

END

SUBROUTINE PEDERV(X,Y,PW)

C .. Scalar Arguments ..

C DOUBLE PRECISION X

C .. Array Arguments ..

C DOUBLE PRECISION PW(3,3), Y(3)

C .. Executable Statements ..

RETURN

END

SUBROUTINE OUT(X,Y)

```

C .. Parameters ..
  INTEGER    NOUT
  PARAMETER  (NOUT=6)
C .. Scalar Arguments ..
  DOUBLE PRECISION X
C .. Array Arguments ..
  DOUBLE PRECISION Y(16)
C .. Scalars in Common ..
  DOUBLE PRECISION H, XEND
  INTEGER    I
C .. Local Scalars ..
  INTEGER    J
C .. Intrinsic Functions ..
  INTRINSIC  DBLE, REAL
C .. Common blocks ..
  COMMON     XEND, H, I
C .. Executable Statements ..
  WRITE (NOUT,FMT=99999) X, (Y(J),J=1,4)
  WRITE (NOUT,FMT=99999) X, (Y(J),J=5,8)
  WRITE (NOUT,FMT=99999) X, (Y(J),J=9,12)
  WRITE (NOUT,FMT=99999) X, (Y(J),J=13,16)
  X = XEND - DBLE(REAL(I))*H
  I = I - 1
  RETURN
C
99999 FORMAT (' ',F7.2,4D13.5)
  END

```

Appendix 3

Numerical Solutions to Differential Equations

Adams Method (Hall and Watt, 1976)

The non-stiff version of Gear's method is based upon ABM methods (Adams-Bashforth-Moulton methods). The ABM method is a combination of the Adams-Bashforth method (AB) and the Adams-Moulton method (AM). The AB method is an explicit linear multi step method (LMM) of Adams type and the AM method is an implicit linear multi step method of Adams type.

The Adams method of k -steps, is defined by the choice of the polynomial, $\rho(\theta)$, given below, to assure strong stability, through all the extraneous roots being at the origin.

$$\rho(\theta) = \alpha_k(\theta^k - \theta^{k-1})$$

Adams-Bashforth-Moulton Method (Mathews, 1992)

The Adams-Bashforth-Moulton method to approximate the solution of the initial value problem $y' = f(t, y)$ and $y(a) = y_0$ over $[a, b]$ is based upon the theorem, given by (1).

$$y(t_{k+1}) = y(t_k) + \int_{t_k}^{t_{k+1}} f(t, y(t)) dt \quad (1)$$

The Adams-Bashforth predictor, given by (2), is based upon the integration of (1) over the interval of $[t_k, t_{k+1}]$ and uses the Lagrange polynomial approximation for $f(t, y(t))$ for the points (t_{k-3}, f_{k-3}) , (t_{k-2}, f_{k-2}) , (t_{k-1}, f_{k-1}) , and (t_k, f_k) .

$$p_{k+1} = y_k + \frac{h}{24}[-9f_{k-3} + 37f_{k-2} - 59f_{k-1} + 55f_k] \quad (2)$$

The Adams-Moulton corrector, given by (3), is also based upon the integration of (1) over the interval of $[t_k, t_{k+1}]$ and uses the Lagrange polynomial approximation for $f(t, y(t))$ for the points (t_{k-2}, f_{k-2}) , (t_{k-1}, f_{k-1}) , (t_k, f_k) , and (t_{k+1}, f_{k+1}) . The new point $(t_{k+1}, f_{k+1}) = (t_{k+1}, f(t_{k+1}, p_{k+1}))$ uses the value p_{k+1} equated by (2).

$$y_{k+1} = y_k + \frac{h}{24}[f_{k-2} - 5f_{k-1} + 19f_k + 9f_{k+1}] \quad (3)$$

The algorithm for the Adams-Bashforth-Moulton method (Mathews, page 472, 1992).

```

INPUT A, B, Y(0)                                {Endpoint and initial value}
INPUT N                                           {Number of steps, N> 3}
H := [B-A]/N                                     {Compute the step size}
T(0) := A, F0 := F(T(0), Y(0))

FOR K = 1 TO 3 DO                                {Either input three additional
  T(K) := A + K*H                                starting values or compute them
  Get Y(K)                                        using the Runge-Kutta method}

F1 := F(T(1), Y(1)), F2 := F(T(2), Y(2)), F3 := F(T(3), Y(3))
H2 := H/24                                       {Saves wasted computations}

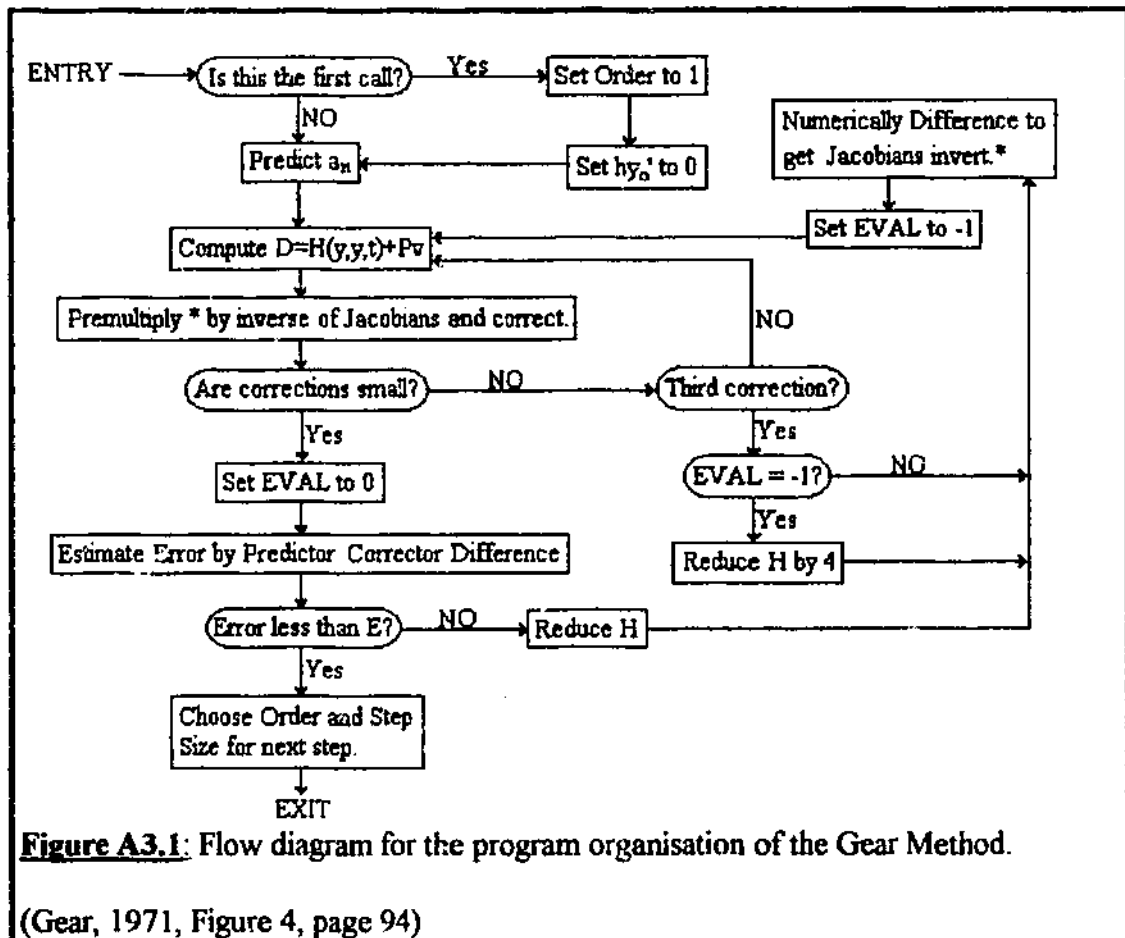
FOR K = 3 TO N-1 DO
  P := Y(K) + H2*[-9*F0+37*F1-59*F2+55*F3]      {Predictor}
  T(K+1) := A + H*[K+1]                         {Next abscissa}
  F4 := F(T(K+1), P)                            {Evaluate f(t,y)}
  Y(K+1) := Y(K) + H2*[F1-5*F2+19*F3+9*F4]      {Corrector}
  F0 := F1, F1 := F2, F2 := F3                  {Update the values}
  F3 := F(T(K+1), Y(K+1))

FOR K = 0 TO N DO                                {Output}
  PRINT T(K), Y(K)

```

The Gear Method (Gear, 1971)

The following figure provides the best description of what the Gear method is. Further details of this method are not provided as they are beyond the scope of this thesis.



APPENDIX 4:

Figures of Model Results

Table A4.1: Legend for figures of results of simulations conducted.			
SIMULATION REFERENCE	INFECTIVE(S) INTRODUCED	NEEDLE SHARING 'PARTNERS'	CONDOM USAGE (% OF THE TIME)
MODEL A:			
Table 4.1	1 male IVDU	1.6 per year	16.5%
Table 4.2	1 male IVDU	1 per year	16.5%
Table 4.3	1 male IVDU	1.6 per year	35.5%
Table 4.4	1 male IVDU	1 per year	35.5%
MODEL B:			
Table 4.5	1 male non-IVDU	1.6 per year	16.5%
Table 4.6	1 male non-IVDU	1 per year	16.5%
Table 4.7	1 male non-IVDU	1.6 per year	35.5%
Table 4.8	1 male non-IVDU	1 per year	35.5%
MODEL C:			
Table 4.9	1 male IVDU 3 male non-IVDUs & 1 female non-IVDU	1.6 per year	16.5%
Table 4.10	1 male IVDU 3 male non-IVDUs & 1 female non-IVDU	1 per year	16.5%
Table 4.11	1 male IVDU 3 male non-IVDUs & 1 female non-IVDU	1.6 per year	35.5%
Table 4.12	1 male IVDU 3 male non-IVDUs & 1 female non-IVDU	1 per year	35.5%

Table A4.2: Legend for figures of results of simulations where modifications on behaviour patterns were made in 1994.

SIMULATION REFERENCE	MODIFICATIONS TO ORIGINAL MODEL *
1994/1	In 1994 the number of needle sharing 'partners' was reduced from 1.6 to 1 per year. Condom usage remains at 16.5% of the time.
1994/2	In 1994 condom usage was increased from 16.5% to 35.5% of the time. Needle sharing 'partners' remains at 1.6 per year.
1994/3	In 1994 the number of needle sharing 'partners' was reduced from 1.6 to 1 per year and condom usage was increased from 16.5% to 35.5% of the time.
1994/4	In 1994 the number of needle sharing 'partners' was reduced from 1.6 to 1 per year and condom usage was increased from 16.5% to 50% of the time.

* Original models are Table 4.1 for Model A, Table 4.5 for Model B and Table 4.9 for Model C.

A4.1 Results from Model A:

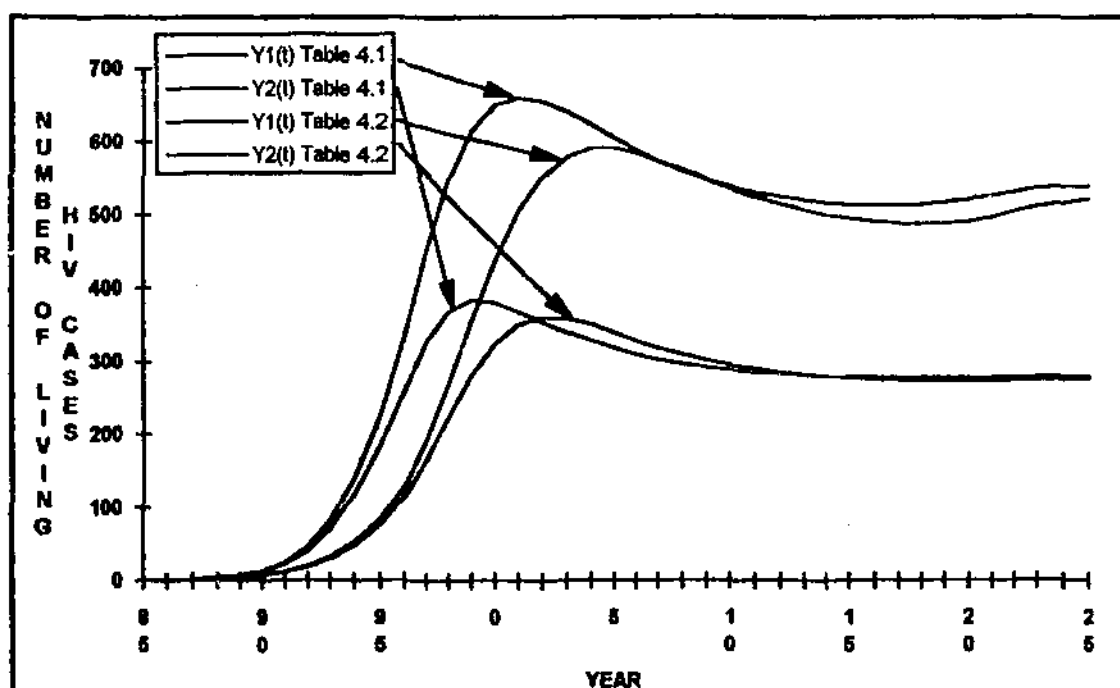
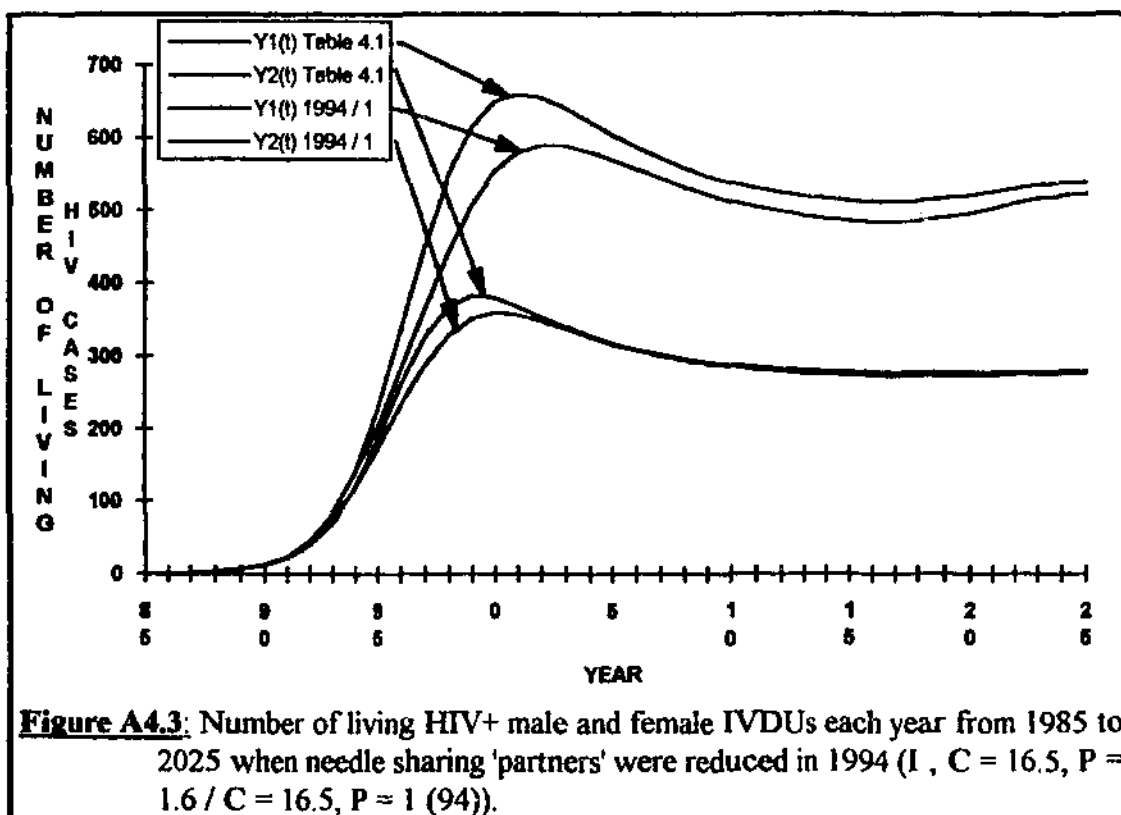
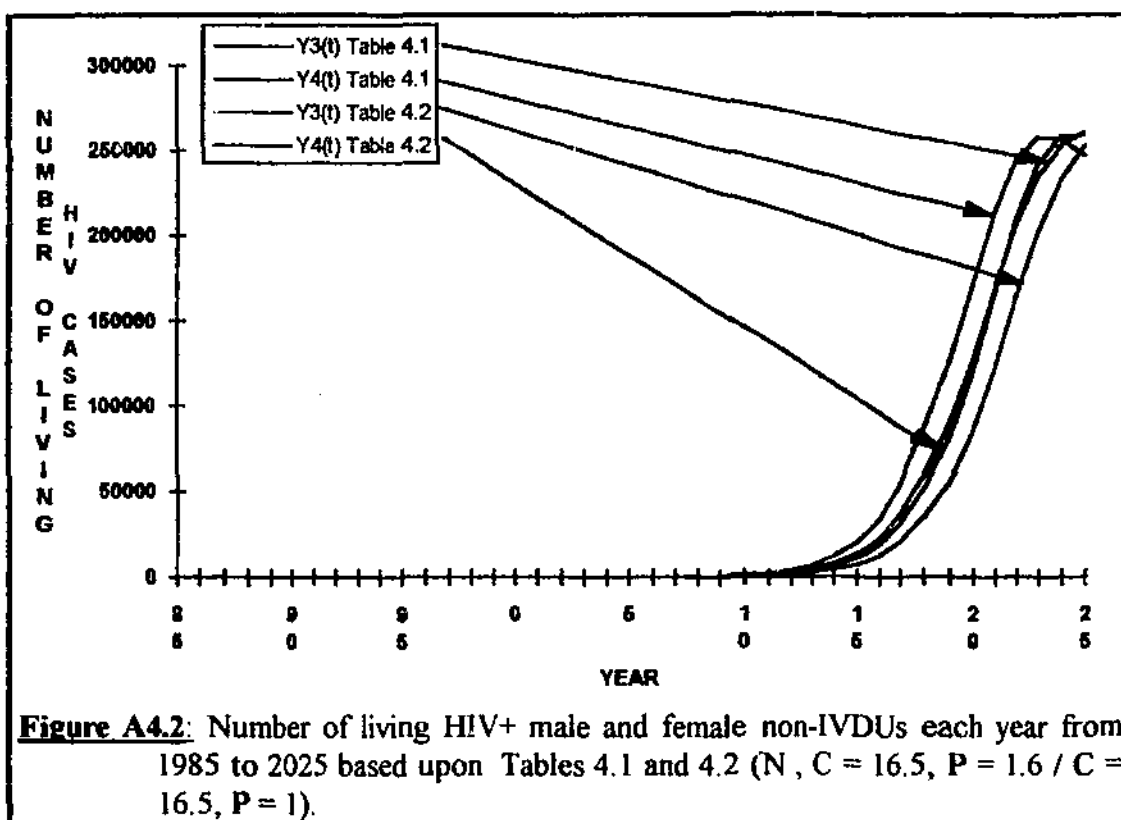


Figure A4.1: Number of living HIV+ male and female IVDUs each year from 1985 to 2025 based upon Tables 4.1 and 4.2 (I, C = 16.5, P = 1.6 / C = 16.5, P = 1).



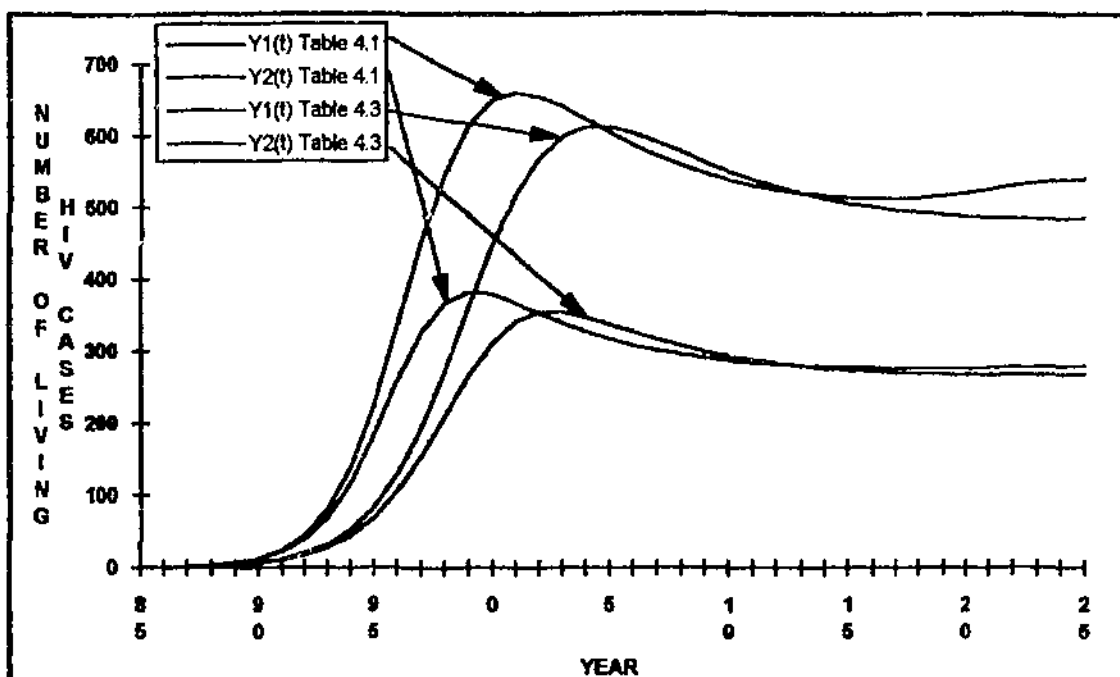


Figure A4.4: Number of living HIV+ male and female IVDUs each year from 1985 to 2025 based upon Tables 4.1 and 4.3 ($I, C = 16.5, P = 1.6 / C = 35.5, P = 1.6$).

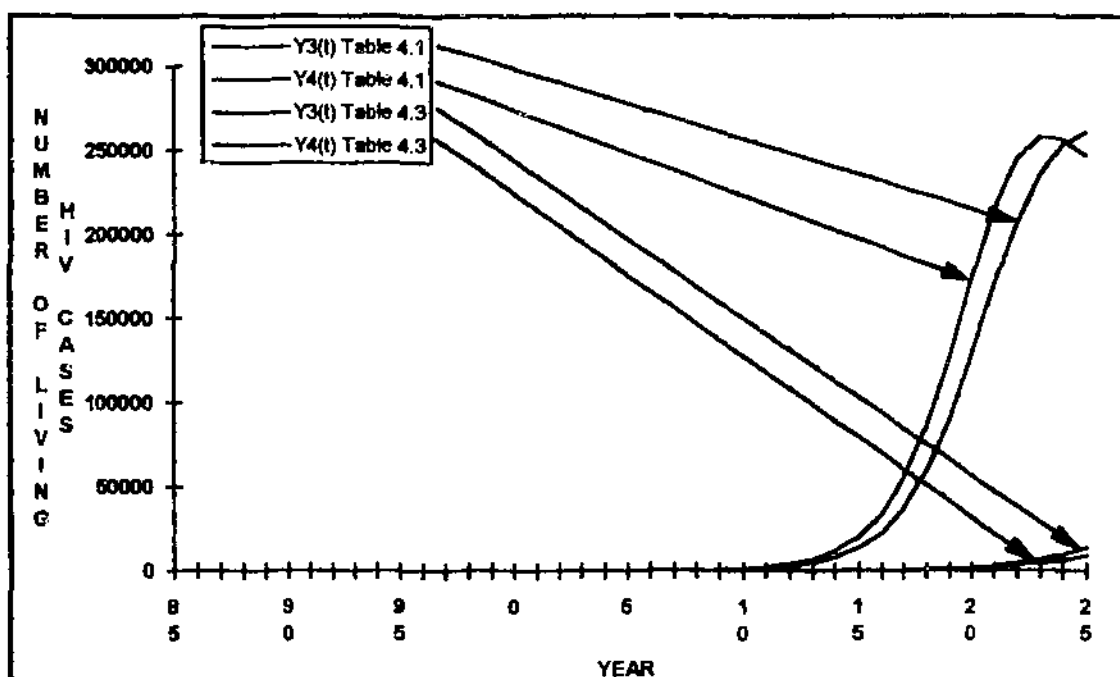


Figure A4.5: Number of living HIV+ male and female non-IVDUs each year from 1985 to 2025 based upon Tables 4.1 and 4.3 ($N, C = 16.5, P = 1.6 / C = 35.5, P = 1.6$).

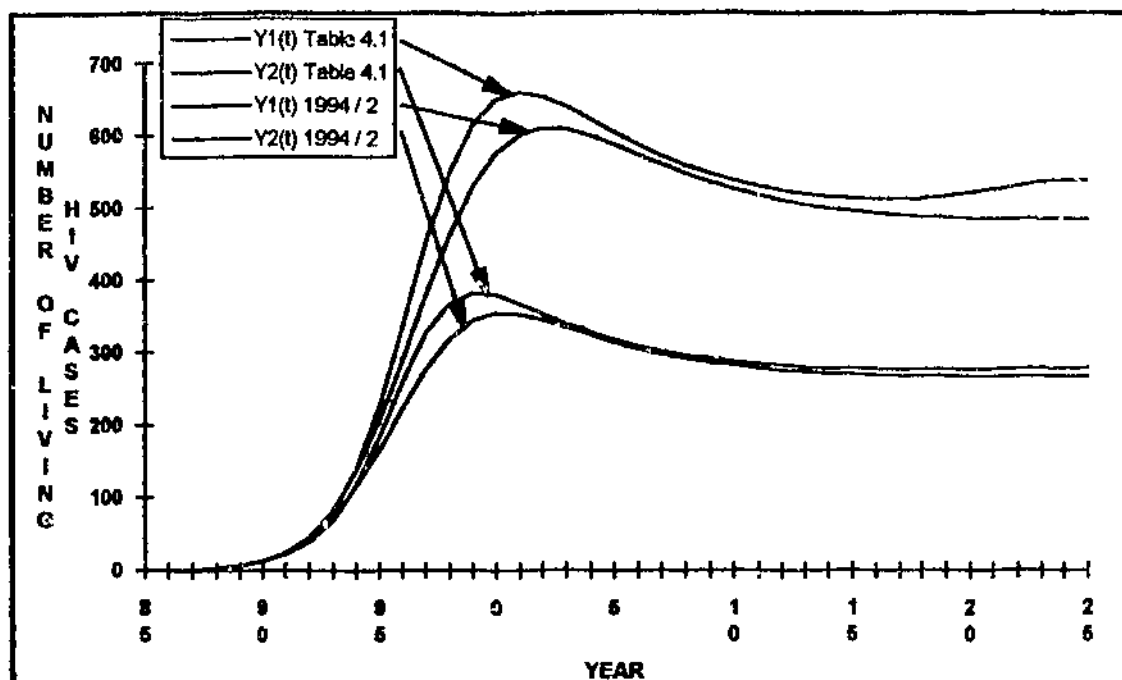


Figure A4.6: Number of living HIV+ male and female IVDUs each year from 1985 to 2025 when condom usage was increased in 1994 ($I, C = 16.5, P = 1.6 / C = 35.5$ (94), $P = 1.6$).

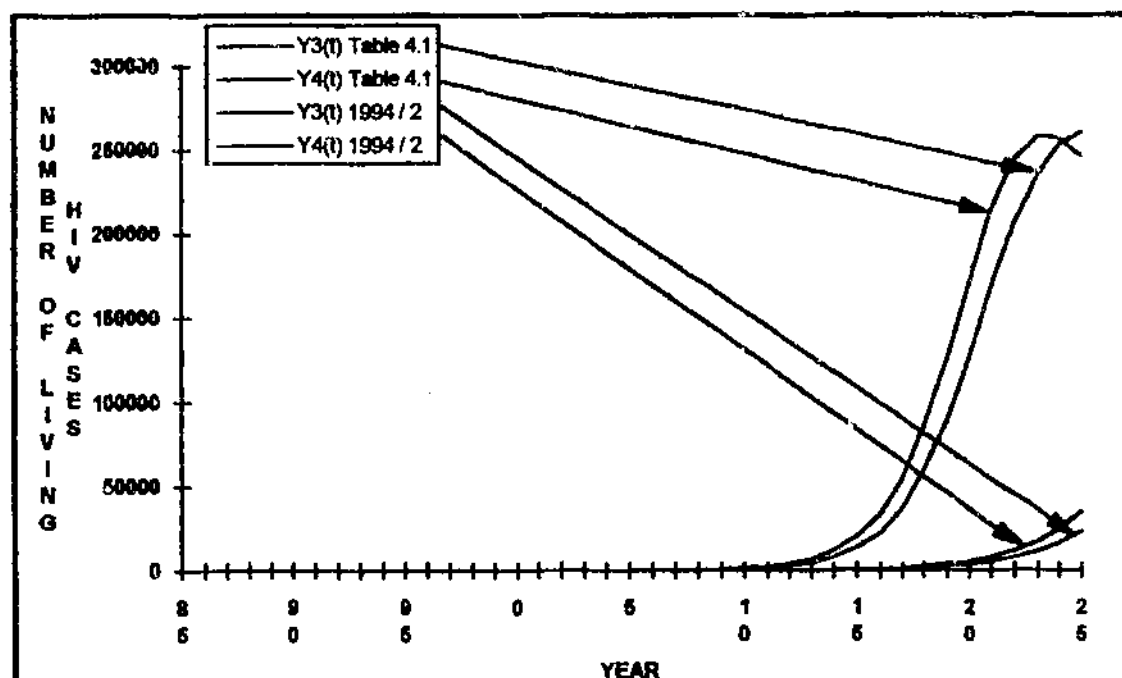


Figure A4.7: Number of living HIV+ male and female non-IVDUs each year from 1985 to 2025 when condom usage was increased in 1994 ($N, C = 16.5, P = 1.6 / C = 35.5$ (94), $P = 1.6$).

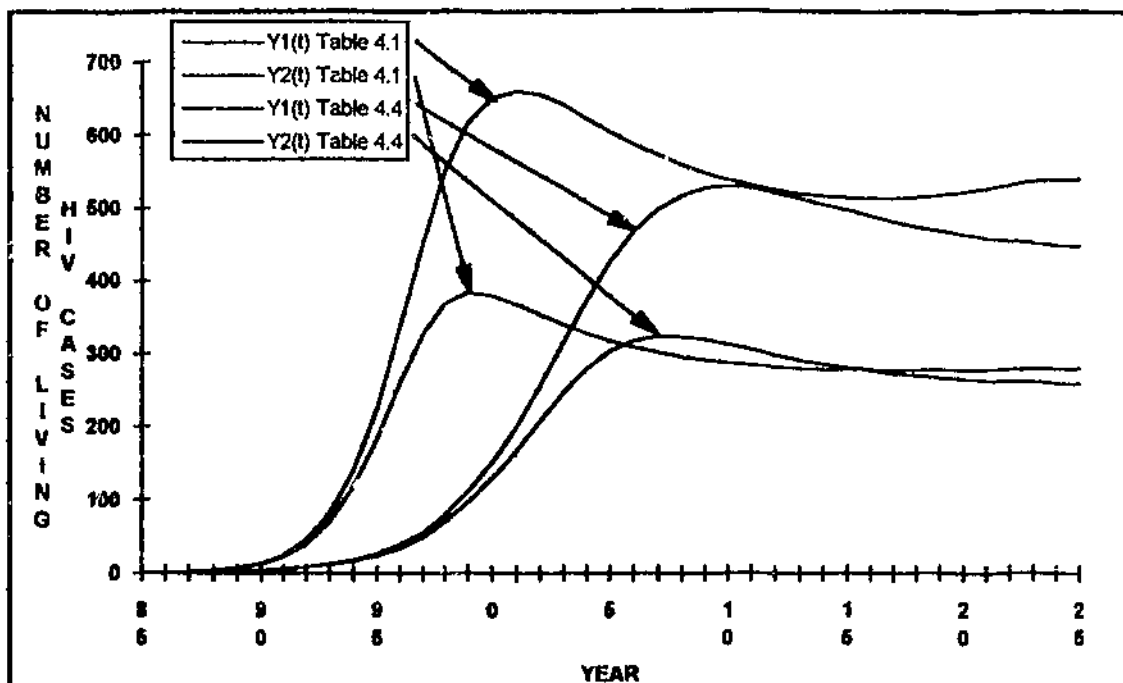


Figure A4.8: Number of living HIV+ male and female IVDUs each year from 1985 to 2025 based upon Tables 4.1 and 4.4 ($I, C = 16.5, P = 1.6 / C = 35.5, P = 1$).

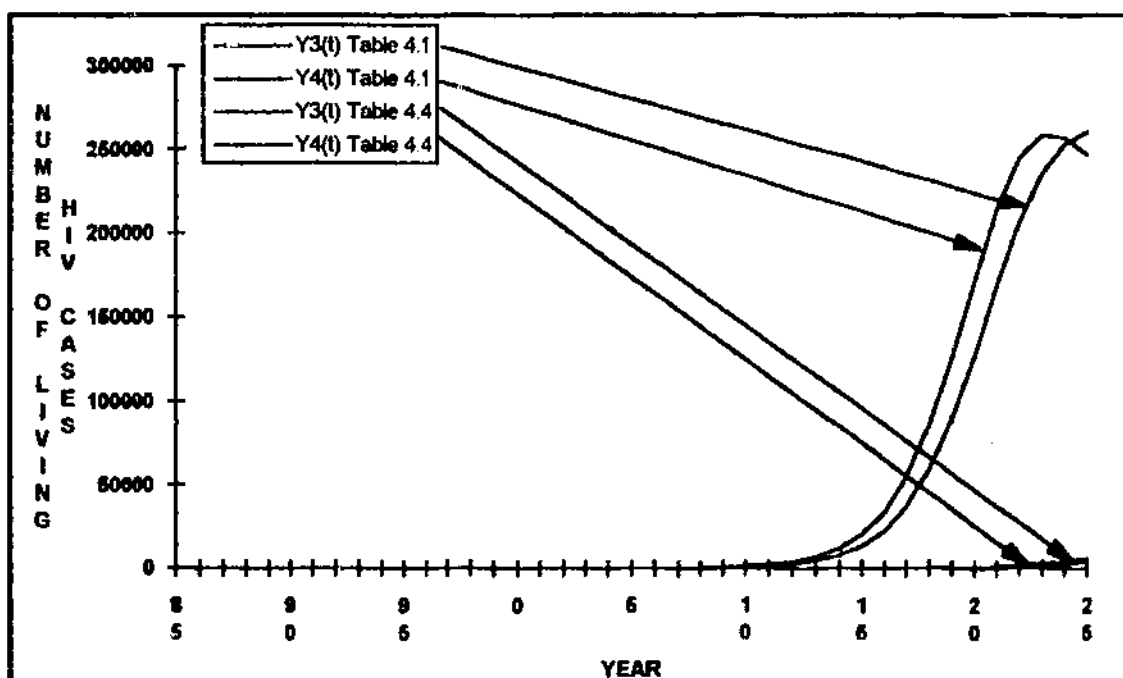


Figure A4.9: Number of living HIV+ male and female non-IVDUs each year from 1985 to 2025 based upon Tables 4.1 and 4.4 ($N, C = 16.5, P = 1.6 / C = 35.5, P = 1$).

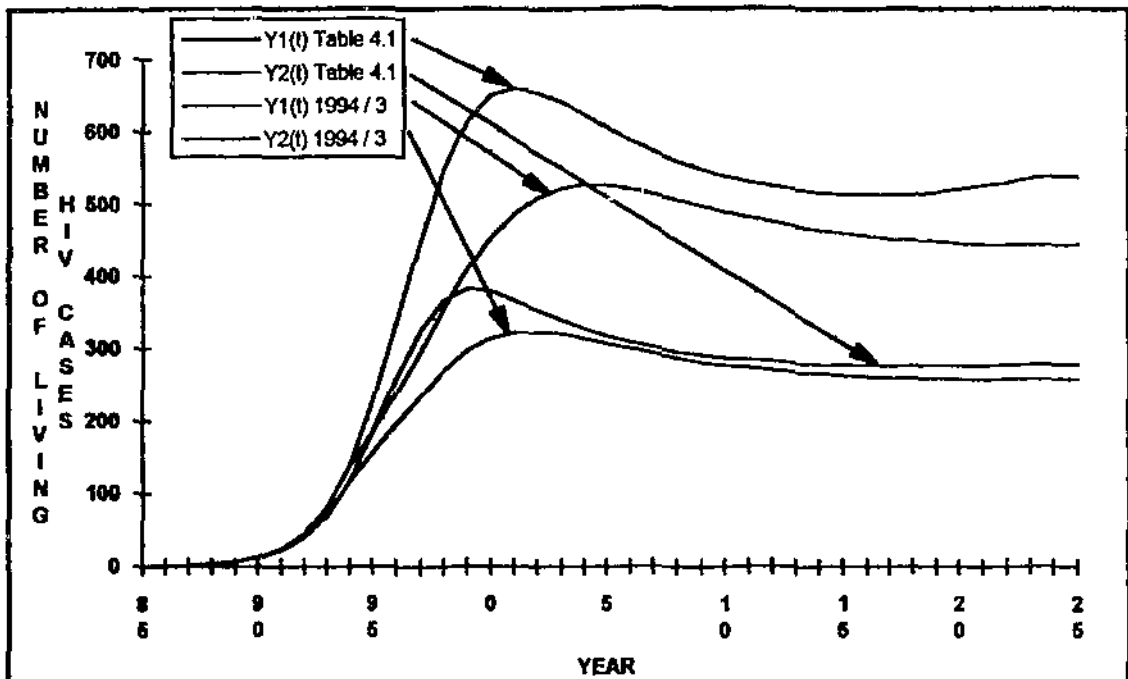


Figure A4.10: Number of living HIV+ male and female IVDUs each year from 1985 to 2025 when needle sharing 'partners' were reduced and condom usage was increased in 1994 ($I, C = 16.5, P = 1.6 / C = 35.5$ (94), $P = 1$ (94)).

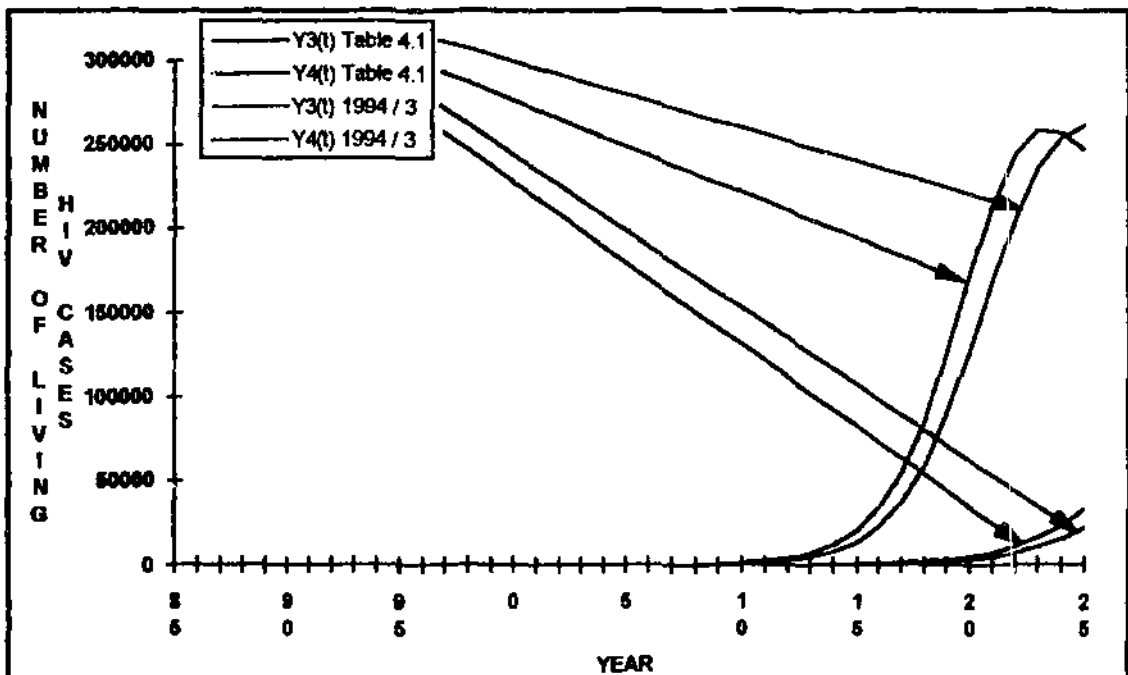


Figure A4.11: Number of living HIV+ male and female non-IVDUs each year from 1985 to 2025 when needle sharing 'partners' were reduced and condom usage was increased in 1994 ($N, C = 16.5, P = 1.6 / C = 35.5$ (94), $P = 1$ (94)).

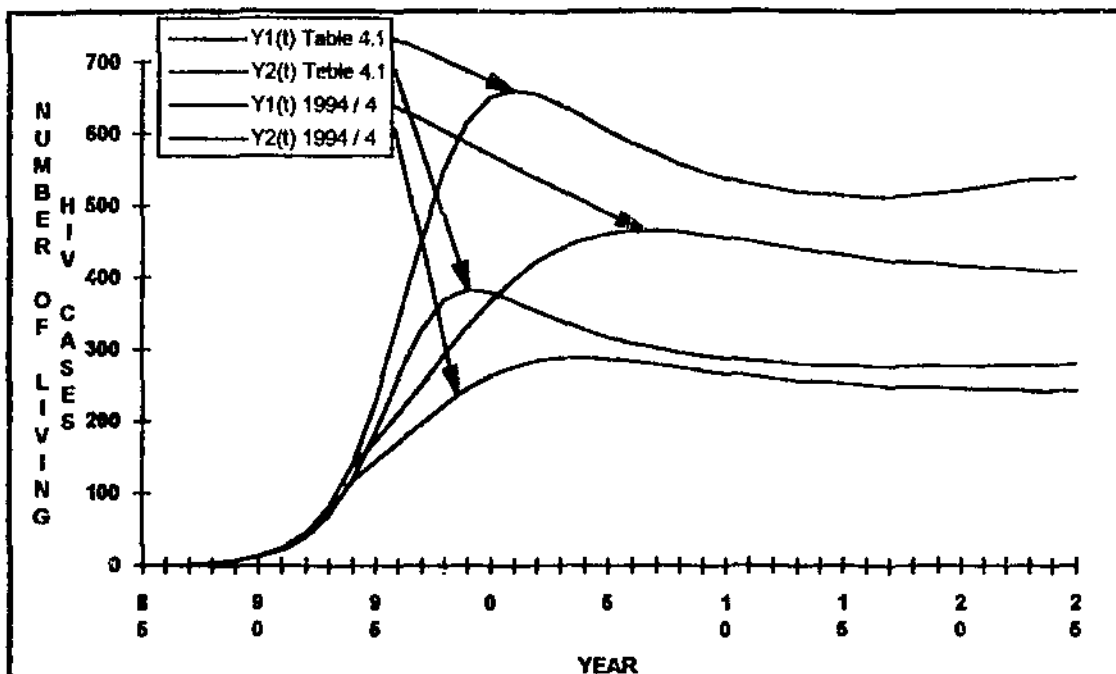


Figure A4.12: Number of living HIV+ male and female IVDUs each year from 1985 to 2025 when needle sharing 'partners' were reduced and condom usage was increased to 50% in 1994 ($I, C = 16.5, P = 1.6 / C = 50 (94), P = 1 (94)$)).

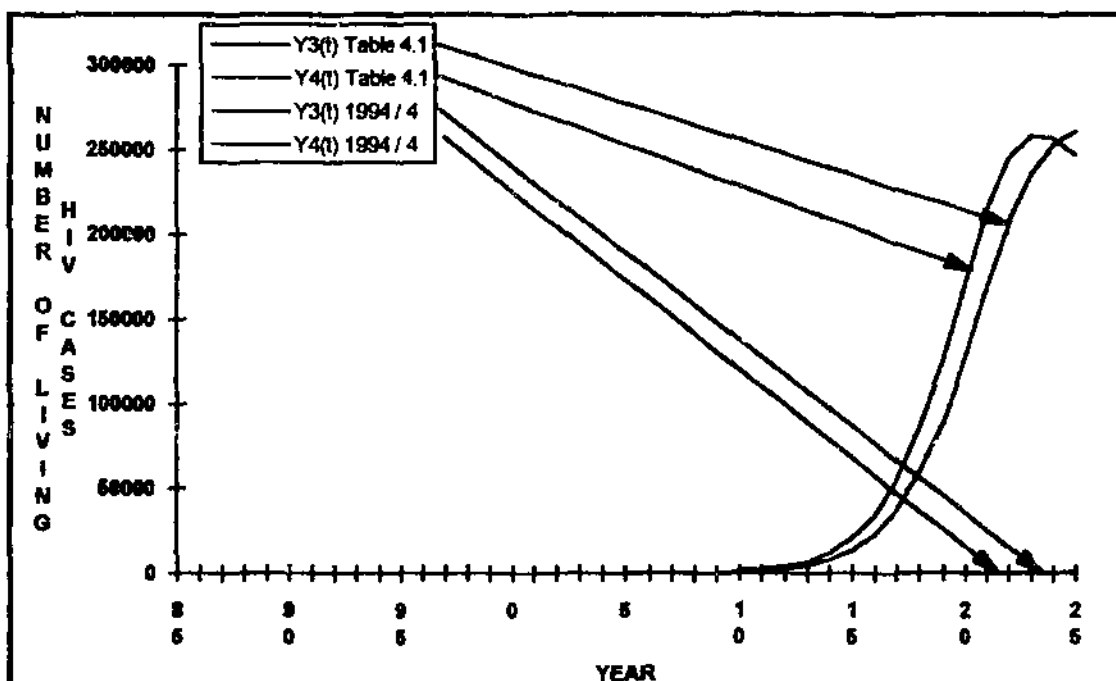


Figure A4.13: Number of living HIV+ male and female non-IVDUs each year from 1985 to 2025 when needle sharing 'partners' were reduced and condom usage was increased to 50% in 1994 ($N, C = 16.5, P = 1.6 / C = 50 (94), P = 1 (94)$)).

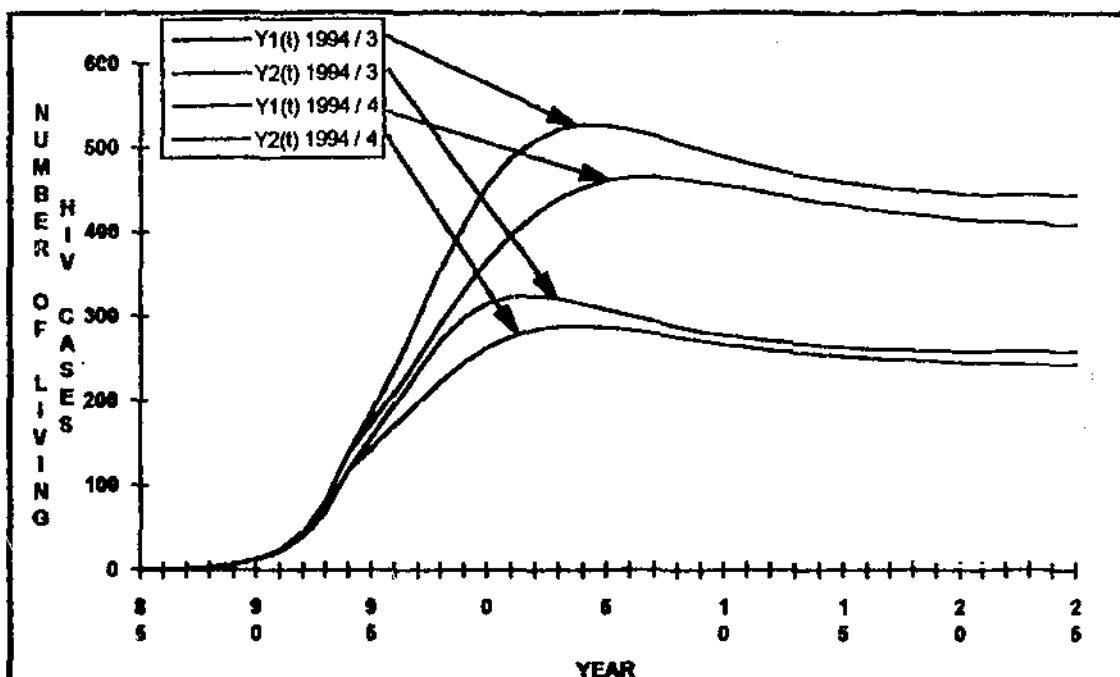


Figure A4.14: Number of living HIV+ male and female IVDUs each year from 1985 to 2025 when needle sharing 'partners' were reduced and condom usage was increased to 35.5% and to 50% in 1994 (I, C = 35.5 (94), P = 1 (94) / C = 50 (94), P = 1 (94)).

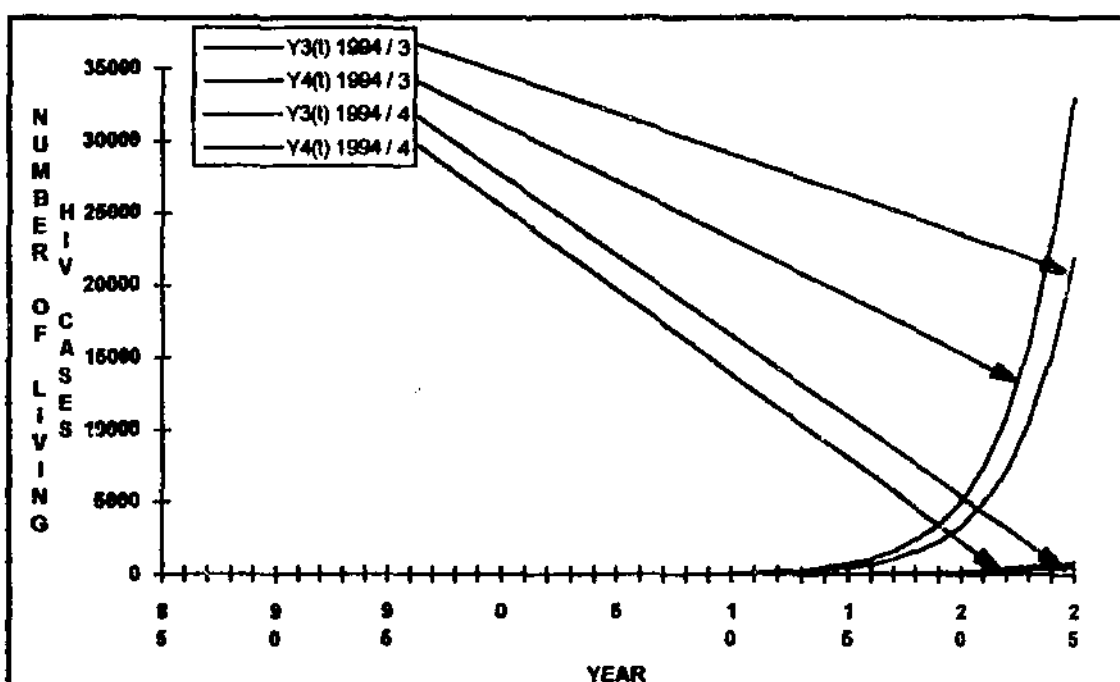
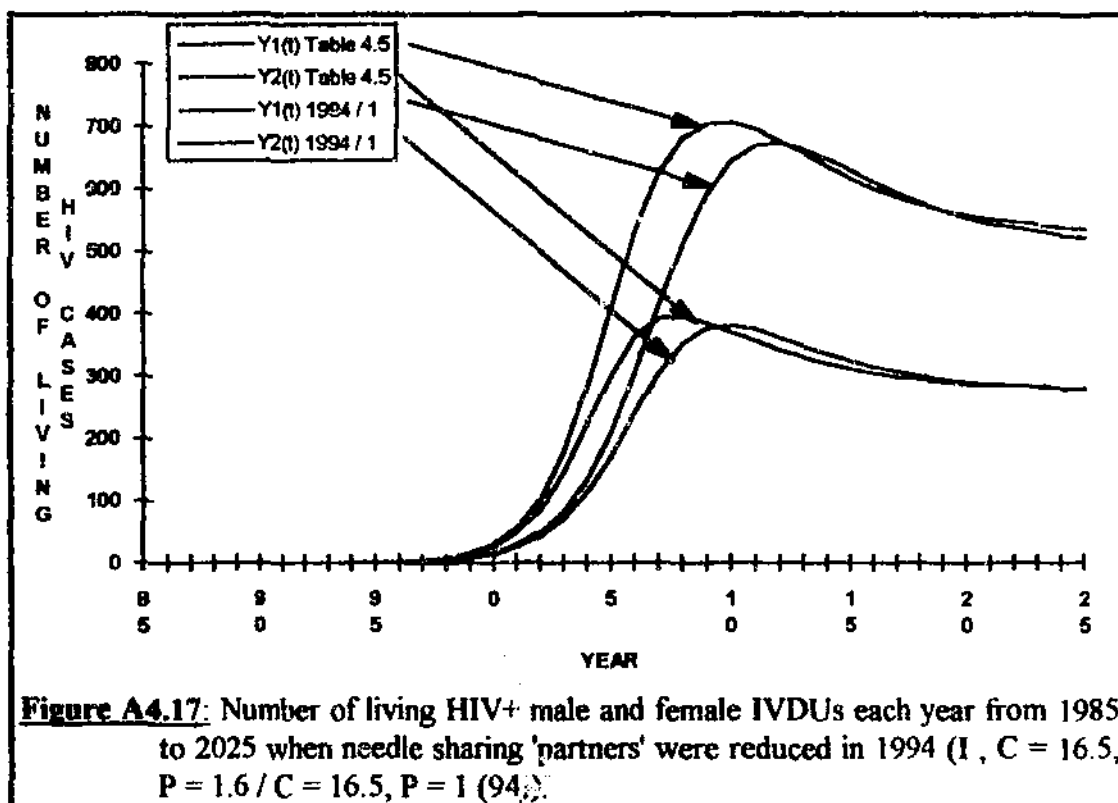
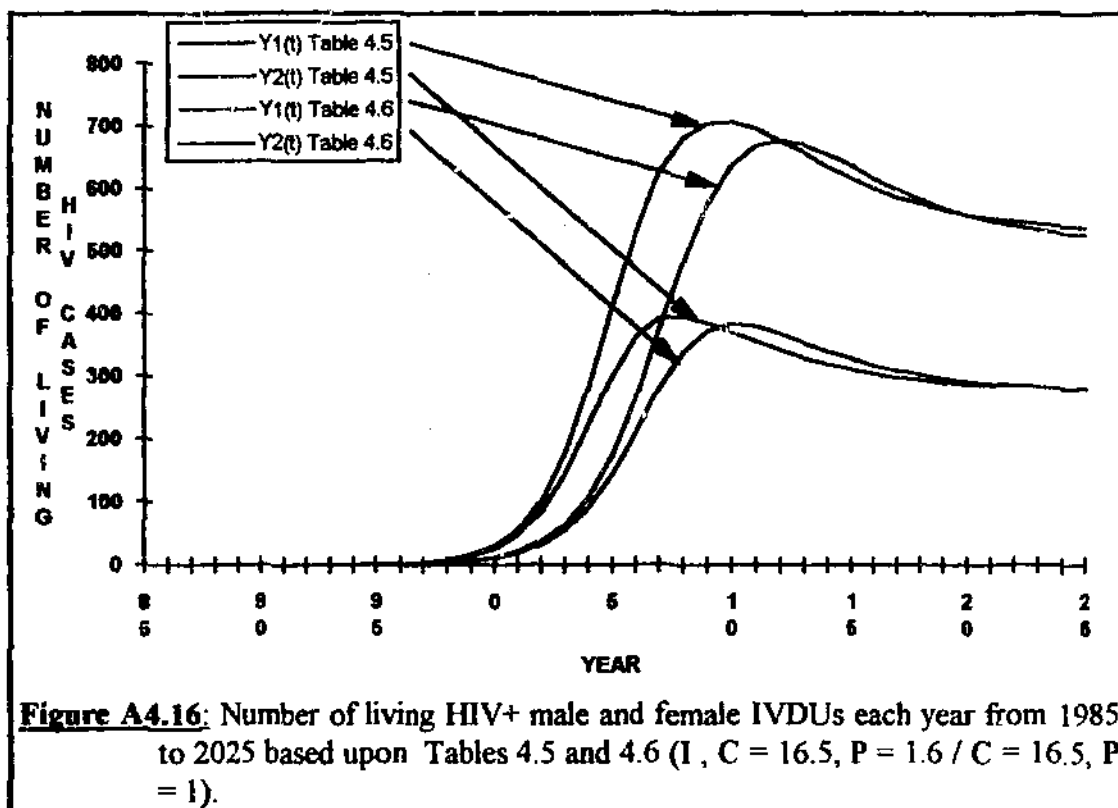
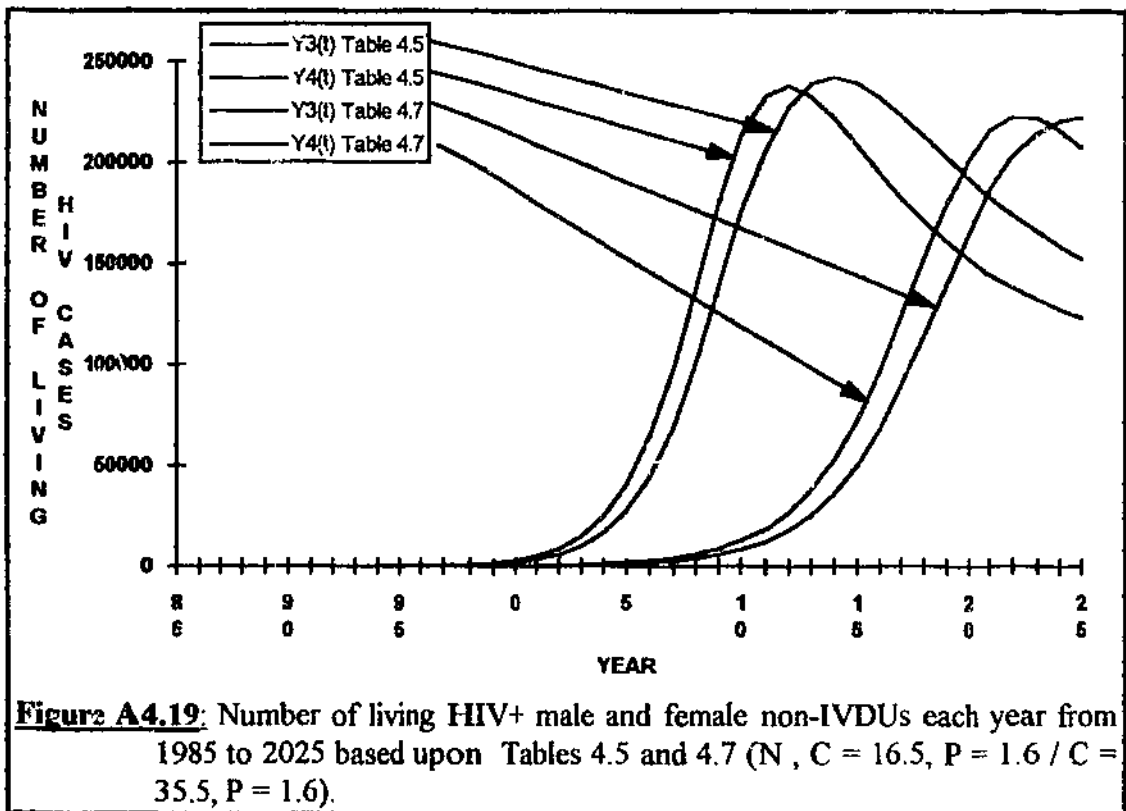
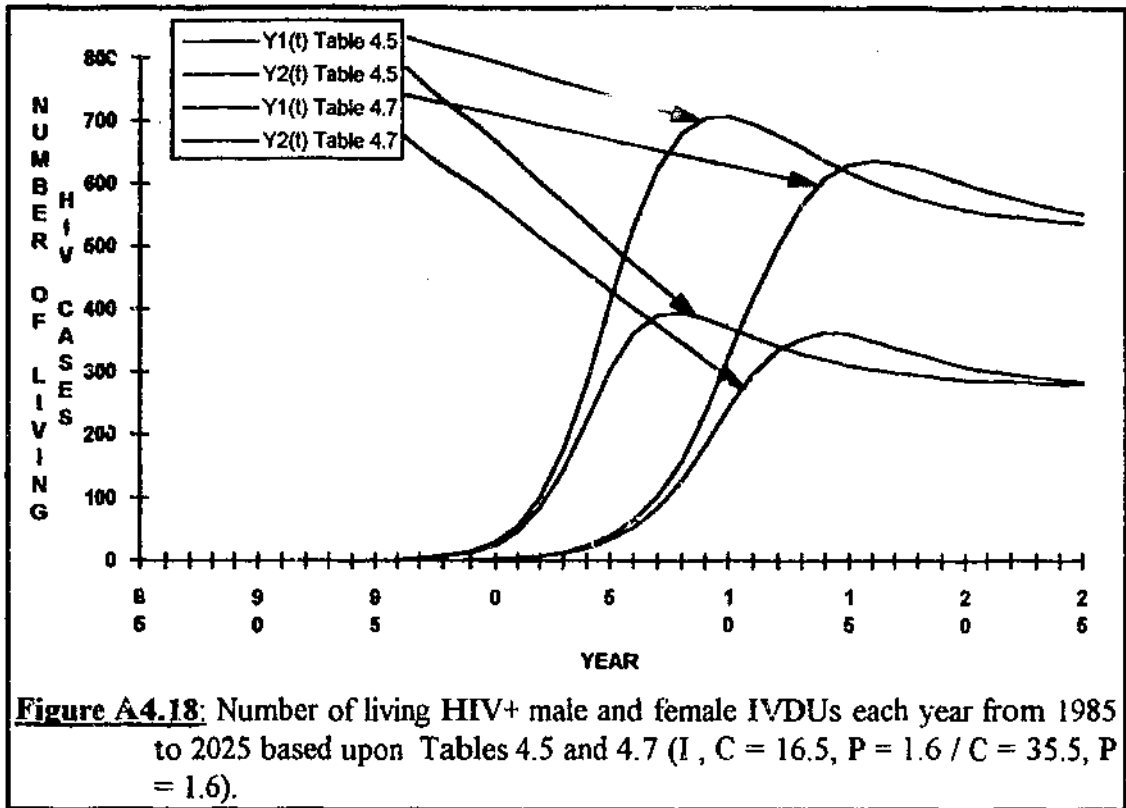


Figure A4.15: Number of living HIV+ male and female non-IVDUs each year from 1985 to 2025 when needle sharing 'partners' were reduced and condom usage was increased to 35.5% and to 50% in 1994 (N, C = 35.5 (94), P = 1 (94) / C = 50 (94), P = 1 (94)).

A4.2 Results from Model B:





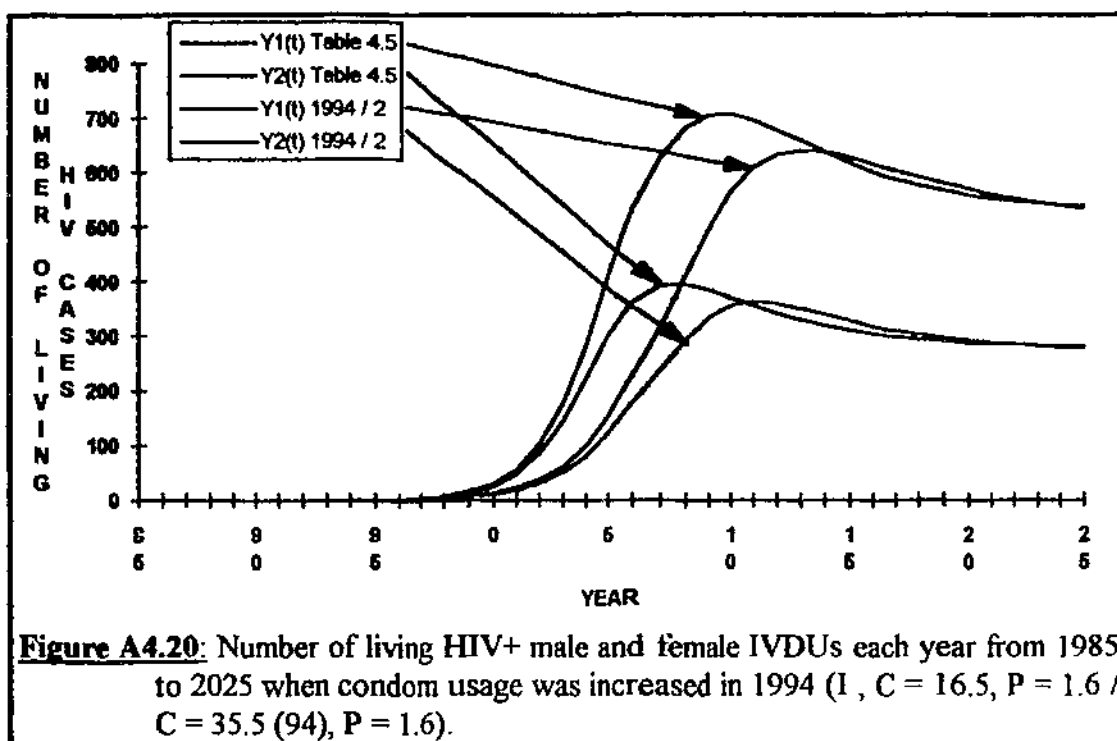


Figure A4.20: Number of living HIV+ male and female IVDUs each year from 1985 to 2025 when condom usage was increased in 1994 (I , $C = 16.5$, $P = 1.6$ / $C = 35.5$ (94), $P = 1.6$).

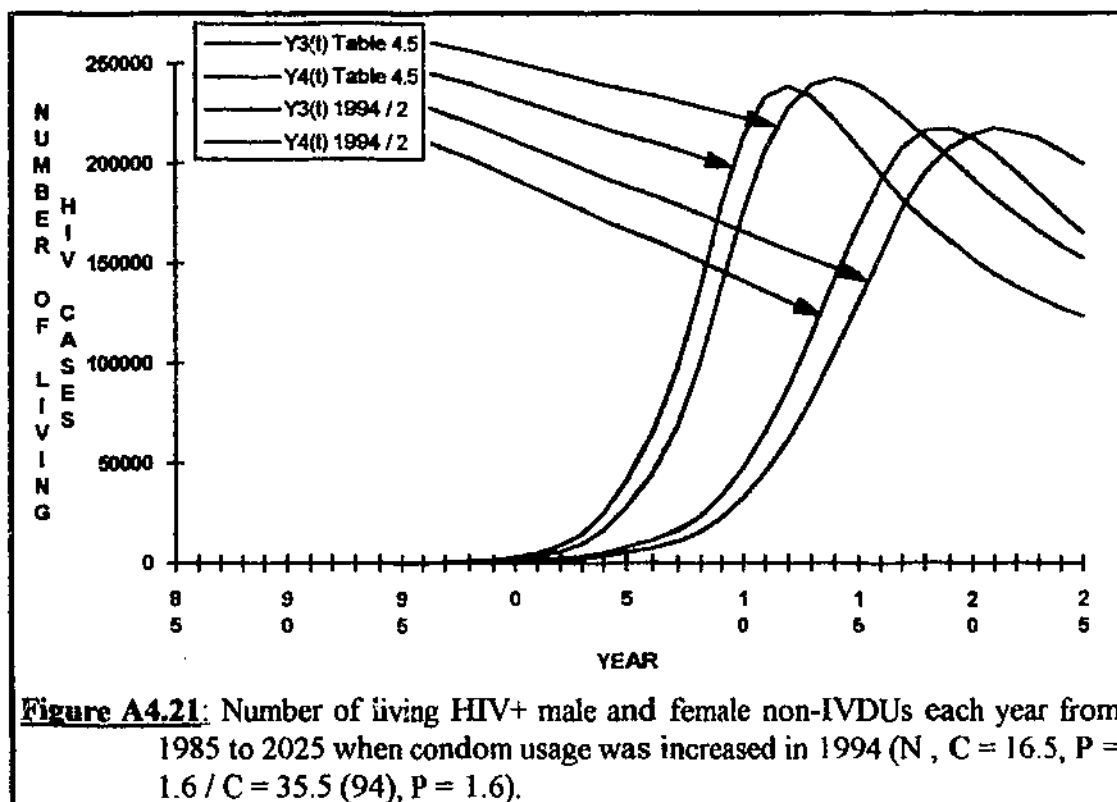
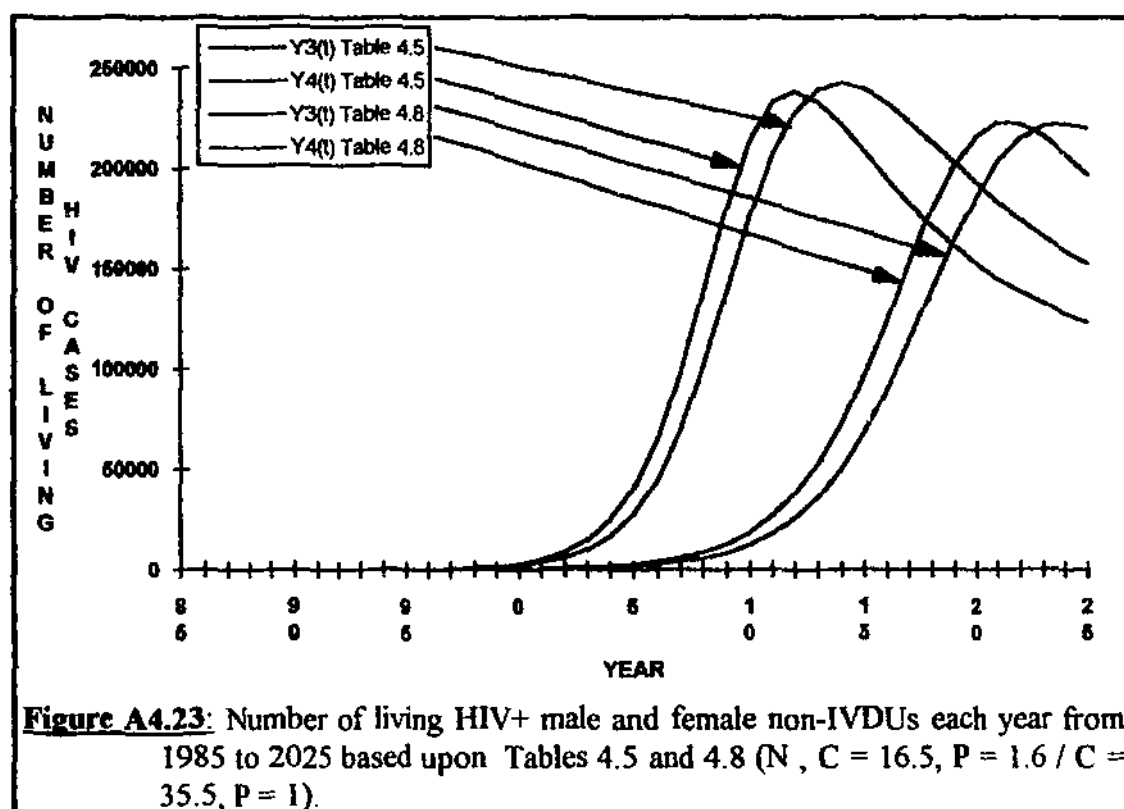
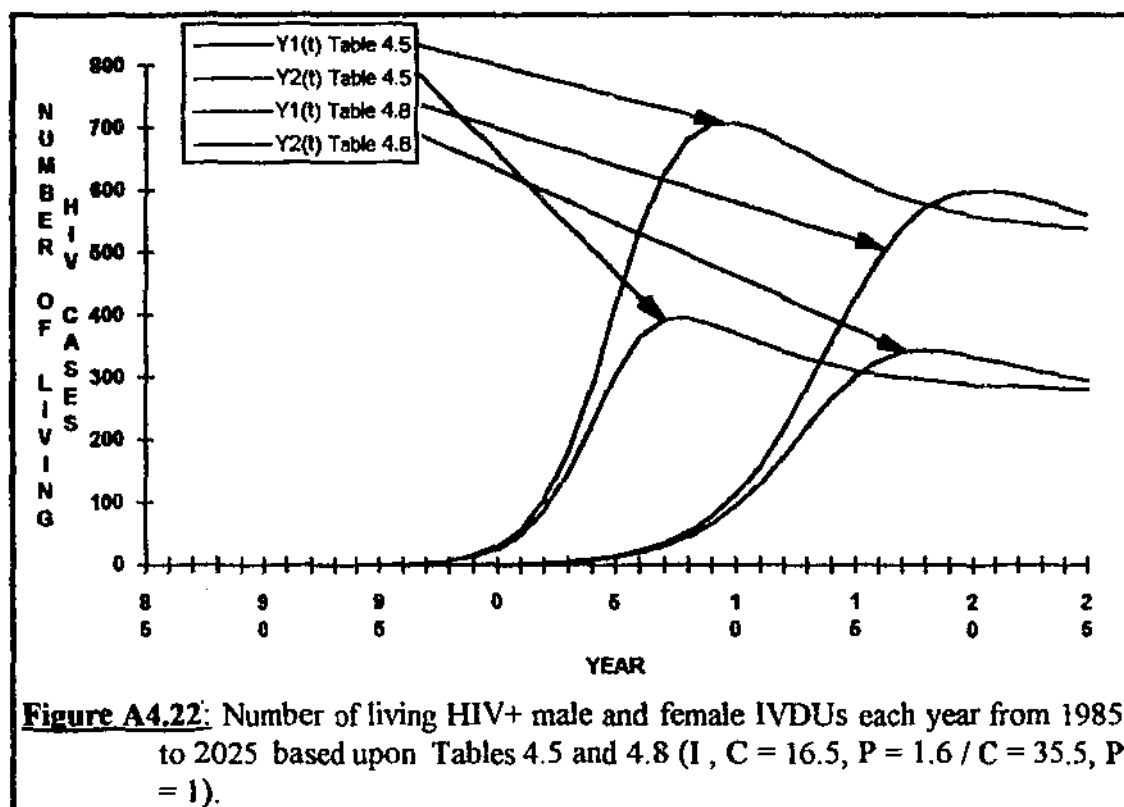


Figure A4.21: Number of living HIV+ male and female non-IVDUs each year from 1985 to 2025 when condom usage was increased in 1994 (N , $C = 16.5$, $P = 1.6$ / $C = 35.5$ (94), $P = 1.6$).



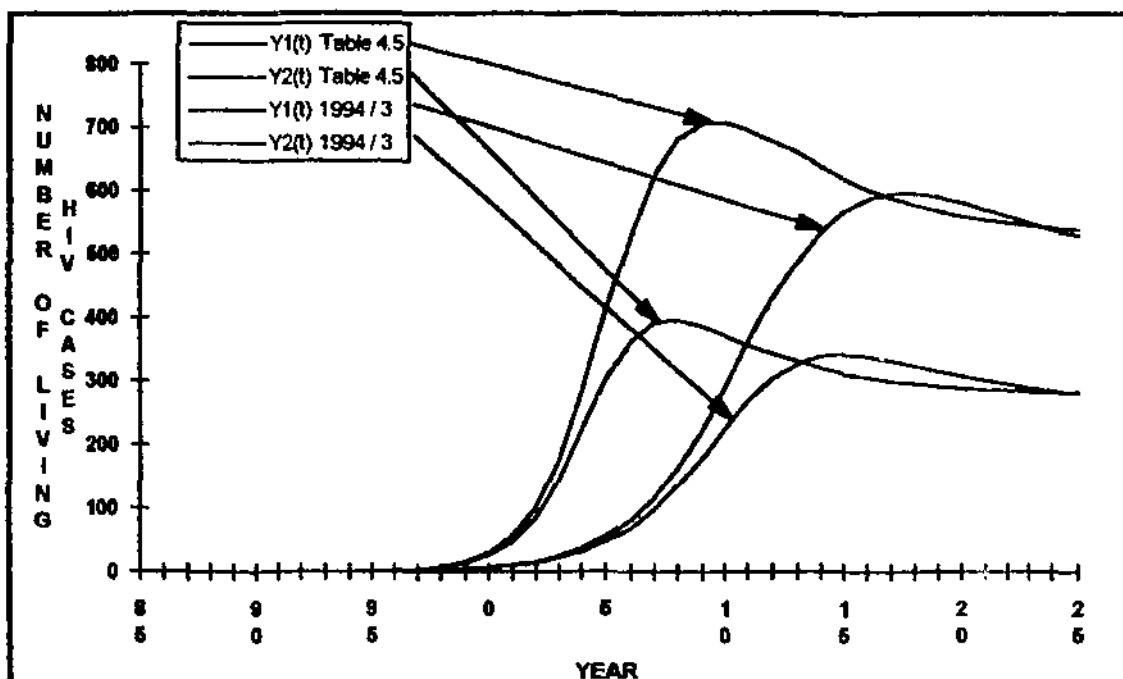


Figure A4.24: Number of living HIV+ male and female IVDUs each year from 1985 to 2025 when needle sharing 'partners' were reduced and condom usage was increased in 1994 ($I, C = 16.5, P = 1.6 / C = 35.5$ (94), $P = 1$ (94)).

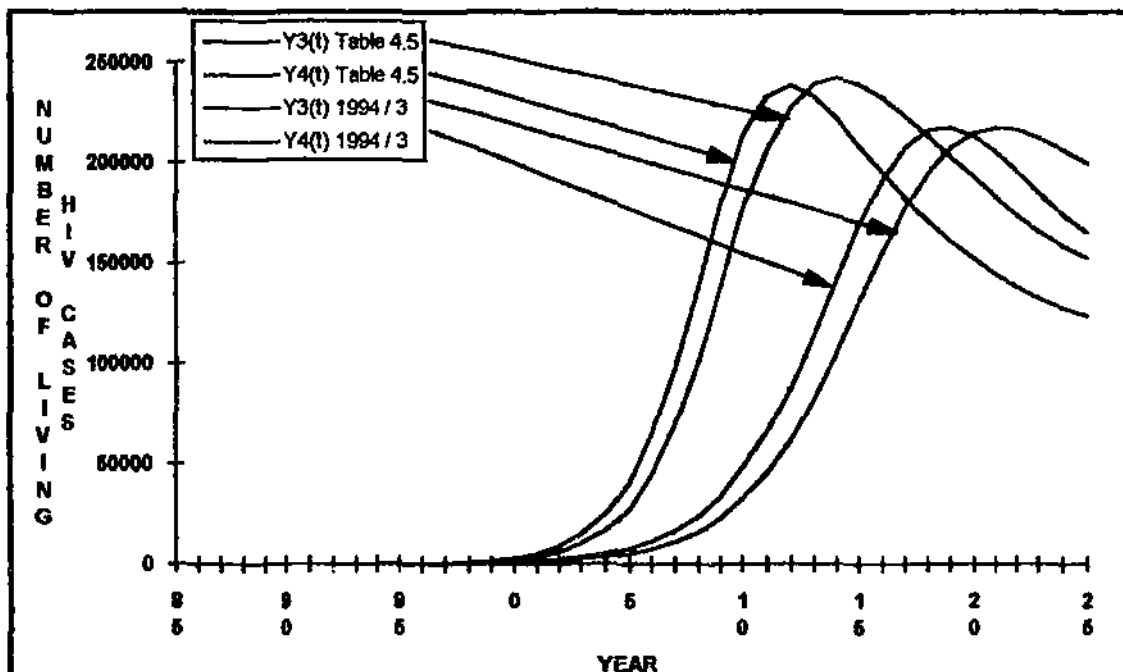
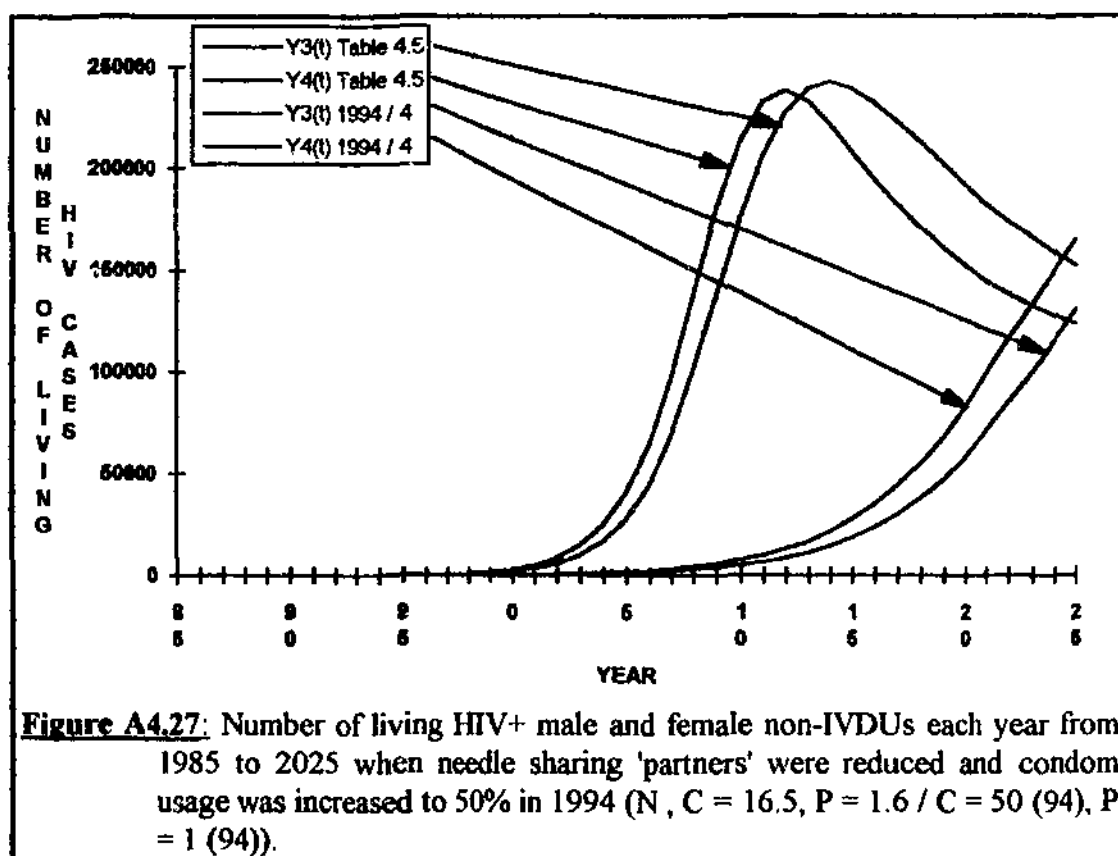
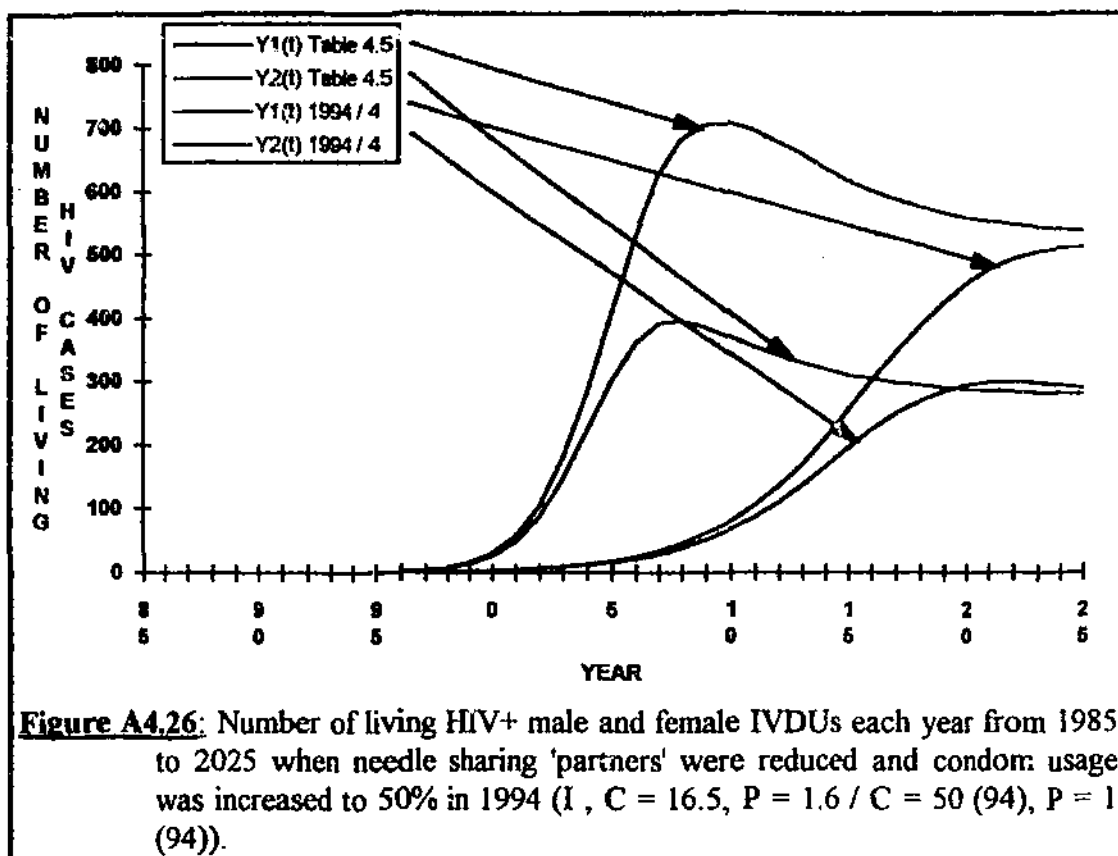


Figure A4.25: Number of living HIV+ male and female non-IVDUs each year from 1985 to 2025 when needle sharing 'partners' were reduced and condom usage was increased in 1994 ($N, C = 16.5, P = 1.6 / C = 35.5$ (94), $P = 1$ (94)).



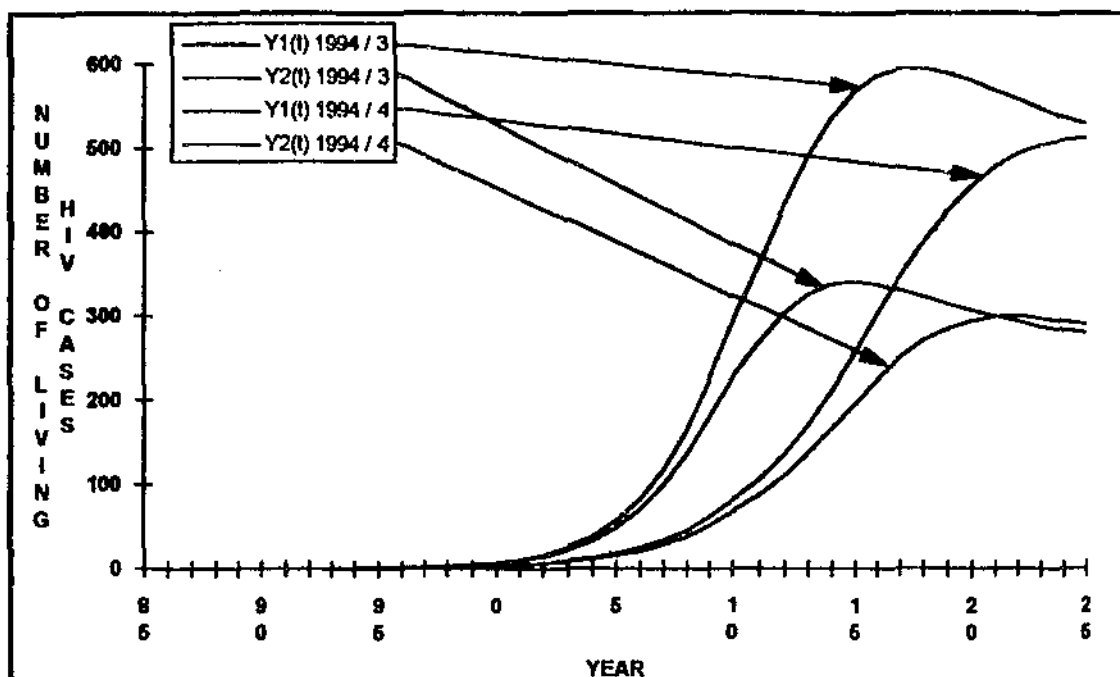


Figure A4.28: Number of living HIV+ male and female IVDUs each year from 1985 to 2025 when needle sharing 'partners' were reduced and condom usage was increased to 35.5% and to 50% in 1994 (I , $C = 35.5$ (94), $P = 1$ (94) / $C = 50$ (94), $P = 1$ (94)).

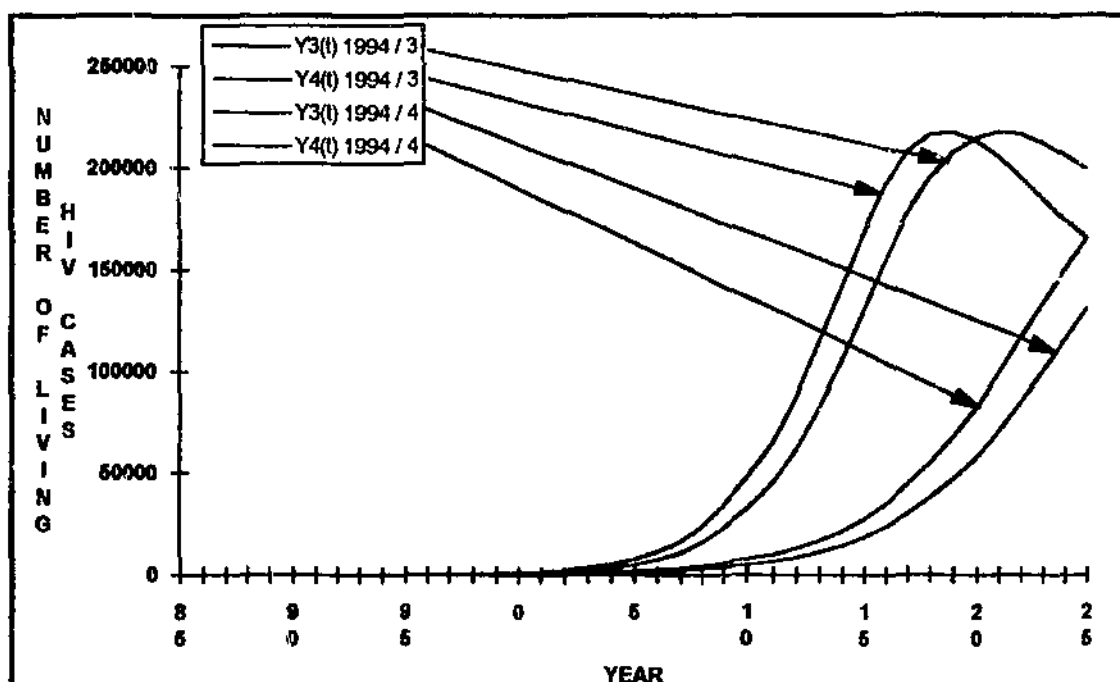
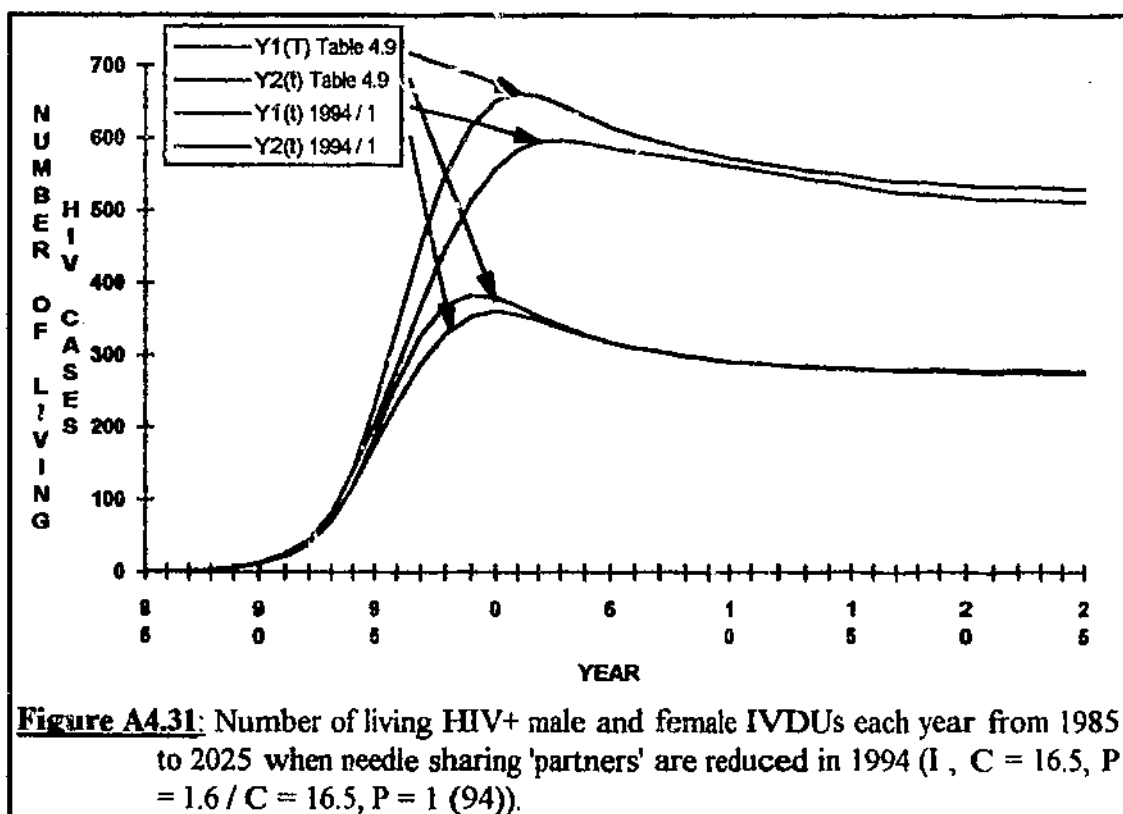
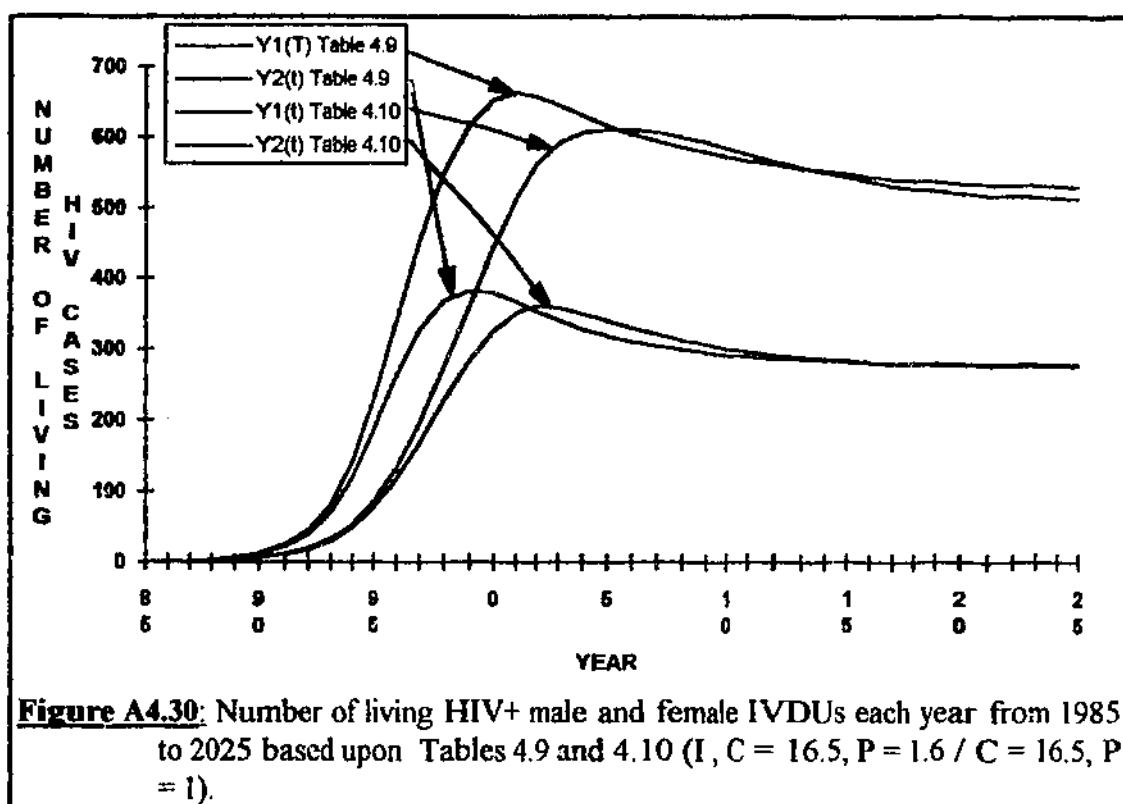


Figure A4.29: Number of living HIV+ male and female non-IVDUs each year from 1985 to 2025 when needle sharing 'partners' were reduced and condom usage was increased to 35.5% and to 50% in 1994 (N , $C = 35.5$ (94), $P = 1$ (94) / $C = 50$ (94), $P = 1$ (94)).

A4.3 Results from Model C:



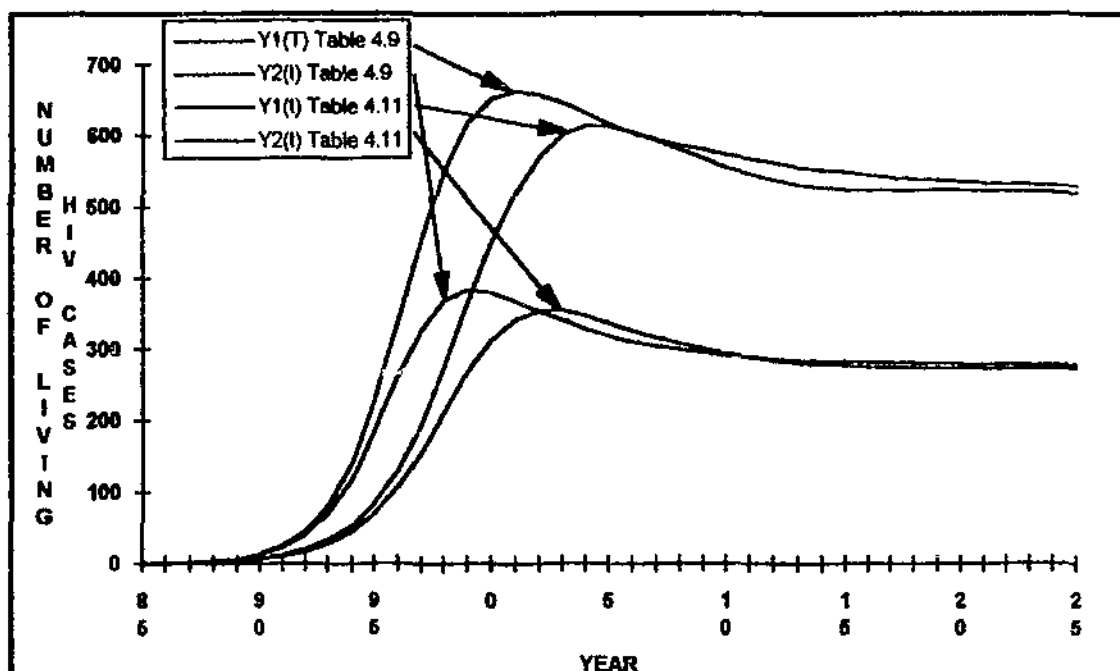


Figure A4.32: Number of living HIV+ male and female IVDUs each year from 1985 to 2025 based upon Tables 4.9 and 4.11 ($I, C = 16.5, P = 1.6 / C = 35.5, P = 1.6$).

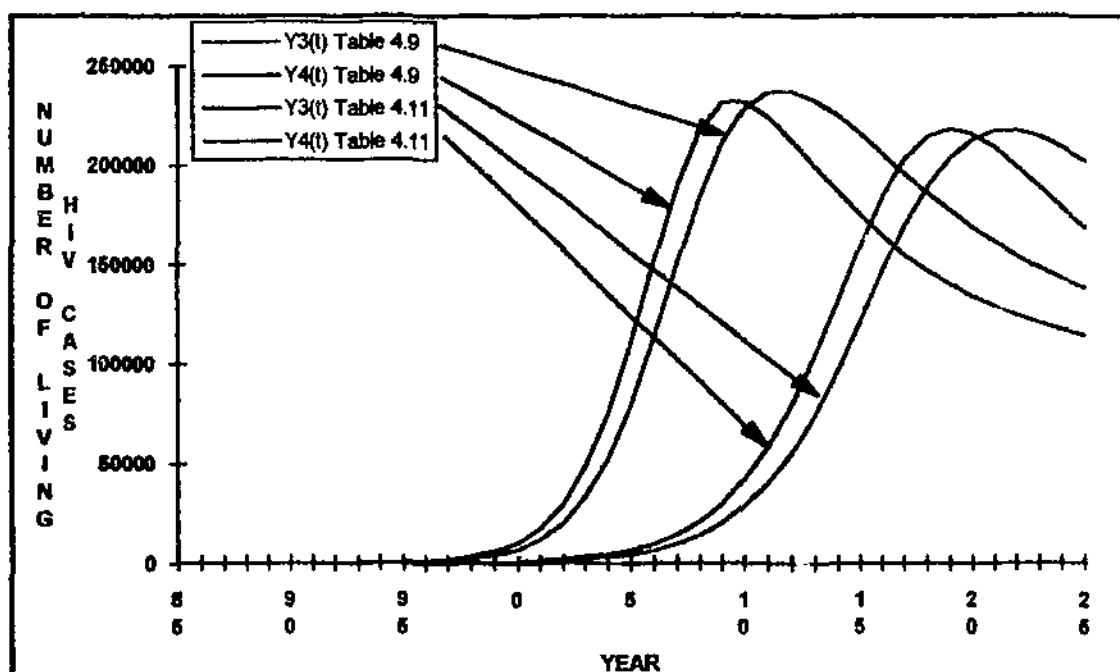


Figure A4.33: Number of living HIV+ male and female non-IVDUs each year from 1985 to 2025 based upon Tables 4.9 and 4.11 ($N, C = 16.5, P = 1.6 / C = 35.5, P = 1.6$).

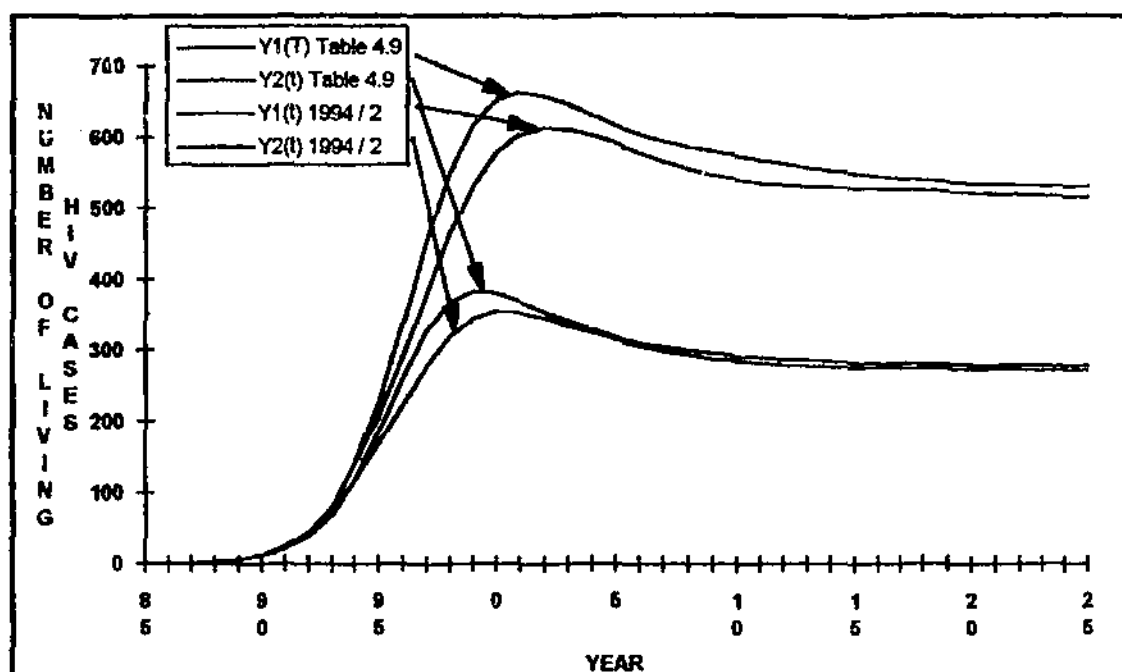


Figure A4.34: Number of living HIV+ male and female IVDUs each year from 1985 to 2025 when condom usage is increased in 1994 (I , $C = 16.5$, $P = 1.6 / C = 35.5$ (94), $P = 1.6$).

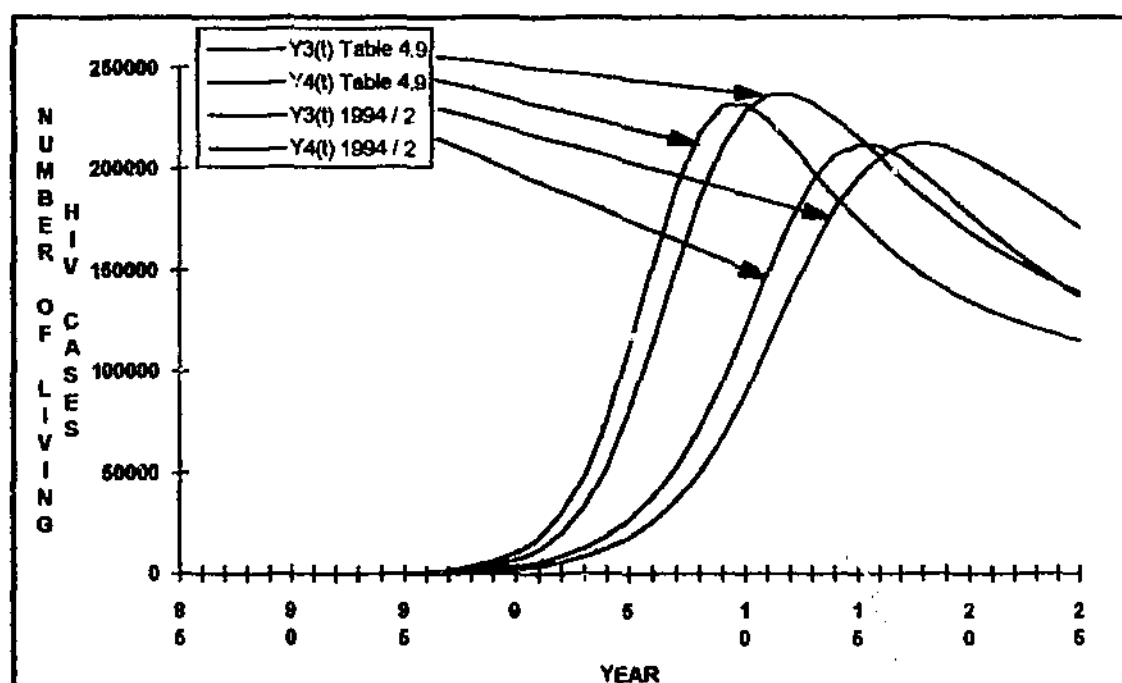


Figure A4.35: Number of living HIV+ male and female non-IVDUs each year from 1985 to 2025 when condom usage is increased in 1994 (N , $C = 16.5$, $P = 1.6 / C = 35.5$ (94), $P = 1.6$).

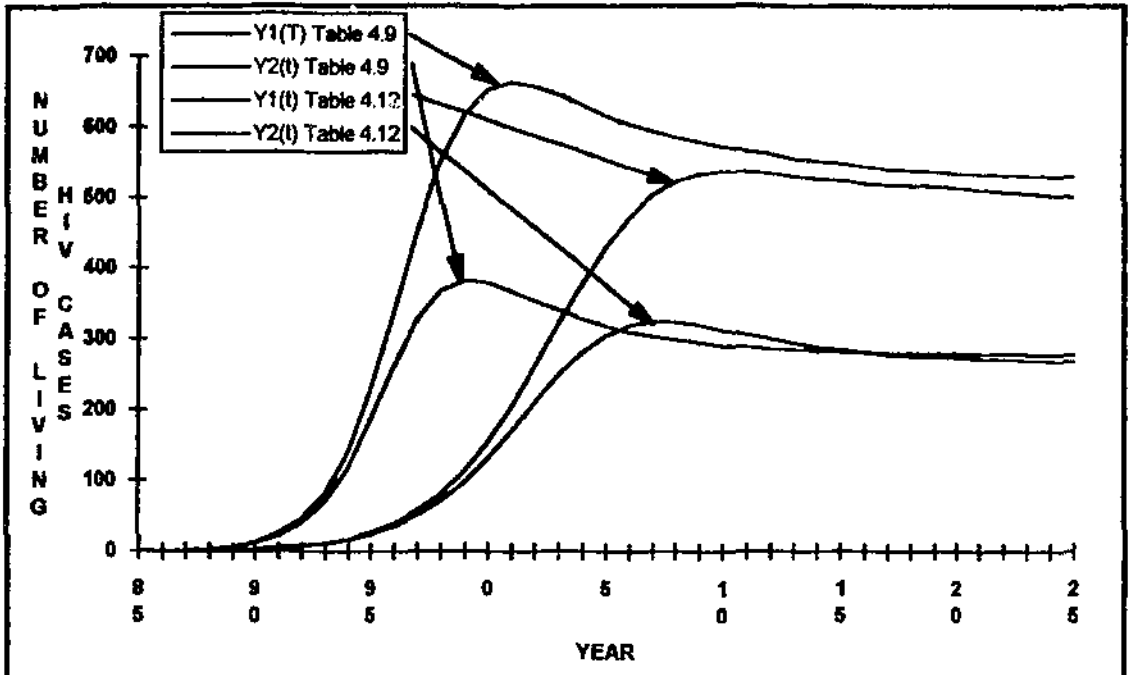


Figure A4.36: Number of living HIV+ male and female IVDUs each year from 1985 to 2025 based upon Tables 4.9 and 4.12 ($I, C = 16.5, P = 1.6 / C = 35.5, P = 1$).

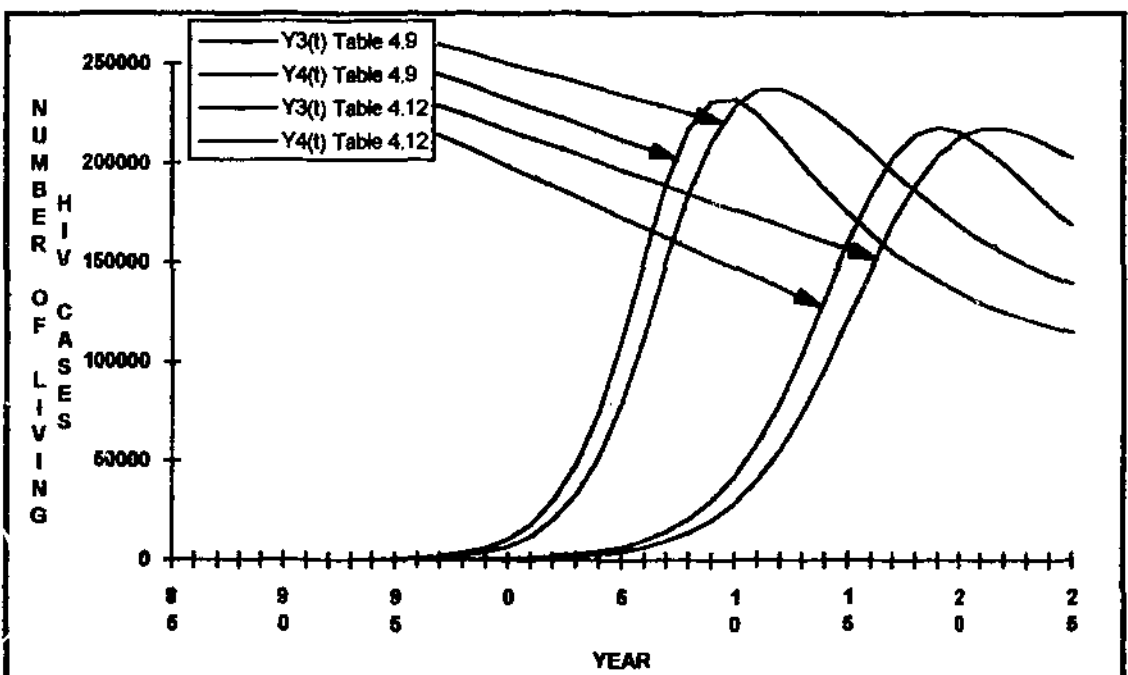
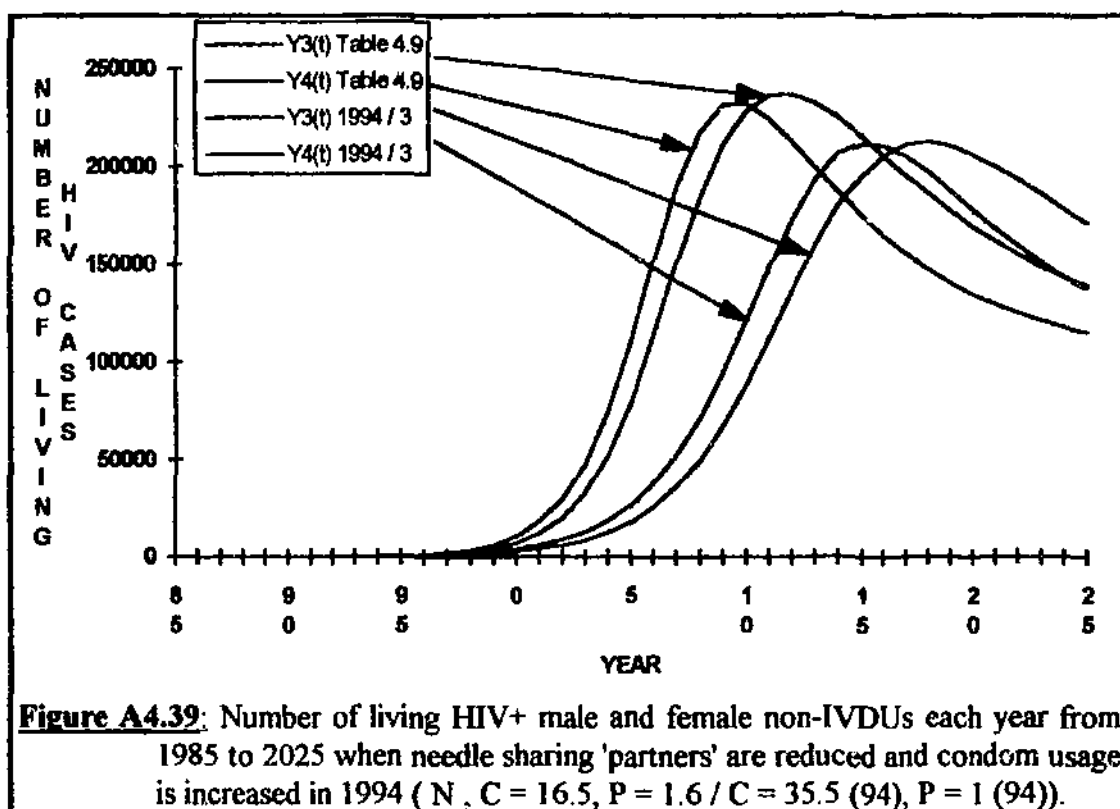
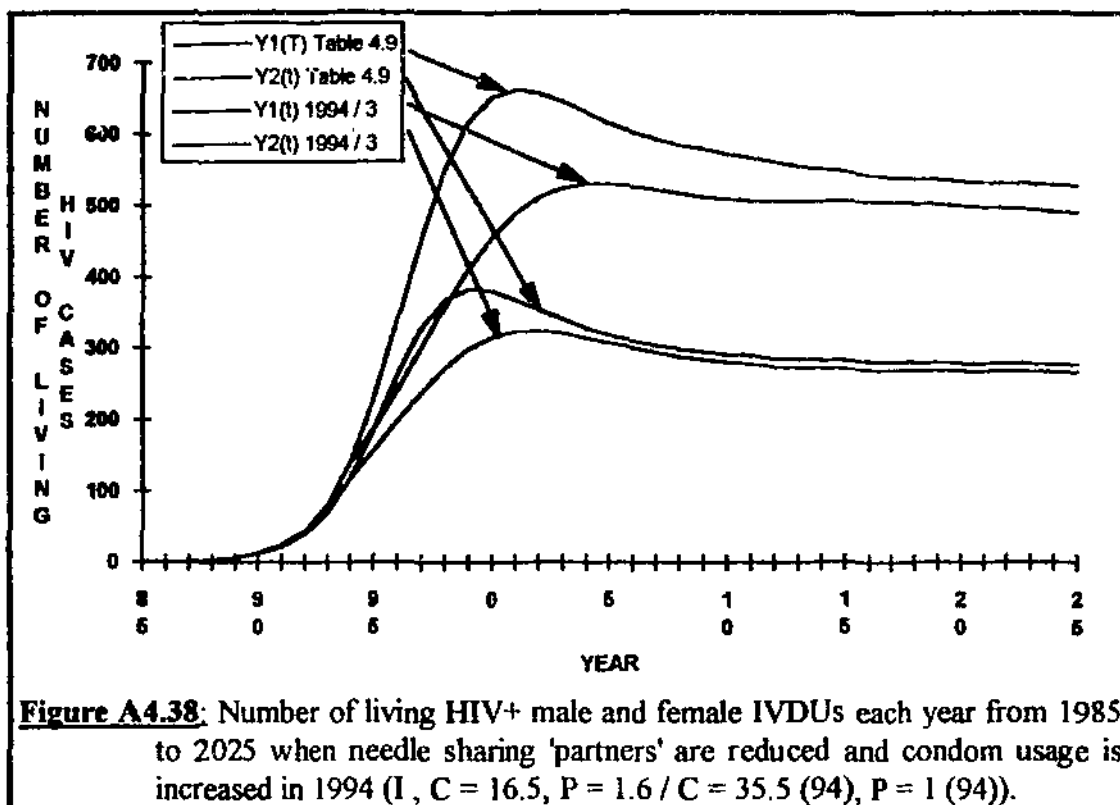


Figure A4.37: Number of living HIV+ male and female non-IVDUs each year from 1985 to 2025 based upon Tables 4.9 and 4.12 ($N, C = 16.5, P = 1.6 / C = 35.5, P = 1$).



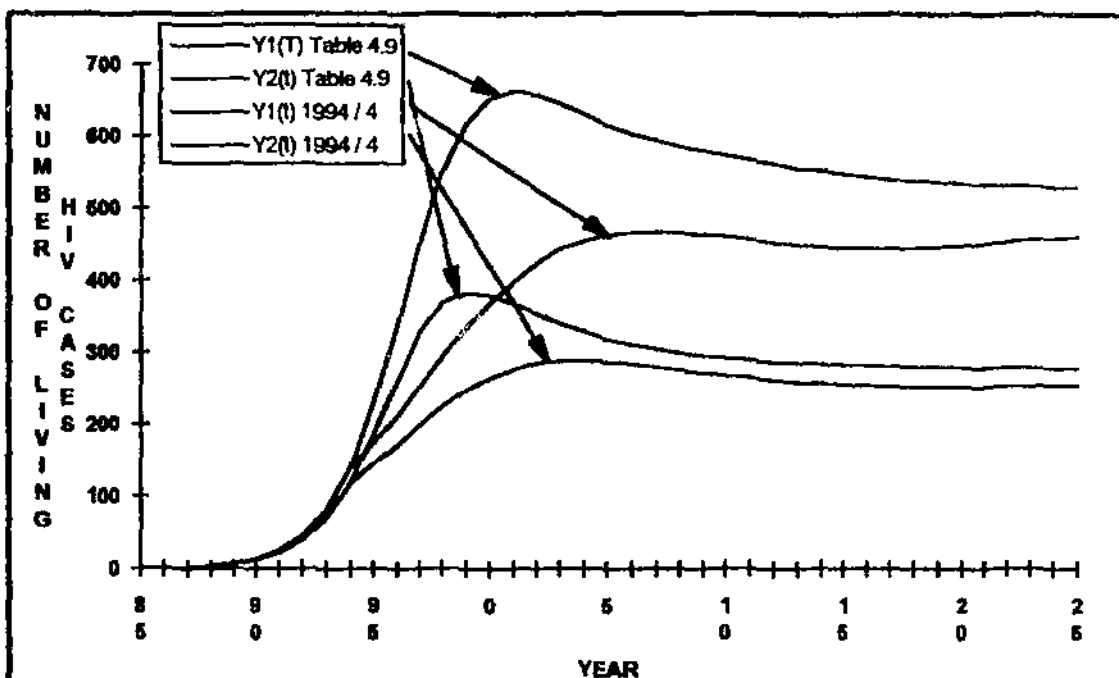


Figure A4.40: Number of living HIV+ male and female IVDUs each year from 1985 to 2025 when needle sharing 'partners' are reduced and condom usage is increased to 50% of in 1994 (I , $C = 16.5$, $P = 1.6$ / $C = 50$ (94), $P = 1$ (94)).

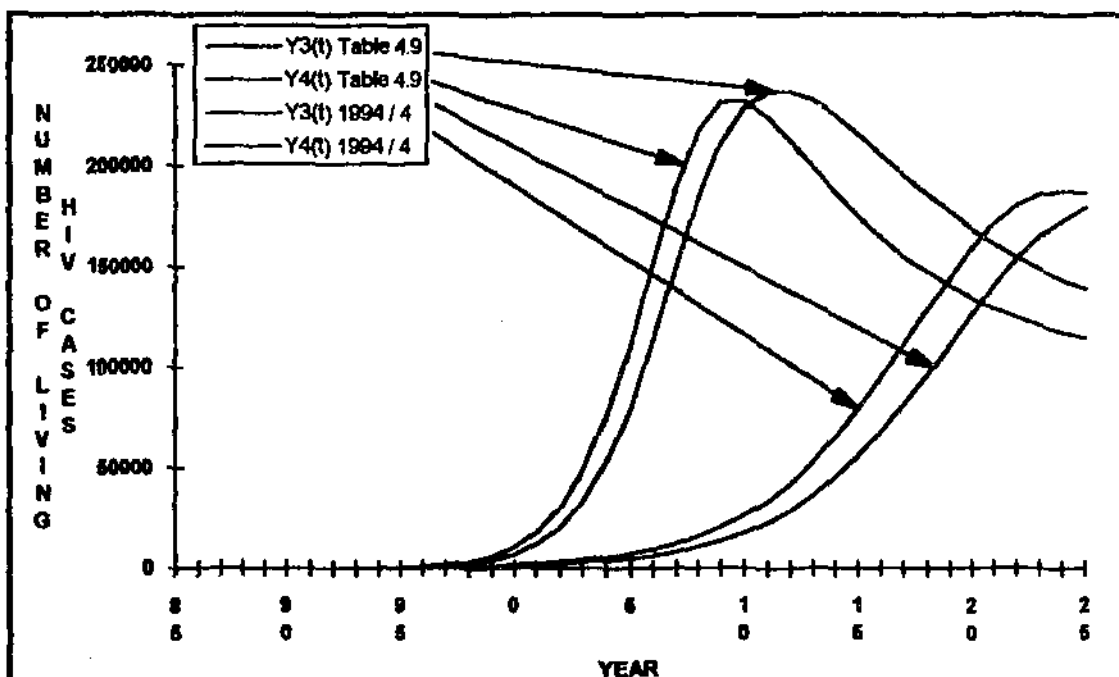


Figure A4.41: Number of living HIV+ male and female non-IVDUs each year from 1985 to 2025 when needle sharing 'partners' are reduced and condom usage is increased to 50% in 1994 (N , $C = 16.5$, $P = 1.6$ / $C = 50$ (94), $P = 1$ (94)).

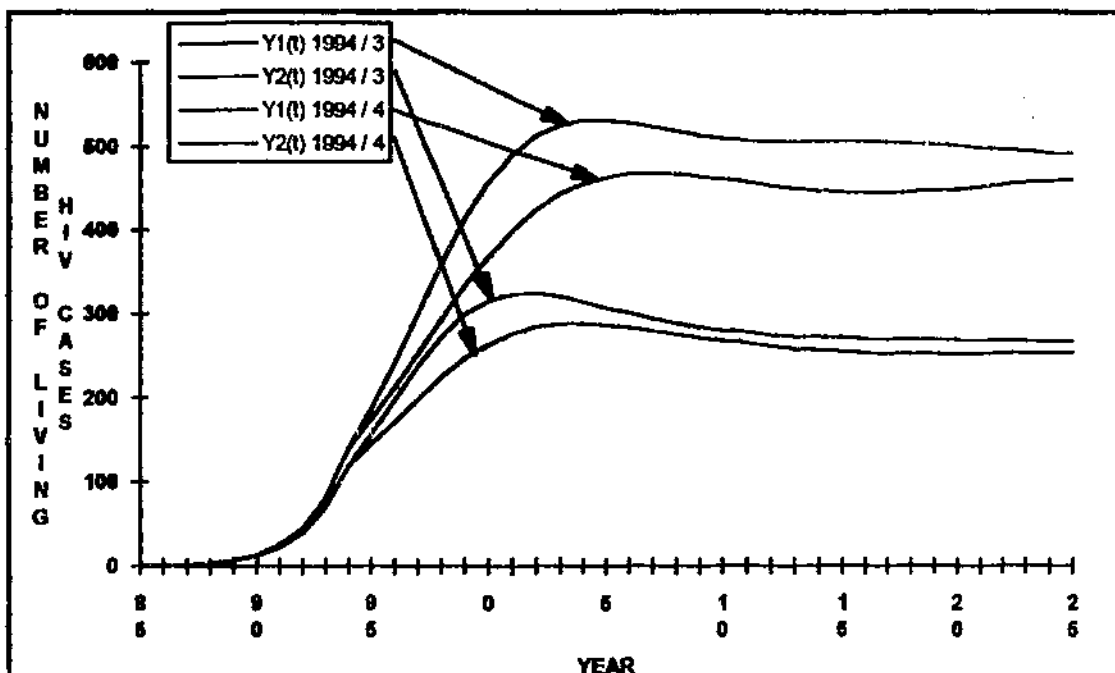


Figure A4.42: Number of living HIV+ male and female IVDUs each year from 1985 to 2025 when needle sharing 'partners' are reduced and condom usage is increased to 35.5% and to 50% in 1994 ($I, C = 35.5 (94), P = 1 (94) / C = 50 (94), P = 1 (94)$).

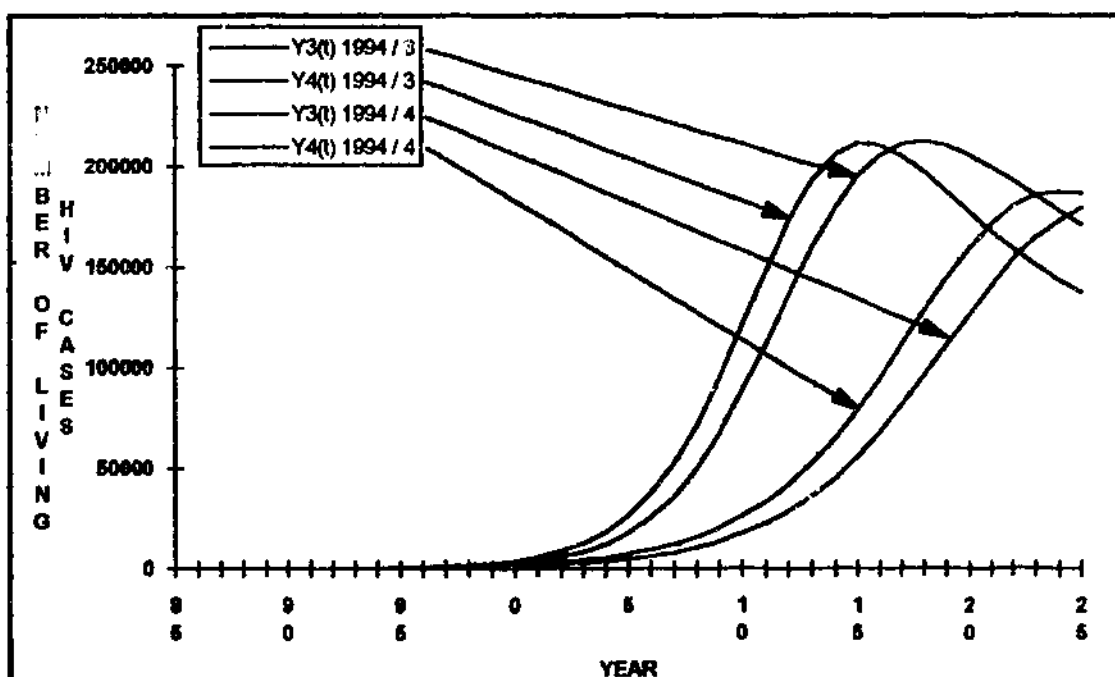


Figure A4.43: Number of living HIV+ male and female non-IVDUs each year from 1985 to 2025 when needle sharing 'partners' are reduced and condom usage is increased to 35.5% and to 50% in 1994 ($N, C = 35.5 (94), P = 1 (94) / C = 50 (94), P = 1 (94)$).

APPENDIX 5:

Epidemic Model for All Sexual Contact

The two models in this appendix follow the same principles as those in chapter 2. The first model considers transmission of the virus through sexual contact only, while the second model also includes transmission through IV drug use. As in the earlier models $X_i(t)$, $Y_i(t)$, $A_i(t)$ are the number of susceptibles, infectives and AIDS cases respectively which are from group i at time t . $P_i(t)$ is the total population of group i at time t . The following model parameters have been used in the two models. The groups used in each model are listed at the start of each section.

β_{mm} : Probability of transmission of the HIV virus through sexual contact from a male infective to a male susceptible.

β_{mf} : Probability of transmission of the HIV virus through sexual contact from a male infective to a female susceptible.

β_{fm} : Probability of transmission of the HIV virus through sexual contact from a female infective to a male susceptible.

β_{ff} : Probability of transmission of the HIV virus through sexual contact from a female infective to a female susceptible.

$\hat{\beta}$: Probability of transmission of the HIV virus through the sharing of needles.

Λ_i : Recruitment rate into group i .

μ_i : Migration rate from group i .

η_i : Average number of needle sharing 'partners' which an individual from group i has in a year ($i = 7$ to 12).

c_{ij} : Average number of sexual partners an individual from group i chooses from group j in a year.

α : Rate of progression of the HIV virus.

δ : Death rate from AIDS.

A5.1 Transmission Through Sexual Contact Only

Group 1 : All male homosexuals.

Group 2 : All female homosexuals.

Group 3 : All male bisexuals.

Group 4 : All female bisexuals.

Group 5 : All male heterosexuals.

Group 6 : All female heterosexuals.

$$\frac{dX_1(t)}{dt} = \Lambda_1 - \beta_{mm}c_{11}X_1(t)\frac{Y_1(t)}{P_1(t)} - \beta_{mm}c_{13}X_1(t)\frac{Y_3(t)}{P_3(t)} - \mu_1X_1(t) \quad (A5.1.1)$$

$$\frac{dX_2(t)}{dt} = \Lambda_2 - \beta_{ff}c_{22}X_2(t)\frac{Y_2(t)}{P_2(t)} - \beta_{ff}c_{24}X_2(t)\frac{Y_4(t)}{P_4(t)} - \mu_2X_2(t) \quad (A5.1.2)$$

$$\begin{aligned} \frac{dX_3(t)}{dt} = & \Lambda_3 - \beta_{mm}c_{31}X_3(t)\frac{Y_1(t)}{P_1(t)} - \beta_{mm}c_{33}X_3(t)\frac{Y_3(t)}{P_3(t)} - \beta_{fm}c_{34}X_3(t)\frac{Y_4(t)}{P_4(t)} \\ & - \beta_{fm}c_{36}X_3(t)\frac{Y_6(t)}{P_6(t)} - \mu_3X_3(t) \end{aligned} \quad (A5.1.3)$$

$$\begin{aligned} \frac{dX_4(t)}{dt} = & \Lambda_4 - \beta_{ff}c_{42}X_4(t)\frac{Y_2(t)}{P_2(t)} - \beta_{mf}c_{43}X_4(t)\frac{Y_3(t)}{P_3(t)} - \beta_{ff}c_{44}X_4(t)\frac{Y_4(t)}{P_4(t)} \\ & - \beta_{mf}c_{45}X_4(t)\frac{Y_5(t)}{P_5(t)} - \mu_4X_4(t) \end{aligned} \quad (A5.1.4)$$

$$\frac{dX_5(t)}{dt} = \Lambda_5 - \beta_{fm}c_{54}X_5(t)\frac{Y_4(t)}{P_4(t)} - \beta_{fm}c_{56}X_5(t)\frac{Y_6(t)}{P_6(t)} - \mu_5X_5(t) \quad (A5.1.5)$$

$$\frac{dX_6(t)}{dt} = \Lambda_6 - \beta_{mf}c_{63}X_6(t)\frac{Y_3(t)}{P_3(t)} - \beta_{mf}c_{65}X_6(t)\frac{Y_5(t)}{P_5(t)} - \mu_6X_6(t) \quad (A5.1.6)$$

$$\frac{dY_1(t)}{dt} = \beta_{mm}c_{11}X_1(t)\frac{Y_1(t)}{P_1(t)} + \beta_{mm}c_{13}X_1(t)\frac{Y_3(t)}{P_3(t)} - (\alpha + \mu_1)Y_1(t) \quad (\text{A5.1.7})$$

$$\frac{dY_2(t)}{dt} = \beta_{ff}c_{22}X_2(t)\frac{Y_2(t)}{P_2(t)} + \beta_{ff}c_{24}X_2(t)\frac{Y_4(t)}{P_4(t)} - (\alpha + \mu_2)Y_2(t) \quad (\text{A5.1.8})$$

$$\begin{aligned} \frac{dY_3(t)}{dt} = & \beta_{mm}c_{31}X_3(t)\frac{Y_1(t)}{P_1(t)} + \beta_{mm}c_{33}X_3(t)\frac{Y_3(t)}{P_3(t)} + \beta_{fm}c_{34}X_3(t)\frac{Y_4(t)}{P_4(t)} \\ & + \beta_{fm}c_{36}X_3(t)\frac{Y_6(t)}{P_6(t)} - (\alpha + \mu_3)Y_3(t) \end{aligned} \quad (\text{A5.1.9})$$

$$\begin{aligned} \frac{dY_4(t)}{dt} = & \beta_{ff}c_{42}X_4(t)\frac{Y_2(t)}{P_2(t)} + \beta_{mf}c_{43}X_4(t)\frac{Y_3(t)}{P_3(t)} + \beta_{ff}c_{44}X_4(t)\frac{Y_4(t)}{P_4(t)} \\ & + \beta_{mf}c_{45}X_4(t)\frac{Y_5(t)}{P_5(t)} - (\alpha + \mu_4)Y_4(t) \end{aligned} \quad (\text{A5.1.10})$$

$$\frac{dY_5(t)}{dt} = \beta_{fm}c_{54}X_5(t)\frac{Y_4(t)}{P_4(t)} + \beta_{fm}c_{56}X_5(t)\frac{Y_6(t)}{P_6(t)} - (\alpha + \mu_5)Y_5(t) \quad (\text{A5.1.11})$$

$$\frac{dY_6(t)}{dt} = \beta_{mf}c_{63}X_6(t)\frac{Y_3(t)}{P_3(t)} + \beta_{mf}c_{65}X_6(t)\frac{Y_5(t)}{P_5(t)} - (\alpha + \mu_6)Y_6(t) \quad (\text{A5.1.12})$$

$$\frac{dA_i(t)}{dt} = \alpha Y_i(t) - (\delta + \mu_i)A_i(t) \quad (\text{A5.1.13})$$

$$P_i(t) = X_i(t) + Y_i(t) + A_i(t) \quad (\text{A5.1.14})$$

for $i = 1$ to 6

A5.2 Transmission Through Sexual Contact and IVDU Use

Group 1 : Male homosexual non-IVDU.

Group 2 : Female homosexual non-IVDU.

Group 3 : Male bisexual non-IVDU.

Group 4 : Female bisexual non-IVDU.

Group 5 : Male heterosexual non-IVDU.

Group 6 : Female heterosexual non-IVDU.

Group 7 : Male homosexual IVDU.

Group 8 : Female homosexual IVDU.

Group 9 : Male bisexual IVDU.

Group 10 : Female bisexual IVDU.

Group 11 : Male heterosexual IVDU.

Group 12 : Female heterosexual IVDU.

$$\begin{aligned} \frac{dX_1(t)}{dt} = & \Lambda_1 - \beta_{mm}c_{1,1}X_1(t)\frac{Y_1(t)}{P_1(t)} - \beta_{mm}c_{1,3}X_1(t)\frac{Y_3(t)}{P_3(t)} - \beta_{mm}c_{1,7}X_1(t)\frac{Y_7(t)}{P_7(t)} \\ & - \beta_{mm}c_{1,9}X_1(t)\frac{Y_9(t)}{P_9(t)} - \mu_1X_1(t) \end{aligned} \quad (A5.2.1)$$

$$\begin{aligned} \frac{dX_2(t)}{dt} = & \Lambda_2 - \beta_{ff}c_{2,2}X_2(t)\frac{Y_2(t)}{P_2(t)} - \beta_{ff}c_{2,4}X_2(t)\frac{Y_4(t)}{P_4(t)} - \beta_{ff}c_{2,6}X_2(t)\frac{Y_6(t)}{P_6(t)} \\ & - \beta_{ff}c_{2,10}X_2(t)\frac{Y_{10}(t)}{P_{10}(t)} - \mu_2X_2(t) \end{aligned} \quad (A5.2.2)$$

$$\begin{aligned}
\frac{dX_3(t)}{dt} = & \Lambda_3 - \beta_{mm}c_{3,1}X_3(t)\frac{Y_1(t)}{P_1(t)} - \beta_{mm}c_{3,3}X_3(t)\frac{Y_3(t)}{P_3(t)} - \beta_{fm}c_{3,4}X_3(t)\frac{Y_4(t)}{P_4(t)} \\
& - \beta_{fm}c_{3,6}X_3(t)\frac{Y_6(t)}{P_6(t)} - \beta_{mm}c_{3,7}X_3(t)\frac{Y_7(t)}{P_7(t)} - \beta_{mm}c_{3,9}X_3(t)\frac{Y_9(t)}{P_9(t)} \\
& - \beta_{fm}c_{3,10}X_3(t)\frac{Y_{10}(t)}{P_{10}(t)} - \beta_{fm}c_{3,12}X_3(t)\frac{Y_{12}(t)}{P_{12}(t)} - \mu_3X_3(t)
\end{aligned} \tag{A5.2.3}$$

$$\begin{aligned}
\frac{dX_4(t)}{dt} = & \Lambda_4 - \beta_{ff}c_{4,2}X_4(t)\frac{Y_2(t)}{P_2(t)} - \beta_{mf}c_{4,3}X_4(t)\frac{Y_3(t)}{P_3(t)} - \beta_{ff}c_{4,4}X_4(t)\frac{Y_4(t)}{P_4(t)} \\
& - \beta_{mf}c_{4,5}X_4(t)\frac{Y_5(t)}{P_5(t)} - \beta_{ff}c_{4,8}X_4(t)\frac{Y_8(t)}{P_8(t)} - \beta_{mf}c_{4,9}X_4(t)\frac{Y_9(t)}{P_9(t)} \\
& - \beta_{ff}c_{4,10}X_4(t)\frac{Y_{10}(t)}{P_{10}(t)} - \beta_{mf}c_{4,11}X_4(t)\frac{Y_{11}(t)}{P_{11}(t)} - \mu_4X_4(t)
\end{aligned} \tag{A5.2.4}$$

$$\begin{aligned}
\frac{dX_5(t)}{dt} = & \Lambda_5 - \beta_{fm}c_{5,4}X_5(t)\frac{Y_4(t)}{P_4(t)} - \beta_{fm}c_{5,6}X_5(t)\frac{Y_6(t)}{P_6(t)} - \beta_{fm}c_{5,10}X_5(t)\frac{Y_{10}(t)}{P_{10}(t)} \\
& - \beta_{fm}c_{5,12}X_5(t)\frac{Y_{12}(t)}{P_{12}(t)} - \mu_5X_5(t)
\end{aligned} \tag{A5.2.5}$$

$$\begin{aligned}
\frac{dX_6(t)}{dt} = & \Lambda_6 - \beta_{mf}c_{6,3}X_6(t)\frac{Y_3(t)}{P_3(t)} - \beta_{mf}c_{6,5}X_6(t)\frac{Y_5(t)}{P_5(t)} - \beta_{mf}c_{6,9}X_6(t)\frac{Y_9(t)}{P_9(t)} \\
& - \beta_{mf}c_{6,11}X_6(t)\frac{Y_{11}(t)}{P_{11}(t)} - \mu_6X_6(t)
\end{aligned} \tag{A5.2.6}$$

$$\begin{aligned}
\frac{dX_7(t)}{dt} = & \Lambda_7 - \beta_{mm}c_{7,1}X_7(t)\frac{Y_1(t)}{P_1(t)} - \beta_{mm}c_{7,3}X_7(t)\frac{Y_3(t)}{P_3(t)} - \beta_{mm}c_{7,7}X_7(t)\frac{Y_7(t)}{P_7(t)} \\
& - \beta_{mm}c_{7,9}X_7(t)\frac{Y_9(t)}{P_9(t)} - \eta_7 \hat{\beta} X_7(t) \left[\frac{\sum_{i=7}^{12} Y_i(t)}{\sum_{i=7}^{12} P_i(t)} \right] - \mu_7X_7(t)
\end{aligned} \tag{A5.2.7}$$

$$\begin{aligned}
\frac{dX_8(t)}{dt} = & \Lambda_8 - \beta_{ff} c_{82} X_8(t) \frac{Y_2(t)}{P_2(t)} - \beta_{ff} c_{84} X_8(t) \frac{Y_4(t)}{P_4(t)} - \beta_{ff} c_{88} X_8(t) \frac{Y_8(t)}{P_8(t)} \\
& - \beta_{ff} c_{8,10} X_8(t) \frac{Y_{10}(t)}{P_{10}(t)} - \eta_8 \hat{\beta} X_8(t) \left[\frac{\sum_{i=7}^{12} Y_i(t)}{\sum_{i=7}^{12} P_i(t)} \right] - \mu_8 X_8(t)
\end{aligned} \tag{A5.2.8}$$

$$\begin{aligned}
\frac{dX_9(t)}{dt} = & \Lambda_9 - \beta_{mm} c_{9,1} X_9(t) \frac{Y_1(t)}{P_1(t)} - \beta_{mm} c_{9,3} X_9(t) \frac{Y_3(t)}{P_3(t)} - \beta_{fm} c_{9,4} X_9(t) \frac{Y_4(t)}{P_4(t)} \\
& - \beta_{fm} c_{9,6} X_9(t) \frac{Y_6(t)}{P_6(t)} - \beta_{mm} c_{9,7} X_9(t) \frac{Y_7(t)}{P_7(t)} - \beta_{mm} c_{9,9} X_9(t) \frac{Y_9(t)}{P_9(t)} \\
& - \beta_{fm} c_{9,10} X_9(t) \frac{Y_{10}(t)}{P_{10}(t)} - \beta_{fm} c_{9,12} X_9(t) \frac{Y_{12}(t)}{P_{12}(t)} \\
& - \eta_9 \hat{\beta} X_9(t) \left[\frac{\sum_{i=7}^{12} Y_i(t)}{\sum_{i=7}^{12} P_i(t)} \right] - \mu_9 X_9(t)
\end{aligned} \tag{A5.2.9}$$

$$\begin{aligned}
\frac{dX_{10}(t)}{dt} = & \Lambda_{10} - \beta_{ff} c_{10,2} X_{10}(t) \frac{Y_2(t)}{P_2(t)} - \beta_{mf} c_{10,3} X_{10}(t) \frac{Y_3(t)}{P_3(t)} - \beta_{ff} c_{10,4} X_{10}(t) \frac{Y_4(t)}{P_4(t)} \\
& - \beta_{mf} c_{10,5} X_{10}(t) \frac{Y_5(t)}{P_5(t)} - \beta_{ff} c_{10,8} X_{10}(t) \frac{Y_8(t)}{P_8(t)} - \beta_{mf} c_{10,9} X_{10}(t) \frac{Y_9(t)}{P_9(t)} \\
& - \beta_{ff} c_{10,10} X_{10}(t) \frac{Y_{10}(t)}{P_{10}(t)} - \beta_{mf} c_{10,11} X_{10}(t) \frac{Y_{11}(t)}{P_{11}(t)} \\
& - \eta_{10} \hat{\beta} X_{10}(t) \left[\frac{\sum_{i=7}^{12} Y_i(t)}{\sum_{i=7}^{12} P_i(t)} \right] - \mu_{10} X_{10}(t)
\end{aligned} \tag{A5.2.10}$$

$$\begin{aligned}
\frac{dX_{11}(t)}{dt} = & \Lambda_{11} - \beta_{fm}c_{11,4}X_{11}(t)\frac{Y_4(t)}{P_4(t)} - \beta_{fm}c_{11,6}X_{11}(t)\frac{Y_6(t)}{P_6(t)} - \beta_{fm}c_{11,10}X_{11}(t)\frac{Y_{10}(t)}{P_{10}(t)} \\
& - \beta_{fm}c_{11,11}X_{12}(t)\frac{Y_{12}(t)}{P_{12}(t)} - \eta_{11}\hat{\beta}X_{11}(t)\left[\frac{\sum_{i=7}^{12}Y_i(t)}{\sum_{i=7}^{12}P_i(t)}\right] - \mu_{11}X_{11}(t)
\end{aligned} \tag{A5.2.11}$$

$$\begin{aligned}
\frac{dX_{12}(t)}{dt} = & \Lambda_{12} - \beta_{mf}c_{12,3}X_{12}(t)\frac{Y_3(t)}{P_3(t)} - \beta_{mf}c_{12,5}X_{12}(t)\frac{Y_5(t)}{P_5(t)} - \beta_{mf}c_{12,9}X_{12}(t)\frac{Y_9(t)}{P_9(t)} \\
& - \beta_{mf}c_{12,11}X_{12}(t)\frac{Y_{11}(t)}{P_{11}(t)} - \eta_{12}\hat{\beta}X_{12}(t)\left[\frac{\sum_{i=7}^{12}Y_i(t)}{\sum_{i=7}^{12}P_i(t)}\right] - \mu_{12}X_{12}(t)
\end{aligned} \tag{A5.2.12}$$

$$\begin{aligned}
\frac{dY_1(t)}{dt} = & \beta_{mm}c_{1,1}X_1(t)\frac{Y_1(t)}{P_1(t)} + \beta_{mm}c_{1,3}X_1(t)\frac{Y_3(t)}{P_3(t)} + \beta_{mm}c_{1,7}X_1(t)\frac{Y_7(t)}{P_7(t)} \\
& + \beta_{mm}c_{1,9}X_1(t)\frac{Y_9(t)}{P_9(t)} - (\alpha + \mu_1)Y_1(t)
\end{aligned} \tag{A5.2.13}$$

$$\begin{aligned}
\frac{dY_2(t)}{dt} = & \beta_{ff}c_{2,2}X_2(t)\frac{Y_2(t)}{P_2(t)} + \beta_{ff}c_{2,4}X_2(t)\frac{Y_4(t)}{P_4(t)} + \beta_{ff}c_{2,8}X_2(t)\frac{Y_8(t)}{P_8(t)} \\
& + \beta_{ff}c_{2,10}X_2(t)\frac{Y_{10}(t)}{P_{10}(t)} - (\alpha + \mu_2)Y_2(t)
\end{aligned} \tag{A5.2.14}$$

$$\begin{aligned}
\frac{dY_3(t)}{dt} = & \beta_{mm}c_{3,1}X_3(t)\frac{Y_1(t)}{P_1(t)} + \beta_{mm}c_{3,3}X_3(t)\frac{Y_3(t)}{P_3(t)} + \beta_{fm}c_{3,4}X_3(t)\frac{Y_4(t)}{P_4(t)} \\
& + \beta_{fm}c_{3,6}X_3(t)\frac{Y_6(t)}{P_6(t)} + \beta_{mm}c_{3,7}X_3(t)\frac{Y_7(t)}{P_7(t)} + \beta_{mm}c_{3,9}X_3(t)\frac{Y_9(t)}{P_9(t)} \\
& + \beta_{fm}c_{3,10}X_3(t)\frac{Y_{10}(t)}{P_{10}(t)} + \beta_{fm}c_{3,12}X_3(t)\frac{Y_{12}(t)}{P_{12}(t)} - (\alpha + \mu_3)Y_3(t)
\end{aligned} \tag{A5.2.15}$$

$$\begin{aligned}
\frac{dY_4(t)}{dt} = & \beta_{ff}c_{4,2}X_4(t)\frac{Y_2(t)}{P_2(t)} + \beta_{mf}c_{4,3}X_4(t)\frac{Y_3(t)}{P_3(t)} + \beta_{ff}c_{4,4}X_4(t)\frac{Y_4(t)}{P_4(t)} \\
& + \beta_{mf}c_{4,5}X_4(t)\frac{Y_5(t)}{P_5(t)} + \beta_{ff}c_{4,8}X_4(t)\frac{Y_8(t)}{P_8(t)} + \beta_{mf}c_{4,9}X_4(t)\frac{Y_9(t)}{P_9(t)} \\
& + \beta_{ff}c_{4,10}X_4(t)\frac{Y_{10}(t)}{P_{10}(t)} + \beta_{mf}c_{4,11}X_4(t)\frac{Y_{11}(t)}{P_{11}(t)} - (\alpha + \mu_4)Y_4(t)
\end{aligned} \tag{A5.2.16}$$

$$\begin{aligned}
\frac{dY_5(t)}{dt} = & \beta_{fm}c_{5,4}X_5(t)\frac{Y_4(t)}{P_4(t)} + \beta_{fm}c_{5,6}X_5(t)\frac{Y_6(t)}{P_6(t)} + \beta_{fm}c_{5,10}X_5(t)\frac{Y_{10}(t)}{P_{10}(t)} \\
& + \beta_{fm}c_{5,12}X_5(t)\frac{Y_{12}(t)}{P_{12}(t)} - (\alpha + \mu_5)Y_5(t)
\end{aligned} \tag{A5.2.17}$$

$$\begin{aligned}
\frac{dY_6(t)}{dt} = & \beta_{mf}c_{6,3}X_6(t)\frac{Y_3(t)}{P_3(t)} + \beta_{mf}c_{6,5}X_6(t)\frac{Y_5(t)}{P_5(t)} + \beta_{mf}c_{6,9}X_6(t)\frac{Y_9(t)}{P_9(t)} \\
& + \beta_{mf}c_{6,11}X_6(t)\frac{Y_{11}(t)}{P_{11}(t)} - (\alpha + \mu_6)Y_6(t)
\end{aligned} \tag{A5.2.18}$$

$$\begin{aligned}
\frac{dY_7(t)}{dt} = & \beta_{mm}c_{7,1}X_7(t)\frac{Y_1(t)}{P_1(t)} + \beta_{mm}c_{7,3}X_7(t)\frac{Y_3(t)}{P_3(t)} + \beta_{mm}c_{7,7}X_7(t)\frac{Y_7(t)}{P_7(t)} \\
& + \beta_{mm}c_{7,9}X_7(t)\frac{Y_9(t)}{P_9(t)} + \eta_7 \hat{\beta} X_7(t) \left[\frac{\sum_{i=7}^{12} Y_i(t)}{\sum_{i=7}^{12} P_i(t)} \right] - (\alpha + \mu_7)Y_7(t)
\end{aligned} \tag{A5.2.19}$$

$$\begin{aligned}
\frac{dY_8(t)}{dt} = & \beta_{ff}c_{8,2}X_8(t)\frac{Y_2(t)}{P_2(t)} + \beta_{ff}c_{8,4}X_8(t)\frac{Y_4(t)}{P_4(t)} + \beta_{ff}c_{8,8}X_8(t)\frac{Y_8(t)}{P_8(t)} \\
& + \beta_{ff}c_{8,10}X_8(t)\frac{Y_{10}(t)}{P_{10}(t)} + \eta_8 \hat{\beta} X_8(t) \left[\frac{\sum_{i=7}^{12} Y_i(t)}{\sum_{i=7}^{12} P_i(t)} \right] - (\alpha + \mu_8)Y_8(t)
\end{aligned} \tag{A5.2.20}$$

$$\begin{aligned}
\frac{dY_9(t)}{dt} = & \beta_{mm}c_{9,1}X_9(t)\frac{Y_1(t)}{P_1(t)} + \beta_{mm}c_{9,3}X_9(t)\frac{Y_3(t)}{P_3(t)} + \beta_{fm}c_{9,4}X_9(t)\frac{Y_4(t)}{P_4(t)} \\
& + \beta_{fm}c_{9,6}X_9(t)\frac{Y_6(t)}{P_6(t)} + \beta_{mm}c_{9,7}X_9(t)\frac{Y_7(t)}{P_7(t)} + \beta_{mm}c_{9,9}X_9(t)\frac{Y_9(t)}{P_9(t)} \\
& + \beta_{fm}c_{9,10}X_9(t)\frac{Y_{10}(t)}{P_{10}(t)} + \beta_{fm}c_{9,12}X_9(t)\frac{Y_{12}(t)}{P_{12}(t)} \\
& + \eta_9\hat{\beta}X_9(t)\left[\frac{\sum_{i=7}^{12}Y_i(t)}{\sum_{i=7}^{12}P_i(t)}\right] - (\alpha + \mu_9)Y_9(t)
\end{aligned}
\tag{A5.2.21}$$

$$\begin{aligned}
\frac{dY_{10}(t)}{dt} = & \beta_{ff}c_{10,2}X_{10}(t)\frac{Y_2(t)}{P_2(t)} + \beta_{mf}c_{10,3}X_{10}(t)\frac{Y_3(t)}{P_3(t)} + \beta_{ff}c_{10,4}X_{10}(t)\frac{Y_4(t)}{P_4(t)} \\
& + \beta_{mf}c_{10,5}X_{10}(t)\frac{Y_5(t)}{P_5(t)} + \beta_{ff}c_{10,8}X_{10}(t)\frac{Y_8(t)}{P_8(t)} + \beta_{mf}c_{10,9}X_{10}(t)\frac{Y_9(t)}{P_9(t)} \\
& + \beta_{ff}c_{10,10}X_{10}(t)\frac{Y_{10}(t)}{P_{10}(t)} + \beta_{mf}c_{10,11}X_{10}(t)\frac{Y_{11}(t)}{P_{11}(t)} \\
& + \eta_{10}\hat{\beta}X_{10}(t)\left[\frac{\sum_{i=7}^{12}Y_i(t)}{\sum_{i=7}^{12}P_i(t)}\right] - (\alpha + \mu_{10})Y_{10}(t)
\end{aligned}
\tag{A5.2.22}$$

$$\begin{aligned}
\frac{dY_{11}(t)}{dt} = & \beta_{fm}c_{11,4}X_{11}(t)\frac{Y_4(t)}{P_4(t)} + \beta_{fm}c_{11,6}X_{11}(t)\frac{Y_6(t)}{P_6(t)} + \beta_{fm}c_{11,10}X_{11}(t)\frac{Y_{10}(t)}{P_{10}(t)} \\
& + \beta_{fm}c_{11,11}X_{11}(t)\frac{Y_{11}(t)}{P_{11}(t)} + \eta_{11}\hat{\beta}X_{11}(t)\left[\frac{\sum_{i=7}^{12}Y_i(t)}{\sum_{i=7}^{12}P_i(t)}\right] \\
& - (\alpha + \mu_{11})Y_{11}(t)
\end{aligned}
\tag{A5.2.23}$$

$$\begin{aligned}
\frac{dY_{12}(t)}{dt} = & \beta_{mf} c_{12,3} X_{12}(t) \frac{Y_3(t)}{P_3(t)} + \beta_{mf} c_{12,5} X_{12}(t) \frac{Y_5(t)}{P_5(t)} + \beta_{mf} c_{12,9} X_{12}(t) \frac{Y_9(t)}{P_9(t)} \\
& + \beta_{mf} c_{12,11} X_{12}(t) \frac{Y_{11}(t)}{P_{11}(t)} + \eta_{12} \hat{\beta} X_{12}(t) \left[\frac{\sum_{i=7}^{12} Y_i(t)}{\sum_{i=7}^{12} P_i(t)} \right] \\
& - (\alpha + \mu_{12}) Y_{12}(t)
\end{aligned} \tag{A5.2.24}$$

$$\frac{dA_i(t)}{dt} = \alpha Y_i(t) - (\delta + \mu_i) A_i(t) \tag{A5.2.25}$$

$$\begin{aligned}
P_i(t) &= X_i(t) + Y_i(t) + A_i(t) \\
&\text{for } i = 1 \text{ to } 12
\end{aligned} \tag{A5.2.26}$$