Understanding heterogeneity in HIV prevalence in rural southwest India and its relationship to female sex work and migration: a mathematical modelling approach

By

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A Thesis Submitted to the Faculty of Graduate Studies in Partial Fulfillment of the Requirements for the Degree of
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A Thesis/Practicum submitted to the Faculty of Graduate Studies of The University of Manitoba in partial fulfillment of the requirement of the degree

MASTER OF SCIENCE

Abstract

Introduction: HIV prevalence in Bagalkot district, a primarily rural area in the southern Indian state of Karnataka, varies considerably by *taluka* (sub-district administrative area). Several key demographic and sexual behavioural risk factors characteristic of the population of female sex workers (FSWs) and male clients, including seasonal migration, may be important determinants of the HIV epidemic in rural India.

Methods: This study used deterministic compartmental mathematical models that incorporated the transmission dynamics of heterosexually transmitted HIV infection to explore the impact of key risk factors in high-risk and general populations and better understand heterogeneity in HIV prevalence among the overall populations across the three *talukas* in Bagalkot district. These included sexual behaviour and demographic factors and factors relating to migration. Three migration scenarios were explored, depending on the population migrating (all males, only clients, or FSWs). Univariate and multivariate sensitivity analysis was conducted to determine the impact of important parameters on HIV prevalence at different stages of the epidemic (2004, peak and equilibrium).

Results: The fraction of infections caused by high-risk groups was much higher than the fraction caused by low-risk groups, even if we assumed that mean FSW-client and client-FSW contact rates were half what is observed (~75%). The mean FSW-client contact rates, FSW sizes and client sizes observed in each *taluka* were potentially all important parameters in explaining why observed HIV prevalence in 2004 was different in the three *talukas*, when the total high-risk partnerships were allowed to vary when we varied these parameter values. If the total number of high-risk partnerships remained fixed, increasing FSW (or client) size actually decreased prevalence as this caused the mean client-FSW contact rate (or mean FSW-client contact rate) to also decline. This suggested that overall prevalence was more sensitive to the mean frequency of high-risk sexual contacts than high-risk population size; this was validated in a sensitivity analysis. Differences in observed HIV prevalence between the three *talukas* could only be explained realistically by migration when a fraction of low-risk males migrated and became clients in the place of migration. Migration of only FSWs or clients had a much more modest impact even with realistic increases in migration-associated risk factors.

Conclusions: The HIV epidemic in Bagalkot district continues to be driven by commercial sex; there is low transmission of HIV in low-risk populations, although low-risk groups are increasingly important later in the epidemic. Since the importance of the mean high-risk contact rates, FSW and client sizes depended on each groups' partners' change in sexual behaviour when parameters were varied, this research highlights the importance of being able to estimate how high-risk behaviour in one high-risk group influences high-risk behaviour in other groups, and if certain risk/sex groups have more influence on high-risk behaviour than others in different populations in rural India. Understanding how local sexual networks are impacted in areas where migration is common is crucial for understanding the impact of migration on HIV transmission, and for designing HIV preventive interventions, both locally and in the migration destination.

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LIST OF ABBREVIATIONS

AIDS: acquired immune deficiency disease
B-C: Base-case
B-CI: Base-case scenario I
B-CII: Base-case scenario II
B-CM: Base-case migration scenario
BDP: Bagalkot Demonstration Project
BSWDP: Bagalkot Sex Workers Demonstration Project
CBHPS: Community-based HIV Prevalence Survey
FHM: female high-risk who migrate (used in flowcharts)
FH: female high-risk (used in flowcharts)
FL: female low-risk (used in flowcharts)
FSW or SW: female sex work (when used as "duration of female sex work")
FSW: female sex worker
HIV: human immunodeficiency virus
ICHAP: India-Canada Collaborative HIV/AIDS Project
KHPT: Karnataka Health Promotion Trust
KSAPS: Karnataka State AIDS Prevention Society
LRF: low-risk female
LRM: low-risk male
MCL: male client
MCRB: male client risk behaviour
MH: male high-risk (used in flowcharts)
MHM: high-risk males who migrate (used in flowcharts)
MLM: low-risk males who migrate (used in flowcharts)
MM: married men
ML: male low-risk (used in flowcharts)
MW: married women
NACO: National AIDS Control Organization
PBS: polling booth survey
POM: place of migration
PRC: population research centre
SAL: sexually active lifetime
SFSWS: Statewide FSW survey
SNA: situation and needs assessment
STD: sexually transmitted disease
STI: sexually transmitted infection
TA: Taluka A
TB: Taluka
TC: Taluka C
UMM: unmarried men
UMW: unmarried women
UNAIDS: United Nations Programme on HIV/AIDS

SUMMARY OF RESEARCH

Introduction: HIV prevalence in Bagalkot District, a primarily rural area in the southern Indian state of Karnataka, varies considerably by Taluka (sub-district administrative area). Several key sexual and behavioural risk factors characteristic of the population of female sex workers (FSWs) and male clients, including seasonal migration, may be important determinants of the HIV epidemic in rural India.

Methods: Transmission dynamics models were developed to explore the impact of key risk factors in high-risk and general populations and better understand heterogeneity in HIV prevalence among the overall populations across the three Talukas. These factors included sexual behaviour and demographic factors for which observed data was different in each of the three Talukas (overall population size, FSW size and client size, mean high-risk contact rate) and additional factors with limited data, including those relating to migration. A univariate sensitivity analysis was conducted on each of these parameters to determine their impact on HIV prevalence at different stages of infection (2004, peak and equilibrium). Additional outcomes included HIV incidence, and the fraction of new infections caused and acquired by high-risk groups. Plausible ranges for behavioural and demographic model parameters for each Taluka were defined from FSW and general population surveys conducted in Bagalkot District, and the available literature.

Results: The fraction of infections caused by high-risk groups was much higher than the fraction caused by low-risk groups, even with half the mean FSW-client and client-FSW contact rates, when it is the lowest (~75%). The mean FSW-client contact rates, FSW sizes and client sizes observed in each Taluka were potentially all important parameters in explaining why observed HIV prevalence in 2004 was different in the three Talukas, when the total high-risk partnerships

were allowed to vary when these parameters vary. It is intuitive that HIV prevalence would increase if total high-risk partnerships increase, since the larger FSW size would be accompanied by an increased FSW-client contact rate and larger client size accompanied by an increased client-FSW contact rate. This analysis, however, showed that the relative differences observed in these parameters, between the three Talukas, were large enough to explain a substantial fraction of the differences in observed 2004 HIV prevalence between the three Talukas, providing that high-risk partnerships also varied. If the total number of high-risk partnerships remained fixed, increasing FSW (or client) size decreased prevalence in high-risk and overall populations, as this caused the mean client-FSW contact rate (mean FSW-client contact rate) to also decline. This suggests that overall prevalence was more sensitive to the mean frequency of high-risk sexual contacts than high-risk population size. This assumption was validated by varying only the mean high-risk contact rates (with fixed high-risk population sizes) and separately, the high-risk population sizes (with fixed mean high-risk contact rate); increasing only the mean contact rates had a much larger effect on increasing 2004 model HIV prevalence than only increasing high-risk population size (greater than double). Seasonal migration (for four months duration) of only clients (men who are clients in the place of origin and the place of migration) in Taluka A required unrealistically high levels of migration-associated risk behaviour (4-fold increase in mean FSW-client contact rate and 70% HIV prevalence in FSWs in 2004 in the place of migration) to bring 2004 model prevalence to within observed prevalence in Taluka B, the Taluka with the next-highest observed HIV prevalence. Increased migrant HIV prevalence only increased overall model HIV prevalence marginally, since local HIV prevalence decreased due to the model assumption that the local male demand for sex work (mean FSW-client contact rate) remained the same regardless of the change in the size of the local high-risk populations. This

means that when clients migrated, there were fewer local clients, which caused the local mean client-FSW contact rate to decline while migrants were away, and lower local model HIV prevalence. In the scenario when males who were not clients in the place of origin became clients in the POM (low-risk clients), much more realistic values for increases in migration-associated risk behaviours increased 2004 model HIV prevalence in Taluka A to within 2004 overall observed values in other Talukas. This was true for both Taluka B (Taluka A required 30% migrant size, 2-fold increase in the mean FSW-client contact rate and 35% prevalence in 2004 in FSWs in the place of migration), with 2.9% HIV prevalence [2.2 – 3.6]%; and Taluka C (Taluka A required 30% migrants, 2-fold increase in the mean FSW-client contact rate and 70% prevalence in 2004 in FSWs in the place of migration), with 4.9% HIV prevalence [3.5 – 6.3]%. Seasonal migration of FSWs caused prevalence to increase even with no increase in migration-associated risk in the place of migration, since fewer local FSWs and the same local FSW-client contact rate caused the local client-FSW contact rate to increase while migrant FSWs were away. The total impact on HIV prevalence of only FSWs migrating was limited by the small FSW population size and because model HIV prevalence in FSWs was initially high. With modest increases in migration levels for FSWs (30% migrants and a two-fold increase in the FSW-client contact rate, four months away and a moderate HIV epidemic in clients in the place of migration), 2004 model HIV prevalence in Taluka A (with lowest overall observed HIV prevalence in 2004) increased to within observed values in Taluka B (2.2%). However, even with unrealistically high migration levels (10-fold increase in the mean client-FSW contact rate in the place of migration), overall model prevalence did not increase above 2.5%. Even with high migration-associated risk behaviour (when migrants were causing a fraction of new yearly infections larger than their population sizes), in order to reverse and eliminate the epidemic in

rural India, it was necessary to introduce high levels of condom use into both local as well as migrant groups, and not only local or migrant groups separately.

Conclusions: The results from this study suggest that the HIV epidemic in Bagalkot District has been (and continues to be) driven by commercial sex, as there was low transmission of HIV in low-risk populations, although low-risk groups were increasingly important later in the epidemic. Since the importance of the mean high-risk contact rates, FSW and client sizes depended on what happened to other populations when these parameters were varied, this research highlights the importance of being able to estimate how high-risk behaviour in one high-risk group influences high-risk behaviour in other groups, and if certain risk/sex groups have more influence on high-risk behaviour than others in different populations in rural India. Of the migration scenarios investigated in this study, differences in observed HIV prevalence between the three Talukas could only be explained realistically when a fraction of low-risk males migrated and became clients in the place of migration. Migration of only FSWs or only clients had a much more modest impact with realistic increases in migration-associated risk factors. Other migration scenarios should be developed and explored in further analysis with increased data on what happens locally when migrants are in the place of migration.

CHAPTER ONE: INTRODUCTION

1.1. The global burden of HIV

HIV/AIDS is one of the most serious and far-reaching epidemics in history. According to the United Nations Programme on HIV/AIDS [UNAIDS] (UNAIDS, 2005), 25 million people had died from acquired immunodeficiency disease syndrome (AIDS) by the end of 2005, 25 years since the identification of the disease. At this time, the number of people living with human immunodeficiency virus (HIV), the pathogen that causes AIDS, had reached 40.3 million [36.7–45.3 million] (UNAIDS, 2005). This number was similar at the end of 2006, when there were about 39.5 million (34.1-47.1 million) people living with HIV (UNAIDS, 2006a). There were fewer people newly infected and lower AIDS related mortality in 2006 compared to 2005 (UNAIDS, 2006a).

The global HIV epidemic is reflective of the many inequalities inherent in the world's societies: countries show much variation in levels of HIV infection, with most of the infections occurring in less developed and poorer countries (UNAIDS, 2005; UNAIDS, 2006a). During the last 25 years of the HIV epidemic, the overall adult HIV prevalence in sub-Saharan Africa is estimated to have reached 8.8%, whereas more than 70% of the world's countries report less than 1% infection among adults (Mills, Saidel, Magnani & Brown, 2004). In 2006, sub-Saharan Africa was home to almost two-thirds (63%) of the total people living with HIV in the world and to almost three-quarters of all AIDS deaths (UNAIDS, 2006a). In Latin America, the Caribbean and North America, the number of new infections remained stable in 2006 compared with 2004; however, there were 70% more new infections in eastern Europe and central Asia in 2006 compared with 2004, 15% more in south and southeast Asia and 12% more in the middle east and north Africa over the same period (UNAIDS, 2006a).

1.2. The burden of HIV in India

In comparison, about 0.9% of adults in India are infected with HIV (UNAIDS, 2006b). However, due to India's considerable population of one billion people, this translates into 5.7 million infected people in 2005. India recently surpassed South Africa, with an estimated 5.5 million infected people, to gain the dubious distinction of the nation with the greatest number of people living with HIV at the end of 2006 (UNAIDS, 2006a). The HIV epidemic is a growing health threat to India's population.

In India to date, most infections have been confined to the more industrialized states in the south and the west. The overall epidemic in India is really a collection of smaller, more localized epidemics, and displays significant heterogeneity based on geographic location and risk behaviour (UNAIDS, 2006b; Blanchard et al., 2005; Becker et al., 2007). Initially, the epidemic was concentrated in densely populated urban areas such as Chennai and Mumbai, major cities in southern India. However, new evidence indicates that the epidemic is becoming established in rural populations as well (India-Canada Collaborative HIV/AIDS Project [ICHAP], 2004a; Becker et al, 2007). Until several years ago, there were very few HIV intervention programmes specifically targeting rural populations in India (ICHAP, 2004a).

1.3. Bagalkot District

The District of Bagalkot, a predominantly rural District located in the southwest Indian state of Karnataka (Figure A1.6 in Appendix 1), was chosen by ICHAP for a pilot programme that integrates District-level community-based rural HIV/AIDS prevention and care, focusing on vulnerable populations such as female sex workers (FSWs), while also covering as much of the wider population as possible. It is currently unclear why the epidemic has spread so quickly in some areas of Karnataka compared with others, and why HIV prevalence in rural areas is so high. From

surveillance conducted in Bagalkot District as part of the pilot programme, there is considerable heterogeneity in levels of infection in the overall population (ICHAP, 2004a; Becker et al., 2007). For example, HIV prevalence was higher in rural areas than in urban areas, and was found to vary across the three Talukas (sub-District administrative areas) surveyed, and as well between villages (20 urban blocks, 10 villages and seven towns were covered) (ICHAP, 2004a; Becker et al., 2007). The overall prevalence in 2004 in Taluka C is highest (4.9%), with Taluka B second (2.9%) and finally, Taluka A (1.2%) (Becker et al., 2007).

Several factors working alone or together may contribute to the different levels of HIV prevalence found in Bagalkot District. Based on the data available, the three Talukas differ with respect to some community-level risk factors (FSW size, client size and the estimated mean FSW-client contact rate). Less reliable data (factors for which we do not have data available for each Taluka) may also explain this heterogeneity in HIV prevalence (duration of female sex work [SW] and male client risk behaviour [MCRB], numbers of low-risk partners, transmission probabilities [and biological cofactors of HIV infection and transmission]), and factors relating to seasonal migration of FSWs and clients (such as size of the migrating populations, levels of sexual behaviour while in the migration destination, duration of migration, or HIV prevalence in high-risk partners in the migration destination). In this study, we seek to understand the impact of these parameters on the HIV epidemic in India by using simple transmission dynamics models and available data to define reasonable values for model parameters and scenarios analyses. Understanding the contribution of these risk factors to the epidemic reaching higher levels in some populations compared with others will assist in developing effective public health strategies for realistic interventions in rural areas. The effect that the epidemic has on India's population could be severe if well-planned and effective prevention programs are not instituted.

1.4. Study objectives and plan of analysis

The main goal of this project was to understand why three different Talukas in Bagalkot District had different HIV prevalence in 2004 in the overall, male and female populations. The intent of this study is to assist in the development of a rural HIV prevention strategy in Karnataka, India, by providing insight into the transmission dynamics of the epidemic in rural areas. The results from the analysis will assist in understanding how to effectively reduce the HIV epidemic in rural areas of India. Core group transmitters (FSWs) and bridging populations (individuals that link FSWs group with the general population, such as male clients of FSWs) will be focused on, as these groups are particularly vulnerable to HIV infection, due to their high-risk behaviour (Blanchard et al, 2005; O'Neil et al, 2004). Using a mathematical modelling approach (Chapters Three and Four), this project addresses the following main question: Which sexual behaviour and demographic characteristics of populations in three Talukas in Bagalkot District, a mostly rural District in Karnataka, India, contribute to explaining heterogeneity in levels of HIV infection? Secondary research questions include the following:

- How do certain demographic and sexual behaviour factors for which we have different data in each Taluka (including overall population size, size of the high-risk populations, and mean FSW-client contact rate) affect the local HIV epidemic in each Taluka?
- How much of the observed difference in HIV prevalence in 2004 between the Talukas is explained by the observed different values for these parameters?
- How do certain demographic and sexual behaviour factors for which we have limited data that is not stratified by Taluka (including mean numbers of low-risk sexual partners, duration of SW, duration of MCRB, transmission probabilities and the overall population growth rate) affect the local HIV epidemic each Taluka?

- How (and by how much) might each of these factors contribute to observed differences in HIV prevalence between the three Talukas?
- How (and by how much) do different patterns of seasonal migration and seasonal migration factors (including size of the migrating populations, duration of seasonal migration, mean high-risk contact rates and HIV prevalence in high-risk groups in the place of migration) affect the local HIV epidemic in Taluka A, the lowest prevalence Taluka?

To answer these research questions, the model was first fit to observed 2004 HIV prevalence data in each Taluka. Univariate sensitivity analysis was then conducted on parameters known to be different in the three Talukas (overall population size, FSW size, client size, FSW-client contact rate). Each parameter value was then adjusted in turn in one Taluka to the other Taluka's value (to identify important characteristics that explain differences in observed 2004 HIV prevalence). The population in each Taluka that acquired and transmitted the most infections was identified (to help direct prevention efforts at certain populations). A univariate sensitivity analysis was then conducted on parameters with limited data (duration of SW and MCRB, transmission probabilities, low-risk partner change rate), including migration parameters (only in Taluka A) and each parameter value was adjusted in turn (to identify important parameters that might explain differences in observed 2004 HIV prevalence). A multivariate sensitivity analysis was then conducted on migration parameters and parameters were adjusted together in Taluka A (identifies combinations of parameters that might have a large impact and explain differences in 2004 observed HIV prevalence). Finally, a simple condom intervention program was introduced into high-risk groups under certain conditions and the theoretical decrease in prevalence due to this intervention was evaluated.

CHAPTER TWO: LITERATURE REVIEW

2.1. Overview of the epidemiology of HIV in India

The first cases of HIV in India were discovered in Chennai, the capital city of the southwest state of Tamil Nadu in 1986 (Solomon, Chakraborty & D'Souza, 2004). Since then, HIV infection has been found in many areas across the country (Solomon et al., 2004). The National AIDS Control Organization (NACO) was founded in 1992 and is India's main governmental body that coordinates surveillance and control of HIV/AIDS in the country. NACO has provided HIV prevalence estimates each year since 1998 at different sentinel surveillance sites for different risk groups by state (NACO, 2004). UNAIDS estimates that a total of 5.7 [3.4 – 9.4] million people were living in with HIV in India by the end of 2006 (UNAIDS, 2006b). This latter value translates into about 0.9% [0.5 – 1.5]% overall prevalence (UNAIDS, 2006b). These aggregate data hide the highly heterogeneous localized epidemics in India, which differ widely in size and pattern. Six states in India (four in the south and two in the north) comprise about 80% of the total number of infections in India (UNAIDS, 2006b). The southern states, including Maharashtra, Andhra Pradesh, Tamil Nadu and Karnataka, contain about 75% of the total HIV cases in India, but represent only 30% of India's population (Arora, 2004). Heterosexual transmission is the main route of HIV transmission in the southern states, with injecting drug use causing the highest proportion in the northern states of Manipur and Nagaland (UNAIDS, 2006b). In these six states, HIV prevalence in pregnant women is greater than 1%, but most infections occur in high-risk groups (UNAIDS, 2006b). The former estimates are taken from antenatal clinics, which are assumed to be representative of the general populations. The HIV epidemic in these states appears to have stabilized, as the HIV prevalence in pregnant women has

remained the same in the last three years (UNAIDS, 2006b). Unfortunately, only about 1.6% of pregnant women with HIV receive access to treatment that reduces the possibility of mother-to-child transmission of HIV and only 7% of infected men and women in India receive access to antiretroviral treatment for HIV (UNAIDS, 2006b).

Levels of HIV infection vary widely between and within groups with different levels of high-risk behaviour. For example, FSW HIV prevalence varies widely across India. In Maharashtra (neighbouring state to Karnataka), HIV prevalence in FSWs was reported to be very high in 2001, at 58.7% (Family Health International, 2001). Within several large cities in Maharashtra, FSW HIV prevalence is much higher than the state average. In Mumbai, FSW prevalence was estimated to be 71% in 1997 (Schwartzlander, Garnett, Walker & Anderson, 2000), 54.3% in 2003 and 44.7% in 2004 (NACO, 2004) and 72% in 2004 (Chattopadhyay & McKaig, 2004. In Pune, FSW HIV prevalence has remained relatively stable over 10 years (46% [39.8 – 52.0]% in 1993 and 50% [32.7 – 67.3]% in 2002; $P = 0.80$) (Brahme et al, 2006).

2.2. Overview of the epidemiology of HIV in Karnataka and Bagalkot District

In Karnataka, one of the high HIV prevalence states in the southwest area of India, HIV surveillance and program implementation services are brought together by the Karnataka Health Promotion Trust (KHPT), a partnership between the Karnataka State AIDS Prevention Society (KSAPS) and the University of Manitoba, Canada (KHPT, n.d.). KSAPS is the government body implementing NACO's National AIDS Control Programme in Karnataka and the University of Manitoba is the chief executing agency for SANKALP, a project for scaling up HIV prevention in Karnataka (funded by the Bill and Melinda Gates Foundation) (KHPT, n.d.). ICHAP, established jointly by the governments of India and Canada in 2001 has implemented three programs in Karnataka, including two in Bagalkot District (see section: "Sources of Data" in Chapter Four). ICHAP was

funded for five years from 2001 – 2005 and its activities were absorbed by KSAPS after 2005. These organizations are the main sources of HIV prevalence data in this study.

According to the KHPT, AIDS is the leading cause of death among people 15-49 years old in Karnataka (KHPT, n.d.). NACO places estimates for antenatal clinic HIV prevalence in Karnataka at 1.3% in 2004, decreasing from 1.8% in 1998 (NACO, 2004); according to the KHPT, antenatal clinic HIV prevalence was 1.5% in 2004, with an estimated 500,000 people living with HIV in the state (KHPT, n.d.). HIV prevalence in antenatal clinics varies widely across Karnataka, with Shimoga District lowest at 0.5% and Belgaum District highest at 4.3% in 2004 (KHPT, n.d). Bagalkot District was fourth highest in 2004, with 2.6% antenatal clinic prevalence (KHPT, n.d).

Several intensive situation and needs assessments of the population were conducted by ICHAP and collaborating organizations in 2004 to collect baseline data on levels of risk behaviour and HIV prevalence in three of the six Talukas in Bagalkot District (ICHAP, 2004b). Overall, male and female HIV prevalence was significantly higher in Taluka C (overall: 4.9% [3.5 – 6.5]%, male: 6.4% [4.2 – 8.6]%, female: 3.2% [1.5 – 4.9]%), compared with Taluka B (overall: 2.9% [2.2 – 3.6]%, male: 3.0% [2.0 – 4.0]%, female: 2.8% [1.8 – 3.8]%), and Taluka A (overall: 1.2% [0.6 – 1.8]%, male: 1.1% [0.2 – 2.0]%, female: 1.3% [0.3 – 2.3]%) (Becker et al., 2007). Rural HIV prevalence was 50% (and significantly) higher in the overall population compared with urban areas (3.6% compared with 2.4%) (Becker et al., 2007). Prevalence was found to vary widely between the 10 villages sampled within the three Talukas, from 8.2% in Vantigodi to 1.0% in Kumbarhal (ICHAP, 2004a; Becker et al, 2007).

In Karnataka, FSW prevalence is estimated to be approximately 23.0% in 2005, with about half (47%) of women who operated out of brothels being HIV infected (Ramesh et al.,

2006). HIV prevalence in STD clinics (which generally measure HIV prevalence within high-risk groups) was 13.5% in 2003 according to KHPT (KHPT, n.d.) and 10.4% in 2003 according to NACO, from 7 sites (NACO, 2004). NACO also reported that HIV prevalence in STD clinics in Karnataka was 16.7% in 1998 and 12.0% in 2004 (NACO, 2004), possibly indicating that the epidemic is declining in high-risk groups in Karnataka, or that fewer high-risk people are being tested (or more have died from HIV infection). In three neighbouring Districts to Bagalkot District, FSW prevalence varies from 15% to 35%, and is 23.4% overall in 2005 (Ramesh et al., 2006). There are no estimates available for HIV prevalence among FSWs in Bagalkot District.

2.3. Characteristics of the study population that may affect the HIV epidemic

- **Demographics**

According to the India government census of 2001, there are approximately 1.65 million people in Bagalkot District, with over 70% of the population living in rural areas; the remaining 29 percent of the population lives in 11 urban towns, and there is no single large urban centre in the District (ICHAP, 2004b). This study will focus on the heterosexual population in the three Talukas in Bagalkot District since it is estimated that approximately 85% of HIV cases in India are caused by heterosexual contact, and mathematical models suggest that the epidemic is driven mainly by heterosexual sex (Venkataramana & Sarada, 2001; Arora, 2004). The overall population sizes in each Taluka are different from one another: Taluka B has the largest population, which is 1.6-fold the size of Taluka A and 1.5-fold the size of Taluka C, whereas Taluka A's population is 0.9-fold the size of Taluka C's (Ramesh, 2004).

- **Sexual behaviour**

Early in the HIV epidemic, simple mathematical models for HIV demonstrated the importance of collecting information on the variance of sexual activity (i.e. heterogeneity versus homogeneity),

as well as the mean sexual contact rate per unit time, due to the influence of these parameters on the initial spread and equilibrium prevalence of HIV/AIDS (Ghani & Boily, 2003). This resulted from the observations that the majority of people in a population have a small number of sexual partners in their lifetime, while a small proportion of individuals (core group transmitters), such as FSWs, have a high number of partners and high rate of partner change (Ghani & Boily, 2003; Lowndes et al., 2003). FSWs are core group transmitters in India's population, as they have a high average frequency of sexual contacts all year round; this creates many opportunities for them to pass on or come in contact with the virus (~10 per week in northern Karnataka) (ICHAP, 2002a). This vulnerable group currently has disproportionately higher levels of infection that the general population in India (see section on: Overview of the epidemiology of HIV in India).

FSWs infect male clients, who may pass the infection onto their wives or other sexual partners (Lowndes et al, 2002). These women, who are traditionally termed "low-risk" (LRF) because their only male partners are their husbands, will have a much heightened risk of becoming infected if their husbands are clients of sex workers. The men who introduce the infection in the low-risk female population are referred to as the "bridging population" (Lowndes et al, 2002). Without the heterogeneity introduced by core and bridging groups and the sexual network linking them to lower risk population, STI and HIV infections may never spread to the low-risk population and may have remained concentrated in the high-risk population, because of the low overall level in sexual activity in the low-risk groups that prevent the establishment of infection (Ghani & Boily, 2003; Lowndes et al, 2002; Nagelkerke et al, 2001; Venkataramana & Sarada, 2001). In Cotonou, Benin, for example, a city with a relatively low HIV prevalence, Lowndes et al (2002) have suggested that most new HIV infections could be caused by core and bridging groups, and a small number by individuals in the general population.

Partnerships between individuals with different risk behaviours (such as those between low-risk females and MCLs) are more formally defined by the sexual mixing pattern and are important to include when modelling STIs (including HIV); mixing patterns have been shown to have a striking influence on the outcomes of STIs (Boily and Anderson, 1991; Garnett & Anderson, 1993; Garnett et al., 1996, as cited in Ghani & Boily, 2003). Assortative mixing patterns (like-with-like), where partnerships with similar sexual behaviour are formed (e.g. number of partnerships, frequency of partner change, between low-risk females and low-risk males, or FSWs and MCLs), increase the likelihood that an infection will establish itself in a population, but the final endemic prevalence is lower than for a less assortative mixing (i.e. disassortative or proportionate mixing), as the infection remains trapped in the population with high sexual partner change (Ghani & Boily, 2003). However, pure assortative sexual mixing patterns are uncommon in real life—individuals in a population do not all exhibit the same type of sexual behaviour, and frequently have partners with a different level of sexual activity (Ghani & Boily, 2003; Lowndes et al, 2002).

It has been estimated that between 50% and 75% all primary and secondary infections of HIV infections in the total population in India could result from contact with commercial sex workers (Arora, Cyriac & Jha, 2004). Bagalkot District's population exhibits a large amount of heterogeneity in levels of sexual behaviour within its Talukas. For example, there is a significant population of rural sex workers, including a substantial proportion of FSWs from the Devadasi tradition, or traditional-based religious sex work (O'Neil et al., 2004). This is the most common form of sex work in the region. Records dating back to the 12th century describe the Devadasi tradition, which is ingrained in the culture and society of Karnataka (O'Neil et al., 2004). The Devadasi tradition involves young women being dedicated by their families into marriage to

gods or goddesses, and subsequently performing various duties within the temples, including sex work (O'Neil et al., 2004; Blanchard et al., 2005). This temple-based sex work, due to its association with religion, has gained an acceptance in society that other forms of sex work do not. In Karnataka, Devadasi sex workers are significantly less likely to report police harassment or client violence than other FSWs (Blanchard et al., 2005). The Devadasi report a higher mean number of partners than other commercial sex workers (Blanchard et al., 2005).

Evidence of MCLs who have partnerships with both low-risk and high-risk individuals (low-risk females and FSWs), constituting a bridging population, has been documented in Bagalkot District and India. Premarital and extramarital sex is considered a demonstration of virility in men in India (ICHAP, 2003). 24.7% of married men in the three Bagalkot District Talukas studied reported that they had visited female sex workers in their lifetimes, with 8.1% of married men stating that they had had sex outside of or before marriage (Ramesh, 2005a). It is important to consider both core and bridging groups in this study since it is clear that male clients in Bagalkot District have partnerships with high-risk women, who have a high level of sexual activity, and with their wives, who generally only have their husbands as partners and who have a low level of sexual activity (ICHAP, 2003).

Risky sexual behaviour, defined as having any risk behaviours, such as sex with a non-regular partner in the last 12 months, having had more than one sexual partner during lifetime and having ever paid/ received money for sex, is in general reported more often in men compared with women in the general population. In Bagalkot District, 7% of total respondents (13% of males and 2% of females) reported that they had ever engaged in risky sexual behaviour in 2004 (ICHAP, 2004). In rural areas this proportion was 9% and in urban areas was 7% (ICHAP, 2004a). Further, rural males were more likely to have risky sexual behaviour than

urban males (15% compared with 12%). There was an even larger variation across villages and towns with respect to the proportion of men engaging in risky sexual behaviour. Men from several villages and towns in Bagalkot District, including Vantigodi (34%), Mirji (20%) and Shirol (29%) all displayed different levels of risky sexual behaviour (ICHAP, 2004a). These figures are from a survey that was administered face-to-face however, and may underestimate the level of risky behaviour in both males and females in the population.

Other characteristics of sexual behaviour, such as the size of the sex worker and client populations, average number of new partners per timeframe (mean FSW-client and client-FSW contact rates) and total number of partnerships, have also been shown to be significant factors in HIV epidemics in mathematical modelling studies. A larger FSW population (assuming a constant mean FSW-MCL contact rate per unit time) caused higher levels of HIV infection in the overall population, as did increasing the FSW-MCL contact rate per unit time (assuming a constant FSW population size), since the total number of FSW-MCL partnerships and levels of sexual activity increased (Ghani & Aral, 2005). However, when the client population size was varied, this parameter had a comparatively small effect on overall prevalence. The size of the FSW population determined the levels of HIV in the infection and the MCL population, as a bridging population, simply determined the level of infection that would be transmitted into the general population (through the low-risk female partners of the MCLs). The type of FSW-MCL relationship (one-time partner versus regular partner) may also be important. The small effect of MCL size on HIV prevalence in Ghani & Aral (2005) was particularly evident when clients consistently chose the same FSW rather than visiting different FSWs.

Other factors, such as concurrency (many partners at the same time), or gaps of time between partnerships, also shape STI and HIV epidemics. In a telephone survey of urban 18-39

years old individuals, the correlation of individual (describing the actions of the partner alone) and partnership (describing the actions of the partner and the respondent) concurrency with an STI diagnosis in the previous year were both shown to be positive, even when lifetime number of sex partners was controlled for (Manhart, Aral, Holmes, & Foxman, 2002). Kraut-Becher and Aral (2003) measured the effect of concurrency and gaps by recording the date of the difference between the date of first sex with a current or most recent partner and the date of last sex with a previous partner, for a cohort of women. Researchers found that for many of these women, the duration of time that passes between different partners is shorter than the mean infectivity periods of many STIs; this increase their risks of STI transmission (Kraut-Becher & Aral, 2003). While concurrency and gaps may be important in determining risk of HIV transmission, the simple models used in this study will focus instead on frequency of partners per year.

- **Migration, mobility and geographic location**

Migration is thought to be a factor in increasing HIV infection in many countries because migrants who leave their families for extended periods of time are more likely to engage in risky sexual behaviour (such as increased numbers of partners in general, and with partners who may have higher rates of HIV infection themselves) when they are away, placing them at greater risk for contracting HIV (Solomon et al, 2004; Lurie et al., 2003). In addition, overall HIV epidemics may increase because sexual networks, which are influenced by the geographic location of individuals (Ghani & Boily, 2003) are made between groups who wouldn't normally interact (Halli, Blanchard, Satihal, & Moses, 2007). Mobile people may also be less aware of their HIV status than non-mobile people, and have less access to STI/HIV-related health services.

Migration has been linked to increased vulnerability to HIV in both industrialized and developing countries (Coffee, Lurie & Garnett, 2007; Lacey & Merrick, 2005; Bronfman, Leyva,

Negroni, Rueda, 2002; McCoy, Weatherby & Yu, 1999; Pison, Le Guenno, Lagarde, 1993, as cited in Boerma et al, 2002). A higher HIV prevalence at the site of migration was shown to increase HIV prevalence in South African migrant workers (Coffee et al., 2007), and these increases were greater if there was an associated increase in high-risk behaviour among migrants (such as partner change rates). Increases in equilibrium (a stable epidemic) HIV prevalence were seen in non-migrant men and women only when there were increases in partner-change rate among migrants in this population (which is seen in observed data) (Coffee et al, 2007). In a Kenyan population, the most important factor associated with males visiting FSWs was making frequent or long journeys away from home (Voeten et al., 2002).

Some contradictions to the commonly perceived idea that migration increases HIV epidemics were found in a pilot project conducted in Bijapur District, a neighbouring District to Bagalkot District, in Karnataka. This study found that the proportion of male migrants visiting FSWs or engaging in casual sex was not significantly higher than the proportion of non-migrants (Halli et al, 2007). Although verbal autopsies of AIDS deaths indicate that migrant status was linked to AIDS in an earlier stage of the epidemic, the correlation between AIDS and being a migrant appears to have decreased in recent years (Halli et al, 2007). However, although condom use is reported more often in migrant males compared to non-migrants, the prevalence of STIs in migrant men was significantly higher than in non-migrant men (Halli et al, 2007).

Mobility is common among sex workers in northern Karnataka, and frequent locations of migration include the major cities of Sholapur, Sangli, Mumbai and Pune. FSWs tend to travel to places with festivals between December and April (festival season), when they are able to make more money (ICHAP, 2003). 83% of sex workers may be out of their village in Bagalkot District at any given time, indicating some level of migration from the District (ICHAP, 2003). Devadasi

sex workers are less likely to travel outside their town or city to other areas in Karnataka, but are more likely to travel outside of the state, especially to Mumbai (Blanchard et al., 2004), where HIV prevalence is higher (Chattopadhyay & McKaig, 2004).

In rural areas of Bagalkot, frequent travel due to work seems to be associated with a higher prevalence of HIV, although the associations were not significant for the total population or the urban population (ICHAP, 2004a; Becker et al, 2007). Of the total population of Bagalkot District, travel for work is common, possibly increasing migrants' exposure to HIV infection compared to non-migrants in the population (ICHAP, 2004a). 5% of rural respondents who reported that they travel regularly for work had HIV, compared with 3% of those who did not report travel. Rural respondents are more likely to travel due to work than urban respondents, with 22% travelling compared with 16%. Duration and location of travel may also be important when considering the impact of migration on sexual network patterns. Of the total respondents, 9% travel daily, 4% travel weekly, 1% travel monthly, and 5% travel once in awhile (ICHAP, 2004a). 7% of total respondents reported that the duration of their travel lasted more than a month during the past year (11% female and 3% of males). 4% of females reported that they had travelled to places within the District, 6% outside the District and 1% outside the state (ICHAP, 2004a). Overall, 18% of people reported that they travel due to work, with 34% being male and 4% female (ICHAP, 2004a). Indian males who migrate may be an important bridging population, as they may infect their wives when they return (Solomon et al, 2004).

- **Biological characteristics**

Considerable evidence exists showing that transmission probabilities of HIV are affected by many factors, including stage of infection, concurrent sexually transmitted infections (STIs), type of sexual intercourse (penile-anal, penile-vaginal, oral, dry), circumcision of males, sex during

menstruation and condom use (Baggaley, Boily, White & Alary, 2005). Wawer et al (2005) showed that in a cohort of HIV-discordant couples in a Ugandan population, HIV-infected people are the most infectious (per sexual contact) during the first several months after infection. Between six and 15 months after seroconversion by the index partner (second stage of infection), individuals are the least infectious, and this rises steadily with increasing viral load through the third stage, during which infectivity is somewhere between that of the first and second stages (Wawer et al., 2005). Transmission from males to females is generally thought to be higher than from females to males, although not all studies show these results (Baggaley et al., 2005). In some partnerships in Bagalkot District, there are many sex acts, in others, there are very few, sometimes only one and this affects transmission probability values. Differences in transmission probabilities between these types of partnerships will be taken into account when estimating the values of these parameters.

CHAPTER THREE: METHOD OF ANALYSIS

3.1. Mathematical modelling method

The influence the factors described in Chapter Two have on HIV transmission and heterogeneity in HIV prevalence in three different Talukas in Bagalkot District (and in India in general), is not well understood, especially in rural areas. Different types of mathematical models can be used to help identify important risks for acquiring and transmitting sexually transmitted infections such as HIV and provide insight into how these factors affect the spread of HIV in different settings (Boily and Anderson 1991, Garnett, 2003). In a study conducted by Turner, Garnett, Ghani, Sterne, and Low (2004), simple deterministic models were used to estimate the impact of various sexual behaviour risk factors (such as levels of sexual activity and reported numbers of partners for men and women) for contracting gonorrhoea in men and women in three different ethnic groups. Ghani and Aral (2005) used a network model to determine how FSW and MCL size, as well as variation in the numbers of MCL partners per FSW (and if the partners were one-time or frequently visited) affected the endemic prevalence of gonorrhoea and herpes simplex virus 2. Coffee et al. (2007) used a deterministic model to assess the impact of various aspects of migration (such as high-risk behaviour and HIV prevalence in the place of migration) on local epidemics South Africa. Some of these studies, and more, were discussed in greater detail in Chapter Two to provide support for the reasoning behind investigating the effects of certain factors on HIV prevalence in Bagalkot District. Mathematical modelling techniques have not been used in depth in India to explore some of the important factors that may contribute to the heterogeneity of the HIV epidemic and this study will therefore add to the body of knowledge encompassing HIV transmission in India.

Mathematical models can also be used to assist in the interpretation of HIV surveillance data, and as predictive tools to understand the likely future of an epidemic and its impact on a population, given the information available at a given moment in time (Garnett, 2002). For example, Venkataramana and Sarada (2001) used estimates of the number of FSWs and their work patterns, and prevalence of HIV and STIs and condom use among FSWs in 1999 (with reasonable estimates for other relevant parameters) to estimate the spread of HIV infection in commercial sex networks until 2005. Anderson, May, and McLean (1998), Anderson, May, Boily, Garnett and Rowley (1991) and Bongaarts (1989) used mathematical models to predict the possible demographic impact of HIV in various settings and circumstances.

Mathematical models have proved useful to assist in the interpretation of reliable and valid descriptive data and are used to help determine which populations would be best targeted with interventions, assisting in the design of intervention programs (Garnett, 2002; Ghani & Boily, 2003; Alary, Lowndes & Boily, 2004). Targeting intervention strategies at core groups, for example, has been shown theoretically to reduce the infection levels in the wider population. A modelling analysis of the dynamics of HIV infection in Cotonou, Benin suggested that the intervention programs aimed at core groups contributed to a reduction in prevalence and incidence of HIV in both the high- and low-risk groups (Boily et al, 2002). This was supported by an empirical study (Lowndes et al, 2002). Nagelkerke et al (2000) suggested that a single intervention (increasing condom use from 33% to 75%) targeted at FSWs in India may drive the epidemic to extinction, and that this single intervention would likely be as effective as several implemented at once. This helps to direct scarce resources toward HIV prevention interventions.

Mathematical models can be useful in situations where it is unfeasible or difficult to institute community-based randomized trials (CBRTs), the current gold standard for evaluating

interventions (Ghani & Boily, 2003). CBRTs offer many advantages; however, they are often very expensive, and they require large sample sizes (Ghani & Boily 2003). There are complex ethical concerns involved with providing one group of individuals with some form of treatment, and one group without. The complex transmission dynamics of HIV also make design and interpretation of CBRTs difficult; this may mean that the conclusions from CBRTs have limited relevance of in the real world (Ghani & Boily 2003). Modelling is also less expensive, has fewer ethical concerns, can be simple or complex, and is easily incorporated into routine monitoring and assessment activities (Ghani & Boily 2003; Garnett, 2002).

Transmission dynamics models, which take into account the biology of the infection and the demographic and behavioural characteristics of the population to describe the spread of infection over time, will be used in this study. Such models reflect the dynamical nature of HIV epidemics and can introduce changes in sexual behaviour and other risk factors over time. These types of models have proved useful to define important epidemiological quantities that summarize the most important determinants of epidemic spread and control (Brunham & Boily, 1993; Garnett, 2002; Ghani & Boily, 2003). One of these well-known quantities is the basic reproductive number, R_0, defined as the average number of secondary infections caused by an infected person introduced into a wholly susceptible population (Garnett, 2002). This parameter predicts whether or not the epidemic will establish itself in a population ($R_0 > 1$), die out ($R_0 < 1$) or remain stable ($R_0 = 1$) (Boily & Alary, 2002). In it simplest form $R_0 = C * B * D$ depends on 1) sexual behaviour (C = average rate of partner change) and the biology of the infection with 2) B = probability of transmission when an infected person encounters a susceptible person and 3) D = duration of infectiousness (Ghani & Boily, 2003). R_0 is central to the prevention of infectious diseases, because ultimately we are trying to reduce R_0 through a change in C, B or D.

In this study, compartmental, population-based deterministic transmission dynamics models are used. Many mathematical modelling studies use these types of models to gain insights into sexually transmitted infections (Boily & Masse, 1997; Renton, Whitaker, & Riddlesdell, 1998; Turner et al., 2004; Coffee et al., 2007). Compartmental mathematical models represent heterogeneity in sexual behaviour by stratifying the population into groups with similar characteristics; population-based models use parameter averages estimated for each of these groups, rather than having individuals and their characteristics be individually simulated (Garnett, 2002). Deterministic models assume "that events are not subject to chance and two realisations of a model using the same parameters and exact starting conditions will give exactly the same results", as opposed to stochastic models, which incorporate some level of randomness (Garnett, 2002). The deterministic models in this study are made up of differential equations (one set for each compartment: shown in Figure A1.5 in Appendix 1), which predict results that are continuous over time, as opposed to models that use difference equations and predict results in discrete units (i.e. units of time) (Garnett, 2002). The equations are non-linear (as opposed to linear), because the transmission of STIs such as HIV are more complex and dependent on both susceptible and infected populations (Garnett, 2002). Results from the model analysis will be both quantitative and qualitative.

3.2. Ethical considerations

This study was given approval from the Health Research Ethics Board (HREB) of the University of Manitoba, Winnipeg, Canada. To estimate values for model parameters in this study, secondary data collected by ICHAP on Bagalkot District, Karnataka was used (ICHAP, 2004a; ICHAP, 2004b; ICHAP, 2003; ICHAP, 2002s), as well as data from literature reviews. ICHAP data were collected in accordance with the ethical requirements of community-based HIV

prevalence studies, and the design and protocols had a prior approval from the HREB as well as the Research Review Board of St. John's Medical College, Bangalore, India (ICHAP, 2004a). Access to the identities of the individuals who were surveyed is not provided, and so confidentiality and anonymity of the individuals will be assured. One ethical issue that must be considered is that researchers must accurately represent the data they are interpreting (Creswell, 2003). To ensure that the results of the analysis and subsequent conclusions were as accurate as possible, a thorough uncertainty analysis was conducted. Since we examined the effects of important parameters in each of three Talukas, there were many opportunities to assess the validity of the data used in this study.

CHAPTER FOUR: MODEL DEVELOPMENT

4.1. Model scenarios

In this chapter, the different model scenarios examined in this study are described. Two base-case scenarios (B-C) without migration were used: base-case scenario I (B-CI) and base-case scenario II (B-CII), and one B-C with migration for each migration scenario (each referred to as B-CM and described in this chapter). In Chapter Five: Model Fitting we describe how B-CII allowed us to fit the model to observed data better than B-CI, and how we used B-CII to examine the independent effects of all non-migration risk factors (including parameters for which we have data for each Taluka: overall population size, size of the high-risk populations, mean high-risk contact rates, as well as parameters for which we do not have data for each Taluka: duration of FSW, duration of MCRB, transmission probabilities, average numbers of low-risk partners). We used B-CM (all migration parameters at base-case values) in Taluka A only to examine the independent effects of all migration parameters (including size of the migrating population, duration of migration, mean high-risk contact rates and HIV prevalence in high-risk groups in the place of migration). We conducted a univariate sensitivity analysis and varied all parameters by the same amount proportionately with other parameters at B-C values.

"B-CI", "B-CII" and "B-CM" refers to the scenario's model structure and the model parameter values associated with each scenario. Table A1.1 in Appendix 1 provides estimates for model parameters used in the mathematical models in this study and indicates whether this data is used for B-CI, B-CII or B-CM (some parameter values are common to all scenarios). Table A1.1 also lists the parameter names used in the model for each parameter value and the final column also describes the references for the parameter estimates as well as the quality of the

data. Some parameters were estimated from data taken directly from Bagalkot District, but other parameters were estimated using data from outside Bagalkot District and in some cases, even outside of India. The parameters were categorized as epidemiological, demographic, sexual behaviour and biological (described in greater detail in this chapter). There is a separate section for migration parameters as this analysis required different model structuring for both MCL and FSW migration. In this chapter, we also describe the major sources of data used to estimate values for model parameters and how B-C values were estimated for each parameter. The model equations (including parameters and scenarios) are shown in Figure A1.5, Appendix 1.

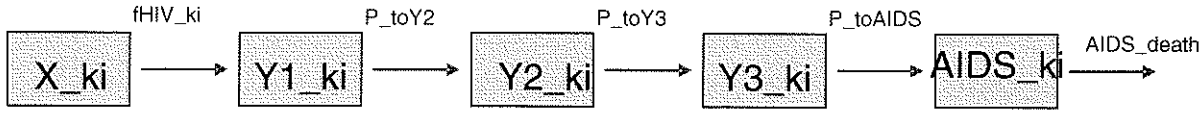
Biological characteristics

All base-case scenarios included natural history characteristics of HIV infection to represent the dynamics of infection accurately (Anderson & May, 1991; Boily et al, 2002). Figure 4.1 represents the progression of individuals of gender "k" (k=M [male] or F [female]) and risk group "i" (i=H [high-risk] or L [low-risk]) through the five stages of HIV infection (red arrows). Each of the bolded square represents a different stage of the disease: susceptible, first infectious stage, second infectious stage, third infectious stage and AIDS, represented by Y1, Y2, Y3 and AIDS in Figure 4.1. Previous mathematical models have shown that it is important to include four stages of infection when modelling HIV (Ghani, & Boily, 2001).

Susceptible individuals can become infected and move to the infected compartment at a per capita rate f_HIV, which corresponds to the risk of infection or force of infection of uninfected individuals (Figure 4.1). The force of infection depends on the frequency of contacts with sexual partners (and other risk behaviours with the partner), the probability that their partner is infected and the probability that the person will become infected upon contact with an infected partner (Anderson & May, 1991; Brauer & Castillo-Chavez, 2001; Ghani & Boily, 2003).

Following infection with HIV, individuals progress through several infection stages before succumbing to the illness. HIV positive individuals can transmit infection during each of the three stages but with different probability (described in section 4.3) (Boily et al, 2002). However, it is assumed that people in the AIDS stage are usually very ill and physically unable to have sexual relationships, and therefore also unlikely to transmit infection (Boily et al, 2002). The rates of progression from one stage to the next are the inverse of the estimated mean duration of time spent in each infected stage. People move from infected stage 1 by a rate p_toY1 (duration=5 months); stage 2 to stage 3 as p_toY2 (duration=8 years); and stage 3 to AIDS as p_toAIDS (1 year); and from AIDS to death (9 months) (Figure 4.1; Figure A1.4, Appendix 1).

Figure 4.1. Flowchart of infection progression



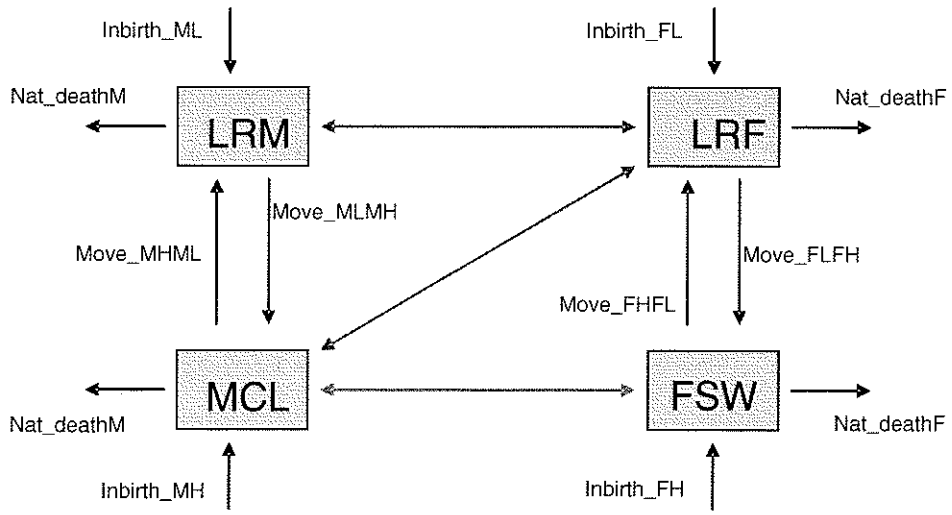
Sexual behaviour

The B-C scenarios (B-CI and B-CII) all include four compartments: low-risk females (LRF) low-risk males (LRM), high-risk females (FSW) and high-risk males (MCL). B-CI assumes lifelong duration of high-risk behaviour (FSWs engage in sex work and MCLs visit sex workers their whole sexually active lives) and B-CII assumes that FSWs and MCLs change behaviour after a mean number of years and become low-risk (they are replaced by the same number of low-risk females and males to balance model equations). In each B-CI and B-CII we examined two different sexual mixing patterns: purely assortative and strongly assortative. In purely assortative mixing, low-risk females (women who have never engaged in sex work), have exclusive long-term relationships with low-risk men (who have never visited a sex worker), who in turn only have long-term exclusive relationships with low-risk females. As well, high-risk males only have

many short-term, one-time contacts with high-risk females, who in turn only have many short-term, one-time contacts with high-risk males.

For non-purely assortative mixing, some low-risk females will have exclusive long-term relationships with low-risk males and some have exclusive long-term relationships with high-risk males. High-risk males can be clients of sex workers (with whom they have many short-term, one-time contacts) and also can have exclusive long-term relationships with low-risk women; thus, high-risk males are a bridging population from the higher-prevalence FSWs to much lower prevalence low-risk females. Other mixing patterns stay the same (i.e. low-risk males only have exclusive long-term relationships with low-risk females; high-risk females only have many short-term, one-time contacts with high-risk males). Figure 4.2 (and Figure A1.1 in Appendix 1) shows the non-purely assortative mixing pattern for B-CII, the model scenario used in all analysis without migration. People move in and out of each population (compartment) by a rate that is inversely proportional to the mean number of years they are sexually active.

Figure 4.2. Flowchart for sexual behaviour (B-CII): demographic movement in/out of each population by sex/ risk group (black single-headed arrows: inbirth_ki=birth rate into the population; nat_deathk=death rate out; blue single-headed arrows, move_kiki=rate of movement from high- to low-risk and replacement; sexual relationships (red double-headed arrows= low-risk relationships; green double-headed arrows=high-risk contacts).



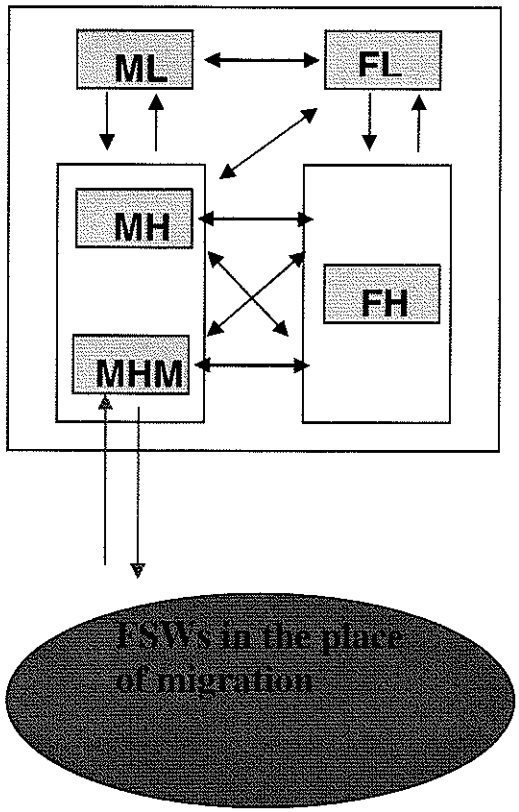
Migration

Only Taluka A was used to examine the effects of seasonal out-migration in Bagalkot District, because model prevalence in Taluka A initially fit best to observed data for all populations with B-CII parameter values. "Seasonal out-migration" is defined as migration that happens only once a year for a duration of less than one month every year. The frequency is always annual. In this analysis, "in-migration" (clients migrating in from a place of origin) is ignored. For all migration scenarios, we assume that there is non-purely assortative mixing and that the duration of high-risk behaviour (MCRB and SW) is less-than-lifelong (B-CII). For each of the three migration scenarios described below, we provide simplified flowcharts. Refer to the Appendix for more detailed flowcharts of male and FSW migration (Figures A2 and A3).

- **Out-Migration Client Scenario I (M.1 and M.2)**

In this scenario, we assume that a proportion of clients migrate (MHM) away once a year and that migrating male clients in the place of origin are also clients in the place of migration (Figure 4.3). All general features of sexual behaviour and biological characteristics remain the same as in B-CII (Figure 4.2), but MCLs are split into migrants and non-migrants. Migrating male clients interact with FSWs in the place of migration with varying number of contacts and with two FSW HIV prevalence scenarios: 1) constant HIV prevalence (M.1) and 2) logistically increasing HIV prevalence (M.2). Out-migrating clients are not replaced during the duration of their out-migration and we assume that the client demand for sex determines the number of local client-FSW contacts (mean FSW-client contact rate remains the same, thus the mean local client-FSW contact rate drops). We also assume that the mean low-risk male per low-risk female partner change rate is unaffected by male migration. Figure A1.2 in Appendix 1 shows a more detailed flowchart detailing important features of male migration in M.1, M.2 and M.3 (next section).

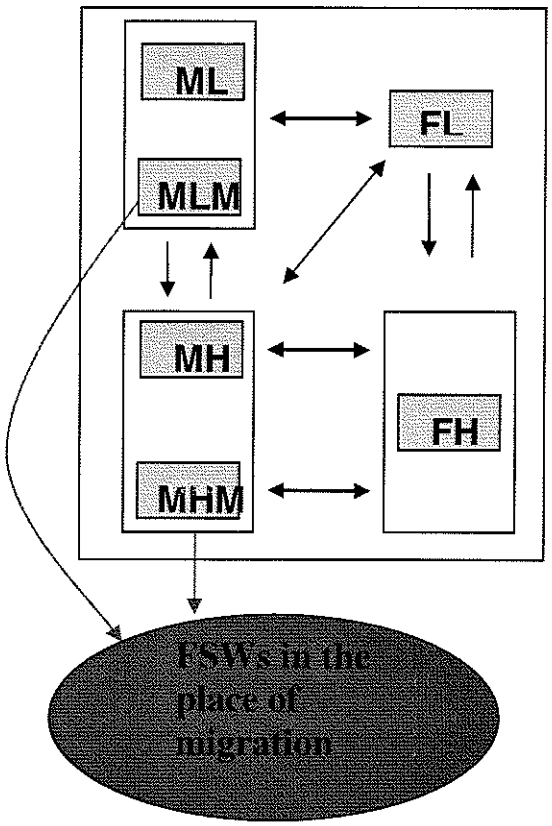
Figure 4.3. Flowchart for sexual behaviour characteristics of male client migration (M.1, M.2)



- **Out-Migration Client Scenario II (M.3)**

In this scenario, instead of just a proportion of local MCLs migrating to the POM, a proportion of all men migrate: both high-risk (MHM) males and low risk males (MLM) can migrate away once a year (Figure 4.4; Figure A1.2 in Appendix 1). Again, all general features of B-CII remain the same (Figure 4.2). All migrating men (MHM and MLM) are clients of FSWs in the POM (again, with varying number of contacts but with only one POM FSW HIV prevalence scenario: logistically increasing HIV prevalence) but MLM are only clients while in the POM. Again, we assume that the client demand for sex determines the number of local client-FSW contacts while clients are in the POM (mean FSW-client contact rate remains the same, thus the mean local client-FSW contact rate drops). We also assume that the mean local low-risk female per male partner change rate is unaffected by low-risk and high-risk male migration.

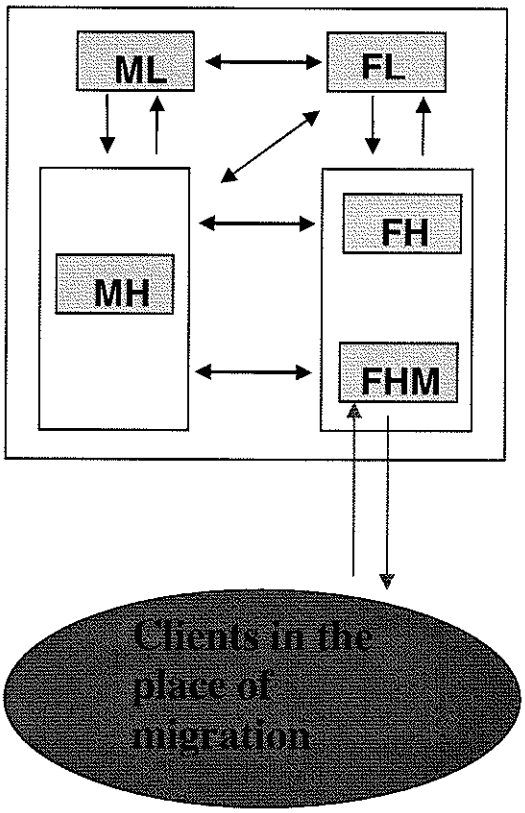
Figure 4.4. Flowchart for sexual behaviour characteristics of all male migration (M.3)



- **Out-Migration FSW Scenario (F.1 and F.2)**

In this seasonal out-migration scenario, a proportion of FSWs migrate away for a set duration of time each year (Figure 4.5; Figure A1.3 in Appendix 1). Again, all general features of B-CII remain the same (Figure 4.2). All of this proportion has sexual contact with clients when they are in the POM. Migrating FSWs interact with MCLs in the POM with varying number of contacts and with two MCL HIV prevalence scenarios: 1) constant HIV prevalence (F.1) and 2) logistically increasing HIV prevalence (F.2). We assume that the client demand for sex determines the number of client-FSW contacts while FSWs are in the POM (mean FSW-client contact rate remains the same, thus the mean local client-FSW contact rate increases.

Figure 4.5. Flowchart for sexual behaviour characteristics of FSW migration (F.1, F.2)



4.2. Sources of data

Model parameter values and initial conditions were estimated from several sources of data, predominantly gathered from the Bagalkot Demonstration Project (BDP) and the Statewide FSW Survey (SFSWS), implemented by ICHAP and the PRC. These initiatives are described in greater detail below. India government census data, NACO data and data gathered from a literature review were also used to estimate some model parameters.

- Bagalkot Demonstration Project

The goal of the BDP was to develop and pilot a programme model for District-level community-based rural HIV/AIDS prevention and care, reaching both vulnerable populations and the general population (ICHAP, 2004b). Beginning in July, 2002, a series of assessments of the area was carried out; data used to estimate parameters for this study will be obtained from these

assessments. The timeline for the assessments that are relevant for this study is as follows: July-September, 2002, a situation and needs assessment (SNA) was carried out in three Talukas in Bagalkot, as well as a behavioural study and sex work survey; early 2003, a Community-Based HIV Prevalence Study (CBHPS) in three Talukas (10 rural villages and 20 urban areas), social mapping of villages in four Talukas; April 2003, household surveys in 308 villages were carried out; September-December, 2003, an SNA for the remaining Talukas in the area in preparation for project expansion to these Talukas; March 2004, social mapping and household surveys of villages in these remaining Talukas.

Collaborating with ICHAP, the Population Research Centre (PRC), Dharwad, undertook the CBHPS, and data were collected within three Talukas in Bagalkot District. The 10 villages in the study were randomly selected from 237 rural villages in the District, and 3300 persons were randomly selected from within these 10 villages. In addition, 20 randomly selected urban blocks were taken from six towns in the District and 3300 persons were randomly selected from these areas. The selection of urban blocks was based on a systematic random sampling procedure (ICHAP, 2004a). The sampling frame was created from a census of households and individuals in these selected villages and urban blocks, and a list of all persons 15-49 years was prepared. Sex, age and marital status were used as stratification variables in the final selection of 3300 persons from the rural villages and 3300 persons from the urban blocks, and a list was created of both of these samples. The sampling procedures in the CBHPS were used so that the data would be representative of the entire population in each Taluka sampled and taken into account into analysis. The survey questionnaire included personal information of respondents (respondent's name, id number, interviewer's name, date of interview, consent); socioeconomic background of the respondent; behavioural characteristics; knowledge and attitudes about STIs, HIV/AIDS and

sexuality; socioeconomic and demographic characteristics of the spouse; and attitudes toward condom use, persons with HIV and sexuality (ICHAP, 2004a). A pre-test of the questionnaire was conducted in two villages near Dharwad, Karnataka. The questionnaire was translated into Kannada, the common local dialect, and the field staff (interviewers, team supervisors and nurses) was trained in asking questions and recording information (ICHAP, 2004a). The interviews were conducted face-to-face (ICHAP, 2004a).

A series of polling booth surveys (PBS) were also conducted as part of the BDP, both in 2004 and 2005, in the three Talukas in Bagalkot District (Kang, 2005). PBS were administered in 12 "cohort" villages (to be included in all subsequent PBS rounds) and 24 cross-sectional villages (new ones to be chosen in all subsequent PBS rounds, with selection by the probability proportional to size sampling method) (Kang, 2005). Participants were selected by a stratified systematic random sampling method conducted in each village, and included members from six demographic groups (Kang, 2005). During PBS, interviewers call out questions to participants, who are physically shielded from other participants, and who answer each question with a separate piece of paper. The interviewer does not know their response to the question, thus facilitating truthful answers and reducing social desirability bias (answering questions according to what the respondent believes will gain a positive response from the interviewer). Data such as the proportion of males who have ever visited FSWs can be obtained from these surveys.

- Statewide female sex worker survey

This baseline survey of FSWs in Karnataka was undertaken by the PRC and ICHAP in 2002, with the goal to collect information on a random sample of FSWs in the state (ICHAP, 2002a). As there was no existing sampling frame for FSWs in the state of Karnataka, detailed mapping was undertaken in collaboration with sex worker collectives in different areas in the state

(ICHAP, 2002a). Mapping was carried out by several groups, who first identified the villages and towns (in each District) in which sex work occurs (ICHAP, 2002b). A list of locations where sex workers work was created for all of the Talukas in each District, and only the locations with at least five sex workers was kept. The total number of sex workers who were members of a collective in each location was estimated by the representatives from that collective, and a best estimate of the number of non-members was made. Researchers then estimated these numbers again, but this time by the place where the sex worker normally worked (home-based, brothel-based, lodge-based, roadside-based, etc.), and corroborated the results for consistency (ICHAP, 2002b). This process was repeated for all of the Talukas within all of the Districts in the state of Karnataka (ICHAP, 2002b).

The baseline survey data were collected by a team of interviewers, consisting of three interviewers and one supervisor (ICHAP, 2002a). Sex workers were contacted initially by facilitators from collectives in their area and if they agreed to take part in the survey, they were contacted by the survey team. The field teams were trained for two weeks in the month of July, 2002. The data was collected between 29 July and 28 October, 2002. The survey covered 1,598 female CSWs, out of which the interviews were successfully completed for 1,512 CSWs or 84 percent of the target sample size. Information on migration patterns of sex workers was collected, including information on destination of migration (ICHAP, 2002a). In addition, the number of clients each FSW had per week was collected during this survey, and the total number of people with whom each sex worker had sex with was collected. While the mapping exercise underestimated the total number of sex workers in the District, behavioural data collected in the survey from the sex workers is reasonably accurate.

4.3. Parameter estimation *(Table A1.1 in the appendix lists all parameter estimates)*

Epidemiological parameters

Initial conditions—to seed the epidemic: We assumed there were initially only one HIV-infected person in each low-risk group and two in each high-risk group in 1976 (*noinf_ki*), ten years before the year that HIV was first detected in India, 1986 (NACO, n.d). The exact number of people initially infected with HIV in Bagalkot District in 1976 is not known. Seeding the model with a small number of people around the time when HIV was detected in India allowed us to validate the model with current estimates of HIV.

Model validation: We validated model predications for HIV prevalence in 2004 with observed 2004 HIV prevalence in the overall, male and female populations in each Taluka as well as in the high-risk populations across all Talukas. Details on how the observed HIV prevalence was estimated are found in Chapter Two: Literature Review.

Demographic and sexual behaviour parameters

Total population

- Rate of movement out of the sexually active population ("death rates")

Males: The median that age that males began sexual activity was 23.9, 22.4, and 21.1 years in Talukas, A, B and C respectively (ICHAP, 2004a). Assuming that sexual life expectancy ended at 49 years (we have data for the population size of the 15-49 years old population in Bagalkot District from Ramesh, 2004) this meant that the sexual life expectancy for males in each Taluka was 25.1, 26.6 and 27.9 years respectively. The death rate (*nat_deathM*) was the inverse of the sexual life expectancy for males in each Taluka.

Females: The median age that females began sexual activity was 16.7, 15.1 and 15.4 years in each respective Taluka (ICHAP, 2004a). Assuming that sexual life expectancy ended at 49 years,

the sexual life expectancy for females in each Taluka was 32.3, 33.9 and 33.6 years respectively, and the death rate (*nat_deathF*) was again the inverse of this value.

- Renewal rates into sexually active population ("birth rates")

The estimated birth rate (*preAIDS_growth*) into Bagalkot District was the exponential growth rate at the start of the epidemic in 1976; for the model we assumed this was 1.9% in Karnataka (the population growth rate in Karnataka in 1981) (Census India, 2001), plus the death rates for each Taluka (see above). The birth rates into each gender/risk group (*inbirth_ki*) is the birth rate multiplied by the proportion of people in that group (*propB_ki)* and the male-to-female ratio of that group (*mal_fem_ratio* and *fem_mal_ratio*).

- Overall population sizes

The total (*ntot*), female (*ntot_f*) and male (*ntot_m*) population sizes (for 15-49 years) were estimated in a census conducted by the BDP (Ramesh, 2004). Using a population pyramid for India from the US Census Bureau (US Census Bureau, 2004) for 2004 that describes the estimated proportion of people by age group and the sexual life expectancy for males and females in each Taluka (see above), we estimated the size of the sexually active population in the three Talukas in Bagalkot District in 2004 (Table A1.1 in the Appendix). We estimated the initial size of the sexually active population in 1986 by using the 2004 population sizes, the growth rates for Karnataka from 1981–1991 (1.9%) and 1991–2004 (1.6%) and by assuming exponential growth of the population from 1986 to 2004 (Table A1.1 in the Appendix).

High-risk groups

When high-risk group parameter values were varied (probabilities of choosing someone from a particular group, mean high-risk contact rates and population sizes in each group), other high-risk parameters must satisfy the following constraint at all times, in order to ensure that the total

number of partnerships that FSWs had with clients was the same as the total number of partnerships that clients had with FSWs.

Equation 4.1. Total numbers of partnerships between FSWs and MCLs

$$p_{kik'i'} * m_{ki} * n_{ki} = p_{k'i'ki} * m_{k'i'} * n_{k'i'}$$

In Equation 4.1, $p_{kik'i'}$ represents the probability that a person of sex k (male or female) and group i (in this case, high-risk) will choose a partner from opposite sex k' and risk group i'; m_{ki} represents the average number of contacts that person of sex k and group i has per year; and n_{ki} represents the population size of group ki.

- FSW size *(propB_FH)*

The numbers of FSWs enumerated in Bagalkot District in 2004 were 295 (0.5%) in Taluka A, 1993 (2.2%) in Taluka B and 1269 (2.1%) in Taluka C. These numbers were revised as FSWs continued to be enumerated, bringing the numbers of FSWs to 837 (1.6%) in Taluka A, 1751 (2.0%) in Taluka B and 2083 (3.6%) in Taluka C. We used the latter estimates, as they provided a better model fit and in the model were represented by a fraction *(propB_FH).*

B-CI, B-CII scenarios: 1.6%: (A); 2.0% (B); 3.6% (C)

Uncertainty ranges: [0.8 – 3.2]% (A); [1.0 – 4.0]% (B); [1.8 – 7.2]% (C)

- Client size in 2004 *(propB_MH)*

Data only existed for the proportion of males reporting ever visiting a FSW (lifetime MCL size) with 11.4% in Taluka A, 13.2% in Taluka B and 18.0% in Taluka C in 2004 (for the total District, this value was 12.8%) (Ramesh, 2005a). The proportion of males visiting FSWs in 2004 (*propB_MH*) was calculated using equation 4.2 and was dependent on the lifetime MCL size

(*prob_everCLI*), the sexual life expectancy of males (*nat_deathM*) and the estimated duration of MCRB (*durationMH*).

Equation 4.2. Estimating the 2004 size of the MCL population

$$r1 = \frac{\ln(1 - prob\_everCLI)}{(-(1/nat\_deathM))}$$

$$r2 = durationMH$$

$$propB\_MH = \frac{r1}{(r1 + r2)}$$

B-CI scenario (lifelong duration MCRB): 10.8% (A); 12.4% (B); 16.6% (C)

B-CII scenario (20, 20 and 22 years duration MCRB): 8.8% (A); 9.6% (B); 14.0% (C)

Uncertainty range (half-to-double the lifetime MCL size for B-CII values): [4.5 – 17.1]% (A); [4.9 – 18.7]% B); [6.9 – 26.0]% (C)

- Mean client per FSW contact rate *(M_FH)*

Data from northern Karnataka (which incorporated a small number of Bagalkot District FSWs) reported that FSWs in 2002 had an average of 10.0 male partners per week, equal to 520.0 per year (ICHAP, 2002a). Of these, FSWs reported that 5.8 per week (301.6 per year) were with one-time clients, 3.8 per week (3.8 – 197.6 per year) were with regular clients and 0.4 per week (0.4 – 20.8 per year) were with husbands/boyfriends/lovers. In order to not underestimate the mean client-FSW contact rate, we therefore used the high-range value (510 – 520) and assumed this held true for 2004 as well as 2002. We used this parameter, as well as the FSW/MCL sizes to estimate the mean FSW per client contact rate per year (FSW-client contact rate), a constant value. In the models, the mean number of MCL partners per FSW per year (client-FSW contact rate) varied over time and was dependent on the latter three parameters. We varied the initial sizes of the FSW/MCL populations so that the 2004 population sizes and the 2004 mean client-FSW contact rate (~510-520) per year fit to the B-CII parameter values.

B-CI, B-CII scenario: 520 MCL partners per FSW per year

Uncertainty range: [260 – 1040] MCL partners per FSW per year

- Mean FSW per client contact rate *(MB_MH)*

Empirical data for this parameter was not available in Bagalkot District (or India), but we estimated this parameter indirectly, using the 2004 sizes of the MCL (*propB_MH*malsz2004*) and FSW (*propB_FH*femsz2004*) populations, and Equation 4.1 and 4.3. The mean FSW-client contact rate was also dependent on the duration of MCRB and thus different for B-CI and B-CII.

Equation 4.3. Estimating the mean number of FSW partners per MCL per year

$$MB\_MH = \frac{propB\_FH * femsz2004 * M\_FH}{propB\_MH * malsz2004}$$

B-CI scenario: 100 (A); 110 (B); 139 (C) FSW partners per MCL

B-CII scenario: 122 (A); 141 (B); 169 (C) FSW partners per MCL

Uncertainty range: [60-220] (A); [70-280] (B); [85-340] (C)

- High-risk sexual mixing probabilities

For purely assortative (B-CI) and non-purely assortative (B-CII) sexual mixing patterns, high-risk females (FSWs) had only have short-term one-time contacts with high-risk males (MCLs), and so the mixing probability (P_FHMH) was 100%. For B-CI, the probability that high-risk males mixed with high-risk females was also 100%; for B-CII, high-risk males had short-term one-time contacts with high-risk females and long-term relationships with low-risk females, thus to balance Equation 4.1, the probability that high-risk males mixed with high-risk females (P_MHFH) was equal to the proportion of total partners per year that high-risk males had with FSWs per year. Because the latter parameter value was very high (~510-520 per year), P_MHFH was approximately equal to 99.3% in each Taluka. This was a strongly assortative mixing pattern. We did not look at the impact of mixing assumptions in this model.

- Duration of female sex work (FSW) *(durationFH)*

There was little data to describe the duration of FSW in Bagalkot District. The mean age of FSWs was 27 years in northern Karnataka (ICHAP, 2002a), and their average age of onset of sexual activity was 17.8 years (ICHAP, 2002a), thus a rough estimate for the average duration of FSW in northern Karnataka was the latter parameter subtracted from the former: 9.2 years (round to 9 years). In Mysore District (southern Karnataka), the estimated duration of FSW was 4.5 years (Ramesh, 2005c.). In Kolkata, the mean duration of FSW was 7.5 years (Pal, et al., 2003). These estimates can help us interpret the range of values explored in Bagalkot District.

B-CI scenario: lifelong

B-CII scenario: 9 years

Uncertainty range:[4.5- 18] years

- Duration of male client risk behaviour (MCRB) *(durationMH)*

There was little data to describe the duration of MCRB in Bagalkot District. The average age of MCLs was 32.6 years in northern Karnataka, as estimated by FSWs in 2002 (we assumed the same in 2004) (ICHAP, 2002a), and the average age of onset of sexual activity in Bagalkot District was 22.5 years (ICHAP, 2004a), so a rough estimate for the mean duration of FSW in northern Karnataka was the latter parameter subtracted from the former: 10.1 years (round to 10).

B-CI scenario: lifelong

B-CII scenario: 10 years

Uncertainty range: [5 – 20] years

When we varied duration of MCRB, we needed to take into account that the size of the 2004 client population would also vary as a result. Additionally, if the client size changed, to balance out numbers of high-risk partnerships (*Equation 4.1*), one of the following parameters must also

vary: 1) the average number of FSW partners per client per year; 2) the average number of client partners per FSW per year; or 3) the size of the FSW population. Since we had empirical data for 2) and 3), we varied 1). Since the number of clients decreased with decreased duration of MCRB, the value for 1) increased, keeping the initial number of high-risk partnerships constant.

Low-risk groups

- Low-risk groups size

The proportion of individuals in low-risk groups by gender (*propB_ML* and *propB_FL*) was estimated by subtracting the proportion of individuals in high-risk groups from 100%. There were thus 98.4%, 98.0% and 96.6% low-risk females in Talukas A, B and C respectively in 2004 and 89.6%, 86.8% and 82% lifetime low-risk males in each Taluka respectively.

- Low-risk partner change rates (*M_ML* and *M_FL*)

There were little data available on the mean low-risk partner change rate (lifetime or annual) in Bagalkot District. In the HIV prevalence report conducted in Bagalkot District in 2004 (ICHAP, 2004a), most married men (MM) and women (MW) reported only being married once (96.6% in all males and 99.2% respectively). In Talukas A, B and C respectively, 1.5%, 2.1% and 2.0% of females and 2.1%, 2.6% and 1.4% of males reported having more than one lifetime sexual partner (ICHAP, 2004a). However, polling booth data suggests that there may be higher levels of low-risk sexual behaviour than was reported in the general population survey. In Talukas A, B and C, 26.0%, 28.0% and 42.3% of MM reported having pre- or extra-marital sex (Ramesh, 2005a). In Bagalkot District, by age group, in 2004, 26.5% of MM ages 15-29 and 31.7% of MM ages 30-49 reported this behaviour; in 2005, 36.9% of MM ages 15-29 and 38.1% of MM ages 30-49. In 2004, 10.9% of MW ages 15-29 and 8.0% of MW ages 30-49 reported this behaviour; in 2005, 11.3% of MW ages 15-29 and 17.2% of MW ages 30-49 (Ramesh, 2005a). 17.4% of all

unmarried men in Bagalkot District reported having pre-marital sex in 2004 and 14.5% in 2005 (Ramesh, 2005a). Additionally, 9.8% of all unmarried women in Bagalkot District reported this in 2004 and 12.4% in 2005 (Ramesh, 2005a). Finally, 24.8%, 26.4% and 40.0% of all rural men in Talukas A, B and C reported having ever been with a non-FSW, non-marital partner and 9.4% of women total reported having pre- or extra-marital sex (unknown how many FSWs in sample) (Ramesh, 2005a).

These data do not tell us the mean number of low-risk partners per male (*M_ML*) or per low-risk female per year (*M_FL*) in Bagalkot District, but they do suggest that males and females may have an average of more than 1-2 per sexually active lifetime (SAL). We initially assumed one low-risk partner per male per SAL (~0.04 per year in each Taluka), but to fit the model to observed data in Taluka B, we increased this to 10 per SAL (~0.4 per year in each Taluka) (see Chapter Five: Model Fitting). The mean number of low-risk female partners per male per year was a constant value and the mean number of male partners per low-risk female per year varied over time, dependent on the former parameter and population sizes.

B-CI scenario: one low-risk female partner per male per SAL

B-CII scenario: 10 low-risk female partners per male per SAL

Uncertainty range: [5 – 20] low-risk female partners per male per SAL

- Low-risk sexual mixing probabilities

Low-risk males had only long-term, exclusive relationships with low-risk females (*P_MLFL*=100% mixing probability) in both B-CI and B-CII. In B-CI, low-risk females only have long-term, exclusive relationships with low-risk males (*P_FLML*=100%). In B-CII, low-risk females had long-term, exclusive relationships with either low-risk males or high-risk males. To balance the total number of low-risk partnerships that low-risk females had with low-risk

males and the total number of low-risk partnerships that low-risk males had with low-risk females (Equation 4.1), as well as the total number of low-risk partnerships that low-risk females had with high-risk males and the total number of low-risk partnerships that high-risk males had with low-risk females (Equation 4.1), we defined the probability that low-risk females mixed with low-risk males as the proportion of total partners per low-risk female that were with low-risk males (*P_FLML*) and the probability that low-risk females mixed with high-risk males as the proportion of total partners per low-risk female that were with high-risk males (*P_FLMH*). For each Taluka, *P_FLMH* was approximately equal to the proportion of high-risk males in the population (~8% – 14% in 2004) over time and fit with observed data from polling booths in 2004 (Ramesh, 2005a), which stated that in Bagalkot District, 12.5% of MW ages 20-29 and 11.3% of MW ages 30-49 had husbands who visit FSWs. Thus *P_FLML* was approximately (~92% - 86% in 2004) over time. The probability that high-risk males mixed with low-risk females (*P_MHFL*) was equal to the proportion of total high-risk male partners per year that were with low-risk females and was very low in each Taluka (~0.3%) since we assumed a high mean FSW-client contact rate (~520). This resulted in strongly assortative mixing between low-risk females and high-risk males. We did not look at the impact of mixing assumptions.

Migration parameters

- Size of the migrating client population (*propmigMH*)

Seasonal migration is widespread in Northern Karnataka, where Bagalkot District is located (Halli et al., 2006); however, there was a lack of data on the extent of migration. A pilot study on migration of clients (Halli et al, 2006) was recently conducted in Bijapur District (neighbouring District just north of Bagalkot). In this study, migration was shown to vary within the district, with 30% to 50% of males migrating in 2005 (did not say if lifetime or in last year); 41% of

married males and 47% of unmarried males. These values may be higher in Bijapur District compared with Bagalkot District because of certain tribes of people who traditionally migrate seasonally in Bijapur District; it is unknown if the same proportion of Bagalkot's population has similar characteristics. In Bagalkot District, 2.4% of men reported travelling for longer than a month in the past year (ICHAP, 2004a). This may be an underestimate if the migrating males in the study were away from Bagalkot District at the time the study took place. In addition, 20.1% of males in Taluka A, 33.6% in Taluka B and 56.1% in Taluka C reported travelling for work in the past year (ICHAP, 2004a). We chose a wide range to explore to reduce model uncertainty.

B-CM: 30%

Uncertainty range: [15 - 60]%

- Size of the migrating FSW population (*propmigFH*)

There was very little data available on the size of the migrating FSW population in Bagalkot District. Between 13% and 18% of FSWs in Northern Karnataka migrate outside of their home Districts (ICHAP, 2002a).

B-CM: 15%

Uncertainty range: [7.5 - 30]%

- Duration of migration

In Bagalkot District, 2.4% of men reported travelling for longer than one month in the past year (no data on mean duration) (ICHAP, 2004a). Frequently, males travelled to construction sites or brick kilns for 3-4 months in summer, or to sugarcane fields for 6 months (ICHAP, 2003). In Bijapur District, males reported migrating for road building and construction for 7 – 8 months (Halli et al, 2006). FSWs frequently travelled during December to February, when there are festivals in different towns or villages. FSWs may also follow males to migration destinations.

B-CM: 4 months

Uncertainty range: [2 – 8] months

- Mean high-risk contact rate in the POM relative to the place of origin (*factorMHO, factorFHO*)

Migrating individuals may engage in higher-risk behaviour when they are in the place of migration (Halli et al, 2006). However, there was a lack of quantitative data on extent of higher-risk behaviour in place of migration for FSWs or MCLs. To assess the impact of this parameter, we assumed a B-CM value of 1.0-fold (the same mean high-risk contact rate in the POM as in the place of origin); greater than 1.0-fold means that there are x-fold greater contacts in the POM compared with the place of origin.

B-CM: 1.0-fold

Uncertainty range: [0.5-fold – 2.0-fold]

- HIV prevalence of FSWs in the place of migration in 2004 (*prev_FH2SA* for constant HIV prevalence, Figure 4.6; *prevFHout* for logistic HIV prevalence, Figure 4.7)

Males in Bagalkot District may migrate outside the state to larger urban centres such as Mumbai, Pune or Sholapur, within the state to other Districts or within the District to other Talukas (ICHAP, 2004a). In India, the FSW HIV prevalence has a wide range, and is estimated at between 2.6% and 60%. In Maharashtra (neighbouring state), NACO estimates put FSW HIV prevalence at 44.7% (NACO, 2004). In Karnataka, this value is estimated at 25.0% and in neighbouring Districts to Bagalkot District, was between 15% and 35% (Ramesh et al, 2006). Figures 4.6 and 4.7 show the HIV prevalence scenarios for FSWs in the POM when we assume constant or gradually increasing (logistic) HIV prevalence.

B-CM (2004 model HIV prevalence in FSWs in the POM): 35%

Uncertainty range: [17% - 70%]

Figure 4.6. Constant HIV prevalence scenario for FSWs in the POM and range explored

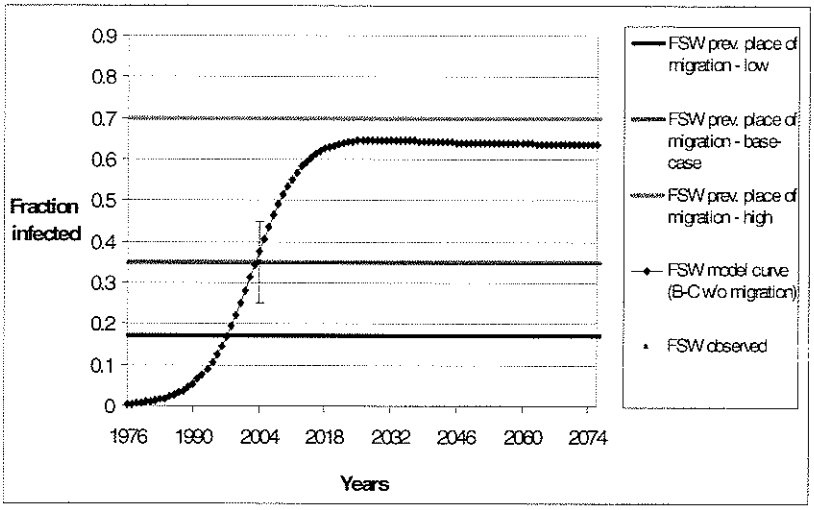
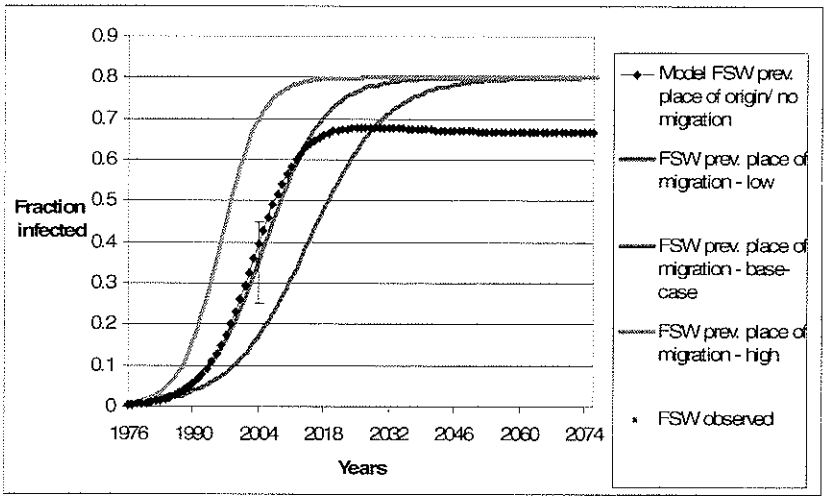


Figure 4.7. Gradually increasing (logistic) HIV prevalence scenario for FSWs in the POM and range explored (for logistic HIV prevalence scenario range, equilibrium value is always 80%)



- HIV prevalence of MCLs in the place of migration (*prev_MH2SA* for constant HIV prevalence; *prevMHout* for logistic HIV prevalence) *(for logistic HIV prevalence, the equilibrium value is always 40%)*

Little data exists for HIV prevalence for MCLs in India. FSWs in Bagalkot District may migrate outside the state to larger urban centres such as Mumbai, Pune or Sholapur or within the state to

other Districts (ICHAP, 2002a), and the HIV prevalence of clients in many of these places is just a guess. The two HIV prevalence scenarios for clients in the POM and the range explored (similar to Figures 4.6 and 4.7) are not shown.

B-CM: 17%

Uncertainty range: [8% – 34%]

Biological parameters

- Disease progression

The course of HIV infection is well-represented by four stages (Ghani & Boily, 2003). Both CD4 counts (a measure of immune system function) and changes in viral load (a measure of the quantity of virus in plasma) have been used as markers for progression of HIV infection, with several recent studies indicating that viral load is a better predictor than CD4 counts (Mellors et al, 2007; Lau et al., 2007). HIV infection progression is commonly represented by four main stages of infection in modelling studies (Baggaley et al., 2005; Boily, Lowndes & Alary, 2002) and we included these four in our model of HIV infection: 1) a very short, acute seroconversion illness, which occurs immediately after initial infection and has the highest probability of transmission; 2) a latent or asymptomatic stage, which generally lasts the longest period of time and has the lowest probability of transmission; 3) a symptomatic stage as the disease advances during which the probability of transmission and duration of time lasts between the values of the first two stages and 4) a full-blown AIDS stage, or advanced HIV infection, during which we assumed people are too sick to transmit infection (Wawer et al, 2005; Longini et al, 1989).

For all stages of infection, we used combined male-to-female and female-to-male per-contact transmission probabilities that were estimated in a Ugandan study for two stages of infection – primary (0.0081) and incubation stages (0.0016), with no known co-factors that

would increase or decrease HIV infection (Wawer et al., 2003 as cited in Baggaley et al., 2005). We broke these into separate male-to-female and female-to-male values based on the assumption that the former value is about half that of the latter (Downs & de Vincenzi, 1996) and that the incubation period is separated into two stages, the latent and symptomatic (second and third) stages of infection, with the symptomatic stage of infection having a higher probability of transmission than the latent stage (Royce et al., 1997). We varied these values slightly to fit to overall model prevalence for stage 1, stage 2 and stage 3 (male-to-female: 0.008, 0.0012, 0.002; female-to-male: 0.0035, 0.0006, 0.001). These are within range of empirical estimates (Baggaley et al., 2005; Royce et al., 1997). We calculated per-partnership transmission in low-risk sexual relationships (Equation 4.4) based on these per-contact values (clients and low-risk males with low-risk females). In Equation 4.4, $R_{kik'i'}$ represents the probability of transmission of HIV from a group of sex "k" and risk level "i" to an individual from group "k'" to "i'" and N represents the number of sex acts in a low-risk partnership, which we estimated to be four per month.

Equation 4.4. Estimating per-partnership transmission probabilities

$$1 - (1 - R_{kik'i'})^N$$

Since the first-stage transmission probabilities were estimated for a five-month seroconversion stage of infection, we also assumed that our first stage of infection lasted 5 months. Some studies have indicated that the duration of AIDS incubation (from infection to development of clinical AIDS) is shorter in poorer countries such as Africa, but other studies indicate that this is not the case (Morgan & Whitworth, 2001). In a Ugandan setting, researchers found that there was only 35% mortality due to AIDS 8 years after patients had seroconverted, and the median estimated survival (from detection of the infection to death) in the cohort is 10

years (Morgan et al., 2000). This is similar to estimates in industrialized countries before antiretroviral treatment, which varied from 8.3 to 12.1 years (Hendricks et al., 1998; Collaborative Group on AIDS Incubation and HIV Survival including the CASCADE EU Concerted Action, 2000; Koblin et al., 1999, as cited in Morgan & Whitworth, 2001). The incubation period for AIDS in this study (comprising the latent, symptomatic and full-blown AIDS stages) was therefore estimated to be about 10 years, within range of these empirical values. We assumed that people remained in the latent stage of the infection for 8 years and the symptomatic stage for 1 year, based on estimates from other modelling studies (Baggaley et al., 2006; Nagelkerke et al., 2001; Boily, Lowndes & Alary, 2002) and by fitting to the model within the constraints of the empirical estimates for incubation period. Survival with AIDS (the final stage of infection) was estimated to be 9 months for our model, near observed estimates in rural Uganda, which were 9.3 months (95% CIs = 4.3–17.3 months) (Morgan et al., 2000) and Thailand (Kitayaporn et al., 1996) and other modelling studies (Baggaley et al., 2006; Nagelkerke et al., 2001; Boily, Lowndes & Alary, 2002).

Rates of movement from one stage to the next (stage 1 to stage 2: *p_toY2*; stage 2 to stage 3: *p_toY3;* stage 3 to AIDS: *p_toAIDS*) were the inverse of these values. In the model, infectivity from each transmission stage is *BYx_kik'i'*, where "x" represented the stage of infection, "k" was one sex and "i" was a risk group, and "k'" and "i'" were other sex and risk groups). When doing analysis, we varied the high-risk and low-risk transmission probabilities together (low, middle, high and "B-C") (see Table A1.1 in Appendix 1 for parameter value estimates).

CHAPTER FIVE: MODEL FITTING

5.1. Base-case scenario I (B-CI)

Initially, the simple assumption was made that FSWs and MCLs (and low-risk females and low-risk males) engaged in high-risk behaviour for their entire sexually active lives (lifelong duration of SW and MCRB=base-case scenario I [B-CI]) for one of two sexual mixing pattern assumptions – purely assortative and strongly assortative. Under the assumption of lifelong duration of high-risk behaviour, a realistic 2004 HIV prevalence could not be reproduced in low-risk groups in any Taluka, due to the low reported number of partners in the general population. This happened for both purely assortative mixing, where there were no partnerships between low-risk females and clients, and strongly assortative mixing, where the low numbers of reported low-risk partners resulted in low mixing probability between low-risk females and MCLs (~12%). HIV prevalence was very low in low-risk groups in all Talukas, even at equilibrium, and was lowest in Taluka A in 2004 (low-risk male: 0%; low-risk female: 0.04%), peak (low-risk male: 0%; low-risk female: 0.10%) and equilibrium (low-risk male: 0%; low-risk female: 0.09%). In the low-risk populations, an epidemic did not initially establish, indicating that $R_0<1$ (the basic reproductive number, see Chapter Three), which is an unrealistic scenario.

With lifelong duration of SW and MCRB, the epidemics become concentrated in the high-risk groups and so despite low-risk HIV prevalence being close to 0% in each Taluka, high FSW and MCL epidemics allowed overall model HIV prevalence (which included both low- and high-risk group epidemics) in 2004 to reach within the 95% CIs for the observed values in Talukas A and C, though not Taluka B. Because Taluka A's overall, female and male observed 2004 HIV prevalence was the lowest of the three Talukas, a lower high-risk HIV prevalence was

required to bring the former populations' model HIV prevalence in 2004 to within observed values. Figures 5.1 and 5.2 show high-risk model HIV prevalence for Talukas A and C: even with similarly high high-risk model HIV prevalence in Taluka B (Figure A2.1 in Appendix 2), overall model HIV prevalence did not reach within range for overall observed HIV prevalence.

Figure 5.1. B-CI Scenario – high-risk male and female prevalence in Taluka A

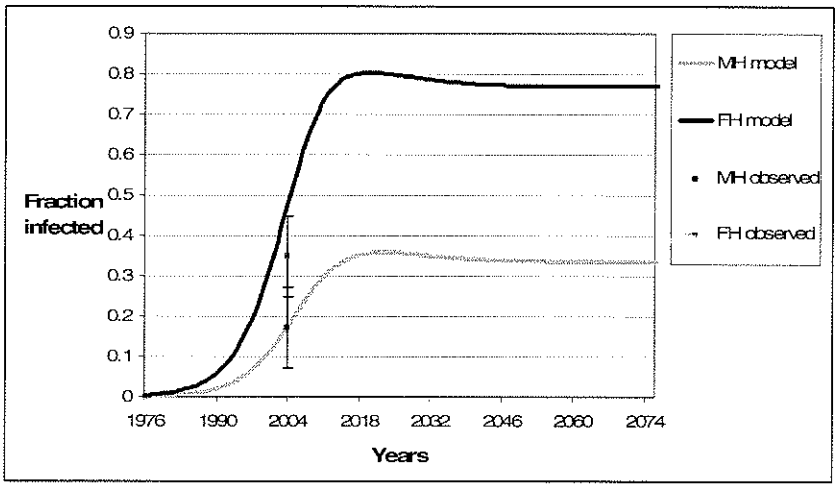
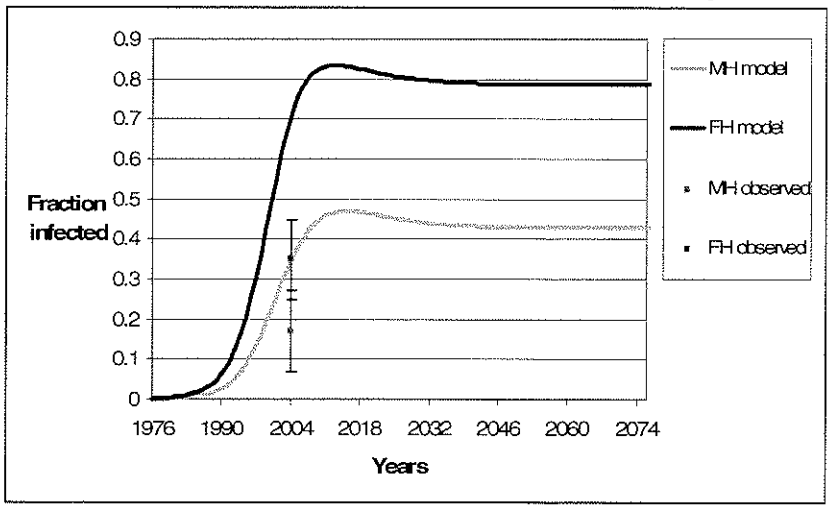


Figure 5.2. B-CI Scenario – high-risk male and female prevalence in Taluka C



5.2. Base-case scenario II (B-CII)

With less than lifelong duration, high-risk people "moved" into low-risk groups (i.e. cease high-risk behaviour) after a certain average duration of time, increasing the epidemic in low-risk groups by providing constant seeding of infections and a better model fit to observed 2004 HIV

prevalence in all Talukas. Since data on the duration of SW and MCRB was limited, we estimated these values as the difference between the mean age of FSWs and MCLs and the estimated mean age of becoming sexually active for FSWs and all males (9 years for FSWs; 10 years for MCLs). Overall model 2004 HIV prevalence was not very sensitive to duration of FSW (see Chapter Seven: Uncertainty Analysis). However, since the mean FSW-client contact rate was inversely dependent on the duration of MCRB, 10 years duration caused very high contact rates (234 for Taluka A, 269 for Taluka B, 344 for Taluka C) and high high-risk model HIV prevalence (increasing low-risk epidemics marginally). Thus, we increased the duration of MCRB to 20 years for Taluka A and B and 22 years for Taluka C to better fit to the observed 2004 HIV prevalence in each population, with a more realistic and lower mean FSW-MCL contact rate (122 for Taluka A, 141 for Taluka B, 169 for Taluka C).

With these values for the duration of FSW and MCRB, Taluka A and C fit better to the observed overall 2004 model HIV prevalence (Taluka A model: 1.2%; Taluka C model: 4.4%) but was still low for Taluka B (Taluka B model: 1.8%). Since 2004 high-risk male and female model HIV prevalence was already high in Taluka B (~22% and 45%), we increased the mean low-risk partner change rate until Taluka B's 2004 model HIV prevalence was 2.2%, within range of observed values (~10 low-risk partners per sexually active lifetime per low-risk male and low-risk female). This negligibly affected high-risk model HIV prevalence, but increased the epidemic in low-risk groups to bring overall (male and female) model HIV prevalence to within observed values (Table 5.1). To keep this consistent across Talukas, we increased the mean number of low-risk partners to the same values in Taluka A and Taluka C, which remained within range of observed values for the overall, male and female populations (Table 5.1). Our final 2004 model HIV prevalence values for Talukas A, B and C are shown in Table 5.1 and the

epidemic curves for overall and high-risk groups in Taluka A, the best-fitting Taluka, are shown in Appendix 2 (Figure A2.2 and A2.3). Some important concerns regarding this final model fit are explored in Chapter Eight: Discussion, Section 8.4: Study Limitations.

Table 5.1: Final B-CII model fit for Taluka A, B and C

Taluka	Population	2004 observed	2004 model	Peak model	Equilibrium model
A	Overall	1.2% [0.6 – 1.8]%	1.5%	2.6% (2021)	2.3% (2074)
	Male	1.1% [0.3 – 1.9]%	1.8%	3.1% (2021)	2.8% (2079)
	Female	1.3% [0.4 – 2.2]%	1.2%	2.1% (2020)	1.9% (2077)
	High-risk male	17% [7 – 27]%	16.4%	31.7% (2027)	30.9% (2091)
	High-risk female	35% [25 – 45]%	37.5%	64.7% (2029)	63.7% (2092)
B	Overall	2.9% [2.2 – 3.7]	2.2%	3.2% (2016)	2.7% (2065)
	Male	3.0% [2.0 – 4.0]	2.6%	3.9% (2016)	3.3% (2064)
	Female	2.8% [1.8 – 3.8]	1.8%	2.7% (2016)	2.3% (2072)
	High-risk male	17% [7 – 27]%	22.3%	36.6% (2062)	35.2% (2082)
	High-risk female	35% [25 – 45]%	46.1%	67.8% (2062)	66.4% (2081)
C	Overall	4.9% [3.6 – 6.6]	5.2%	5.9% (2010)	4.7% (2063)
	Male	6.4% [4.2 – 8.6]	6.0%	6.7% (2010)	5.4% (2071)
	Female	3.2% [1.5 – 4.9]	4.7%	5.2% (2010)	4.2% (2065)
	High-risk male	17% [7 – 27]%	35.8%	43.2% (2015)	40.9% (2069)
	High-risk female	35% [25 – 45]%	61.7%	69.9% (2015)	67.2% (2069)

Re-fit model due to model error

Initially, we assumed the mean duration of infection for each stage was six months, 6.25 years, 2 years and 1 year, from literature and previous modelling studies. Late in the analysis, it was discovered that values for Stages 2 and 3 had accidentally been switched. We re-fit the model using the values described in Chapter Four (and Table A1.1 in Appendix 1), with slightly increased first-stage high-risk transmission probabilities (M-F: 0.0083 from 0.0057; F-M: 0.0041 from 0.0031) to fit the model to observed 2004 prevalence in the overall, male, female and high-risk populations. This model fit was only marginally different from the initial B-CII model fit (<0.2% difference in 2004, peak and equilibrium) and thus results from the following analysis remain valid, though actual values for outcomes such as prevalence were slightly different.

CHAPTER SIX: RESULTS

6.1. Empirical data stratified by Taluka

Univariate sensitivity analysis was conducted on main characteristics relating to sex work known to be different in the three Talukas (FSW size, client size, mean FSW-client contact rate) as well as overall population size. Each parameter value was first adjusted in turn in one Taluka to the other Taluka's value (to identify characteristics that explain differences in observed 2004 HIV prevalence). Each parameter value was then varied the same proportionately from the estimated B-CII value (from half-to-double the B-CII value) and ranked based on its impact on 2004, peak and equilibrium model HIV prevalence. Six different were developed (Table 6.1) to satisfy the constraint of keeping the total number of partnerships that FSWs have with clients the same as the total number of partnerships clients have with FSWs (equation 4.1).

Table 6.1. Scenarios (partnerships vary versus constant) when varying FSW/MCL size and number of high-risk partners. Red highlights the parameters of interest.

Parameter Scenario	FSW size	MCL partners per FSW	MCL size	FSW partners per MCL	Number high-risk partnerships
PV1	Constant	[redacted]	Constant	[redacted]	Varies
PV2	[redacted]	Constant	Constant	[redacted]	Varies
PV3	Constant	Varies	[redacted]	Constant	Varies
PV4	[redacted]	Constant	[redacted]	Constant	Varies
PC1	[redacted]	Varies	Constant	Constant	Constant
PC2	Constant	Constant	[redacted]	Varies	Constant

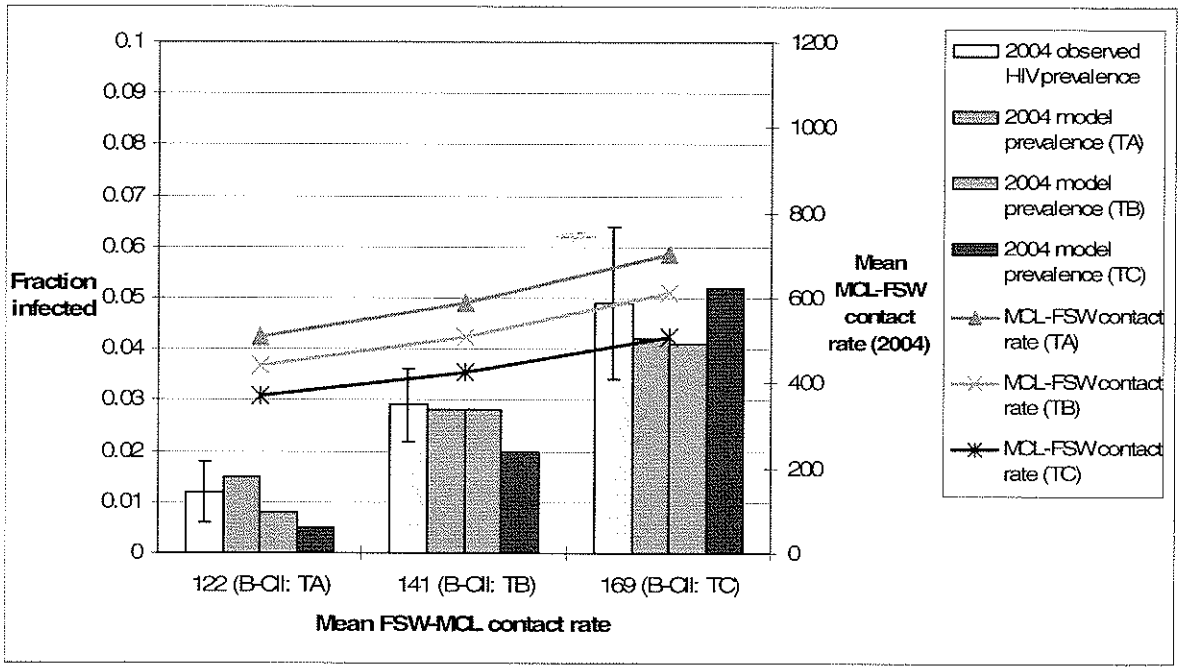
6.1.1. Understanding heterogeneity

When the total number of high-risk partnerships increased when FSW size, MCL size and the mean high-risk contact rates increased (PV1, PV2, PV3), each of these parameters alone could easily explain much of the differences in 2004 HIV prevalence observed between the Talukas.

- **Mean FSW-client contact rate (partnerships vary: PV1)**

Figure 6.1.1 shows how 2004 model HIV prevalence in the overall population varied as the mean FSW-MCL contact rate was exchanged in each Taluka for another Taluka's values.

Figure 6.1.1. 2004 overall model HIV prevalence and mean MCL-FSW contact rate per year in all three Talukas as each Taluka's mean FSW-MCL contact rate per year is exchanged for the others' B-CII values (partnerships vary: scenario PV1).



When Taluka A's mean FSW-MCL contact rate was increased to 141 (Taluka B's B-CII values), while keeping FSW and MCL sizes constant, the mean MCL-FSW contact rate per in 2004 increased from ~510 to ~590 and the overall 2004 model prevalence increased to 2.8% (B-CII: 1.5%), within range of Taluka B's overall observed HIV prevalence in 2004 (2.9% [2.2 – 3.6]%) (Figure 6.1.1); male 2004 model prevalence increased to 3.4% (B-CII: 1.8%), within the range of Taluka B's observed values (3.0% [2.0 – 4.0]%) and female prevalence increased to 2.3% (B-CII: 1.2%), within the range of Taluka B's observed female prevalence (2.8% [1.8 – 3.8]%). Since the number of high-risk partners increased, high-risk 2004 model prevalence also showed significant increases. Male client and FSW 2004 model prevalence increased to 30.7%

(B-CII: 16.3%) and 61.3% (B-CII: 37.2%) respectively, above estimated observed values for MCLs (17% [7 – 27]%) and FSWs (35% [25 – 45]%).

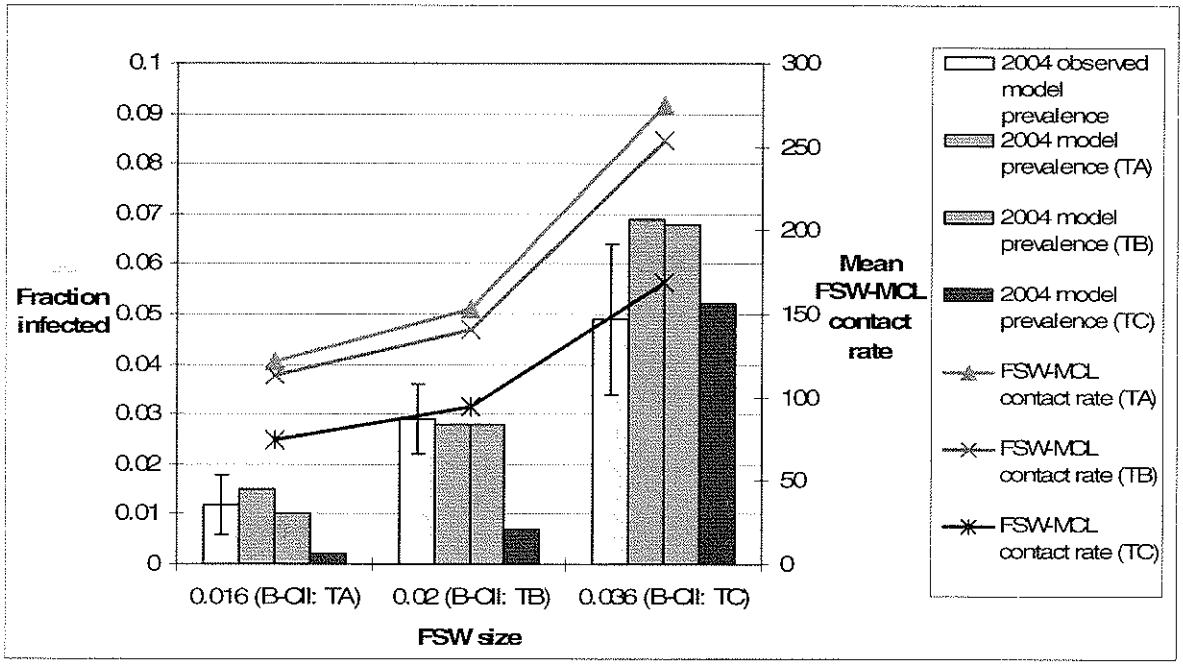
Taluka C's larger estimated mean FSW-MCL contact rate (169) may also explain its higher 2004 HIV prevalence compared with Taluka A. With Taluka C's values for this parameter, Taluka A's overall model 2004 prevalence increased to 4.2% (B-CII: 1.5%), within observed 2004 overall prevalence in Taluka C (4.9% [3.5 – 6.5]%) (Figure 6.1.1). Male and female model prevalence in 2004 in Taluka A increased within range of Taluka C's observed 2004 female prevalence at 5.1% (observed: 6.4% [4.2 – 8.6]%) and 3.5% (observed: 3.2% [1.5 – 4.9]%) respectively. High-risk male and female 2004 model prevalence again increased above the range of estimated observed values, at 43.5% and 74.8% respectively. Similarly, when Taluka B's B-CII mean FSW-MCL contact rate (141) was replaced with Taluka C's (169), the mean MCL-FSW contact rate in 2004 increased (~604 per year from B-CII=~510) and overall, male and female model 2004 prevalence increased to 4.1% (Figure 6.1.1), 5.0% and 3.4% respectively, all within range of Taluka C's observed prevalence. Again, there were large increases in high-risk model HIV prevalence in 2004, above estimated observed values.

When Taluka B's and C's mean FSW-MCL contact rates were lowered to Taluka A's, with the mean MCL-FSW contact rates in 2004 reduced accordingly (440 and 369 respectively), overall 2004 model prevalence in Talukas B and C both reduced to 0.8% and 0.5%, though only the former is within range of Taluka A's overall observed 2004 prevalence (1.2% [0.6 – 1.8]%) (Figure 6.1.1). Taluka C's overall 2004 model prevalence became slightly too low (2.0%) to be within the range of Taluka B's observed prevalence when Taluka C's mean FSW-MCL contact rate was decreased to Taluka B's (141 from 169), with a subsequent decrease in the mean MCL-FSW contact rate (~426 per year from ~510).

- **FSW size (partnerships vary: PV2)**

Figure 6.1.2 shows how 2004 model HIV prevalence in the overall population varied as 2004 FSW size was exchanged in each Taluka for another Taluka's, when total partnerships varied.

Figure 6.1.2. 2004 overall model HIV prevalence and mean FSW-MCL contact rate per year in three Talukas as each Taluka's FSW size is exchanged for the others (partnerships vary: PV2)



When Taluka A's FSW size was increased from 1.6% to 2.0% (Taluka B's B-CII values) and total high-risk partnerships varied, the mean FSW-MCL contact rate increased to 153 and the overall 2004 model prevalence increased to 2.8% (Figure 6.1.2), within range of Taluka B's overall observed HIV prevalence in 2004; male and female 2004 model prevalence increased to 3.3% (B-CII: 1.8%) and 2.4% (B-CII: 1.2%), within range of Taluka B's observed values. Since the mean FSW-MCL contact rate increased as FSW size increased, high-risk 2004 model prevalence also showed large increases – MCL and FSW 2004 model prevalence increased to 29.6% (B-CII: 16.3%) and 55.6% (B-CII: 37.2%) respectively, above estimated observed values.

When Taluka A had Taluka C's FSW size (3.6%) and the mean FSW-MCL contact rate increased accordingly (to 275), Taluka A's overall model 2004 prevalence increased to 6.9% (B-

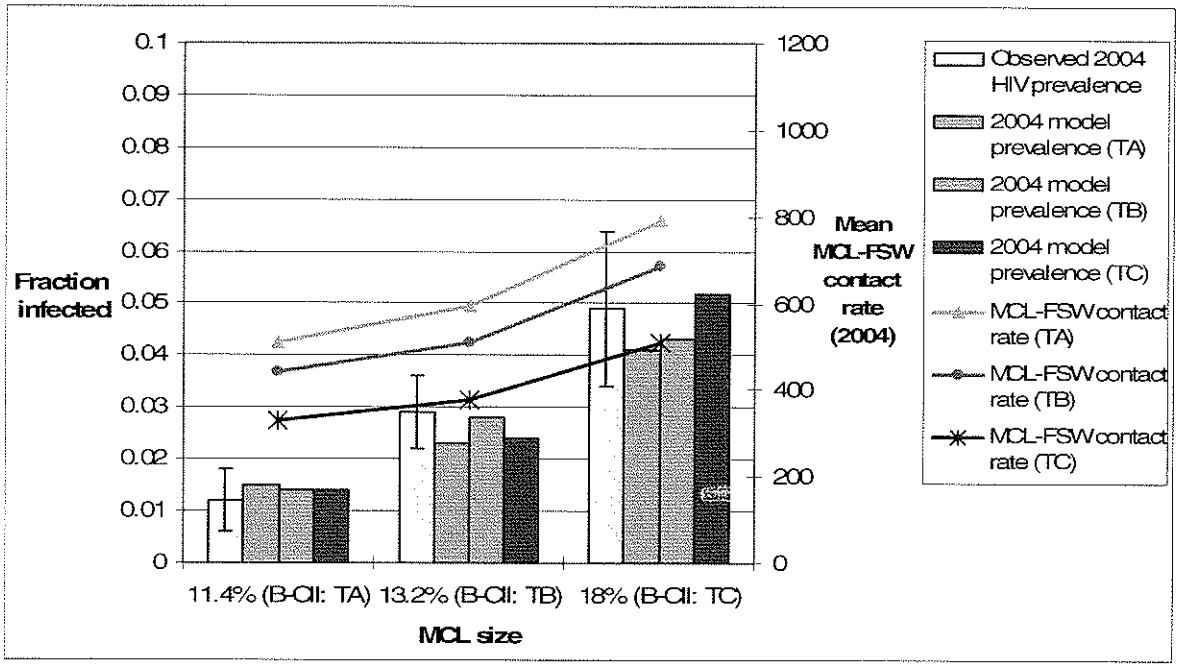
CII: 1.5%) (Figure 6.1.2), higher than observed 2004 overall prevalence in Taluka C. Female model prevalence in Taluka A also increased above range of Taluka C's observed 2004 female prevalence to 6.8%, while male model 2004 prevalence remained within range of Taluka C's observed 2004 male prevalence at 7.1%. Again, high-risk male and female 2004 model prevalence increased above range of the estimated observed values, at 57.0% and 74.1% respectively. When Taluka B's FSW size (2.0%) was replaced with C's (3.6%) (Figure 6.1.2), the mean FSW-MCL contact rate increased (254 per year from B-CII=141) and overall and female model 2004 prevalence increased above range to 6.8% and 6.4% respectively; male model 2004 prevalence remained within range at 7.3%.

FSW size may also help explain the differences in 2004 observed prevalence between Talukas B and C (with higher FSW sizes of 2.0% and 3.6% respectively) and Taluka A. When Taluka B's and C's FSW sizes were decreased to Taluka A's (1.6%), and the mean FSW-MCL contact rate reduced accordingly (113 and 75 respectively), overall 2004 model prevalence in Taluka B and C reduced to 1.0% and 0.2% respectively (Figure 6.1.2). Taluka B's 2004 model observed prevalence remained within Taluka A's overall observed 2004 prevalence (1.2% [0.6 – 1.8]%), but Taluka C's became too low. Similarly, Taluka C's overall 2004 model prevalence became too low (0.7%, B-CII: 5.2%) when Taluka C's FSW size was decreased to Taluka B's (2.0%, B-CII: 3.6%) (Figure 6.1.2), with a subsequent decrease in the mean FSW-MCL contact rate (to 94 per year; B-CII: 169).

- **MCL size (partnerships vary: PV3)**

Figure 6.1.3 shows how 2004 model HIV prevalence in the overall population varied as the lifetime (and 2004) MCL size was exchanged in each Taluka for another Taluka's, assuming total FSW-MCL partnerships were allowed to vary along with MCL size (PV3).

Figure 6.1.3. 2004 overall model HIV prevalence and mean MCL-FSW contact rate in 2004 in three Talukas as each Taluka's MCL size is exchanged for the others (partnerships vary: PV3)



When Taluka A's lifetime MCL size increased from 11.4% (2004 = 8.8%) to 13.2% (2004 = 10.3%), (Taluka B's B-CII values) and total high-risk partnerships varied, the mean MCL-FSW contact rate also increased, from ~510 to ~590 and overall 2004 model prevalence increased to 2.3%, within range of Taluka B's overall observed HIV prevalence in 2004 (Figure 6.1.3); male and female 2004 model prevalence increased to 2.9% (B-CII: 1.8%) and 1.9% (B-CII: 1.2%), within range of Taluka B's observed values. Since the mean MCL-FSW contact rate increased as MCL size increased, high-risk 2004 model prevalence showed large increases. With 13.2% MCLs, high-risk male and female 2004 model prevalence increased to 22.8% (B-CII: 16.3%) and 52.0% (B-CII: 37.2%) respectively, slightly above estimated observed values.

Taluka C's larger MCL size (18.0%) may explain its higher 2004 HIV prevalence compared with Taluka A. When Taluka A had Taluka C's MCL size and total high-risk partnerships varied, Taluka A's mean MCL-FSW contact rate increased to 789 in 2004 and overall model 2004 prevalence increased to 4.1% (B-CII: 1.5%), which is within observed 2004

overall prevalence in Taluka C (Figure 6.1.3). Male and female model prevalence in 2004 in Taluka A increased to within range of Taluka C's observed 2004 HIV prevalence at 5.4% and 3.1% respectively. High-risk male and female 2004 model prevalence also increased above the range of the estimated observed values, at 31.5% and 69.8% respectively. Similarly, when Taluka B's MCL size (13.2%) was replaced with Taluka C's (18.0%), the mean MCL-FSW contact rate increased (~685 per year from B-CII=~510) and overall, male and female model 2004 prevalence increased to 4.3% (Figure 6.1.3), 5.5% and 3.3% respectively, all within the range of Taluka C's observed 2004 HIV prevalence.

Larger MCL size may also help explain the higher in 2004 observed prevalence in Talukas B and C compared with A (18.0% and 13.2% compared with 11.4%). When Taluka B's and C's MCL sizes were decreased to A's (11.4%), the mean MCL-FSW contact rate reduced accordingly (441 and 328 respectively) and overall 2004 model prevalence in Talukas B and C both reduced to 1.4%, within range of A's overall observed 2004 prevalence (1.2% [0.6 – 1.8]%) (Figure 6.1.3). Similarly, Taluka C's overall 2004 model prevalence became within range (2.4%, B-CII: 5.2%) when Taluka C's MCL size was decreased to B's (13.2% from 18.0%), with a subsequent decrease in the mean FSW-MCL contact rate (to 378 per year from ~510).

- **FSW or MCL size (total high-risk partnerships fixed: PC1 and PC2)**

Observed differences in FSW and MCL size did not explain differences in 2004 overall HIV prevalence between the three Talukas with fixed total high-risk partnerships (Figures A2.5 and A2.6 in Appendix 2). Larger FSW or MCL size with fixed number of high-risk partnerships was associated with decreases in the mean high-risk contact rate and so 2004 model prevalence decreased with increased FSW or MCL size. This did not correspond with observed trend of larger FSW and client sizes in the Talukas with higher observed 2004 HIV prevalence.

- **Overall population size**

We show in the next section that a larger overall population size delayed the epidemic, causing peak and equilibrium model prevalence to be reached later. Since Taluka B had the largest population (larger than Taluka C by 1.5-fold) and had lower overall, male and female 2004 observed HIV prevalence than Taluka C (overall 2.9% [2.2 – 3.6]% in Taluka B; 4.9% [3.5 – 6.3]% in Taluka C), overall population sizes in Talukas B and C were switched to see how much of the difference in 2004 model HIV prevalence could be explained by total population size.

When Taluka B's overall population size was switched with Taluka C's smaller population size (smaller by 0.7-fold), Taluka B's overall, male and female model HIV prevalence in 2004 increased marginally from 2.2% to 2.4%, 2.6% to 2.9% and 1.8% to 1.9%. These are small increases, and not enough to explain the difference between 2004 model HIV prevalence between the two Talukas. When Taluka C's overall population size was increased to Taluka B's (larger by 1.5-fold), Taluka C's overall, male and female model HIV prevalence in 2004 decreased from 5.2% to 4.9%, 6.0% to 5.6% and 4.6% to 4.3%. Although a larger population size in Taluka C did decrease 2004 model HIV prevalence by delaying the epidemic slightly (peak and equilibrium reached later), these were small decreases and again not enough to explain the difference between 2004 model HIV prevalence between the two Talukas.

6.1.2. Understanding where to focus prevention efforts

Figures 6.1.4, 6.1.5 and 6.1.6 show the absolute effects (red=increase; peach=decrease) on 2004, peak and equilibrium overall model HIV prevalence respectively when the parameters with different data in each Taluka (and also the mean numbers of low-risk partners per low-risk

person for comparison) were varied from half-to-double B-CII values for Taluka A. Tables A2.1, A2.2 and A2.3 in Appendix 2 summarize these effects for each Taluka.

Figure 6.1.4. 2004 model HIV prevalence in Taluka A in the overall population when each parameter varied through its estimated range; black line=base-case overall model prevalence

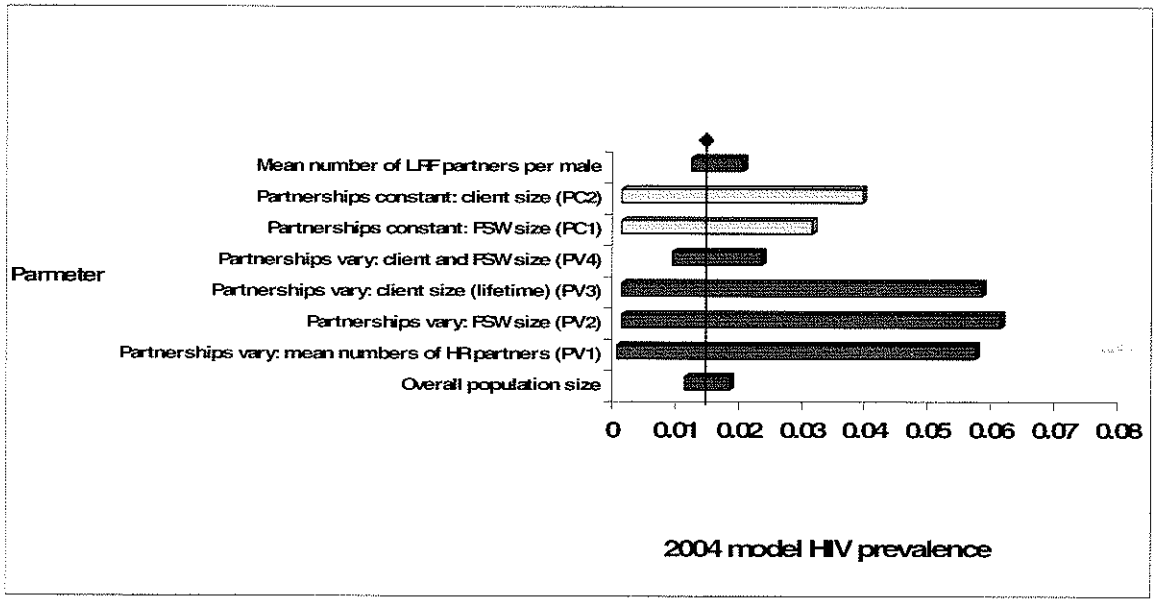


Figure 6.1.5. Peak model HIV prevalence in Taluka A in the overall population when each parameter varies through its estimated range; black line=base-case overall model prevalence

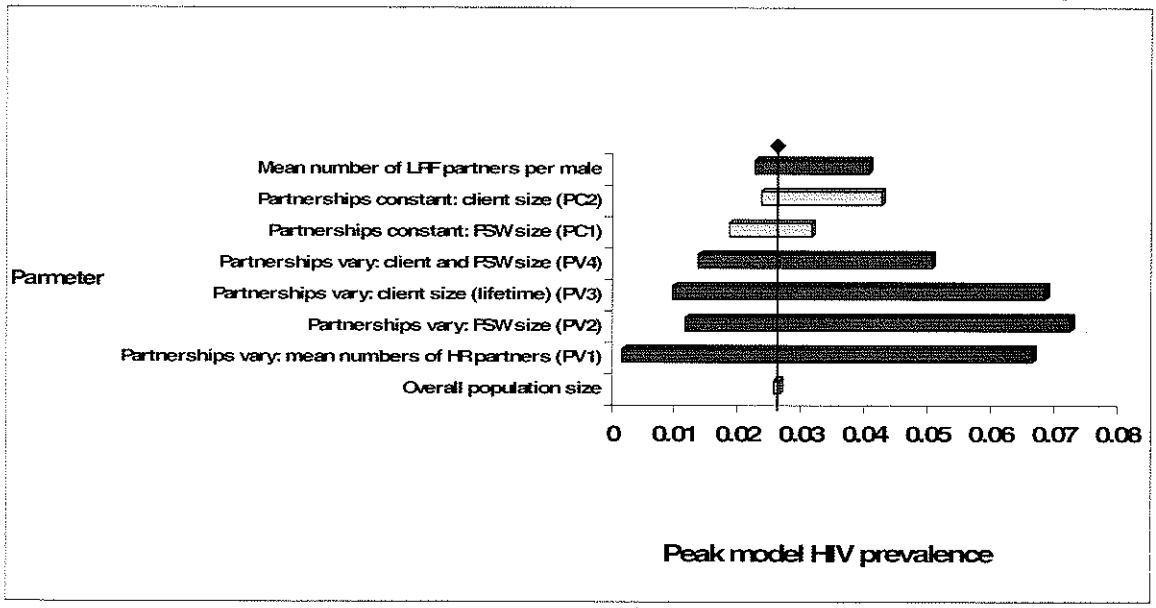
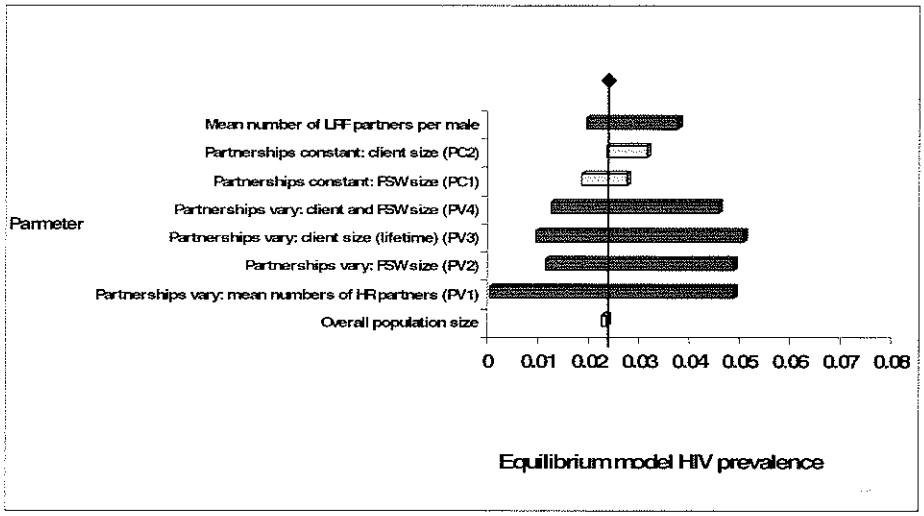


Figure 6.1.6. Equilibrium model HIV prevalence in Taluka A in the overall population when each parameter varies through its estimated range; black line=base-case overall prevalence



- ***Largest impact on increasing HIV prevalence***

The mean FSW-client contact rate, FSW size and client size had the largest impact on increasing 2004, peak and equilibrium model HIV prevalence in the overall population, if the total number of high-risk partnerships in the population also increased when these parameters increased (Figures 6.1.4 – 6.1.6). Increasing the mean FSW-client contact rate was important in increasing prevalence when high-risk partnerships varied because the mean client-FSW contact rate also increased; increasing the FSW size was important with varied high-risk partnerships because this also increased the mean FSW-client contact rate; and increasing the client size was important with varied high-risk partnerships because this also increased the mean client-FSW contact rate.

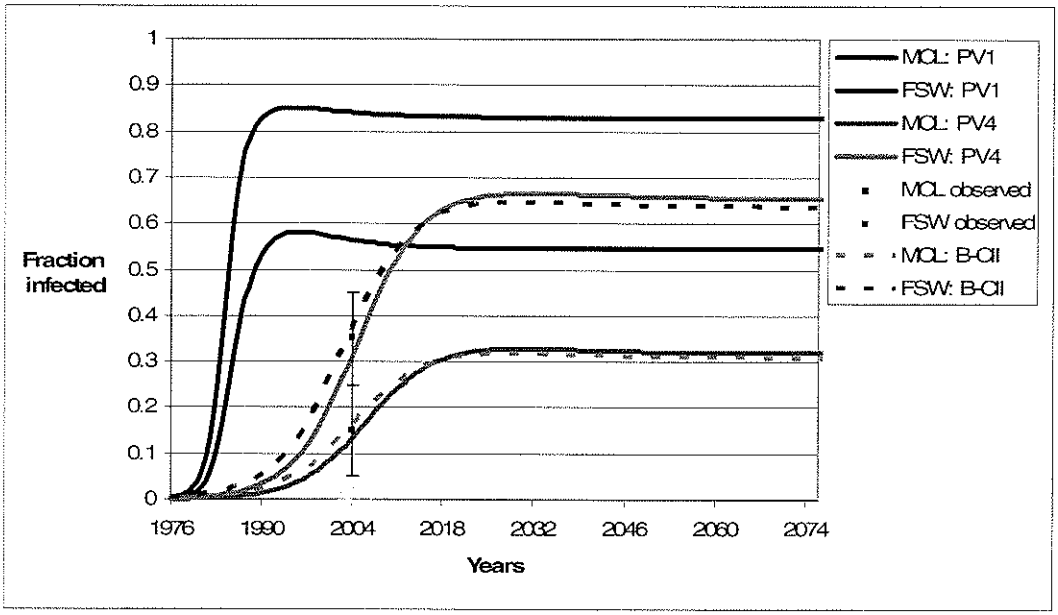
HIV prevalence was more sensitive to the mean high-risk contact rate compared with FSW or client size: when FSW and client size varied together (with fixed mean high-risk contact rate), this caused smaller increases in overall HIV prevalence than when only the mean high-risk contact rates varied and high-risk population sizes were fixed (Figure 6.1.4 – 6.1.6).

The epidemic shape was also most affected when just high-risk partners were varied and least affected when just high-risk population size varied, even though there was the same

proportionate increase in the total FSW-MCL partnerships. The epidemic peak was reached earliest when there was double the high-risk contact rate (PV1) in each Taluka (in 1993 in Taluka A, in 1992 in Taluka B and in 1990 in Taluka C). At double the FSW size and MCL size with a corresponding increase in the mean contact rate in the opposite risk group (PV2 and PV3), the epidemic peak occurred nearer to 2004 (2x FSW size: in 2001 in Taluka A, in 1999 in Taluka B and in 1996 in Taluka C; 2x MCL size: in 2008 in Taluka A, in 2004 in Taluka B, in 2002 in Taluka C). At double the high-risk population sizes (PV4), the epidemic peak occurred latest of the four scenarios where total FSW-MCL partnerships vary (in 2023 in Taluka A, in 2019 in Taluka B and in 2013 in Taluka C). All of these results can be compared with the base-case scenario (B-CII), in which overall peak prevalence was reached in 2021 in Taluka A, 2016 in Taluka B and 2010 in Taluka C (see Tables A2.1-A2.3 in Appendix 2).

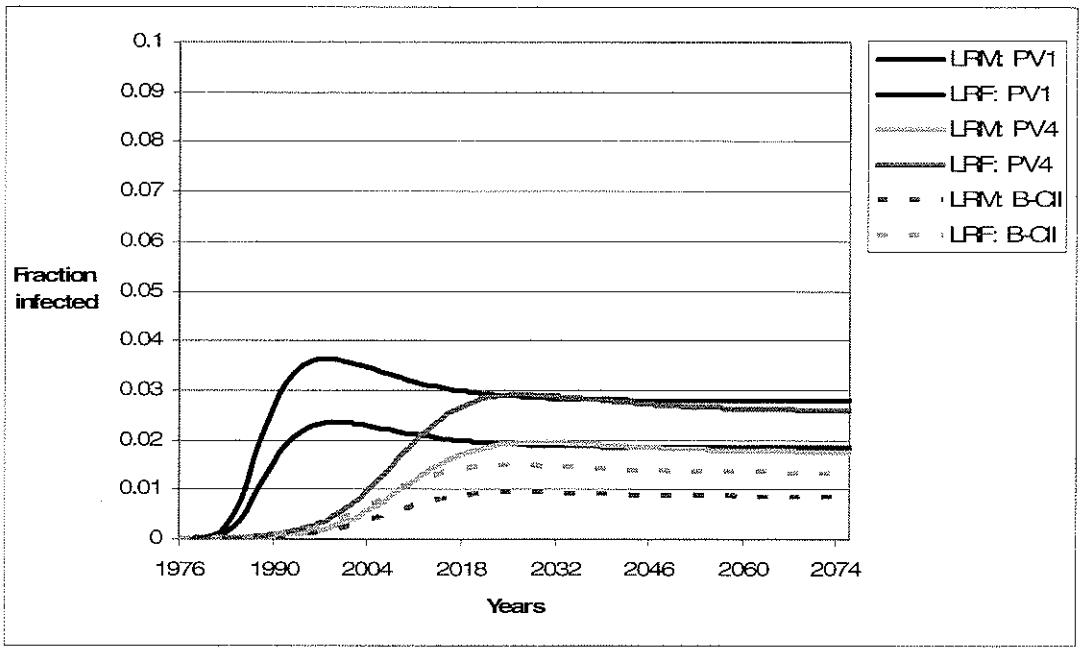
The effects on overall prevalence can be better understood by looking at the effects on high-risk and low-risk epidemics. Increasing just high-risk contact rates (PV1) caused the high-risk epidemic to become concentrated in the high-risk populations; an increase in just the high-risk population size (PV4), however, had a negligible effect on high-risk prevalence compared with the base-case scenario (B-CII). Figure 6.1.7 shows what happens to the high-risk epidemics in Taluka A when there are double the mean high-risk partner rate (PV1) with fixed high-risk population sizes and double the FSW and client sizes with fixed high-risk contact rate (PV4), compared to the base-case scenario. Similar results were found for other Talukas (not shown).

Figure 6.1.7. High-risk model HIV prevalence at double the mean number of high-risk partners (PV1) and high-risk population size (PV4), compared to the base-case scenario (B-CII)



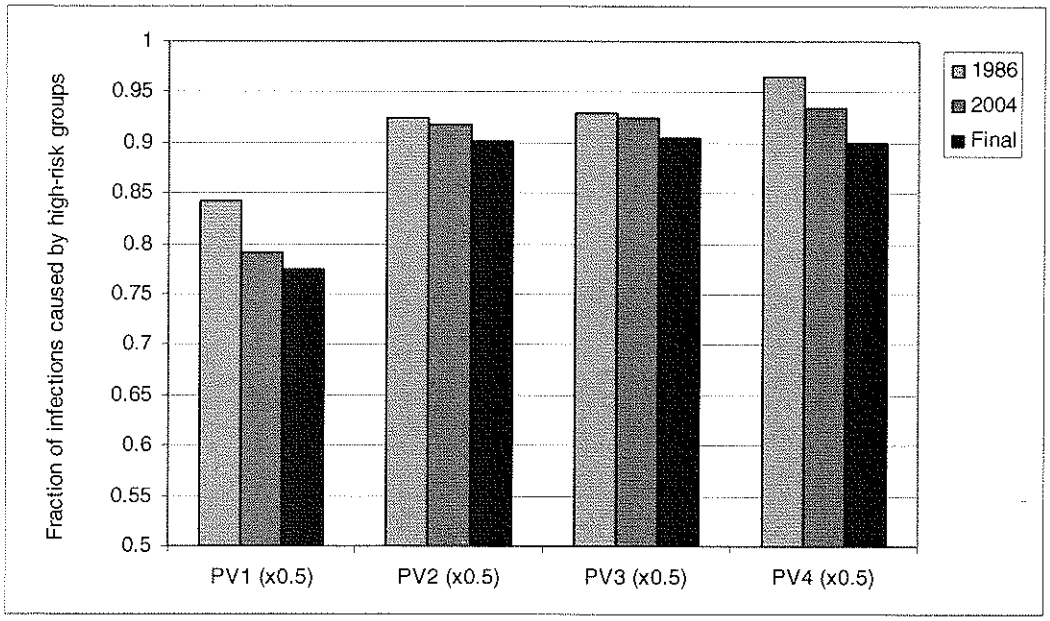
Increasing just high-risk contact rates (PV1) caused the low-risk epidemic to also peak earlier than the base-case scenario, as low-risk females were increasingly infected by the higher-prevalence clients (through the bridging effect). However, the low-risk epidemics peaked later than the high-risk epidemics because the mean duration of high-risk behaviour (before moving into low-risk groups) was 9 and 20 years for FSWs and clients. An increase in just the high-risk population size (PV4), however, increased the peak and equilibrium model prevalence in low-risk groups but had a small effect on changing the shape of the epidemic compared to the base-case scenario, B-CII (peak and equilibrium prevalence was reached at about the same time as B-CII). Almost all of the impact of increased high-risk population size was due to increases in the low-risk epidemic, but the impact of increased mean high-risk contact rate was due to both high- and low-risk epidemics. Figure 6.1.8 shows what happens to the low-risk epidemics when there are double the mean high-risk partner rate (PV1) with fixed high-risk population sizes and double the FSW and client sizes with fixed high-risk contact rate (PV4), compared to B-CII.

Figure 6.1.8. Low-risk model HIV prevalence at double the mean high-risk contact rates (PV1) and high-risk population sizes (PV4).



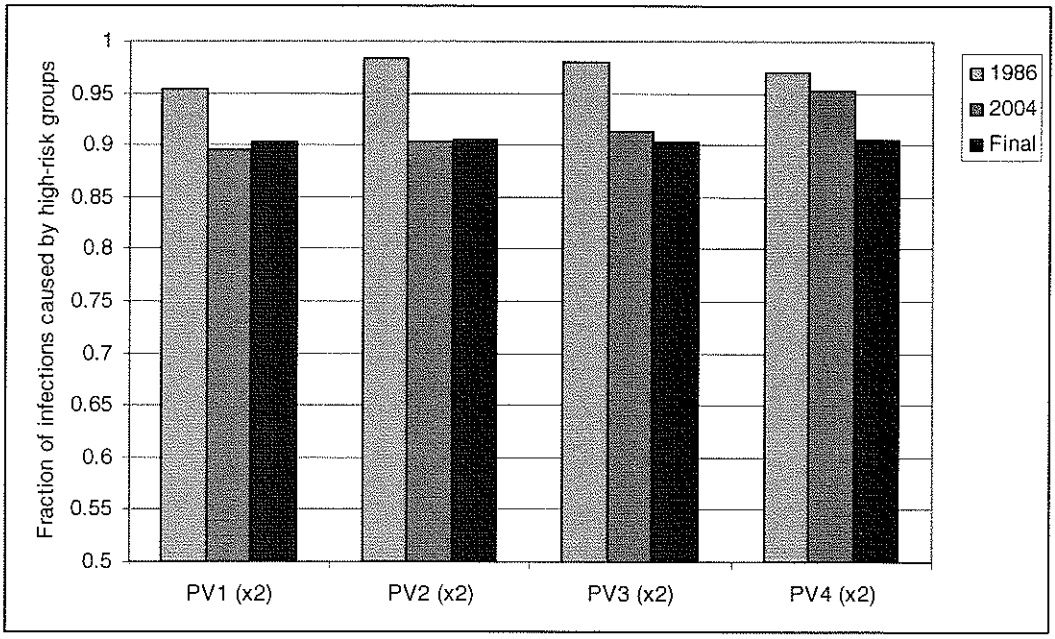
Even when FSW or client size, or mean FSW-client rate is half B-C values, a high fraction (>75%) of new yearly infections in 1986, 2004 and at equilibrium was still caused by high-risk groups, since there was very high transmission potential in high-risk groups and very low transmission within low-risk groups. Figure 6.1.9 shows the fraction of infections due to high-risk groups in Taluka A with half the high-risk contact rate (PV1: with fixed FSW/client size), half the FSW size and mean FSW-client contact rate (PV2: with fixed client size and mean client-FSW contact rate), half the client size and mean client-FSW contact rate (PV3: with fixed FSW size and mean FSW-client contact rate) and half the FSW and client size (PV4: with fixed high-risk contact rates). The fraction of infections due to high-risk groups was lowest with half the mean high-risk contact rates (PV1), with about 84%, 79% and 77% of infections due to high-risk groups in 2004, peak and equilibrium and was highest with half the high-risk population sizes (PV4), with about 97%, 93% and 90% of infections due to high-risk groups in 2004, peak and equilibrium (Figure 6.1.9). Similar results were found for other Talukas (not shown).

Figure 6.1.9. Fraction of new infections due to high-risk groups with half the values of the relevant high-risk parameters, in 1986, 2004 and equilibrium in Taluka A



When FSW or client size, or mean FSW-client rate is double B-C values, the fraction of new yearly infections caused by high-risk groups was even higher in 1986, 2004 and at equilibrium (Figure 6.1.10) when total high-risk partnerships were allowed to vary (PV1-PV4).

Figure 6.1.10. Fraction of new infections due to high-risk groups with double the values of the relevant high-risk parameters, in 1986, 2004 and equilibrium in Taluka A



These results indicate that high-risk groups in Bagalkot District would be effective to target with HIV prevention interventions, even if there are half the size of these key high-risk sexual behaviour parameters, and would have reduced effectiveness over time, or if the mean high-risk contact rate is much less than what is observed.

- ***Largest impact on decreasing overall model prevalence***

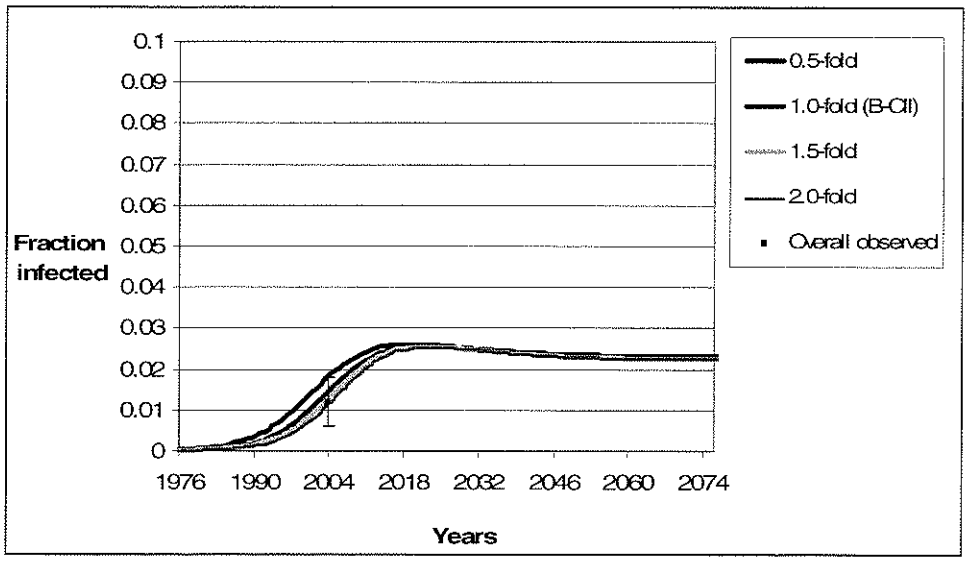
When total high-risk partnerships are fixed when FSW or client size is varied, increasing these parameters from half-to-double the base-case values caused the largest decreases in 2004, peak and equilibrium model HIV prevalence (Figures 6.1.4-6.1.6). With fixed high-risk partnerships, increasing FSW size (with fixed client size and mean FSW-MCL contact rate, PC1) caused decreases in overall model prevalence, since the MCL-FSW contact rate decreased; increasing client size (with fixed FSW size and mean MCL-FSW contact rate, PC2) caused decreases in overall model prevalence, since FSW-MCL contact rate decreased. Here, the effect of larger bridging population (clients) was overcome by the smaller FSW-MCL contact rate. Varying MCL size had a slightly larger impact on decreasing model prevalence in 2004 compared with the FSW population size (decreased by 3.8% compared with 3.0%), at peak (1.9% compared with 1.4%). The same change in HIV prevalence occurred at equilibrium (0.9%) (Figure 6.1.6).

- ***Smallest impact on increasing model HIV prevalence***

Increasing the overall population size in each Taluka from half-to-double the B-CII values caused the HIV epidemic in the overall population to grow more slowly, with peak and equilibrium being reached later, but marginally different. This happened because we still seeded the epidemic with the same small number of initial infections, but a larger total population size spread out the acquisition and transmission of infections and it took longer for the epidemic to establish. In Taluka A, for example, when the overall population was increased from half of its

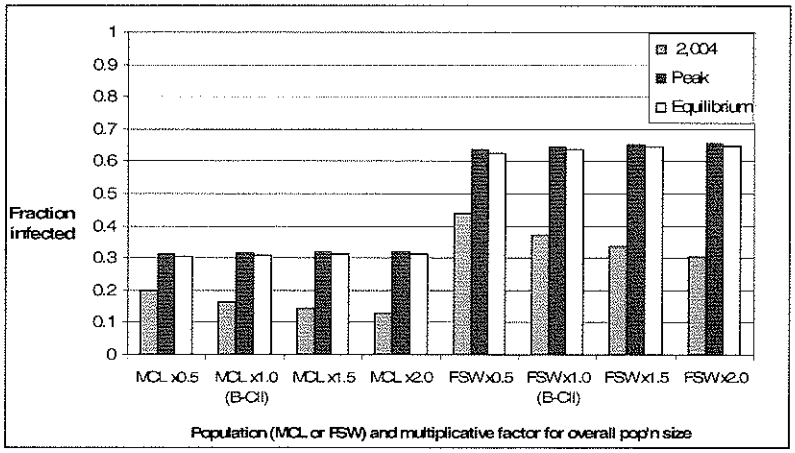
estimated value to double the estimated value (no other parameters change when overall population size changes), this caused overall model HIV prevalence in 2004 to decrease from 1.8% to 1.1% in 2004 (B-CII: 1.5%), with negligibly different peak being reached later (2.6% in 2018 to 2.5% in 2022, B-CII: 2.6% in 2021) and negligibly different equilibrium HIV prevalence being reached later (2.3% in 2063 to 2.2% in 2071, B-CII: 2.3% in 2067). Figure 6.1.11 shows how overall prevalence varied in Taluka A along with overall population size. Results from all Talukas are shown in Tables A2.1-A2.3 Appendix 2.

Figure 6.1.11. Overall model HIV prevalence in Taluka A as overall population size varied from half-to-double the B-CII values



Each population exhibited this same pattern, including both low-risk and high-risk populations. For example, Figure 6.1.12 shows how high-risk peak and equilibrium HIV prevalence varied negligibly with increased overall population size, but 2004 model prevalence is higher earlier in the epidemic because the epidemic is delayed with higher overall population size (peak and equilibrium reached later: MCLs: 2030 versus 2025 and 2082 versus 2076; FSWs: 2031 versus 2027 and 2088 versus 2079).

Figure 6.1.12. High-risk peak and equilibrium model HIV prevalence in Taluka A as overall population size varied from half-to-double the B-CII values



6.2. Migration

Since data on migration is limited, the analysis on migration in this study is explanatory, intended to provide an idea of the potential impact of several different levels and patterns of migration in rural India. The migration factors examined included size of the migrating high-risk populations, duration of migration, mean high-risk contact rate in the place of migration (POM) and HIV prevalence in high-risk groups in the place of migration. Five different out-migration scenarios were developed and are summarized in Table 6.2.2.

To assess the independent impact of each migration risk factor, all other model parameters were initially calibrated to base-case migration (B-CM) parameter values (Table 6.2.1 and in Table A1.1 in Appendix 1) in Taluka A with lowest 2004 observed HIV prevalence.

Table 6.2.1. Base-case migration parameters and range explored

	Migrating FSW size	Migrating male/MCL size	Duration of time away	Mean number of HR partners in POM	HIV prevalence in FSWs, POM (2004)	HIV prevalence in FSWs, POM (final)	HIV prevalence in MCLs, POM (2004)	HIV prevalence in FSWs, POM (final)
B-CM	15%	30%	4 months	1.0-fold	35%	80%	17%	40%
Range	[7.5 –30]%	[15 – 60]%	[2 – 8] months	[0.5 – 2.0]-fold	[17 – 70]%	80%	[8 – 35]%	40%

To understand why HIV prevalence is different in the three Talukas, we determined if any of the migration factors, when varied alone or together, could bring overall model prevalence in Taluka A to within range of the 2004 observed HIV prevalence in other Talukas in Bagalkot District. To understand where to focus prevention efforts, we determined which migration factor had the greatest effect on 2004, peak and equilibrium model prevalence and ranked each migration parameter within each of the five migration scenarios (Table A2.4 in Appendix 2). We developed low, moderate and high migration (all migration parameters at low, moderate or high), in each of the three migration scenarios that assume a gradually increasing (logistic) epidemic in high-risk groups in the POM (M2, M3 and F2), and determined which risk group is transmitting and acquiring the most infections, to help direct prevention efforts at certain populations. Finally, we compared the effectiveness of a simple condom intervention program, introduced in 2004 in clients and FSW, on HIV prevalence in low-risk females across migration scenarios.

In all migration scenarios we assumed that the local mean MCL-FSW contact rate was determined by the male demand for sex work – thus, when MCLs migrated, the local mean MCL-FSW contact rate decreased and when FSWs were away, the local mean MCL-FSW contact rate increased proportionately to the size of the migrating population (See Table 6.2.2 below and Chapter Four, Section 4.1).

Table 6.2.2. Five migration scenarios explored

Migration scenario	Migrating high-risk population	Mean local FSW-MCL contact rate when migrants are away	Mean local MCL-FSW contact rate when migrants are away	Total number FSW-MCL partnerships	HIV prevalence in POM
M.1	MCLs	Constant	decreases	Varies	Constant
M.2	MCLs	Constant	decreases	Varies	Logistic
M.3	All males	Constant	decreases	Varies	Logistic
F.1	FSWs	Constant	increases	Constant	Constant
F.2	FSWs	Constant	increases	Constant	Logistic

6.2.1. Understanding heterogeneity

- **Migrating client scenarios: M.1 & M.2**

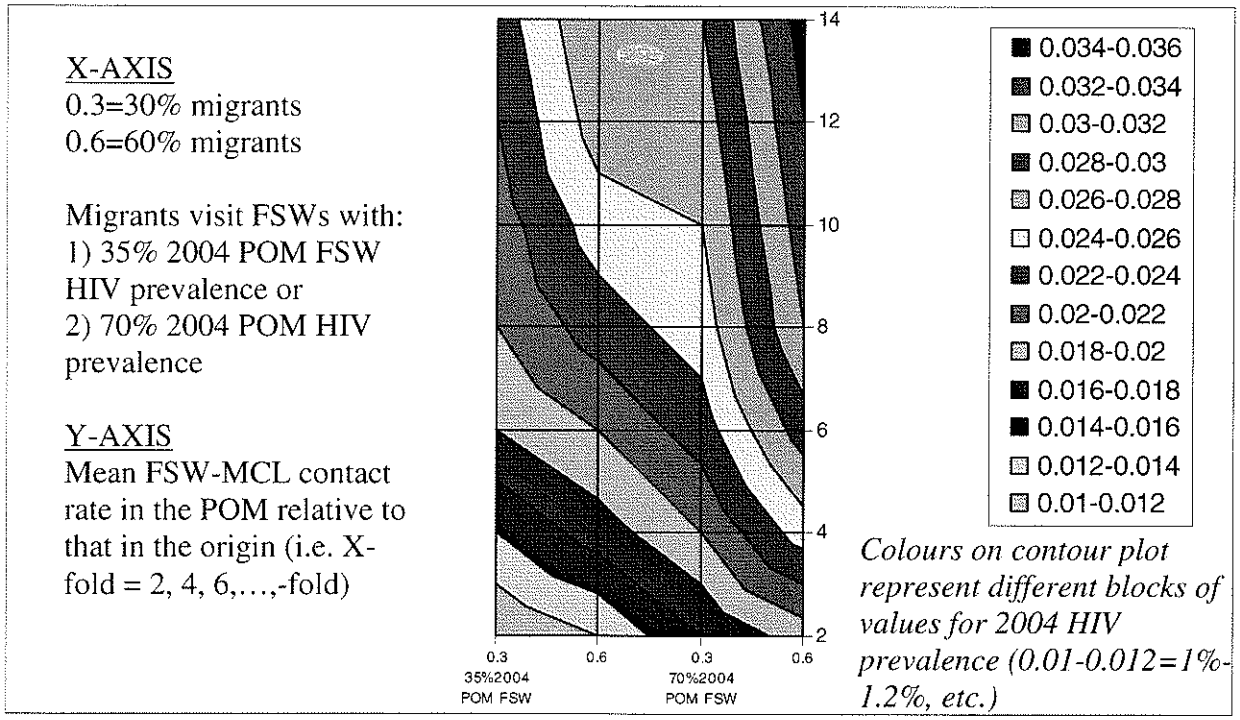
In the presence of base-case migration (B-CM), 2004 model HIV prevalence in Taluka A's overall population increased to 1.7% in M.1 and decreased to 1.0% in M.2, from a B-CII (without migration) value of 1.2%. In both scenarios, even increasing the mean FSW-MCL contact rate in the POM to double the B-CM values, while holding all other parameters at B-CM values, was not sufficient to bring Taluka A's overall model 2004 prevalence to reach Taluka B's observed prevalence (2.9% [2.2 – 3.6]%). This is true at all points of the epidemic, even at peak (Table A2.4 in Appendix 2). Overall 2004 model HIV prevalence reached within range of Taluka B's observed 2004 HIV prevalence when we varied migration parameters higher than double the base-case values or when they were varied together (multivariate analysis).

For M.1, holding B-CM values constant for other migration parameters (Table 6.2.1), increasing the mean FSW-MCL contact rate in the POM alone to four-fold that in the place of origin caused overall 2004 model HIV prevalence to reach 2.2% from 1.7% (within Taluka B's range). This is likely unrealistic: the estimated local mean FSW-MCL contact rate in Taluka A is already 120 per year (10 per month). With four-fold the number in the POM, MCLs would have 40 FSW contacts per month on average for the four months duration in the POM. Data was not available on the level of increase in the mean number of high-risk partners clients have while in the POM (in India or in other countries), but it seems that greater than one per day is high.

Figure 6.2.1 is a contour plot for scenario M.2, showing the combination of migrant size, mean FSW-MCL contact rate and HIV prevalence in FSWs in the POM (assuming four months duration away) required to bring overall 2004 model HIV prevalence in Taluka A to within range of Taluka B's or C's 2004 observed HIV prevalence (at least 2.2% or 3.5%). For M.2, increasing

the FSW-MCL contact rate in the POM alone to four-fold that in the place of origin caused overall 2004 model HIV prevalence to reach 1.7% from 1.0%, not within Taluka B's range. It was necessary to increase the FSW-MCL contact rate in the POM to 12-fold that in the place of origin to reach 2.2% in 2004. This is even less realistic, requiring MCLs to have 122 FSW contacts per month for four months duration in the POM: more than four FSW contacts per day.

Figure 6.2.1. Contour plot for the scenario when **only clients** *migrate (M.2), showing the combination of migrant size, mean FSW-MCL contact rate in the POM (relative to that in the place of origin) and HIV prevalence in FSWs in the POM required to bring overall 2004 model HIV prevalence in Taluka to other Talukas' observed values.*



With all other migration parameters at B-CM values in M.1 (see Table 6.2.1 for values), even increasing the constant HIV prevalence in the POM to 100% (clearly an unrealistic value) did not increase Taluka A's overall 2004 model prevalence to within Taluka B's observed values (increased it to 2.0% in 2004). Similarly, no values for POM FSW HIV prevalence alone in M.2, with a gradually increasing (logistic) HIV prevalence in FSWs in the POM, brought overall model HIV prevalence in Taluka A to within Taluka B's observed values.

However, in M.1, when both migration-associated risk factors were increased to double their B-CM values (2.0-fold FSW partners and 70% constant HIV prevalence in the POM), with 30% migrants and four months duration away, overall 2004 model prevalence in Taluka A again increased to 2.2% (within Taluka B's observed range). This seems more realistic: MCLs would only have 20 FSW contacts per month in the POM. A 70% constant HIV prevalence in FSWs since the start of the epidemic is clearly already an unrealistic assumption, although FSWs in Mumbai (a place commonly reported as being a site for migrating males) have been reported to have as high as 70-80% HIV prevalence within the past 10 years. In M.2, even when FSW prevalence in the POM hit 70% in 2004 (80% at equilibrium), a very high increase in the mean FSW-MCL contact rate was required in the POM (5.0-fold) to reach 2.2% in 2004 (Figure 6.2.1), which resulted in an mean of 50 per month for four months duration, which again seems high.

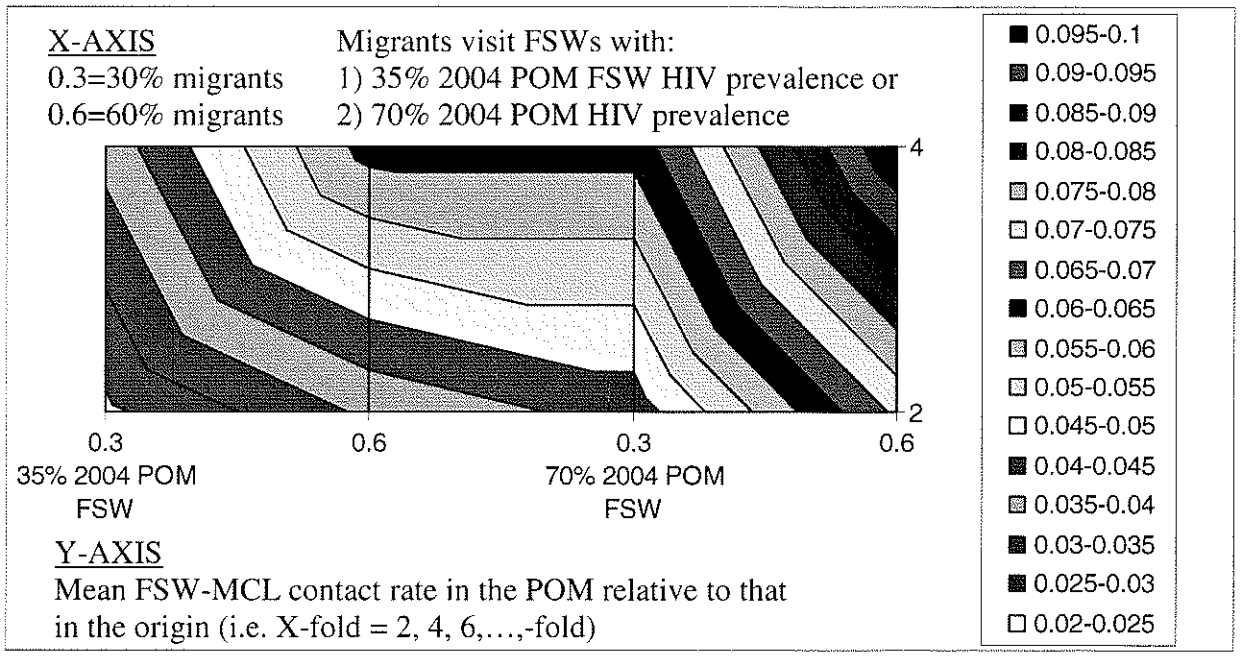
We might expect that higher levels of risk behaviour in the POM compared with in the place of origin would mean that increasing the size of the migrating MCL population or the duration of time away would substantially increase the modeled epidemic – however, the subsequent decrease in the local mean MCL-FSW contact rate while MCLs are in the POM was larger with increased migrant size and time away, countering the effects of higher migration-associated risk behaviour. For example, in M.1, with double the mean number of partners and HIV prevalence in the POM (70%), doubling MCL size from 30% to 60% only increased 2004 overall model HIV prevalence to 2.3% from 2.2%. Doubling MCL size and increasing the duration of time away to eight months from four months still only caused 2004 model HIV prevalence to reach 2.3%. This highlights the importance of understanding what happens to high-risk sexual behaviour in local high-risk groups while migrating groups are in the POM.

Migrating male scenarios: M.3

In scenario M.3, where a fraction of all males (including low-risk males and MCLs) were assumed to migrate, overall 2004 model prevalence did not reach Taluka B's observed 2004 HIV prevalence in the presence of base-case migration (Table 6.2.1 for values), reaching 1.5% in 2004. However, with base-case migration, the epidemic in Taluka A reached 2.2% in 2007 (within range of Taluka B's 2004 overall observed HIV prevalence) and 3.5% in 2018 (within range of Taluka C's 2004 observed overall HIV prevalence: 4.9% [3.5 – 6.3]%), suggesting that the epidemics in Talukas B and C could have begun earlier than Taluka A (by 3-12 years).

Figure 6.2.2 is a contour plot for scenario M.3, showing the combination of migrant size, mean FSW-MCL contact rate in the POM (relative to that in the place of origin) and HIV prevalence in FSWs in the POM required to bring overall 2004 model HIV prevalence in Taluka A to within range of Taluka B's or C's 2004 observed HIV prevalence (at least 2.2% or 3.5%).

Figure 6.2.2. Contour plot for the scenario when **all males** *migrate, showing the combination of migrant size, mean FSW-MCL contact rate in the POM (relative to that in the place of origin) and HIV prevalence in FSWs in the POM required to bring overall 2004 model HIV prevalence in Taluka A to within range of other Talukas' 2004 overall observed HIV prevalence.*



Colours on contour plot represent different blocks of values for 2004 HIV prevalence (0.01-0.012=1%-1.2%, etc.)

In scenario M.3, for B-CM values for all other parameters (see Table 6.2.1 for values), increasing the mean FSW-client contact rate in the POM alone to double that in the place of origin caused overall 2004 model HIV prevalence to reach 2.4% from 1.5% (within Taluka B's range) (Figure 6.2.2; Table A2.4 in Appendix 2). This may be realistic – with two-fold the number of FSW contacts in the POM as in the place of origin, MCLs would have about 20 FSW contacts per month for the duration of time they were in the POM. With four-fold the mean FSW contacts that MCLs have in the POM, overall 2004 model HIV prevalence increased to 3.7%, which is within range of Taluka C's 2004 observed values.

With other migration parameters at B-CM values in M.3 (see Table 6.2.1 for values), increasing the gradually increasing (logistic) HIV prevalence in FSWs in the POM to double the B-CM values in 2004 (70% in 2004 and 80% at equilibrium) increased Taluka A's overall 2004 model prevalence to within Taluka B's observed values (increased it to 2.7% in 2004) (Figure 6.2.2; Table A2.4 in Appendix 2). Increasing POM FSW prevalence to 98% in 2004 and 100% at equilibrium was required to bring overall model prevalence in Taluka A in 2004 to within Taluka C's observed 2004 overall values (to 3.5%), but this is of course, very unrealistic.

Since increasing the size of the migrant population and the duration of migration increased the epidemic in the overall population (mainly because it was more heavily weighted with low-risk migrant males, who had much higher HIV prevalence than non-migrant low-risk males, due to their interactions with FSWs in the POM) almost to the extent that 2004 overall model HIV prevalence reached within Taluka B's observed range (reached 2.1% in 2004 with 60% migrants or eight months duration in the POM, even with the same level of migration-associated risk behaviour in the POM as in the place of origin), we expected that increasing the size of the migrating MCL population or the duration of time away would substantially increase

the modeled epidemic. This would override the subsequent decrease in the local mean MCL-FSW contact rate while MCLs are in the POM, which was larger with increased migrant size and time away, countering the effects of migration-associated risk behaviour. This was confirmed with double the mean FSW-MCL contact rate in the POM and double the 2004 FSW prevalence in the POM (70% in 2004, 80% at equilibrium): overall model HIV prevalence increased remarkably with increased migrant size or duration of time away. When MCL size was increased from half-to-double the B-CM values (15 to 60%, B-CM: 30%), this increased 2004 overall model HIV prevalence from 2.8% to 7.1%. Peak prevalence increased from 3.5% (2012) to 8.7% (2011); equilibrium prevalence increased from 2.9% in 2050 to 6.9% in 2060. Overall 2004 model prevalence was within Taluka B's observed 2004 prevalence with just 15% migrating males and with 30% migrating males was within Taluka C's overall observed prevalence (4.0%).

It is clear from this analysis that "low-risk clients" and their sexual behaviour in the place of migration may mean that they act as a very important bridging population in Bagalkot District. However, in our analysis we assumed 15% - 60% low-risk male clients and this may be an overestimate of the number of low-risk clients in our population. Halli et al (2007) showed in focus groups in Bijapur District (neighbouring District to Bagalkot) that there was ~7.0% difference between married males reporting visiting FSWs in place of migration compared to place of origin (29.4% versus 23.0%, ~28% increase). Additional analysis should be conducted on an increased range of values for the size of low-risk clients, and for values that differ from the numbers of clients migrating who are clients in both the POM and the place of origin.

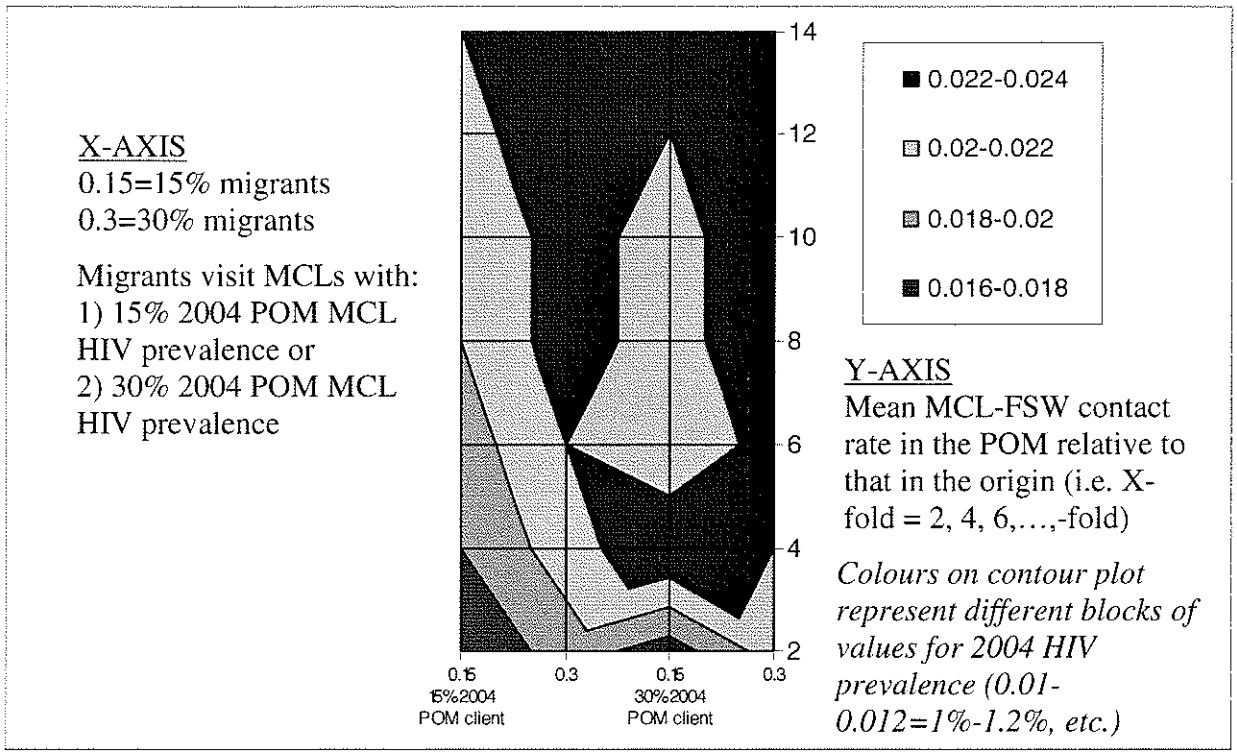
- **Migrating FSW scenarios: F.1 & F.2**

In F.1, only when migrating FSW size was varied to double its B-CM values (30%), assuming B-CM for all other migration parameters (see Table 6.2.1 for values), did Taluka A's overall 2004

model HIV prevalence increase to 2.2%, just within Taluka B's overall observed values (Table A2.4 in Appendix 2). When the B-CM values for duration of migration, mean MCL-FSW contact rate in the POM and HIV prevalence of MCLs in the POM were doubled, each independently brought overall observed HIV prevalence to 2.0%, 2.1% and 2.1% respectively.

Assuming a gradually increasing (logistic) HIV epidemic in MCLs in the POM (F.2), none of the FSW migration parameters, even when varied to double their B-CM values, could bring Taluka A's overall model HIV prevalence to within Taluka B's overall observed values. Figure 6.2.3 is a contour plot for scenario F.2, showing the combination of migrant size, mean MCL-FSW contact rate in the POM (for four months duration away) and HIV prevalence in clients in the POM required to bring overall 2004 model HIV prevalence in Taluka A to within range of Taluka B's or C's 2004 observed HIV prevalence (at least 2.2% or 3.5%).

Figure 6.2.3: Contour plot for the scenario when **only FSWs** *migrate, showing the combination of migrant size, mean MCL-FSW contact rate in the POM (relative to that in the place of origin) and HIV prevalence in clients in the POM required to bring overall 2004 model HIV prevalence in Taluka A to within range of other Talukas' 2004 overall observed HIV prevalence.*



In F.1, when the mean MCL-FSW contact rate in the POM was increased alone to four-fold that in the place of origin, overall 2004 model HIV prevalence increased to 2.2% from 2.0% (within Taluka B's range, but not a substantial increase). This seems unrealistically high: the estimated local MCL-FSW contact rate in 2004 is already high, at 510-520 per year (~10 per week and 40 per month). With four-fold the number in the POM, FSWs would have 40 MCL contacts per week (~5-6 per day) and 160 per month, which seems high, though data is not available to confirm this. For overall model HIV prevalence to reach Taluka B's observed 2004 HIV prevalence in F.2, it was required to increase the mean MCL-FSW contact rate in the POM to even higher, at 14-fold that in the place of origin (Figure 6.2.3) to reach 2.2% from 1.5%.

However, even increasing the mean MCL-FSW contact rate in the POM to 20-fold that in the place of origin did not increase overall 2004 model HIV prevalence to within Taluka C's overall observed values in 2004 in either FSW migration scenario (see Figure 6.2.3 for F.2). This caused migrating FSW model HIV prevalence to increase to final prevalence (90-95%) only a few years after the epidemic began. Since we only assumed that 15% of FSWs migrate and there were only 837 FSWs in Taluka A in 2004 to begin with, this meant that only 125 FSWs were migrants. Even with almost all of the FSWs infected, since FSWs were assumed to only have high-risk partnerships with MCLs, the epidemic did not easily leak into the low-risk population.

Unless the proportion of FSWs migrating is much larger than estimated, it seems that overall model HIV prevalence in 2004 is unlikely to reach much above 2.2%, and certainly not within Taluka C's overall observed HIV prevalence in 2004. In F.2, when all other migration parameters are at the high values of the uncertainty ranges (double the B-CM values), a larger proportion of migrating FSWs did cause an increase in overall 2004 model HIV prevalence, compared with when other migration parameters are at B-CM values. Increasing the migrating

FSW population from half-to-double the B-CM values (15% - 30%, B-CM: 15%) caused 2004 overall model HIV prevalence to increase from 1.6% to 2.2%, B-CM: 1.8% (no migration: 1.2%), within Taluka B's overall observed values but still not within Taluka C's. Peak prevalence increased less, from 2.0% - 2.3% and so did equilibrium prevalence, from 1.7% to 1.9%. In F.1, when the migrating FSW population was saturated with HIV (85% model prevalence in 2004; 90-95% at equilibrium) due to an extremely and unrealistically high mean MCL-FSW contact rate in the POM (20-fold that in the place of origin) and 15% constant HIV prevalence in MCLs in the POM, even increasing the migrating FSW size to 90% for a duration of eight months only increased 2004 overall model HIV prevalence to 3.3% (too low for Taluka C's overall observed prevalence), with peak occurring in 1995 (4.4%) and an equilibrium HIV prevalence of 3.0% in 2030. Migrating FSW and non-migrating HIV prevalence increased 92.5% and 90.7% respectively in 2004, having already reached the peak/equilibrium prevalence by about 1987. Similar results are obtained in F.2.

6.2.2. Understanding where to focus prevention efforts

- **Base-case migration increased 2004 model prevalence compared with no migration**

In the presence of base-case migration for all migration scenarios except M.2 (only male clients migrate; logistic increasing HIV prevalence in FSWs in the POM), the modelled epidemic grew more quickly and thus 2004 overall model HIV prevalence was lower in the absence of migration compared to some migration (1.2% compared with B-CM: 1.7% in M.1; 1.5% in M.3; 2.0% in F.1; 1.5% in F.2) (see Table A2.4 in Appendix 2).

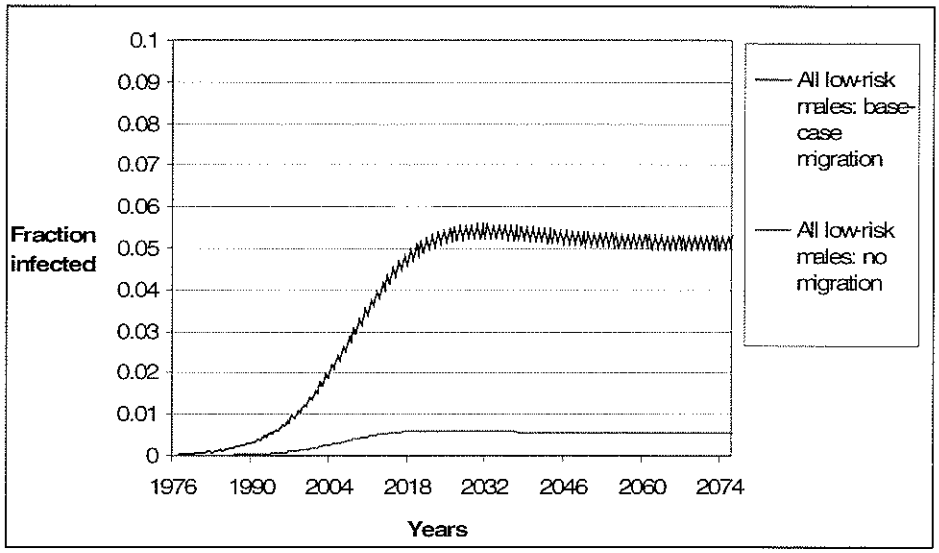
In M.1 this happened because for B-CM, we assumed that HIV prevalence of MCLs in the POM was constant at 35%. Early in the HIV epidemic, the constant FSW HIV prevalence in

the POM was higher than in the place of origin, so HIV prevalence increased in migrating MCLs and the overall population. Later in the epidemic, this constant POM-FSW HIV prevalence was lower than in the place of origin (~70% at equilibrium) and so B-CM caused lower HIV prevalence in migrating MCLs. This happened even though when migrant MCLs were away, the mean local MCL-FSW contact rate dropped proportionately to the size of the migrating population (30%: from ~510 to ~350 in 2004) and the mean FSW-MCL contact rate in the POM was the same as in the place of origin. In M.2 however, a more realistic, gradually increasing (logistic) HIV prevalence was assumed in FSWs in the POM, and since this epidemic curve was similar to the curve seen in FSWs in the place of origin, did not have the same effect early in the local epidemic as constant POM-FSW prevalence. Due to local MCL-FSW contact rate dropping with client migrated, B-CM, 2004, peak and equilibrium HIV prevalence decreased.

In M.3, a logistic increasing HIV prevalence was also assumed in FSWs in the POM, and the mean MCL-FSW contact rate locally also decreased when males were in the POM. Because of this, the total MCL population (including migrating and non-migrating MCLs) and the FSW population had decreased 2004, peak and equilibrium model HIV prevalence in the presence of B-CM compared with no migration. However, the presence of B-CM in M.3 caused higher 2004, peak and equilibrium HIV prevalence compared with no migration because low-risk males as well as MCLs migrated, and engaged in client behaviour in the POM. Total low-risk male HIV prevalence increased greatly in the presence of some low-risk males migrating, to about 2% model prevalence in 2004, 5.5% at peak and 5% at equilibrium with base-case migration (0.3%, 0.6% and 0.5% respectively without migration: Figure 6.2.4). Migrating low-risk males reached 6% model HIV prevalence in 2004, and a peak/equilibrium prevalence of almost 20%. Low-risk males who did not migrate also showed slightly increased long-term model HIV prevalence with

base-case migration compared with no migration (peak: increases from 0.6% to 0.8%, equilibrium increases from 0.5% to 0.7%), as a result of the increased epidemic in low-risk females (peak: 0.4% to 0.5%, equilibrium: 0.3% to 0.4%), who had long-term relationships with low-risk males who migrate as well as low-risk males who do not migrate.

Figure 6.2.4. Total low-risk male (migrating and non-migrating) model HIV prevalence in the presence of base-case migration in M.3 and no migration



In F.1, constant HIV prevalence is assumed in MCLs in the POM, and thus contributes to the increase in 2004 model HIV prevalence, which happened even though for B-CM we assume that the mean MCL-FSW contact rate in the POM is the same as in the place of origin. Early in the epidemic, the constant MCL HIV prevalence (17%) in the POM was higher than in the place of origin, so HIV prevalence increased in migrating FSWs, but later, MCL HIV prevalence in the POM was lower (~30% at equilibrium) and so a larger migrating FSW size or duration of migration resulted in lower HIV prevalence in migrating FSWs. In addition, when FSWs were away, since we assumed that the male demand for sex remained constant in the place of origin regardless of the number of local FSWs (who are not replaced when FSWs migrate), the mean local MCL-FSW contact rate increased proportionately to the size of the migrating population

(15%: from ~510 to ~600). In F.2 however, a more realistic, gradually increasing (logistic) HIV prevalence was assumed in MCLs in the POM, and since this epidemic curve is similar to the curve seen in MCLs in the place of origin, does not have the same effect early in the local epidemic as constant POM-MCL prevalence. Unlike with male migration, the increase in the local MCL-FSW contact rate when migrating FSWs are away caused 2004, peak and equilibrium model HIV prevalence to increase in the presence of base-case migration, above prevalence in the absence of migration (Table A2.4 in Appendix 2).

- **Largest effect when both low- and high-risk males migrate (scenario M.3)**

The largest impact on overall 2004, peak and equilibrium model HIV prevalence by far happened when a proportion of all males migrated (MCLs and low-risk males) and were clients in the POM (Scenario M.3). This was true of all of the migration parameters investigated, although higher levels of migration-associated risk behaviour caused the greatest increases in HIV prevalence in the overall population earlier in the epidemic, in 2004: increased HIV prevalence in the POM, when varied from half-to-double the B-CM value (17% - 70% 2004 HIV prevalence in FSWs in the POM; always the same equilibrium model HIV prevalence: 80%), had the largest effect on 2004 model HIV prevalence (increased by 1.6%, from 1.1% to 2.7% in 2004, B-CII: 1.2%, B-CM: 1.5%). Next was the mean FSW-client contact rate in the POM (increased by 1.4%, from 1.0% to 2.4%), duration of migration (increased by 0.9%, from 1.2% to 2.1%) and finally, migrating male size (increased by 0.8%, from 1.3% to 2.1%).

Figures 6.2.5-6.2.7 show the absolute effect on 2004, peak and equilibrium model HIV prevalence (black=increase; white=decrease) caused by each migration parameter (increased from half-to-double the B-CM values) in the three migration scenarios with gradually increasing (logistic) modelled epidemic assumed in high-risk groups in the POM (M.2, M.3 and F.2).

Figure 6.2.5. 2004 model HIV prevalence in the overall (total) population when each migration parameter in each migration scenario varied through its estimated range (all other migration parameters at B-CM values)

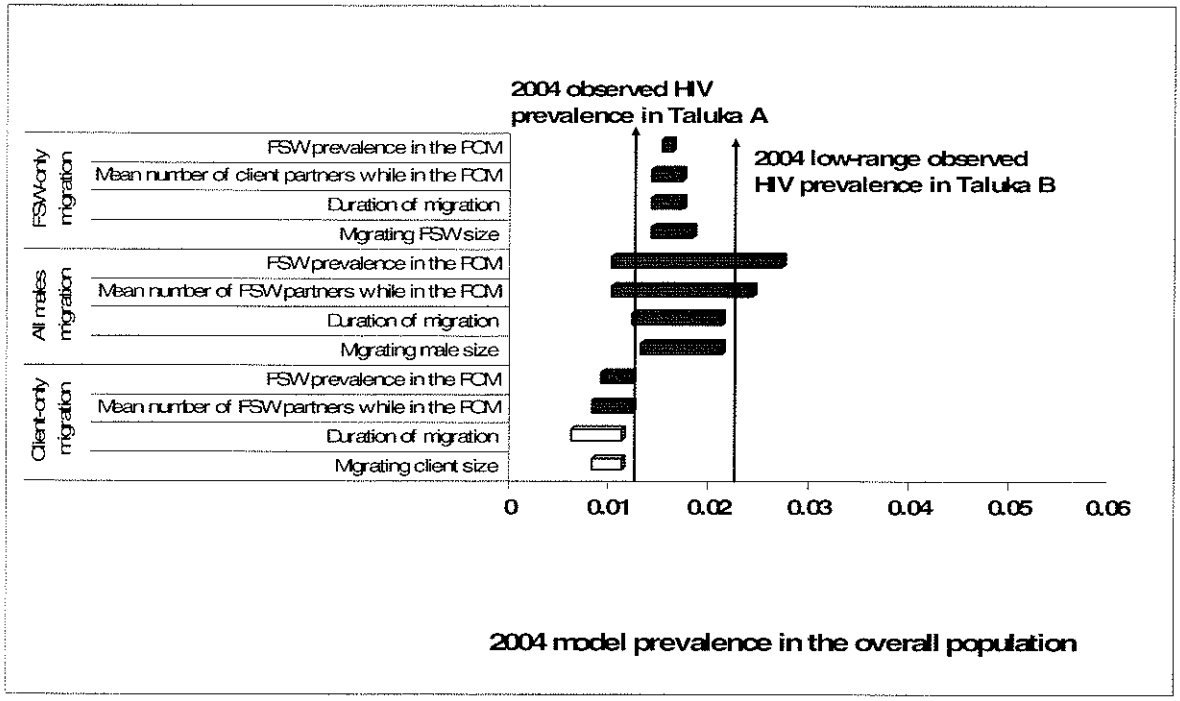


Figure 6.2.6. Peak model HIV prevalence in the overall (total) population when each migration parameter in each migration scenario varied through its estimated range (all other migration parameters at B-CM values)

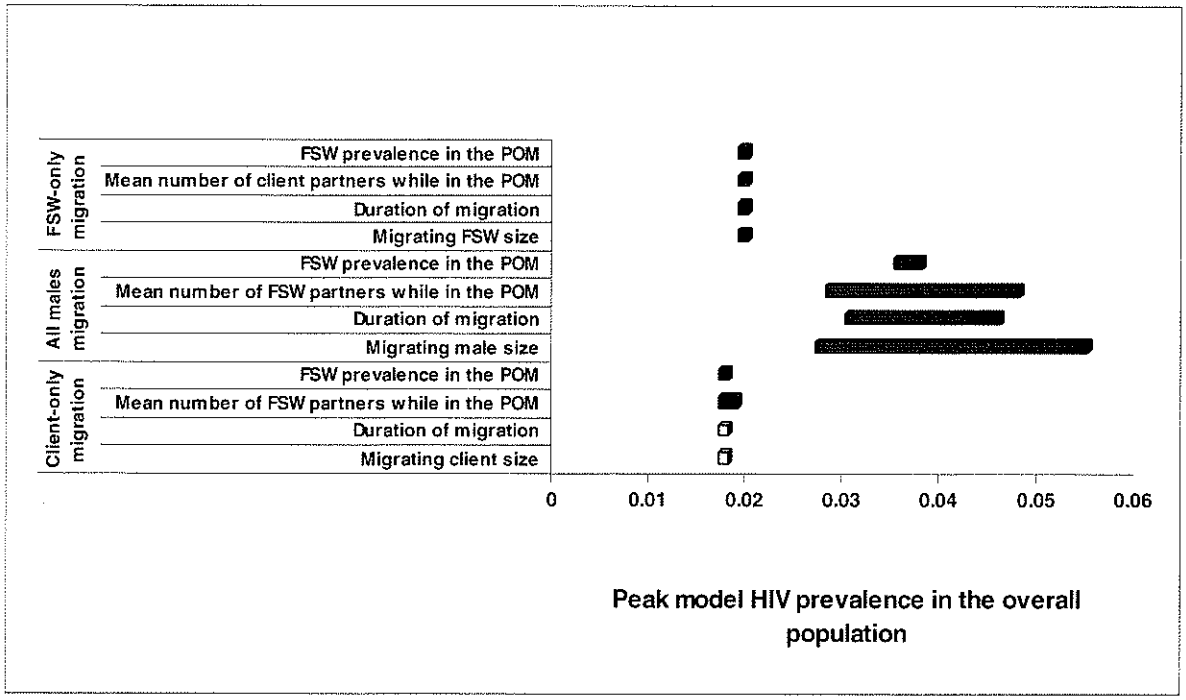
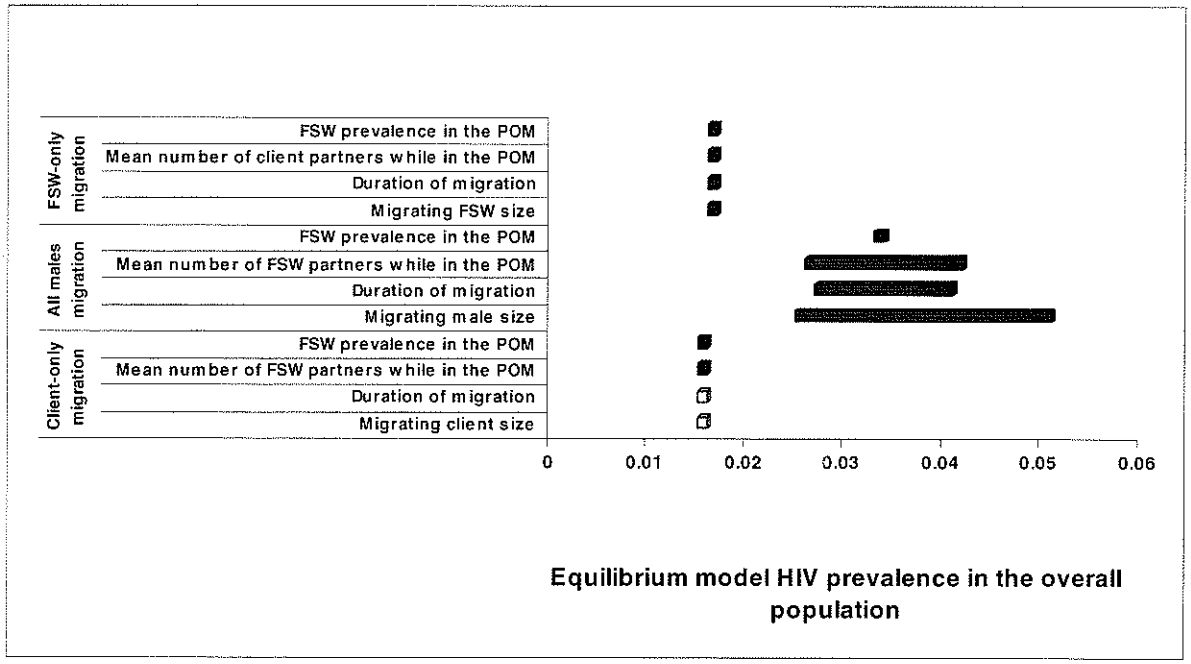


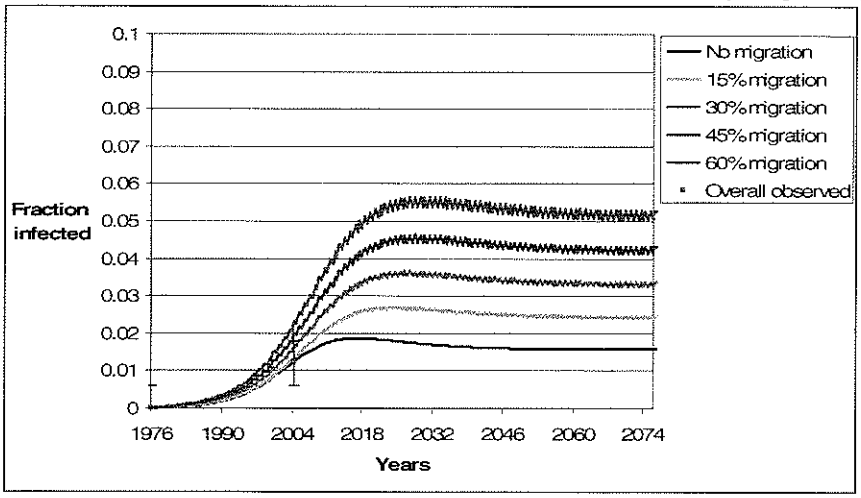
Figure 6.2.7. Equilibrium model HIV prevalence in the overall (total) population when each migration parameter in each migration scenario varied through its estimated range (all other migration parameters at B-CM values)



Interestingly, although migration in the presence of higher levels of migration-associated risk behaviour caused the largest increases in HIV prevalence in 2004 (Figure 6.2.5), the size of the migrating male population became the most important factor in increasing HIV prevalence later in the epidemic (Figure 6.2.6, 6.2.7). Because we assume the same equilibrium HIV prevalence in FSWs in the POM (80%), even though 2004 HIV prevalence varied from half-to-double the B-CM values in this analysis, the effects on peak and equilibrium prevalence in the overall Taluka A population is negligibly different (increased by 0.3% and 0.1%, compared to 2.8% and 2.6% for size of migrating male population, Figure 6.2.6, 6.2.7). Overall model HIV prevalence increased with a larger migrating male size because this weighted the overall population more heavily with a large population (migrant low-risk males) that engaged in high-risk behaviour and had increased HIV infection. In addition, a larger population of low-risk males who migrated caused slight increases in infection in low-risk females (peak: 0.5% in 2022

from 0.4% in 2015 [no migration]; equilibrium: 0.5% in 2050 from 0.3% in 2048 [no migration]). This happened even though we assumed that there was no increase in high-risk behaviour in the POM (B-CM values for other parameters: same mean FSW-client contact rate and HIV prevalence in FSWs in the POM as in the place of origin; 4 months duration of migration) and the mean MCL-FSW contact rate in the place of origin decreased proportionately to the size of the migrating client population. Figure 6.2.8 shows how peak and equilibrium model HIV prevalence in the overall population increased compared with earlier in the epidemic, with increased migrant size.

Figure 6.2.8. Overall model HIV prevalence as the size of migrating males varies



Long-term prevalence rates in non-migrant, low-risk males were elevated over 1% only when the migrating male population was the largest (60%), contributing to the largest increases in peak and equilibrium prevalence in the overall population (5.5% and 5.1% for highest migrating male size). Surprisingly, both short and long-term low-risk female prevalence was relatively unaffected by the significant increases seen in the low-risk migrant male population and remained less than one percent over all time regardless of the size of the migrating male population, duration of time away or migration-associated risk behaviour. This is likely because the mean low-risk partner change rate per year was very low (~one low-risk partner per sexually

active lifetime), so even if there was high prevalence in low-risk clients, infection would not transmit very much to low-risk female population.

Long-term prevalence rates in both non-migrant and migrant MCLs however, were less affected by migrating client size and noticeably highest with increased risk behaviour in the POM (high-risk contacts in the POM were double that in the place of origin), with maximum peak and equilibrium prevalence reaching 31.6% (2034) and 30.8% (2076) respectively in non-migrant MCLs (B-CM=29.4% [2037] and 28.8% in 2100) and 30.1% and 39.6% (2033) and 39.1% (2076) among migrant MCLs (B-CM=30.7% [2042] and 30.1% in [2100]), even though we held other parameters constant at B-CM values.

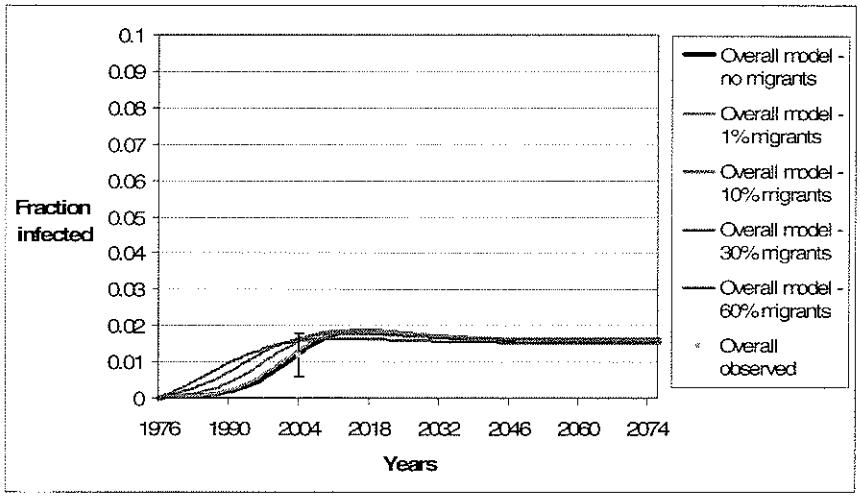
- **Effects of migration small for other migration scenarios**

When only a proportion of MCLs or FSWs were assumed to migrate 30% seasonally (M.1 and M.2; F.1 and F.2) and other migration parameters were set at B-CM values, when each of the migration parameters was varied independently from half-to-double the B-CM values, the absolute changes in 2004, peak and equilibrium HIV overall model prevalence were small, at <0.5% (Figures 6.2.5-6.2.7; Table A2.4 in Appendix 2).

Migrating client scenarios (M.1 and M.2)

In the first MCL migration scenario (M.1: see Table 6.2.2) with a proportion of MCLs migrating and a constant HIV prevalence in FSWs assumed in the POM (35%), where there were no increases in migration-associated risks (B-CM values: see Table 6.2.1), the independent impact of the size of the migrating client population and the duration of migration was negligible in 2004 (overall 2004 model prevalence remained unchanged at 1.5% as each parameter increased from half-to-double the B-CM values and was highest at B-CM values: 1.7%; peak prevalence decreased from 1.8% to 1.6%; equilibrium remained constant at 1.6%) (Figure 6.2.9).

Figure 6.2.9. Overall model prevalence as the size of the migrating MCL population varies, assuming constant HIV prevalence in FSWs in the place of migration

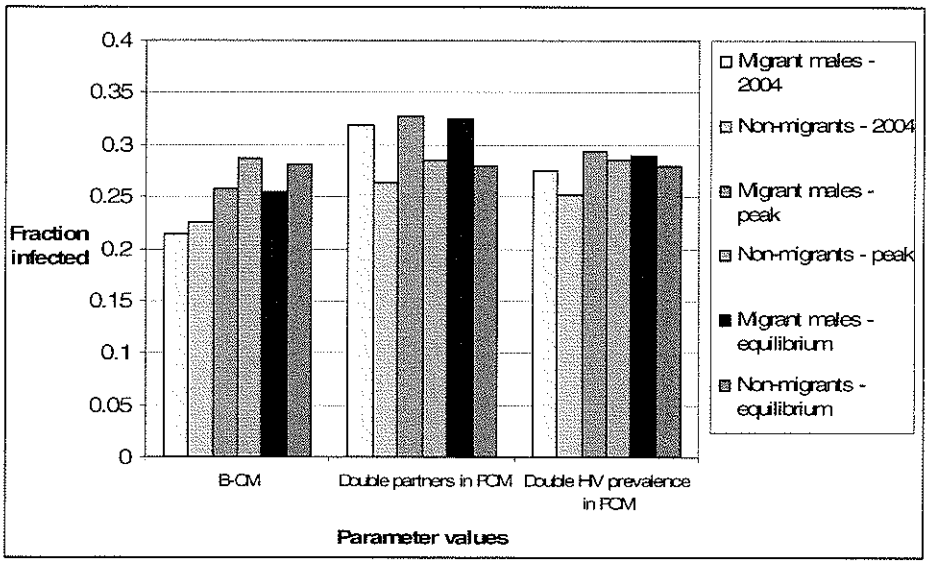


In the second MCL migration scenario (M.2), in which a gradually increasing logistic HIV prevalence in high-risk groups in the POM is assumed as opposed to a constant HIV prevalence, the independent impact of varying migrating population size and duration of migration from half-to-double the B-CM values caused small absolute decreases in overall model HIV prevalence in 2004, peak and equilibrium (Figure 6.2.5-6.2.7).

This happened because a larger migrating MCL size and duration of time away caused a negligibly different epidemic in MCLs who migrate as those who do not migrate, since the B-CM values for the mean FSW-MCL contact rate and HIV prevalence in FSWs in the POM were the same as in the place of origin. This also caused a smaller epidemic in local non-migrating MCLs, since the mean local FSW-MCL contact rate remained constant while MCLs are in the POM and thus the mean local MCL-FSW contact rate decreased (as already discussed in the previous section). Not surprisingly, however, prevalence in migrating high-risk males increased with increased risky behaviour (increased [double] the number of FSW partners that MCLs have and HIV prevalence in FSWs in the POM) in the migration site, as they became infected faster than non-migrating males, seeding infections in local FSWs and low-risk females, and increasing

peak and equilibrium prevalence in the overall population. Figure 6.2.10 shows what happens in migration scenario M.1 when each of these parameters were doubled (with all other migration parameters at B-CM values), in different risk groups, compared with when there are B-CM values for all parameters. Migrant males have substantial gains in 2004, peak and equilibrium prevalence from the B-CM scenario with twice the mean FSW POM partners (2004: ~10%; peak, equilibrium: ~8%) rather than twice the FSW POM HIV prevalence (2004, peak, equilibrium: ~5%), but the effect of twice each of these parameters on the non-migrant male and overall population is negligibly different.

Figure 6.2.10. Model HIV prevalence in different risk groups in the base-case (B-CM and with either double the mean FSW-MCL contact rate or the 2004 FSW prevalence in the POM).



The effect of doubling the migration-associated risk behaviour in the POM caused a surprisingly small effect on overall HIV prevalence, however, considering that doubling the mean FSW-MCL contact rate (mean: 121 per year to 242 per year or ~10 to 20 per month) and HIV prevalence in the POM (35% to 70% in 2004) was quite a substantial increase. The epidemic in low-risk males and females did not reach over 0.6%, even with increased risk in the POM for either of the two MCL migration scenarios.

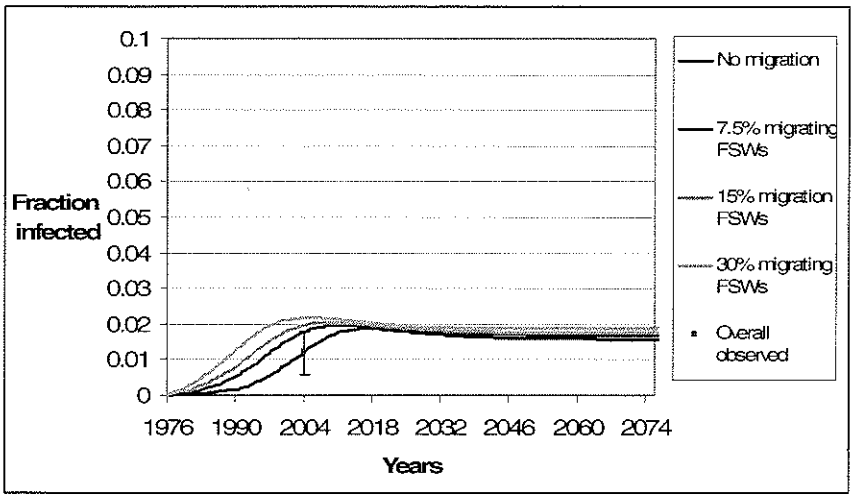
This is likely because of the low numbers of low-risk partners assumed (~one per sexually active lifetime) and low mixing between low-risk females and MCLs, as well as the size of the migrating client population being a small proportion of the total population (30% migrating males of 11.4% lifetime clients = 3.4% total migrating lifetime clients; 30% of 8.8% MCLs in 2004, assuming 20 years duration of MCRB, thus, 2.6% migrating MCLs in 2004).

Migrating FSW scenarios (F.1 and F.2)

FSW migration had a larger effect on overall model HIV prevalence than MCL migration, insofar as doubling each migration parameter alone brought 2004 model prevalence to higher values than with male migration, when compared with the base-case scenario in the absence of migration. FSW size had the largest effect in both scenarios: with double the FSW size investigated (30%) in F.1 (constant HIV prevalence in MCLs in the POM), 2004 model prevalence increased to 2.2% (B-CM: 2.0%, B-CII: 1.2%) and in F.2 (logistic increasing HIV prevalence in MCLs in the POM), 2004 model prevalence increased to 1.8% (B-CM: 1.5%, B-CII: 1.2%). This happened even though when FSW size was varied, there was no increase in migration-associated risk behaviour (the same mean MCL-FSW contact rate and HIV prevalence in MCLs in the place of origin as in the POM). Figure 6.2.11 shows how overall model HIV prevalence varied while migrating FSW size increased in F.1. FSW size caused greater increases than MCL size although only half as many FSWs migrated as MCLs because (as discussed above) we assumed that the male demand for sex remains the same in the place of origin when migrating FSWs were away, resulting in higher number of average local client contacts per FSW in Taluka A while migrating FSWs were away (total high-risk partnerships remain the same). As discussed in the previous section, the gains in overall model prevalence early in the epidemic with larger FSW size and duration of time away in F.1 were due to the simple assumption of

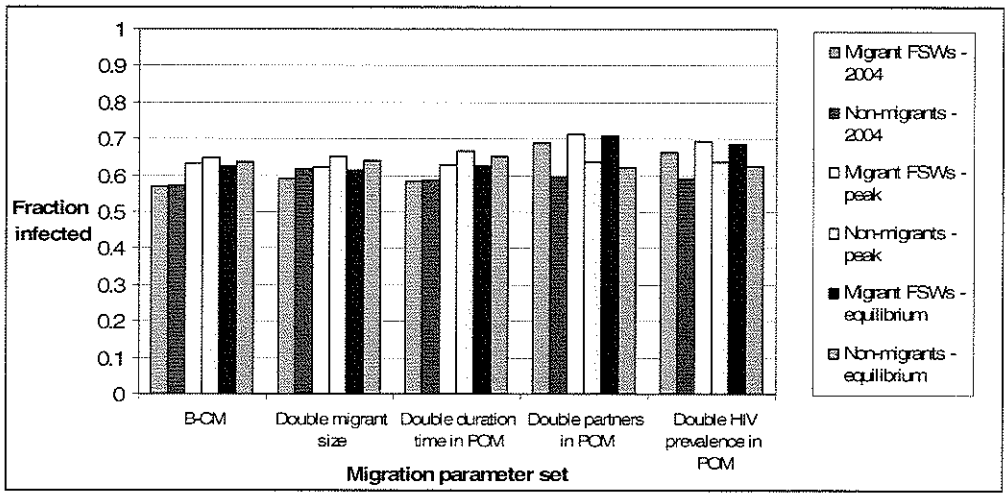
constant HIV prevalence in MCLs in the POM (17%), which was higher than local MCL prevalence early in the epidemic. FSW migration again had a smaller impact with a more realistic, logistic HIV prevalence curve in MCLs in the POM (B-CM: 17% in 2004; 40% at equilibrium) as each migration parameter increased (Figures 6.2.12-6.2.14; Table A2.4).

Figure 6.2.11. Overall model HIV prevalence as migrating FSW size increased, with constant HIV prevalence in MCLs in the POM (F.1)



Only when there were increases in migration-associated risk behaviour, however, did long-term migrating FSW model HIV prevalence increase above that in non-migrating FSWs. For example, in F.1, when the migrant FSW size was increased to its highest value (30%, B-CM: 15%) with all other parameters at B-CM values, 2004, peak and equilibrium model prevalence were 59.0%, 62.1% and 61.2% in migrating FSWs and 61.9%, 65.2% and 63.9% in non-migrant FSWs; when the mean MCL-FSW contact rate in the POM was doubled compared to the local MCL-FSW contact rate, 2004, peak and equilibrium model prevalence were 68.9%, 71.3% and 70.9% in migrating FSWs and 59.5%, 63.5% and 62.3% in non-migrant FSWs. Figure 6.2.12 shows 2004, peak and equilibrium model HIV prevalence in both migrating and non-migrating FSWs when each migration parameter is doubled in F.1. Similar results were found for F.2.

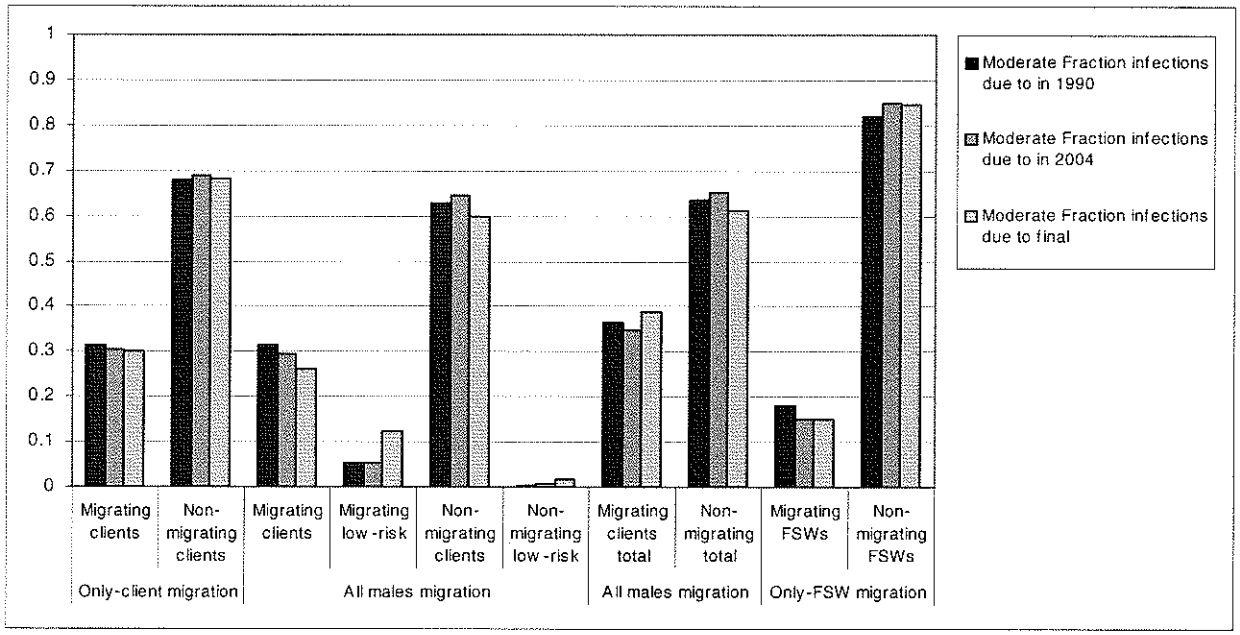
Figure 6.2.12. Model HIV prevalence in migrating and non-migrating FSWs in the B-CM and with double the B-CM of each migration parameter (all others held at B-CM values)



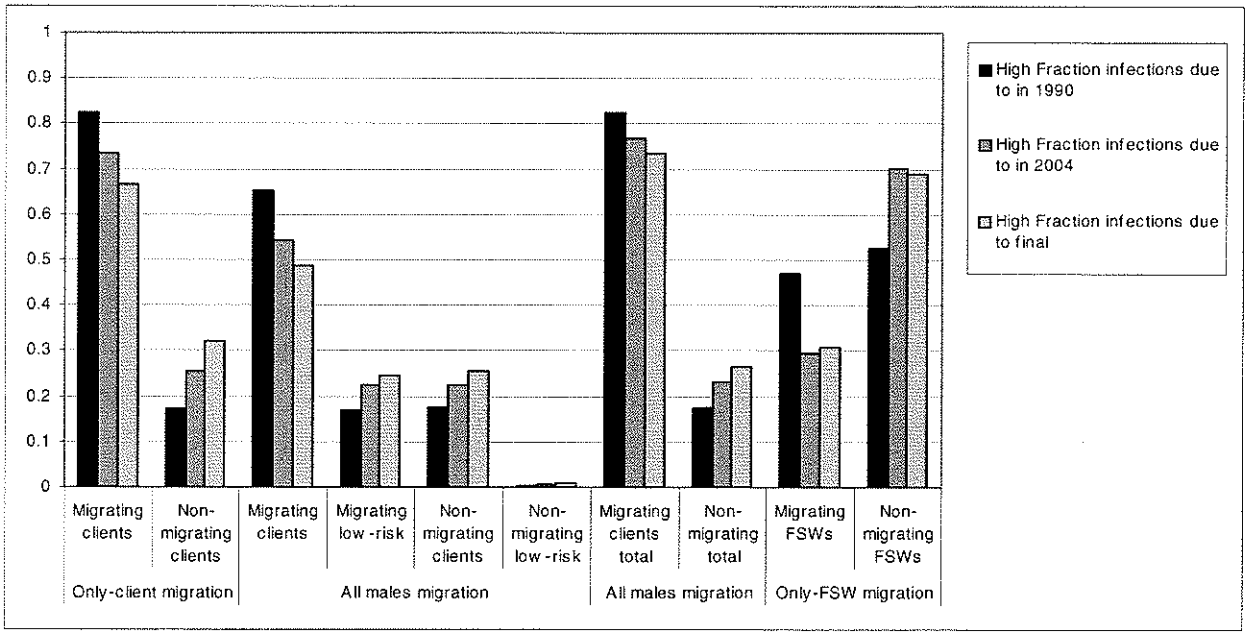
Fraction of new infections acquired and transmitted

We used low, moderate and high migration (all migration parameters low, B-CM or high: Table 6.2.1), in scenarios M.2, M.3 and F.2 (Table 6.2.2) to see which population was causing and gaining the most infections and help direct prevention efforts at certain populations. With low or moderate migration, the fraction of new infections transmitted by migrating populations (when in the place of origin) was about the same as their population sizes – what we would expect if migrating and non-migrating populations had the same likelihood of passing on infection (Figure 6.2.13 for moderate migration). However, with high migration, since migration-associated risk behaviour was higher in the POM than in the place of origin, the fraction of new infections transmitted by migrating groups increased above their population sizes, particularly early in the epidemic (Figure 6.2.14). With high migration in each scenario, migrating clients and FSWs transmitted more infections earlier in the epidemic compared with non-migrating groups, and this trend reversed later in the epidemic. Interestingly in scenario M.3, low-risk male clients did not follow the same trend as clients – low-risk male clients transmitted more infections later in the epidemic, just like non-migrating low-risk males.

Figures 6.2.13. Fraction of new infections due to migrant/ non-migrant high-risk groups (moderate migration)



Figures 6.2.14. Fraction of new infections due to migrant/ non-migrant high-risk groups (high migration)



Although the fraction of all new yearly infections acquired in migrating populations (when in their place of origin) was about the same as their population sizes with low or moderate

migration (Figure 6.2.15), with high migration the fraction of new infections found in migrating groups was lower than their population sizes (fraction of the total population).

Figure 6.2.15. Fraction of new infections found in high-risk groups (moderate migration)

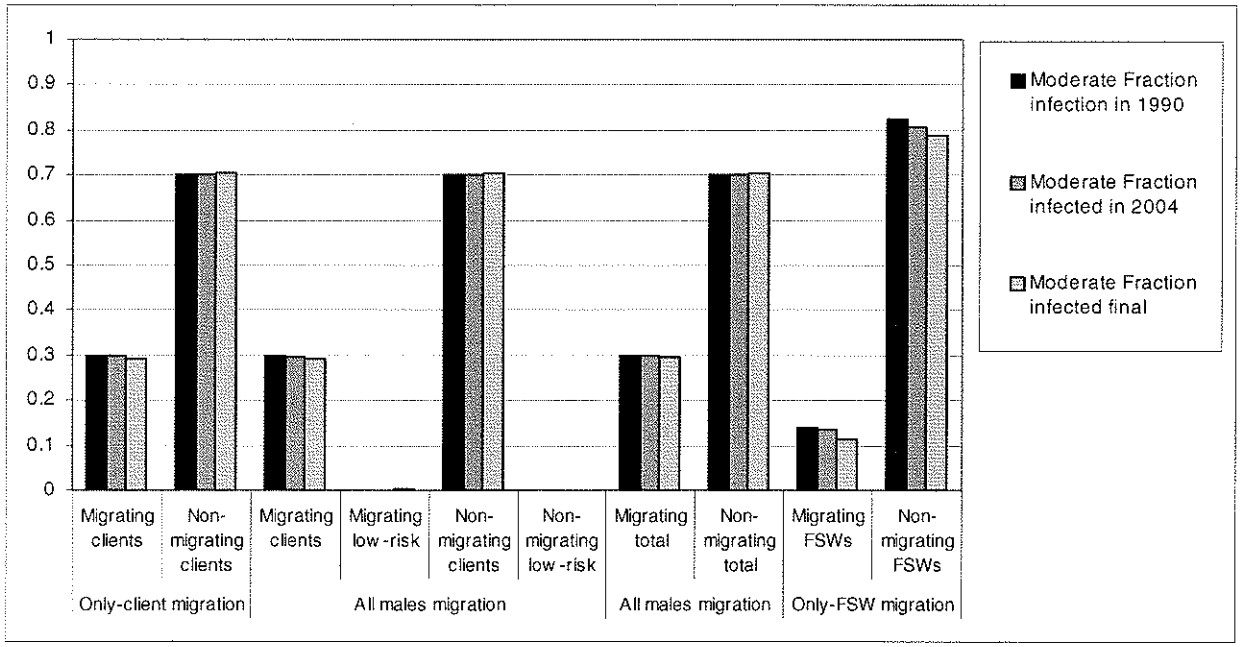
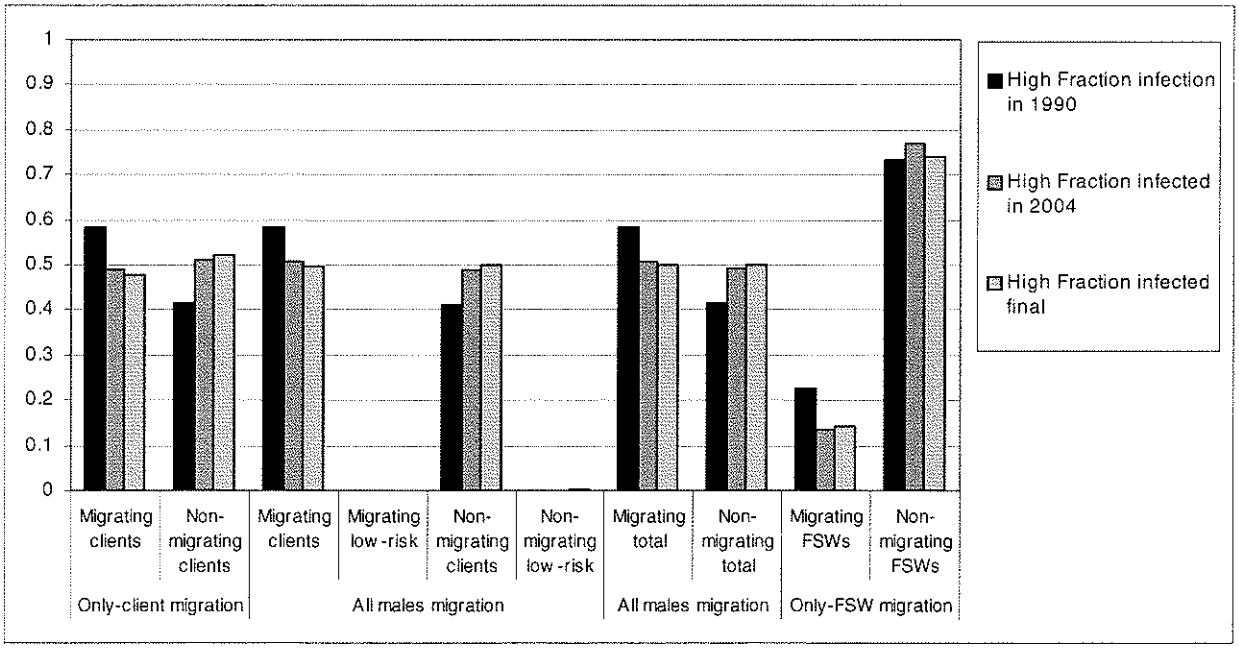


Figure 6.2.16. Fraction of new infections found in high-risk groups (high migration)



As the epidemic progressed (from 1990 to 2004 to final), for low and moderate migration, the fraction of new infections increased and was higher in males (about 58% to 68%),

while it decreased in females (about 42% to 32%) in each migration scenario. However, with high migration, in the two scenarios in which clients or clients and low-risk clients migrate, the fraction of new infections in males was lower than in low or moderate migration while still increasing over time (only-clients: 42% to 52%; all-males: 38% to 45%). In contrast, when only FSWs migrate, the fraction of new infections in males was higher than in low or moderate migration (only-FSWs: 66% to 73%) and still higher than in females (32% to 27%).

Effects of a simple condom intervention program

In each migration scenario, with moderate migration (B-CM values, Table 6.2.1), introducing a simple condom prevention program into only migrating high-risk populations had a smaller impact than when condoms were introduced into only non-migrating groups. As condom use increased from 0% to 75% in migrating groups in scenarios M.2, M.3 and F.2, low-risk female prevalence in 2020 decreased by 15.8%, 26.4% and 7.3% respectively; with increased migration-associated risk behaviour in the POM, low-risk female prevalence in 2020 decreased only slightly more than all migration parameters at B-CM values (17.8%, 33.7%, 9.7%). As condom use increased in only non-migrating groups, low-risk female prevalence in 2020 decreased by 58.9%, 47.7% and 62.2%. Figures 6.2.17 and 6.2.18 show what happened to low-risk female prevalence when a fraction of all males migrate (M.3), when condoms were introduced into only migrating high-risk groups (Figure 6.2.17) or non-migrating high-risk groups (Figure 6.2.18) (not shown for M.2 or F.2). Similar results were seen in local FSWs (Figures A2.6 and A2.7, Appendix 2). Only when condoms were introduced into both migrating and non-migrating groups did the epidemic curve decrease consistently over time at 75% condom use for local low-risk females and FSWs: as condom use increased, low-risk female prevalence in 2020 decreased by 80.4%, 77.4% and 71.4% (Figure 6.2.19). Figure A2.8 shows this in FSWs (Appendix 2).

Figure 6.2.17. Low-risk female prevalence with varied migrant-only condom use (M.3)

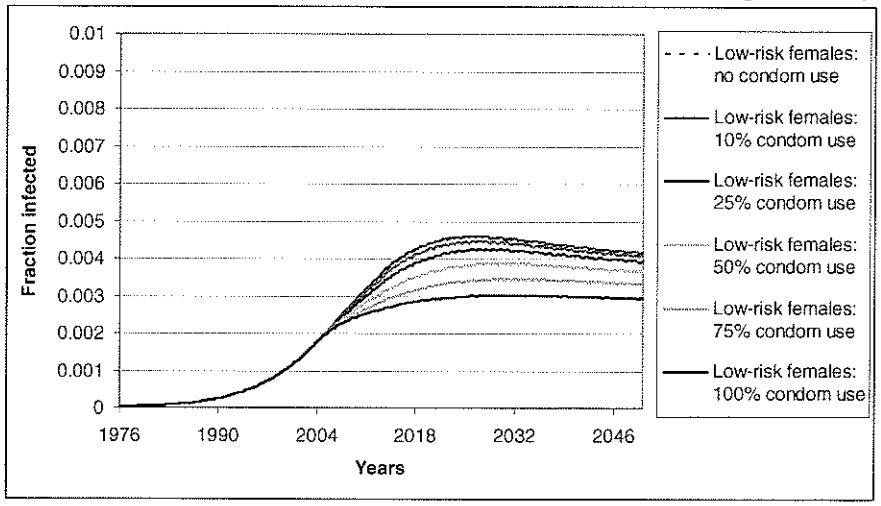


Figure 6.2.18. Low-risk female prevalence with varied non-migrant-only condom use (M.3)

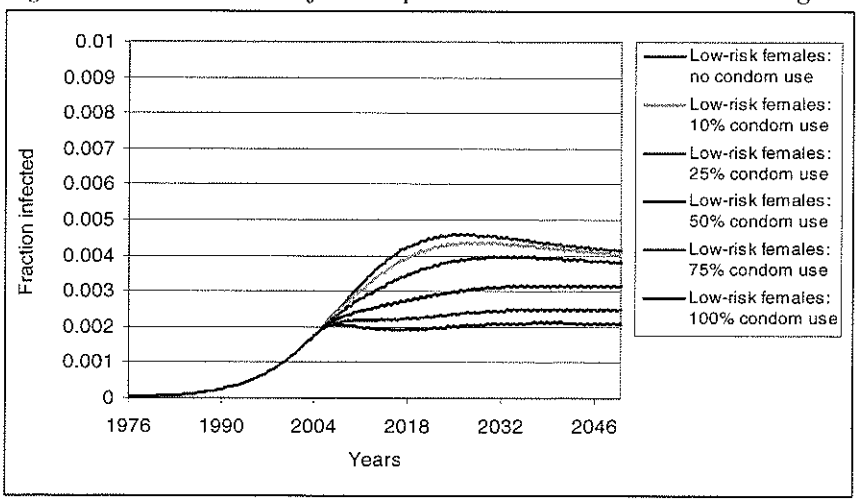
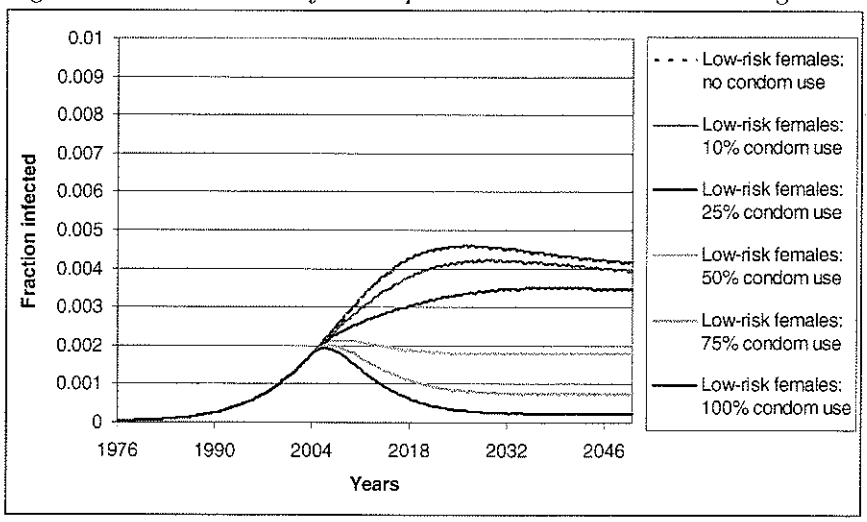


Figure 6.2.19. Low-risk female prevalence with varied all high-risk group condom use (M.3)



CHAPTER SEVEN – UNCERTAINTY ANALYSIS

We conducted uncertainty analysis on duration of FSW and MCRB, mean numbers of low-risk partners, transmission probabilities, and population birth rate. The main results are summarized:

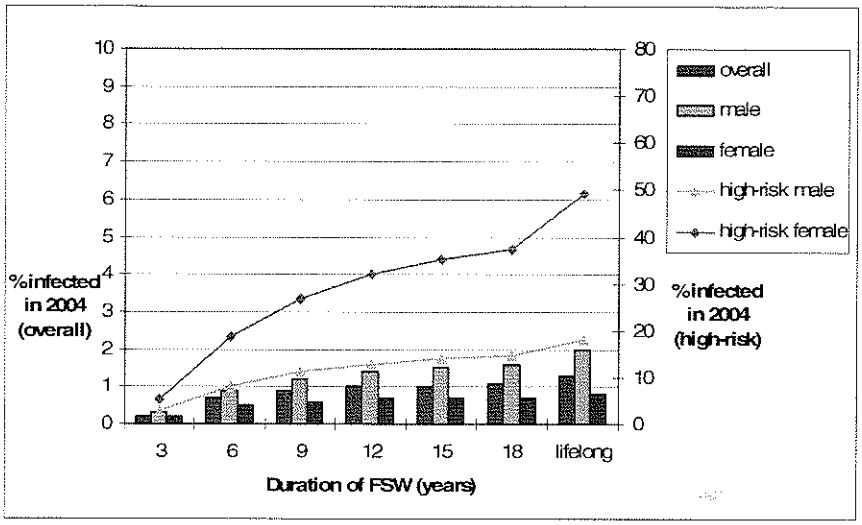
- Within a reasonable range of values for the duration of female sex work (6 to 15 years, base-case value=9 years), there was only a change in 2004 model HIV prevalence of 0.4% in Talukas A and B, 0.5% in Taluka C. A shorter duration of FSW caused lower 2004 and peak model HIV prevalence, and a negligibly different equilibrium model HIV prevalence.
- FSW and client 2004 model HIV prevalence (and also the overall population) was sensitive to the duration of male client risk behaviour since a shorter duration (for the same lifetime client size) resulted in smaller 2004 client size and much larger (less realistic) mean numbers of FSW partners per client; a longer duration of MCRB helped the model (with already high high-risk prevalence in 2004) fit to observed 2004 high-risk HIV prevalence.
- Overall 2004 model HIV prevalence was less sensitive in 2004 when the mean low-risk partner change rate varied from five to 20 per sexually active lifetime than at peak and equilibrium (increased by 0.8%, 1.8% and 1.9% in Taluka A respectively). This was mainly due to substantial late-epidemic low-risk epidemic increases. Low-risk (and overall) model HIV prevalence was increasingly sensitive with higher numbers of low-risk partners.
- Overall 2004 model HIV prevalence was very sensitive (particularly in the later stages of the epidemic), to the four transmission probability sets developed in this study, when there were 10 low-risk partners per sexually active lifetime, and much less when there was only one.
- Varying the overall birth rate through a wide range (half-to-double the base-case estimated value) caused negligible variations in 2004, peak and equilibrium model HIV prevalence.

- **Sensitivity to duration of FSW**

In the overall population, shorter duration of FSW (decreased from 18 to 4.5 years, B-CI: lifelong, B-CII: 9 years) resulted in lower 2004, peak and equilibrium prevalence (the latter two reached later), since FSWs had less time to become infected by MCLs, thus lowering their HIV prevalence and the entire population's (for this analysis we assumed a lifelong duration of MCRB). In Taluka A, for example, 2004 model HIV prevalence in the overall population decreased from 0.9% to 0.3%, while peak decreased from 1.8% to 1.6% and equilibrium remained the same at 1.5% (Tables A2.2 and A2.3 in Appendix 2 show these results for Talukas B and C). For all Talukas, the highest overall HIV prevalence in 2004 was with lifelong duration of FSW (B-C: 1.3% in Taluka A, 1.7% in Taluka B and 4.2% in Taluka C).

Overall 2004 model HIV prevalence was less sensitive to duration of FSW with higher values for this parameter. Varying duration of FSW from 6 to 15 years in only resulted in a change in 2004 model HIV prevalence of 0.4% in Taluka A, 0.4% in Taluka B and 0.5% in Taluka C, so we can be confident that near the B-CII value for duration of FSW (9 years), overall model prevalence in each Taluka is not sensitive to this parameter. However, larger changes in 2004 model prevalence occurred when duration of FSW was short – in only a three-year period, from three to six years, overall prevalence increased about 0.5% in Taluka A (Figure 7.1) and Taluka B and 1.2% in Taluka C. If duration of FSW is lower than we estimate to be in each Taluka, then our base-case model prevalence in 2004 and peak may be substantially lower than we expect it to. However, equilibrium model prevalence would not change much regardless of the duration of FSW.

Figure 7.1. Relative increase in 2004 model HIV prevalence in Taluka A in different risk groups by duration of FSW

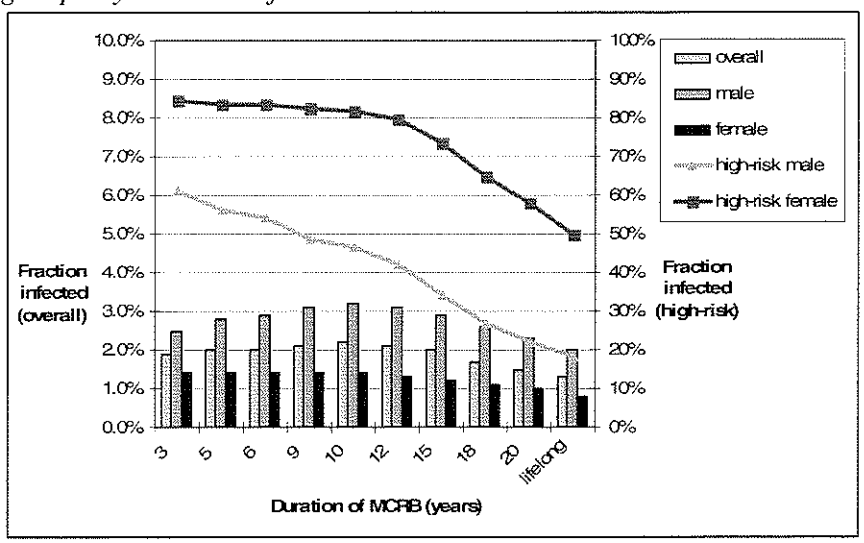


- **Sensitivity to duration of MCRB**

In contrast to the effects of decreasing duration of FSW, a shorter duration of MCRB resulted in higher 2004 and peak HIV prevalence in the overall, male and female populations, which was reached earlier, since a shorter duration of MCRB caused a smaller 2004 MCL size and higher mean FSW-MCL contact rate (assuming that partnerships remain constant when MCL size varies). The yearly MCL population size was dependent on the duration of MCRB – when the duration of MCRB decreased, the 2004 size of the MCL population decreased. For example, in Taluka A, when the duration of MCRB was decreased from double to half its B-CII value (20 to 5 years; B-CII: 10 years; B-C: lifelong), yearly size of the MCL population decreased from 8.8% to 2.4% (B-CII: 4.6%; B-C: 10.8%) and overall model HIV prevalence in 2004 increased from 1.5% to 2.0% (with peak decreasing from 2.5% to 1.9% and equilibrium decreasing from 1.7% to 1.5%), shown in Figure 7.2. Similar changes were seen in Talukas B and C (Tables A2.2, A2.3 in Appendix 2) Since we assumed that the size of the FSW population and the average number of MCL partners per FSW remained the same, the number of FSW partners per MCL per year increased with decreasing MCL size, from 120 per year to 448 per year (B-CII: 229; B-C: 98).

Larger effects were seen earlier in the epidemic compared with later; equilibrium HIV prevalence increased only slightly, and was reached earlier. This happened because the effects of the smaller MCL size (and higher numbers of high-risk partners) were weaker later in the epidemic, as they were slightly offset by the effects of a shorter duration of MCRB – similar to duration of FSW, a shorter duration of MCRB would result in a shorter duration of time to become infected by FSWs, and fewer infections in this population if there weren't also a higher number of high-risk partners (which did not happen as duration of FSW is decreased).

Figure 7.2. Relative increase in 2004 overall model HIV prevalence in Taluka A in different risk groups by duration of MCRB

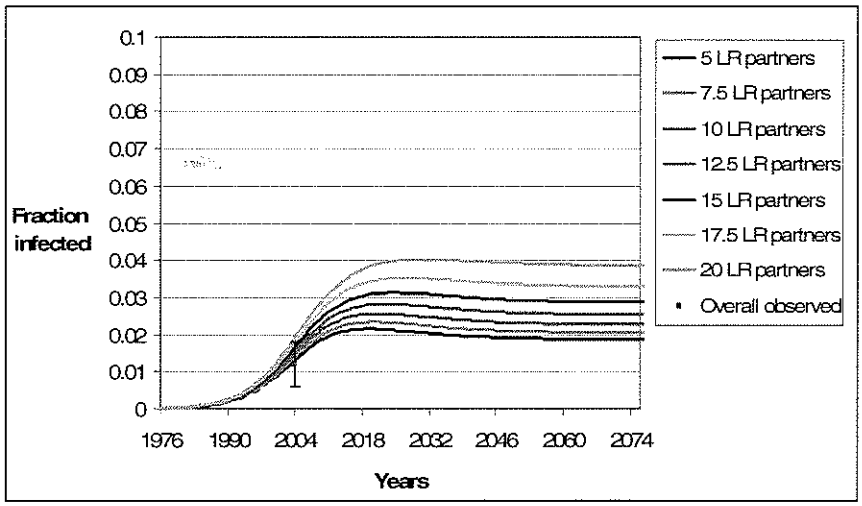


- **Sensitivity to average numbers of low-risk partners**

Overall model prevalence was more sensitive to the mean numbers of low-risk partners at peak and equilibrium compared with 2004. For example, increasing the mean LRM partners per LRF per sexually active lifetime from half-to-double the B-CII value in Taluka A (5 to 20, B-CII: 10) resulted in higher overall 2004 HIV prevalence (1.3% to 2.0%, B-CII: 1.5%), peak prevalence (2.2% to 4.0%, B-CII: 2.6%), reached later (2019 to 2029, B-CII: 2020) and equilibrium prevalence (1.9% to 3.8%, B-CII: 2.3%), reached later (2061 to 2087, B-CII: 2068) (Figure 7.3).

Additionally, incremental increases in low-risk partners increased slightly with higher numbers of low-risk partners – i.e. in Taluka A, from 5 to 7.5 partners, 2004, peak and equilibrium prevalence increased from 1.3% to 1.4%, 2.2% to 2.3% and 1.9% to 2.1% (differences of 0.1%, 0.1% and 0.2%), but from 17.5 to 20 partners, these values increased from 1.8% to 2.0%, 3.5% to 4.0% and 3.3% to 3.8% (differences of 0.2%, 0.5% and 0.5% respectively).

Figure 7.3. Overall prevalence in Taluka A as the number of LRM partners per LRF increased



The patterns seen in overall prevalence are influenced mostly by low-risk male and female prevalence and much less by high-risk male and female prevalence, which was not sensitive to increases in the numbers of low-risk partners. In Taluka A, from 5 – 20 low-risk partners, low-risk male peak prevalence increased significantly and was reached later, from 0.7% in 2022 to 2.1% in 2036 (B-CII: 0.9% in 2025) and equilibrium prevalence increased from 0.6% in 2058 to 2.1% in 2104 (B-CII: 0.9% in 2065). Low-risk female peak and equilibrium prevalence also increased and was reached later (peak: 0.9% in 2020 to 3.3% in 2033, B-CII: 1.5% in 2023; equilibrium: 0.8% in 2067 to 3.2% in 2086, B-CII: 1.3% in 2084).

High-risk male peak prevalence increased negligibly and was reached at the same time, from 31.5% in 2028 to 32.8%% in 2028 (B-CII: 31.7% in 2028) and equilibrium prevalence increased from 30.6% in 2098 to 32.1% in 2093 (B-CII: 30.9% in 2083). High-risk female peak

prevalence increased from 64.4% (2029) to 65.9% (2030) (B-CII: 64.7% in 2029) and equilibrium prevalence increased from 63.4% (2084) to 65.0% (2100) (B-CII: 63.7% in 2085).

The number of LRF partners per LRM may explain some of the differences in 2004 overall observed HIV prevalence between the three Talukas, but it likely does not explain much if our model is accurate in timing of the epidemic (i.e. it is still increasing in 2004 in Bagalkot District). High values for the mean numbers of LRF partners per LRM were required to bring each Taluka to within range of the other Talukas' observed 2004 HIV prevalence. Taluka A required 25 (from 10) to reach 2.2%, within range of Taluka B's observed prevalence and 36 to reach 3.6%, within range of Taluka C's overall observed 2004 HIV prevalence. Taluka B required 30 (from 10) to reach within range of Taluka B's observed prevalence.

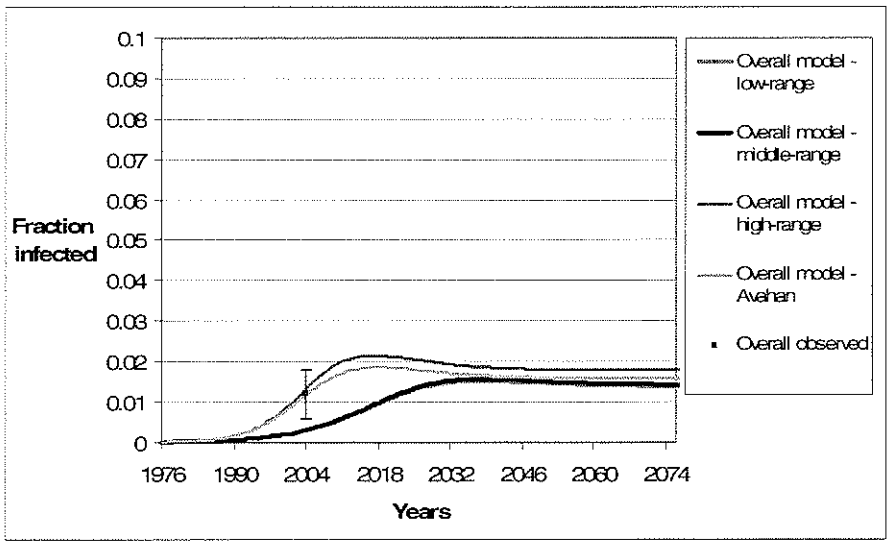
- **Sensitivity to transmission probabilities**

From a review of transmission probabilities (TPs) for HIV (Baggaley et al, 2005), we developed four sets of TPs for high- and low-risk HIV transmission. These are detailed in Table A1.1 in Appendix 1 and discussed further in Chapter Four: Model Development. To summarize, we termed the four sets: high-range, middle-range, low-range and base-case (B-C). Our B-C TP set was estimated from based on a resource-poor setting (Uganda) in per-contact partnerships where there were no reported risk factors for increased transmission. Our B-C TPs allowed us to fit each model to observed overall, female and male 2004 HIV prevalence in each Taluka. In this section, we show the different results in two scenarios: when we assumed one low-risk female partner per low-risk male per year (used in our first base-case scenario [B-CI] and the migration scenarios) and ten low-risk female partners per low-risk male per year (used in second base-case scenario [B-CII], in which we assessed the impact of parameters in this study).

- **B-CI (one low-risk female partner per low-risk male per year)**

In all three Talukas, there was negligible difference in 2004 overall model HIV prevalence using the B-C TP set and the high-range TP set (peak and equilibrium was slightly higher for the high-range TP set), and there was negligible difference in overall 2004, peak and equilibrium model HIV prevalence using the low-range and middle-range TP sets. However, there were noticeable differences between the high range/ B-C sets and the low/ middle range sets. Figure 7.4 shows how overall model HIV prevalence varies according to which TP set used in Taluka A (Figures A3.1 and A3.2 in Appendix 3 show this in Talukas B and C).

Figure 7.4. Overall model prevalence in Taluka A for different transmission probability sets with B-CI values for low-risk partners (one low-risk female partner per male per year)



In Taluka A for the overall, male and female populations, 2004 model prevalence was lower for the low- and middle-range TP sets (0.3%, 0.4%, 0.2%) compared with high-range (1.3%, 1.8%, 0.9%) and B-C (1.2%, 1.6%, 0.8%) TP sets respectively. Gains in model HIV prevalence were due to increased epidemics in both low- and high-risk populations. In 2004 and equilibrium, model prevalence was lower for the low-range TP set (2004: LRM: 0.07%; LRF: 0.06%; MCL: 3.9%; FSW: 10.1%; equilibrium: LRM: 0.5%; LRF: 0.4%; MCL: 25.9%; FSW: 59.6%) and middle-range TP sets (2004: LRM: 0.07%; LRF: 0.06%; MCL: 3.9%; FSW: 10.2%;

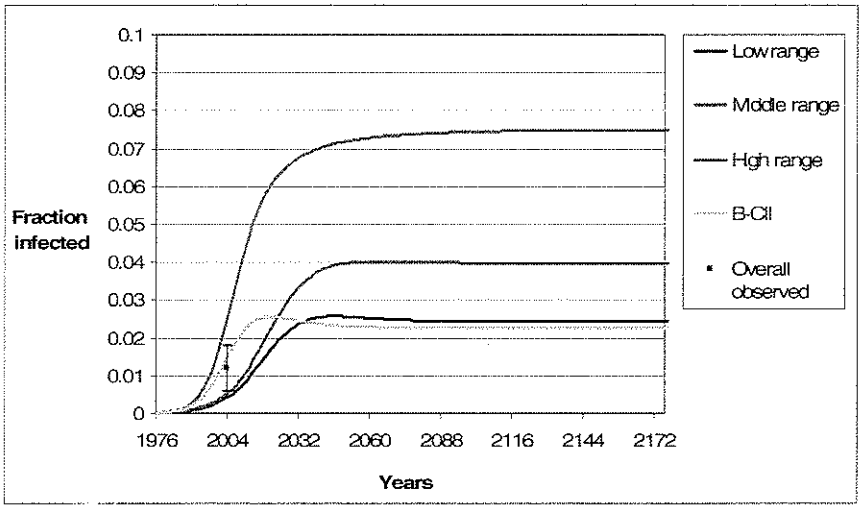
equilibrium: LRM: 0.4%; LRF: 0.9%; MCL: 25.9%; FSW: 59.7%) compared with the high-range (2004: LRM: 0.3%; LRF: 0.3%; MCL: 17.6%; FSW: 39.7%; equilibrium: LRM: 0.5%; LRF: 0.6%; MCL: 34.1%; FSW: 67.7%) and B-C TP set (2004: LRM: 0.3%; LRF: 0.2%; MCL: 15.7%; FSW: 37.4%; equilibrium: LRM: 0.5%; LRF: 0.4%; MCL: 30.59%; FSW: 65.4%).

For most of the TP estimates for risk/gender groups, the B-C estimates were lower than or very similar to the middle-range estimates, although HIV prevalence was higher at 2004, peak and equilibrium for the B-C set (Figure 7.4). However, for the initial stage of transmission for both M-F and F-M, the B-C estimates are higher (M-F B-C: 0.0052; middle-range: 0.0031, F-M B-C: 0.0031; middle-range: 0.0015). This may indicate that the first stage of transmission in high-risk groups and the TP values associated with this first stage are important in the transmission dynamics of HIV. The first stage may be the shortest stage, but almost doubling the TPs in the first stage for high-risk transmission has this large effect on HIV prevalence in 2004 in all Talukas (and less at peak and equilibrium).

- **B-CII (ten low-risk female partner per low-risk male per year)**

In all three Talukas, when we assume that there are a higher number of low-risk female partners per male per year (10), there is a much larger difference in the model epidemic curves with the four different model parameter sets, compared with when we assume a lower number (1). Figure 7.5 shows how overall model HIV prevalence varied according to which TP set used in Taluka A (Figures A3.3 and A3.4 in Appendix 3 show this in Talukas B and C).

Figure 7.5. Overall model prevalence in Taluka A for different transmission probability sets with B-CII values for low-risk partners (ten low-risk female partners per male per year)



In Taluka A for the overall, male and female populations, 2004 model prevalence was still lower for the low-range (0.4%, 0.5%, 0.4%) and middle-range TP sets (0.5%, 0.5%, 0.5%) compared with the high-range (2.5%, 2.6%, 2.4%) and B-C (1.5%, 1.8%, 1.3%) TP sets respectively (only fitting to observed prevalence in the B-C scenario). Of the four parameter sets, only B-C values produced overall, male and female model prevalence in 2004 that was within range of observed values. Equilibrium model HIV prevalence in the overall populations varied substantially, with B-C TP set the smallest in the overall, male and female populations (2.3%, 2.8%, 1.9%), with low-range (2.4%, 2.7%, 2.2%) slightly higher and middle-range (4.0%, 3.9%, 4.0%) and high-range (7.5%, 7.0%, 7.8%) being much higher. Gains in model HIV prevalence were due to increased epidemics in both low- and high-risk groups. In 2004 and equilibrium, model prevalence was lower for the low-range (2004: LRM: 0.1%; LRF: 0.2%; MCL: 4.1%; FSW: 10.2%; equilibrium: LRM: 1.1%; LRF: 1.8%; MCL: 26.3%; FSW: 58.2%) and middle-range TP sets (2004: LRM: 0.2%; LRF: 0.4%; MCL: 4.2%; FSW: 10.6%; equilibrium: LRM: 2.3%; LRF: 3.6%; MCL: 27.7%; FSW: 59.8%) compared with high-range (2004: LRM: 0.9%; LRF: 1.8%; MCL: 19.6%; FSW: 41.9%; equilibrium: LRM: 5.0%; LRF: 7.3%; MCL: 37.8%;

FSW: 69.4%) and B-C TP set (2004: LRM: 0.4%; LRF: 0.7%; MCL: 16.5%; FSW: 37.7%; equilibrium: LRM: 0.9%; LRF: 1.3%; MCL: 30.9%; FSW: 63.7%).

When each TP set within this B-CII scenario is compared with the B-CI scenario (one LRF partner per LRM), it is clear that the gains were from low-risk groups rather than high-risk. Figure 7.6 illustrates how low-risk male and female 2004 model HIV prevalence increased across scenarios in Taluka A only; Figure 7.7 shows how MCL and FSW 2004 model HIV prevalence increased across scenarios. While 2004 model HIV prevalence in low-risk groups (and thus overall groups) is more sensitive to the different TP sets with higher number of low-risk partners, equilibrium model HIV prevalence is more sensitive in low-risk groups (Figure 7.8) than high-risk groups (Figure 7.9).

Figure 7.6. Low-risk model prevalence in 2004 with each different TP set (low-range, middle-range, high-range and B-C) under different scenario assumptions (B-CI and B-CII)

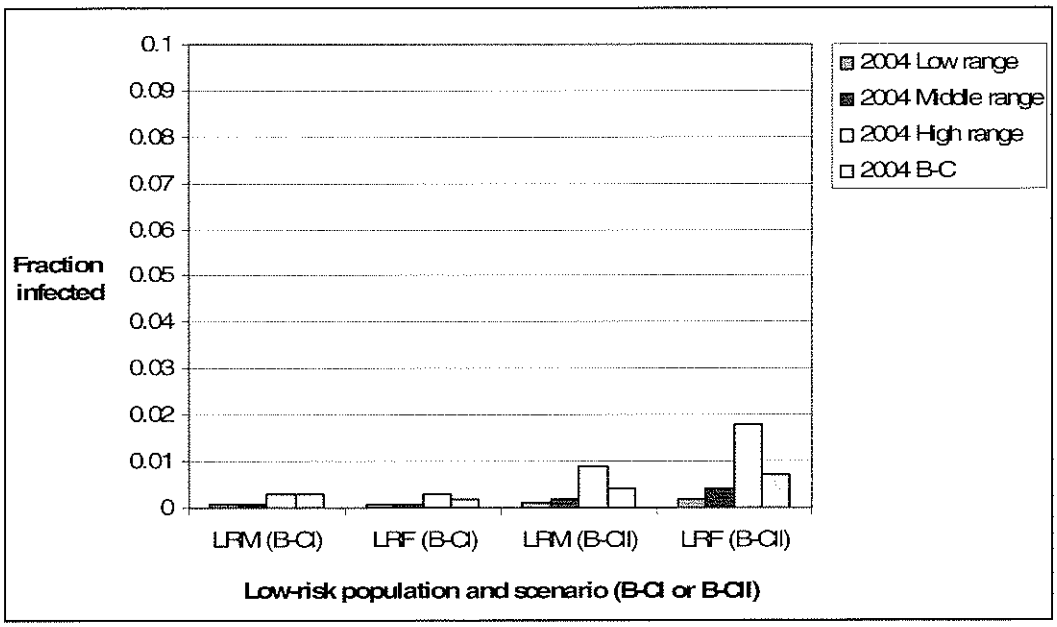


Figure 7.7. High-risk model prevalence in 2004 with each different TP set (low-range, middle-range, high-range and B-C) under different scenario assumptions (B-CI and B-CII)

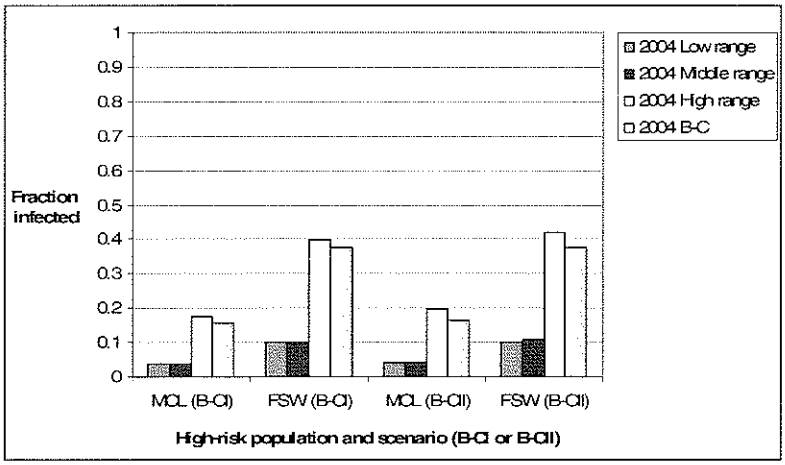


Figure 7.8. Low-risk model prevalence in equilibrium with each different TP set (low-range, middle-range, high-range and B-C) under different scenario assumptions (B-CI and B-CII)

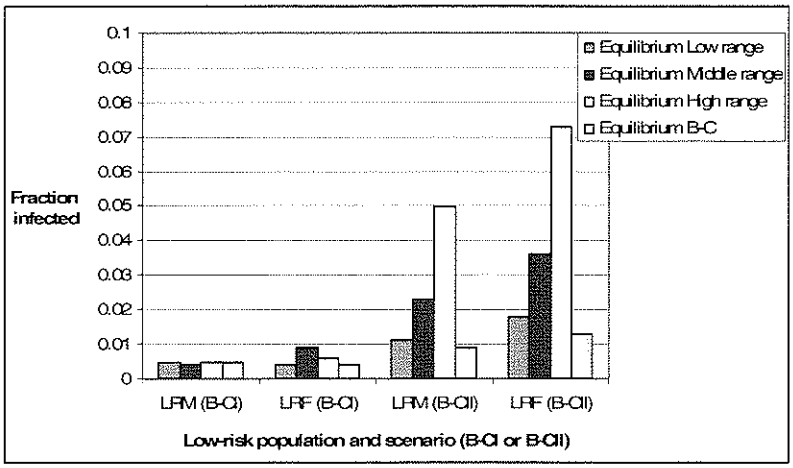
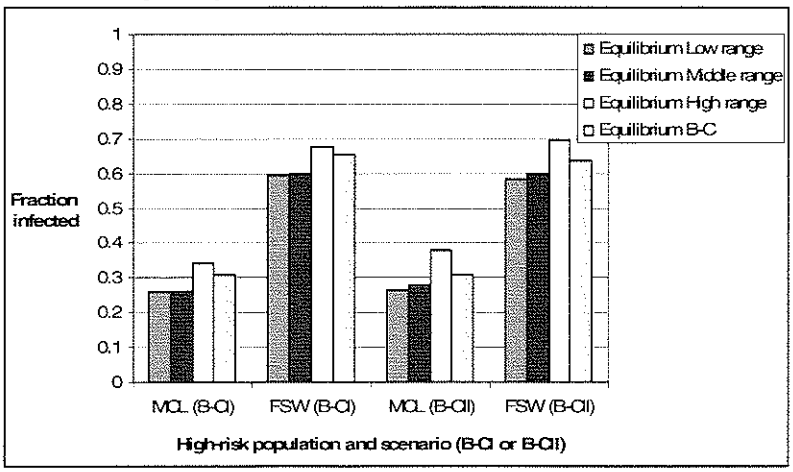


Figure 7.9. High-risk model prevalence in equilibrium with each different TP set (low-range, middle-range, high-range and B-C) under different scenario assumptions (B-CI and B-CII)



In this analysis of different TP sets using ten LRF partners per LRM, we find that only in the first infected stage are the B-C low-risk transmission probabilities (from both M-F and F-M) higher than the low-range TP set. The rest of the stages of infection in the low-range set and all of the stages of infection in the middle- and high-range set are higher than in the B-C TP set (Table A1.1, Appendix 1).

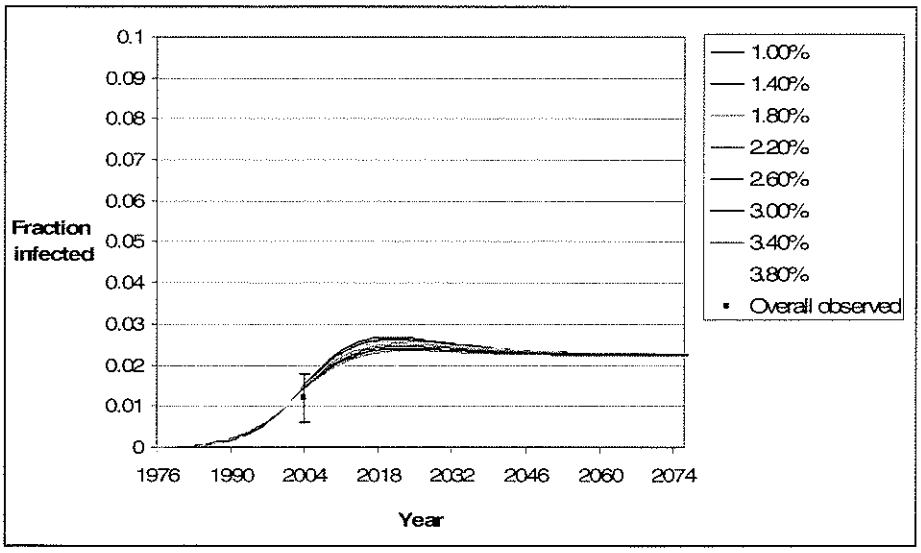
Our results suggest that when there are increased numbers of low-risk partners (increased low-risk sexual activity) in a population, then low-risk TPs, particularly the later stages of infection, become more important in the modeled HIV epidemic. Low-risk TPs play a larger role in long-term prevalence in our B-CII analysis (ten LR partners) while high-risk TPs affect short-term model prevalence in our B-CI analysis (one LR partner). These results are supported by our previous analysis of low-risk partners, where we found that increasing the mean number of low-risk partners in a population by the same increments could cause model prevalence to vary more with increased value for low-risk partners, particularly long-term model prevalence (see Figure 7.3). In addition, we found that increasing high-risk partners concentrated the epidemics in high-risk groups, causing peak and equilibrium to be reached higher and earlier in high-risk and overall populations (see Chapter Six: Results, sub-section 6.1.1, scenario PV1).

- **Sensitivity to population growth rate**

We assumed an initial 1.9% population growth rate that was estimated from India Census data for the state of Karnataka (1981 – 1991). We were not able to find the population growth rate for 1976, the year that our epidemic is assumed to begin. The actual growth rate in Bagalkot District in 1976 (without AIDS deaths) may be higher or lower than 1.9%. We varied the population growth rate from half-to-double the base-case values (1.0% to 3.8%).

Negligible changes in HIV prevalence were seen in 2004 for all populations as the population growth rate was increased through these values (overall model 2004 HIV prevalence: decreased by 0.2% in Talukas A and B and 0.5% in Taluka C). Figure 7.10 shows how the epidemic curve changed in the overall population in Taluka A as population growth rate varied. Within each population, larger changes were seen in peak and equilibrium prevalence, with a larger population growth rate causing a smaller peak and equilibrium prevalence in both low- and high-risk groups (Figure A3.5 in Appendix 3 shows how peak and equilibrium model HIV prevalence varied in high-risk groups for Taluka A). Since the model predicted an epidemic that was still increasing in 2004 and had not reached peak or equilibrium in any Taluka, the population growth rate value did not affect model fit to observed prevalence and likely does not contribute to the heterogeneity in 2004 HIV prevalence in each Taluka. If the epidemic is actually at the peak or equilibrium, population growth rate may contribute slightly to heterogeneity in HIV prevalence. However, it seems unlikely that population growth rate varies substantially between Talukas due to their close proximity and demographic similarities.

Figure 7.10. Overall model prevalence with varying population growth rate



CHAPTER EIGHT – DISCUSSION AND CONCLUSIONS

8.1. Explaining heterogeneity

- **Parameters with different values in each Taluka**

We conducted a univariate analysis on the sexual behaviour factors for which we had different date in each Taluka, adjusting each Taluka's value in turn to another's value and measuring the change in 2004 overall model HIV prevalence.

We obtained interesting results, which showed that the different observed values for the mean FSW-client contact rates, FSW sizes and client sizes in each Taluka were potentially all important parameters in explaining the different observed HIV prevalence in each Taluka, when the total high-risk partnerships were allowed to vary when these parameters varied. It is intuitive that HIV prevalence will increase if total high-risk partnerships increase, since a larger FSW-client contact rate is therefore accompanied by a larger client-FSW contact rate, larger FSW size is accompanied by increased FSW-client contact rate and larger client size is accompanied by increased client-FSW contact rate. However, this analysis showed that, providing total high-risk partnerships vary, the relative differences observed in these parameters between the three Talukas were large enough to explain a substantial fraction of the differences in observed 2004 HIV prevalence between the three Talukas.

High-risk behaviour was also associated with HIV infection in a general population study in Bagalkot District. For example, women were more likely to be infected with HIV if they had received money for sex (Becker et al., 2007). Other factors that might predispose women to exploitation and becoming involved in the sex trade, such as being of a lower caste (Hindu religion) and being widowed or divorced, was also significantly associated with HIV infection in

both women and men (Becker et al., 2007). Men who had experienced symptoms of sexually transmitted infections (indicating higher-risk behaviour) were significantly more likely to be HIV-infected than men who did not, and men had higher prevalence than women (though this wasn't significant), particularly the 25-39 years old age group. Both men and women who reported more than one sex partner had significantly higher levels of HIV infection, although significantly more men than women reported having multiple sex partners (Becker et al., 2007).

Our conclusions that: "If Taluka "i" had Taluka "j's" FSW size, MCL size or mean FSW-client contact rate, then Taluka "i" would have had HIV prevalence similar to Taluka "j's" depend on the assumption that total high-risk partnerships vary – i.e. an increase in risky sexual behaviour in one high-risk population (larger FSW or client size, higher volume of high-risk partners in one group) would also be accompanied by an increase in risky behaviour in the other high-risk population. These patterns are indicated by the observed data, in which the Taluka with the highest observed HIV prevalence (Taluka C) also had the largest proportion of FSWs and lifetime clients, and the Taluka with the lowest observed HIV prevalence had the smallest proportion of FSWs and lifetime clients in their overall population (Taluka A). Because we assume the same mean client-FSW contact rate in 2004 in each Taluka, Taluka C also had the highest mean FSW-client contact rate and Taluka A had the lowest.

In addition, anecdotal evidence suggests that some villages in Bagalkot District are "sex work villages", where the volume of high-risk activity is higher than in other areas (Bagalkot District site visit, 2005). FSWs may travel outside their home villages to these nearby "sex work villages" to avoid letting their families know they practice sex work. In this case, since these areas would be known as common places of gathering for FSWs, it might also encourage higher levels of client behaviour. Taluka C, with the highest fraction (and number) of FSWs than other

Talukas measured in Bagalkot District, might have a higher number of sex work villages. In support of this, Taluka C has the highest number of villages with 10 or greater FSWs per village (40), followed by Talukas B (35) and A (11) (Blanchard et al., 2005). Taluka C also has the highest number of villages with 20 or more FSWs per village (36, or 47%), with Taluka B second (21, or 32%) and finally, Taluka A (11, or 11%). Little is known about how many men are clients of FSWs in each Taluka each year. In 2004, Taluka C had the highest fraction of men who report ever being a client (18.0%) compared with Taluka B (13.2%) and Taluka A (11.4%) (Ramesh, 2005a), but because of Taluka B's larger population size, Taluka B has the highest number of men who report ever being a client (~8800), with Taluka C slightly lower (~8500) and Taluka A the lowest (~5400).

Populations in Bagalkot District with a regular influx of long- or short-term migrants might have developed higher local levels of high-risk behaviour over time as well and higher rates of HIV infection. Studies regularly find that HIV prevalence is higher in migrants versus non-migrants. For example, one study in a rural area of Lesotho, South Africa, found that there was a high prevalence of HIV in the migrant labour force working on a dam (5.3%), but a much lower prevalence in villages surrounding the area (0.8%), which were not involved in the dam's construction, indicating the higher-risk behaviour was ongoing in the site of migration (Kravitz et al, 1995). In a study of migrant and non-migrant men in South Africa, migrant men were more likely to have regular partners outside of their place of origin, mostly at the place of migration, and more likely to have casual sex partners and higher HIV prevalence than non-migrant men (Lurie et al, 2003). Talukas B and C are known as more fertile, wealthier areas, with more employment opportunities, possibly drawing a large male migrant population from nearby Talukas (sugarcane fields, construction, etc.) (ICHAP, 2003). In-migration was not examined in

this study, but may be an important factor in Talukas with higher observed HIV prevalence if an influx of migrants causes increased volume of sex work or population of sex workers locally.

In transactional sex, women provide a service that men purchase and thus the male demand for sex work seems to be a likely factor in determining a population's total volume of sex work, particularly in populations such as India where the status of women is low and many women participate in the sex work trade for financial reasons, and sometimes by coercion (Blanchard et al, 2005; Dandona et al, 2006). Many FSWs in Karnataka (45%) report starting sex work because of their low financial status, as well as listing reasons such as desertion by their husbands, indebtedness and widowed status (ICHAP, 2002b). If an elevated demand for sex work causes FSWs to have more client partners, or influences more women to go into sex work (larger FSW size), then the male demand for sex work is likely an important factor in Bagalkot's HIV epidemic. FSWs may, however, have more control over the number of men who become their clients than these data indicate. If this were the case, and FSWs' demand for a higher volume of clients caused an increase in the number of clients or the mean number of FSW contacts that each client has, then the observed differences in these parameters in Bagalkot District would explain much of the differences in observed 2004 HIV prevalence.

However, if total number of high-risk partnerships in Bagalkot District's population was fixed, then a change in the male demand for sex work would prompt a proportionately similar, but directionally opposite change in client size. In this case, FSWs have the same number of client contacts and about the same client volume regardless of the size of the client population, with clients forced to reduce their FSW volume with a larger client population. Because of the survival aspect to sex work, this seems less likely than the opposite situation (if total high-risk partnerships remain constant) where a higher number of FSWs have a smaller number client

contacts per year and a lower number of FSWs have a higher volume of client contact, with the male demand for sex work the same regardless of the number of FSWs or their sexual contacts.

Nevertheless, if the total number of high-risk partnerships were fixed when high-risk population size or mean high-risk contact rate changes, then the model results suggest that these are not important factors in the modelled epidemic and there must be other drivers of the epidemic in Bagalkot District. The epidemic may have begun several years earlier in Taluka C compared with Taluka B or A, which could explain differences in 2004 HIV prevalence if the epidemic was still increasing in 2004 or if Taluka C's epidemic had peaked by that time (or if the epidemic was decreasing in 2004 and Taluka C's epidemic began later than Taluka B's or A's). The mean numbers of low-risk partners may also play a larger role depending on the actual timing of the epidemic, which we discuss below. We found that a larger population size delayed the HIV epidemic in the overall population (and all sub-populations), but affected peak and equilibrium model HIV prevalence negligibly. Since Taluka C had higher 2004 observed HIV prevalence in the overall, male and female populations and a smaller population than Taluka B, we hypothesized that overall observed HIV prevalence in 2004 in Taluka B might have been as high as Taluka C's if its population was smaller (and vice versa). However, when we varied the total population size in Talukas B and C, we found that the changes in model prevalence in 2004 were too small to explain all of the differences in 2004 observed prevalence between the two Talukas (varied by <0.4%). It seems unlikely that overall population size has played a large role in the differences in observed HIV prevalence in 2004 between the three Talukas.

- **Parameters without different values in each Taluka (uncertainty analysis)**

We conducted a univariate uncertainty analysis on data for which we did not have data that was different in each Taluka (it was estimated across Talukas). Neither the duration of FSW nor the

duration of MCRB alone is likely to account for the difference in HIV prevalence between the three Talukas, based on model results. Even varying the duration of FSW or MCRB from half-to-double the base-case values resulted in small changes in predicted 2004 HIV prevalence. While we do not have empirical data to describe differences in the average duration of high-risk behaviour between Talukas, it is likely that it does not differ by more than a few years, and not by enough to cause the differences in 2004 HIV prevalence observed between the Talukas.

We had little data on the actual numbers of low-risk partners per sexually active lifetime (SAL) in each Taluka and have discussed how we arrived at our first estimation (one low-risk partner per SAL) and our second (ten low-risk partner per SAL) in Chapter Five: Model Fitting. Increasing the average number of low-risk female partners per male per SAL (and thus the low-risk partner change rate per year) caused an increase in 2004, peak and equilibrium prevalence in all risk groups, but mostly in low-risk groups. Thus, it is possible that the Talukas with higher observed HIV prevalence in 2004 also have higher mean numbers of low-risk partners. However, we do not have empirical data to support this, and to describe differences between each Taluka.

In fact, the differences in values required to cause the differences in overall 2004 HIV prevalence observed between the Talukas are fairly large. Varying the mean number of low-risk female partners per low-risk male per SAL from five to 20 (half-to-double the B-CII value of 10) per SAL did not bring predicted 2004 HIV prevalence in any Taluka to within range of any other Taluka's overall observed HIV prevalence. Taluka A would require about 25 per SAL (almost one mean new LRF partner per male per year) to bring its overall model HIV prevalence to within range of Taluka B's (a difference of 15 LRF partners per male per SAL) and 36 mean LRF partners per male per SAL for Taluka A to reach Taluka C's overall observed 2004 HIV prevalence (a difference of 26 mean LRF partners per male per SAL). For Taluka B's overall

model prevalence in 2004 to reach Taluka C's, the mean number of LRF partners per male per SAL needed to be increased to 30 (from B-CII=10, a difference of 20). These large differences in the average number of LR partners are likely too high to be realistic, and thus the mean low-risk partner change rate is likely not a key factor in 2004 HIV prevalence in Bagalkot District.

However, varying the mean number of LRF partners per male per SAL had a much larger impact on overall model HIV prevalence at peak and equilibrium than in 2004, where the mean numbers of high-risk partners had a much larger impact earlier in the epidemic. The infection became concentrated in high-risk groups very quickly, because the mean high-risk contact rate per year was relatively much higher than the mean low-risk partner change rate per year and took longer to establish in low-risk groups because the low-risk population sizes were relatively much larger than the high-risk population sizes (we showed earlier that a larger population size delays the epidemic). Since our base-case epidemic model curves predicted an epidemic that was still increasing in 2004, varying high-risk sexual behaviour parameters had a large impact on 2004 prevalence. If the epidemics in Bagalkot District have actually reached equilibrium, then mean number of low-risk partners per low-risk person in Bagalkot District could have a larger impact.

- **Migration**

Based on model results, low-risk and high-risk client seasonal out-migration (when a fraction of local non-clients and clients migrate and become clients in the place of migration) seems to be the most likely scenario for explaining differences in HIV prevalence between the three Talukas, particularly when migrant clients had a higher level of risk behaviour (numbers of FSW partners in the POM relative to that in the place of origin; HIV prevalence of POM FSWs relative to the place of origin) at the site of migration compared with clients at the site of origin. In this case, large differences in 2004 model HIV prevalence are seen both compared to the base-case

scenarios with and without migration, and are enough that Taluka A's model prevalence increased within observed prevalence in Taluka B, with 2.9% [2.2 – 3.6]% (2-fold the number of FSW in the POM as in the place of origin increased 2004 model HIV prevalence to 2.4%; 70% FSW prevalence in 2004 in the POM increased overall model prevalence to 2.7%; these values were compared with base-case scenarios with migration, 1.5% and without migration, 1.2%). Higher-risk behaviour in seasonal out-migrants at the site of migration may have been a significant factor in increasing 2004 prevalence in Talukas B and C compared with Taluka A. Increases in migration-associated risk behaviour at the site of seasonal migration was shown to be a major factor in spreading the HIV epidemic in rural South Africa, rather than by connecting areas with low prevalence to areas with high HIV prevalence (Coffee et al, 2007).

It was possible for overall model HIV prevalence in 2004 to reach Taluka C's overall observed prevalence in 2004 in this migration scenario (M.3) with higher numbers of FSW contacts per client in the POM (4-fold, or 40 FSW contacts per month for four months duration), or a 60% migrant size combined with 2-fold the numbers of FSW contacts in the POM as in the place of origin, or when 30% migrants interacted with 2-fold the number of FSWs in the POM as they do in the place of origin, and those FSWs had 70% HIV prevalence in 2004.

When only local clients migrated, to bring overall 2004 model HIV prevalence in Taluka A to within Taluka B's observed values, it was necessary to increase the mean number of FSW partners per MCL in the POM (4-fold assuming constant HIV prevalence in FSWs in the POM; 12-fold assuming a more realistic, logistic epidemic). With all other migration parameters at B-CM values, even increasing the constant HIV prevalence in the POM to 100% (clearly an unrealistic value) could not increase Taluka A's overall 2004 model prevalence to within Taluka B's observed values.

When we varied migration parameters independently to double their B-CM values (all other migration parameters at B-CM values), FSW migration could only bring the modeled epidemic in Taluka A within observed range of Taluka B's 2004 HIV prevalence with a large proportion of FSWs migrating (60%) and a constant HIV prevalence assumed in clients in the POM (15%). A constant HIV prevalence of 15% in clients in the POM since the start of the epidemic is clearly an unrealistic assumption, and FSW migration had a smaller impact in 2004 when the POM client epidemic was gradually increasing (logistic – reaching 15% in 2004 and 40% at equilibrium) and thus more realistic. It was not possible to bring overall model HIV prevalence in 2004 to reach Taluka C's observed 2004 prevalence when only FSWs migrated; the impact of FSW migration was limited by the size of the FSW population, as there are only 837 FSWs in TA in 2004, or 1.6% of the female population. FSW migration may have had a small impact because we only looked at seasonal out-migration – additional data indicates that blocks of FSWs migrate into different populations in Bagalkot District; their size is independent of the size of the local FSW population. This often happens during festival seasons, and the client volume locally increases. It would be interesting to explore further migration scenarios, including in-migration scenarios, to determine the potential impact of migration on heterogeneity in HIV prevalence in Bagalkot District. However, this would require detailed further data.

While we do not have much evidence for the levels and patterns of migration in Bagalkot District, since 2004 model HIV prevalence in Taluka A remained within range of its observed 2004 values (for all migration scenarios except F.1), it is possible to conclude that there is some seasonal out-migration of low-risk males and clients in Bagalkot District that contributes to differences in HIV prevalence between the three Talukas. Travel due to work was associated with HIV infection in a general population survey of Bagalkot District, although not significantly

(Becker et al., 2007). We also found that base-case migration of a fraction of all males did not raise 2004 model HIV prevalence very much compared to the base-case scenario without migration, but became more important later in the epidemic. Results from Becker et al (2007) may reflect that the effects of male migration on the epidemic in Bagalkot District may not be substantial at this stage of the epidemic. However, the low numbers of migrants in the survey (ICHAP, 2004a) suggests that conclusions regarding the lack association of migration with HIV infection cannot be taken too strongly without further study. Seasonal out-migrants may not have been included in this study if they were at the site of migration when the study took place. Conclusions about the effects of migration increasing HIV prevalence depend on whether or not the migration levels and patterns differ across the three Talukas – we have no reason to assume that seasonal out-migrants from one Taluka are prone to higher high-risk behaviour than from other Talukas while in the place of migration. Larger proportions of seasonal out-migrants had a large impact in increasing the epidemic, particularly in the low-risk population at peak and equilibrium, compared with the same proportionate increases in migration-associated risk behaviour and so it is possible that higher levels of seasonal out-migration occur in Talukas with higher HIV prevalence; however, this contradicts qualitative evidence suggesting that Talukas with higher HIV prevalence are wealthier, more fertile Talukas that actually draw migrants in rather than push them out for employment (ICHAP, 2003). Low levels of seasonal out-client migration in Taluka A might have contributed to its lower observed HIV prevalence if migrant clients were not replaced while they were away and there were subsequently lower numbers of client partners per local FSW.

8.2. Ranking of parameters

- **Parameters with different values in each Taluka**

Assessing the impact of high-risk group population size and numbers of high-risk contacts is complicated, as varying any of these parameters also influences the total number of FSW-client partnerships. Six scenarios were developed based on what happened to other parameters when one of these sexual behaviour parameters was varied (see Table 6.1 in Chapter Six: Results).

The size of the 2004 FSW population had the largest effect on increasing 2004 model prevalence in each Taluka when total FSW-client partnerships were allowed to vary (scenario PV2), although the differences were small between the effects of FSW size, MCL size (PV3) and the mean high-risk contact rate (PV1) when these parameters increased from half-to-double their base-case values. In effect, not just FSW or MCL size is being varied – the mean number of high-risk partners that the opposite high-risk group has is also being varied.

While two-fold the base-case (B-CII) FSW or MCL size increased 2004 model HIV prevalence more than two-fold the mean high-risk contact rate, this is in part because of how the epidemic's shape over time varied when these parameters were varied. In the three Talukas, the epidemic's peak occurred nearer to 2004 when there was double the FSW size (between 1996-2001) and MCL size (2002-2008), and earlier with double the mean high-risk contact rate (1990-1993). At peak and equilibrium, the mean high-risk contact rate had a larger effect than high-risk population size. This indicates that overall model HIV prevalence is more sensitive to the mean high-risk contact rate than high-risk population size.

These results are supported when total FSW-MCL partnerships are assumed to vary with FSW or MCL size, and the mean number of high-risk partners per FSW and MCL are held constant (PV4). Here, increasing the size of one high-risk population also increases the other by

the same proportionate amount in order to keep total high-risk partnerships equal. Overall model HIV prevalence in 2004, peak and equilibrium still increased, but these were much less dramatic compared with the first three scenarios (PV1, PV2, PV3). In PV4, the peak and equilibrium prevalence were higher, but the epidemic was delayed with increased FSW/MCL size rather than being higher and reached earlier, as in the other three scenarios.

It is interesting that varying the FSW size/ FSW partners of MCLs (PV2) had approximately the same impact on overall 2004, peak and equilibrium model HIV prevalence as MCL size/ MCL partners of FSWs (PV3), even though there were far fewer FSWs in the population than clients – in 2004, there were about 5-fold the number of clients as FSWs in Bagalkot and Taluka Bs and about 4-fold in Taluka C. It makes sense, however, that varying FSW size and MCL size by the same proportionate amount would have approximately the same effect on HIV prevalence. The force of infection for each risk/gender group determines how many people in each group are infected over the course of an epidemic, and is dependent on its mean number of high-risk contacts (with each group it would come in contact with), the infected population size (of all other groups our population of interest would come in contact with), the transmission probability of HIV (in that particular partnership in each stage of infection) and the probability that one group would come in contact with another. Doubling the FSW population (assuming total FSW-MCL partnerships vary) also doubles the mean number of FSW partners that MCLs have and doubling the MCL population doubles the mean number of MCL partners that FSWs have; in each case, this doubles the force of infection in both FSWs and MCLs, and there is about the same effect on model prevalence.

The size of the FSW population was shown to have the largest impact on increasing an STI epidemic in Ghani and Aral (2005). However, this was due to the model assumption that the

mean number of FSW partners per client remained the same regardless of FSW or client size – thus, when FSW size varied, total high-risk partnerships varied (corresponding to scenario PV2 in this study) but when client size varied, total high-risk partnerships remained constant (PC2 in this study). When we vary FSW or MCL size and total FSW-MCL partnerships remained fixed (PC1 and PC2), increasing MCL or FSW size by double the base-case values caused a decrease in overall model 2004 HIV prevalence (since the associated number of high-risk contacts also declined by half to keep total partnerships constant) that was smaller in absolute value compared with PV2 or PV3, but larger than in PV4. Later in the epidemic, however, only very modest changes were observed in PC1 and PC2, and all four of the scenarios where FSW-MCL partnerships were allowed to vary caused larger absolute changes in overall model HIV prevalence in peak and equilibrium.

Of the data for which we had strong data stratified by Taluka, overall population size had the smallest effect on 2004 model prevalence, with negligible effect on peak and equilibrium model HIV prevalence.

- **Parameters without different values in each Taluka (uncertainty analysis)**

In the uncertainty analysis (Chapter Seven), we examined the effects of varying some of the parameters for which we did not have data stratified by Taluka, by the same proportionate amount we vary the parameters for which we did have strong data stratified by Taluka (half-to-double the estimated B-CII values). While all of these parameters are measured differently and the absolute effects on HIV prevalence outcomes may be dependent on the starting (base-case) values, which were estimated as best as possible from available data, because of the wide range of values for which we varied each parameter in each Taluka, we can nevertheless be more

confident of our results because the same patterns are exhibited in each Taluka. Conducting the same analysis in each Taluka is therefore essentially another form of uncertainty analysis.

The duration of MCRB and FSW consistently produced the smallest variations in HIV prevalence in 2004 overall model HIV prevalence, and had the smallest effect in peak and equilibrium (after overall population size: see previous section). In each Taluka, increasing the duration of FSW from 4.5 years to 18 years (B-CII: 9 years) caused a larger absolute change in 2004 HIV prevalence (increased) than duration of MCRB (decreased), but both parameters caused less than one percent difference when varied through the uncertainty range. 2004 HIV prevalence was highest with lifelong duration of FSW and MCRB, but a shorter duration of these parameters could cause higher peak and equilibrium prevalence. The duration of MCRB produced larger effects on peak prevalence than duration of FSW (since a shorter duration of MCRB also caused a smaller 2004 MCL size, assuming our base-case values for lifetime client size, and thus an unrealistically high FSW-MCL contact rate, assuming the same number of MCL partners per FSW), but effects on equilibrium prevalence were minor for both duration of MCRB and FSW. Varying the mean number of low-risk female partners per low-risk male from half-to-double the B-CII values (5 – 20, B-CII: 10) consistently (in each Taluka) varied (increased) 2004, peak and equilibrium model HIV prevalence by more than the absolute changes caused by overall population size (increased) or duration of FSW (increased) or MCRB (decreased), but by less than any of the sexual behaviour parameters varied in our scenarios.

- **Migration**

Within each of the migrating male scenarios (M.1=only MCLs migrate, constant HIV prevalence in FSWs in the POM, M.2=only MCLs migrate, logistic HIV prevalence in FSWs in the POM and M.3=low-risk males and MCLs migrate, logistic HIV prevalence in FSWs in the POM),

increased migration-associated risk behaviours consistently caused the greatest impact on increasing 2004 model HIV prevalence. In M.1, increasing the mean number of FSW partners that MCLs have in the POM from half-to-double the B-CM values (0.5-2.0-fold, B-CM: 1.0-fold) increased 2004 overall model HIV prevalence by 0.3%; in M.2 this increased by 0.4%; in M.3, this increased by 1.4%. Increasing FSW model HIV prevalence in the POM (2004: 17 – 70%, B-CM: 35%) caused overall model HIV prevalence in 2004 to increase by 0.4% in M.1, 0.3% in M.2 and 1.7% in M.3. At the epidemic peak and equilibrium, these parameters had same small (but largest relative to other parameters) effects on increasing model HIV prevalence in M.1 and M.2. Increasing migrant MCL size (from 15% to 60%, B-CM: 30%) and duration of migration (2 to 8 months, B-CM: 4 months) actually decreased 2004, peak and equilibrium model HIV prevalence by <0.4% in both M.1 and M.2 (except duration of migration in M.2, which decreased 2004 model HIV prevalence by 0.5%). In M.3, the size of the migrant population (varied from 15% to 60%, B-CM: 30%) had the largest impact on increasing peak (by 2.8%) and equilibrium (by 2.6%), then the mean number of FSW partners of MCLs in the POM for peak (by 2.0%) and equilibrium (by 1.6%), then the duration of time away (varied from 2 to 8 months, B-CM: 4 months) on increasing peak (by 1.6%) and equilibrium (by 1.4%) and finally HIV prevalence in the POM on increasing peak (by 0.3%) and equilibrium (by 0.1%).

In the migrating FSW scenarios (F.1=only FSWs migrate, constant HIV prevalence in MCLs in the POM; F.2=only FSWs migrate, logistic HIV prevalence in MCLs in the POM), the independent impact of varying any of the migration parameters caused small variations in model HIV prevalence, varying 2004 prevalence more compared with peak and equilibrium model HIV prevalence, but still by <0.5%. The size of the migrating FSW size had the largest effect on increasing overall 2004 model HIV prevalence (by 0.4% in both F.1 and F.2). This is because

when FSWs migrate, the model assumptions is that the average number of client contacts per FSW remaining in the place of origin increases proportionately to the size of the migrating population and time away. In F.1 and F.2, increasing the duration of time away (2 to 8 months, B-CM: 4 months) and the mean number of MCL partners that FSWs have caused the next largest impact on 2004 model HIV prevalence (increase by 0.3%), with peak and equilibrium model prevalence increasing by similar negligible amounts.

8.3. Implications of findings

This research has several implications from an HIV prevention program planning perspective in rural southwest India. Since the HIV epidemic in Bagalkot District is very heterogeneous on a Taluka level, it is important to define different factors that contribute to higher HIV prevalence in some areas than others to provide insight as to where to focus HIV prevention interventions.

We hypothesized that since across the three Talukas, larger proportions of FSWs and MCLs, and the estimated FSW-MCL contact rate were accompanied by higher 2004 observed HIV prevalence, these factors would likely be associated with the increase in HIV prevalence. In this study, the size of the high-risk populations was shown to be an important driver of the HIV epidemic in rural India, if a change in the FSW or client size was accompanied by a change in the mean numbers of high-risk partners in the opposite high-risk population (total high-risk partnerships vary). It is intuitive that HIV prevalence would increase if total high-risk partnerships increase, since larger FSW-client contact rate is then accompanied by larger client-FSW contact rate, larger FSW size is accompanied by increased FSW-client contact rate and larger client size is accompanied by increased client-FSW contact rate. However, this analysis showed that the relative differences observed in these parameters between the three Talukas were

large enough to explain a substantial fraction of the differences in observed 2004 HIV prevalence between the three Talukas, providing that high-risk partnerships also varied.

The mean high-risk contact rate was a more important factor early in the epidemic than FSW and client size: varying contact rates only had a larger impact on 2004 and peak prevalence compared with varying FSW and client sizes only. If the mean high-risk contact rates had been set at half their base-case estimated values since the epidemic began, the modelled epidemic would have essentially never established in any of the Talukas. Even when FSW or MCL size was set to half the base-case values, an epidemic always established in each Taluka and reached near or over 1% in the overall population by the epidemic's final prevalence.

Core groups, such as FSWs and clients are frequently identified as being the highest risk for acquiring and transmitting HIV infection, with clients being a bridging group to the general population via contacts with sex workers and with low-risk, regular female partners (Lowndes et al., 2002; Voeten et al., 2002; Anderson & May, 1991; Anderson et al., 1991; Plummer et al., 1991; Kreiss et al., 1986). Using the best available data across the three Talukas, we also found this to be the case in Bagalkot District. The fraction of new infections acquired by low-risk groups was consistently low, but increased over the course of the epidemic (from 10% early in the epidemic to almost 30% in each Taluka by the epidemic's final prevalence) when we assumed increased numbers of low-risk partners (10) in each Taluka, our main base-case scenario (B-CII) developed to fit the model in all Talukas to observed HIV prevalence (when we assumed only one low-risk partner in each Taluka, the fraction of new infections in low-risk groups was only 1-3% early in the epidemic and 3-6% at the epidemic's equilibrium). The role of the bridging client population seemed to be larger in Taluka C, with the highest client and FSW prevalence. Even though we assumed the same numbers of low-risk partners across the

three Talukas, the low-risk epidemic in Taluka C was much higher than in the other two Talukas (by at least 2.6-fold and 2.8-fold in low-risk males and females in 2004; by at least 1.6-fold and 1.8-fold in low-risk males and females later in the epidemic). This was also likely influenced by the slightly larger probability that low-risk females had sexual relationships with male clients, because of Taluka C's larger client size. A study in Cotonou, Benin, suggests that the number of new infections generated by high-risk groups is much higher than is transmitted by the general population, which accounts for only a small proportion of new infections (Lowndes et al., 2002). In the male population in Ghana, over 80% of the prevalent cases of HIV infection were shown to be due to sexual interactions involving transactional sex (Cote et al., 2004). Even though we assumed an increased number of low-risk partners in our base-case scenario, the fraction of infections caused by women and men involved in transactional sex was near to 95% early in the epidemic (in 2004) and about 85% when the epidemic had reached its final prevalence (higher when we assumed fewer low-risk partners, remaining near 100% for the entire epidemic). This remained true even when we varied FSW and client size through a wide range of values.

Our results indicated that FSW and MCL size was not important in defining the different HIV epidemics across Talukas if there is no related change in the mean number of high-risk partners by the opposite high-risk group (total high-risk partnerships remained constant). We have already discussed the likelihood of this scenario in a previous section (8.1). Nevertheless, even if the size of the high-risk population or numbers of high-risk partners are not important factors in explaining the differences in HIV prevalence in 2004 between the three Talukas, data available still suggest that high-risk populations are a driving factor in the epidemic in rural India. However, other factors may have a larger impact than is accounted for in the model. Despite the estimated low infection levels due to and found in low-risk populations in this

modelling study, much of this may be due to the data available underestimating behaviour that may place low-risk groups at higher risk for HIV infection in rural India. More evidence is emerging that married women have pre- or extra-marital relationships, particularly when they are married to husbands who migrate away for long periods of time (Halli et al, 2007). The contribution of homosexual male-with-male partnerships may also be a driving factor in the epidemic that we have overlooked: for example, in 2004/2005, 7.2%/12.0% of men in Taluka A, 4.3/11.0%% in Taluka B and 7.2%/5.7% in Taluka C, reported ever having sex with another man (Ramesh, 2005a). In the general population in rural India, there is a low fraction of circumcised men, early age of onset of sexual activity for women, large differences in the mean age of marriage between young women and older men, and a lack of condom use in marital partnerships, and these different levels of these factors found in different areas of Bagalkot District may account for more of the differences in overall model HIV prevalence than we can account for in this simple model.

There is a common belief that seasonal out-migration of men is a driving factor in the HIV epidemic in rural India, particularly in Northern Karnataka, where Bagalkot District is located. At this time, empirical data on migration in Bagalkot District is limited and analysis on migration was conducted to assess the likely impact of only a few patterns of migration in rural India. Migration of only clients or only FSWs was found to only have a marginal impact on the local epidemic, and only when low-risk clients migrated to become clients in the POM were important differences seen. However, model results depended heavily on the assumption that a fraction of the total existing local population out-migrated from their home Talukas for several months duration. It is also thought that groups of migrants travel in and out of different areas at different times of the year for work (such as FSWs during festival seasons or MCLs for seasonal

labour), and that the groups' sizes are unrelated to the size of the local populations. FSW and MCL migration might have had more of an impact than indicated by model results if this pattern of migration was taken into account. In addition, much of the increases in model HIV prevalence (although small) for FSW migration are due to the increase in the number of client contacts in local FSWs when migrating FSWs are away (we assumed that migrating FSWs were not replaced and the local male demand for sex work remains same). If migrating FSWs or MCLs are replaced by others, the model would produce different results; this illuminates the importance of collecting data on local sexual behaviour dynamics while migrants are away.

Only when migration-associated risk behaviour was increased in the POM compared to the home Taluka did the fraction of new infections due to migrating groups increase to higher than their population sizes (relative to non-migrating groups). Increased levels of migration (high compared to moderate or low) caused a larger fraction of new infections in females when clients or clients/low-risk males migrate, but a larger fraction of new infections in males when only FSWs migrate. This indicates that if there is a larger migrating population with higher levels of migration-associated risk behaviour in the POM, migrating populations have a larger impact on the populations with whom they have local sexual relationships. This highlights the importance of collecting data quantifying the differences in sexual behaviour of migrants when they are in the POM relative to when they are in their places of origin.

Studies have found that targeting only core groups can reverse the serious outcome of an HIV epidemic and drive it to extinction, particularly if transaction sex acts are targeted earlier in the epidemic (Nagelkerke et al., 2002; Boily et al., 2002). A mathematical modelling study examined the potential impact of interventions at FSWs and clients in both Bagalkot and Mysore Districts in Karnataka, and found that focusing interventions at high-risk groups could be very

effective at reducing the overall epidemic in these areas, due to the low estimated transmission among low-risk groups in these areas (Williams et al., 2006). Given the importance of the contribution of core and bridging groups indicated by our results, high-risk groups would clearly be the most effective to target with intervention programs and should continue to be a priority for HIV-prevention-related resource allocation. Results from Williams et al. (2006) suggested that a simple condom intervention program would be the more effective individual intervention, and also that interventions would be more effective if introduced in areas with smaller numbers of FSWs (Mysore compared with Bagalkot District). These results suggest that interventions introduced into Talukas A and B could have a greater impact than if introduced in Taluka C, and this should be investigated more thoroughly in future studies. In all Talukas, FSW and their clients should be targeted as early as possible, as they generate a smaller proportion of new infections later in the epidemic; however, their contributions at all stages of the epidemics should be noted and addressed with prevention programs.

A simple condom intervention program had the largest theoretical effect (when introduced into migrants only) when both low-risk and high-risk males migrated compared to other migration scenarios, which makes sense because it was in this scenario that migration had the largest impact compared with the other scenarios (only a fraction of local FSWs or MCLs migrating). If a simple intervention is introduced into non-migrating groups only, there is a much larger impact in the latter two scenario compared with the former. Results suggest that the epidemic can be lowered dramatically in low- and high-risk females if a simple condom intervention program is introduced into non-migrants only or the whole high-risk population, but has a much smaller effect if it is introduced only in the migrating populations.

8.4. Directions for further study

Further research could be undertaken in Bagalkot District to explore the impact of HIV prevention interventions in populations with different structures and levels of migration in rural India. We determined that some risk factors in Bagalkot District would have more of an impact on overall, peak and equilibrium HIV prevalence than others, but that model HIV prevalence in 2004 would fit to observed prevalence for many different sets of parameters and assumptions for the model structure. For example, 2004 model prevalence fit to observed values in Taluka A for four of the five migration scenarios, when we assumed base-case migration parameters, as well as when we assumed that there was no migration activity in Taluka A at all. Evaluating the effectiveness of HIV prevention interventions in each of these scenarios would be helpful to explain why prevention programs seem to work better in some populations than others.

Additionally, it would be useful to compare and contrast different effects of introducing different interventions into high-risk groups or low-risk groups. There are several interesting interventions that could be evaluated in mathematical models of the HIV epidemic in rural India in addition to condom interventions. The population in this area of rural India is largely uncircumcised, and circumcision of men has recently been shown to significantly reduce the transmission of HIV in Kenya (Bailey, et al., 2007). In addition, while a trial on the effectiveness of a new microbicide was recently halted because women using microbicides were showing higher rates of HIV than women who were not (Horwood, 2007), it is still feasible that an effective microbicide could be developed. Further research could therefore explore whether microbicides, circumcision or condoms would best reduce HIV prevalence and incidence in rural India, under different assumptions for the model structure (while also evaluating how realistic it would be to introduce these types of interventions into populations in rural India).

Additional research could be conducted to explore the reasons for heterogeneity in HIV prevalence between the three Talukas using stronger empirical data, which would reduce model uncertainty. Particularly, migration in rural India is thought to be a major factor in helping spread HIV and although we explore the effects of migration in different migration scenarios, we were limited by the lack of available data on migration in Bagalkot District. A study entitled "The Corridors Project" is currently underway to explore the levels and patterns of migration within northern Karnataka and southern Maharashtra (Public Affairs, University of Manitoba, 2006). This analysis would be greatly enhanced with additional empirical data on the patterns and amount of migration in Bagalkot District. Particularly, it would be interesting to know the extent that migration-associated risk behaviour is higher than high-risk behaviour in the place of origin – for example, by what factor, if any, are the numbers of high-risk partners per client or per FSW increased while the individual is away from their familiar environments.

We explored the effects of annual seasonal migration only, where clients or FSWs migrate away from their home Taluka for a certain block of months per year, with a frequency of only once per year. It would be interesting to explore the effects of more frequent migration, where clients are able to return home several times in the middle of their long time away and resume sexual relationships with their low-risk female partners (wives, for example). It would be useful to examine the effects of increased frequency of migration causing migrant men to return home at different stages of HIV infection – HIV is much more easily transmissible in the few months after initial infection and frequent visits to their wives, with whom they are less likely to use condoms, would increase the chance they would be more infective during sexual contact.

Similarly, it would be useful to understand better the effects of high- and low-risk HIV transmission probabilities on HIV infection in Bagalkot District and rural India. The

transmission probability values we used in this study allowed us to fit the model well to observed 2004 HIV prevalence in each Taluka. However, as we showed in the uncertainty analysis, peak and equilibrium prevalence (as well as shape and timing of the epidemic) can be significantly affected by the transmission probability set used. Ours were estimated from empirical data taken from Thailand, which assumed there were no factors present that increased transmission probabilities (sexually transmitted infections, violent sex) nor factors that decreased transmission probabilities (condom use). Clearly, these factors exist in Bagalkot District and it would be very useful to explore how important the transmission probabilities are at each stage of infection and how these are affected by the duration of the stage of infection.

8.5. Study limitations

Mathematical models

Mathematical models provide a framework within which researchers can communicate and understand the complex the acquisition and transmission dynamics of infectious diseases in a population (Garnett, 2002). Models provide valuable insight into the mechanisms that influence these dynamics, and how infectious diseases might best be controlled (Garnett, 2002; Boily & Masse, 1997). The predictions of mathematical models can be very useful, for example, in situations where it is difficult to implement an empirical study. This is particularly true when the disease of interest is HIV/AIDS, an illness which can have serious adverse outcomes, especially in poorer countries. Many issues may discourage researchers from conducting an empirical study on a population in this case: the duration of time in a longitudinal study required to follow participants, the cost of the study and the ethical considerations. Practically and ethically, some questions are best answered by using a theoretical mathematical modeling framework.

However, mathematical models represent very complex sexual behaviour dynamics of people in a population, and the reliability of the model is only as good as the assumptions upon which it is based, including the model structure and the model parameter estimates. A common concern regarding models is that they are unable to encompass the almost-limitless types of sexual networks and the specific sexual behaviour characteristics of a population under study. These concerns have validity, as mathematical models are often a heavy simplification of these characteristics. A balance must be struck between creating a model that is complex enough to accurately estimate and represent the characteristics of a population and the dynamics of acquisition and transmission of infection, while also remaining simple enough for researchers and policy-makers to effectively interpret and communicate results, to make predictions about the likely course of an epidemic under certain conditions and to provide realistic options regarding how to control the spread of disease (Anderson & May, 1991).

Type and structure of models

The models used in this study were simple deterministic compartmental mathematical models. Some researchers feel that deterministic models do not represent the dynamics of HIV infection well, since these models are unable to capture the randomness of sexual interactions in a population, and that stochastic models of HIV infection should be used instead (Wai-yuan, 2000). The merits of deterministic versus stochastic (versus statistical models, or the state space model) models are too complex to debate adequately in this thesis. However, deterministic models have been successfully used in similar studies on HIV and other STIs (see Chapter Four). The transmission dynamics models used in this study were simple, but encapsulated the main aspects of the population under study, and fit well to observed data. Models were formulated to best use the epidemiological, demographic and sexual behaviour data that was available; more

complex models would likely require that more parameter values be estimated from data outside the population of interest, limiting the relevance of the conclusions to the area.

Nonetheless, it is important to recognize the limitations of some of the characteristics and assumptions of the model in this study. For example, the simple assumption that a rural Indian population can be well-represented by only two risk groups in a heterosexual population may not accurately represent the transmission dynamics of HIV infection or best predict the HIV epidemic in rural India. The model included four main population groups (low-risk males and females; high-risk males and females), with the low-risk male, high-risk male and high-risk female groups being split into migrating versus non-migrating populations. It may be more appropriate to include additional risk groups, with sexual behaviour that is between high- and low-risk. This idea is not backed up by traditional ideas of Indian culture, where it is assumed that wives are monogamous within marriages, or not sexually active, although some husbands may visit female sex workers (for simplicity, our model captures this scenario). Our model assumes that low-risk women have a low average number of only long-term partnerships and it may be important to include a compartment of generally low-risk women who occasionally have one-time, higher risk relationships with men, as suggested by some studies (Halli et al., 2007).

We may also be overlooking male-to-male transmission – homosexuality is technically outlawed in India, but male-to-male sexual relationships are relatively common. Although our model structure was based on evidence suggesting that most transmission of HIV is heterosexual (Arora, et al., 2004), the sexual network between males may be a significant hidden pathway of HIV transmission (Ramesh, 2005a), as we have already discussion in section 8.3.

Additionally, we assumed that FSWs only have high-risk ("one-time") partnerships with clients, and that all of their reported partners per week (10 in northern Karnataka) were one-time

partnerships with MCLs. The numbers of reported partners per week were actually broken down into several categories when the data were collected: FSWs were asked how many partners they had in the previous week, and of these partners, how many were 1) one-time partners, 2) regular partners and 3) husbands, lovers or boyfriends (ICHAP, 2002a). There was not enough information on the two latter types of FSW partners. For example, a "regular partner" was not defined explicitly in the survey. FSWs defined themselves what a regular partner was, and so a regular partner could be someone a FSW sees greater than once every week, month, three months, etc. Also, we did not have information on how many of these relationships are monogamous from the male partner's perspective; anecdotal evidence suggests that some husbands of FSWs are not in only one relationship with one sex worker. Even if long-term partnerships between FSWs and men were included in the model (decreasing the number of short-term partnerships), the overall epidemic might have been relatively unaffected (many one-time contacts with clients at a lower transmission probabilities per contact might have the same effect as fewer long-term partnerships with higher transmission probabilities per partnership, assuming there are multiple contacts with the same person in long-term partnerships).

Some or all of these additional sexual networks might be worth investigating in further studies. More information is needed before they can be adequately incorporated into our model.

Quality of data used in models

The quality of a model's predications can be enhanced or hampered by the quality of data available to incorporate into the model. Valid data on sexual behaviour in particular can be difficult to obtain in many populations, as sexuality is frequently a sensitive issue in many countries (Fenton, et al., 2001). This is especially true in countries such as India, where many people, especially those living in rural areas, live within a sexually conservative culture. For

women, disclosing sensitive information on sexual behaviour and HIV/AIDS can result in violence, discrimination and neglect (Bharat, 1996).

Different methods of collecting data produce different accounts of the levels of sexual behaviour risk factors in a population. In Bagalkot District polling booth studies provided much higher estimates for the proportion of men who report ever visiting FSWs, compared to face-to-face questionnaire administration. The method of collecting data using polling booths provides an anonymity that facilitates more accurate reporting of sexual behaviour characteristics; in this method, a question is read aloud to people in a group, who drop their answer off in boxes (Kang, 2005). Their answers cannot be linked to their person, which is a limitation for multivariate data analysis, but may allow for more accurate point estimates of a certain sexual behaviour characteristic. In the face-to-face questionnaire study in Bagalkot District, 1.9% of men reported that they had ever paid/received money for sex, while using polling booth methodology, this increased dramatically to 12.8% (ICHAP, 2004a; Ramesh, 2005a). In a study conducted in Bijapur, a neighbouring District to Bagalkot District, a higher proportion of men in focus groups reported visiting commercial sex workers compared with men in polling booths (Halli et al, 2007). Whether or not this is an increase in accuracy, however, is questionable, since men are notorious for over-reporting their levels of sexual behaviour, and it is possible that this would only be facilitated in the presence of other men as a result of their desire to impress others.

Other factors may influence the accuracy of data used in this study. The increase in the visibility and acceptance of sex work as facilitated by the increasing presence of sex work collectives in India (KHPT, n.d.) may have influenced the changing estimates of the numbers of FSWs enumerated in the population, which have increased over the last few years. Stigma related to having HIV in Bagalkot District has also reportedly decreased due to the presence of

organizations that aim to support and empower people living with HIV/AIDS in India, such as the Indian Network of People Living with HIV/AIDS (Indian Network of People Living with HIV/AIDS, n.d.) or the SAATHII group (Solidarity and Action Against the HIV Infection in India); this may increase the willingness of people to best tested for HIV and other factors.

The data considered the strongest in this study were overall population sizes in each Taluka (which were gathered is a census as part of the sampling procedures in the HIV prevalence study conducted by ICHAP in 2004), FSW size (which has been enumerated several times) and the mean numbers of partners per FSW. There were several limitations to the data on male client size – first, it is likely underestimated and second, the yearly numbers of clients in each Taluka was not directly collected – men were normally asked to report if they had "ever visited commercial sex workers" or "ever paid or received money for sex". The 2004 male client size was determined via an equation that was dependent on the proportion of men who had ever visited sex workers, as well as the estimated number of sexually active years and the mean number of years during which they had visited sex workers (time they started visiting sex workers subtracted from time they stopped being clients). Very little data exists for the latter two parameters, and we estimated high values for them so that the 2004 client size was larger and mean numbers of FSW partners per MCL was lower, while fitting to observed HIV prevalence.

Concerns regarding model fitting

There were some concerns with the final initial model fit used for analysis. Even with less-than-lifelong duration of high-risk behaviour, it was difficult to fit to 2004 observed HIV prevalence in the three Talukas, because of the low levels of sexual behaviour reported in low-risk groups and the high levels of sexual behaviour reported in high-risk groups. The assumption of an average of 10 low-risk partners per SAL was perceived as too high. As described in Chapter

Four, Section 4.3: Parameter Estimation, most people in Bagalkot District's general population reported a low number of lifetime sexual partners, although other data indicates that some people may have greater than one or two per SAL. One of the difficulties fitting the model to observed HIV prevalence is that we only had one data point for the overall, male and female populations (2004), to fit observed prevalence in each Taluka. Clearly, there could be many different model epidemic shapes that fit to 2004 observed data. HIV prevalence data for Bagalkot District and Karnataka indicates that the HIV epidemic may have stabilized by 2004, reaching equilibrium, where the model used in this study predicts an epidemic still increasing in 2004.

In addition to some of the model structure and data assumptions described already, other factors not taken into account in the model may help explain why the modeled epidemic did not reach equilibrium by 2004, despite using observed data for demographic and behavioural model parameters available. We did not take into account certain factors that might affect transmission probabilities, including condom use, levels of STIs, or type of sex practiced; these might be different across Talukas. Condom use was reported to be as high as 70-80% in FSWs in 2002 in northern Karnataka (ICHAP, 2002b). Within the general married population in Bagalkot District, 26.3% of males and 23.2% of females report that they have ever used a condom (Ramesh, 2005a). 23.3% of all people in Bagalkot District in 2004 and 26.2% in 2005 report any symptoms of STIs, as defined as genital ulcers, burning urination or white discharge (Ramesh, 2005a) and 16.6% of FSWs in Bagalkot District reported that they had experience STI symptoms (Ramesh, 2005b). Also in the FSW population in Bagalkot District, 15.9% reported that their clients had ever asked them for anal sex (Ramesh, 2005b).

Since condom use decreases transmission and STIs and anal sex likely increases transmission, there might be a small effect overall on transmission probabilities and the shape of

the modeled epidemic. Instead, increases or decreases in levels of these factors at different points of time might have had a greater impact on the epidemic's shape. In the modeling software used (Berkeley Madonna), it is difficult to represent changes in model parameters at certain points over time and so factors whose values might be time-dependent would remain constant over the whole epidemic. Condom use has likely increased in frequency of use since HIV was detected in India's populations; mean numbers of high-risk contacts may have been higher before HIV was detected and would increase the transmission of HIV and make the peak and equilibrium prevalence be reached earlier (as we showed in our analysis); STIs may have been higher before HIV was detected and attention was drawn to preventing STIs along with HIV. If the model had included assumptions for transmission was higher than is predicted earlier in the epidemic, with increasing condom use later in the epidemic, then the epidemic might have reached equilibrium prevalence earlier, nearer to 2004. It has also been hypothesized that the bridging role of clients as they pass infections (acquired from FSWs) to their low-risk female (mostly marital) partners is becoming weaker due to increased condom use in both FSWs and clients, as HIV prevention has gained more attention. Since the mean age at marriage for males is much higher than females, younger women, who are thought to be biologically more susceptible to HIV infection, were becoming infected at higher rates than older females, fueling the epidemic's increase. If the model could take into account the different risk for becoming infected for different age groups (in the past increased for younger low-risk females), and also the change in the level of risk over time (decreased for younger males and low-risk females), then the modeled epidemic's equilibrium may have fit closer to equilibrium in 2004.

Despite the limitations regarding the model's structure, estimated values of parameters and the modeled epidemic's shape, the model fit observed 2004 prevalence data in each Taluka

using the best data available when this study was conducted. We were thus able to conduct univariate and multivariate sensitivity analyses and assess the impact of important factors thought to be drivers of the epidemic in rural India; the study objectives were therefore satisfied.

8.6. Conclusions

The HIV/AIDS epidemic in India has been the source of great concern in recent years, as India has one of the highest numbers of persons living with HIV/AIDS in the world. India's large, dense population, weak health care system, high levels of poverty, malnutrition and generally low status for women, has many risk factors that contribute to high levels of HIV infection, and there is a worry that the epidemic could explode to devastating effect. Yet the epidemic has materialized with vastly different patterns and levels of severity, across geographic space and within different populations. The heterogeneity in India's HIV epidemic highlights the need to understand the relative contribution of risk factors to higher levels of HIV infection in some areas compared with others, in order to develop effective site-specific prevention interventions.

Heterogeneity in HIV prevalence is observed even in Bagalkot District, a mostly rural District in northern Karnataka, a southern Indian state with high HIV prevalence in the general population. This transmission dynamics mathematical modelling study provides evidence of the importance of the contribution of core and bridging groups (female sex workers and their clients) to different levels of HIV infection in three Talukas with varying HIV prevalence in Bagalkot District. The fraction of infections due to high-risk groups was much higher than low-risk populations, in which there was low transmission of infection. We found in this study, that if Taluka "i" had the same female sex worker population size, client population size, or estimated mean number of high-risk partners as Taluka "j", then the modelled epidemic in Taluka "j" produced the same 2004 HIV prevalence in Taluka "j" as observed in Taluka "i", indicating that

the impact of each of these parameters could explain a substantial fraction of differences in HIV prevalence between Talukas. This was dependent on an increase in one of these factors in the total number of high-risk partnerships varying when one of these parameters varied; thus, an increase in one of these factors in one high-risk population being accompanied by an increase in one of the factors in the other high-risk population. Interestingly, if total number of high-risk partnerships remained fixed, increasing female sex worker or male client size decreased prevalence, since the number of high-risk contacts in the opposite high-risk population also decreased. The mean numbers of low-risk partners, duration of female sex work and male client risk behaviour, and overall population size were varied through realistic values, and we showed that these were unlikely to explain differences in 2004 HIV prevalence between Talukas.

While the size of the female sex worker population is confidently estimated in Bagalkot District, we are less confident with the client size and the mean numbers of high-risk contacts that female sex workers or male clients have on a Taluka level. Since overall model prevalence was shown to be most sensitive to these parameters, additional data on the population sizes of female sex workers and clients would help reduce model uncertainty and help assess the relative importance of these parameters. Since model results were also sensitive to the sexual behaviour scenario explored (total high-risk partnerships vary versus total high-risk partnerships remaining constant when female sex worker size, client size or high-risk contact rates are varied), this also highlights the importance of measuring not only the values of high-risk behaviour parameters in one population relative to another (which whom the first interacts via sexual relationships), but also how the dynamics of sexual behaviour in these sexually interacting populations are affected when the values of these sexual behaviour parameters change, in populations in rural India.

This study showed that seasonal out-migration may be an important risk factor for the spread of HIV in rural India, provided that a fraction of males who are not clients in the place of origin become clients in the place of migration (both low-risk and high-risk males migrate). This was true even if there was no increase in migration-associated risk behaviour in the place of migration (mean numbers of high-risk contacts in the place of migration relative to that in the place of origin; HIV prevalence in high-risk groups in the place of migration). However, increases in migration-associated risk behaviour had the largest impact early in the epidemic on the overall population, while the size of the migrating population had more of an impact on long-term epidemic prevalence. Seasonal migration of local clients only had a relatively modest impact on the HIV epidemic in India, unless clients had much higher (likely unrealistic) risk behaviour in the place of migration. Seasonal migration of only female sex workers had a greater impact on HIV prevalence than client-only migration (even if female sex workers did not increase risky sexual behaviour in the migration site), because of the model assumption that the demand for sex work was determined by male clients when female sex workers or clients migrated away from their home Talukas. When female sex workers migrated, the average number of client contacts per local female sex worker increased proportionately to the migrant FSW size; when clients migrated, the average number of client contacts per female sex worker decreased proportionately to migrant client size. The effect of female sex worker migration was limited because of the small female sex worker size relative to the overall population. Even with high levels of migration-associated risk, interventions were much more effective if introduced into non-migrants only compared to migrants only. Additional detailed data on the local sexual behaviour dynamics when migrants are away and on differential levels of risk behaviour in migrants is needed to better evaluate how migration affects rural India's HIV epidemic.

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APPENDIX 1: Parameter table, flowcharts & maps of study area

Table A1.1. Table of model parameters

Parameter definition	Scenario	Taluka A	Taluka B	Taluka C	Model symbols	Type of data and reference
EPIDEMIOLOGICAL PARAMETERS						
To seed the epidemic: Number of initial HIV infections in 1976						
FL	B-CI, B-CII	1			no_infFL	DD4 [72]
ML	B-CI, B-CII	1			no_infML	
FH	B-CI, B-CII	2			no_infFH	
MH	B-CI, B-CII	2			no_infMH	
FL	B-CM	1			no_infFL	
ML	B-CM	1*(1-propmigML)			no_inf_ML	
FH	B-CM	2*(1-propmigFH)			no_infFH	
MH	B-CM	2*(1-propmigMH)			no_infMH	
MLM	B-CM	1*(propmigML)			no_infMLM	
FHM	B-CM	2*(propmigFH)			no_infFHM	
MHM	B-CM	2*(propmigMH)			no_infMHM	
For model validation: (Observed prevalence of HIV 2004) [95% CI]	B-CI, B-CII, B-CM					
Total population		1.2% [0.6 – 2.1]	2.9% [2.2 – 3.7]	4.9% [3.6 – 6.6]		DD1 [83]
All males		1.1% [0.2 – 2.0]	3.0% [2.0 – 4.0]	6.4% [4.2 – 8.6]		DD1 [83]
Low-risk males		>1.3%	>2.8%	>3.2%		ID1 [83]
All females		1.3% [0.3 – 2.3]	2.8% [1.8 – 3.8]	3.2% [1.5 – 4.9]		DD1 [83]
Low-risk females		<1.1%	<3.0%	<6.4%		ID1 [83]
Client		17.0% (7.0% - 27.0%)				ID2 [52, 71-73]
FSW		35% (25% - 45%)				DD2 [43, 79]
DEMOGRAPHIC PARAMETERS						
Size of 15-49 years population, 2004	B-CI, B-CII, B-CM					
Total population		117312	180575	119659		DD1 [83]

Females		57383	89349	59247		DD1 [83]
Males		59929	91226	60412		DD1 [83]
Male-to-female ratio		0.49	0.49	0.50		*calculated using
Female-to-male ratio		0.51	0.51	0.50		above data
Size of sexually active population, 2004	B-CI, B-CII, B-CM					
Total		91708	155308	101009	Ntot	DD1 [83], IDI [95]
Ratio with TA		1.0-fold	1.6-fold	1.1-fold		*calculated using
Ratio with TB		0.6-fold	1.0-fold	0.7-fold		above data for each
Ratio with TC		0.9-fold	1.5-fold	1.0-fold		Taluka
Females		53653	89260	58358	Ntot_F	DD1 [83], IDI [95]
Males		38055	66048	42651	Ntot_M	DD1 [83], IDI [95]
Male-to-female ratio		0.640	0.425	0.422	mal_fem_ratio	*calculated using
Female-to-male ratio		0.360	0.575	0.578	fem_mal_ratio	above data
Size of FSW (2004)	B-CI, B-CII, B-CM	837	1751	2083	N_FH	DD1 [13]
Fraction FSW (2004)	B-CI, B-CII, B-CM	1.6%	2.0%	3.6%	propB_FHT	*calculated (DD1
[range]		[0.8 – 3.2]%	[1.0 – 4.0]%	[1.8 – 7.2]%		[13])
Fraction ever-MCL	B-CI, B-CII, B-CM	11.4%	13.2%	18.0%	prob_everCLI	DD1 [80]
[range]		[5.7 – 22.8]%	[6.6 – 26.4]%	[9.0 – 36.0]%		
Fraction MCL (2004)					propB_MHT	
Lifelong MCRB	B-CI	10.8%	12.4%	16.6%		*calculated (Eq. 4.2)
22 years MCRB	B-CII, B-CM	-	-	13.5%		*calculated (Eq. 4.2)
[range]		-	-	[6.9 – 26.0]%		
20 years MCRB	B-CII, B-CM	8.8%	9.6%	-		*calculated (Eq. 4.2)
[range]		[4.5 – 17.1]%	[4.9 – 18.7]%	-		
Fraction low-risk female (2004)	B-CI, B-CII, B-CM		1-propB_FHT		propB_FL	*calculated using propB_FHT
Fraction low-risk male (2004)	B-CI, B-CII, B-CM		1-propB_MHT		propB_ML	*calculated using propB_MHT

Exponential growth rate 1981 – 1991, Karnataka	B-CI, B-CII, B-CM	1.9%				DD4 [27]
Exponential growth rate, 1991 – 2001 (assume to 2004, lack of data), Karnataka	B-CI, B-CII, B-CM	1.6%				DD4 [27]
Growth rate, total population 1986 – 2004 (in the absence of AIDS deaths)	B-CI, B-CII, B-CM	1.9%			preAIDSgrowth_ki	DD4 [27]
SEXUAL BEHAVIOUR PARAMETERS						
Median age of onset of sexual activity Males Females	B-CI, B-CII, B-CM	 23.9 years 16.7 years	 22.4 years 15.1 years	 21.1 years 15.4 years		 DD1 [44] DD1 [44]
Median age of onset of sexual activity Males Females FSWs		22.5 years 15.7 years 17.8 years				 DD1 [44] DD1 [44] DD3 [47]
Sexual life expectancy Males Females	B-CI, B-CII, B-CM	 25.1 32.3	 26.6 33.9	 27.9 33.6	 nat_deathM nat_deathF	 ID1 [44] ID1 [44]
Mean age of FSWs Mean age of MCLs		27 years 32.6 years				DD3 [47] DD3 [47]
Mean duration of high-risk behaviour MCRB [range]	 B-CI B-CII, B-CM (initial) B-CII, B-CM (fit to model)	 lifelong 10.1 years [5 – 20] years 20 years	 lifelong 10.1 years [5 – 20] years 20 years	 lifelong 10.1 years [5 – 20] years 22 years	move_MHML	 model assumption ID3 [44, 47] fit to model

FSW [range]	B-CI B-CII, B-CM	lifelong 9.0 years [4.5 – 18.0] years			move_FHFL	model assumption ID3 [47]
Mean number of low-risk partners per sexually active lifetime (SAL) Low-risk males (constant over time) [range] Low-risk females (varies over time) [range]	B-CI B-CII, B-CM (fit to model) B-CI B-CII, B-CM	1 per SAL 10 per SAL [5 – 20] per SAL ~1 per SAL ~10 per SAL ~[5 – 20] per SAL			MB_ML M_FL	ID1 [44] fit to model ID1 [44] fit to model
Mean number of high-risk partners per year MCLs (constant over time) lifelong MCRB 22 years MCRB [range] 20 years MCRB [range]	B-CI B-CII, B-CM B-CII, B-CM	100 per year - - 119 per year [60 – 220] per yr	110 per year - - 141 per year [70 – 280] per yr	139 per year 169 per year [85 – 340] per yr - -	MB_MH	*calculated using Eqs. 4.1-4.3 and ID1 [13, 80], ID3 [47]
FSWs (varies over time) [range]	B-CI, B-CII, B-CM	~510 per year [255 – 1020] per year			M_FHT	DD3 [47]
Sexual mixing: *Purely assortative (i.e. lifelong duration of MCRB, FSW)* ML with FL ML with FH FL with ML FL with MH MH with FL	B-CI	100% 0% 100% 0% 0%			P_MLFL P_MLFH P_FLML P_FLMH P_MHFL	Assumed for B-CI model structure

MH with FH		100%	P_MHFH	
FH with ML		0%	P_FHML	
FH with MH		100%	P_FHMH	
Sexual mixing: Strongly assortative (i.e. <lifelong duration of MCRB, FSW)	B-CII			Indirectly estimated based on parameter values for mean numbers of partners and population sizes.
ML with FL		100%	P_MLFL	
ML with FH		0%	P_MLFH	
FL with ML		~ 84% – 91% in 2004	P_FLML	
FL with MH		~ 9% – 14% in 2004	P_FLMH	
MH with FL		~ 0.03% constant	P_MHFL	
MH with FH		~ 99.97% constant	P_MHFH	
FH with ML		0%	P_FHML	
FH with MH		100%	P_FHMH	
MIGRATION PARAMETERS (only Taluka A)				
Fraction migrating high-risk groups				
MCLs	B-CM	30% [15 – 60]%	propmigMH	ID1 [44], ID2 [41]
FSWs	B-CM	15% [7.5 – 30]%	propmigFH	ID1 [47]
Duration of migration				
MCLs	B-CM	4 months [2 – 8] months	time_depart/ time_return/ i/ o	ID1 [44], ID2 [41]
FSWs	B-CM	4 months [2 – 8] months	""	ID1 [47]
Number of contacts in POM				
MCLs	B-CM	1.0-fold [0.5 – 2.0]-fold	M_MHFHO/ factorMHO	model assumption
FSWs	B-CM	1.0-fold [0.5 – 2.0]-fold	M_FHMHO/ factorFHO	model assumption
HIV prevalence in POM (constant)				

MCLs	B-CM	17% [8 – 35]%	prev_MH2SA	ID2 [41, 52, 71-73]
FSWs	B-CM	35% [17 – 70]%	prev_FH2SA	DD2 [47, 43, 79]
HIV prevalence in POM (logistic)				
MCLs (2004)	B-CM	17% [8 – 35]%	prevMHout	ID2 [41, 52, 71-73] (model assumption)
MCLs (equilib.)	B-CM	40%		
FSWs (2004)	B-CM	35% [17 – 70]%	prevFHout	DD2 [47, 43, 79] (model assumption)
FSWs (equilib.)	B-CM	80%		
Sexual mixing: Strongly assortative (i.e. <lifelong duration of MCRB, FSW) with migration - when MCLs/FSWs are in the POM	B-CM			Indirectly estimated based on parameter values for mean numbers of partners and population sizes.
ML with FL			P_MLFL	
ML with FH			P_MLFH	
FL with ML		100%	P_FLML	
FL with MH		0	P_FLMH	
MH with FL		~ 91% in 2004	P_MHFL	
MH with FH		~ 9% in 2004	P_MHFH	
FH with ML		~ 0.03% (const.)	P_FHML	
FH with MH		~ 99.97% (const.)	P_FHMH	
migration		0		
MLM with FL		100%	P_MLMFL	
FL with MLM			P_FLMLM	
MHM with FL		0%	P_MHMFL	
FL with MHM		0%	P_FLMHM	
MHM with FH		0%	P_MHMFH	
FH with MHM		0%	P_FHMHM	

FHM with MH		0%	P_FHMMH	
MH with FHM		0%	P_MHFHM	
- when MCLs/FSWs		0%		
are in BT		0%		
ML with FL			P_MLFL	
ML with FH			P_MLFH	
FL with ML		100%	P_FLML	
FL with MH		0%	P_FLMH	
MH with FL		P_FLML*(1-propmigML)	P_MHFL	
MH with FH		P_FLMH*(1-propmigMH)	P_MHFH	
FH with ML		0.03%	P_FHML	
FH with MH		P_MHFH%*(1-propmigFH)	P_FHMH	
migration		0		
MLM with FL		P_FHMH*(1-propmigMH)	P_MLMFL	
FL with MLM			P_FLMLM	
MHM with FL		100%	P_MHMFL	
FL with MHM		P_FLMLM*propmigML	P_FLMHM	
MHM with FH		0.03%	P_MHMFH	
FH with MHM		P_FLMH*(propmigMH)	P_FHMHM	
FHM with MH		99.97%	P_FHMMH	
MH with FHM		100%	P_MHFHM	
		100%		
		P_MHFH*propmigFH		
BIOLOGICAL PARAMETERS				
Duration of HIV infection	B-CI, B-CII, B-CM			ID6 [8, 9, 18, 61, 67, 68, 70] *see Chapter Five: 5.2 for explanation for "re-fit model"
Stage I		6 months (re-fit model: 5 months)	ptoY2_ki	
Stage 2		6.25 years (re-fit model: 8 years)	ptoY3_ki	
Stage 3		2 years (re-fit model: 1 year)	ptoAIDS_ki	
AIDS		1 year (re-fit model : 9 months)	AIDSdeath_ki	
Transmission probabilities – per-partnership(B-C)	B-CI, B-CII, B-CM			ID6 [8] *calculated using Eq.4.4
Male-to-female				
Stage 1		0.3651	BY1_MLFL	

Stage 2		0.1191	BY2_MLFL	
Stage 3		0.1985	BY3_MLFL	
AIDS		0		
Female-to-male				
Stage 1		0.2464	BY1_FLML	
Stage 2		0.0621	BY2_FLML	
Stage 3		0.1035	BY3_FLML	
AIDS		0		
Transmission probabilities – per-contact (B-C)				
Male-to-female				DD6 [8]
Stage 1		0.0057 (0.0083: refit model)	BY1_MHFH	
Stage 2		0.0012	BY2_MHFH	*see Chapter Five: 5.2 for explanation for "re-fit model"
Stage 3		0.0020	BY3_MHFH	
AIDS		0		
Female-to-male				
Stage 1		0.0031 (0.0041: refit model)	BY1_FHMH	
Stage 2		0.0006	BY2_FHMH	
Stage 3		0.0010	BY3_FHMH	
AIDS		0		
Transmission probabilities – per-partnership				ID6 [8] *calculated using Eq.4.4
Male-to-female				
Stage 1		0.517 [0.332-0.641]	BY1_MLFL	
Stage 2		0.337 [0.250-0.437]	BY2_MLFL	
Stage 3		0.496 [0.315-0.578]	BY3_MLFL	
AIDS		0		
Female-to-male				
Stage 1		0.259 [0.166-0.321]	BY1_FLML	
Stage 2		0.169 [0.125-0.219]	BY2_FLML	
Stage 3		0.248 [0.158-0.289]	BY3_FLML	
AIDS		0		
Transmission probabilities – per-				

contact				ID6 [8]
Male-to-female				
Stage 1		0.0031 [0.0028- 0.0034]	BY1_MHFH	
Stage 2		0.0016 [0.0013-0.0019]	BY2_MHFH	
Stage 3		0.0018 [0.0015- 0.0021]	BY3_MHFH	
AIDS		0		
Female-to-male				
Stage 1		0.0015 [0.0012-0.0018]	BY1_FHMH	
Stage 2		0.0008 [0.0005-0.0011]	BY2_FHMH	
Stage 3		0.0010 [0.0007-0.0013]	BY3_FHMH	
AIDS		0		

Legend

DD1: estimated directly using data from Bagalkot District
DD2: estimated directly using data from Districts near Bagalkot District
DD3: estimated directly using data from Karnataka
DD4: estimated directly using data from high-risk states near Karnataka (Maharashtra, Tamil Nadu, Andhra Pradesh)
DD5: estimated directly using data from India
DD6: estimated directly using data from other resource-poor countries (not India)
ID1: indirectly estimated using data from Bagalkot District
ID2: indirectly estimated using data from Districts near Bagalkot District
ID3: indirectly estimated using data from Karnataka
ID4: indirectly estimated using data from high-risk states near Karnataka (Maharashtra, Tamil Nadu, Andhra Pradesh)
ID5: indirectly estimated using data from India
ID6: indirectly estimated using data from other resource-poor countries (not India)

Figure A1.1. Flowchart for sexual behaviour (B-CII): demographic movement in/out of each population by sex/ risk group (black single-headed arrows: inbirth_ki=birth rate into the population; nat_deathk=death rate out; blue single-headed arrows, move_kiki=rate of movement from high- to low-risk and replacement; sexual relationships (red double-headed arrows= low-risk relationships; green double-headed arrows=high-risk contacts). See Chapter Four: Model Development, for description of the structure of the model.

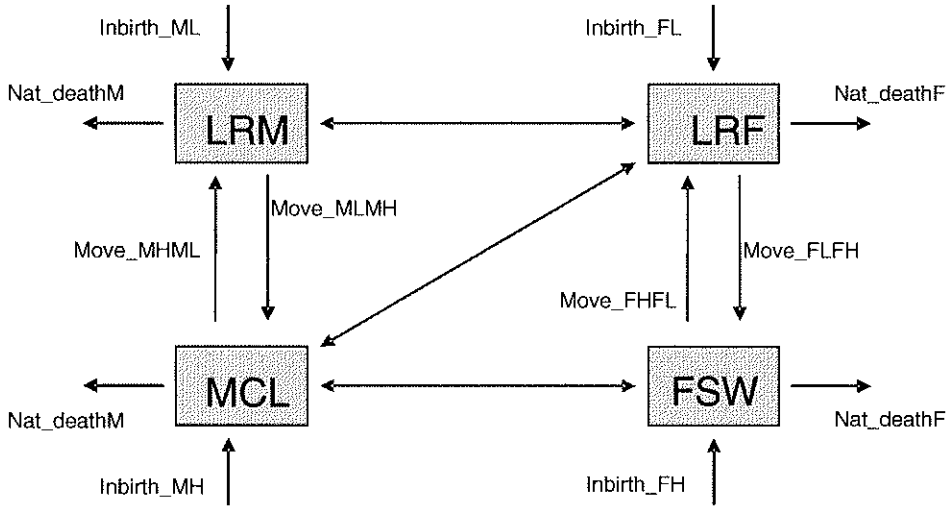


Figure A1.2. A more detailed version of Figures 4.3 and 4.4, representing sexual behaviour in the local population (FL=low-risk females; FH=high-risk females; ML=low-risk males; MH=high-risk males) and the migrating populations (low-risk males [MLM] and high-risk males [MHM]) and the demographic movement in and out of each population (single-headed black arrows) and the sexual relationships assumed (double-headed black arrows). Duration of high-risk behaviour is less than lifelong (single-headed red arrows). In M1 and M2, only high-risk males migrate; in M3, low-risk males also migrate. See Chapter Four for description of model.

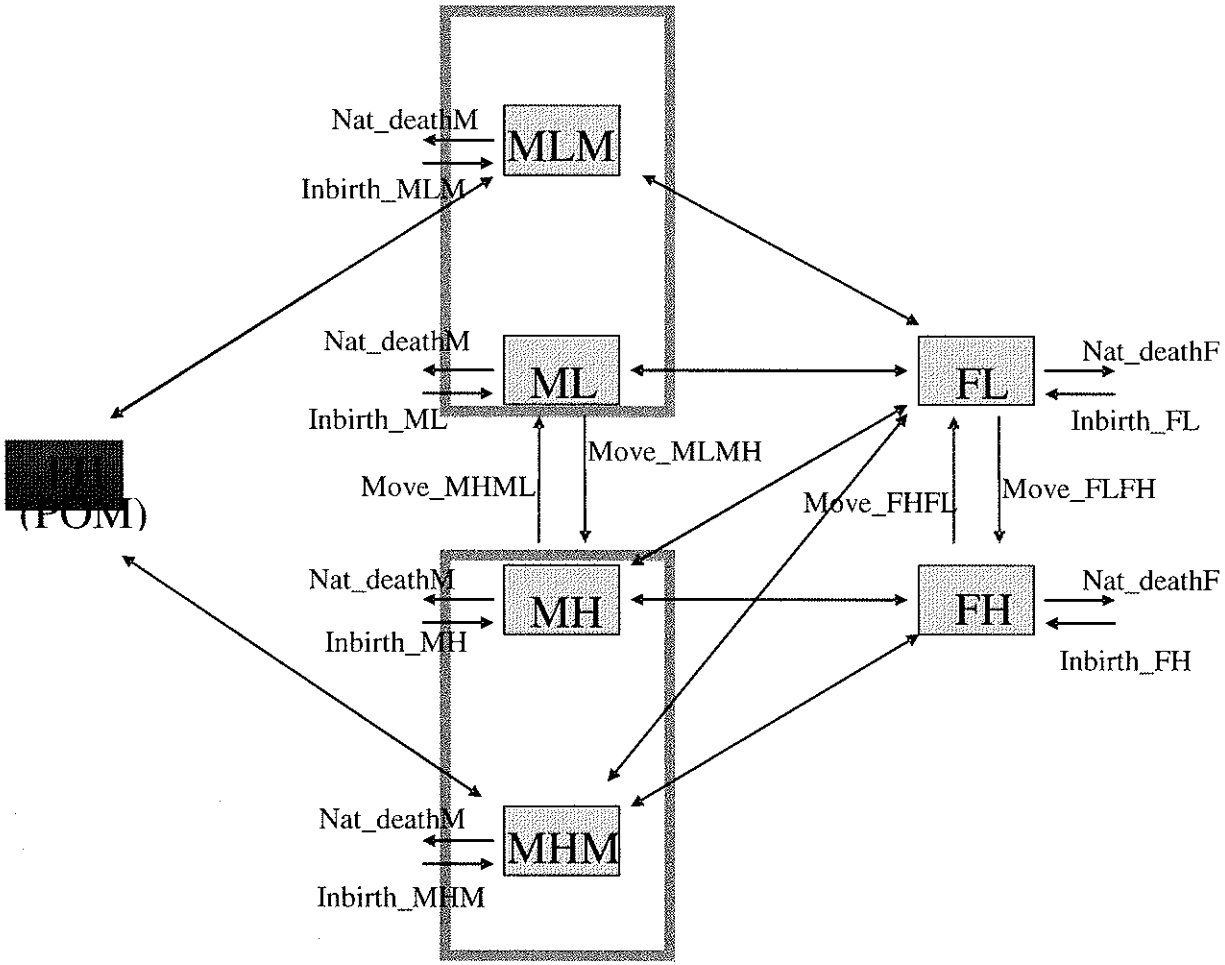


Figure A1.3. A more detailed version of Figure 4.5, representing sexual behaviour in the local population (FL=low-risk females; FH=high-risk females; ML=low-risk males; MH=high-risk males) and the migrating population (high-risk females [FHM]) and the demographic movement in and out of each population (single-headed black arrows) and the sexual relationships assumed (double-headed black arrows). Duration of high-risk behaviour is less than lifelong (single-headed red arrows). See Chapter Four.

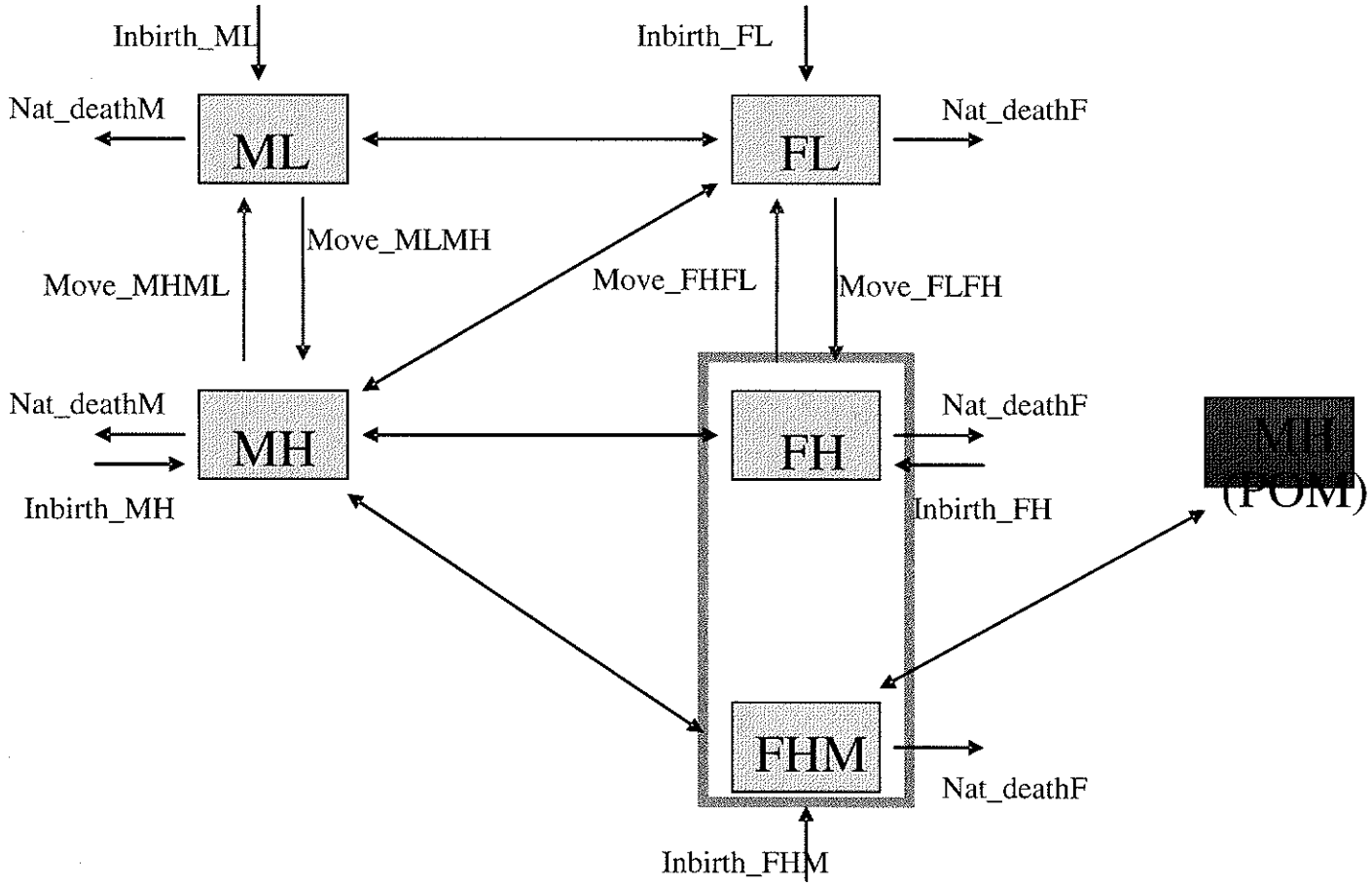


Figure A1.4. A more detailed version of Figure 4.1, which incorporates all of the features of infection progression in each sex (k = male or female) and risk group (i = high-risk or low-risk) and shows the demographic movement in and out of each population (single-headed black arrows) and movement from one infected stage to the next by a rate equal to the inverse of the mean duration of time spent in each stage (single-headed green arrows: X = susceptible; Y1 = infected, stage 1; Y2 = infected, stage 2; Y3 = infected, stage 3; AIDS = infected, AIDS stage). See Chapter Four: Model Development, for description of the structure of the model.

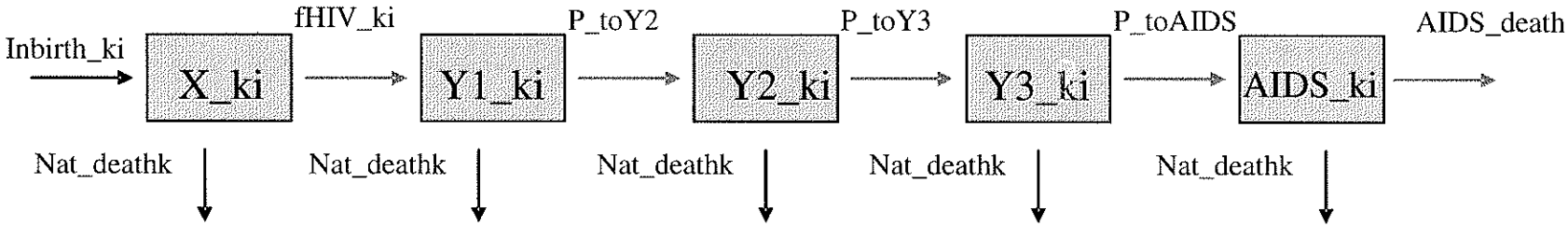


Figure A1.5. Equations used in model

Equation set 1. Low-risk equations

$$\frac{dX_{kl}}{dt} = in\_birth_{kl} * N_{tot} - f\_HIV_{kl} * X_{kl} + move_{khkl} * \sum X_{kh} - move_{klkh} * (\frac{\sum N_{kh}}{XY123_{kl}}) - nat\_death_{k} * X_{kl}$$

$$\frac{dY1_{kl}}{dt} = f\_HIV_{kl} * X_{kl} - p\_toY2 * Y1_{kl} + move_{khkl} * \sum Y1_{kh} - move_{klkh} * (\frac{\sum N_{kh}}{XY123_{kl}}) - nat\_death_{k} * Y1_{kl}$$

$$\frac{dY2_{kl}}{dt} = p\_toY2 * Y1_{kl} + p\_toY3 * Y1_{kl} + move_{khkl} * \sum Y2_{kh} - move_{klkh} * (\frac{\sum N_{kh}}{XY123_{kl}}) - nat\_death_{k} * Y2_{kl}$$

$$\frac{dY3_{kl}}{dt} = p\_toY3 * Y3_{kl} + p\_toAIDS * Y3_{kl} + move_{khkl} * \sum Y3_{kh} - move_{klkh} * (\frac{\sum N_{kh}}{XY123_{kl}}) - nat\_death_{k} * Y3_{kl}$$

$$\frac{dAIDS_{kl}}{dt} = p\_toAIDS * Y3_{kl} + move_{khkl} * \sum AIDS_{kh} - (AIDS\_death + nat\_death_{k}) * AIDS_{kl}$$

Equation set 2. High-risk equations

$$\frac{dX_{kh}}{dt} = in\_birth_{kh} * N_{tot} - f\_HIV_{kh} * X_{kh} - move_{khkl} * X_{kh} + move_{klkh} * (\frac{N_{kh}}{XY123_{kl}}) - nat\_death_{k} * X_{kh}$$

$$\frac{dY1_{kh}}{dt} = f\_HIV_{kh} * X_{kh} - p\_toY2 * Y1_{kh} - move_{khkl} * Y1_{kh} + move_{klkh} * (\frac{N_{kh}}{XY123_{kl}}) - nat\_death_{k} * Y1_{kh}$$

$$\frac{dY2_{kh}}{dt} = p\_toY2 * Y1_{kh} + p\_toY3 * Y1_{kh} - move_{khkl} * Y2_{kh} + move_{klkh} * (\frac{N_{kh}}{XY123_{kl}}) - nat\_death_{k} * Y2_{kh}$$

$$\frac{dY3_{kh}}{dt} = p\_toY3 * Y3_{kh} + p\_toAIDS * Y3_{kh} - move_{khkl} * Y3_{kh} + move_{klkh} * (\frac{N_{kh}}{XY123_{kl}}) - nat\_death_{k} * Y3_{kh}$$

$$\frac{dAIDS_{kh}}{dt} = p\_toAIDS * Y3_{kh} - move_{khkl} * AIDS_{kh} - (AIDS\_death + nat\_death_{k}) * AIDS_{kh}$$

Definitions

k = sex (male [m] or female [f])
i = risk group (high- [h] or low-risk [l])\

Demographics

X_{ki} = number of susceptible individuals

$Y1_{ki}$ = number of individuals in infected stage 1

$Y2_{ki}$ = number of individuals in infected stage 2

$Y3_{ki}$ = number of individuals in infected stage 3

$AIDS_{ki}$ = number of individuals in the AIDS stage of infection

$Ntot$ = total population

N_{ki} = the total population in each risk/gender group

$XY123_{ki}$ = the total population in the susceptible and first three infected stages (not the AIDS stage)

$inbirth_{ki} = preAIDSgrowth_k * propB_{ki} * ratio_{fm\,/\,mf}$

$preAIDSgrowth_k = nat\_death_k * pop\_growth$

$propB_{ki}$ = proportion of people in each risk/gender group

$ratio_{fm\,/\,mf}$ = ratio of females to males/ males to females

pop_growth = population growth rate in the absence of AIDS

$move_{khkl} = \frac{1}{duration_{kh}}$ = the mean rate at which people move from high-risk groups to low-risk groups

$move_{klkh} = move_{khkl}$ the mean rate at which people move from low-risk groups to high-risk groups (replace high-risk)

$duration_{kh}$ = the mean duration of time high-risk individuals remain high-risk before becoming low-risk

$nat\_death_k = \frac{1}{sexuallyactive}$ = the inverse of the mean number of sexually active years, the natural death rate of the population

$$AIDS\_deaths = \frac{1}{time\_todeathAIDS}$$ = the inverse of the mean duration of time living with AIDS until death

Acquisition and transmission

$fHIV_{ki}$ = the force of infection

$fHIV_{fi} = M_{fi} * \sum_{i}(P_{fimi} * Exposure\_to_{mi})$ #note : fimi can equal flml, flmh or fhmh for strongly assortative mixing

$fHIV_{mi} = M_{mi} * \sum_{i}(P_{mifi} * Exposure\_to_{fi})$ #note : mifi can equal mlfl, mhfl or mhfh for strongly assortative mixing

M_{fi} = the mean number of partners that females (low- or high-risk) have with males from low- or high-risk groups

$$M_{fi} = \frac{numberpartnership_{mifi}}{XY123_{fi}}$$

$numberpartnership_{mifi} = M_{mi} * XY123_{mi} * P_{mifi}$

M_{mi} = the mean number of partners that males (low- or high-risk) have with females from low- or high-risk groups

P_{fimi} = the probability that female individuals in risk group i have sexual relationships with male individuals in risk group i

P_{mifi} = the probability that male individuals in risk group i have sexual relationships with female individuals in risk group i

$$Exposure\_to_{mi} = \frac{(BY1_{mifi} * Y1_{mi} + BY2_{mifi} * Y2_{mi} + BY3_{mifi} * Y3_{mi})}{XY123_{mi}}$$

$$Exposure\_to_{fi} = \frac{(BY1_{fimi} * Y1_{fi} + BY2_{fimi} * Y2_{fi} + BY3_{fimi} * Y3_{fi})}{XY123_{fi}}$$

$BY1_{mifi}$ = the probability of transmission of HIV in the first infected stage from males in one risk group to females in one risk group

$BY2_{mifi}$ = the probability of transmission of HIV in the second infected stage from males in one risk group to females in one risk group

$BY3_{mifi}$ = the probability of transmission of HIV in the third infected stage from males in one risk group to females in one risk group

$BY1_{fimi}$ = the probability of transmission of HIV in the first infected stage from females in one risk group to males in one risk group

$BY2_{fimi}$ the probability of transmission of HIV in the second infected stage from females in one risk group to males in one risk group

$BY3_{fimi}$ the probability of transmission of HIV in the third infected stage from females in one risk group to males in one risk group

Migration-specific parameters:

- Demographic

When we include a migrating population of either a proportion of high-risk males, high- and low-risk males, or high-risk females, the above equations remain the same, but we have an additional set for each migrating population. In this case, the 'i' for risk group is: high-risk who migrate [hm] or high-risk who do not migrate [h]; low-risk who migrate [lm] or low-risk who do not migrate [l]

- Force of infection

When we include migration in the model, the force of infection for migrating groups is the same as for non-migrating groups, with an additional term to take into account the sexual relationships in the place of migration. When the migrating groups are in the home Taluka, the additional term is set to zero; when migrating groups are in the place of migration, the part of the force of infection for relationships in the home Taluka is set to zero. This is accomplished using a set of "pulses" in Berkeley Madonna:

Pulses:

init o = 0
init i = 1
d/dt(o) = PULSE(1,1976+time_depart,1) + PULSE(-1,1976+time_return,1)
d/dt(i) = PULSE(-1,1976+time_depart,1) + PULSE(1,1976+time_return,1)
i = time that migrants are in the home Taluka
o = time that migrants are in the place of migration

time_depart = time of the year (in fraction form) that the migrating population leaves
time_return = time of the year (in frction form) that the migrating population returns (i.e. if leaving for six months, the migrating population might leave at 0.1 and return at 0.6, so that 0.5 or 50% of the year is spent in the place of migration and 50% spent in the place of origin)

$$fHIV_{fmi} = i * \left(M_{fi} * \sum_{i} (P_{fimi} * Exposure\_to_{mi}) \right) + o * \left(M_{mhfho} * P_{mhfho} * Exposure\_to_{fho} \right)$$ #note : fimi can equal flml, flmh or fhmh for strongly assortative mixing

$$fHIV_{mhi} = i * \left(M_{mi} * \sum_{i} (P_{mifi} * Exposure\_to_{fi}) \right) + o * \left(M_{fhmho} * P_{fhmho} * Exposure\_to_{mho} \right)$$ #note : mifi can equal mlfl, mhfl or mhfh for strongly assortative mixing

M_{mhfho} = mean number of FSW partners that clients (or low-risk males) have in the place of migration

M_{fhmho} = mean number of client partners that FSWs have in the place of migration

P_{mhfho} = probability that clients (or low-risk males) have FSW partners in the place of migration (equal to 100%)

P_{fhmho} = probability that FSWs have client partners in the place of migration (equal to 100%)

$Exposure\_to_{mho} = BY_{mho} * prev_{mho}$

$Exposure\_to_{fho} = BY_{fho} * prev_{fho}$

BY_{mho} = probability of infection by high-risk males in the place of migration

BY_{fho} = probability of infection by high-risk females in the place of migration

$prev_{mho}$ = 1) constant HIV prevalence in clients in the place of migration or 2) prevalence defined by the following logistic curve:

$prev_{fho}$ = 1) constant HIV prevalence in FSWs in the place of migration or 2) prevalence defined by the following logistic curve:

$$\frac{d}{dt}(prev_{mho}) = rad_{mho} * prev_{mho} * \frac{(k_{mho} - prev_{mho})}{k_{mho}}$$

$$\frac{d}{dt}(prev_{fho}) = rad_{fho} * prev_{fho} * \frac{(k_{fho} - prev_{fho})}{k_{fho}}$$

rad_{mho} = a factor that determines the speed of the epidemic growth in clients in the place of migration

rad_{fho} = a factor that determines the speed of the epidemic growth in FSWs in the place of migration

k_{fho} = "carrying capacity" of logistic curve epidemic – the equilibrium model HIV prevalence in clients in the place of migration

k_{fho} = "carrying capacity" of logistic curve epidemic – the equilibrium HIV model prevalence in FSWs in the place of migration

- High-risk partners

When migrant clients (or clients and low-risk males) are in their home Talukas or in the place of migration, we keep M_{fi} (the mean number of partners that females (low- or high-risk) have with males from low- or high-risk groups) constant. This means that when migrant clients (or clients and low-risk males) are in the place of migration, since there are fewer males but the same number of mean FSW partners per client, the mean number of client partners per FSW in the home Taluka will decrease proportionate to the size of the migrating client population for the duration of time clients are in the place of migration:

$$M_{fi} = i * \left(\frac{numberpartnership_{mifi}}{XY123_{fi}} \right) + o * (1 - propmig_{mh}) * \left(\frac{numberpartnership_{mifi}}{XY123_{fi}} \right)$$

$propmig_{mh}$ = the proportion of clients migrating

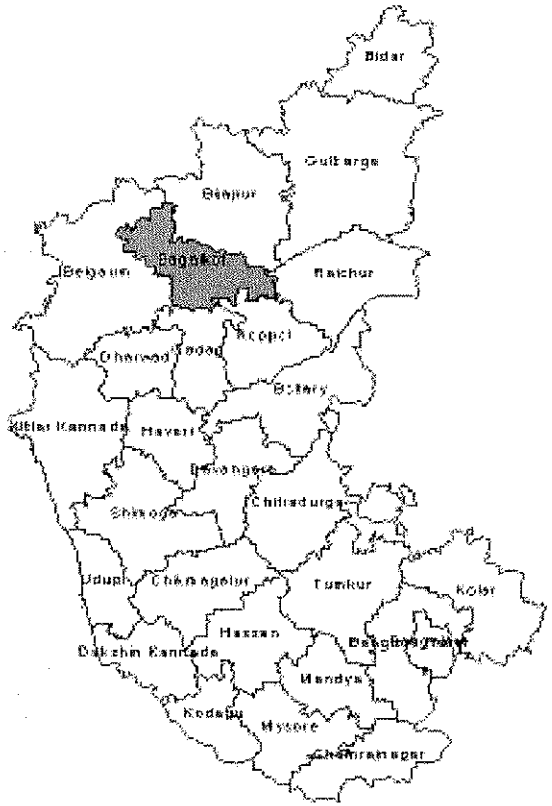
However, when FSWs migrate, there will be fewer FSWs but the same number of mean FSW partners per client, so the mean number of client partners per FSW in the home Taluka will increase proportionate to the size of the migrating client population for the duration of time FSWs are in the place of migration:

$$M_{fi} = i * \left(\frac{numberpartnership_{mifi}}{XY123_{fi}} \right) + o * (1 + propmig_{fh}) * \left(\frac{numberpartnership_{mifi}}{XY123_{fi}} \right)$$

$propmig_{fh}$ = the proportion of FSWs migrating

We assume that even when low-risk males migrate and are clients in the place of migration, the mean number of low-risk female partners per low-risk male does not change, and neither does the mean number of low-risk male partners per low-risk female. This is because we assume that low-risk groups have long-term partnerships that wouldn't be affected in the low-risk male was a migrant or not (i.e. marital relationships).

Figure A1.6. Map of Karnataka (left) showing Bagalkot District highlighted; map of Bagalkot District (right) showing where its six Talukas are located; three Talukas in Bagalkot District are of interest in this study



Map of Karnataka

APPENDIX 2: Additional results from Chapters Five and Six

Figure A2.1. B-CI Scenario - high-risk male and female prevalence in Taluka B

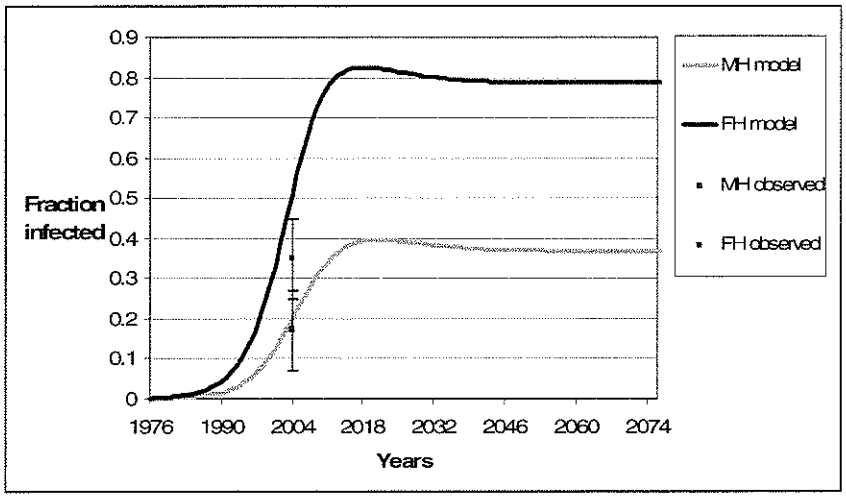


Figure A2.2. B-CII Scenario – overall, male and female prevalence in Taluka A

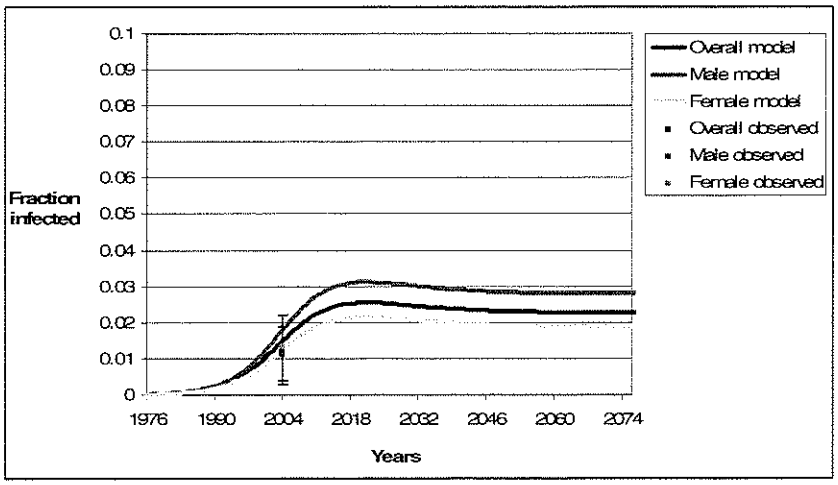


Figure A2.3. B-CII Scenario - high-risk male and female prevalence in Taluka A

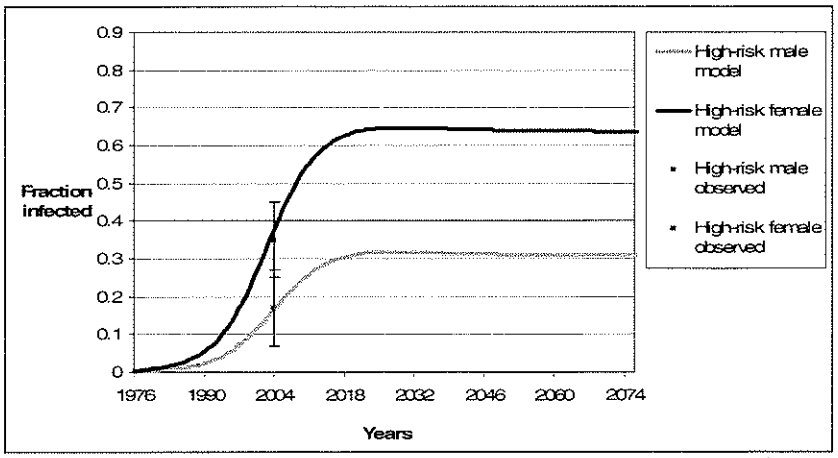


Figure A2.4. 2004 overall model HIV prevalence and average number of MCL partners per FSW in all three Talukas as each Taluka's FSW size is exchanged for the others (partnerships constant, so average number of MCL partners per FSW decrease with increasing FSW size)

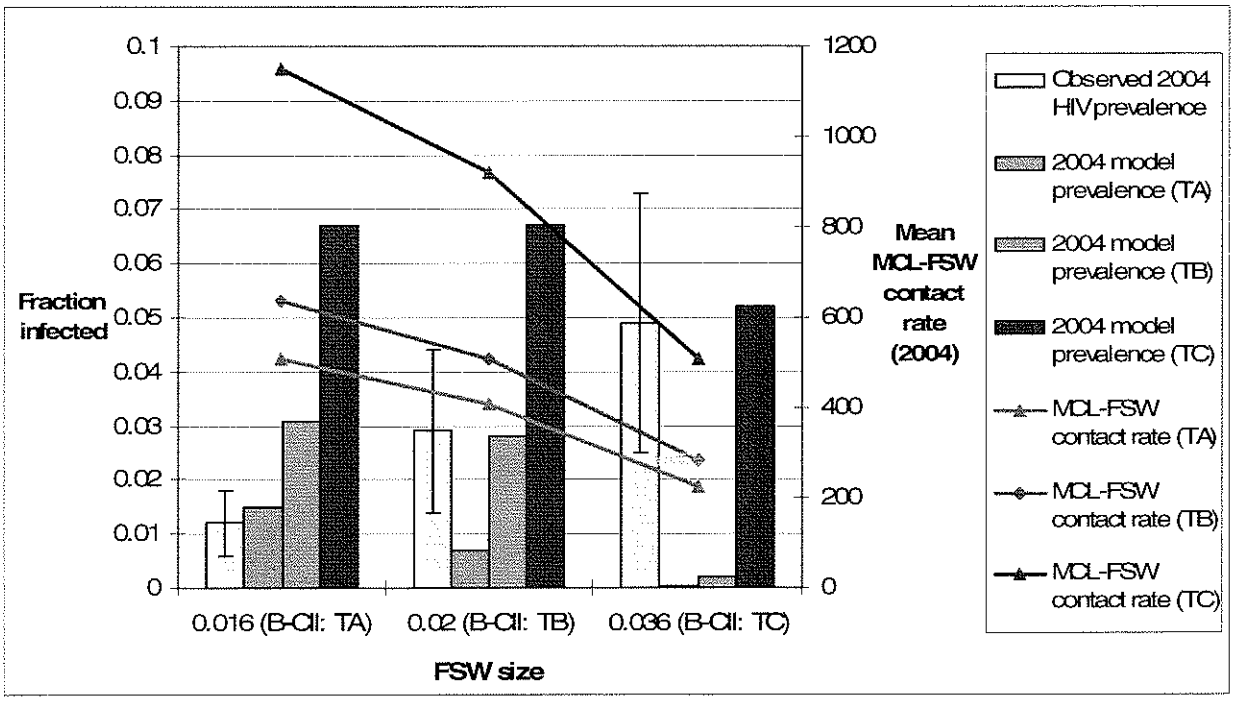


Figure A2.5: 2004 overall model HIV prevalence and average number of FSW partners per MCL in all three Talukas as each Taluka's MCL size is exchanged for the others (partnerships constant, so average number of FSW partners per MCL decrease with increasing MCL size)

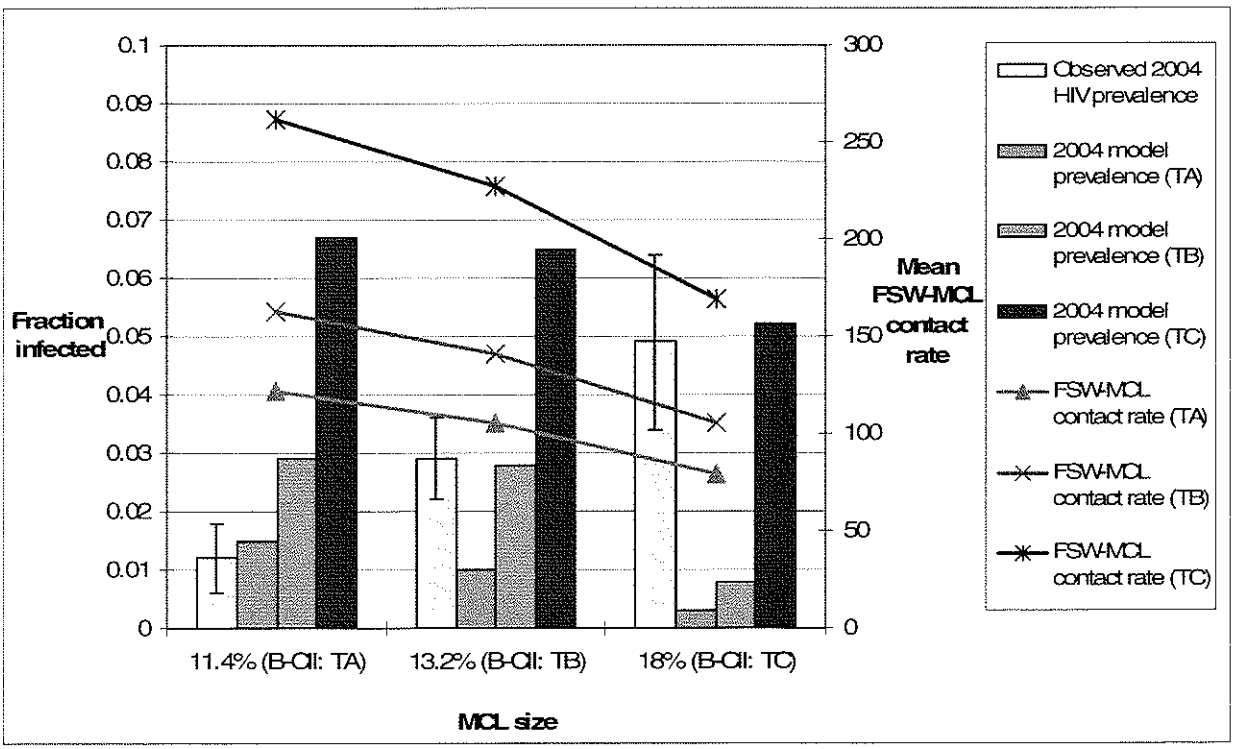


Figure A2.6. FSW prevalence with varied condom use in migrants only in Taluka A (M.3)

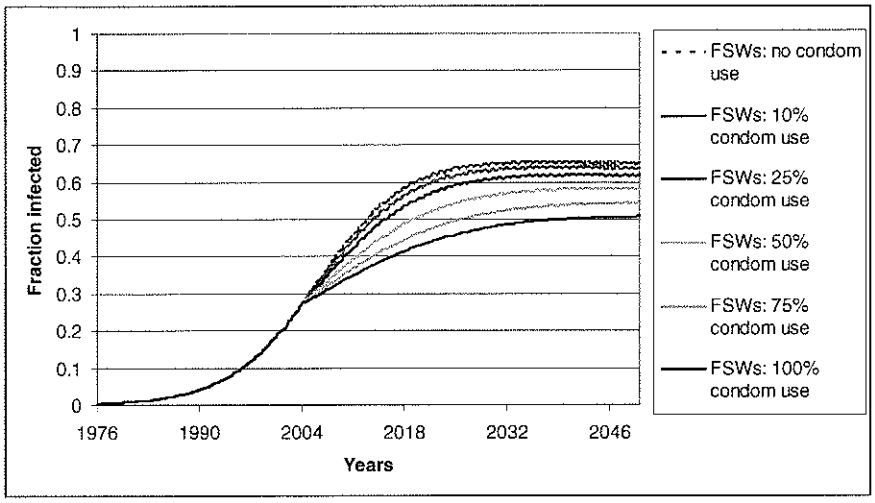


Figure A2.7. FSW prevalence with varied condom use in non-migrants only in Taluka A (M.3)

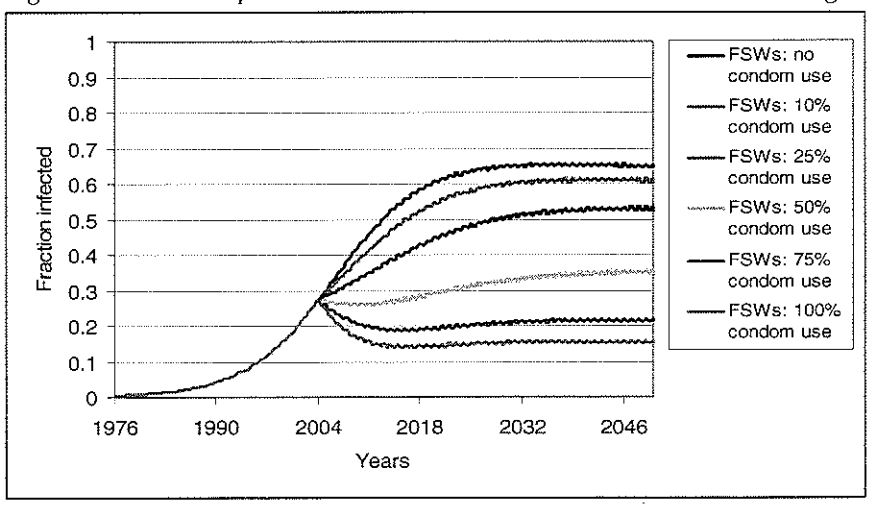


Figure A2.8. FSW prevalence with varied condom use in all high-risk groups in Taluka A (M.3)

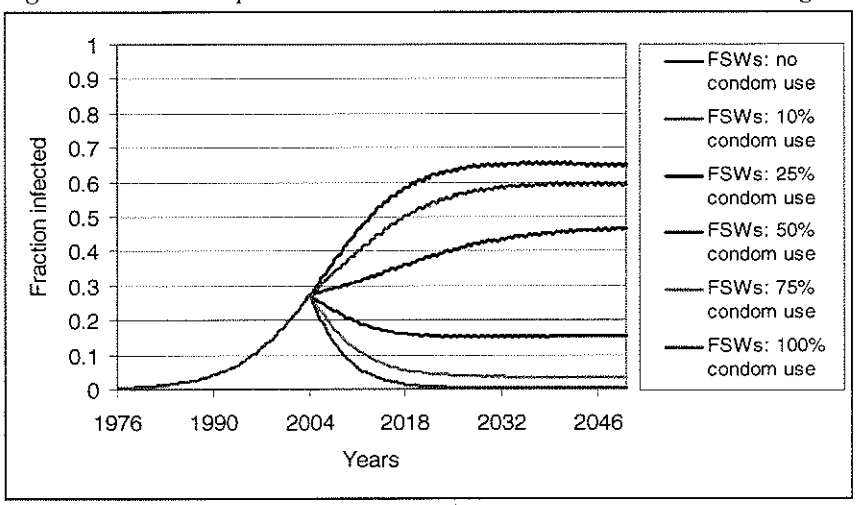


Table A2.1. Taluka A: Overall model 2004, peak and equilibrium HIV prevalence and ranking of parameters

Ranking (abs effect: range)	**Parameter**	**Scenario**	**B-C value [range]**	**Other parameters varied [range]**	**2004 [range]**	**Peak [range]**	**Equilibrium [range]**
9 (increase)	Duration of FSW (years)	B-CI	lifelong [4.5 – 18]	none	1.3% [0.3 – 0.9]%	1.9% [1.6 - 1.8]%	1.5% [1.5 - 1.5]%
10 (decrease)	Duration MCRB (years) (partnerships const)	B-CI	lifelong [5 – 20]	2004 MCL size: 10.8% [2.4 - 8.8]%; Mean FSW partners/ MCL: 99 [457 – 122]	1.3% [2.0 – 1.5]%	1.9% [2.5 – 1.9]%	1.5% [1.7 – 1.5]%
8 (decrease)	Overall population size	B-CII	1.0-fold [0.5 – 2.0]-fold	none	1.5% [1.8 – 1.1]%	2.5% (2020) [2.6 (2018) – 2.5 (2022)]%	2.3% (2074) [2.3 (2087) – 2.2 (2070)]%
3 (increase)	Partnerships vary: mean numbers of FSW partners/ MCL	B-CII (PV1)	122 [61 – 244]	Mean number of MCL partners per FSW (2004): ~510 [255 – 1020]	1.5% [0.01 – 5.7]%	2.5% (2020) [0.01 (1979 – 6.5 (1993)]%	2.3% (2074) [~0 (2007) – 4.8 (2049)]%
1 (increase)	Partnerships vary: FSW size (2004)	B-CII (PV2)	1.6% [0.8 – 3.2]%	Mean FSW partners /MCL 122 [61 – 244]	1.5% [0.1 – 6.1]%	2.5% (2020) [1.1 (2500) – 6.2 (2001)]%	2.3% (2074) [1.1 (2500)– 4.8 (2052)]%
2 (increase)	Partnerships vary: MCL size (lifetime)	B-CII (PV3)	11.4% [5.7 – 22.8]%	Mean MCL partners/ FSW (2004): ~510 [255 – 1020]	1.5% [0.1 – 5.8]%	2.5% (2020) [0.9 (2100) – 5.8 (2008)]%	2.3% (2074) [0.9 (2100) – 5.0 (2063)]%
6	Partnerships vary: MCL size	B-CII (PV4)	11.4% [5.7 – 22.8]%	FSW size: 1.6% [0.8 – 3.2]%	1.5% [0.9 – 2.3]%	2.5% (2020) [1.3 (2017) – 5.0 (2023)]%	2.3% (2074) [1.2 (2082) - 4.5 (2075)]%
5 (decrease)	Partnerships constant: FSW size (2004)	B-CII (PC1)	1.6% [0.8 – 3.2]%	Mean number of MCL partners per FSW (2004): ~510 [1020 – 255]	1.5% [3.1 – 0.1]%	2.5% (2020) [3.1 (2005) – 1.8 (2150)]%	2.3% (2074) [2.7 (2056)– 1.8 (2150)]%
4 (decrease)	Partnerships constant: MCL size (lifetime)	B-CII (PC2)	11.4% [5.7 – 22.8]%	Mean number of FSW partners per MCL (const.) 122 [244 – 61]	1.5% [3.9 – 0.1]%	2.5% (2020) [4.2 (1997)– 2.3 (2110)]%	2.3% (2074) [3.1 (2050)– 2.3 (2150)]%
7 (increase)	Mean number of LRF partners per male	B-CII	10 [5 – 20]	Mean number of male partners per LRF in 2004: ~10 [5 – 20]	1.5% [1.2 – 2.0]%	2.5% (2020) [2.2 – 4.0]%	2.3% (2074) [1.9 – 3.8]%

Table A2.2. Taluka B: Overall model 2004, peak and equilibrium HIV prevalence and ranking of parameters

Ranking (abs effect: range)	**Parameter**	**Scenario**	**B-C value [range]**	**Other parameters varied [range]**	**2004 [range]**	**Peak [range]**	**Equilibrium [range]**
7 (increase)	Duration of FSW (years)	B-CI	lifelong [4.5 – 18]	none	1.7% [0.6 – 1.4]%	2.3% [2.2 – 2.3]%	1.7% [1.9 – 1.8]%
9 (decrease)	Duration MCRB (years) (partnerships const)	B-CI	lifelong [5 – 20]	2004 MCL size: 12.4% [2.6 – 9.6]%; Mean FSW partners/ MCL: 108 [519 – 141]	1.7% [2.4 – 2.2]%	2.3% [3.2 – 2.5]%	1.7% [2.1 – 1.9]%
7 (decrease)	Overall population size	B-CII	1.0-fold [0.5 – 2.0]-fold	none	2.2% [2.6 – 1.8]%	3.2% (2016) [3.3 (2015 – 3.1 (2017)]%	2.7% (2076) [2.8 (2061 – 2.7 (2069]%
3 (increase)	Mean FSW partners/ MCL	B-CII (PV1)	141 [70 – 282]	Mean number of MCL partners per FSW (2004): ~510 [255 – 1020]	2.2% [0.01 – 6.7]%	3.2% (2016) [0.01 (1980)– 8.0 (1992)]%	2.7% (2076) [~0 (2006)– 5.6 (2050)]%
1 (increase)	Partnerships vary: FSW size (2004)	B-CII (PV2)	2.0% [1.0 – 4.0]%	Mean FS W partners/ MCL (const.) 141 [71 – 282]	2.2% [0.1 – 7.6]%	3.2% (2016) [1.5 (2072) – 7.9 (1999)]%	2.7% (2076) [1.4 (2100) – 5.8 (2062)]%
2 (increase)	Partnerships vary: MCL size (lifetime)	B-CII (PV3)	13.2% [6.6 – 26.4]%	Mean MCL partners/ FSW (2004): ~510 [255 – 1020]	2.2% [0.1 – 7.2]%	3.2% (2016) [1.2 (2065)– 7.2 (2005)]%	2.7% (2076) [1.2 (2100)– 5.9 (2065)]%
6 (increase)	Partnerships vary: MCL size	B-CII (PV4)	13.2% [6.6 – 26.4]%	FSW size: 2.0% [1.0 – 4.0]%	2.2% [1.3 – 3.4]%	3.2% (2016) [1.6 (2014) – 6.3 (2019)]%	2.7% (2076) [1.4 (2065) – 5.3 (2074)]%
4 (decrease)	Partnerships constant: FSW size (2004)	B-CII (PC1)	2.0% [1.0 – 4.0]%	Mean MCL partners/ FSW (2004): ~510 [1020 – 255]	2.2% [3.9 – 0.1]%	3.2% (2016) [3.9 (2003)– 2.4 (2079)]%	2.7% (2076) [3.1 (2052)– 2.3 (2150)]%
5 (decrease)	Partnerships constant: MCL size (lifetime)	B-CII (PC2)	13.2% [6.6 – 26.4]%	Mean number of FSW partners per MCL (const.) 141 [282 – 71]	2.2% [3.8 – 0.1]%	3.2% (2016) [4.1 (1998)– 3.1 (2074)]%	2.7% (2076) [3.0 (2044)– 2.9 (2150)]%
8 (increase)	Mean LRF partners/ male	B-CII	10 [5 – 20]	Mean number of male partners per LRF in 2004: ~10 [5 – 20]	2.2% [2.0 – 2.7]%	3.2% (2016) [2.8 – 4.7]%	2.7% (2076) [2.3 – 4.3]%

Table A2.3. Taluka C: Overall model 2004, peak and equilibrium HIV prevalence and ranking of parameters

Ranking (abs effect: range)	**Parameter**	**Scenario**	**B-C value [range]**	**Other parameters varied [range]**	**2004 [range]**	**Peak [range]**	**Equilibrium [range]**
7 (increase)	Duration of FSW (years)	B-CI	lifelong [4.5 – 18]	none	4.1% [2.9 – 3.8]%	4.3% [4.0 – 4.3]%	3.2% [3.3 – 3.1]%
8 (decrease)	Duration MCRB (years) (partnerships const)	B-CI	lifelong [5 – 20]	2004 MCL size: 16.6% [3.4 – 12.5]%; Mean FSW partners/ MCL: 137 [182 – 662]	4.1% [4.0 - 4.6]%	4.3% [5.7 - 4.6]%	3.2% [3.4 - 3.2]%
7 (decrease)	Overall population size	B-CII	1.0-fold [0.5 – 2.0]-fold	none	5.2% [5.7 – 4.6]%	5.8% (2010) [6.0 (2009) - 5.7 (2012)]%	4.7% (2063) [4.8 (2081) - 4.5 (2064)]%
3 (increase)	Mean number of FSW partners per MCL	B-CII (PV1)	169 [85 – 238]	Mean number of MCL partners per FSW (2004): ~510 [255 – 1020]	5.2% [0.02 – 10.8]%	5.8% (2010) [0.06 (2500) – 13.5 (1990)]%	4.7% (2063) [0.06 (2500) – 9.2 (2065)]%
1 (increase)	Partnerships vary: FSW size (2004)	B-CII (PV2)	3.6% [1.8 – 7.2]%	Mean number of FSW partners per MCL (const.) 169 [85 – 238]	5.2% [0.4 – 12.9]%	5.8% (2010) [2.6 (2047) – 14.3 (1996)]%	4.7% (2063) [2.5 (2095) – 10.1 (2063)]%
2 (increase)	Partnerships vary: MCL size (lifetime)	B-CII (PV3)	18.0% [9.0 – 36.0]%	Mean number of MCL partners per FSW (2004): ~510 [255 – 1020]	5.2% [0.4 – 12.5]%	5.8% (2010) [2.3 (2038)– 12.6 (2002)]%	4.7% (2063) [2.1 (2090) – 9.7 (2073)]%
4 (increase)	Partnerships vary: FSW size	B-CII (PV4)	3.6% [1.8 – 7.2]%	MCL size (lifetime): 18.0% [9.0 – 36.0]%	5.2% [2.9 – 9.1]%	5.8% (2010) [3.0 (2008) – 11.2 (2013)]%	4.7% (2063) [2.4 (2057) – 9.0 (2018)]
4 (decrease)	Partnerships constant: FSW size (2004)	B-CII (PC1)	3.6% [1.8 – 7.2]%	Mean number of MCL partners per FSW (2004): ~510 [1020 – 255]	5.2% [6.7 – 0.5]%	5.8% (2010) [6.9 (2000) – 4.7 (2044)]%	4.7% (2063) [5.3 (2055)– 4.2 (2105)]%
5 (decrease)	Partnerships constant: MCL size (lifetime)	B-CII (PC2)	18.0% [9.0 – 36.0]%	Mean number of FSW partners per MCL (const.) 169 [85 – 238]	5.2% [6.5 – 0.6]%	5.8% (2010) [7.4 (1995)– 5.6 (2043)]%	4.7% (2063) [5.2 (2050) – 5.1 (2106)]%
6 (increase)	Mean number of LRF partners per male	B-CII	10 [5 – 20]	Mean number of male partners per LRF in 2004: ~10 [5 – 20]	5.2% [4.7 – 6.9]%	5.8% (2010) [5.1 – 8.2]%	4.7% (2063) [4.0 – 7.4]%

Table A2.4. Taluka A results: migration parameters

Ranking (absolute effect on 2004 model prev) [range]	Scenario (all B-CM)	Parameter	B-C value [range]	Other parameters varied [range]	B-C overall 2004 HIV prevalence, B-CM [range]	B-C overall peak HIV prevalence [range]	B-C overall equilibrium HIV prev. [range]
10 (constant)	MCLs migrate: constant HIV prevalence in POM	Migrating MCL size	30% [15 – 60]%	Mean number MCL partners per FSW (M_FH) : propmigMH*M_FH while MCLs in POM 2004: ~153 [77 – 306]	1.2% 1.7% [1.5 – 1.5]%	1.9% 1.8% [1.8 – 1.6]%	1.6% 1.5% [1.5 – 1.5]%
10 (constant)		Duration of migration	4 months [2 – 8] months	None	1.2% 1.7% [1.5 – 1.5]%	1.9% 1.8% [1.8 – 1.6]%	1.6% 1.5% [1.5 – 1.5]%
7 (increase)		Number of high-risk contacts in POM	1.0-fold [0.5 – 2.0]-fold	None	1.2% 1.7% [1.6 – 1.9]%	1.9% 1.8% [1.6 – 1.9]%	1.6% 1.5% [1.5 – 1.7]%
6 (increase)		HIV prevalence in POM	35% [17 – 70%	None	1.2% 1.7% [1.5 – 1.9]%	1.9% 1.8% [1.7 – 2.0]%	1.6% 1.5% [1.5 – 1.7]%
8 (decrease)	MCLs migrate : logistic HIV prevalence in POM	Migrating MCL size	30% [15 – 60]%	Mean number MCL partners per FSW (M_FH) : propmigMH*M_FH while MCLs in POM 2004: ~153 [77 – 306]	1.2% 1.0% [1.1 – 0.8]%	1.9% 1.8% [1.8 – 1.7]%	1.6% 1.5% [1.6 – 1.5]%
5 (decrease)		Duration of migration	4 months [2 – 8] months	None	1.2% 1.0% [1.1 – 0.6]%	1.9% 1.8% [1.8 – 1.7]%	1.6% 1.5% [1.6 – 1.5]%
6 (increase)		Number of high-risk contacts in POM	1.0-fold [0.5 – 2.0]-fold	None	1.2% 1.0% [0.8 – 1.2]%	1.9% 1.8% [1.7 – 1.9]%	1.6% 1.5% [1.5 – 1.6]%
7 (increase)		HIV prevalence in POM (2004)	35% [17 – 70%	None	1.2% 1.0% [0.9 – 1.2]%	1.9% 1.8% [1.7 – 1.8]%	1.6% 1.5% [1.5 – 1.6]%
3 (increase)	All males migrate : logistic HIV	Migrating MCL size	30% [15 – 60]%	Mean number MCL partners per FSW (M_FH) : propmigMH*M_FH while	1.2% 1.5% [1.3 – 2.1]%	1.9% 3.7% [2.7 – 5.5]%	1.6% 3.3% [2.5 – 5.1]%

	prevalence in POM			MCLs in POM 2004: ~153 [77 – 306]			
4 (increase)		Duration of migration	4 months [2 – 8] months	None	1.2% 1.5% [1.2 – 2.1]%	1.9% 3.7% [3.0 – 4.6]%	1.6% 3.3% [2.7 – 4.1]%
2 (increase)		Number of high-risk contacts in POM	1.0-fold [0.5 – 2.0]-fold	None	1.2% 1.5% [1.0 – 2.4]%	1.9% 3.7% [2.8 – 4.8]%	1.6% 3.3% [2.6 – 4.2]%
1 (increase)		HIV prevalence in POM (2004)	35% [17 – 70%	None	1.2% 1.5% [1.0 – 2.7]%	1.9% 3.7% [3.5 – 3.8]%	1.6% 3.3% [3.3 – 3.4]%
6 (increase)	FSWs migrate : constant HIV prevalence in POM	Migrating FSW size	15% [7.5 – 30]%	None	1.2% 2.0% [1.8 – 2.2]%	1.9% 2.0% [2.0 – 2.2]%	1.6% 1.8% [1.7 – 1.9]%
9 (increase)		Duration of migration	4 months [2 – 8] months	None	1.2% 2.0% [1.9 – 2.0]%	1.9% 2.0% [2.0 – 2.1]%	1.6% 1.8% [1.7 – 1.8]%
7 (increase)		Number of high-risk contacts in POM	1.0-fold [0.5 – 2.0]-fold	None	1.2% 2.0% [1.8 – 2.1]%	1.9% 2.0% [2.0 – 2.1]%	1.6% 1.8% [1.7 – 1.8]%
6 (increase)		HIV prevalence in POM	17% [8 – 35]%	None	1.2% 2.0% [1.7 – 2.1]%	1.9% 2.0% [2.0 – 2.1]%	1.6% 1.8% [1.7 – 1.8]%
7 (increase)	FSWs migrate : logistic HIV prevalence in POM	Migrating FSW size	15% [7.5 – 30]%	None	1.2% 1.5% [1.4 – 1.8]%	1.9% 1.9% [1.9 – 2.0]%	1.6% 1.7% [1.6 – 1.7]%
7 (increase)		Duration of migration	4 months [2 – 8] months	None	1.2% 1.5% [1.4 – 1.7]%	1.9% 1.9% [1.9 – 2.0]%	1.6% 1.7% [1.6 – 1.7]%
7 (increase)		Number of high-risk contacts in POM	1.0-fold [0.5 – 2.0]-fold	None	1.2% 1.5% [1.4 – 1.7]%	1.9% 1.9% [1.9 – 2.0]%	1.6% 1.7% [1.6 – 1.7]%
9 (increase)		HIV prevalence in POM (2004)	17% [8 – 35]%	None	1.2% 1.5% [1.5 – 1.6]%	1.9% 1.9% [1.9 – 2.0]%	1.6% 1.7% [1.6 – 1.7]%

APPENDIX 3: Additional results from Chapter Seven

Figure A3.1: Overall model HIV prevalence with four different HIV transmission probability sets (low-range, middle-range, high-range and B-C) in Taluka B (one low-risk partner)

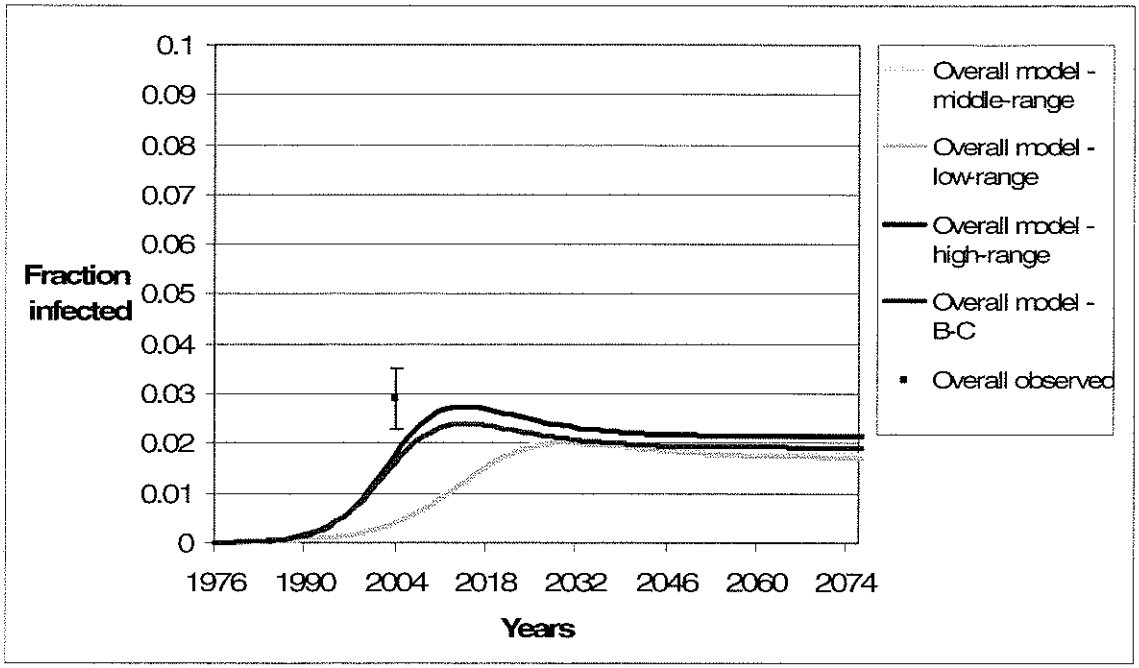


Figure A3.2: Overall model HIV prevalence with four different HIV transmission probability sets (low-range, middle-range, high-range and B-C) in Taluka C (one low-risk partner)

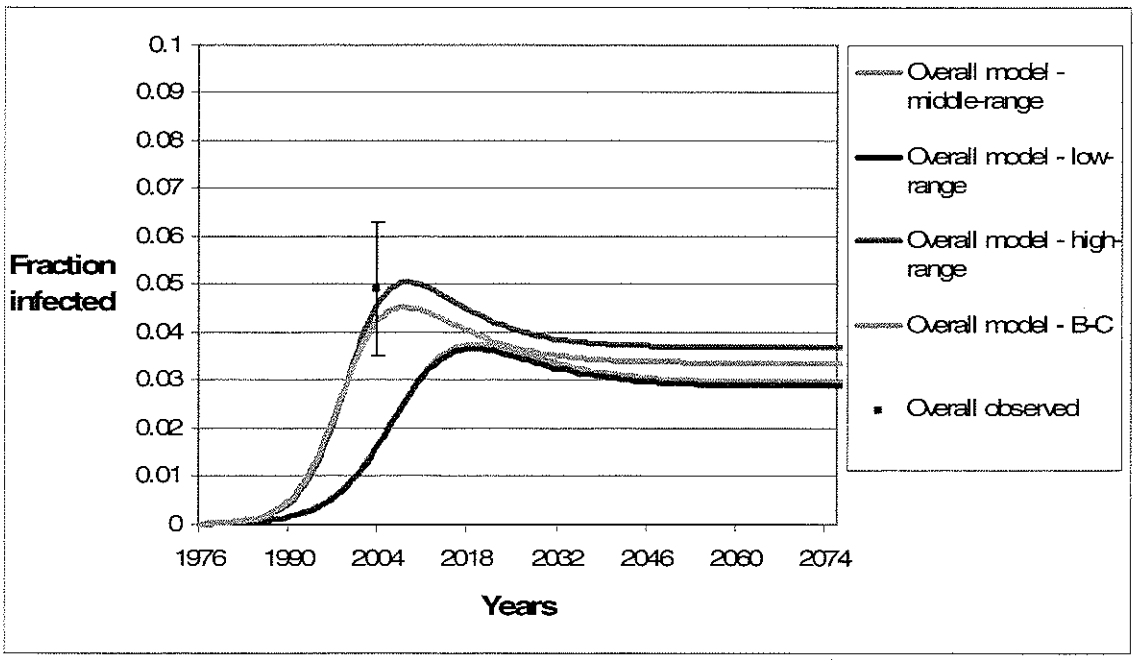


Figure A3.3. Overall model HIV prevalence with four different HIV transmission probability sets (low-range, middle-range, high-range and B-C) in Taluka B (ten low-risk partners)

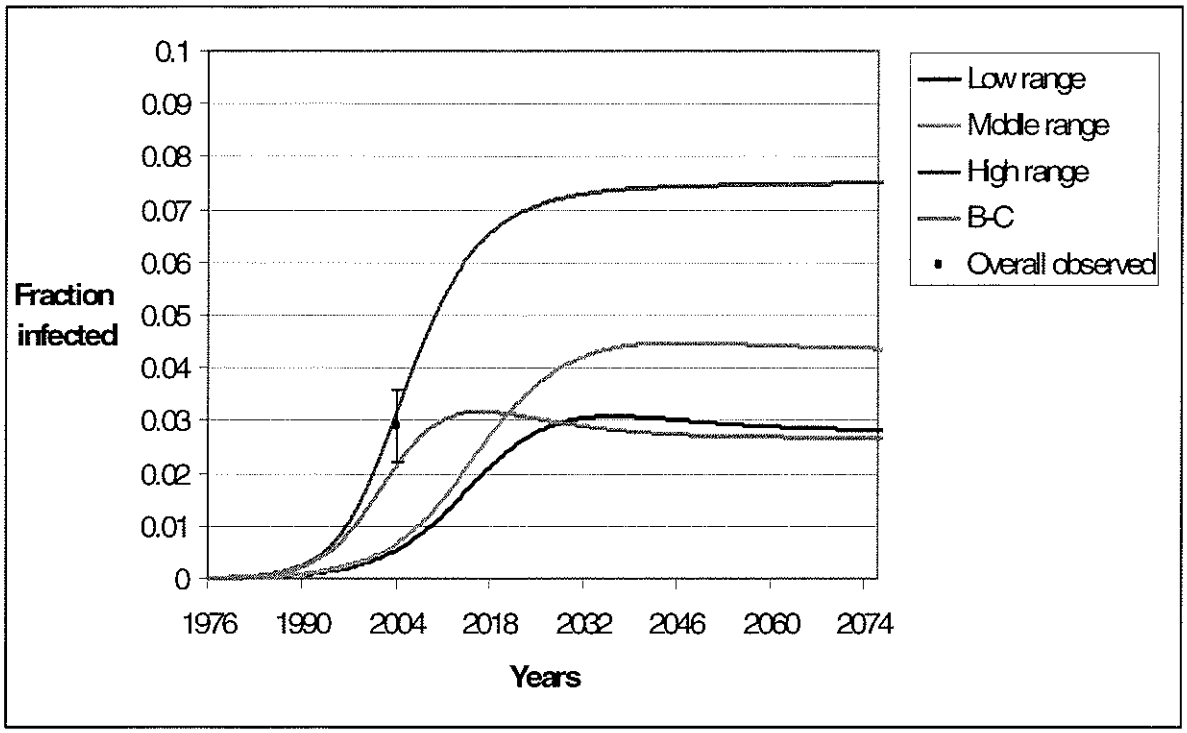


Figure A3.4. Overall model HIV prevalence with four different HIV transmission probability sets (low-range, middle-range, high-range and B-C) in Taluka C (ten low-risk partners)

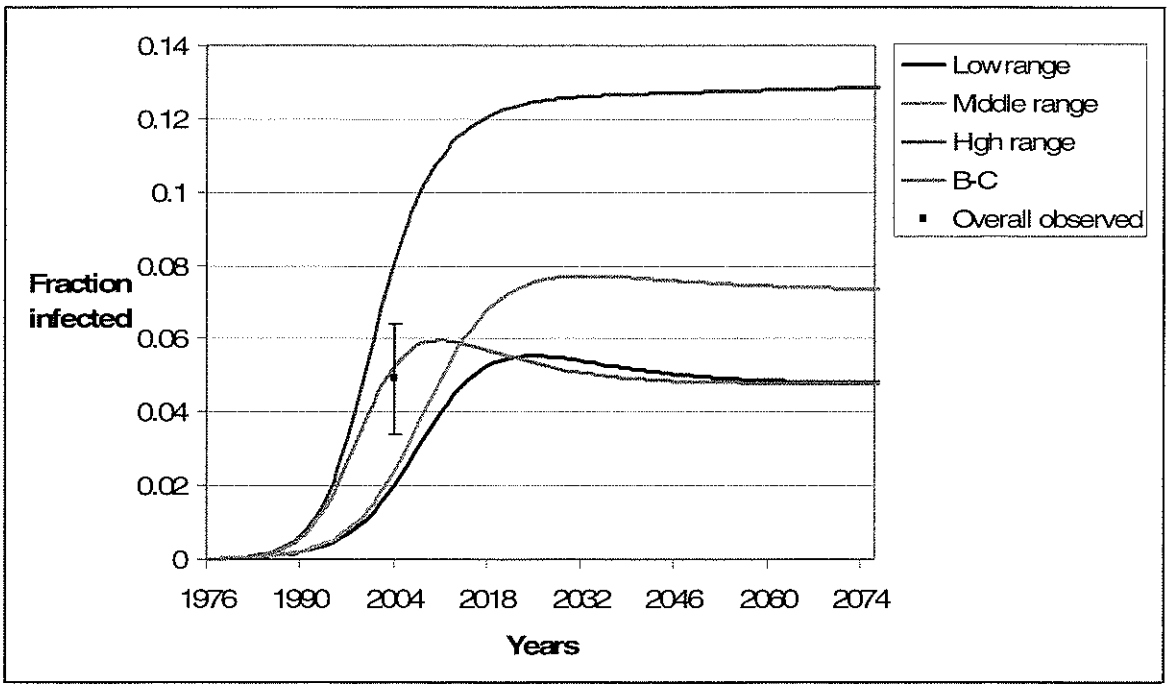


Figure A3.5. Peak and equilibrium HIV prevalence in high-risk populations with varying population growth rate