

**THE DYNAMICS OF HIV/AIDS IN GHANA WITHIN THE SUB-
SAHARAN AFRICA CONTEXT**

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ABSTRACT

The overall aim of this thesis is to understand the factors determining the spatial patterning of the HIV/AIDS epidemic in sub-Saharan Africa in order to inform public health policy debates. The justification for the thesis emerges from the uncertainties around the epidemiology of HIV/AIDS in sub-Saharan Africa and the lack of research to justify the assumptions that underpin policy formulation.

The study is organised at two different scales making up the two parts of the thesis. The first part is at the sub-Saharan African level and applies epidemic modelling to AIDS incidence data from 41 sub-Saharan African countries. The output of the single region model produced disease parameters which were fed into the multiregion model to determine transmission pathways and infection transfers between countries. The second part is at sub-national level and uses a population-based behavioural survey, conducted in the Krobo district in Ghana, designed to explore the socio-economic, demographic and political contexts in which the epidemic continues to thrive despite universal awareness of the consequences of AIDS.

The main findings of the modelling are that the HIV epidemic started both in Ghana and D.R.Congo in 1974. This means that the epidemic occurred years earlier before the date of the first recorded AIDS cases and that the epidemic went unnoticed for a very long period of time. Whereas the epidemic spread from D.R.Congo to countries in East, Central and Southern Africa at the start of the epidemic, Ghana was the source of the infection for countries in the West African sub-region. At the peak of the epidemic however, South Africa and Tanzania become the main nodes of infection transfer to countries in the Central, Eastern and Southern African sub-regions while Cote d'Ivoire becomes the main node for infection transfer to countries in the West African sub-region.

The population-based survey of the Krobo district explored the infection transfer depicted by the modelling exercise from Ghana to Cote d'Ivoire at the start of the epidemic and shows that this was mediated by migrant workers, especially commercial sex workers, from the Krobo district in Ghana. However, at the peak of the epidemic, migrants returning to Ghana, again mainly commercial sex workers, mediated a re-infection from outside back into the Krobo district and subsequently the whole of Ghana. The findings from the population-based survey show that the reason for the continued spread of the virus is not a lack of knowledge or awareness about HIV. The main factors identified as causing the increase in the spread of the epidemic, through high risk unprotected sex, are high unemployment, low income levels, low status of women, low educational status and a false sense of security among those in stable relations. Other cultural factors such as belief in witchcraft, stigma and living arrangements were identified as factors influencing the epidemic spread. These factors, which make individuals vulnerable, need to be addressed if interventions are to have any meaningful impact.

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DEDICATION AND ACKNOWLEDGEMENT

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Elvis Nuro Amoakwa.

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Elvis Nuro Amoakwa.

CHAPTER ONE

1.0 INTRODUCTION

1.1 Background

As the world enters the third decade of the AIDS epidemic, the evidence of its impact is undeniable. Wherever the epidemic has spread unchecked, it is robbing countries of resources and capacities on which human security and development depend. In some regions, HIV/AIDS, in combination with other crises, is driving ever-larger numbers into destitution and is undermining previous health gains.

By the mid-twentieth century, drugs and vaccine development suggested that the world might be entering a period when the battle against infectious diseases could be won. This led to the Alma Ata Declaration of "Health for all by the year 2000" later renamed as Primary Health Care for all. This declaration reflected the extent to which the world thought it had conquered infectious diseases and hoped to eliminate them for humankind in the years ahead.

However such hopes were dashed with the advent of the HIV/AIDS epidemic as well as the re-emergence of some of the old diseases with new and drug resistant strains that are difficult to combat such as Ebola, Lassa fever, Marburg fever and multi-drug resistant TB. Also of concern are the rise of antibiotic resistant bacteria, new strains of salmonella, bovine spongiform encephalopathy and related human form, new variant Creutzfeld-Jacob Disease (nvCJD) and recently the Severe Acute Respiratory Syndrome (SARS) emerging as the first global epidemic in the twenty-first century. AIDS is the only disease to have a dedicated United Nations Organisation (UNAIDS) charged with the aim of confronting it. For the first time in the field of public health a syndrome defined as sexually transmitted produced a qualitative change in the institutions devoted to its control (Austoker, 1992). The fortieth World Health Assembly declared its deep concern that HIV/AIDS has assumed a pandemic proportion affecting all regions of the world and represents a threat to the attainment of health for all (World Health Assembly, 1987).

What was thought to be a disease affecting the homosexual community has now become a global pandemic with grave consequences on global population. One disturbing fact about the HIV/AIDS pandemic is that there is no cure yet and unlike other infectious diseases where some of the susceptible population may become immune, all cases result in death. Also unlike other infectious diseases, HIV/AIDS transmission depends on varied sexual and social contacts which involve exchange of body fluids. This makes it difficult to forecast the epidemic accurately due to the varied social backgrounds of the susceptible populations in different communities and regions around the world. Therefore, the solution to the HIV/AIDS epidemic is not just about medicine or public health. Its origin lies far back in time and deep within the structures of social, economic and cultural life (Barnett and Whiteside, 2002).

Although HIV was first reported in 1979, the actual date for the inception of the disease is not known but it is estimated to have been in existence decades earlier. Also the magnitude and the directionality of the disease are not known. This is particularly the case in sub-Saharan Africa where the disease is having severe demographic and social consequences such as an emerging unbalanced age structure (Caldwell 1997, Ainsworth and Teokul, 2000). The adoption of preventative behaviour is the only protection against the virus, but despite an almost universal awareness of the serious consequences of AIDS and of the sexual transmission of HIV there is no widespread adoption of preventative measures in sub-Saharan Africa. This indicates the importance of additional influences on behaviour which need greater understanding (Wolf and Blanc, 2000; Schroder, Hobfall and Jackson, 2001). As efforts mount to unravel the peculiar nature of the HIV/AIDS pandemic and the apparent failure of current interventions to halt or minimise the spread of the HIV/AIDS epidemic in sub-Saharan Africa, tradeoffs among different potential interventions and other critical policy decisions must be based on the best possible information on the magnitude and directionality of HIV transmission pathways.

Population-based epidemiological data are limited in sub-Saharan Africa. Incidence data in representative study samples are also rare due to the difficulty of direct measurement of population incidence and the high cost and long follow-up periods required for cohort studies. AIDS notification data also represent a fraction of new

cases of AIDS and are subject to reporting delays. Information on HIV/AIDS-attributable mortality is also essential for assessment of the epidemiological situation but vital registration systems have extremely limited coverage in most sub-Saharan Africa. In this context, the use of a modelling approach can make a valuable contribution towards a better understanding of the prevalence and trends in the HIV epidemic (Thomas, 1992).

From the start of the AIDS pandemic, individual behaviour has been put forward implicitly and explicitly as the main explanatory concept for understanding the epidemiology of HIV infection and in particular for the rapid spread and high prevalence in sub-Saharan Africa. In the United States, the gay community was particularly affected at the start of the epidemic. The epidemic therefore became seen as a gay plague with AIDS a consequence of individual behaviour and interventions targeted at individuals comprising the gay community. In the same way when the epidemic was identified among intravenous drug users in the US and Europe, attempts were made to provide syringes to drug users in an effort to stop the spread of the HIV virus through needle exchange. Commercial sex workers and their clients constitute a third group targeted for interventions in high income countries due to their high risk activities. Current interventions in the form of safer sex promotion including abstinence, mutual faithfulness to one sexual partner and condom use are all based on the notion that individual human behaviour is the main explanatory factor for the HIV epidemiology. This has had implications for the international response to AIDS in other countries and has influenced public health policy and strategy as well as the design of prevention and care interventions at national, community and individual level. However, individual behaviour alone cannot possibly account for the exceptionally rapid spread of the HIV epidemic in developing countries, especially in sub-Saharan Africa which accounts for two-thirds of all cases in the world.

1.2 The Aim of the Thesis

Given the uncertainties around the epidemiology of HIV/AIDS in sub-Saharan Africa and the lack of research to justify the assumptions that underpin policy formulation, the overall aim of this thesis is to understand the factors determining the spatial patterning of the HIV/AIDS epidemic in sub-Saharan Africa in order to inform public

health policy debates. This overall aim is addressed through four specific research objectives:

- To identify source locations and dates in sub-Saharan Africa for the inception of the HIV epidemic
- To predict the dates for each country in sub-Saharan Africa when the HIV epidemic will become endemic in a scenario of no intervention
- To identify transmission routes in sub-Saharan Africa at both the start and at the peak of the HIV epidemic
- To explore the processes by which the HIV epidemic is spreading through a case study of Krobo district, Ghana
- To identify the implications of the study for public health policy debates on HIV/AIDS

The study is organised on two different scales making up the two parts of the thesis. The first part is on the sub-Saharan African level. As a result of poor epidemic surveillance systems and inadequate data about the HIV/AIDS epidemic in sub-Saharan Africa, the magnitude and directionality of the epidemic spread is uncertain. The first part of the thesis therefore applies epidemic modelling to AIDS incidence data from 41 sub-Saharan African countries. Modelling the epidemic in this way provides estimates of the date of inception of the epidemic, date of forecast endemic state and the size of the risk population in various countries in sub-Saharan Africa. It also estimates the transmission probability of infection transfer between Susceptibles and infectives in a sub-region where the spread of the epidemic is mainly heterosexual. The model outputs help to estimate the transmission pathways from the start of the epidemic and how these pathways were differentiated at peak prevalence between the countries of sub-Saharan Africa. Lastly the thesis explores the implications of the modelling outputs for future AIDS incidence.

The second part is at sub-national level. This part aims to understand the observed spatial patterning of the epidemic as depicted in the modelling process and to explain the apparent failure of several policies and interventions put in place to mitigate the spread. These aims are addressed through a population based behavioural survey taking the Krobo District in Ghana as a case study. The survey is designed to explore

the socio-economic, demographic and political contexts in which the epidemic continues to thrive despite universal awareness of the consequences of AIDS.

1.3 The structure of the Thesis

Chapter 1 places the emergence of HIV/AIDS into its context of a time when public health had high hopes of eliminating infectious diseases. The need to understand the trend and pattern of the epidemic in a continent where prevalence data were limited and the role of modelling in filling the gap are stressed.

Chapter 2 reviews the emergence of the HIV/AIDS epidemic. The first section examines how the epidemic started, the current global situation and hypothesised origin of the virus. This is followed by evidence of HIV virus in Africa in the pre-AIDS era. The biochemistry of the HIV virus is examined for us to understand how the epidemic spread. This is followed by the global typology of the HIV/AIDS epidemic in order to put the African epidemic into perspective. Lastly the HIV surveillance system in Africa and how this has affected the epidemic spread in Africa is reviewed.

Chapter 3 considers conceptual issues in HIV and AIDS epidemiology. The focus of this chapter is to explain the various frameworks through which the spread of HIV/AIDS is explained. This forms the basis upon which the spread of the epidemic in sub-Saharan Africa and Ghana will be explored. The common research approaches to the spread of HIV/AIDS are discussed: Cultural (behavioural); political economy; vulnerability. The effects of migration and pre- and post-independence African history and the concept of vulnerability are discussed further.

Chapter four provides an overview of the HIV/AIDS epidemic in Ghana within the sub-Saharan African context. The first part examines the epidemiological situation in Ghana and a review of the national response to the HIV/AIDS epidemic in Ghana. This is followed by a background of the epidemic in the Krobo district in Ghana, the chosen case study area. The final section reviews the demographic and socio-economic characteristics of the Krobo district in order to put the fieldwork into perspective.

Chapter five presents the primary research of the thesis at the sub-Saharan African level. Epidemic modelling systems are fitted to AIDS incidence data for 41 sub-Saharan African countries in order to derive estimates for the disease parameters in order to help understand the magnitude and directionality of the epidemic spread in sub-Saharan Africa. The first part of this chapter traces the evolution of epidemic modelling systems and provides the theoretical background that underpins the modelling process used in this chapter. This is followed by description of the single and multiregion models developed by Thomas (1992, 2001), Smith and Thomas (2001) and Smith (2003). This is followed by fitting the single region model in order to estimate disease parameters, which are then fitted into the multiregion specification to determine transmission pathways of the epidemic. The results of the modelling process are then interpreted.

Chapter six presents the design of the primary research of the thesis carried out at the sub-national level. A population based survey is designed to explore the processes taking place in everyday life that underpin the magnitude and directionality of the epidemic spread as depicted in the modelling processes. The survey is carried out in the Krobo district of Ghana using a combination of interviews and questionnaires. The first section of this chapter deals with the methodological issues of the fieldwork. Issues arising out of the fieldwork follow this.

Chapter seven presents the result of the quantitative analysis and discussion and conclusion. The first part of this chapter presents the results of the quantitative element of the survey. First the general demographic and socio-economic characteristics of the respondents are described. This is followed by bivariate and multivariate analysis of factors influencing the spread of the epidemic. The chapter again concludes with a discussion of the policy implications of the results.

Chapter eight presents the results of the qualitative analysis. The views of HIV/AIDS service organisations on the factors causing the rapid spread of the epidemic are presented. Themes arising from the interview are then discussed and conclusion drawn.

Chapter nine draws together the two parts of the study; the modelling output and the population based survey and conclude with an overview discussion of the implications of the study's results for public health policy and strategies for disease prevention and control.

CHAPTER TWO

2.0 EMERGENCE OF HIV/AIDS EPIDEMIC

2.1 Introduction

In order to understand the spatial epidemiology of HIV and AIDS and how it is spreading, especially in sub-Saharan Africa, the focus of the thesis, this chapter first examines how the epidemic started, the current global spread and the hypothesised sources of infection. This is followed by the aetiology of the epidemic, how the epidemic progresses from HIV to AIDS as well as cofactors of disease progression. The modes of transmission of the epidemic are then considered in order to understand the reasons for and the modes of spread of the epidemic in sub-Saharan Africa. Lastly, the surveillance system for monitoring the HIV/AIDS epidemic, again with particular focus on sub-Saharan Africa, is reviewed.

The epidemic of human immunodeficiency virus (HIV) infection and acquired immunodeficiency syndrome (AIDS) emerged in the last quarter of the 20th century and within less than two decades has affected over 190 countries (Mertens et al., 1995). The first cases of AIDS were recognised because of an unusual clustering of diseases such as Kaposi's sarcoma and Pneumocystis Carinii Pneumonia (PCP) in young homosexuals in the USA. Up to this time, Kaposi's Sarcoma had been a rare form of relatively benign cancer that tended to occur in distinct subgroups of the population such as older men of Mediterranean origin. However by March 1981, at least eight cases of a more aggressive form of Kaposi's sarcoma had occurred among young gay men in New York (Hymes, Green and Marcus, 1981). Concurrently, there was an increase in both California and New York in the number of cases of a rare lung infection, PCP (Morbidity and Mortality Weekly Report (MMWR) 5 June 1981). In June 1981, the Centre of Disease Control in Atlanta published a report about the occurrence without identifiable cause of PCP in five men in Los Angeles (MMWR, 1981). This report is sometimes referred to as the 'beginning' of AIDS but it might be more accurate to describe it as the beginning of an awareness of AIDS in the USA.

In December 1981, it was clear that the disease affected other population groups when the first cases of PCP were reported in intra-venous (IV) drug users (Masur et al

1982). HIV was later also reported to be occurring in haemophiliacs and recipients of blood transfusion (MMWR, 1992). Subsequently the syndrome was identified among infants born to mothers who used drugs. It was apparent that this was not only a 'gay' disease. The illness was seen simultaneously in a number of locations outside the United States. In Zambia, a significant number of cases of Kaposi's sarcoma were reported at the University Teaching Hospital in Lusaka and results from research into a wave of deaths in south Uganda confirmed the existence of AIDS there too (Kaleebu et al, 2002). Hooper (1999) documented similar recognition of the disease in Tanzania, Congo and Rwanda. Outside Africa, AIDS cases were identified in all Western countries, Australia, New Zealand and some Latin American countries. By 1981 there was global recognition of the syndrome and clinicians knew what to look for. The search for the origin and the cause of the HIV/AIDS epidemic therefore became a matter of concern for scientists.

Since the beginning of the epidemic, more than 25 million people have died of AIDS and the total number of AIDS death in 2005 was 3.1 million of which 2.6 million were adult and about 570000 were children below the age of fifteen (UNAIDS, 2005). As of December 2005, an estimated 40.3 million people were living with HIV/AIDS (see table 2.1).

Table 2.1 Global summary of the AIDS epidemic

Number of people living with HIV in 2005	Total	40.3 million (36.7–45.3 million)
	Adults	38.0 million (34.5–42.6 million)
	Women	17.5 million (16.2–19.3 million)
	Children under 15 years	2.3 million (2.1–2.8 million)
People newly infected with HIV in 2005	Total	4.9 million (4.3–6.6 million)
	Adults	4.2 million (3.6–5.8 million)
	Children under 15 years	700 000 (630 000–820 000)
AIDS deaths in 2005	Total	3.1 million (2.8–3.6 million)
	Adults	2.6 million (2.3–2.9 million)
	Children under 15 years	570 000 (510 000–670 000)

Source: UNAIDS, 2005

These include 2.3 million children less than fifteen years of age and 17.5 million women, 38 million adults. About 5 million people were thought to have been infected with HIV during 2005 alone (UNAIDS, 2005). It is estimated that about 14,000 new HIV infections occurred every day in 2005. Ninety five percent of them occurring in developing countries (UNAIDS 2005). Although there are reports of HIV/AIDS levelling off in some countries, the epidemic is still in its early phase and there could be 68 million HIV related death between 2000 and 2020 unless prevention and treatment programmes to combat the disease are expanded drastically (UNAIDS, 2005).

Eastern Europe and Central Asia is experiencing the fastest growing epidemic. The number of people living with HIV in 2002 stood at 1.6 million. HIV/AIDS is expanding rapidly in the Baltic States, the Russian Federation and several Central Asian republics. This is as a result of economic and social changes which has giving rise to conditions and trends that favour the rapid spread of HIV such as wide social disparities, limited access to basic services and increased migration.

In Asia and the Pacific, 7.4 million people are now living with HIV (UNAIDS, 2005). The growth of the epidemic in this region is largely due to the growing epidemic in China where a million people are now living with HIV and where official estimates foresee a manifold increase in that number over the coming decade. There remains a potential for growth in India where about 4 million people are living with HIV. However, with the exception of Cambodia, Myanmar and Thailand, national HIV prevalence levels remain comparatively low in most countries of Asia and the Pacific. However, in large countries, notably India and China, aggregate prevalence rates mask the experience of serious localised epidemics affecting many millions of people. India's national adult HIV prevalence rate of less than 1 percent offers little indication that an estimated 3.97 million people were living with HIV at the end of 2001, the second largest national number in the world after South Africa. In the case of China, official estimates put the number of people living with HIV/AIDS at 1 million in mid-2002 with the number of reported new HIV infections having risen about 17 percent in the first sixth months of 2002 (UNAIDS, 2005).

In Sub-Saharan Africa the AIDS crisis continues with an estimated 3.2 million new infections in 2005 and about 25.8 million people now living with HIV/AIDS (UNAIDS, 2005). Recent antenatal clinic data show that several countries in southern Africa now have prevalence rates among pregnant women exceeding 30 percent, a level previously only recorded in Botswana (UNAIDS, 2005). In West Africa at least five countries can be said to be experiencing serious epidemics with adult HIV prevalence exceeding five percent. On the more optimistic side, HIV prevalence in Uganda continues to fall while there is evidence that prevalence among young people especially women is dropping in some other parts of the continent.

In the Middle East and North Africa, HIV prevalence continues to be low in most countries in the region although increasing numbers of infection are being detected in several countries including the Islamic Republic of Libyan Arab Jamahiriya and Pakistan. The epidemic's advance is most marked in countries such as Djibouti, Somalia and the Sudan which hitherto were among the low incidence countries.

The epidemic is well established in Latin America and the Caribbean. An estimated 1.8 million adults and children are living with HIV in this region. Twelve countries in this region have an estimated HIV prevalence of 1 percent or more among pregnant women. In several Caribbean countries, adult HIV prevalence rates are surpassed only by the rates in sub-Saharan Africa making this the second most affected region of the world. HIV is now the leading cause of death in some of these countries. Haiti remains the worst affected followed by the Bahamas with adult prevalence rates of over 6 and 3.5 percent respectively.

In high-income countries a total of 1.6 million people are now living with HIV and an estimated 30,000 people died of AIDS in 2005. The introduction of antiretroviral therapy since 1995/96 has drastically reduced HIV/AIDS mortality although this trend has begun to level off in the past two years. Longer survival of people living with HIV/AIDS has led to a steady increase in the number of people living with the virus in high-income countries. A larger proportion of new HIV infections are occurring through heterosexual intercourse in several countries, estimated at 59% of all new infections in 2001. In the United Kingdom, as in some other European countries, a large proportion of this heterosexual transmission is found in persons who originated

from or have lived or visited areas where HIV prevalence is high (WHO/UNAIDS, 2005). Although the HIV epidemic has spread to all the continents of the world, the origin and source of the HIV/AIDS epidemic is not certain and there is the need to review literature on the possible origin of the virus in order to understand the epidemic spread.

2.2 The Origin of the HIV Virus

Debates about the origin of AIDS have sparked considerable interest and controversy since the beginning of the epidemic. The first known cases of AIDS occurred in the USA in 1979 but little is still known about the source of the disease. Several theories have been proposed. First, as argued by the majority of the explanations, the etiologic agent may represent a more virulent mutant variant or recombinant of an organism that was either previously infecting the same population without causing disease or had a distinct profile of disease pathology. Secondly, the organism may have been introduced from a relatively isolated population of people who had developed a resistance to the lethality of the agent. Thirdly, the organism may have been introduced to humans from another species. This is becoming widely accepted as explaining the human immunodeficiency virus type 2 (HIV 2) and is increasingly likely for HIV1. There are others who believe that the origin of the HIV virus is iatrogenic or man-made while some religious groups suggest that AIDS and HIV are acts of punishment from God for immoral practices such as homosexuality, drug use and sexual promiscuity.

Scientists have long recognised the ability of certain viruses and other diseases to pass from animals to humans, a process referred to as zoonosis. According to this theory, once an animal disease has infected people, it then mutates and may be passed on from human to human. For example the Ebola virus and Marburg filovirus are two diseases among many which were introduced to humans from animal through zoonotic transfer (Martini, 1969; WHO, 1979). Although it has not been proven that HIV came from primates, an SIV has been known to infect humans (MMWR, 1992) lending credence to the idea that HIV came into human population through zoonotic transfer. The exact route of the transfer of HIV from primates to humans is not known. There are some who believe that HIV jumped to humans as a result of a bite

or after a hunter became infected while capturing a monkey or ape or preparing its flesh for eating.

Advocates of the genocidal hypothesis are of the view that HIV was introduced into Africa from the west by an accident such as oral polio vaccine or intentionally by the United States of America's Central Intelligence Agency (CIA). The emergence of HIV/AIDS in sub-Saharan Africa coincided with the depopulation policy institutionalised by the then United States administration with primary funding from Rockefeller Foundation and the Merk Fund (Horowitz, 2001). Such coincidence coupled with the staunch advocacy of African depopulation by Henry Kissinger, the then National Security Advisor of the United States administration, made developing countries, especially those in Africa suspicious giving rise to the theory that the origin of HIV could be linked to the CIA's biological weapons programme and vaccine manufacture and that the possibility that HIV virus was released intentionally could not be ruled out (Hooper, 1999).

However, some scientists argue that such conspiracy theories are unfounded because natural transmission of HIV repeatedly occurs (Weiss, 2001). Others propose that increased unsterilised injection in Africa during the period 1950-1970 accidentally provided the agent for SIV human infection to emerge as epidemic HIV in the modern era (Drucker et al, 2002). Moreover, Burr et al (2001) argue that the large number of HIV-1 subtypes cannot be attributed to a transfer of the HIV virus between species but that the likely source of the multiple subtypes of HIV can be attributed to the vaccine hypothesis. Those in favour of the natural evolution theory of HIV claim that the discovery of HIV in a 1959 blood sample from Leopoldville (Zaire) indicates 1931 as an approximate date of HIV origination (Zhu et al, 1998; Yusim et al, 2001). This means that HIV originated well before the polio vaccination or the special virus cancer programme implicated in the iatrogenic origin theory.

A more convincing vaccination hypothesis involves Hepatitis B. Horowitz, (2001) linked Hepatitis B vaccination to the cause of HIV/AIDS. He supported the view that African chimpanzees used in the manufacture of the Hepatitis B vaccines during the early 1970s were the cause of the zoonosis transfer of HIV virus. He argued that

human Hepatitis B viruses cultured *in vivo* in chimpanzees were returned to humans whose infected blood serum was then pooled to develop four different strains of experimental Hepatitis B vaccine. These pooled serum-derived Hepatitis B vaccines were tried between 1970 and 1975 on gay males in New York City and males in Central Africa and are argued to contribute to causing the HIV virus origin (Hamilton, 2001). The geographical area where the oral polio vaccine trials and Hepatitis B vaccine trials were tested in Africa is consistent with countries in Africa where HIV/AIDS prevalence is very high, Such as Senegal, Cote d'Ivoire, Uganda, Kenya, Swaziland, North-eastern South Africa and eastern Zaire (Horowitz, 2001). The argument for a vaccine related origin may suggest that the Hepatitis B vaccine pilot studies may have activated an endogenous or exogenous HIV-related retroviral gene in one or more of the primates fulfilling the 'starburst phylogeny' antecedents advanced by Burr, Hyman and Myer (2001).

2.3 Evidence of HIV/AIDS in Africa: Pre AIDS era

Once the causative virus of AIDS had been isolated in the early 1980s, and the characteristic symptoms defined, researchers began to look for evidence of both AIDS and HIV infection prior to the 1980s. Medical and historical records were searched for evidence of illness in the pre-AIDS era based on the CDC clinical definition of AIDS. When the HIV virus was isolated, similarities between HIV and certain viruses found in African green monkeys were soon recognised. As a result Africa emerged as the main possible geographic origin of the HIV virus. Much of the serological analysis therefore was focused on testing stored African serum samples for antibodies to HIV.

The earliest suggested timing for the occurrence of AIDS in Africa is that of Ablin et al (1985) who comments on references to $\bar{A} \bar{A} \bar{A}$ disease in the literature of ancient Egypt which is translated as poison or semen. This led to the speculation that this is a possible prototype AIDS that may have forced or reinforced the homosexual taboo of ancient Egypt. Other records of AIDS in Africa include that of Goldwater (1986), who records the case of a male from Accra, Ghana, who presented in 1920 with bronchial candidiasis and pulmonary tuberculosis. Similarly, Clumeck et al (1983) observed AIDS-like disorders in African patients before HIV was identified. About forty-two of the earliest 200 AIDS cases recognised in Europe were in Africans

(AIDS-Forschung, 1987). Also studies in Zaire and Rwanda confirmed the endemic nature of HIV infection and AIDS in Central Africa (Piot et al, 1984).

Central Africa has been suspected as the origin of HIV-1 but disputed evidence for early HIV-1 infection in Central Africa dates from the 1950s. Antibodies to HIV-1 were found at the west Nile district of northern Uganda in samples collected between 1972 and 1973 (Saxinger et al, 1985). Nzilambi et al (1988) detected an antibody positive prevalence rate of 0.8 percent from a study of 659 serum samples collected in 1976 from the rural equator province of Zaire. Between 1962 and 1976, Evidence of seven HIV infections including three likely AIDS cases originating in Zaire and Burundi were found in samples collected between 1962 and 1976 (Prentice, 1986; Sonnet et al, 1987; Smith et al, 1988). The most generally accepted work which points to HIV-1 circulation in Central Africa in the mid-1970s is that of Getchell et al (1987) who isolated HIV-1 in a stored serum sample collected in Zaire in 1976.

Unlike HIV-1, HIV-2 has been hypothesised to originate from West Africa. Ancelle et al (1987) observed a heterosexual Portuguese couple that presented with HIV-2 related AIDS in 1985 and 1986. This infection is believed to have occurred in Guinea-Bissau between 1966 and 1969. There was another record of HIV-2 infection in a Portuguese male in Guinea in 1966 (Courouce, 1987; Saimot et al, 1987; Bryceson et al, 1988). Retrospective serological analysis on 3177 stored West African sera samples from 1966 to 1977 from eleven West African countries also revealed that 10 samples were HIV-2 positive the earliest coming from Ivory Coast (Cote d'Ivoire) (Kawamura et al., 1989).

Other evidence of HIV infection, which may be traced back to an African source, comes from other parts of the world. For example Froland et al. (1988) claim a Norwegian family of three died in the mid 1970s following an infection of the husband as early as 1966 in Africa. Also Bygberg (1983) recorded a plausible case of AIDS in a Danish surgeon who had worked in Zaire in 1976. Some of the serological analyses conducted in Africa are summarised in Table 2.2.

Table 2.2 Some Results of Retrospective Serological Analysis for HIV-1 Infection

Date	Country	Subjects	No. tested No.positive (%)	Confirmatory test
1959	Central Africa	Urban/rural	672/1(0.15)	WB,IFA,RIPA
1959	South Africa	Xhosa Bushmen	146/0	WB,IFA,RIPA
1969	Mozambique	Rural Bantu	118/0	WB,IFA,RIPA
1970	Zaire	Urban, pregnant	805/2(0.25)	WZ,IFA,RIPA
1976	Zaire	Rural	659/5(0.76)	WB
1976	Zaire	Urban	454/6(1.3)	WB
1976-77	Gambia	Children	92/0	IFA
1976-85	Liberia	Villagers	935/0	IFA
1978	Ivory Coast	Villagers	1195/0	IFA
1980	Zaire	Urban, pregnant	498/15(3.0)	WZ,IFA,RIPA
1980	Kenya	Male, Chancroid	118/0	IFA,RIPA
1981	Gabon	Clinic attendees	855/2(0.23)	IFA
1981	Senegal	General population	789/1(0.13)	RIPA
1981	Kenya	Students	300/0	RIPA
1981	Kenya	Urban, pregnant	111/0	IFA,RIPA
1981	Kenya	Female prostitutes	(4.0)	IFA,RIPA
1981	Kenya	FemaleSTDpatients	(4.0)	IFA,RIPA

Source: AIDS-Forschung, 1987

NB: WZ-Wellozyme, WB-Western blot, RIPA-Radioimmunoprecipitation essay, IFA- Immunofluorescenenassay

The above evidence suggests that HIV might have existed as endemic in Africa for decades or centuries and that the change of traditional lifestyle in the post war period might have hastened the spread. Prentice (1986) observed that HIV existed in isolated tribal communities in Burundi. It is worth noting that despite the considerable knowledge researchers have gained since the inception of HIV/AIDS epidemic, a definite source for the HIV virus continues to elude them.

Given the uncertainties and controversy surrounding possible AIDS cases before 1980 and the controversy surrounding the African origin hypotheses, it is impossible to be sure of the spread of HIV between Central and West Africa. Modelling which forms the main crust of the thesis offers an alternative approach to understanding HIV epidemiology in the pre AIDS era.

2.4 Aetiology of HIV/AIDS

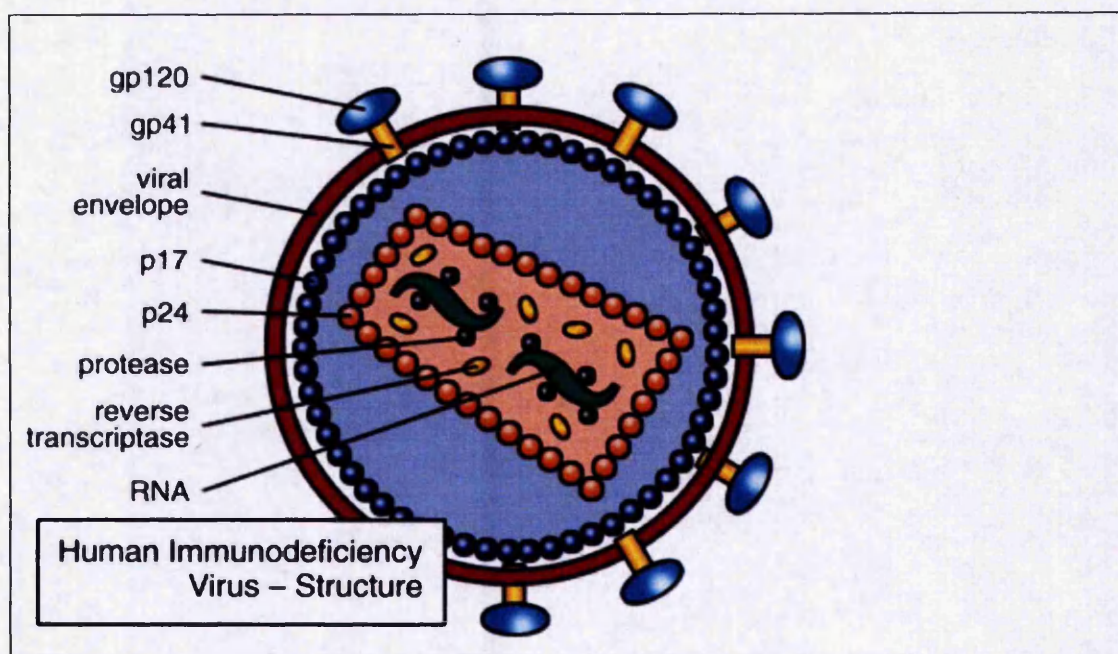
This section reviews the isolation of the HIV virus, its virology and immunology, the viral life cycle and stages of disease progression and the dynamic interplay of HIV

with its cellular host. This provides the biological basis for understanding the spread of the virus and how to control the epidemic. Once the new syndrome had been identified, the pace of scientific and epidemiological activity to identify the cause of the disease increased. By June 1983, a team of French scientists lead by Luc Montagnier identified the virus we now know as HIV-1 (Barre-Sinoussi, Chermann, and Rey et al (1983) and named it lymphadenopathy-associated virus (LAV). In April 1984, the United States Health and Human Services Secretary announced that Dr Robert Gallo of the National Cancer Institute had isolated the virus which caused AIDS and named the virus HTLV-111 (Culliton, 1984). In January 1985, a number of detailed reports were published concerning LAV and HTLV-111 and by March, it was clear that the viruses were the same (Marx, 1988).

There was however significant disagreement about the name of the virus at this time. As the French insisted on LAV (lymphadenopathy-associated virus, Gallo's group used HTLV-3 (human T-cell lymphotropic virus, type 3). In May 1986, the International Committee on the Taxonomy of Viruses ruled that both names should be dropped and the dispute solved by a new name HIV (Human Immunodeficiency Virus) (Coffin, Haase and Levy et al, 1986). In 1985, a second Human Immunodeficiency Virus, HIV-2 was identified.

HIV-1 and the less common HIV-2 belong to the family of retroviruses. HIV-1 contains a single-stranded RNA genome that is 9 kilo bases in length and contains 9 genes that encode 15 different proteins (illustrated in Figure 2.1) (Muesing, Smith and Cabradilla et al, 1985; Gallo, Wong-Staal, and Montagnier et al, 1988). The major viral proteins are classified as structural proteins (Gag, Pol, and Env), regulatory proteins (Tat and Rev), and accessory proteins (Vpu, Vpr, Vif and Nef). Like all retroviruses, HIV replicates within a living host cell and uses the host cell's RNA as a template to make DNA.

Figure 2.1 Two-dimensional diagram of HIV-1



Source: Gelderblom, HR, AIDS, 5, 1991

HIV infection begins when an HIV particle encounters a T-Helper cell with a surface molecule called CD4. The virus particle uses gp120 to attach itself to the cell membrane and then enters the cell (Kwong, Wyatt and Robinson et al, 1998). Within the cell, the virus particle releases its RNA and the enzyme reverse transcriptase then converts the viral RNA into DNA. This new HIV DNA then moves into the cell's nucleus where, with the help of the enzyme integrase, it inserts into the host cell's DNA (Chan and Kim, 1998). Once it is in the cell's genes, HIV DNA is called a provirus. The HIV provirus is replicated by the host cell, which can then release infectious virus particles. Once HIV enters a T helper cell, HIV takes over the cell and the virus replicates. In this process, the infected cell dies. The new virus then seeks out new T-helper cells to infect.

Individuals infected with HIV show both cellular and hormonal immune response to the virus but these responses are unable to prevent the ultimate progression of disease in the great majority of infected individuals. Cellular responses are mediated by cytotoxic T-lymphocytes (CD8 cells) and helper T lymphocytes (CD4 cells). The cytotoxic T-lymphocytes inhibit HIV replication both directly by recognising and

killing infected cells and indirectly by producing soluble chemokine antiviral factor (Haynes, Pantaleo, and Fauci., 1996).

CD4 responses to HIV are important in viral control and strong HIV-specific CD4 responses are associated with lower HIV viral loads (Rosenberg and Walker, 1998). CD4 cells respond to HIV antigens presented in conjunction with major histocompatibility complex (MHC) class II molecules on the surface of infected cells.

Because productive HIV infection occurs in activated CD4 cells, infection and killing of CD4 cells that are responding to HIV infection itself may cause a selective decrease in the number of HIV-specific CD4 cells. HIV can also exist in nonactivated CD4 cells in a preintegrated form, which can become integrated if activation occurs within a few days (Perelson, Essunger and Cao et al, 1997). Also as some of the activated, infected CD4 cells differentiate into resting memory CD4 cells, they may carry copies of the HIV genome in a post integrated form that can persist for decades (Wong, Hezareh and Gunthard et al, 1997).

Current antiretroviral medications cannot efficiently eliminate the virus from a cell in the resting state leading to persistence of infection even in the presence of suppressive therapy (Wong et al 1997). Moreover HIV continues to evolve under the selection pressure of the immune response that occurs in each infected individual and the mutations in the viral epitopes recognised by the immune system may enable the virus to escape the control of the even broad and robust CD4 and CD8 HIV-specific responses (McMichael and Rowland-Jones, 2001).

Depletion of CD4 lymphocytes is the hallmark of HIV infection and predicts an individual's risk for infection with opportunistic pathogens as well as other complications of HIV disease. Evidence has shown that both increased peripheral destruction and decreased production of CD4 cells likely play a role in this decline (Rowland-Jones, 1999; Mohri, Perelson and Tung et al 2001).

2.5 Difficulties in viral control

The difficulty in eliciting broadly neutralising antibody responses against HIV has posed a particularly difficult challenge to the development of a protective HIV vaccine. Humoral immunity appears to be less effective in controlling viremia than cellular responses as HIV is remarkably effective at evading host antibody responses and broadly neutralising antibodies are rare (Peterlin and Trono, 2003).

Due to a high rate of mutation, the HIV virus is able to refine and optimise its interactions with various host proteins and pathways thereby promoting its growth and spread. The virus ensures that the host cell survives until the viral replicative cycle is complete. HIV also establishes stable latent forms that support the chronic nature of infection. Eradication of the virus appears unlikely until effective methods are developed to purge these latent viral reservoirs. Virally induced cell death is not limited to the infected target cell but also involves uninfected bystander cells (Finkel, Tudor-Williams, and Banda, et al, 1995).

Future therapies will likely target viral proteins other than the reverse transcriptase, protease and integrase enzymes. Clinical trials are already underway to study small molecules or short peptides that block the binding of HIV to cell surface chemokine receptors or interfere with the machinery of viral-host cell fusion. Although not as advanced in development, small molecules have been found that block Tat transactivation and Rev-dependent export of viral transcripts from the nucleus to the cytoplasm (Chao, Fujinaga, and Marion et al, 2000; Wolff, Sanglier, and Wang, 1997). As a proof of principle, dominant-negative mutants of Tat, Rev, and Gag proteins have been shown to block viral replication. By increasing the number of antiviral compounds available to target different steps in the viral replicative cycle, in particular drugs that can be deployed in developing countries, research at the cellular level can serve to extend survival and to improve the quality of life for infected individuals and to inhibit the spread of AIDS.

2.6 Epidemiology of Disease Progression in HIV

HIV disease is a continuum of progressive damage to the immune system from the time of infection to the manifestation of severe immunologic damage by opportunistic infections, neoplasms, wasting, or low CD4 lymphocyte count that define AIDS (CDC, 1993). The official CDC case definition for HIV/AIDS is presented in Table (2.2). The time it takes to progress along this continuum ranges from one year or less to an unknown upper limit that has reached about twenty years in a few individuals (Osmond, 1998)

The effect of HIV on the immune system is monitored by measuring the CD4 (helper) lymphocyte count in the blood. A normal CD4 count between approximately 600 and 1200 cells/ μ L indicates that the immune system has not undergone sufficient damage to put the individual at risk for opportunistic illness. Such individuals are unlikely to require treatment. CD4 counts less than 350 cells/ μ L indicate that some impairment of immune function is present and ART should be considered. CD4 counts of less than 200 cells/ μ L indicate imminent risk of serious opportunistic infections or other complications of HIV disease and prompt treatment is recommended (Dybul, Fauci, and Bartlett, et al 2002).

Untreated HIV disease is chronic and progressive and the continuum can be divided into three stages: Primary HIV Infection; Clinical Latency Stage; Clinical Disease Progression Stage. Primary HIV infection often marked by an influenza-like acute viral syndrome is followed by a period of clinical latency typically lasting several years during which high levels of viral replication and CD4 cell turnover lead to progressive immune dysfunction, eventually resulting in clinical disease progression. The distinction between HIV infection and AIDS is important as it has clinical and prognostic implications as well as utility in research.

Table 2.3: The 1993 AIDS Surveillance Case Definition of the U.S Centres for Disease Control and Prevention.

A diagnosis of AIDS is made whenever a person is HIV positive and

1. He or she has a CD4+ cell count less than 200 cells/ μ L, or
2. His or her CD4+ cells account for less than 14 percent of all lymphocytes or
3. That person has been diagnosed with one or more of the AIDS-defining illness listed below

AIDS-defining illness:

4. Candidiasis of bronchi, trachea, or lungs
5. Candidiasis, oesophageal
6. Cervical cancer, invasive
7. Coccidioidomycosis, disseminated
8. Cryptococcosis, extrapulmonary
9. Cryptosporidiosis, chronic intestinal
10. Cytomegalovirus disease (other than liver, spleen, or lymph nodes)
11. Cytomegalovirus retinitis (with loss of vision)
12. Encephalopathy, HIV related
13. Herpes simplex: chronic ulcers or bronchitis, pneumonitis, or esophagitis
14. Histoplasmosis, disseminated
15. Isosporiasis, chronic intestinal
16. Kaposi sarcoma
17. Lymphoma, Burkitt
18. Lymphoma, immunoblastic
19. Lymphoma, primary, of brain (primary central nervous system lymphoma)
20. Mycobacterium avium complex or disease caused by M. Kansasii, disseminated
21. Disease caused by Mycobacterium tuberculosis, any site (pulmonary or extrapulmonary)
22. Disease caused by Mycobacterium, other species, or unidentified species, disseminated
23. Pneumocystis jirovecii (formerly carinii) pneumonia
24. Pneumonia, recurrent
25. Progressive multifocal leukoencephalopathy
26. Salmonella septicaemia, recurrent
27. Toxoplasmosis of brain (encephalitis)
28. Wasting syndrome caused by HIV infection

Additional illness that are AIDS defining in children, but not adults

29. Multiple, recurrent bacterial infections
30. Lymphoid interstitial pneumonia/pulmonary lymphoid hyperplasia

Source: Centres for Disease Control. 1993 revised classification system for HIV infection and expanded surveillance case definition for AIDS among adolescents and adults. MMWR Morb Mort WKLY Rep: 41(RR-17): 1-19

Primary HIV infection is defined as the time period from initial infection with HIV to the development of an antibody response detectable by a standard test. Data from prospective evaluations of populations at risk for HIV infection demonstrate that about 87 percent of individuals who acquire HIV may experience some symptoms of primary HIV infection (Schacker, Collier, and Hughes et al, 1996). The acute viral syndrome of primary HIV infection which is referred to as seroconversion illness was

first defined in 1985 with symptoms resembling those of mononucleosis appearing within days to weeks following exposure to HIV (Cooper, Gold, and Maclean, et al, 1985; Khan, and Walker, 1998).

Symptoms may be mild or severe and may last from a few days to several weeks with an average duration of 14 days. The most common presenting symptom is fever, seen in over 75 percent of patients (Vanhems, Dassa and Lambert, et al, 1999). Other commonly reported symptoms include fatigue, lymphadenopathy, headache and rash. The rash, which is present in 40-80 percent of cases, may be evanescent, is typically maculopapular in character and typically involves the trunk (Vanhems, Dassa and Lambert, et al, 1999). Evaluation of cohorts from Kenya and India found more frequent reports of joint pains, night sweats and mucosal candidiasis and less frequent rash and pharyngitis (Bolinger, Brookmeyer and Mehendale, et al, 1997; Lavreys, Thompson, and Martin, et al, 2000).

During primary HIV infection, plasma viral load often reaches very high levels in the order of millions of RNA copies/mL (Daar, Moudgil, and Meyer, et al 1991; Piatak, Saag, and Yang et al., 1993). Thus, for individuals in whom primary HIV infection is clinically suspected, HIV RNA assays which have a sensitivity approaching 100 percent and specificity of 97.4 percent should be included in the diagnostic evaluation (Hecht, Busch, and Rawal, et al, 2002). The high levels of viremia seen in primary HIV infection do not persist, however, providing evidence of a host immune response capable of bringing the infection under some degree of control at least in the short term (Daar, Moudgil and Meyer, et al 1991).

After the period of acute HIV infection during which the CD4 count and the viral load change dramatically, a relative equilibrium between viral replication and the host immune response is reached and individuals may have no clinical manifestations of HIV infection.

The time between initial infection and the development of AIDS may be long averaging 10 years even in the absence of treatment (Bachetti and Moss, 1989). Despite the relative clinical latency of this stage of HIV infection, viral replication and CD4 cell turnover remains active with millions of virions produced and destroyed

each day (Ho, Neumann, and Perelson et al, 1995). During this period, most infected individuals will have progressive loss of CD4 lymphocytes and perturbation of immune function and on the average CD4 counts will drop by 50-90 cell/ μ L per year in asymptomatic individuals usually with an acceleration of this rate over time (Philip, 1992; Lefrere, Morand-Joubert and Mariotti et al 1997; Koblin, Van Benthem and Buchbinder, et al 1999). In adults, progression from infection to clinical AIDS is rare in the first two years of infection. Rapid disease progression is reported in infants infected by blood transfusion (van den Berg, Gerritsen and van Tol, et al, 1994).

In a cohort of HIV seroconverters who were identified in a retrospective analysis of stored serum samples from hepatitis B vaccine trials in the 1970s, about 87 percent of infected individuals had developed AIDS by 17 years postseroconversion. Twelve percent maintained a CD4 count above 500 cells/ μ L at 10 years but only 3 percent maintained a CD4 count above 500 cells/ μ L at 16 years after seroconversion (Buchbinder and Kaslow, 1998).

During chronic HIV infection, HIV RNA levels in plasma correlate with the rate of CD4 decline with higher plasma viral loads predicting more rapid progression to AIDS and death (Mellors, Kingsley and Rinaldo, et al, 1995; O'Brien, Blattner and Waters et al, 1996).

Recent research has shown that HIV is not dormant during this stage, but is very active in the lymph nodes. Large amounts of T helper cells are infected and die and large amount of virus is produced.

According to CDC criteria, AIDS is defined by either diagnosis of one of the AIDS-defining conditions, or by measurement of CD4 levels of less than 200 cells/ μ L (see. Tables 2.2). Over time the immune system loses the ability to contain HIV due to the cumulative damage to the lymph nodes and tissues. Also at this stage HIV mutates becoming more pathogenic leading to more T helper cell destruction while the body fails to keep up with replacing the T helper cells that are lost. As the immune system becomes more and more damaged, the illnesses that occur become more and more severe leading eventually to an AIDS diagnosis.

Progression to AIDS from the time of infection occurs on the average two years earlier when defined by laboratory criteria (CD4 levels of less than 200 cells/ μ L) compared with clinical criteria, that is the development of an opportunistic illness (Longini, Clark and Gardner et al, 1991; Osmond, Charlebois and Lang, et al, 1994).

Survival time from the development of AIDS varies according to the AIDS-defining event. For example in the Multicenter Hemophilia Cohort Study, median survival after a single AIDS-defining condition ranged from 3 to 51 months for the 10 most common conditions and the median survival time after diagnosis of AIDS in the United States prior to the availability of antiretroviral treatment was 10-12 months (Gail, Tan and Pee et al, 1997).

2.7 Cofactors of Disease Progression

Endogenous biological or psychological factors, other infections, behaviours or other environmental factors that alter the natural history of HIV infection may be cofactors for disease progression. Understanding the cofactors for HIV disease may shed light on the pathogenesis of HIV as well as on identifying potential interventions that could slow disease progression. The factors covered in this section include host factors, viral factors, co infections and long-term nonprogressors.

A number of host factors influence HIV disease progression. Individuals who acquire HIV at an older age tend to have more rapid disease progression and shorter survival times (Moss, Bacchetti and Osmond, et al, 1988; Bacchetti Osmond and Chaisson, et al 1988). The effect of age is seen in prospective studies of HIV-infected haemophiliacs because the subjects in those studies represent a wide range of ages. Age remains an independent cofactor for disease progression in haemophiliac cohorts after adjustment for HIV RNA blood level (O'Brien et al, 1996). The age effect was similar in haemophiliacs and homosexual men suggesting that it is an endogenous host factor that acts as a cofactor in all HIV transmission groups. (Rosenberg, Geodert and Biggar, 1994).

Genetic differences in human leukocyte (HLA) antigens have also been observed as a potential cofactor. Kaslow et al (1990) found an association between the presence of the HLA antigens A1, Cw7, B8, and DR3 and the rate of CD4 lymphocyte decline in homosexual men. Louie et al. (1991) were unable to find the same associations as Kaslow's team whilst similar findings were reported by Steel et al. (1996) in a study of 18 haemophiliacs, although these investigators did not look at the Cw7 antigen. These genes have been associated with disorders of suppressor cell activity and they might affect HIV disease progression through autoimmune or immune activation phenomena.

Behavioural or psychological host factors can influence HIV disease progression. More rapid HIV disease progression has been reported with unprotected anal intercourse, smoking, depression and poor nutrition (Moseson et al, 1989; Royce and Winkelstein 1990, Vittinghoff et al, 2001). For example Royce et al (1990) observed that there is a link between smoking and increased rate of disease progression. Burack et al(1993) also reported an association with depressive symptoms and more rapid loss of CD4 lymphocytes in San Francisco Men's Health Study. Moseson (1989) observed that malnutrition as a cofactor is known to affect immunocompetence before AIDS epidemic so it is likely that severe malnutrition of the type observed in developing countries may accelerate HIV disease.

Viral subtypes have been observed to be an important determinant of disease progression. For example, faster rates of disease progression have been observed in Ugandan individuals infected with subtype D compared with subtype A isolate (Kaleebu, French and Mahe et al, 2002). Additionally, rare individuals who are infected with variant HIV strains particularly those with nef gene product may experience slower disease progression (Kirchhoff, Greenough and Brettler et al, 1995).

Saravolatz et al (1996) observed that patients with prior histories of opportunistic infections have higher mortality rate. The development of opportunistic infections during HIV disease does not only indicate the degree of immunosuppression but may also influence disease progression itself. Saravolatz et al (1996) observed that in a

large sample of 5,204 HIV-infected patients mortality rates were higher in all CD4 strata for patients with a history of opportunistic infections prior to entry in the study. For example, oral disease is among the earliest clinical manifestation of HIV and the presence of oral candidiasis and hairy leukoplakia has been associated with a higher risk of subsequent disease (Katz et al, 1992). For many of these infectious agents, the relationship with HIV disease may be synergistic. HIV-mediated immunosuppression changes the host control of infectious agents resulting in disease and the disease process in turn activates HIV thus hastening the rate of immunosuppression.

Buchbinder and Kaslow (1998) observed that a small subset of individuals infected with HIV, probably less than five percent, remain free of symptoms, achieve good control of HIV viral replication and maintain high CD4 counts in the absence of antiretroviral medications over many years of infection. However, some individuals initially identified as long-term nonprogressors (LTNPs) have experienced disease progression over time. In general, LTNPs appear to have strong cellular immune responses to a variety of HIV antigens (Rosenberg et al 1997).

2.8 Modes of Transmission of the Virus.

Prior to the identification of HIV-1 as the causal agent for AIDS, a variety of etiologic theories were considered. Such theories included cytomegalovirus infection (Gottlieb et al, 1981), drug use such as amyl nitrates, or an unidentified sexually transmitted agent (Marmor et al, 1982). HIV has now been isolated from blood, seminal fluid, saliva, tears, and breast milk of infected individuals (Geier, Gurtler and, Klauss et al, 1992). HIV-1 DNA sequences have also been detected in pre-ejaculatory fluid (Ilaria, Jacobs and Polsy et al, 1992). In genital fluids, HIV may be found in both cell-free and cell-associated compartments but it is unknown which is responsible for productive infection (Alexander, 1990). Viral concentrations in tears and saliva are comparatively low and there are substances in saliva that appear to inhibit infectivity. No cases of HIV infection have been documented to arise from contact with non-bloody saliva or tears.

The commonest mode of transmission of the virus throughout the world is by sexual intercourse whether anal or vaginal. Transmission also occurs through the receipt of infected blood or blood products, donated organs and semen through the sharing or reuse of contaminated needles by injecting drug users or for therapeutic procedures and from mother to child at birth and via breast milk.

Sexual transmission is the most common route of spread of human immunodeficiency virus (HIV) with heterosexual transmission of HIV infection accounting for about 90 percent of those infected in 1992 and over 75 of those infected to date worldwide (World Health Organisation, 2005).

Unprotected anal intercourse is the most effective mode of sexual transmission of HIV (Detel, English, and Visscher, et al 1989). It is practiced not only by homosexual and bisexual men but also by heterosexual couples. Whereas this practice seems to be unusual in sub-Saharan Africa, it may play a significant role in the spread of HIV in Latin America, in selected Caribbean countries and in some Asian cities (Parker, and Tawil, 1991). Expression of a homosexual lifestyle is severely repressed in most developing countries. Although data on sexual behaviour in men underestimate homosexual activity for this reason, indicators such a declining male-to-female ratio among AIDS cases suggest that heterosexual intercourse has become the driving force of the epidemic worldwide.

Vaginal-penile heterosexual intercourse is an efficient mode of transmission of HIV, particularly from infected woman to man (Johnson, and Laga, 1988). However the efficiency of heterosexual transmission can be enhanced in the presence of well-defined biological risk factors, including more advanced immunodeficiency in the infecting partner, the presence of conventional STDs in either partner, lack of circumcision, anal intercourse, and sex during menses or use of vaginal products (Piot, Laga, and Ryder, et al, 1990). Evidence indicates that the presence of ulcerative and non-ulcerative STDs facilitates sexual transmission of HIV and more importantly, STD control does have an impact on the rate of HIV infection (Ghys, Fransen, and Diallo, et al, 1997). Probably a rampant and sustained heterosexual epidemic in developing countries is only possible because of a common occurrence of such

amplifying factors in addition to high-risk sexual behaviour patterns. At population level the overall efficiency of heterosexual transmission may increase as the epidemic progresses as more infected people become immunodeficient. This may be a relevant factor currently in Africa. It has also been observed that HIV acquisition is not only as a result of individual risk behaviour but also is determined by 'societal' vulnerability such as urbanisation, single-sex migration, or low status of women, factors that increase 'individuals' vulnerability to HIV (Craiel, Buve, and Awusabo-Asare, 1997).

Vertical transmission from mother to child, during or after pregnancy, is the second most common mode of spread of HIV. It is a major source of HIV infection in children and has become a public health problem especially in Africa. Frequencies of mother-to-child transmission observed in Africa are higher compared with those found in the Western world (Ryder, and Temmerman, 1991). In the absence of interventions, mother to child transmission occurs in approximately 25 percent of live births to HIV-infected mothers (Connor, Sperling, and Gilbert et al, 1994). About one-third of cases of mother-to child transmission result from breast-feeding and the risk increases with the duration of breast feeding (Richardson, John-Stewart, and Hughes et al, 2003). Thus intervention to prevent mother to child transmission at delivery may be largely negated if mothers are not provided with safe alternatives to breast-feeding. This risk factor is particularly important in mothers who become infected during late pregnancy or lactation. In this situation, the risk of transmission through breast milk may be as high as 50 percent (Van de Perre, Simonon, and Msellati, et al, 1991). However the rate of postnatal transmission of HIV-1 from mothers who were already infected before pregnancy is lower, estimated between 5 and 15 percent (Wiktor, Ekpini, and Nduati, 1997). Although mother-to-child transmission of HIV-2 undoubtedly can occur, it seems unusual with a much lower rate of transmission than for HIV-1 (Adjorlolo, De Cock, and Ekpini, et al, 1994).

Blood transfusion is the most efficient of all the modes of HIV transmission. Almost all recipients of HIV-seropositive blood become infected (Colebunders, Ryder, and Francis, et al, 1991). Whereas transmission by contaminated blood transfusion has become unusual in most Asian and Latin American countries, blood transfusion remains the third most common route of HIV infection in Africa after heterosexual and perinatal transmission, especially among children. This is largely because of an

inability to implement screening of blood donors and other measures to ensure a safe blood supply as a result of weak health care systems (Van de Perre, Diakhate, and Watson-Williams, 1997).

Intravenous, illegal drug use is mainly associated with the major routes of drug trafficking and recent shifts in several populations from smoking or inhaling drugs to injection (Des Jarlais, Freidman, and Choopanya et al, 1992). In Latin America and the Caribbean, sharing of injection equipment among drug users is the most common route of HIV transmission by blood. The spread of HIV-1 among injecting drug users in Bangkok in the late eighties parallels closely the patterns of spread of HIV-1 among injecting drug users in several cities in the industrialised world. Imprisonment and injecting with equipment previously used by multiple drug users were the strongest predictors of HIV-1 infection (Choopanya, Vanichseni, and Des Jarlais, et al, 1991).

The role of injections administered for medical reasons in the spread of HIV in the developing countries is not well documented but it is probably low (Van de Perre, Diakhate, and Watson-Williams, 1997). However outbreaks in Eastern Europe demonstrate that injection-associated nosocomial HIV transmission is possible and it may be a source of HIV infection in the community (Hersh, Popovici, and Apetrei, et al, 1991). The occupational risk to health care workers from needle sticks involving known HIV-positive source patients was found to be 0.33-0.5 percent in data prior to the availability of anti-retroviral therapy (Cardo, Culver, and Ciesielski et al, 1997). Factors increasing the risk of HIV acquisition from an occupational needle stick include deep injury, injury with visibly bloody device or injury with a device that had been previously used in the source patient's vein or artery (Cardo, et al, 1997).

2.9 Global patterns of HIV spread.

Three epidemiological patterns have been defined by Chin and Mann (1988) on a global scale, pattern 1, pattern 11 and pattern 111, each with a well defined spatial distribution.

Pattern 1

In pattern 1 regions, the spread is primarily through homosexual activity and IVDU. Males dominate amongst those infected with a male to female ratio of about 10-15:1. Regions with pattern 1 spread are the United States, Western Europe and Australia. The early stages of the epidemic in South Africa were characterised by this pattern.

Pattern 11

Pattern 11 spread is primarily through heterosexual intercourse with the male to female ratio at about 1:1. As a result of vertical transmission, pattern 11 spread is also characterised by an increasing incidence of paediatric AIDS. Regions affected by pattern 11 spread are sub-Saharan Africa, Latin America and India. Over time pattern 11 has overtaken pattern 1 as the principal mode of the spread in terms of absolute numbers affected.

Pattern 111

In pattern 111 regions, the spread of the virus is relatively small compared to pattern 1 and pattern 11 areas. Often infected needles are a possible cause of the spread of the virus or the virus is contained within a small prostitute population. Countries of South East Asia, especially Thailand with its active sex tourism industry (Usher, 1992), are most at risk. Eastern Europe as well as Russia falls into this category.

Whilst providing a useful categorisation of different profiles of the disease geographically, recent evidence indicates that such a sharp categorisation of the epidemic pattern may have become blurred with pattern 11 dominating as the major method for the spread of the epidemic even in the industrialised west and the United States of America.

2.10 HIV/AIDS surveillance systems

The surveillance of HIV/AIDS epidemic is crucial in monitoring and planning interventions to control the epidemic but the surveillance of HIV unlike other pandemics posed a formidable challenge to epidemiologists and public health officials. First, although HIV/AIDS was discovered in 1979, the virus was not isolated

until 1983. Prior to the isolation of the HIV virus, it was difficult to monitor the epidemic since the causative agent was not known. Secondly, the long incubation period means that AIDS cases represent HIV infection that occurred some years before which makes timely monitoring and surveillance difficult. Also the epidemiology of the HIV virus was not fully understood in the initial stages of the epidemic and there was no standardised method of surveillance system in place worldwide. Although the epidemic has now entered its third decade, surveillance continues to pose a problem especially in resource-constrained countries such as sub-Saharan Africa.

The following sections review the HIV surveillance systems in sub-Saharan Africa and other available survey data to assess the adequacy and gaps in knowledge for planning purposes.

2.10.1 AIDS Case Reporting

At the start of the epidemic, AIDS case reporting was the main mode of surveillance of the epidemic in sub-Saharan Africa. Although universal AIDS case reporting is a rich source of information for advocacy and planning interventions, sub-Saharan Africa's experience with it has proved of limited value for national responses to HIV/AIDS (WHO, 2005). This is because AIDS case data in sub-Saharan Africa have been grossly affected by under-reporting making it impossible to determine either the burden or trends in AIDS cases in countries in the sub-region. Moreover, AIDS cases represent infections that occurred many years before. Since HIV infection is latent, countries that focus only on AIDS case surveillance are likely to miss potential hidden epidemics and associated risk factors (WHO, 2005). This may result in delays in providing timely action.

2.10.2 Morbidity and Mortality from HIV/AIDS

Another system of surveillance of the HIV/AIDS epidemic is that of morbidity and mortality from HIV/AIDS but sub-Saharan African countries are countries with poor vital registration systems. Salomon and Murray, (2001) observed that not all illnesses are reported to the hospital. Secondly, traditional medicines are more affordable and accessible hence people do not patronise the hospitals for cases to be captured. Also

deaths that occur outside the hospital are not reported nor recorded and in most cases people are buried without a death certificate. In sub-Saharan Africa about two-thirds of the general population live in a rural setting without access to vital registration systems. The stigma attached to HIV/AIDS is an additional deterrent for people to report AIDS case morbidity or mortality of relatives and friends to the vital registry. This means that deaths as a result of HIV/AIDS are under reported and as a result morbidity and mortality data on HIV/AIDS cannot be used to determine the prevalence of HIV/AIDS in the sub-region (Salomon and Murray,2001).

2.10.3 HIV surveillance among ANC attendees

In most countries worldwide, HIV surveillance systems rely on monitoring prevalence among Antenatal Clinic Attendees (ANC). This is because the prevalence of HIV among pregnant women is a good indicator of the epidemic's spread in the general population since the level of HIV infection among pregnant women is similar to that in the general population of men and women aged 15-49 years (Zaba and Gregson, 1998). Blood samples taken from pregnant women during routine antenatal care are unlinked and screened anonymously for HIV antibodies. Several studies in sub-Saharan Africa have compared HIV prevalence in ANC attendees and the general population. In five sites in Tanzania, Uganda and Zambia, the overall HIV prevalence in ANC has been found to be 10-30 percent lower than that in women in the general population (Fylkesnes, et al, 1998; Kwesigabo, et al, 2000). The HIV prevalence in teenagers in ANC was higher than that in women of the same age in the population in two areas in Zambia and similar to that in women in the population in the study area in Uganda (Kilan et al, 1999).

HIV surveillance among ANC attendees remains vital to monitoring HIV trends and the epidemic progress. Most countries in sub-Saharan Africa continue to rely mainly on ANC attendees to monitor both trends in the HIV infection and its magnitude. In 2004, a review of the quality of HIV surveillance worldwide concluded that the trend among 53 countries with generalised epidemics most of them in Africa has been towards improved HIV surveillance systems with 58 percent of the countries having fully implemented systems an increase from 24 to 31 countries (Aceijas et al.2004).

There are several potential biases in inferring general population HIV prevalence from findings among ANC attendees (Boisson, et al, 1996. Zaba, and Gregson, 1998). In the first place not all pregnant women attend ANC and attendance is likely to vary by age, locality, socio-economic status, education level, parity, ethnic group and religion, factors that are likely to be associated with HIV status (Zaba and Gregson 1998). In most parts of sub-Saharan Africa, Traditional Birth Attendants (TBAs) play a very important role in antenatal care especially in the rural areas where accessibility to the hospital in term of distance and cost are concern. Most people therefore prefer to use the TBAs who are readily accessible and are known in the localities. This means that the sentinel surveillance centres will capture only a small fraction of pregnant women. Besides that ANC only see sexually active women and those who are fertile but Carpenter (1997) and Gray, et al (1998) observed that HIV-positive women have lower fertility rate than their non-HIV positive counterparts. HIV infection may reduce fertility directly or indirectly through association with other sexually transmitted diseases and for social as well as biological reasons.

Although subject to biases, ANC-based sentinel surveillance forms the basis for national estimates of HIV prevalence in sub-Saharan African countries. Given the fact that vital registration system is poorly developed in Africa and AIDS case reporting is of limited use due to under-reporting, ANC-based surveillance provides the best estimates for monitoring the both the trends and magnitude of HIV infection. The epidemic can be monitored from the same sentinel site for several years to determine the pattern and trend of infection and the geographical distribution and coverage of the sentinel sites can be increased and thus makes spatial and temporal comparison about the epidemic spread possible.

2.10.4 Population-based HIV serosurveys

Population-based surveys are a very important method of HIV surveillance but due to the intrusive nature and high cost and follow-ups, few countries in sub-Saharan Africa have used it as a way of HIV surveillance. In the epidemic's early stages in the late 1980s only four countries (Burundi, Cote d'Ivoire, Rwanda and Uganda conducted national population-based surveys. In the last five years, there has been renewed interest in conducting national population-based HIV serosurveys in the WHO

African Region where out of the 46 countries, 17 have carried them out since 2001 (WHO,2005). Countries in the sub-region have adopted two major approaches to conducting national population-based serosurveys. Cameroon, Ghana, Guinea, Kenya, Lesotho, Malawi, Senegal, mainland Tanzania and Zambia have added an HIV testing component to the already-planned Demographic and Health Surveys, now widely referred to as DHS+. Burundi, Niger, Sierra Leone, South Africa, Uganda, Zanzibar-Tanzania and Zimbabwe have carried out special HIV/AIDS surveys, some of them called AIDS Indicator Surveys (AIS). The choice of what type of approach to adopt depends on the survey's objectives and availability of financial resources.

Population-based surveys have two main advantages over ANC HIV surveillance. First, they can provide information on both men and women unlike the exclusively female sample attending ANC. Secondly, the sampling techniques applied in population-based surveys include rural populations living in remote places not covered by ANC sentinel surveillance (Zaba and Gregson, 1998).

Population-based surveys should not however be considered as the gold standard even if they are conducted on a much wider cross-section of the population than ANC sentinel surveillance. The biases in the population-based survey include non-response rate and they exclude persons living in special institutions such as schools, hostels, and hospitals. Also, for population-based surveys at a national scale to be cost-effective, they need to be repeated every 5-10 years and thus are not ideal for monitoring trends as it will take at least 15 years to get sufficient data points to establish a trend (WHO, 2005)

2.10.5 Special populations surveys/studies

Studies and surveys on special population subgroups constitute another method of HIV surveillance. Special populations are defined by UNAIDS as those populations at higher risk of HIV infection such as those whose sexual behaviours make them vulnerable. These subpopulations include sex workers and their clients, long distance truck drivers, the military, STD patients, migrant labourers and youth. In countries of low-level epidemics, monitoring of HIV, STIs and sexual behaviours in special populations such as sex workers, military recruits, long distance truck drivers and STI

patients who are at higher risk for HIV infection is the backbone of HIV surveillance. Studies conducted in sub-Saharan region show that there is substantial sexual mixing between population at higher risk for HIV infection and the general population. For example a study conducted in Cotonou in the republic of Benin and in Accra in Ghana shows that the transmission in the capital cities was due to clients of sex workers transmitting HIV to their partners (Lowndes et al; 2002; Cote et al, 2004). Thus HIV infection in the population at risk has a bearing on the dynamics of the spread of HIV. Monitoring of HIV prevalence trends in these special populations is useful for determining the hotspots of infection and hence directing the design of appropriate and timely interventions in contexts both of generalised and low-level infection.

2.10.6 Behavioural surveys

Another indirect method of surveillance of the HIV epidemic is the collecting of behavioural data. Information on behaviours contributes to understanding HIV infection trends. The Demographic and Health Surveys (DHS) are the main source of data on behaviours in the region. These surveys are conducted every five years in many countries including sub-Saharan Africa region. Other sources include the Behavioural Surveillance Survey (BSS) and the UNICEF Multiple Indicator Cluster Surveys. However because it takes some time before any noticeable change in behaviours is registered among the general population, behaviour surveys need to be repeated at least at a five-year interval in the general population in order for them to detect behaviour trend (WHO, 2005). BSS as a surveillance method is therefore not suitable in determining the epidemic trend especially in the short term but may be used to supplement other surveillance systems to fill in gaps in knowledge about the spread of the epidemic.

2.10.7 Challenges facing HIV surveillance in sub-Saharan Africa

Evans et al (2002) observed that as a chronic infection with a prolonged latent period of incubation, HIV requires multiple sources of surveillance information for its changing epidemiology to be well understood. Even though the various surveillance systems are used in Africa, they are not without problems.

First, when the epidemic surfaced in the early 1980s, the virus was associated with certain social and cultural behaviours often termed risk-behaviours such as homosexuality and intravenous drug users (IVDU). In Africa, HIV/AIDS was associated with people who had returned from their travel abroad and with prostitutes. In the early phase of the epidemic, surveillance programmes were therefore geared towards these risk groups and before it was realised that HIV/AIDS was a problem for the general population, the epidemic had spread to a pandemic proportion.

Secondly, while developed countries already had infrastructure in place for epidemic surveillance due to experience with past epidemics and only had to adjust programmes already in place for surveillance of other infectious diseases, developing countries and sub-Saharan African countries in particular had to develop surveillance system from the scratch (WHO, 2005) Again, before the surveillance systems became operational, the epidemic had spread to the general population and has attained pandemic proportions.

A third significant factor affecting HIV surveillance in sub-Saharan Africa is that of political instability, bad governance and conflicts and wars. The HIV/AIDS epidemiological surveillance update for the WHO African region, 2005, indicates that countries such as Liberia, Somalia, Guinea Bissau and Equatorial Guinea had no ANC HIV sentinel surveillance system in place while other countries in the sub-region such as Democratic Republic of Congo, Congo, Burundi, Rwanda and Eritrea had only few sentinel sites for monitoring the epidemic (Table 2.4) In such countries where instability and civil war are accompanied by mass population movements of refugees across borders, epidemic surveillance becomes difficult at exactly the time when favourable conditions are created for the spread of HIV among the population.

The WHO update on HIV/AIDS, 2005, also shows that although ANC sentinel surveillance has been established in many countries in sub-Saharan Africa, it is not yet able to function as required due to resource constraints; for many countries in the sub-region the last round of sentinel surveillance was conducted in 2001 and 2002 (WHO, 2005)

Table 2.4 ANC HIV sentinel surveillance activity in the WHO African region by country

Countries	Total population (thousands)	Urban population (%)	ANC coverage (%)	Year of last ANC round and data available	No. of ANC sites	HIV data available on young people Yes or No
Southern Africa	121 356	42				
Angola	14 078	35	-	2004	25	N
Botswana	1 795	49	97	2003	21	Y
Lesotho	1 800	29	88	2003	6	Y
Malawi	12 337	15	90	2003	19	N
Mozambique	19 182	33	71	2004	35	N
Namibia	2 011	31	91	2004	24	N
South Africa	45 214	58	94	2003	400	Y
Swaziland	1 083	27	-	2004	17	Y
Zambia	10 924	40	96	2002	24	Y
Zimbabwe	12 932	36	93	2004	19	Y
Eastern Africa	189 056	22				
Burundi	7 068	9	79	2003	7	Y
Eritrea	4 297	19	49	2003	12	Y
Ethiopia	72 420	16	27	2003	65	Y
Kenya	32 420	34	76	2003	35	Y
Rwanda	8 481	6	92	2003	24	Y
United Republic of Tanzania	37 671	33	93	2004	57	Y
Uganda	26 699	15	91	2002	17	N
Central Africa	89 847	37				
Cameroon	16 823	50	75	2002	27	Y
Central African Republic	3 912	42	67	2002	48	Y
Chad	8,854	24	42	2003	16	Y
Congo	3 818	66	-	2002	5	N
Democratic Republic of the Congo	54 417	31	-	2004	12	N
Equatorial Guinea	507	49	37	No system	-	na
Gabon	1 351	82	94	2003	3	N
Sao Tome and Principe	165	48	-	No system	-	na
Western Africa	284 009					
Algeria	33 339	58	58	2000	5	N
Benin	6 918	43	80	2003	39	Y
Burkina Faso	13 393	17	61	2004	12	Y
Cape Verde	473	64	99	2004	5	N
Côte d'Ivoire	16 897	44	88	2004	12	N
Gambia	1 462	31	-	2004	6	N
Ghana	21 377	36	88	2004	34	Y
Guinea	8 620	28	71	2004	18	N
Guinea Bissau	1 538	32	62	No system	-	na
Liberia	3 487	45	83	No system	-	na
Mali	13 409	31	47	2002	9	Y
Mauritania	2 980	59	48	2001	13	N
Niger	12 415	21	41	2000	5	Y
Nigeria	127 177	45	64	2003	86	Y
Senegal	10 339	48	77	2003	12	N
Sierra Leone	5 168	37	68	2003	8	N
Togo	5 017	34	82	2004	19	Y
Indian Ocean	20 004					
Comoros	790	34	74	2004	3	N
Madagascar	17 901	30	73	2003	94	N
Mauritius	1 233	42	-	No system	-	na
Seychelles	80	65	-	2003	ns	ns
WHO African Region	704 272	36				

Y=Yes, N=No, na=not available, ns=not specified

source: WHO, 2005

Notwithstanding the challenges facing the surveillance of the HIV/AIDS epidemic in sub-Saharan Africa, some countries have been able to improve upon their surveillance system and to increase the number of ANC sentinel surveillance sites. The introduction of less invasive techniques to collect specimens in population-based HIV surveys, such as collection of blood onto filter paper using a finger prick (dried blood spots) and a saliva based test (mucosal transudate) enable more countries in the sub-region to conduct population-based HIV surveillance. Out of the 46 countries forming the WHO African Region, 17 have carried out a population-based survey in the last five years, while Eritrea and Guinea have conducted population-based surveys in sub-populations considered to be vulnerable and at higher risk to HIV infection (WHO 2005 update).

Although, considerable efforts are being made to improve upon HIV/AIDS surveillance in the sub-Saharan African region, and to provide reliable data on the epidemic for planning intervention, the current surveillance infrastructure in place and the limited resources cannot cope with the increasing infection rate of the epidemic. The gaps in data regarding, the source of the epidemic, dates for the inception of the epidemic and the pattern and trends in its spread a result of the poor surveillance system can be augmented with estimates generated using modelling. Based on the available data obtained from the existing ANC surveillance systems, epidemic modelling system which forms the core of this study would be used to generate the needed estimated about the magnitude and trend of the epidemic in sub-Saharan Africa for planning and disease control.

2.11 Summary

The review above shows that although HIV was discovered in 1979, a definite source of the virus and the date for the inception of the epidemic is not known. Besides, the epidemic has spread to all countries in the world with sub-Saharan Africa alone accounting for two-thirds of all cases in the world. This led to some researchers hypothesising that the epidemic started in sub-Saharan Africa. The main mode of the spread of the virus is heterosexual and again this led to the hypothesis that the main explanations to the rapid spread of the virus in sub-Saharan Africa can be based on individual human behaviour and culture but again the processes involved in the

epidemic spread is unclear. There are others who believe that individual human behaviour plays out in a particular socio-economic and political environment that makes people vulnerable to the HIV virus. The review also shows that the rapid spread of the epidemic as well as rapid disease progression may be due to the presence of particular co-factors of disease progression namely presence of opportunistic infections but again this plays out in the socio-economic and political environment that make people vulnerable to opportunistic infections and hence AIDS. The review also shows that though the effective way of controlling the virus is through prevention, most of the prevention programmes based on behaviour change have failed to reduce the spread of the virus but the reason for the apparent failure is unclear. To address these gaps in the knowledge of the HIV epidemiology in sub-Saharan Africa, some of the theoretical approaches used to explore the epidemiology of HIV are reviewed in the proceeding chapter.

CHAPTER THREE

3.0 HYPOTHESISED CAUSES AND APPROACHES EXPLAINING THE SPATIAL EPIDEMIOLOGY OF HIV/AIDS IN SUB-SAHARAN AFRICA

3.1 Introduction.

Africa is a continent with 52 countries with diverse cultural, social as well as great spatial diversity. Such diversity makes the understanding of both the epidemiology of HIV and the reported effects of the epidemic difficult to comprehend. In order to understand the spatial epidemiology of HIV/AIDS in sub-Saharan Africa, the first part of this chapter presents the pre-independence African history and how this may have shaped the nature of the HIV/AIDS epidemic as seen today in sub-Saharan Africa. The second section presents the spatial variability of the HIV/AIDS epidemiology based on UNAIDS 2004 prevalence estimates. This is followed by a review of the hypothesised cause of the epidemic spread namely; the military involvement hypothesis, migrant labour hypothesis and the truck town hypothesis and how these help in explaining the epidemic spread using different case studies in sub-Saharan Africa. Lastly, in order to understand the rapid spread of the HIV virus in sub-Saharan Africa and to explore the processes which create such risk environments, many approaches have been adopted. These include the cultural (behavioural) approach, political economy approach and the vulnerability approach based on policy options, choice or focus. These approaches are reviewed and a synthesised framework based on these approaches is adopted for explaining the epidemic spread in the Krobo district, the chosen study area. The chapter ends with a discussion and conclusion.

3.2 Pre Independence African history and the spread of HIV/AIDS in sub-Saharan Africa.

'Epidemics do not just happen and are not random events, they have histories. Histories of infectious diseases reflect the ways in which channels and paths of infection have been created as part of material and cultural lives of societies (McNeil, 1976)' These statements presupposes that there are processes through which epidemics start and spread. The foundation of the continental wide risk to HIV/AIDS

can be traced back to the slave trade period and its associated disruption and dislocation of African societies. Oliver, (1991 in Barnet and Whiteside, 2002) observed that demand for labour to work on plantations in America led European merchants turned to the west coast of Africa from today Senegal to Angola to meet this demand. On the east coast of Africa, the Arab traders made similar forays into the hinterland to meet demand for slaves around the Indian Ocean. Thus from both west and east coast, the slave trade extended disruption through African intermediaries far into the heart of the continent. This created a sustained disruption and dislocation of African societies that created the risk environment for epidemics to thrive. Thus it could be argued that epidemics in Africa are a product of histories that have made the continent's societies 'unhealthy' (Barnet and Whiteside, 2002). This is the foundation for the disorder, inequality, exploitation and poverty in which an epidemic such as HIV/AIDS could grow and thrive (Barnet and Whiteside, 2002). Against this backdrop, the African HIV/AIDS epidemic has to be seen in the broad spectrum of African history (Barnet and Whiteside, 2000). Aspects of this dislocation as they are manifesting today have been exacerbated by a combination of war or civil strife, food shortage and population displacement which is termed 'complex emergencies' (Toole, 1999; Burkle, 1999). The processes through which these complex emergencies create the risk environment have been variously hypothesised by Smallman-Raynor and Cliff, 1991 (the military/civil war hypothesis), Larson, 1990 (migrant labour hypothesis) and Wood, 1988 (the truck town hypothesis). A review of these hypotheses enables us to understand the spatial spread of HIV/AIDS in sub-Saharan Africa.

3.3 Spatial Spread of HIV in sub-Saharan Africa

Figure 3.4 is based on UNAIDS sentinel surveillance data and it shows the estimated number of adults who were HIV positive in 2003. There is a marked variation in HIV prevalence between Western, Central, Eastern and Southern Africa sub-region. Southern Africa is the most highly infected region with Swaziland and Botswana recording an adult HIV prevalence rate above 30 per cent, while Lesotho, Zimbabwe, South Africa and Namibia had prevalence rate above 20 per cent (see figure 3.1 and Table 3.1). In east Africa, Malawi recorded the highest HIV prevalence rate of 14.2 per cent followed by Tanzania 8.8 percent while in Central Africa; Central Africa

Republic recorded the highest HIV prevalence of 13.5 percent. In West Africa, Cote d'Ivoire had the highest prevalence of 7.0 per cent followed by Nigeria 5.4 percent. In all West Africa is less affected by HIV as compared to east, central and South Africa. Within the regions, there are marked variations in prevalence. For example Angola had relatively lower prevalence as compare to neighbouring Namibia and Zambia and in west Africa, the disparity is seen in Benin with lower prevalence while neighbouring Nigeria had a relatively higher prevalence (see figure 3.1. and table 3.1).

In order to understand the observed spatial variation in HIV prevalence in sub-Saharan Africa, the military involvement hypothesis, migrant labour hypothesis and the truck town hypothesis is explored to enable us gain plausible explanation to how the observed pattern of prevalence may have evolved.

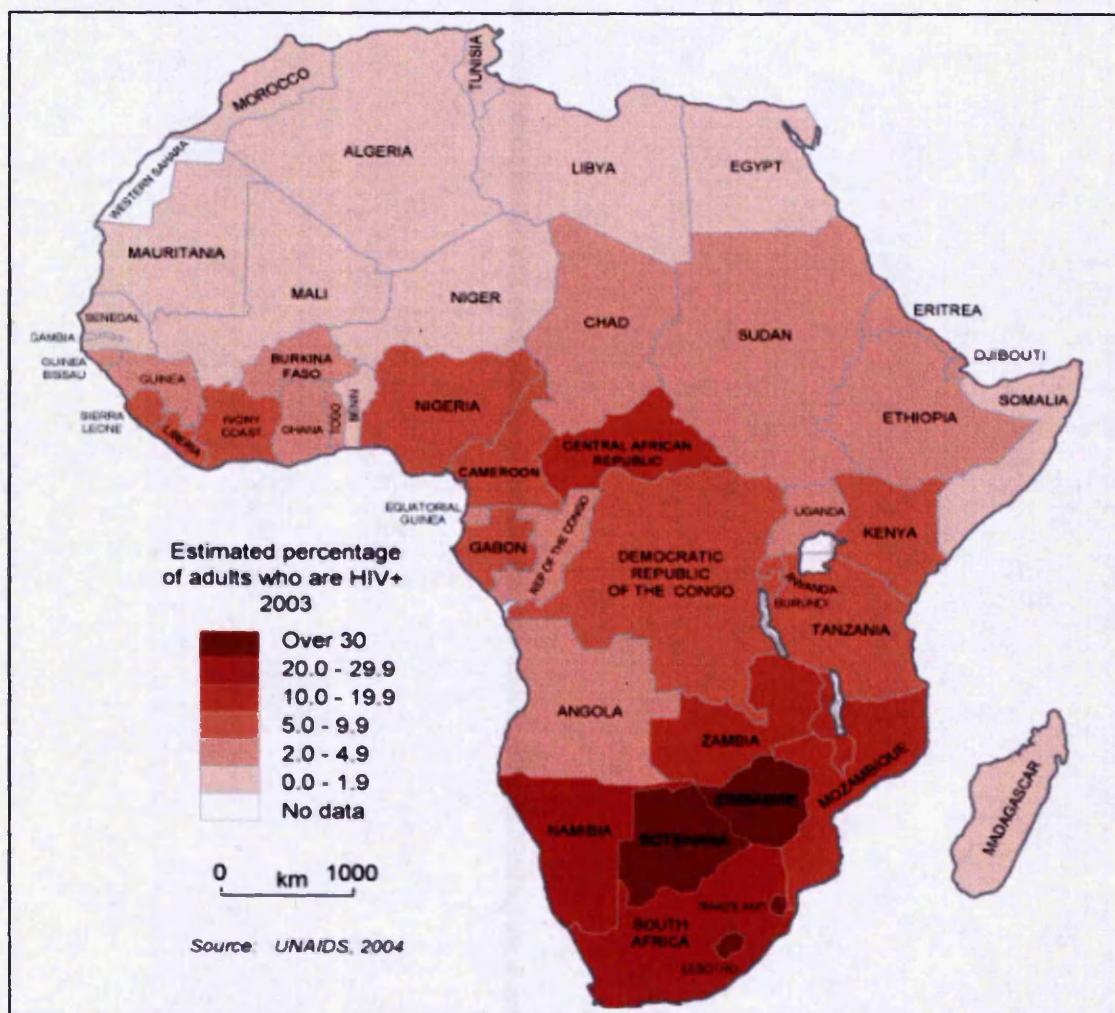


Figure 3.1 estimated percentage of adults who are HIV+ as at 2003.
Source:UNAIDS, 2004.

Table 3.1 Estimated number of adults living with HIV/AIDS

Country	Adult prevalence	Country	Adult prevalence
Angola	3.9	Lesotho	28.9
Benin	1.9	Liberia	5.9
Botswana	37.3	Madagascar	1.7
Burkina Faso	4.2	Malawi	14.2
Burundi	6.0	Mali	1.9
Cameroon	6.9	Mauritania	0.6
Cen Africa Repub	13.5	Mozambique	12.2
Chad	4.8	Namibia	21.3
Congo	4.9	Niger	1.2
Cote d'Ivoir	7.0	Nigeria	5.4
Dem Rep Congo	4.2	Rwanda	5.1
Djibouti	2.9	Senegal	0.8
Eritrea	2.7	South Africa	21.5
Ethiopia	4.4	Swaziland	38.8
Gabon	8.1	Togo	4.1
Gambia	1.2	Uganda	4.1
Ghana	3.1	U A Rep Tanzania	8.8
Guinea	3.2	Zambia	16.5
Kenya	6.7	Zimbabwe	24.6

Source: UNAIDS, 2004

3.4 Hypothesised causes of HIV spread

Three main hypotheses have been proposed to explain the spatial spread of HIV in sub-Saharan Africa namely the military involvement hypothesis, migrant labour hypothesis and truck town hypothesis. The proceeding section explores these hypotheses and how they help in understanding the epidemic spread in sub-Saharan Africa.

3.4.1 The military involvement hypothesis

The military involvement hypothesis takes into account the historical link between servicemen, prostitutes and the spread of STDs. Conflict has been demonstrated to result in the increased sexual mixing of military with civilian population especially in areas of high military concentration for extended period of time (Wollants et al, 1995). Military personnel have frequently used rape to terrorise and drive a population from an area (Stiglmayer, 1994). Secondly during their long periods away from home and family, military and police personnel engage the services of prostitutes or local women to meet their sexual needs (McCarthy et al 1989). It has also been observed by McKinley, (1998) that in conflict times, men with weapons and power opportunistically exploit available women. During the Liberian civil war, nearly half of the civilian women and girls were estimated to have been sexually abused in the first five years of the war (Bauer, 1998). Women raped by military and police personnel are at increased risk of contracting HIV and the risk increases if there are multiple perpetrators or when the women are held by military personnel for prolonged period for sexual purposes as has been reported in emergency situations (Salama et al, 1999). In sub-Saharan Africa where conflict situations involving the military are rampant, one of the processes of the epidemic spread may be due to the increased interaction between civilian population and the military deployed in conflict situation in the sub-region. It has been observed by Smallman-Raynor and Cliff, (1991) that there is a significantly positive relationship between the ethnic composition of the Ugandan National Liberation Army and the current reported spatial pattern of clinical AIDS in Uganda.

Sex as a survival strategy in times of conflicts has also been a risk factor in the spread of HIV/AIDS. Vuylsteke et al, (1996) observed that in an economic and politically vulnerable communities, the use of sex as a survival strategy is a risk factor for HIV transmission particularly in Africa. Among refugee and internally displaced populations, poverty as a result of severing of livelihood strategies and asset loss may compel women to participate in commercial sex or to exchange sex for food and other assets (Asenkeyne et al, 2002). The sexual mixing between the host population, the refugee population and the military on one hand and the sexual mixing between the service personnel and the population of their home country on their return in both

conflict and post-conflict situation may possibly account for the relatively high rate of HIV prevalence in sub-Saharan Africa where conflict are rampant (Salama et al, 1999).

Smallman-Raynor and Cliff (1991) observed that considering the incubation period for HIV which is around eight to ten years, it is likely that current HIV incidence and trends may be due to infection during the conflicts from the continent during the past two decades and thus understanding the processes that created the risk environment will help us to understand the current epidemic spread.

3.4.2 Migrant Labour Hypothesis

The 'migrant labour hypothesis' says that HIV spread from areas of labour demand in urban regions to areas of labour supply in rural regions through return migration. The literature highlights two distinct but related reasons why the migrant labour system in general encourages risky sexual behaviour. First, the separation of familial and stable sexual relationship and second the presence of commercial sex in the migrant receiving areas (Gebrekristos, 2001). Circular forms of sexual networks between rural areas and labour centres has developed where migrants have sexual partners at labour centres while continuing a long distance relationship with their regular partners back in the rural areas. Although less is known about the partners of the migrants in the rural areas, there is some evidence that they also have other sexual partners while their husbands are away in the cities (Gebrekristos, 2001). These migrants come back home when contracts are ended or frequently visit their families in the villages. This sexual networking and multi-partner sex engaged in by migrants increase their risk to HIV infection and thus become agents of HIV spread. It should be noted however that migrant labour is neither the only nor the major avenue for the spread of HIV. Long-distance road haulage and cross-border itinerary traders may play a significant role in spreading HIV (Chirwa, 1995).

3.4.3 Truck Town Hypothesis

The Truck town hypothesis proposes that the distribution of HIV and AIDS reflects the diffusion process in which major roads act as the main corridors of virus spread. One of the main occupations that has been associated with increased risk of HIV

infection is long distance truck driving, a profession that requires prolonged absence from home and family (Bwayo et al, 1994). HIV spreads outwards along principal corridors of trade and smuggling traffic. As the truckers stop along these trucking routes, towns develop along those routes and female commercial sex workers soon follow into those towns. These truckers and their assistants often have a higher disposable income than the communities through which they pass. As a result of poverty and social disruption along transport corridors, local women along the stops see truckers as a ready source of scarce income (Bwayo et al, 1994). This coupled with the fact that many of the truckers have little knowledge of HIV prevention often leads to unprotected sex which exposes them to a higher risk of HIV infection. An early study of truck drivers in Uganda demonstrated an HIV seroprevalence rate of 35 percent (Carswell et al, 1989). Also in East Africa, it was observed by (Bwayo et al, 1994) that 27 percent of truckers and their assistants were found to have HIV antibodies. A study of Thai long haul truck drivers found that 86 percent of the single men and 63 percent of the married men had commercial sex. In India, long distance truck drivers during their journey pick up sex workers from '*dhabas*' which usually provide food, rest, alcohol and drugs on road side, use them and leave them at some other *dhaba* where they are used by other drivers and local youth (Sigh et al, 1992). In terms of geographic distribution of HIV, truckers may be a particularly important core group.

3.4.4 Case Study 1: Ugandan Civil War and HIV

Hooper (1999) observed from anecdotal studies that warfare and civil unrest in Uganda forced refugees from Uganda to flee to neighbouring Congo, Tanzania and Burundi between 1959 and 1964. These movements caused social disruption as a result of men being separated from women and children during the war. These women and children became vulnerable to sexual abuse and rape in the refugee camps. Although there is no clinical evidence or laboratory-based evidence, it has been hypothesised that these refugees may have been HIV positive and later developed AIDS (Barnett and Whiteside, 2002). The Rakai district in Uganda for example where the invasion army from Tanzania stayed was the first region to report AIDS cases in Uganda, Carswell, (1989) and Hooper, (1999). Also during the Great African War in the Congo, forces from Uganda, Zimbabwe, Angola, Namibia and Rwanda

were involved and this contributed to population movement across this huge region spanning many millions of square kilometres in East and Central Africa. These soldiers engaged the services of prostitutes and in most cases raped vulnerable women and children. Some of the vulnerable women and girls have to exchange sex for survival. The war and its resultant migration and disruption of livelihood created patterns of sexual behaviour and mixing risk that resulted in the spread of the HIV virus across east and central Africa.

Another plausible dimension of the HIV spread in this region is the development of trucking routes which run through the port of Mombassa, through Nairobi to Kampala, on north to Sudan, on west and south to Rwanda, Burundi and Zaire. Kyewalyanga (1976) observed that the illicit trade along this route resulted in younger women becoming prostitutes selling sexual services in return for cash while others became 'kept women' in stable non-resident relationship along the tracking route.

The opportunities for the more casual types of sexual relationship increased along the main transport routes in the 1970's thus encouraging greater mixing between different groups. The high rate of sexual partner change, high mixing of partners across geographical areas, large number of concurrent partnerships, geographical mobility and gender relations rooted in local traditions which was disrupted by disorder increased the levels of individual and social susceptibility to STDs including HIV (Barnett and Whitside, 2002).

3.4.5 Case Study 2: D.R.Congo

In the Democratic Republic of Congo, violence, corruption and war created a risk environment for HIV/AIDS to thrive. The geographical position of the Democratic Republic of Congo is such that it shares border with nine countries and its ties with these countries make it a fertile grounds for HIV/AIDS to spread from there to neighbouring countries in Central Africa and the rest of eastern and southern Africa.

A violent history, constant unrest since independence in 1960 and a state corrupt to the core set the condition in place for an epidemic of HIV/AIDS. The troubled history

of the Congo continues to the present day. Kabila overthrew President Mobuto in 1997 only to be killed within four years. Most of the country is not controlled by the central government and a multi-country war is waging across the Congo involving the armies of the countries in the sub-region including Angola, Congolese, Rwandan, Tanzanian and Zimbabwean army (Barnett and Whiteside, 2002).

The socio-economic instability, war and the resultant displacement of populations, as well as sexual mixing between the armies of the countries involved in the wars has hastened the spread of HIV especially as military forces have higher infection rate than the civilian population. For example it is estimated that HIV prevalence in Angolan and Congolese armies ranges between 40-60 percent while in the Tanzanian army it is between 15-30 percent and it may be as high as 80 percent in the Zimbabwean army (Mills, 2000).

3.4.6 Case study 3: South Africa

The explanation to the South African epidemic is a complex mix of the consequence of labour migration both internal and international, and internal conflict and oppression of the apartheid system of government. In the first place, the South African black population were forced into crowded, impoverished homelands, which led to the breakdown of traditional cultural structures and livelihood. Adults, mainly men, migrated to urban areas to work in white owned factories and mines and live in single-sex hostels. The law prevented them from bringing their families to town. Gilgen et al (2001) observed that if people are put in circumstances where they cannot maintain stable relationship, where they are mobile, where life is risky and pleasures are few then sexually transmitted diseases will be rampant. Apart from the indigenous black population, South Africa attracted migrants from as far as Malawi, and Angola, West Africa and India who came to work. In such conditions the construction and reconstruction of sexuality occurs (Campbell, 2001). In such a high sero-prevalence area, where working and living conditions encourage sexual mixing the risk to HIV infection is very high. Chirwa (1995) observed that infected men return to their home countries where local epidemics are established. Secondly, conflict resulting from the cycle of oppression and resistance led to widespread destruction and disruption of civil society. The South African society

became highly militarised. Armed forces proliferated including the defence force, homeland armies, liberation movements, self defence units and political militias as well as vigilante groups and unofficial police and intelligence units. Crime and gun violence are now endemic in South Africa as a result gun rape has become a potent method of spreading HIV (Institute of Security Studies, 1999). Apart from the internal conflicts, wars were fought by South African army in Angola, Mozambique and Namibia. These soldiers may have been infected with HIV due to contact with prostitutes and might have infected their wives on their return from the war. Thus the break down of family structure in the rural areas and towns, government policy towards the black population and the violence that accompanied the end of apartheid combined to create risk environment for the spread of HIV.

3.4.7 Case study 4: Migrant labour from Ghana to West African states.

In the case of Ghana, labour migration was not fuelled by conflict or wars but by economic decline and its resultant unemployment (Fosu, 1992). Migration was seen as a survival strategy for families to enable them to cope with the difficult economic conditions. Large scale labour migration both skilled and unskilled began in the late 1970s and early 1980s. West African countries notably Cote d'Ivoire and Nigeria were the main destination countries (Anarfi, 1982). Anarfi observed that in the early 1980s, about 300 Ghanaians were migrating to Nigeria every day and by December 1980, about 150,000 Ghanaians had registered with the Ghana High Commission in Lagos. Similarly the 1975 census of Cote d'Ivoire recorded over 42000 Ghanaians in that country and by 1980, the number of Ghanaians in Cote d'Ivoire was estimated between 500,000 and 800,000 (Anarfi and Awusabo-Asare, et al, 2000). Contrary to the popular view that women emigrate less, in the case of Ghana, women dominated short distance emigration to nearby countries accounting for 64, 57, and 56 per cent respectively of the Ghanaian emigrants in Cote d'Ivoire, Burkina Faso and Togo whereas a higher proportion of men travelled further field (Anarfi et al, 2003). Kanyenze (2004) observed that migrant women are often excluded from the formal labour market of the country of destination and often end up in low status; low paid and isolated work which increases their vulnerability to HIV infection. In terms of age female migrants were younger with a mean age between 15 and 24 years while for the men it was between 25 and 34 years (Anarfi et al, 2003). Most of these women were unskilled and ended up as sex workers especially in Cote d'Ivoire where most of the

commercial sex workers were Ghanaians notably from the Krobo district in Ghana (Anarfi, 1993). Brummer, (2002) observed that the situations migrants experience and the behaviours they engage in during and after the migration process are influenced by their characteristics such as sex, age, marital status and educational level. Whereas the men leave their wives and children behind in Ghana, the women especially the Krobo leave their husbands behind in search of jobs. The separation from families and regular partners as a result of migration often leads to risky sexual behaviour (Thiam et al, 2004). This is especially the case for male migrants because of traditional definition of masculinity which usually encourages many sexual contacts for men (Brummer, 2002) and as observed by Ateka (2001) fidelity is not a virtue among African men. Thiam et al (2004) observed that in the context of circulatory labour migration, migrant men are not entirely to blame for the frequency of HIV among their wives. Sex is often used as a surviving strategy by women while their husbands are away especially among those women who do not receive economic support from their migrant husbands. Many migrants spend long time away from home and this situation creates sexual mixing among migrants and the indigenous population and prostitutes and thus created the risk environment for STDs including HIV to spread. These migrants are both at high risk of HIV infection as well as being agents of the spread of HIV in their communities of origin to which they occasionally return.

3.5 Explanatory Frameworks for HIV spread

Depending on policy options, choice and focus of a particular research, three main approaches have been adopted to explain the processes of HIV spread. These approaches are cultural (behavioural) approach, political economy approach and the vulnerability approach which are reviewed in the section below to enable us understand the processes of the epidemic spread in the Krobo district in Ghana.

3.5.1 Cultural (behavioural) Approach

One of the main approaches used in explaining HIV/AIDS spread is the behavioural one. Cultural variables are the main focus of this approach which studies the sexuality and the psychology of individual sexual behaviour. The core of this thinking is that individual sexual behaviour is the main determinant for the spread of HIV and that in

sub-Saharan Africa where the epidemic spread is mainly heterosexual, the epidemic can be controlled by advocating behaviour change based on Abstinence, Behaviour Change and Condom Use (ABC) as an intervention to the spread of HIV. As a result of this perspective, vulnerable groups such as commercial sex workers and the gay community were targets for behaviour change through educational campaigns as a means of modifying those behaviours that put them at risk to HIV infection. In Africa, cultural practices such as polygamous, as opposed to monogamous marriages and the practice of inserting foreign bodies, such as herbs, into the vagina to increase sexual pleasure in some societies were associated with the rapid spread of HIV. Another cultural practice that became a subject of increasing interest was circumcision in males which appears to be protective against HIV infection to a certain extent (Caldwell and Caldwell, 1996). Female circumcision and other forms of female genital mutilation in Africa on the other hand increase the risk of HIV transmission due to rupturing of scar tissue and increased vulnerability of the vaginal wall. Much debate still surrounds the influence of such culturally specific sexual practices and individual human behaviours on the spread of HIV/AIDS. It is in this context that some explanations are made regarding the promiscuity of African men and the tolerance of the African societies towards multiple sexual partners. Also early epidemiological reports, for example of AIDS cases in Zaire, were worded in a way that reinforced cultural and gender stereotypes of assumed black, sexual immorality. The shortcomings of the ethics and reasoning behind such a research methodology is evident by the apparent failure of current interventions in Africa based on behaviour change. For example, in a situation of extreme poverty, vulnerable girls may exchange sex for money irrespective of the risk involved. There is therefore the need to broaden the research horizon to include the political and economic structures in which human behaviour takes place and their possible influence on HIV epidemiology.

3.5.2 The Political Economy Approach

Another major way of explaining the African AIDS crisis is the political economy approach which focuses on the importance of macro issues and their impact on the HIV epidemiology, highlighting economic and political processes such as the debt crisis, poverty, urbanisation and government policy and how such factors influence

the AIDS epidemic (Webb, 1997). This approach situates epidemiology in a historical, economic and political context and has a strong focus on power relations within societies. Here individual human behaviour is deemed to have been determined by global economic and political structures that act at international and national scale but also locally (Lurie, 2001). It is based on this thinking that the sub-Saharan African epidemic is often blamed on the international economic and political structures which are beyond the influence of political leaders and policy makers in Africa

The rapid spread of HIV/AIDS in sub-Saharan Africa as reviewed in the previous chapter may therefore be due to the fact the deterministic nature of both approaches according to Webb (1997) cannot account for the variability found at a local scale but the point is reiterated that the variables determining HIV epidemiology are not just physiological and psychological but also sociological, economic, and political and always rooted in historical context.

Webb conceptualises the social epidemiology of HIV as the study of a constantly changing interrelationship between culture, individual action (agency) and socio-political factors (Fig 3.2). Instead of a narrow focus on one aspect of HIV/AIDS epidemiology, the epidemiologist has to look at the interplay of different factors that facilitate the spread of HIV.

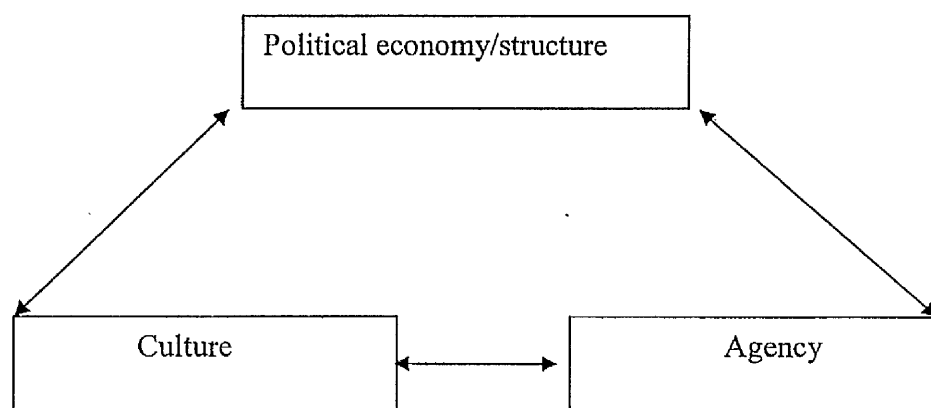


Figure 3.2. Conceptualisation of the study themes within HIV epidemiology (adapted from Schoepf, 1991).

Due to the inadequacies inherent in the two approaches, HIV/AIDS epidemiology should be redefined in terms of subject rather than approach. Thus any approach, which examines some determinants while ignoring others, must be avoided because the determinants act in relation to each other not as separate entities. Conceptualisation of HIV epidemiology must therefore include all cultural, structural and behavioural factors. Thus a holistic approach within a given context would avoid the problems related to the different approach.

In HIV epidemiology simplistic causal relationships do not exist as the number of acting variables is too large to define precisely and by their very nature many have no quantitative value. Because the epidemic has such a considerable but again unquantifiable behavioural aspects, proxy measures of perception, influences on behaviour, knowledge and priorities are needed to produce an estimation of the nature and interaction of the determinants in any one setting. This has important implications for the understanding of HIV epidemiology as behavioural and structural determinants vary across time and space. For example in one village a certain set of factors determines the spread of the virus but elsewhere different sets are likely to be relevant. The likelihood of transmission may have a geography of its own and may be linked with both the incidence of opportunistic infections and STDs. As the determinants of the epidemiology of HIV vary across time and space, the exact factors relating to each incidence of transmission are unique. The identification of causal relationships is thus impossible within a positivist framework and therefore a broader concept of causality is needed. It is therefore crucial to approach AIDS as a disease of society, of political economy and culture both of which can change rather than simply as a virus spread by individuals (Schoepf, 1991). Roundy (1978) demonstrated how the transmission of communicable disease in a rural Ethiopian community varied according to precise location within the community and the different degree of interaction between vectors and their host. The same is true for HIV spread and intervention at a scale of an urban township or compound must be sensitive to the conditions within that community. The assumption that what will work in one place will necessarily work in another must therefore be avoided and is the task of those involved in prevention programmes to understand what particular aspect of a prevention initiative are replicable.

3.5.3 Vulnerability Approach.

Explaining the epidemiology of HIV/AIDS epidemic in sub-Saharan Africa through the concept of vulnerability offers an approach that can combine structural and behavioural determinants across different spaces, scales and time. In the context of the African HIV/AIDS epidemic, vulnerability is related to environmental factors that leave individuals or groups at risk of HIV infection. Webb (1997) defines these risk situations as socially and geographically defined zones where the capacity of the individual to respond effectively to a health threat is reduced. It is in these situations that people are vulnerable. The role of poverty, civil disruption, conflict, gender inequality, migration and population mobility and the processes through which these factors create risk environment are explored.

The links between poverty and HIV/AIDS are complex and not fully understood, however poverty has played and continues to play an important role in the spread of HIV (Whiteside, 2001). Colvin et al (2001) observed that there is a strong relationship between poverty and HIV/AIDS. Colvin argued that in situation of dire poverty, the risk of HIV infection would not be the main concern of people. Poverty can therefore drive women and young girls into prostitution or selling sex for food, money or services. Also, poor people may have less access to health care and condoms (Evian, 1993; Ateka, 2001; Buve et al., 2002). At the household level, it is clear that HIV/AIDS is likely to sustain and even increase poverty. For example illness and caring for the ill forces productive members of the household to give up their jobs with consequent loss of income and labour. At the same time, the household will have to spend more on health care, medicine and funeral services (Booyesen et al., 2002).

Conflict and political instability is another factor which creates the risk environment for HIV/AIDS to thrive. Countries experiencing political and /or economic instability have been more vulnerable to the spread of disease such as HIV/AIDS (Kalipeni, 2000). During conflict or war, the people involved tend to be extremely vulnerable to HIV infection. Soldiers for example living in a stressful environment, separated from

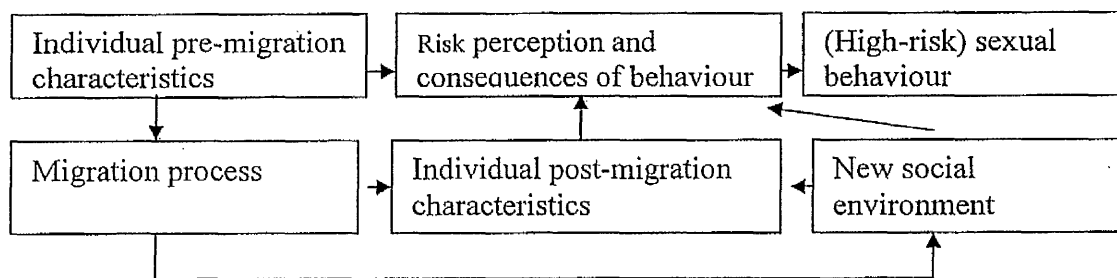
their families are more likely to engage in unsafe sexual activities while the civilians might get exposed to sexual violence and have to survive in situation of extreme poverty which may force them to sell sex (Ateka, 2001; Buve et al., 2002). During conflicts many families and communities are disrupted and women and children are separated from their male partners or parents. These women and children are extremely vulnerable to sexual violence and exploitation. Also health services and the prevention of STDs are not always available and HIV/AIDS is not foremost on the minds of refugees since survival is their ultimate priority (Carballo et al, 2001; Decosas et al., 1997; Tarantola, 1999).

The subordinate social and economic role of women in many African countries creates risk environments for HIV transmission. Gender inequalities are acute in many African societies particularly the patriarchal societies. Cultural and social systems have strict rules concerning female sexuality. Women in these societies have little control over their sexual lives and the sexual lives of their husbands or partners (Buve et al., 2002; Evian, 1993). While women are traditionally and culturally supposed to remain monogamous, their husbands are allowed by tradition to have as many wives or partners as they desire. This culturally prescribed lack of control on their sexual relationships has made women more vulnerable to HIV infection (Buve et al, 2002). This picture of gender inequity is also apparent in the economic sphere. Women are often dependent on men for financial support and need approval from men before they can obtain credit from the financial institutions and consequently find it difficult to enforce condom use or refuse sex (LeBeau et al 2001).

Migration is another factor that creates a risk environment for migrants to be vulnerable. Migrants are more vulnerable to HIV infection than people who do not move (Decosas, 1995, Kane et al 1993; Nunn et al, 1995; Gilgen et al 2001 and Lurie, 2001). This vulnerability is not the direct result of mobility. It is via circumstances and events related to the migration process that high risk of HIV infection is caused by population mobility (Decosas et al, 1997). It is thus the situations encountered and the behaviours possibly engaged in during mobility that increases vulnerability and risk regarding HIV/AIDS (UNAIDS, 2001b). For example Neequaye et al (1988) observed that many Ghanaian women who migrated to Cote d'Ivoire in the early 1970s and 1980s due to the economic crisis of the time could not find jobs and ended

up as sex workers for survival. In this study, of the 242 females who were infected with HIV, 199 (82percent) were prostitutes returning from Cote d'Ivoire with the disease in search for cheaper medical care. Brockerhoff et al, (1999), observed that there are three factors related to migrant sexual behaviour: migrant pre-migration characteristics; changes in individual characteristics due to migration; exposure to a new physical and social environment. These factors play part in the construction of certain perception of risk and eventually have an effect on the actual sexual behaviour of migrants. Figure 3.3 conceptualises the influence of migration on sexual behaviour and how it can impact on the epidemiology of HIV/AIDS.

Figure 3.3 The Influence Of Migration On Sexual Behaviour.



Source: Brockerhoff et al., 1999

The pre-migration individual characteristics include sex, age, marital status, educational level, ethnicity, social status and economic prospects. These characteristics influence the decision to migrate and migrant's risk of HIV infection. The migration process itself changes some of these individual characteristics into post-migration individual characteristics. The most commonly cited post-migration characteristics are separation from regular sexual partner and family. Further more migrants' income is likely to increase in the case of labour migration and decrease in the case of involuntary migration. The third mechanism that links migration with sexual behaviour is the change of environment. The milieu in which migrants are living is likely to differ from the social and cultural environment at home and migrants may not have access to good sexual health services. Migration is very much gendered which can result in a gender imbalance in both the sending and receiving areas (Brockerhoff et al., 1999; Decosas, 1999). Moreover a new social environment can result in a lack of social support, which has been linked to risk-taking behaviour (Campbell, 2001).

The behaviours that migrants exhibit during migration and the extent to which such behaviour increase their vulnerability vary from person to person and from place to place. For example illegal migrants are faced with a different set of risks than others. High risk situations encountered by truck drivers are not comparable to those faced by another groups for example migrant mine workers. Decosas et al (1997), Girdler-Brown, (1998) and Tarantola, (1999) observed that HIV/AIDS education, health services including STD treatment and condoms are not always available to migrant population. This lack of access to public services may be due to cultural, social and language barriers that make communication problematic and lack of money to buy the available services or, in the case of undocumented migrants, their illegal status. Moreover migrants experience many problems living in a new environment, which may influence their mental and physical health. High-risk behaviour such as sex with multiple partners is not solely the result of migration. It is also the result of alienation, of loneliness, of being separated from family and regular partner, as well as breakdown of traditional family units. Women and men leave their familiar environment with traditional norms and values and feel they are freed from personal and community sexual constraint. Also the anonymity of being a foreigner can increase risky sexual activities (Decosas et al 1997; Evian, 1993; Girdler-Brown, (1998).

Migration in sub-Saharan Africa is to a large extent sex segregated. For example in southern Africa migrant mine workers from neighbouring countries who move to work in the mines in South Africa are mainly men. This gender-based migration results in gender disequilibria in both the destination and sending countries (Decosas, 1997). This gender disequilibria and the predominant involvement of young adults who are sexually active create an enabling environment for high rate of partner change and commercial sex. Local women may exchange sex for money, goods, physical security, jobs and transport (Evian, 1993; Tarantola, 1999).

3.6 A Framework for Analysis

This study is thus based on a synthesis of these three approaches to explore the epidemiology of HIV/AIDS in Africa (see fig 3.4) The focus is on how individual

sexual behaviour in the form of the number of sexual partners, sex with casual partners, frequency of sex and condom use is influenced by the environment in which an individual finds themselves in. The synthesised framework explores the relationship between individual sexual behaviour, the broader socio-cultural, economic and political structures and how local environmental conditions combines to create the risk environment for the HIV virus to spread. The outcome of the interaction between these structures provides a holistic view about the nature and pattern of the epidemic spread as well as an in-depth understanding of the reasons for the rapid increase in the epidemic spread in sub-Saharan Africa.

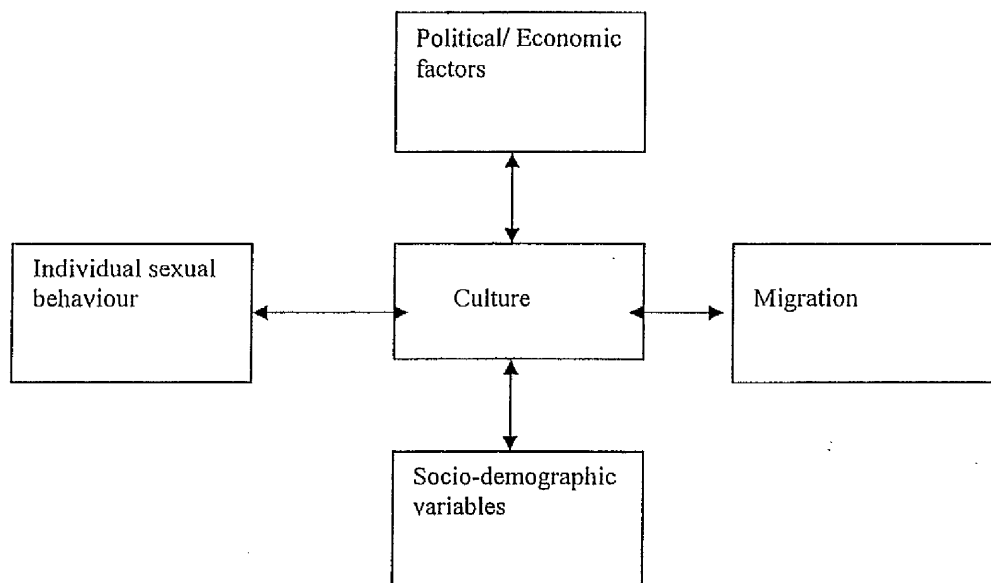


Figure 3.4 Synthesised framework based on the behavioural, political economy and the vulnerability explanations of the HIV/AIDS spread (adapted and modified from Webb, 1997).

3.7 Discussion:

The hypotheses and case studies reviewed show that whereas conflict may increase vulnerability to HIV infection, exposure opportunities may increase or decrease during conflict and post-conflict periods depending on the context in which the conflict occurred. During the war in Angola for example, mobility was limited as most of the population were concentrated in small 'islands of security' around provincial capitals (Mock et al, 2004). This resulted in low HIV seroprevalence as shown in figure 3.1 and table 3.1. In the case of Rwanda, the reverse is the case. Here the sentinel surveys among pregnant women and STI patients showed prevalence rates exceeding 30 percent before the 1994 genocide (WHO, 2003). Here, because

HIV was already so prevalent, community isolation did not curtail the local spread of the disease. In contrast, prevalence rates in Mozambique in sentinel surveys of high risk populations appear to have remained relatively low until the war ended at which point the resumption of normal patterns of social mixing occurred alongside a marked rise in prevalence rates (WHO, 2003). Thus, when initial low prevalence is combined with isolation for a long period of time, HIV progression at the population level may be low. The above analysis shows that even though war and conflict created the risk environment for HIV to spread, the extent of the spread may be context specific and this may account for the spatial variability in HIV prevalence in conflict affected zones in sub-Saharan Africa.

The higher HIV prevalence level in the southern African sub-region on the other hand reflects a complex mix of labour migration and internal conflict due to the oppressive regime and the resultant militarization of the society. Here migrant workers from neighbouring countries with higher HIV prevalence resulted in sexual mixing thereby increasing the risk to HIV infection among the migrant workers, the indigenous population and their communities back home (Chirwa, 1995). The militarization of the society also resulted in sexual violence which may have resulted in increased risk to HIV infection.

In West Africa, labour migrant and civil war may account for the high HIV prevalence in the region. Cote d'Ivoire until recently had enjoyed relative stability in the sub-region and had received migrant workers from Ghana, Burkina Faso, Liberia and other neighbouring countries. One would have expected the HIV prevalence rate to have been low but it is the country with the highest HIV prevalence in West Africa followed by Nigeria. Sexual mixing between migrant workers, commercial sex workers and the local population increase the risk to HIV infection (Anarfi, 1993). In the case of Liberia, the civil war and its resultant displacement of society and the interaction between military personnel and combatants resulted in sexual mixing which increased the risk to HIV infection (UNGA, 2002)

Though the epidemic had not spread as expected in Ghana, the potential for the epidemic to explode is high especially as Ghana shares border with Cote d'Ivoire a country with the highest prevalence in West Africa. Returned migrants from Cote

d'Ivoire in particular and Nigeria to Ghana may have been the agent for the initial spread of the epidemic in Ghana. As observed from the case studies, the main destination countries for Ghanaian migrants were Cote d'Ivoire and Nigeria which happen to be the two countries with the highest HIV prevalence level in West Africa (UNAIDS, 2005). In support of this argument is the observation that about two-thirds of the prostitutes in Cote d'Ivoire were Ghanaians mostly from the Krobo district of Ghana (Anarfi, 1993).

Although Mohammed Ali et al (1990 in Smallman-Raynor and cliff, 2004) observed that major roads were acting as a principal corridors for the spread of HIV in Central Africa at the initial stages of the epidemic, the impact of this hypothesis as an explanatory factor for the current spatial pattern of the HIV epidemic may be minimal. This is because the road infrastructure is not well developed and decades of conflicts in most part of sub-Saharan Africa has destroyed the existing road networks. Although trucking routes may have helped in the epidemic spread in the Great Lake region and Uganda in particular, the truck town hypothesis failed to explain the district to district variability in AIDS incidence in Uganda (Smallman-Raynor and Cliff, 2004).

From the above review, it is evident that different researchers tend to adopt different frameworks in explaining the HIV/AIDS epidemiology probably based on their aims and objectives and their particular interest. However, due to the complex nature of the HIV/AIDS epidemiology in sub-Saharan Africa, one theoretical stand point may not be able to address the broad issues relating to the African epidemic. This may account for the apparent failure of current interventions to mitigate the epidemic spread. The cultural approach for instance focuses on behaviour change as a means of solving the HIV crisis without taking into consideration the economic and political context in which individual behaviour takes place. The political economy approach on the other hand restricts explanatory factors to micro issues where individual behaviour is influenced by global economic and political structures acting on international, national and local levels. A major weakness of the political economy approach is the fact that less emphasis is placed on the environmental factors that leaves individuals and groups at risk which is in the domain of the vulnerability framework.

3.8 Conclusion

The review of the pre-independence African history shows that the continent-wide risk environment may have been created through the impact of slavery and colonisation and may have been exacerbated by post-independent civil strife, conflicts and its resultant population displacements. In order to explain the spatial patterning of the epidemic, three major explanatory hypothesis have been proposed; the military involvement hypothesis; migrant labour hypothesis; truck driver hypothesis. The extent to which any of these approaches explains the spatial epidemiology of HIV in sub-Saharan Africa is context specific. In Central Africa, war and conflict were the main factors creating the risk environment for the epidemic to spread. In Southern Africa, migrant labour was the main factor creating the risk environment for the epidemic to spread even though internal conflict and its resultant militarization of the society compounded the already dire situation by increasing the risk to HIV infection. In the case of West Africa, migrant labour was the main factor creating the risk environment for the epidemic to spread however, the recent civil wars in the sub-region may have aggravated the situation by increasing the risk level.

Whereas countries with relative stability in the Southern African sub-region, such as Botswana, are among the countries with the highest HIV prevalence, in West Africa, Ghana which has enjoyed relative stability for the past three decades and borders Cote d'Ivoire, the country with the highest HIV prevalence level in West Africa, is among the countries with the lowest HIV prevalence rate in West Africa. The location of Ghana and the fact that the bulk of Ghanaian migrants live in Cote d'Ivoire and Nigeria, the two most highly infected countries in West Africa makes Ghana a candidate for an explosion in HIV prevalence. Ghana is therefore chosen as a case study at the sub-national level to explore the processes of the epidemic spread using the Krobo district from where two-thirds of commercial sex workers in Cote d'Ivoire come (Anarfi, 1993).

In this chapter we have also explored the theoretical issues relating to the spread of HIV namely the behavioural, political economy and vulnerability approach. It was observed that due to the complex nature of the HIV/AIDS epidemiology especially in

sub-Saharan Africa, individual approaches do not adequately address the reasons for the continual increase in the epidemic spread despite the numerous interventions to mitigate its spread. A synthesised framework based on the three approaches was adopted for an in-depth understanding of the epidemic in the Krobo district in Ghana.

CHAPTER FOUR

4.0 HIV/AIDS IN GHANA WITHIN SUB-SAHARAN AFRICA CONTEXT

4.1 Background

In order to understand the epidemiology of HIV/AIDS in sub-Saharan Africa within the national context of Ghana, there is the need to understand the epidemiological situation in Ghana as well as the background of the epidemic in the Krobo district in Ghana, which is chosen as a case study. The first part of this chapter examines the epidemiological situation in Ghana. The second part analyses the epidemic situation in the Krobo district and the extent to which the theoretical frameworks as discussed in the conceptual chapter applies to the Ghanaian situation.

The first official AIDS cases in Ghana were reported in 1983 and were associated with women who had travelled overseas (Anarfi, 1993). Although overall levels of infection have remained relatively low, the disease is firmly established within the general population and shows no signs of stabilising. Several factors place Ghana at risk for further spread of the disease such as lack of information on the most vulnerable populations, inaccurate perception of personal risk, marriage practices and gender relations such as polygamy, early marriage, women's subservience to men, widespread poverty, stigma and discrimination towards those living with HIV/AIDS (UNAIDS, 2005). Of particular importance is the geographical position of Ghana which shares a common border with Cote d'Ivoire which is the most infected country in West Africa and has historical and economic link with Nigeria the second most infected region in West Africa.

In Ghana just like other parts of sub-Saharan Africa, population migration, urbanisation, economic, socio-cultural, gender and human rights issues created the enabling environment for HIV/AIDS to thrive. One feature that put Ghana at a higher risk to HIV infection is the high mobile population. As noted in the preceding chapter, migrant labour to Cote d'Ivoire and Nigeria from Ghana increases the risk of infection transfers from these highly infected countries to Ghana (Anarfi, 1989) especially that of women migrants to Abidjan as sex workers. Within Ghana, rural-urban migration

particularly by the youth in search for non-existing jobs end up stranded in cities and thus further expose them to the risk of transactional sex. Street children are another risk group that are at increasing risk to HIV infection through transactional sex. There is also rural-rural migration to market places. In most of these areas sleeping conditions are deplorable and casual sex is rife. Long distance drivers, uniformed service personnel and itinerant traders are particularly exposed to the risk of casual sex and hence to HIV/AIDS. These features of the Ghanaian population make them more vulnerable to HIV infection.

The age structure of the population also shows that 42 percent of the total population are less than 15 years old (Ghana Statistical Service, 1998). Such a youthful population has got a very great potential for the epidemic spread if left unchecked. The economically active and reproductive age-group (15-49) is the worst hit by the HIV infection. Even though children under one year old constitute 4 percent of the total population, they account for 15 percent of HIV/AIDS cases in Ghana (Ministry of Health, 2003). This means that mother to child transmission constitute a second major mode of the epidemic apart from heterosexual transmission in Ghana. This situation poses an enormous threat to the gains made in reducing infant mortality rate. To date, there are no policies and strategies aimed at reducing mother to child transmission. Children aged 5-14 therefore present a 'Window of Hope' for managing the HIV/AIDS crisis because they have generally not begun their sexual lives. Increased attention to this age group is critical for the future response to the epidemic. These characteristics of the Ghanaian epidemic show that there is a strong potential for the epidemic to explode if left unchecked hence the need to explore the processes by which the HIV epidemic is spreading in Ghana.

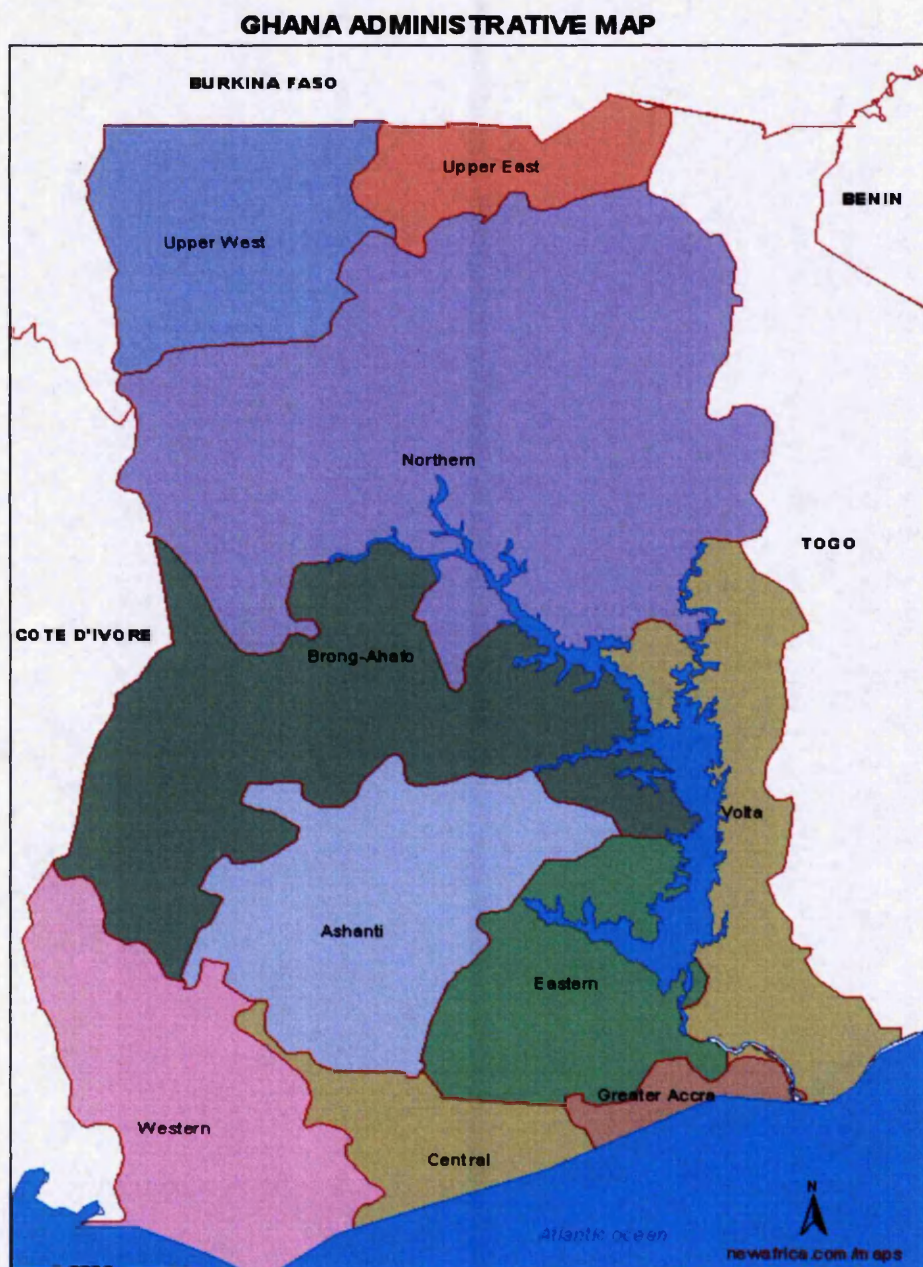


Figure 4.1 Administrative map of Ghana

4.2 Epidemiological Situation

According to Ghana National AIDS Control Programme, approximately 400,000 adults were living with HIV/AIDS as at the end of 2003 (see Table 4.1). Surveillance data from antenatal sites put the 2002 HIV prevalence at 3.4 percent, which is more or less constant since standardised estimates began in 1992.

Table 4.1 Estimated National HIV Prevalence

	Best estimates2002-2003	2003Low-High Range
Estimated Adult(15-49 Prevalence	3.4-3.6	2.8-4.2
Number of Infected Adults(15-49	336,000-352,000	282,000-423,000
Number of Infected Children	23,500-26,000	21,000-31,000
Number of AIDS Deaths	26,000-29,000	23,500-35,300

Source: National AIDS/STD Control Programme, 'Estimating National HIV Prevalence' Technical Report, 11/03.

There is spatial variation in the prevalence of HIV across the regions of Ghana. In 2002, it ranged from a low of 1.7 percent in the northern region to a high of 6.5 percent in the eastern region (see. fig. 4.1). Prevalence rates are higher in densely populated areas particularly in regional capitals such as Kumasi, Koforidua and Accra. HIV prevalence is also higher in mining towns such as Obuasi and Tarkwa as well as border towns and along main transport routes (UNAIDS 2003).

The epidemic is primarily spread through heterosexual transmission, which accounts for about 80 percent of infections (NACP, 2005). Mother-to-child transmission accounts for 15 percent of infection. Two-thirds of reported AIDS cases since the beginning of the epidemic have occurred in women though such a trend is levelling off. Transmission through other routes including blood transmission and contaminated needles is considered to be low although few data are available. Prevalence rate is highest in identified vulnerable populations such as commercial sex workers. In 1999, prevalence among commercial sex workers was 77 percent in Accra and 82 percent in Kumasi (NACP, 2005). Other high risk population groups are truck drivers among which only 56 percent used condom during the last higher risk sex. Two-thirds of policemen had had sex with non regular partners during the previous year but condom use is thought to be high (98 percent during commercial sex, and 83 percent during

higher risk sex. Miners are another group who are at greater risk as condom use remains low amongst them during commercial sex (UNAIDS, 2003).

The distribution of HIV/AIDS in Ghana is higher in densely populated areas. Higher numbers of case occur in the southern regions of the country particularly in densely populated regional capitals such as Kumasi, Koforidua and Accra (see figure 4.1). Prevalence of HIV/AIDS is also very high in mining towns like Obuasi and Tarkwa as well as in border towns (NACP, 2005). The implications are that in high prevalence areas, a two-pronged approach is required. The first is to intensify action towards the reduction of transmission within these towns and the second is to provide care and support for people living with HIV/AIDS (PLWHA) as well as support for people affected by HIV/AIDS like orphans. As rural areas have been less affected by the HIV/AIDS epidemic, primordial preventive efforts in these areas need to be enhanced.

Even though poverty levels have been estimated to drop from 36 percent in 1987/88 to 29.4 percent in 1998/99, there are regional as well as rural-urban variations (Ghana Statistical Service et al 2004). This wealth differential causes people to migrate from the rural areas to the urban centre and hence create sexual mixing which increases the risk to HIV infection. Poverty and other economic pressures on individuals in the country constitute major factor in the spread of HIV/AIDS. For example high youth unemployment, limited job opportunities and the rising cost of living are aspect of the poverty cycle that promotes transactional sex and early sexual relations (Ghana Demographic and Health Survey, 2003). As evident in other countries, the spread of HIV/AIDS also impacts on both the supply and demand aspect of education. The gains made in the education sector such as increased enrolment to above 70 percent will become difficult to sustain because increasing number of HIV/AIDS cases among teachers will reduce their availability to the educational sector. Secondly there will be declining enrolment either through rising infection rates among the youth or as more families begin to require children to care for the sick family members (UNAIDS, 2003).

Ghana has a diverse ethnic and cultural composition associated with different cultural practices and political orientations. A common feature of traditional life is the strong

communal and family support system such as the extended family system. However in recent years with urbanisation and the consequent rural-urban migration the extended family system and its values are being eroded with its attendant sexual mixing which increases the risk to HIV infection. Another price being paid by the erosion of the traditional family structure is the inadequate social and family support for PLWHA and their carers. Other socio-cultural factors such as stigma and denial make the care and support for PLWHA and their carers a daunting challenge (Ministry of Health, 2001) Polygamy, sexual attitude and belief systems, which underline gender inequalities, makes it difficult for women to negotiate issues about sex, reproduction and condom use. Prevailing belief systems are also important in reporting and management of STDs. Ghanaians are generally religious. The principal religions are traditional, Christianity and Islam. However religious bodies have not been adequately used to mobilise and respond appropriately to the HIV/AIDS epidemic and to help reduce the stigma and denial associated with HIV infection.

Anecdotal evidence shows that the economic cost of HIV/AIDS to employers though not determined in terms of care, absenteeism and re-training is high and continue to rise. HIV/AIDS is expected to put severe stress on families, the health sector and other sectors of the economy. Recent estimates indicate that the annual cost of treatment of opportunistic infections in an AIDS patient can be as high as 4.2 million Cedis per person (Nabila, Antwi, et al 2001). The cost of providing health care for AIDS patients alone far exceeds other health care cost thus affecting the provision of primary health care to the general population.

Finally, the rising number of AIDS orphans will put enormous pressure on households, communities and the nation as a whole. This is worse in rural areas where about 66 percent of Ghana's population lives and where levels of poverty are high because the majority are employed mainly in primary agricultural production (Akwara, and Gabriel et al, 2005).

4.4 Review of the national response to HIV/AIDS in Ghana

HIV/AIDS in Ghana was first managed as a disease rather than a developmental issue. The national response has consequently been medically oriented and directed by the

Ministry of Health (MOH). The earliest national response was the establishment of the National Advisory Commission on AIDS (NACA) in 1985 to advise government on HIV/AIDS issues. In 1987 the National AIDS Control Programme (NACP) was established as an arrangement within the Ministry of Health for both implementation and co-ordination of HIV/AIDS programmes. Later it became obvious that the complex multi-facet and multi dimensional nature of the epidemic require a developmental-holistic-co-ordinated and multi-sectoral approach. This realisation led to the establishment of the Ghana AIDS Commission (GAC) as a multi-sectoral body for policy formulation, co-ordination, supervisory and resource mobilisation. Aside the NACP and the GAC, there are several Non-Governmental Organisations (NGOs) as well as public sector organisations involved in HIV/AIDS activities.

Key interventions underpinning HIV/AIDS control in Ghana have been safer sex promotion, preventive clinical interventions, Care for People Living with HIV/AIDS (PLWHA), and Legal and ethical issues (Ministry of Health, 2001). Efforts at promoting safer sex have centred on three main messages-abstinence, mutual faithfulness to one sexual partner and condom use. Though efforts have been intensified, through both print and electronic media, perception of personal risk remains low. Peer education, school based programmes and workplace activities have targeted specific groups. The use of social marketing strategies has improved access and utilisation of condoms.

Within the clinical setting, the response has been focused on broad activities that reduce HIV transmission such as management of Sexually Transmitted Diseases (STDs) and ensuring safe blood transfusion. Overall management of STDs in the health sector both private and public orthodox and traditional providers remains weak and linkages among the providers need to be strengthened (NACP, 2004). Substantial progress has been made to improve the capacity of the health sector to manage STDs. Programmes targeting commercial sex workers have also been initiated and are being implemented.

In the provision of safe blood transfusion, blood is now screened for HIV and Hepatitis B prior to transfusion. There are however occasional shortages of testing kits. A number of Traditional Birth Attendants (TBAs), *Wanzams* (traditional

circumcisers), traditional healers, barbers and beauticians have been given training in infection control.

The provision of an effective and integrated continuum of care for PLWHA in health institutions and at home has not received adequate attention. Health workers still lack the confidence to care for PLWHA for fear of being infected. Similarly, communities and households are yet to come to grips with the fact that they need to handle PLWHA and to give them the attention and care they require as any other patient without any feeling of stigma on the infection. Home-based care remains the least developed component of the continuum of care and requires special attention. A limited number of private hospitals are providing care and support to PLWHA through outreach programmes. Religious, traditional, political as well as community and district system will need to be mobilised to support and sustain home-based care.

Although the government of Ghana has put some measures in place to mitigate the spread of the HIV virus, the epidemic continue to increase especially in the eastern region of Ghana of which the Krobo district is the most affected (National AIDS/SDTs Control Programme, 2005). The national response shows that individual behaviour remains the main focus of intervention. The macro economic issues that make individuals and communities vulnerable to HIV infection are not targeted. The synthesises framework discussed in the theoretical chapter which encompasses the economic and political environment in which individuals operate and the risk factors in the Krobo region of Ghana is explored to understand the processes for the epidemic spread in the Krobo district of Ghana using a population based survey and an in-depth interview

4.5 The Krobo District within the Ghanaian context.

The Krobo District in Ghana is synonymous to HIV/AIDS. The district is highest hit with the HIV/AIDS epidemic with a prevalence rate of 7.0 percent, which is over and above the national rate of 3.6 percent (NACP, 2002). The district has also experience the migration of women to the neighbouring Ivory Coast, which has one of the highest prevalence rates in the West Africa as sex workers. Dzokoto et al (2002) observed

that the high prevalence documented in this part of Ghana seems to be to some extent a consequence of the construction of the Akosombo dam in the 1960s. The flooding of the land, the failure of the resettlement schemes and the ensuing poverty prompted economically driven migration, especially to Cote d'Ivoire where many migrants became infected with HIV. Local transmission then followed. Anarfi (1989) observed that about three-quarters of the prostitutes in Cote d'Ivoire were Ghanaians most of whom returned to Ghana to die of AIDS. It is possible that the sex workers were already infected with the virus in Ghana before travelling to Cote d'Ivoire. Anecdotal studies show that symptoms of HIV/AIDS were prevalent especially among the Krobo tribe in Ghana who coincidentally forms the bulk of the sex workers in Cote d'Ivoire even long before they started migrating to Cote d'Ivoire. This implies that the epidemic might have started in Ghana, diffused to Cote d'Ivoire and spread out to other countries including Ghana at the peak of the epidemic.

4.6 Demographic and Socio-Economic Characteristics of the Krobo District.

The Krobo Region is made up of Manya Krobo district and Yilo Krobo district. The two districts are located at the eastern part of the Eastern Region of Ghana along the south-western part of the Volta River (ref figure 4.1). The district covers an area of 2281 square kilometres

The 2000 population census indicates a population size of 240408 for the entire Krobo region. This is made up of 154301 for Manya Krobo with 76070 male and 78321 females and 86107 for Yilo Krobo with 4180 males and 44277 females. The sexually active population (15-49 years) is a significant 46 percent. The population growth rate is 1 percent for the district as compared with the regional rate of 1.4 percent (Ghana Statistical Service, 2000). Somanya and Odumasi-Krobo are the district capitals for Yilo Krobo and Manya Krobo respectively.

The average household size in the district is 7.5. This figure is higher than both the regional and national average of 4.6 and 5.1 respectively. The large household size is a reflection of the social structure of the society. Thus despite modernisation and subsequent erosion of the extended family household composition, the household in

the Krobo district still maintains its traditional nature. Furthermore women head about 40 percent of households in the urban areas of the district.

The main occupation of the people in the district is farming and the average per capita income is estimated at US\$112 per year. Thus the average Krobo earns less than one dollar a day (Manya Krobo District Development Plan, 2004). The district is 60 percent rural but about 40 percent of the population lives in the district capitals. The urban population in the district increased from 11 percent in 1970 to 23 percent in 1984 and 40 percent in 2000. The continuous decrease in the rural population over the years could be attributed to low yield from farming activities due to poor soil fertility. As a result people migrate to the district capital to seek employment, learn trade or engage in petty trading. The result of rural depopulation has resulted in food shortage since the bulk of the food is produced in the rural areas. Increased urbanisation is therefore a factor that needs to be considered in explaining the fast changing HIV-related socialisation process particularly in the district capital.

The landscape of the district is generally undulating with several streams most of which drain to the Volta lake. The relatively hilly nature of the upper part of the district coupled with the intensive farming using traditional practices has led to severe erosion and deforestation. The lower part of the district, which is relatively flat, suffers from flooding during the raining season.

There is a large out of school population, a factor endorsed by stakeholders as facilitating the rapid spread of HIV. About 34 percent of children of school going age are out of school. At the secondary school level about 47 percent are out of school the majority of who is the girl child (Manya Krobo District Assembly, 2004). Among those who are enrolled at school a large number of them do not attend school regularly especially in the farming season when they contribute to family labour. Also walking distance to school is so high in the rural areas, a major limitation in improving access.

4.7 The Krobo Culture

The Krobos have a culture that has survived to date. However certain cultural practices and customary practices require adaptation with the advent of HIV/AIDS. Notably are '*dipo*', '*Lapomi*', and '*kaduba flame*'. The spread of HIV/AIDS in the Krobo District may be related to certain cultural practices (Teyegaga, 1984). One of these is '*dipo*' the initiation rite, which used to be practiced all over Ghana for girls aged between 14 and 20 years. A hundred years ago the rite lasted over a year and included tuition in housework, cooking and parenting. At the end of the year the girls emerged skilled and well fed ready to be courted by the young men and married. They underwent a series of test to prove that they had learned well and receive small scarification marks on their wrists and at the back of the waist. These signified to a man that the girl had finished *dipo* and could be courted.

In 1892, the British colonial administration banned *dipo* associating it with sorcery and witchcraft. The missionaries did not also accept *dipo* although attempts were made to integrate the rite into the church, the church did not accept the 'pagan' rituals and slaughter of goats. Once the practice was banned it went underground. Now many mothers perform the rite to their daughters very young so they can take the child to church (Teyegaga, 1984).

By the 1950s *dipo* lasted only a fortnight and involved girls as young as eight years old. The Krobo people are the only tribe in Ghana who still keep this tradition which now last for only five days and involves girls as young as five years (Teyegaga, 1984). Each year members of the Krobo people from all over Ghana and abroad bring their daughters to the Krobo land in April and May to undertake this ritual. There is a strong cultural feeling and belief that a girl who has not undergone the *dipo* ritual is not a Krobo and is not ritually clean. Even very poor parents will borrow money to pay for the cost of the ritual and have their daughter's rite performed.

In the past, girls who had not undergone the *dipo* custom and became pregnant were banished from the tribe and the town in which they live (Teyegaga, 1984). Even though people are no longer banished from towns girls who do not undergo the *dipo* custom at present times are neglected by their families and are not accepted by their

own nuclear as well as extended families and have no right to inherit family property and in some case cannot enter their family homes (Teyegaga 1984). This threat helped to protect girls from early pregnancy in the past but nowadays with girls performing *dipo* even before puberty, early pregnancy is no longer frowned upon. Once a girl has undertaken *dipo*, she may begin sexual activity. Puberty rites are therefore undertaken by girls long before they reach puberty and there is greater sexual permissiveness which has implication for the spread of HIV/AIDS but until recently the Krobos had not associated AIDS with sexual activity. The Krobos recognised 'korni doorlee' 'lean' as a new disease but they related it to evil spirits punishing them for wealth gained abroad rather than sexual activity (Yilo Krobo District Assembly, 2003).

Another aspect of *dipo* custom that may relate to HIV/AIDS is the scarification process and the shaving of heads of girls. In order to identify girls who have undergone the *dipo* rites from those who have not, the hair of the girls are shaved and scarification marks are made on their wrists and at the back of their waist using one knife during the ceremony. The scars signify to a man that the girl had finished *dipo* and could be courted. This practice is not in line with HIV/AIDS prevention methods.

Another aspect of the Krobo culture, which may increase the risk of HIV/AIDS is '*kaduba fiame*'. As part of their treatment and healing protocols, traditionalist, herbalist and spiritualist use scarification '*Kaduba Fiame*' which has been practiced for centuries however the use of unsafe knives for such operations increases the risk of transmission of HIV. Circumcision at home by '*Wanzams*' local tribes men who carry out circumcision faces similar challenge.

Also the bride price in the Krobo land is relatively expensive and due to that most young men are not able to marry. The result of this is many single mothers who have had children outside traditional marriage. The rule is that unless the bride price is paid for the woman's family, the father cannot claim the child as his. *Lapomi* therefore involves a father performing such rites. However most men are not able to perform the rites and as such the women become single parents and in most situation end up in the sex industry or have multiple sexual relations which predisposes them to HIV infection (Teyegaga, 1984). Another challenge is the unsafe handling of dead bodies.

The tradition is to lay dead bodies in state and pay their last respect without considering the risk of infection.

4.8 The HIV/AIDS situation in The Krobo District

The Krobo district is the first district in Ghana where the first cases of HIV/AIDS in Ghana were reported. The district is also the first to see the emergence of HIV/AIDS organisations both local, non-governmental and international organisations. The development of such AIDS services organisations therefore reflects the localised epidemiology of HIV infection in the Krobo district. In 1988, the district medical doctor in charge of the Krobo district Dr Margaret Mensah established an AIDS Clinic at Agormanya hospital as well as introduced a home-base care for HIV/AIDS patients due to pressure on existing facilities at the Agormanya hospital. The presence of the AIDS clinic as well as the home-based care attracted media attention and this resulted in NGO,s and International organisations using the district for research on HIV/AIDS in Ghana. As at February 2004 when the fieldwork was conducted, there were about 32 Non Governmental Organisations (NGO,s) and 21 Community Based Organisation (CBO,s) working on HIV/AIDS and related issues in the Krobo district (Manya Krobo District Administration, 2004).

Sentinel surveillance data in the district over the year's revealed HIV prevalence rates above that of the entire country. HIV prevalence rates in the district ranged from 18 percent in 1982 to 6.6 percent in 2001 and 7.0 percent in 2002. The national prevalence rate was 3.6 percent in 2002 (NACP, 2002).

All age groups and gender in the Krobo districts have been affected or infected by HIV/AIDS. The 15-24 year groups are the most affected by the disease. Data from St Martin's Hospital at Agormanya shows HIV prevalence of 11.4 percent for women aged 15-24 receiving antenatal care (NACP, 2002). The disturbing situation represents a major threat for the youth who constitute more than half of the total population of the district. A cluster of potential risk groups includes commercial drivers, traders, barbers, farmers, hairdressers, artisans and apprentices and commercial sex workers.

The extent to which the unique characteristics of the Krobo people described in this section impinge upon the spread of HIV is yet to be unravelled. A survey of the general population and the HIV/AIDS prevention and treatment organisations in the district is undertaken to examine factors affecting the spread of the epidemic in the district. Result of the survey will shed light on the outcome of the modelling process.

4.9 Overview summary of the three preceding chapters

Chapter two shows that although there have been many studies and debates about the source and origin of the HIV virus the exact source of the virus and the possible origin and date of inception of the virus continue to elude them. Secondly factors determining the spatial spread of the epidemic are not known and worse still there is weak surveillance system in sub-Saharan Africa. Given these uncertainties and the fact that the epidemic continue to spread, chapter three examined the common research approaches to the spread of HIV/AIDS. It was observed that each of the approaches fails to fully explain the spatial epidemiology of the virus hence the need for synthesised framework which looks beyond individual human behaviour to include socio-economic, political and environmental risk factors under pinning the epidemic spread. In order to understand the processes of the epidemic spread on sub-national level, chapter four presented the demographic and socioeconomic characteristics and the epidemiological situation of Ghana and the Krobo district. It was observed from the review that the geographical location of Ghana and the socio-economic and demographic characteristics of Ghana in general and the Krobo district in particular makes it vulnerable to HIV spread but the exact processes for the epidemic spread is not known.

Chapter five will model the epidemic to enable us identify the source region and predict dates for the inception of the HIV virus in sub-Saharan Africa. By modelling the epidemic, we will be able to predict the dates in each country in sub-Saharan Africa when the epidemic will become endemic in a scenario of no intervention. The last section of the modelling chapter will help us identify the transmission routes at the start and at the peak of the epidemic. The modelling exercise which is the core of the thesis will help us to trace the epidemic from its source and to understand the

routes through which it spread and the possible processes underlying its spread in order to inform public health policy.

It was observed from the review of the national response to AIDS in Ghana that prevention policies are still based on change in human behaviour. In order to understand spatial patterning depicted in the modelling process and the exact processes of HIV spread, and the reasons for the apparent failure of current interventions, population based survey of the general population and an in-depth interview of the HIV/AIDS prevention and treatment organisations in the Krobo district in Ghana will be carried out.

CHAPTER FIVE

5.0 EPIDEMIC MODELLING SYSTEMS

5.1 Introduction

In order to understand the magnitude and directionality of the HIV/AIDS epidemiology in a continent where data on the epidemic are limited, modelling the epidemic can provide the needed estimates for planning intervention and disease control. The first part of this chapter reviews the evolution of epidemic modelling and provides the theoretical background that underpins the modelling process used in this chapter. The next section describes the single region community models and multiregion model and its application to this study. This is followed by the application of the multiregion model developed by Thomas (1992; 2001), Smith and Thomas (2001) and Smith (2003) to HIV/AIDS incidence data on sub-Saharan Africa. This task entails fitting single region modelling system to HIV/AIDS incidence data. This allows estimates of transmission probability, start dates of the epidemic and the risk population to be estimated. Results from the single region model are then imputed into the multiregion model to determine the transmission pathways at the start of the epidemic and peak prevalence pathways within sub-Saharan Africa.

5.2 Background

The application of mathematics to the study of infectious diseases appears to have been initiated by Bernoulli (1760) after which there was a long gap until the middle of the nineteenth century when in 1840 William Farr fitted a normal curve to smoothed quarterly data on deaths from smallpox in England and Wales over the period 1837-1839 (Bernoulli 1760, Farr 1840 in Thomas 1992). John Brownlee further developed this descriptive approach by fitting pearsonian frequency distribution curves to a large series of epidemics.

The descriptive approach adopted by Farr and Brownlee was in contrast with the quantitative approach adopted by Hamer and Ross (1906, 1909, in Thomas 1992). These were the first to formulate specific theories about the transmission of infectious disease in simple but precise mathematical statements and to investigate the properties

of the resulting models. The work of Hamer and Ross in conjunction with that of Ross, Hudson and Soper (1917), and (1929) in Thomas (1992) provided the firm theoretical framework for the investigation of observed patterns of epidemics.

In 1906, Hamer postulated that the course of an epidemic depends on the rate of contact between susceptible and infectious individuals. This notion has become one of the most important concepts in mathematical epidemiology often referred to as 'the mass action principle' in which the net rate of spread of infection is assumed to be proportional to the product of the density of susceptible people times the density of infectious individuals. However, in 1908, Ross translated the problem into a continuous-time framework in his pioneering work on the dynamics of malaria. Soper (1929) who deduced the underlying mechanisms responsible for the periodicity of epidemics as well as establishing the celebrated threshold theory improved upon the ideas of Hamer (1906) and Ross (1908). From the early stages, it became clear that variation and the elements of chance were important determinants of the spread and persistence of infection and this led to the development of stochastic theories.

Much recent work on epidemic modelling has focused on the application of control theory to epidemic models (Wickwire, 1977 in Thomas 1992). The work of Cliff et al (1986) was on the study of the spatial spread of disease while that of Hethcote et al, (1981); Aron and Schwartz (1984) was on the investigation of the mechanisms underlying recurrent epidemic behaviour. Anderson et al (1991) extended the threshold theory to encompass more complex deterministic and stochastic models. Following the pioneering work of Bailey (1975 in Thomas 1992) on developing general stochastic models for infectious disease, more specific simulation models have been proposed for toxoplasmosis (Papoz et al, 1986,) measles (Riley, Murphy and Riley, 1978; Levy, 1984) influenza (Longini et al, 1984), poliovirus, enteric virus and vaccination strategies (Eckerman, Elveback and Fox, 1984). This style of research has been termed 'structural' modelling (Bailey et al, 1974) because the models try to capture the underlying disease mechanism.

Epidemiologists on the other hand, have tended to apply structural models to mimic the temporal incidence of disease in closed communities and with some notable

exception (Longini et al, 1985), spatial variations in incidence have been studied through the use of statistical methods (Schenzle et al, 1979).

In recent years there has been a revival of academic interest in the development of modelling systems for the diffusion of infectious disease (Thomas, 1988a). Variety of models has also been developed to mimic the dynamics of sexually transmitted infections and some of these have been designed to explore the efficiencies of various control options (Cook and York, 1973; York, Hethcote and Nold, 1978).

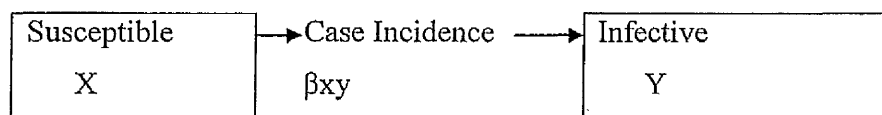
Modelling a sexually transmitted disease which is a subject of this chapter is much complex because many socio-economic factors are thought to be responsible for the recent rise in the global prevalence of sexually transmitted diseases. These include increasing urbanisation, migrant labour, expanding tourist travel and new attitude towards sexual behaviour in modern societies. For example Gonorrhoea has distinct epidemiological characteristics when compared with bacterial infection. In the first place, the disease occurs only within the sexually active portion of a community. Secondly the duration of infection is long and differs substantially between male and female individuals. Lastly, acquired immunity to reinfection is virtually non-existent and hence recovered individuals pass back to the susceptible pool.

Modelling the HIV/AIDS epidemic is much complex because AIDS, unlike gonorrhoea, is not simply a heterosexually transmitted disease but passes between homosexuals, between bisexuals and heterosexuals, between heterosexuals, between mother and foetus and between drug users who share syringes and needles. There are many different kinds of pair contact and not simply the male-female contacts which are usually considered in the common sexually transmitted diseases. The transmission risks are also asymmetric being greater as a rule for one member of the pair if the other is infectious than the other way round. These complications take the calculations beyond the domain of simple algebra and require the construction of a computer programme (Knox 1986).

5.3 Community Models

The first attempts to model the spread of disease through a population were based on infectious diseases which occurred in regular cyclical incidence in a host population. The simplest form of the recurrent epidemic model concentrates solely on the transfer of the agent from susceptible to infective and is attributed to Bailey (1975) but is based on the idea of Hamer (1906) and Soper(1929). The model is based on the assumption of homogeneous mixing meaning that each individual in a community meets every other individual in a given unit of time. However no account is taken of the distances separating the individuals. For this reason the representation is said to be space less and the model is only applicable to a closely-knit population. The model is constructed to project the values of the susceptible and infective populations together with their characteristic incidence through time.

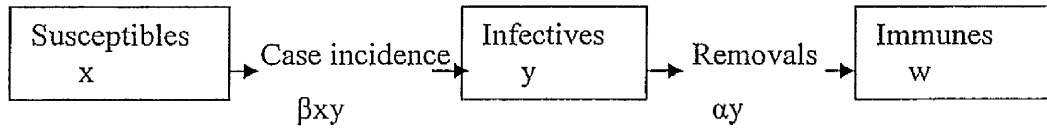
The simple model is formed by two state variables susceptible and infective, which are linked by a single transactional relationship, disease incidence as illustrated below.



The state diagram asserts that case incidence is always transferred from the Susceptibles to the infectives. The symbol β represents the infectivity rate and that if the number of contacts xy is multiplied by the infectivity rate (βxy), the result is the expected number of new cases of disease or the predicted disease incidence (Thomas, 1992).

A further development on the recurrent model is the general epidemic model. The general epidemic model recognises that infectives can be removed from circulating population through death, isolation or the termination of communicability. The general community model therefore introduces a removal rate (α) which will remove αy individuals from the infective population and transfer them to the immune population(w). α takes on the value from 0 to 1 and when $\alpha=0$, no infectives are

removed and when $\alpha=1$, all infectives are removed after dt , thus indicating their infectiousness is lost in a minuscule amount of time (Smith, 2003). This means that the rate of change in the infective population is now dependent on both the input of new cases and the output of removals. This gives a state diagram, with mathematical notation:



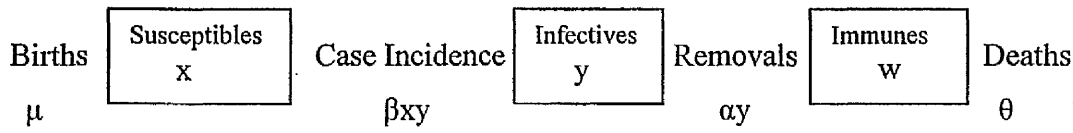
The state diagram defines the differential coefficients for this system as

$$\frac{dx}{dt} = -\beta xy \quad (1)$$

$$\frac{dy}{dt} = \beta xy - \alpha y \quad (2)$$

$$\frac{dw}{dt} = \alpha y \quad (3)$$

The single and general community models as described above failed to predict the continuous transmission often observed when studying infectious diseases. Hamer (1906) and Soper (1929) therefore introduced births at the rate μ into the susceptible population and death rate (θ) to the immune section. This means that given a suitable time span, births will be equal to deaths thus keeping the population constant. The introduction of these two demographic rates results in the following state diagram as well as a differential coefficients.

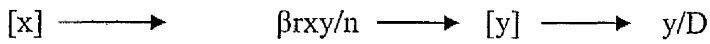


$$\frac{dx}{dt} = \mu - \beta xy \quad (4)$$

$$\frac{dy}{dt} = \beta xy - \alpha y \quad (5)$$

$$\frac{dw}{dt} = \alpha y - \theta \quad (6)$$

The models described above have been adapted to model HIV/AIDS epidemic by Anderson et al (1988) and Isham (1988) in a single community of gay men as outlined in Thomas (1999a). If this community numbers n individuals and y represent those who are infectious, at any point in time t , the transmission of HIV is represented by the following state diagram



Here the expression $\beta rxy/n$ measures the incidence of HIV. Within this term, r is the number of sexual partners acquired per unit of time. Then, y/n is the probability a new partner is infectious and rx/n is the total number of partnerships between Susceptibles and infectives. The removal of infectives from active circulation depends the average duration of the period of communicability D such that y/D counts the number removed in each interval of time. (Thomas, 1999a).

The state diagram defines the differential coefficients for this system as

$$Dx/dt = - \beta rxy/n \quad (7)$$

$$Dy/dt = \beta rxy/n - y/D \quad (8)$$

Setting equation (8) to zero and rearranging provides the following equilibrium for the susceptible population

$$X^0 = n(\beta r D)^{-1} \quad (9)$$

The reproduction rate for this system may be derived as an approximation to the simulated start of an epidemic when a single infection occurs in a wholly susceptible population. In this case, the values of the epidemic variables will be given by $X=n-1$ and $y=1$, this cause the transactions through the infectious population to simplify to βr and $1/D$. Therefore if $1/D \geq \beta r$, an epidemic cannot begin because the index case will be removed from circulation before being replaced by another. This theory leads to the formula for the reproduction rate (R_0) of HIV infection being given by

$$R_0 = \beta r D \quad (10)$$

Here, R^0 is seen to be the average number of Susceptibles infected by the index case during the period of communicability. Consequently, R^0 must be greater than unity for an epidemic to begin. Substituting $R^0 = 1$ in equation (9) shows that

$$n^* = x^0 \quad (11)$$

is the critical value(*) population threshold above which an epidemic may begin (Thomas, 1999a).

A further development of the modelling systems is the introduction of the principles of finite differencing which is used to unravel the most complex model structures using computer simulation (Thomas and Hugget, 1980)

For example if the simple model is considered, the application of finite differencing method will result in the differential coefficient of the simple model re-expressed as a difference coefficient of the form

$$\frac{\Delta x}{\Delta t} = -\beta xy \quad (12)$$

λd_{ij})(Thomas,1988b). Thus the number of contacts decreases with the distance between region centroids (d_{ij}) according to a negative exponential distribution with a constant parameter, λ . The multiregion state equations that are equivalent to those for the community recurrent model may now be written as

$$\frac{dx_i}{dt} = \mu n_i - \beta_i x_i \sum_{j=1}^m y_j \exp(-\lambda d_{ij}) - \theta x_i, \forall i$$

$$\frac{dy_i}{dt} = \beta_i x_i \sum_{j=1}^m y_j \exp(-\lambda d_{ij}) - \alpha y_i - \theta y_i, \forall i$$

5.5 Multiregion Model for HIV/AIDS

The adaptation of the multiregion epidemic model designed to mimic the transmission of HIV/AIDS and predict the incidence of infection among those at risk is attributed to Anderson (1988) and Isham (1988) who described a model that is applicable to a single community of gay men. This multiregion model illustrated above employs the principle of spatial interaction theory to represent regional contacts rates and follows a long lineage of multiregion epidemic systems (Baroyan and Rvachev, 1967; Murray and Cliff, 1977 ; Longini et al, 1986; Toubiana and Vibert, 1998).

Such models have been extended by Thomas (1994) to provide a framework for modelling the HIV/AIDS pandemic. This model has been refined by Thomas and Smith (2000) by introducing the principle of bilateral travel that ensures that the predicted contacts between regions are symmetrical in each direction of travel. This revised specification has also been adapted to include disaggregating of the population by both risk behaviour and region in Thomas (1999a and 1999b). This improved version enables infection transfer to be calibrated by making the distinction between local and international journeys (Smith and Thomas, 2001).

$$\frac{\Delta y}{\Delta t} = \beta xy \quad (13)$$

As Δt is a measurable interval, equation (12) and (13) can be rearranged to give the following expressions

$$\Delta x = (-\beta xy) \Delta t \quad (14)$$

$$\Delta y = (\beta xy) \Delta t \quad (15)$$

Which calculate the changes in the sizes of the susceptible, Δx , and infective, Δy , populations Δy (Thomas, 1992).

5.4 Multiregion Models

The epidemic models described above assess the spread of infectious disease through a single community where there is homogeneous mixing among members of the community and each member has equal chance of meeting any other individual. However such simplified models are not applicable when analysing epidemics across communities. If the geographical area of interest is extensive, then it is appropriate that the mixing of the population should be heterogeneous with contact frequencies declining with distance separating individuals. Thomas (1988b) observed that the recurrent model can be extended to simulate the spread of an infectious disease through a system of m regions by introducing the idea borrowed from spatial interaction theory to estimate the flow of disease contact between pairs of regions. Such models are termed aggregative or multi-region models.

A multiregion representation is made by first subscripting the regional population totals ($n_i, \forall i$) and epidemic variables ($x_i, y_i, \forall i$), such that contacts between susceptibles living in region i and infectives in region j are calculated from $x_i y_j \exp(-$

Even though this specific multiregion model has been applied to the HIV/AIDS epidemic in Western Europe (Smith and Thomas, 2001) no attempt has been made to apply it to the epidemic in sub-Saharan Africa. The focus of the next section is to apply this model to the HIV/AIDS epidemic in sub-Saharan Africa where the outcome of such a modelling process will shed light on the magnitude and transmission pathways of the epidemic.

5.6 Application of the Multiregion Modelling System to AIDS Incidence Data for Sub-Saharan Africa

In the first part of this section, HIV/AIDS epidemic is regarded as a single entity and risk of infection is analysed as the likelihood of infection transfer between one region and another. This perspective suggests the outcome of prevention initiatives is also affected by passive demographic attributes like distance from the transmission pathway and lack of access to travel facilities, which combine to protect many communities from exposure to HIV infection (Smyth and Thomas, 1996a).

The key focus of this section is the application of a multiregion epidemic model to estimate the direction and strength of infection transfers between the countries of sub-Saharan Africa and examine the implication of these results for disease control. The extent to which the diffusion of AIDS might have been affected by changing accessibility characteristics of the population in countries of sub-Saharan Africa is explored. This concern arises because of recent urbanisation in Africa with its resultant rural-urban migration in search of jobs in the cities and the breakdown of traditional society, wars and conflict and consequent forced migration and displacement of people, improvement in means of transport and mass tourism within Africa. Before then Africans were living in closed communities and travel opportunities between countries and within countries were restricted. It is therefore hypothesised that these new travel patterns are likely to have important implication for the transmission of HIV in a continent where AIDS incidence accounts for three quarters of the world total.

The result of the various scenarios of the modelling process is presented in the following order:

1. Single region predictions by country
2. Some estimation issues specific to the sub-Saharan Africa context.
3. A multiregion solution with international travel proportions defined at a set rate and AIDS epidemic size set as the main attraction factor
4. A multiregion solution with international travel proportions defined and total population set as the main attraction factor rather than AIDS epidemic
5. Simulate pandemic started at different locations and recorded time elapsed to first infection in each of the countries

5.7 Calibration Procedure

5.7.1 Single Country Model Process

The construction of the single region specification was achieved using the Fortran programmes `cufit.for` and `com.for` which simulate the algorithm. (see appendix 2 and 3). For each country, a text file was created which consist of the recorded AIDS incidence by each year of reporting obtained from the WHO epidemiological facts sheets (WHO, 2002). This file was then incorporated into the Fortran programme `cufit.for` and run to obtain the value of n_i which best represent the size of the risk population. This process is then repeated using a second iteration mimicked in the Fortran programme, `com.for` to find the value of β_i that best represent the transmission probability within the given region i .

The next step is to find the year of initial HIV infection, $t = 0$. The application of this procedure assume the first HIV infection in each region occurred $D + A = f$ years prior to the firsts recorded AIDS incidence to define $(t = 0) = t_{\text{AIDS}} - f$, where t_{AIDS} represent the year in which the initial AIDS cases occurred in a particular country, $f = 6$. The Fortran programme was run with the appropriate number of entries in the text file to represent $(t=0)=t_{\text{AIDS}}-f$ with $f=6$. To test for the possibility that unrecorded cases of AIDS could have occurred, the entire procedure was repeated with $f=7,8,\dots$ until the globally minimum value of x^2 was found. The number of entries in each text file representing the reported AIDS cases was adjusted to mimic the

additions or subtraction of time. The Fortran programme was then run after each adjustment to find the optimum value. The year identified as corresponding to $(t=0)=t_{\text{AIDS}-f}$ (where $f=6$) was found to define the optimum timing for the series based upon the maximum degree of fit between predicted and observed incidence in most cases which can be suggested as representing an accurate data reporting service. The estimate of the date of the first HIV infections is taken to be f years prior to the first recorded AIDS incidence in most cases.

The establishment of the values of $t=0$, β , and n for each region allow the construction of the single region outputs. Fortran programme is used to simulate the epidemic in a single country over time. The simulation generated is then incorporated into an SPSS file and then graphed to show the incidence of HIV, Predicted AIDS cases and recorded AIDS cases, from $t=0$ to the time of the endemic equilibrium when AIDS incidence is first predicted to become extinct.

5.7.2 Multiregion Application Process

The multiregion model process uses the Fortran programme `airfit.for` (see appendix 4). The text file for the simulation contains the spatial co-ordinates of the major population centres, total population (millions), size of risk population (n_i) and transmission probability, (β_i), international travel proportion (q_{i2}), initial number of HIV infections ($y_{it}=0$) and the number of years between initial HIV infection and peak HIV prevalence as predicted by the single region specification.

These country estimates contained in the text file are then entered into the multiregion setting designed to find the optimum values for the decay parameter (λ) and travel proportions (q_{ij} , $\forall i, j$). The simulation process in the Fortran programme entails inputting the Partnership rate(r), Period of communicability (D), Period of antigen Suppression (A), and the number of regions in the simulation (m). The values assigned to these are constants and are defined as 10, 2, 4 and 41 respectively. The input of the distance decay parameter (λ) for the international and local travel are set at 0.30 and 7 respectively. After imputing these values, the number of time intervals required is entered. This was set at 50 to give the epidemic 50 years to run one course. The number of start regions is then decided. This enables analysis of epidemics with

simultaneous starts in more than one region (see appendix 5a, 5b and 5c). The start regions and the number of infections, $y_{it}=0$ are then inputted and the programme simulates one course of the epidemic through the region. The result obtained is then mapped according to the direction of infection flow between countries. Initial HIV infection date and peak HIV prevalence date are also shown by year together with an indication of the number of years (+ or -) difference between the figures for that particular country.

5.7.3 Fitting AIDS Incidence Data

The sub-Saharan regional system comprises the $i = 41$ countries that report annual incidence to the WHO (2000a) and these observed time series for each country i are denoted by $AIDS(t)_i, \forall ti$. With respect to these data, a calibration procedure has been designed to find terms of unknown or uncertain value that best fit predicted AIDS incidence to these observed values. This procedure operates in two stages: first, a single region version of the epidemic model described in the appendix is employed in an algorithm to find unknown epidemiological or disease parameters separately for each country; and second, the complete multiregion specification of this model is then used to estimate spatial parameters representing the degree of population mixing between the countries (see appendix 1).

More specifically, the initial algorithm searches for optimum values of the following terms: an estimate of the transmission probability (β_i) that is assumed to vary nationally either through viral mutation or geographical differences in the susceptibility to infection; an estimate of the population at risk to HIV infection in each country (n_i) defined as the predicted cumulative incidence of AIDS at the completion of the forecast epidemic cycle; and last, an estimate of the date of the initial HIV infections in each country ($t = 0$). These optima are identified by an iterative routine that minimises the value of the goodness of fit statistic

$$\chi^2(i) = \sum_f^g [AIDS(t)_i - (w(t)/A)]^2 / (w(t)/A), \quad \forall i, \quad (1)$$

Where $t = f$ is the time of the first officially recorded AIDS cases in each country and $t = g$ is the time when the last recorded AIDS cases were available. In Equation (1), $w(t)/A$ denotes the predicted incidence of AIDS in country i at time t (see the

appendix for the complete definition of this expression). Other epidemiological model parameters employed in this algorithm are given estimates taken from published sources. The duration of the periods of communicability ($D = 2$ years) and antigen suppression ($A = 4$ years) are drawn from research into viral abundance and antigen concentrations in patients after HIV infection (Anderson and May, 1991). The average value of $r = 10$ partners per year for those at high risk is taken from surveys of sexual behaviour made at different times during the course of the epidemic (see Smith and Thomas, 2001). The mixing of the risk populations is estimated by a second algorithm that finds optimum values for the distance decay parameter (λ) and the external travel proportions ($q_i, \forall i$). This routine employs a spatial search procedure that minimises the value of the statistic

$$T = [\sum_i |P(O)_i - P(E)_i|] / I. \quad (2)$$

Here, $P(O)_i$ is the observed (O) time of peak (P) AIDS incidence in country i and $P(E)_i$ is the corresponding predicted (E) peak time obtained from the multiregion model. T , therefore, is seen to be the average time difference between observed and predicted peak AIDS incidence. Moreover, the minimisation of T is subject to the constraint $q_i \leq 0.5, \forall i$, to ensure that the external travel proportion for each country is always less than the internal proportion. The full specification of this model, which is adapted from Thomas (2001), Smith and Thomas (2001) and Smith (2003), is in Appendix 1.

5.8 Results

5.8.1 Single Country Model Output

The graphical outcomes of fitting the single region epidemic model to AIDS series for eight selected countries using SPSS are shown in Figure 5.1. There is evidence of lack of a down turn, underreporting as well as missing data in some of the series but most of the series shows a good fit as in most of the western European series (Thomas, 2001).

The graphical outcome of fitting the single region epidemic model to AIDS series shows that caseloads reported for Mozambique and Botswana were still increasing by the year 2000 and 1998 respectively and their risk populations are fitted without knowledge of the date of peak AIDS incidence (Fig. 5.1). Secondly the results show evidence of underreporting in some countries such as Nigeria and South Africa (Fig 5.1). In South Africa, AIDS cases increased from 887 in 1992 to 1882 in 1993, 3816 in 1994 and 4219 in 1995 but decreased to 738 in 1996. In Nigeria, HIV cases increased from 3815 in 1997 to 18490 in 1998 but the figure dropped to 3661 in 2001. These drastic and sudden changes in AIDS cases cannot be attributed to a fall in incidence and may be due to underreporting. The graphical output also shows that in some countries there are missing data for the early phase of the epidemic. However, the erratic incidence data are replicated by the model, as is the case for Liberia and Somalia (Fig 5.1).

Results from some of the series do show a recent downturn in observed AIDS incidence, which is similar to that of Western Europe. For example, the graphical outcome for Ghana and Cote d'Ivoire represent a good fit (Fig 5.1). Most series including Ghana and Cote d'Ivoire exhibit declining annual incidence by 2001. In the case of Ghana and also for Cote d'Ivoire, this downturn had occurred by 1999.

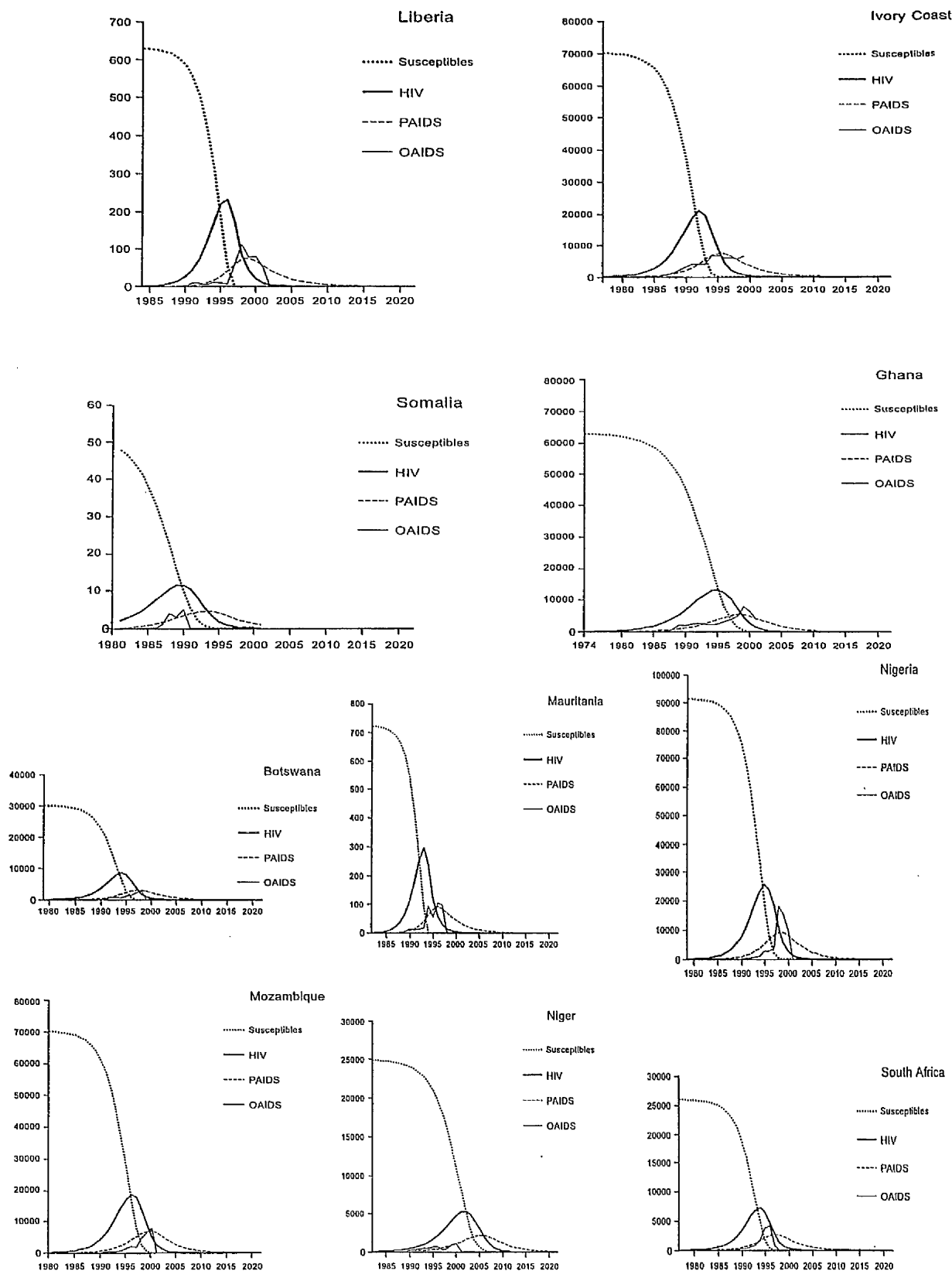


Figure 5.1 Single region output for selected countries as graphed using SPSS (HIV-predicted prevalence; PAIDS-predicted AIDS incidence; OAIIDS-observed AIDS incidence)

While annual AIDS incidence has started declining in some countries, the caseload of AIDS is still increasing in many countries and the date for peak AIDS incidence is still not known. The estimated dates of first HIV infection indicate an epidemic originating in the Democratic Republic of Congo in 1974 and diffusing to all countries in East, Central and Southern Africa by 1985. In the case of West Africa the estimated date of the first HIV infection also occurred in 1974 and by 1985 had diffused to other countries in the sub-region

The estimated date of first HIV infection obtained from the single region country model (Table 5.1) indicate that beginning in the Democratic Republic of Congo in Central Africa and Ghana in West Africa HIV infection first occurred in all sub-Saharan African Countries between 1974 and 1985.

The calibrated values of the transmission probability for each country are reasonably consistent with an estimate of $\beta=0.1$ derived early in the US epidemic from a large sample of seropositive individuals (May et al 1989). Higher values of transmission probabilities are obtained for countries with erratic and short duration of recorded AIDS incidence series such as Somalia and Congo (Table 5.1) and such countries require larger transmission probabilities to predict their apparent speedier epidemics (Smith and Thomas, 2001). The estimated date of forecast endemic state $T=h$ for countries of sub-Saharan Africa (Table 5.1) is between 2011 and 2031. The estimated date for the peak of the epidemic in sub-Saharan Africa is between 1983 and 2003 (Table 5.2). It was observed from the results that the epidemic peaked earlier in Central Africa than the rest of the sub-region. The estimated peak date for Congo was 1983, Zambia 1987, and Uganda and Malawi 1988, but for most of the countries, the estimated peak period of HIV/AIDS occurred between the early to mid 1990s (Table 5.2).

Table 5. 1 Epidemic Dates and Statistics fitted by the Single Country Model

Country	Estimated date of first HIV infection T=0	Date of first recorded AIDS cases (t=f)	Date of last recod AIDS Cases T=g	Date of forecast endemic state T=h	Transmission probability β	Size of risk population(n)	χ^2	Min χ^2/n
1 Zimbabwe	1981	1987	1998	2030	0.07881	7500	3336	0.44
2 Namibia	1985	1992	2000	2031	0.14921	41800	1243	2.97
3 Rwanda	1975	1983	1999	2024	0.10107	27600	0.5915	2.14
4 D R C	1974	1986	2000	2025	0.09598	95000	0.42885	4.51
5 Malawi	1979	1985	1999	2025	0.15530	58000	0.5605	9.66
6 Kenya	1980	1986	1998	2029	0.07887	81500	0.7042	8.64
7 S.Africa	1977	1982	1996	2025	0.10165	26000	0.4920	1.89
8 Swaziland	1981	1987	1999	2026	0.11126	10000	0.1028	0.00
9 Uganda	1977	1983	1999	2024	0.13464	59000	0.3565	6.04
10 Burundi	1974	1986	2000	2025	0.09032	31000	0.8071	2.60
11 Ethiopia	1979	1986	2001	2027	0.09503	100400	0.15540	1.54
12 Eritrea	1982	1988	1999	2024	0.12513	11000	0.197	1.79
13 Botswana	1979	1987	1998	2027	0.10166	30000	0.1016	3.38
14 Cent Africa Rep	1980	1987	1996	2017	0.17403	8400	0.7511	8.94
15 Zambia	1978	1984	1997	2022	0.17410	46500	0.15697	3.37
16 U R Tanzania	1976	1983	2000	2025	0.10826	145000	0.38391	2.64
17 Djibouti	1981	1988	1998	2016	0.14142	2400	321	0.00
18 Angola	1977	1985	2000	2022	0.09446	10000	0.936	0.00
19 Congo	1979	1986	2001	2018	0.82780	41000	0.1066	0.00
20 Mozambique	1980	1986	2000	2028	0.09700	70000	0.4461	6.37
21 Lesotho	1979	1986	2000	2027	0.09109	35000	0.3275	9.35
22 Somalia	1981	1987	1990	2001	0.22893	26	2	7.69
23 Equitorial Guinea	1982	1988	2000	2018	0.10754	1600	98	0.06
24 Togo	1980	1987	2000	2023	0.12741	13900	955	6.87
25 Mali	1978	1985	1999	2019	0.11465	6300	387	6.14
26 Senegal	1977	1986	1999	2016	0.11057	3400	428	0.12
27 Sieraleon	1977	1987	1998	2011	0.10771	400	95	0.23
28 Burkina Faso	1978	1986	2001	2025	0.10395	21300	1215	5.70
29 Nigeria	1981	1986	2001	2015	0.10162	92000	25765	0.28
30 Mauritania	1981	1988	1996	2012	0.13318	720	54	0.07
31 Gabon	1975	1980	2001	2023	0.09401	10500	304	2.89
32 Liberia	1985	1991	2001	2015	0.12361	630	85	0.13
33 Chad	1980	1986	2001	2026	0.10798	19400	1285	6.62
34 Ghana	1974	1986	2001	2022	0.08544	63000	8035	0.12
35 Cote d'Ivoire	1977	1986	1999	2027	0.10421	70000	3980	5.68
36 Benin	1979	1985	2000	2022	0.10175	7500	404	5.38
37 Guinea	1980	1987	2000	2026	0.10535	13400	366	2.73
38 Guinea B	1979	1987	1999	2014	0.12578	1400	435	0.31
39 Niger	1982	1987	2000	2030	0.08589	25000	4137	0.16
40 Gambia	1980	1989	1998	2016	0.10297	1200	735	0.61
41 Cameroon	1980	1986	1998	2029	0.10296	47000	2041	6.38
42 Sudan	1977	1986	2001	2016	0.09533	5000	830	0.166

Table 5.2. Estimated HIV Peak Year and Predicted Cases

COUNTRY	PEAK YEAR	PREDICTED HIV CASES
SOMALI	1990	11
SOUTH AFRICA	1994	7410
BOTSWANA	1994	8551
MOZAMBIQUE	1996	18505
NIGERIA	1995	26170
ANGOLA	1995	2533
CONGO	1983	45388
ETHIOPIA	1996	25569
NIGER	2002	5311
CAMEROON	1995	13668
DEMOCRATIC REPUBLIC CONGO	1991	24636
RWANDA	1991	7776
SIERRA LEON	1991	123
LIBERIA	1996	232
EQUATORIAL GUINEA	1996	491
GAMBIA	1995	342
LESOTHO	1997	8376
ERITREA	1995	4020
DJIBOUTI	1992	1047
GABON	1998	2623
MALAWI	1988	26399
NAMIBIA	1995	18419
COTE D'VOIRE	1992	20694
CHAD	1995	5946
MAURITANIA	1992	294
NAMIBIA	1995	18441
CENTRA AFRICAN REPUBLIC	1989	4369
ZIMBABWE	2003	13307
U R TANZANIA	1990	45350
BURUNDI	1993	7224
UGANDA	1988	24414
SWAZILAND	1996	3253
KENYA	2003	14488
ZAMBIA	1987	23344
BURKINA FASO	1993	6287
SENEGAL	1991	1097
MALI	1991	2082
TOGO	1992	5342
GUINEA BISSAU	1991	524
GUINEA	1996	3935
BENIN	1995	2139
GHANA	1995	13253
SUDAN	1994	1286

5.8.2 Multiregion Model Output

A multiregion solution with international travel proportions defined at a set rate and AIDS epidemic size set as the main attraction factor

The result of the multiregion model simulation of the initial pathway and peak pathway for an epidemic with AIDS specified as both the risk population and travel attraction is presented in the section below. Also displayed is the predicted date of the first HIV infection in each country in the multiregion model and the lags (-) and leads (+) between this date and the single-country model estimate.

The estimated date of first HIV infection obtained from the single region country model (Table 5.1) indicate an epidemic originating in DR Congo and Ghana simultaneously in 1974 that had spread to all countries of sub-Saharan Africa by 1985.

5.8.3 Multiregion Solution with Epidemic Start at D R Congo.

The first application of the multiregion model mimics an epidemic that starts in D R Congo in 1974. D R Congo has been hypothesised as the source of the HIV infection (Gould, 1993). Table 5.1 and Figure 5.1(a) show the multi region fit statistics for the simulation with D R Congo as the start region.

The infection pathway displays a characteristic hub and spoke pattern where the infection diffuses from the D R Congo to other countries in the sub-region. In this solution, D R Congo is the main hub, which served as the initial source of infection in sub-Saharan Africa. From the source region in D.R.Congo, the virus diffuses to the neighbouring Congo within the period of four years and by the 6th and 7th year has spread to Central African Republic, Angola, Zambia and Namibia. Between eight and ten years of its inception, the virus has diffused to all countries in Sub-Saharan Africa. The transmission of HIV to Zambia established a second node for the transfer of infection to Zimbabwe, Swaziland and South Africa. From the source region the virus diffused relatively faster to Central, Eastern and Southern African countries than West African countries (Fig 5.1(a)).

It is worth noting however that the links characterising any starting pathway is influenced by the location of the selected source region (Smith and Thomas, 2001). For this reason, the linkage pattern generated at peak HIV prevalence provides a more distinctive visualisation of infection transfers when sufficient time has elapsed for the source region effect to have been absorbed into the more general system structure.

Fig 5.1(b) shows the result from the same simulation with the source of infection at Peak HIV prevalence the number of years taken for the epidemic to Peak in each country. Here the influence of D.R.Congo is much reduced and three major hubs are established. D.R. Congo becomes the main source of infection in Central Africa. Tanzania has become a major hub infecting East Africa and Ethiopia becoming a secondary node for transmission to the Sudan, Eritrea and Djibouti while Swaziland serves as a secondary node of infection to Botswana, South Africa and Lesotho. In West Africa on the other hand, Cote D'Ivoire becomes the major hub of infection with Nigeria acting as a secondary node for transmission to Burkina Faso and Niger.

The timing errors at peak prevalence range between -5 to +8. The major inaccuracies occur in Zimbabwe +8, Kenya +7 and Senegal -5. Each area in the sub-region has got both countries with a high and low degree of accuracy. For example in East Africa, Ethiopia has a value of 0 whereas Kenya has a value of +7. In the Central Africa region, Central Africa Republic has a value of 0, whereas Congo has a value of +7. In West Africa similar pattern occurs. Whereas the Benin has a value of 0, Senegal has a value of -5.

The number of years taken for the epidemic to peak also differs from country to country. On the average it took between 15 and 23 years for the epidemic to peak in sub-Saharan Africa. The earliest time for the epidemic to peak occurred in Zambia and the Central Africa Republic. Here the epidemic peaked within the first 15 years of its inception. However, in countries such as Ghana and the Gambia it took 23 years for the epidemic to peak.

Table 5.3 A multiregion fit statistics with AIDS epidemic size set as the main attraction factor start region 2(D R Cong)

Solution with AIDS epidemic size set as the main attraction factor start region 2(D.R.CONGO)				
Country	P(O)i	P(E)i	Pi	q i2
1. Ghana	1995	1997	-2	0.1
2. DRC	1991	1992	-1	0.1
3. Zimbabwe	2003	1995	8	0.1
4. Namibia	1995	1991	4	0.1
5. Rwanda	1991	1994	-3	0.1
6. Malawi	1988	1991	-3	0.1
7. Kenya	2003	1996	7	0.1
8. South Africa	1994	1992	2	0.1
9. Swaziland	1996	1994	2	0.1
10. Uganda	1988	1992	-4	0.1
11. Burundi	1993	1995	-2	0.1
12. Ethiopia	1996	1996	0	0.1
13. Eritrea	1995	1994	1	0.1
14. Botswana	1994	1994	0	0.1
15. Cent Africa Rep	1989	1989	0	0.1
16. Zambia	1987	1989	-2	0.1
17. Tanzania	1990	1994	-4	0.1
18. Djibouti	1992	1993	-1	0.1
19. Angola	1995	1994	1	0.1
20. Congo	1983	1979	4	0.1
21. Mozambique	1996	1995	1	0.1
22. Lesotho	1997	1995	2	0.1
23. Equatorial Guinea	1996	1994	2	0.1
24. Gabon	1998	1995	3	0.1
25. Chad	1995	1994	1	0.1
26. Mauritania	1992	1994	-2	0.1
27. Cote D'Ivoire	1992	1996	-4	0.1
28. Guinea	1996	1996	0	0.1
29. Guinea Bissau	1991	1995	-4	0.1
30. Sudan	1994	1996	-2	0.1
31. Nigeria	1995	1995	0	0.1
32. Sierra Leon	1991	1996	-5	0.1
33. Burkina Faso	1993	1996	-3	0.1
34. Senegal	1991	1996	-5	0.1
35. Gambia	1995	1997	-2	0.1
36. Liberia	1996	1995	1	0.1
37. Mali	1991	1995	-4	0.1
38. Togo	1992	1993	-1	0.1
39. Cameroon	1995	1994	1	0.1
40. Niger	2002	1997	5	0.1
41. Benin	1995	1995	0	0.1
λ			0.1000	
Min. T.,years			2.4146	

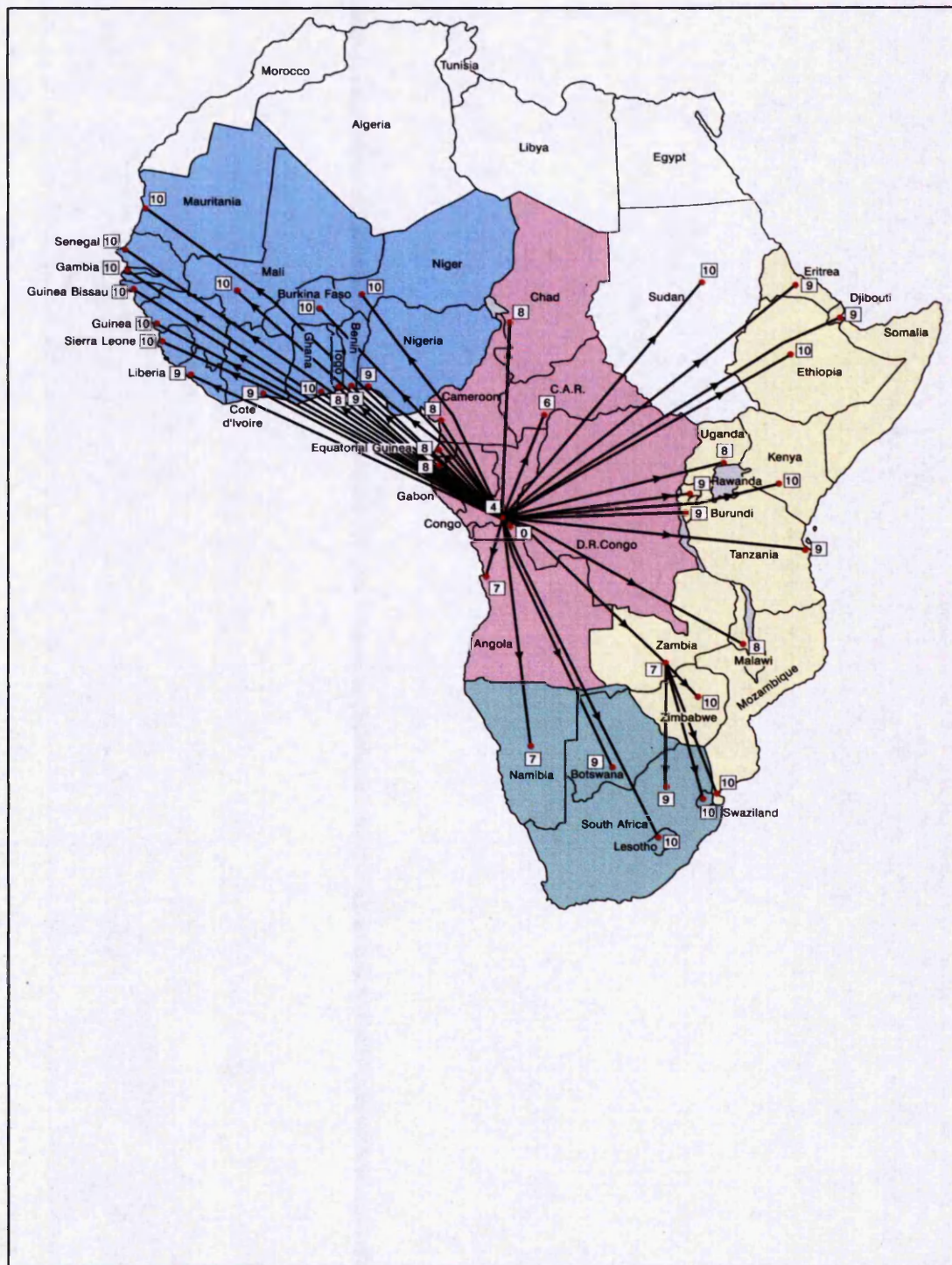


Figure 5.1(a). Starting pathway of first HIV infection mapped for the scenario utilising AIDS incidence as the attraction factor.

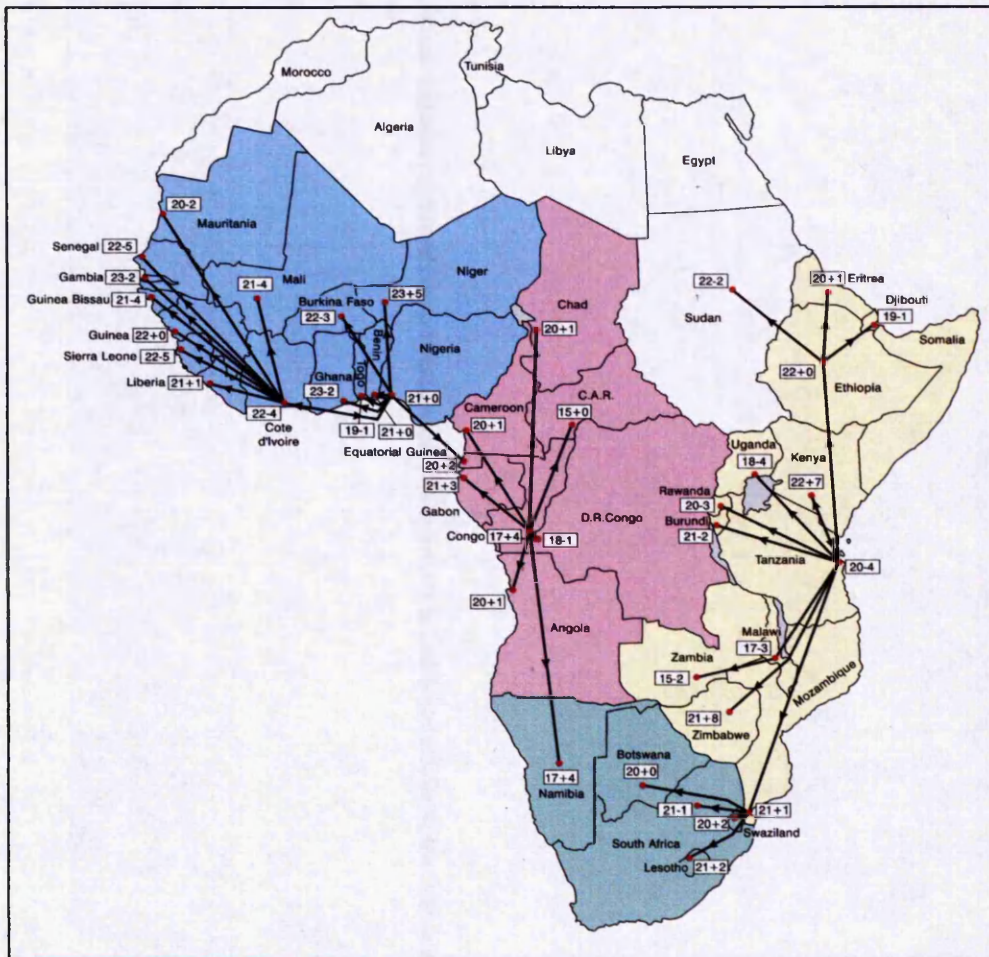


Figure 5.1(b). Principal infection vectors at peak HIV prevalence for the scenario utilising AIDS incidence as the attraction factor

5.8.4 Multiregion Solution with Epidemic Start in Start Region 1(Ghana)

This solution is simulated for an epidemic started in 1974 in Ghana and is illustrated in figure 5.2(a) and 5.2(b). The pathway of this solution displays a characteristic hub and spoke pattern (Bryan and O' Kelly, 1999). Three hubs dominate the pathway. Ghana serves as the source of initial infection in all West African countries, then the transmission of HIV to Democratic Republic of Congo establishes a node for the transfer of infection to East and Southern Africa. From Democratic Republic of Congo, the epidemic spread to Zambia where another node is established for the transfer of infection to East and Southern African countries. Two linear routes are also

evident in the network. These are the path formed between Ghana, Central Africa Republic and the Sudan on one hand and the path between the Democratic Republic of Congo, Uganda, Ethiopia and Djibouti on the other.

From the initial start in Ghana, the virus takes seven years to diffuse to Togo and by the 8th year it has spread to Cote d'Ivoire, Nigeria, Benin, Burkina Faso, Mali, Mauritania, Senegal, the Gambia, Guinea Bissau and Liberia. By the 9th and 10th years the virus has spread to Guinea, Sierra Leone and Niger. Between seven and ten years, the virus has diffused to all countries in West Africa. The virus then spreads to Central Africa between ten and eleven years and by the 13th and 14th year has reached East and Southern Africa. Starting from Ghana, the virus diffuses to all Sub-Saharan Africa between seven and fourteen years (Ref. fig 5.2a).

The simulated epidemic structure at peak HIV prevalence is illustrated in figure 5.2b. Here the equivalent linkage pattern is mapped at the time of peak HIV prevalence when the influence of the source region has been largely absorbed into the more general system structure. In this peak network, the influence of the source region (Ghana) is lost and Cote d'Ivoire becomes a node for infection transfer to countries in West Africa. In Central Africa, the Democratic Republic of Congo remains a major source of infection to all countries in Central Africa. The link between Democratic Republic of Congo and Zambia is cut and Tanzania becomes the major source of infection to all countries in East and South Africa except Namibia, which still receives infection from Democratic Republic of Congo.

The timing errors at peak prevalence range between -8 and +5. The major inaccuracies occur in Tanzania with -8, Rwanda -7 and Zimbabwe +5. With the exception of Niger and Liberia, the predicted peak of the epidemic in West Africa occurred latter than estimated. In the case of Central Africa, just as West Africa, the predicted peak occurs later than estimated with the exception of Gabon and Equatorial Guinea where the predicted and estimated peak was the same. In Central and Southern Africa a similar pattern is shown. Here with the exception of Kenya, Zimbabwe and Namibia, the predicted peak occurred later than estimated.

The peak of the epidemic occurred earlier in some countries than others. For example it took 17 years for the epidemic to peak in Central African Republic but it took 25 years for the epidemic to peak in Kenya and Lesotho (see. Fig 5. 2b).

Table 5.4. A multiregion fit statistics with AIDS epidemic size set as the main attraction factor start region 1(Ghana)

Solution with AIDS epidemic size set as the main attraction factor .start region 1(Ghana)				
Country	P(O) _i	P(E) _i	P _i	q _{i2}
1.Ghana	1995	1996	-1	0.1
2.DRC	1991	1997	-6	0.1
3.Zimbabwe	2003	1998	5	0.1
4.Namibia	1995	1994	1	0.1
5.Rwanda	1991	1998	-7	0.1
6.Malawi	1988	1994	-6	0.1
7.Kenya	2003	1999	4	0.1
8.South Africa	1994	1998	-4	0.1
9.Swaziland	1996	1998	-2	0.1
10.Uganda	1988	1996	-8	0.1
11.Burundi	1993	1998	-5	0.1
12.Ethiopia	1996	1999	-3	0.1
13.Eritrea	1995	1997	-2	0.1
14.Botswana	1994	1998	-4	0.1
15.Cent Africa Rep	1989	1991	-2	0.1
16.Zambia	1987	1992	-5	0.1
17.Tanzania	1990	1998	-8	0.1
18.Djibouti	1992	1996	-4	0.1
19.Angola	1995	1998	-3	0.1
20.Congo	1983	1984	-1	0.1
21.Mozambique	1996	1998	-2	0.1
22.Lesotho	1997	1999	-2	0.1
23.Equatorial Guinea	1996	1996	0	0.1
24.Gabon	1998	1998	0	0.1
25.Chad	1995	1997	-2	0.1
26.Mauritania	1992	1993	-1	0.1
27.Cote D'Ivoire	1992	1996	-4	0.1
28.Guinea	1996	1997	-1	0.1
29.Guinea Bissau	1991	1994	-3	0.1
30.Sudan	1994	1999	-5	0.1
31.Nigeria	1995	1996	-1	0.1
32.Sierra Leon	1991	1996	-5	0.1
33.Burkina Faso	1993	1996	-3	0.1
34.Senegal	1991	1996	-5	0.1
35.Gambia	1995	1997	-2	0.1
36.Liberia	1996	1994	2	0.1
37.Mali	1991	1995	-4	0.1
38.Togo	1992	1993	-1	0.1
39.Cameroon	1995	1997	-2	0.1
40.Niger	2002	1998	4	0.1
41.Benin	1995	1996	-1	0.1
λ			0.1000	
Min,T.,years			3.1951	

q_{i2}= the international travel proportions estimated for the listed solution.

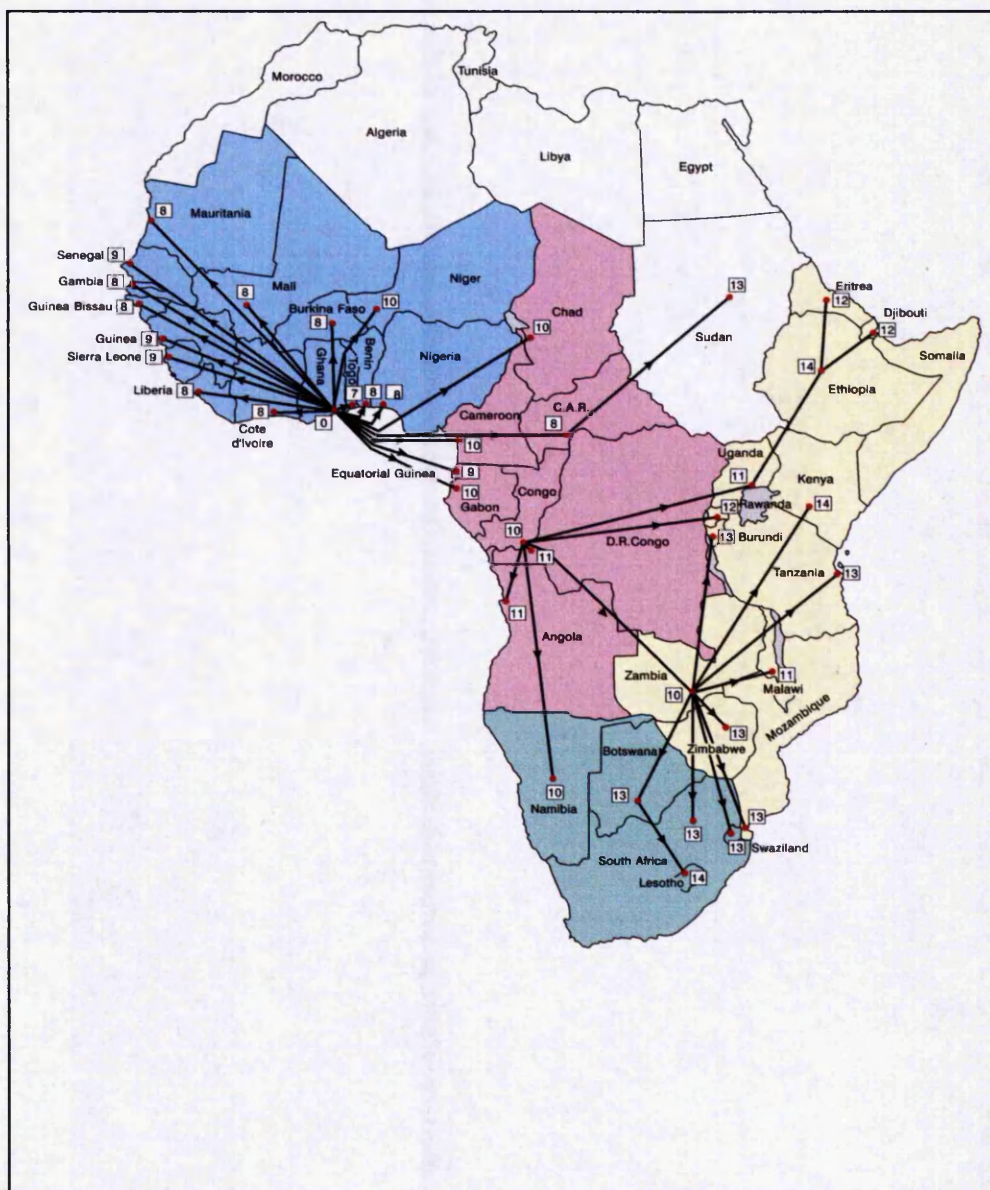


Fig 5.2 (a). Starting Pathway of First HIV Infections Fitted To AIDS Incidence

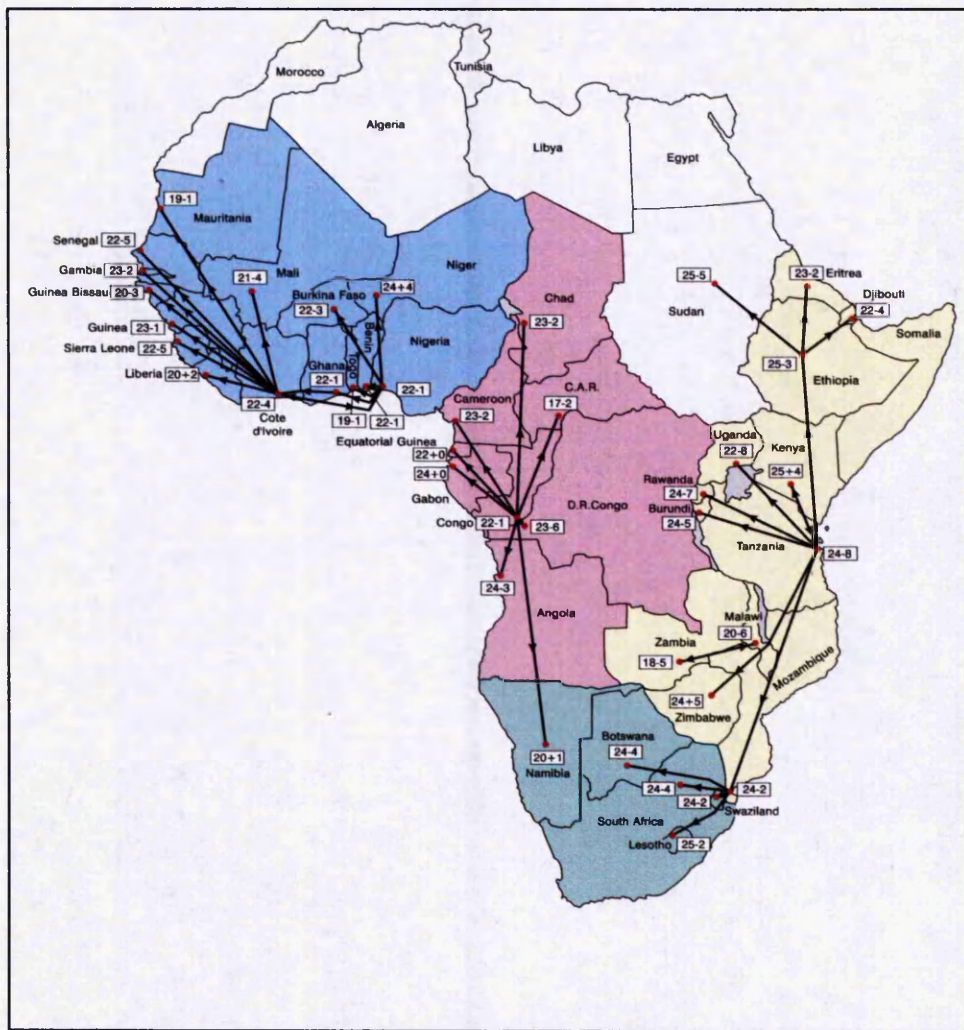


Figure 5.2(b). Principal infection vectors at peak HIV prevalence fitted to AIDS incidence

5.8.5 Multiregion Solution with Epidemic Start in Both Region 1 and 2 (Ghana and D R Congo)

Here the epidemic is simulated to have started from both Ghana and D R Congo in 1974 concurrently. The pathway for this solution also displays the hub and spoke pattern with two hubs dominating the pathway. Ghana serves as the main source of initial infection in West Africa from where HIV is transmitted to other countries in West Africa. From the initial start in Ghana, the virus takes six years to infect neighbouring Togo and by the 7th year has spread to Cote d'Ivoire, Liberia, Mali, Benin and Nigeria. By the 8th year the virus had spread to all countries in West Africa. In the case of Central, East and South Africa, from the initial source in D R Congo, the virus takes four years to infect neighbouring Congo and by the 6th year has spread to Central Africa Republic and by the 7th year has spread to Cameroon, Equatorial Guinea, Gabon, Angola, Namibia, and Zambia and by the 8th, 9th, and 10th year has spread to every country in East, and Southern Africa.

The simulated pathway at the peak HIV prevalence when the influence of the source region is largely absorbed into the more general system structure is illustrated in figure 5.3b. Here the influence of Ghana and Democratic Republic of Congo is lost and for West Africa Cote d'Ivoire serves as the node for infection transfer to other West African countries. The link between Democratic Republic of Congo and East Africa is cut and instead, Tanzania becomes a node for infection transfer to Eastern and Southern Africa. Democratic Republic of Congo remains as a node infecting Congo, Gabon, Equatorial Guinea, Cameroon, Angola and Central Africa Republic. The infection transfer between Democratic Republic of Congo and Chad is redirected between Nigeria and Chad but the link between Democratic Republic of Congo and Namibia is maintained.

The timing errors at peak prevalence are generally smaller with the exception of Niger, Kenya and Zimbabwe where this peak occurs six, seven and eight years earlier than estimated. Between fourteen and twenty four years HIV has peaked in all sub-Saharan Countries. In Central and East Africa, the epidemic peaked earlier than West Africa, for example HIV peaked in Central Africa Republic within 14 years and by the 15th, 17th, and 18th year has peaked in Zambia, Congo and Uganda respectively. In West Africa the epidemic first peaked in Togo within eighteen years.

Table5.5. A Multiregion Fit Statistics with AIDS Epidemic size set as the Main attraction factor Start Region 1&2(Ghana and D R Congo)

Solution with AIDS epidemic size set as the main attraction factor start region 1&2 (Ghana and D R Congo).				
Country	P(O)i	P(E)i	Pi	q i2
1.Ghana	1995	1995	0	0.1
2.DRC	1991	1992	-1	0.1
3.Zimbabwe	2003	1995	8	0.1
4.Namibia	1995	1998	-3	0.1
5.Rwanda	1991	1994	-3	0.1
6.Malawi	1988	1991	-3	0.1
7.Kenya	2003	1996	7	0.1
8.South Africa	1994	1992	2	0.1
9.Swaziland	1996	1994	2	0.1
10.Uganda	1988	1992	-4	0.1
11.Burundi	1993	1995	-2	0.1
12.Ethiopia	1996	1996	0	0.1
13.Eritrea	1995	1994	1	0.1
14.Botswana	1994	1994	0	0.1
15.Cent Africa Rep	1989	1988	1	0.1
16.Zambia	1987	1989	-2	0.1
17.Tanzania	1990	1994	-4	0.1
18.Djibouti	1992	1993	-1	0.1
19.Angola	1995	1994	1	0.1
20.Congo	1983	1979	4	0.1
21.Mozambique	1996	1995	1	0.1
22.Lesotho	1997	1995	2	0.1
23.Equatorial Guinea	1996	1993	3	0.1
24.Gabon	1998	1994	4	0.1
25.Chad	1995	1994	1	0.1
26.Mauritania	1992	1993	-1	0.1
27.Cote D'Ivoire	1992	1994	-2	0.1
28.Guinea	1996	1995	1	0.1
29.Guinea Bissau	1991	1993	-2	0.1
30.Sudan	1994	1996	-2	0.1
31.Nigeria	1995	1994	1	0.1
32.Sierra Leon	1991	1995	-4	0.1
33.Burkina Faso	1993	1995	-2	0.1
34.Senegal	1991	1995	-4	0.1
35.Gambia	1995	1997	-2	0.1
36.Liberia	1996	1993	3	0.1
37.Mali	1991	1994	-3	0.1
38.Togo	1992	1992	0	0.1
39.Cameroon	1995	1994	1	0.1
40.Niger	2002	1996	6	0.1
41.Benin	1995	1994	1	0.1
λ			0.1000	
Min.T., years			2.3171	

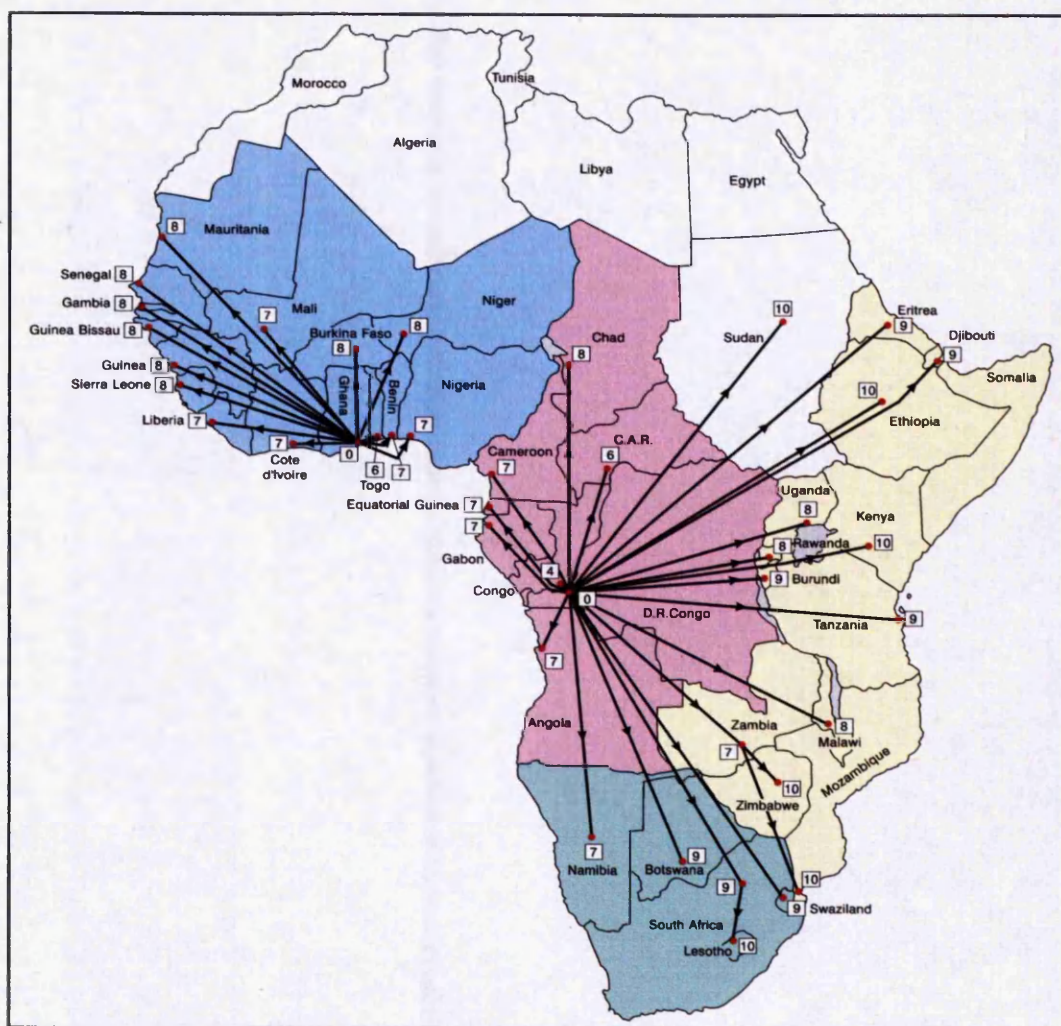


Figure 5.3(a). Starting pathway of first HIV infection fitted to AIDS incidence

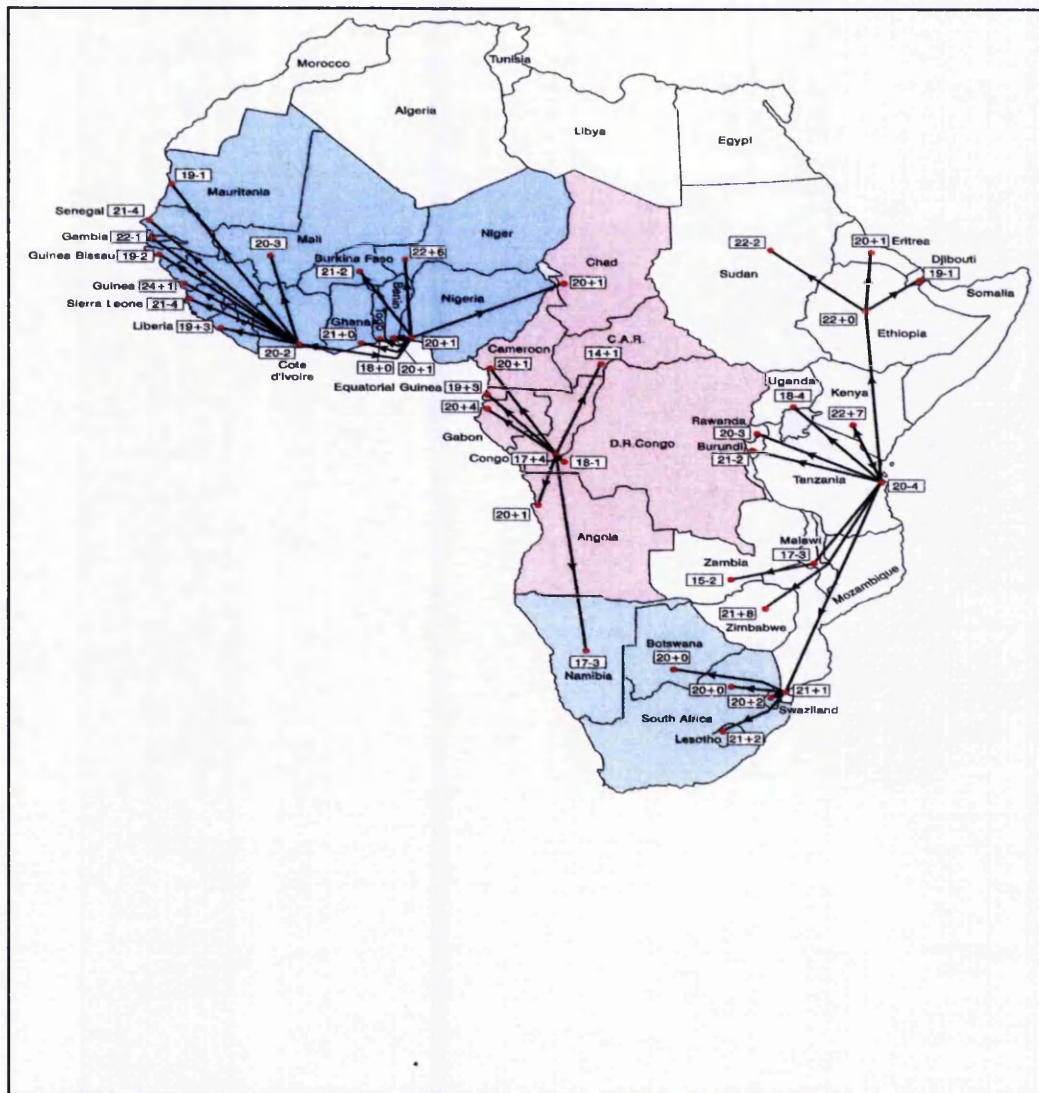


Figure 5.3(b). Principal Infection Vectors At Peak HIV Prevalence Fitted To AIDS Incidence

5.9 Population Driven Specification

The population driven simulations assume that the total population of each country is the attraction factor (mi) and discount the influence of the risk population. This is done to find out whether, simulations using total population of a country as an attraction factor will give a more accurate multiregion prediction when compared with the single region output and secondly, to find out whether such simulation changes the infection pathways and the speed with which HIV diffuses through sub-Saharan Africa.

5.9.1 Multiregion Solution with Epidemic Start in Start Region 2(D R Congo)

Using total population as the attraction factor changes the initial infection pathway in West Africa and part of East Africa (Figure 5.4(a)). But the speed of infection is almost the same as the previous simulation using AIDS as the attraction factor as most countries are infected in the same year or are one year earlier or latter. The infection pathway is completed in year eleven in this simulation as compared to ten years in the previous simulation. The only country with a marked difference is Angola where it took only four years for HIV to diffuse to from D R Congo instead of seven years from the previous simulation. Table.5.6. and Figure 5.4(a) show that using the total population as an attraction factor as opposed to the risk population as the attraction factor increases the speed of the epidemic in sub-Saharan Africa. The only exceptions are Swaziland, Eritrea, Botswana, Tanzania, Djibouti and Lesotho, where HIV peak prevalence occurs earlier than the previous simulation.

The infection pathway in West Africa moves from D R Congo to Nigeria from where all of the West African countries with the exception of Cote d'Ivoire, Liberia and Sierra Leone which continue to receive infection from D R Congo as the previous simulation. Also D R Congo does not infect Eritrea and Djibouti as it did in the previous simulation, instead the virus first infects the Sudan and Ethiopia from where the virus is passed on to Eritrea and Djibouti. Also in this simulation D R Congo infects South Africa from where infection is transferred to Lesotho, Swaziland, Mozambique and Botswana unlike the previous simulation where the virus first

infects Zambia after which it is passed on to South Africa, Swaziland and Mozambique. It is also evident from this simulation that there are two main hubs, D R Congo and Nigeria unlike the previous simulation with D R Congo as the main hub. Here D R Congo does not infect many countries as the previous simulation.

HIV infection pathway at the peak of HIV prevalence differs from the previous simulation using risk population as the attraction factor. In West Africa Nigeria becomes the main hub from which countries in the sub-region are infected instead of Cote d'Ivoire in the previous simulation. The influence of D R Congo as a hub is reduced but the infection pathway in East Africa remains operational except that the link between East and Southern Africa is curtailed. South Africa becomes a node infecting Namibia, Botswana, Lesotho Swaziland and Mozambique as compared with the previous simulation using the risk population as the attraction factor where Mozambique infects the countries of southern Africa.

The result from the simulation using total population as an attraction factor shows a relatively bigger value of the Min T years value of 2.87804 than the value of the previous simulation using the risk population as an attraction factor with a Min.T years value of 2.4146. This is an indication that the simulation using total population as an attraction factor is less accurate compared to the previous simulation using risk population as an attraction factor.

Table 5.6 Result generated for the Scenario using total Population as attraction factor
(Start Region 2-D R Congo)

Solution with Population as the main attraction factor selected start region 2 (D R Congo)				
Country	P(O)i	P(E)i	Pi	q i2
1.Ghana	1995	1996	-1	0.1
2.DRC	1991	1987	4	0.1
3.Zimbabwe	2003	1991	12	0.1
4.Namibia	1995	1991	4	0.1
5.Rwanda	1991	1994	-3	0.1
6.Malawi	1988	1991	-3	0.1
7.Kenya	2003	1996	7	0.1
8.South Africa	1994	1992	2	0.1
9.Swaziland	1996	1995	1	0.1
10.Uganda	1988	1992	-4	0.1
11.Burundi	1993	1995	-2	0.1
12.Ethiopia	1996	1996	0	0.1
13.Eritrea	1995	1995	0	0.1
14.Botswana	1994	1995	-1	0.1
15.Cent Africa Rep	1989	1987	2	0.1
16.Zambia	1987	1989	-2	0.1
17.Tanzania	1990	1995	-5	0.1
18.Djibouti	1992	1994	-2	0.1
19.Angola	1995	1989	6	0.1
20.Congo	1983	1977	6	0.1
21.Mozambique	1996	1995	1	0.1
22.Lesotho	1997	1996	1	0.1
23.Equatorial Guinea	1996	1993	3	0.1
24.Gabon	1998	1993	5	0.1
25.Chad	1995	1993	2	0.1
26.Mauritania	1992	1994	-2	0.1
27.Cote D'Ivoire	1992	1995	-3	0.1
28.Guinea	1996	1996	0	0.1
29.Guinea Bissau	1991	1995	-4	0.1
30.Sudan	1994	1993	1	0.1
31.Nigeria	1995	1993	2	0.1
32.Sierra Leon	1991	1992	-1	0.1
33.Burkina Faso	1993	1995	-2	0.1
34.Senegal	1991	1995	-4	0.1
35.Gambia	1995	1996	-1	0.1
36.Liberia	1996	1992	4	0.1
37.Mali	1991	1994	-3	0.1
38.Togo	1992	1993	-1	0.1
39.Cameroon	1995	1992	3	0.1
40.Niger	2002	1996	6	0.1
41.Benin	1995	1993	2	0.1
λ			0.1800	
Min T.,years			2.8780	

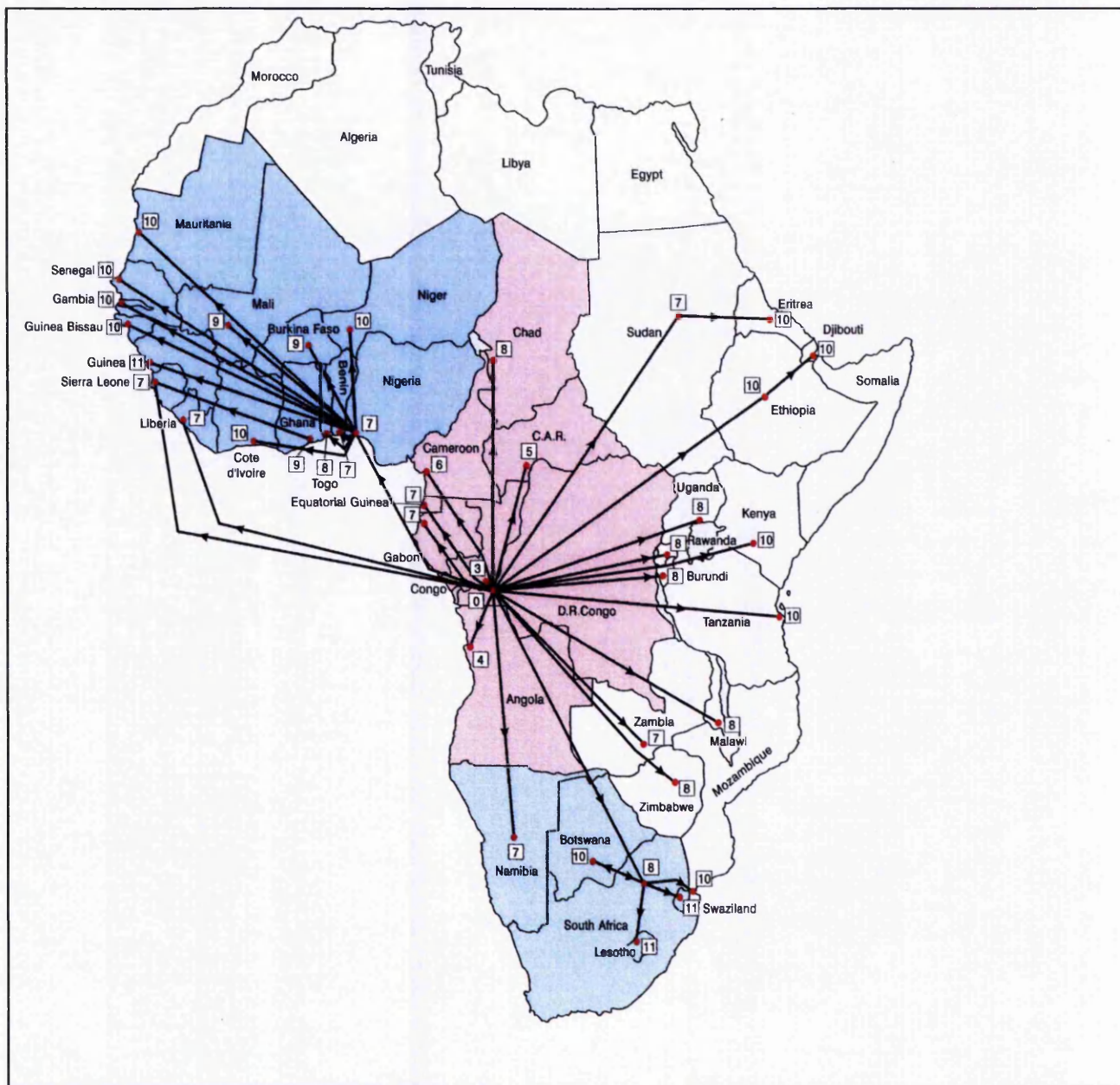


Figure 5.4 (A). Initial HIV infection vectors mapped for the scenario utilising total population as attraction factor

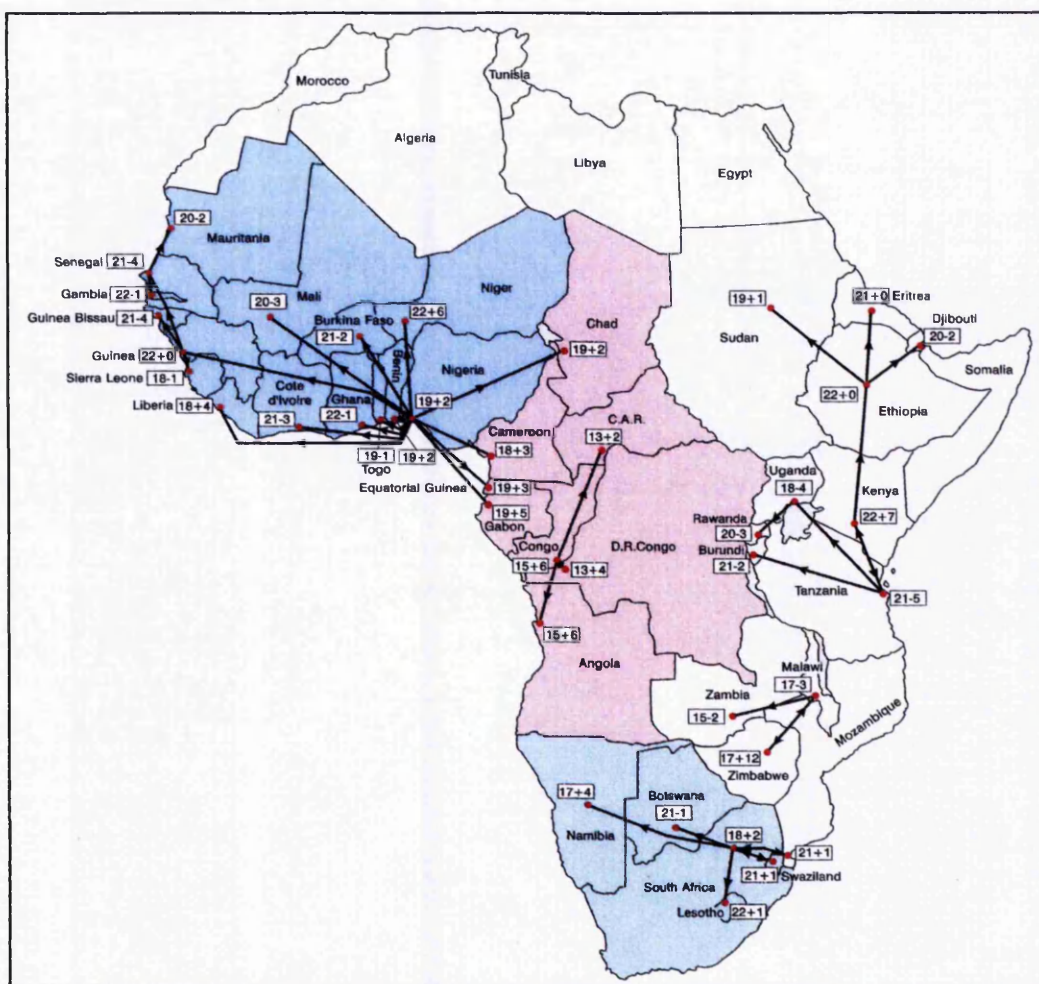


Figure 5.4(B) Infection vectors at HIV peak prevalence for the scenario utilising total population as attraction factor

5.9.2 Multiregion Solution with Epidemic Start in Start Region 1(Ghana)

This solution is simulated for an epidemic started in Ghana in 1974 using total population as the attraction factor instead of risk population. Figure 5.5a and table 5.7. show that using total population as an attraction factor changes the infection pathway through sub-Saharan Africa and the speed with which it diffuses. The infection pathway is completed by the 18th year as opposed to the 14th in the previous simulation based on risk population as attraction factor. However most countries are infected in the same year especially in West Africa. Countries in the East and Southern Africa are infected latter than the previous simulation and at a slower speed.

This is particularly evident in Lesotho and Swaziland where infection occurred in the 18th and 17th year as compared to the 14th and 13th year in the previous simulation.

There is a marked difference in the infection pathway as compared to the previous simulation. Here the link between Ghana on one hand and Mali, Burkina Faso, Niger, Chad, Cameroon, Gabon and Equatorial Guinea on the other hand is cut. Instead, Nigeria becomes a hub from which the virus spreads to such countries. The link between Ghana and Countries to the west of Ghana is however maintained. In Central Africa, the virus spreads from D R Congo to Uganda but the link between Uganda and Ethiopia is cut and instead redirected to Kenya. Here the infection spreads from Central African Republic to Ethiopia through the Sudan. Infection transfer between D R Congo and Zambia remains the same as the previous simulation but the influence of Zambia as a hub in the sub-region is much reduced as compared to the previous simulation. Here infection spreads from Zambia to Zimbabwe, Malawi and Tanzania and the link between Zambia and South Africa is cut and instead South Africa receives infection transfer from Namibia. The infection then spread from South Africa to Botswana, Mozambique, Swaziland and Lesotho.

The infection pathway at the peak of HIV prevalence (Figure 5.5b) and (table 5.7) show that using total population as opposed to the risk population as an attraction factor reduces the speed of the epidemic in Namibia, Rwanda, Malawi, Kenya, Swaziland, Burundi, Ethiopia, Eritrea, Botswana, Zambia, Djibouti, Congo, Mozambique and Lesotho. On the other hand, the speed of the epidemic is increased in countries such as D R Congo, Zimbabwe, Uganda, Tanzania, Angola, Guinea, Sudan, Nigeria, Sierra Leone, Senegal, Gambia, Liberia, Mali and Benin. All the other countries have dates of peak HIV prevalence that are the same as those predicted in the previous solution (Table 5.4). The values predicted for the timing of peak HIV prevalence show marked difference with the accuracy of the initial HIV infection dated with a range of -9 to +6 with the major inaccuracies occurring in Rwanda with -9, Malawi with -9 and Zimbabwe with +6.

The source of infection at peak HIV prevalence differs from the previous simulation using risk population as the attraction factor. Figure 5.5b shows that Nigeria is the main source of infection at peak HIV prevalence for West African countries using

total population as the attraction factor unlike the previous solution where Cote d'Ivoire was the main source of infection at peak HIV prevalence. Also the influence of D R Congo as a source of infection is reduced and the link between D R Congo and Namibia, Angola, Chad, Gabon, Equatorial Guinea, Cameroon and Chad is cut. In East Africa, the influence of Tanzania as a hub is also reduced as infection transfers to Malawi, Mozambique, Zimbabwe and Ethiopia is cut. In Southern African sub-region, South Africa replaces Mozambique as the main hub infecting Lesotho, Namibia, Swaziland, Botswana and Mozambique.

The result shows that using total population instead of total risk population as an attraction factor produces a multiregion simulation which is relatively less accurate than a simulation using risk population size as the dominant factor in contact formation with a Min.T.years value of 3.26829 instead of 3.1951 in the previous simulation.

Table 5.7 Result generated for the scenario using total population as attraction Factor
(Start Region 1-Ghana)

Solution with Population as the main attraction factor selected start region 1 (Ghana)				
Country	P(O)i	P(E)i	Pi	q i2
1.Ghana	1995	1996	-1	0.1
2.DRC	1991	1996	-5	0.1
3.Zimbabwe	2003	1997	6	0.1
4.Namibia	1995	1996	-1	0.1
5.Rwanda	1991	2000	-9	0.1
6.Malawi	1988	1997	-9	0.1
7.Kenya	2003	2002	1	0.1
8.South Africa	1994	1998	-4	0.1
9.Swaziland	1996	2001	-5	0.1
10.Uganda	1988	1988	0	0.1
11.Burundi	1993	2001	-8	0.1
12.Ethiopia	1996	2001	-5	0.1
13.Eritrea	1995	2000	-5	0.1
14.Botswana	1994	2001	-7	0.1
15.Cent Africa Rep	1989	1991	-2	0.1
16.Zambia	1987	1995	-8	0.1
17.Tanzania	1990	1990	0	0.1
18.Djibouti	1992	2000	-8	0.1
19.Angola	1995	1996	-1	0.1
20.Congo	1983	1985	-2	0.1
21.Mozambique	1996	2001	-5	0.1
22.Lesotho	1997	2002	-5	0.1
23.Equatorial Guinea	1996	1996	0	0.1
24.Gabon	1998	1998	0	0.1
25.Chad	1995	1997	-2	0.1
26.Mauritania	1992	1993	-1	0.1
27.Cote D'Ivoire	1992	1996	-4	0.1
28.Guinea	1996	1996	0	0.1
29.Guinea Bissau	1991	1994	-3	0.1
30.Sudan	1994	1998	-4	0.1
31.Nigeria	1995	1995	0	0.1
32.Sierra Leon	1991	1992	-1	0.1
33.Burkina Faso	1993	1996	-3	0.1
34.Senegal	1991	1995	-4	0.1
35.Gambia	1995	1996	-1	0.1
36.Liberia	1996	1992	4	0.1
37.Mali	1991	1994	-3	0.1
38.Togo	1992	1993	-1	0.1
39.Cameroon	1995	1997	-2	0.1
40.Niger	2002	1998	4	0.1
41.Benin	1995	1995	0	0.1
λ			0.1800	
Min. T.,year			3.2683	

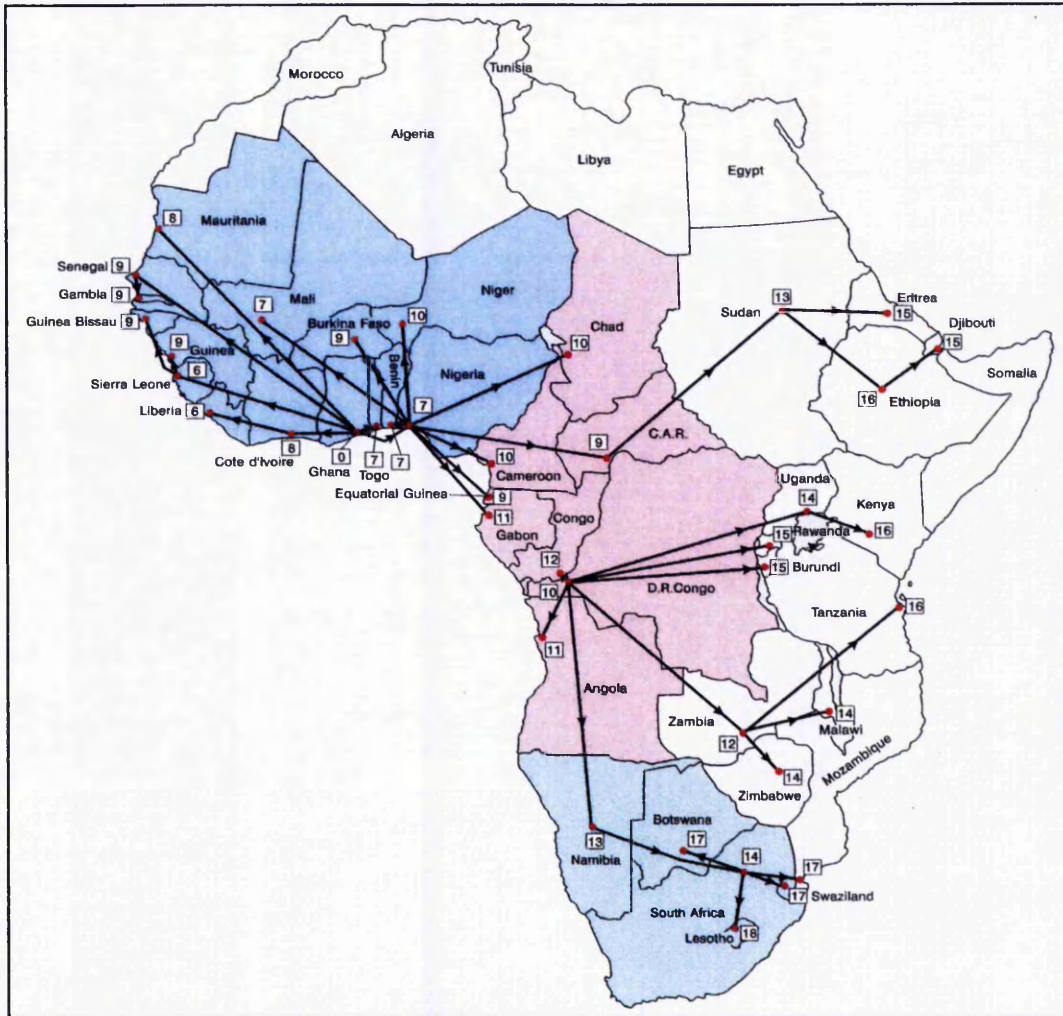


Figure 5.5(A). Initial HIV infection vectors mapped for the scenario utilising total population as attraction factor

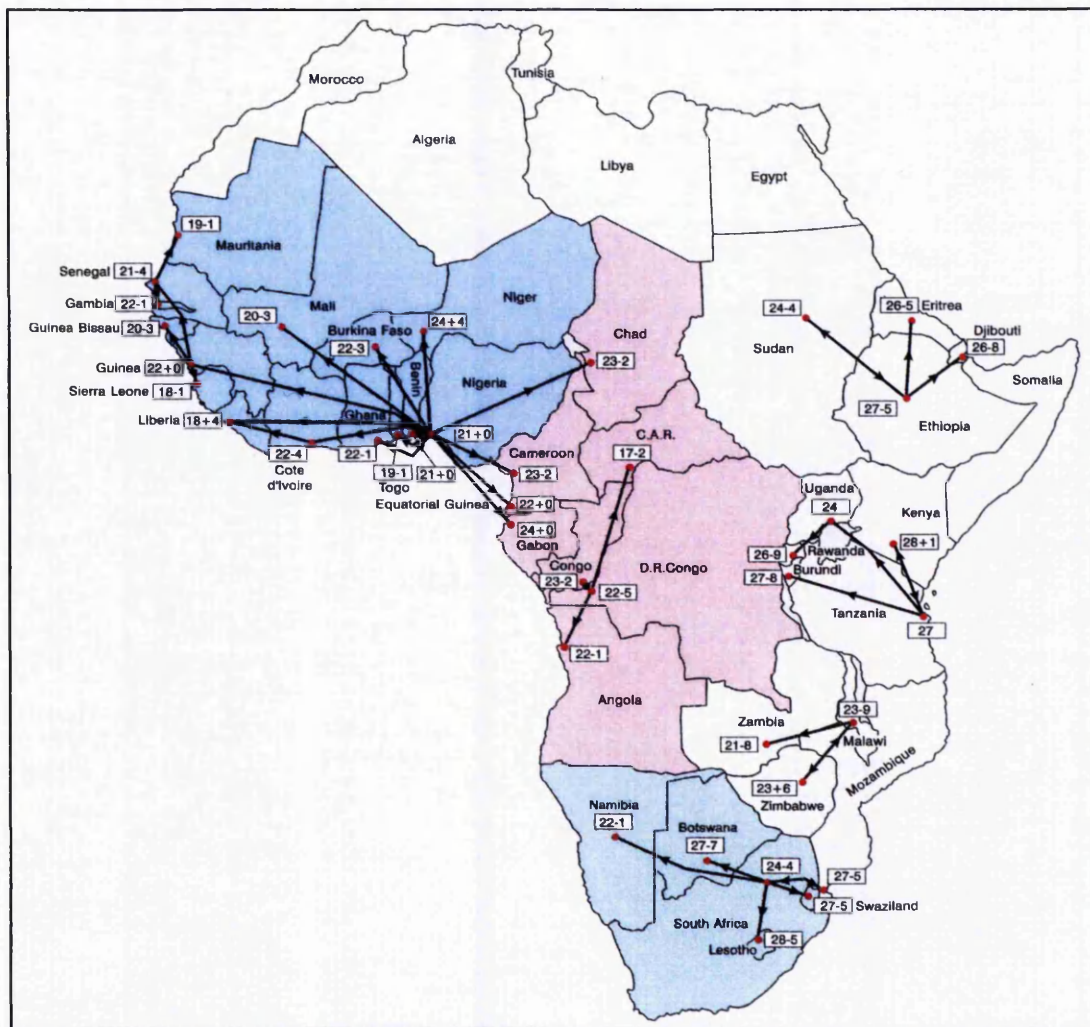


Figure 5.5(B). Infection vectors at HIV peak prevalence for the scenario utilising total population as attraction factor

5.9.3 Multiregion Solution with Epidemic Start In Start Region 1&2(Ghana and D R Congo)

This solution is simulated for an epidemic started both in Ghana and the Democratic Republic of Congo in 1974 simultaneously. Figure 6.6(a) indicates that using total population as the attraction factor changes the infection pathway as well as the speed of the epidemic. In this simulation there is an infection transfer between West Africa and Central Africa unlike the previous simulation using risk population as attraction factor where the pathways of the epidemic clearly differ. Here there is an infection transfer between D R Congo and Nigeria from where the epidemic spread to other West African countries. There is also an infection transfer between Nigeria and Cote

d'Ivoire. Sierra Leone and Liberia receive infection transfer directly from Ghana unlike the previous solution where Ghana infects all countries in West Africa at the start of the epidemic. The infection transfer between D R Congo and the rest of Africa remains the same as the previous solution except that here the Sudan infects Eritrea instead of receiving direct infection from D R Congo as per the previous solution using risk population as the attraction factor. The influence of South Africa in the infection transfer is increased as South Africa infects Botswana, Mozambique, Swaziland and Lesotho unlike the previous simulation where infection transfer is direct from D R Congo to countries in South Africa. In this simulation apart from the two main start regions of D R Congo and Ghana, the influence of Nigeria and South Africa in the infection transfer becomes prominent.

The infection pathway is completed by the 11th year in this simulation as compared to the 10th year in the previous simulation. Even though most countries are infected in the same year, the speed of infection in this simulation is faster in most countries than the previous simulation. For example the epidemic diffused from Ghana to Sierra Leone and Liberia within five years whereas it took seven and eight years in the previous simulation. In the case of Mauritania, Senegal, Gambia, Guinea Bissau and Guinea it took between eight and nine years for the epidemic to diffuse to Nigeria instead of eight years from Ghana in the previous simulation which is relatively shorter in terms of spatial distance. In Central Africa, infection transfer from D R Congo to neighbouring Congo, Central African Republic, Cameroon, Equatorial Guinea and Chad occurred one year earlier than the previous simulation using risk population as the attraction factor. Whereas it took 4 years for the epidemic to spread to Angola from D R Congo in this simulation, it took 7 years in the previous simulation when risk population was used as the attraction factor. It is in South Africa that differences in timing of the epidemic occur. For example Lesotho and Swaziland are infected in the 11th year instead of the 10th and 9th year in the previous simulation.

Table 5.8 indicates that using total population as the attraction factor as opposed to the risk population as an attraction factor increases the speed of the epidemic in many countries. About three-quarters of the countries in sub-Saharan Africa have dates of peak HIV prevalence that are the same or earlier than those predicted in the previous simulation. However, there is a greater change in peak HIV prevalence timing

especially in Zimbabwe with a value of +12. The level of accuracy in this simulation is indicated by the Min. T value of 2.8292 as compared to 2.3170 in the previous simulation.

An alternative representation of the epidemic structure is illustrated in Figure 5.6b. Here the equivalent linkage pattern is mapped at the time of peak HIV prevalence when the source region influence has been largely absorbed into the general system structure. The West African hub shows a major change. Here Nigeria instead of Cote d'Ivoire is the major source of infection at the peak of the epidemic. In Central Africa the influence of D R Congo is reduced as it infects only Angola, Central Africa Republic and Congo as compared to the previous simulation using risk population as attraction factor where D R Congo infects Angola, Namibia, Congo, Gabon, Equatorial Guinea, Cameroon and Central Africa Republic. The Tanzanian hub is still in operation but here the link with Zambia, Zimbabwe, Malawi and Mozambique is curtailed. The Southern hub demonstrates a major change as South Africa becomes the dominant source of infection for Lesotho, Swaziland, Namibia and Botswana instead of Mozambique in the previous simulation.

Table 5.8 shows that the epidemic in sub-Saharan Africa is affected much more when using total population as the attraction factor because of the relative proportions of risk population and total population. Table 5.8 shows that some countries tend to have much lower values for n_i/P because of relatively lower case loads. Using population as an attraction factor in contact formation therefore results in proportionately much more contact formation compared to using risk population and thus moves the epidemic faster while recording no major differences in timing.

Table 5.8. Result generated for the scenario using total population as attraction factor
(Start region 1&2-Ghana and D R Congo)

Solution with Population as the main attraction factor selected start region 1 &2(Ghana and D R Congo)				
Country	P(O)i	P(E)i	Pi	q i2
1.Ghana	1995	1994	1	0.1
2.DRC	1991	1987	4	0.1
3.Zimbabwe	2003	1991	12	0.1
4.Namibia	1995	1991	4	0.1
5.Rwanda	1991	1994	-3	0.1
6.Malawi	1988	1991	-3	0.1
7.Kenya	2003	1996	7	0.1
8.South Africa	1994	1992	2	0.1
9.Swaziland	1996	1995	1	0.1
10.Uganda	1988	1992	-4	0.1
11.Burundi	1993	1995	-2	0.1
12.Ethiopia	1996	1996	0	0.1
13.Eritrea	1995	1995	0	0.1
14.Botswana	1994	1995	-1	0.1
15.Cent Africa Rep	1989	1992	-3	0.1
16.Zambia	1987	1989	-2	0.1
17.Tanzania	1990	1995	-5	0.1
18.Djibouti	1992	1994	-2	0.1
19.Angola	1995	1989	6	0.1
20.Congo	1983	1977	6	0.1
21.Mozambique	1996	1995	1	0.1
22.Lesotho	1997	1996	1	0.1
23.Equatorial Guinea	1996	1992	4	0.1
24.Gabon	1998	1993	5	0.1
25.Chad	1995	1993	2	0.1
26.Mauritania	1992	1992	0	0.1
27.Cote D'Ivoire	1992	1994	-2	0.1
28.Guinea	1996	1995	1	0.1
29.Guinea Bissau	1991	1993	-2	0.1
30.Sudan	1994	1993	1	0.1
31.Nigeria	1995	1992	3	0.1
32.Sierra Leon	1991	1991	0	0.1
33.Burkina Faso	1993	1994	-1	0.1
34.Senegal	1991	1994	-3	0.1
35.Gambia	1995	1995	0	0.1
36.Liberia	1996	1990	6	0.1
37.Mali	1991	1993	-2	0.1
38.Togo	1992	1991	1	0.1
39.Cameroon	1995	1992	3	0.1
40.Niger	2002	1995	7	0.1
41.Benin	1995	1992	3	0.1
λ			0.1800	
Min.T.,years			2.8293	

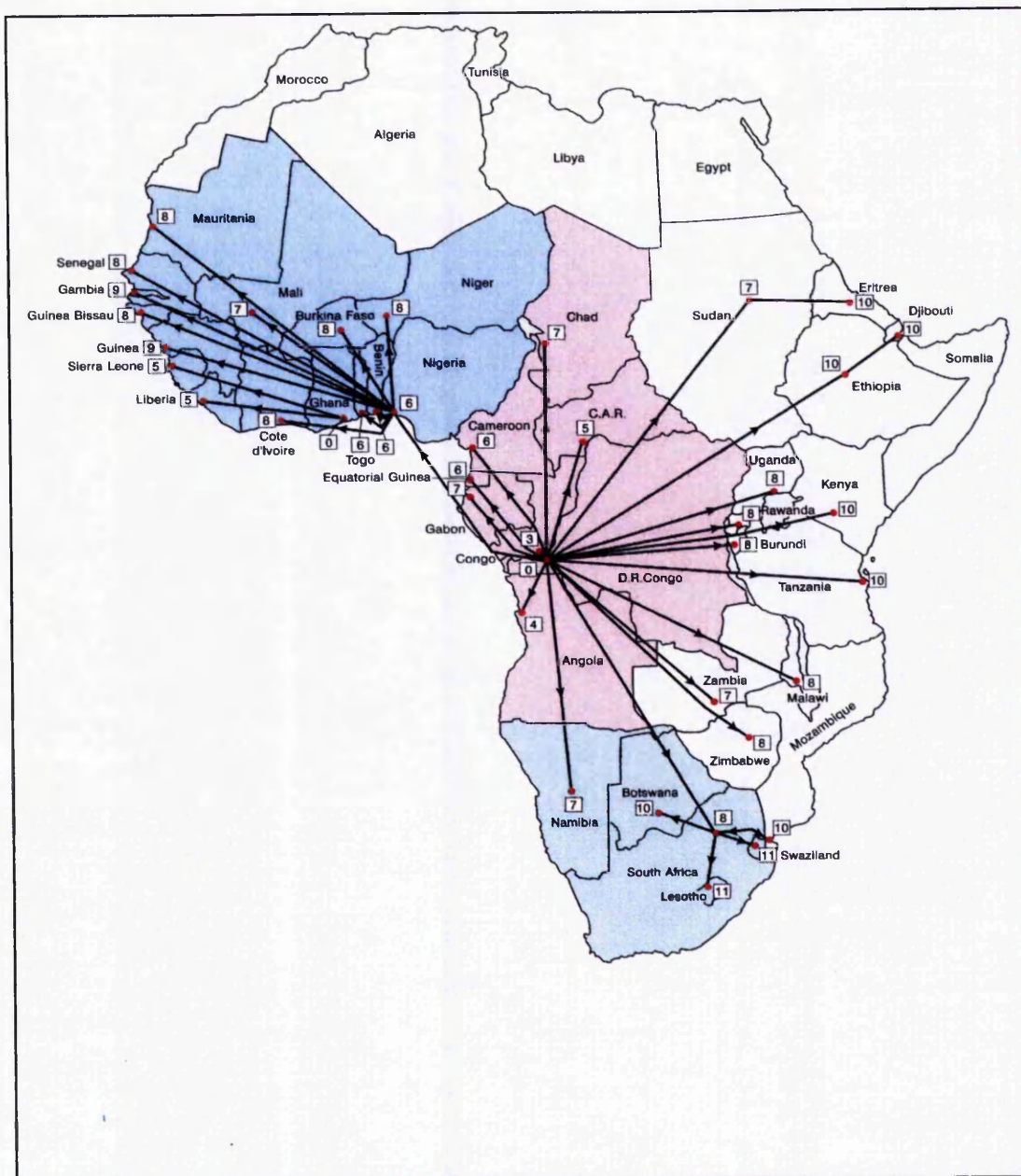


Figure 5.6(a): Initial HIV infection vectors mapped for the scenario utilising total population as attraction factor

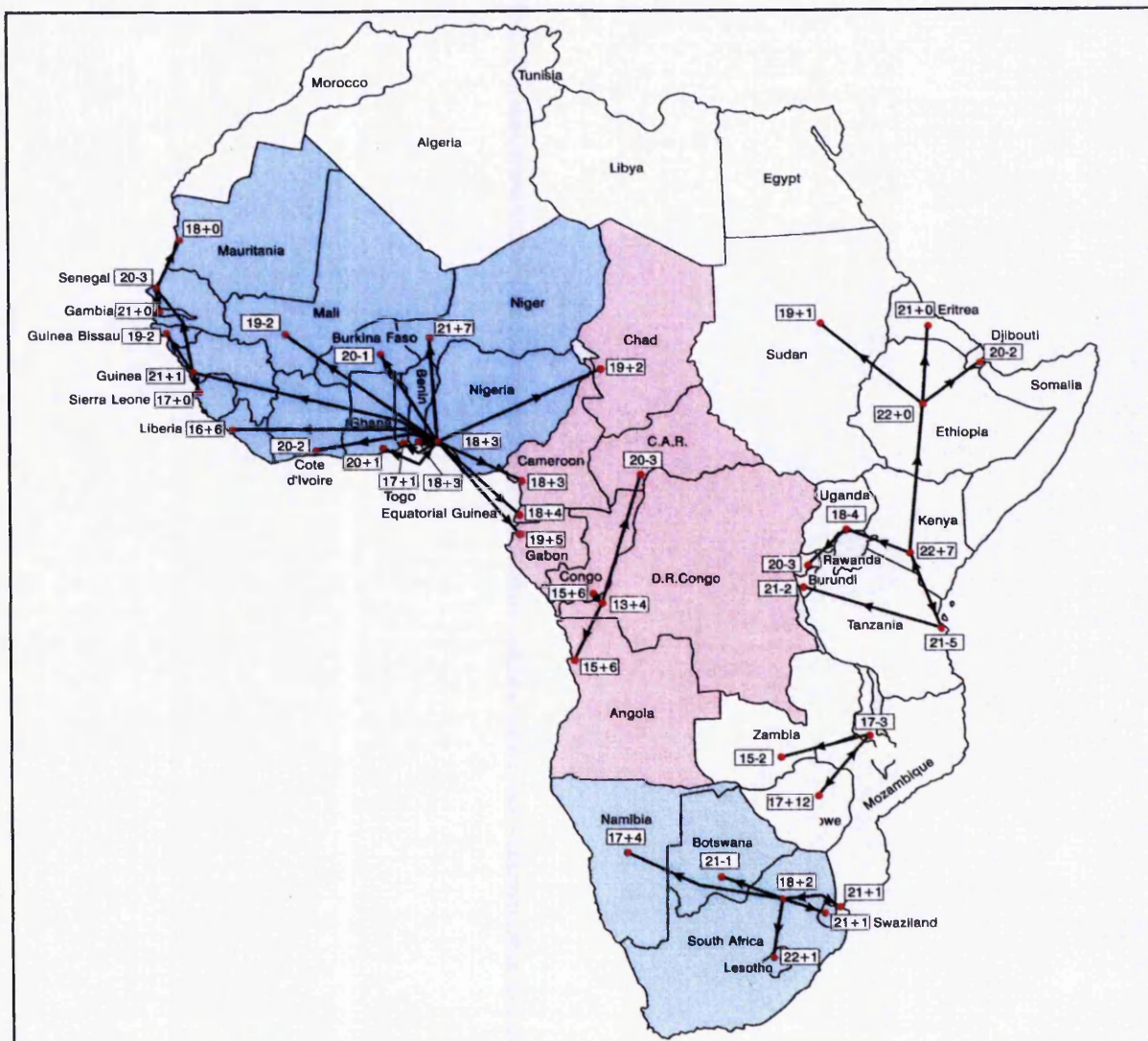


Figure 5.6(B). Infection vectors at HIV peak prevalence for the scenario utilising total population as attraction factor.

Table 5.9 Selected National Data

Country	Size of Risk Population(ni)	Total Population(P)(millions)	Those at risk per million population(ni/P)
1.Ghana	63000	19.73	3193
2.D.R.Congo	95000	52.52	1809
3.Zimbabwe	7500	12.85	584
4.Namibia	41800	1.78	23483
5.Rwanda	27600	7.94	3476
6.Malawi	58000	11.57	5013
7.Kenya	81500	31.29	2605
8.South Africa	26000	43.79	594
9.Swaziland	10000	0.93	10753
10.Uganda	59000	24.02	2456
11.Burundi	31000	6.50	4769
12.Ethiopia	100400	64.45	1558
13.Eritea	11000	3.81	2887
14.Botswana	30000	1.55	19355
15.Cent Africa Republic	8400	3.78	2222
16.Zambia	46500	10.64	4370
17.Tanzania	145000	35.96	4032
18.Djibouti	2400	0.64	3750
19.Angola	10000	13.52	740
20.Congo	41000	3.11	13183
21.Mozambique	70000	18.64	3755
22.Lesotho	35000	2.05	17173
23.Equatorial Guinea	1600	0.47	3404
24.Gabon	10500	1.26	8333
25.Chad	19400	8.13	2386
26.Mauritania	720	2.27	317
27.Cote D'Ivoire	70000	16.34	4284
28.Guinea	13400	8.27	1620
29.Guinea Bissau	1400	1.22	1147
30.Sudan	5000	31.80	157
31.Nigeria	92000	116.92	787
32.Siera Leon	400	4.58	87
33.Burkina Faso	21300	11.85	1797
34.Senegal	3400	9.66	352
35.Gambia	1200	1.33	902
36.Liberia	630	3.10	203
37.Mali	6300	11.67	540
38.Togo	13900	4.65	2989
39.Cameroon	47000	15.20	3092
40.Niger	25000	11.22	2228
41.Benin	7500	6.44	1164

5.10 Discussion

5.10.1 The Magnitude and Directionality of HIV/AIDS Epidemic in Sub-Saharan Africa.

The previous section presents the results of the single region epidemic modelling of HIV/AIDS in Sub-Saharan Africa as well as estimation issues relating to sub-Saharan Africa. It also shows the multiregion models for scenarios where risk population is used as an attraction factor and where total population is used as the attraction factor. In both scenarios the source regions were varied to see the effect on the pathway of infection at the start of the epidemic and at the peak prevalence. This was done with the intention of evaluating the effect of population migration, urbanisation, wars and conflicts and socio-economic factors on the spread of HIV/AIDS in sub-Saharan Africa.

It is evident from the single region model that the HIV/AIDS epidemic is estimated to have started in 1974 in Democratic Republic of Congo and Ghana concurrently. This implies that the epidemic may have developed from two sites and that the sources of the Central Africa epidemic may be Democratic Republic of Congo while that of West Africa might be Ghana. This partly explains the predominance of HIV 1 in Central, Eastern and Southern Africa while HIV2 predominates in West Africa. The long incubation period estimated for HIV implies that AIDS cases recorded in the early 1980s were probably caused by infections during the 1970s as estimated by the single region model. Earlier review of the epidemic surveillance system in Africa suggests that the AIDS epidemic went unnoticed due to the poor surveillance system in sub-Saharan Africa. The calibrated values of the transmission probability obtained from the modelling process for sub-Saharan Africa are high and consistent with a reliable estimate of 0.1 derived in the U S epidemic (May et al, 1989). In a continent where HIV/AIDS is spread mainly heterosexually, such high values for transmission probability partly explains the high incidence and prevalence of the epidemic. It is evident from the calibrated values of the single region model that the date for the forecast endemic state of the epidemic $T=h$ ranges between 2011 and 2031 and that if interventions are not put in place, the pandemic will pose a major developmental challenge in sub-Saharan Africa.

Even though the modelling process has been applied to the countries of Western Europe (Smith, 2003), there are some estimation issues that are specific to the African context. Whereas the epidemic in Western Europe has reached its peak and started to fall and stabilise, in Sub-Saharan Africa, there is no downturn in some countries. There is also evidence of underreporting of caseloads in some countries as well as missing data for countries ravaged with conflicts and wars. However many countries do show recent downturn in observed AIDS incidence just as in Europe. The calibration procedure for the single region model therefore needs to take into consideration such anomalies in choosing disease parameters that fit the African situation.

The multiregion model presented two scenarios for infection transfers within sub-Saharan African countries. The simulated epidemic for both scenarios shows that at the start of the epidemic, the source region influences the infection pathways but at the peak of the epidemic, secondary nodes are established from where the epidemic spreads. For the risk population based scenarios and starting from D R Congo the epidemic spread to all countries in sub-Saharan Africa. However at the peak of the epidemic, Cote d'Ivoire becomes the main source of infection in West Africa while D R Congo continues to infect Central Africa. In East and South Africa, Tanzania becomes the main source of infection transfer. If the epidemic is simulated from the start region Ghana, the directionality of infection transfers changes. Here Ghana infects all countries in West Africa at the start of the epidemic. It then infects D R Congo from where it spreads to Zambia and then redistributes to Southern Africa and East Africa. At the peak of HIV prevalence however the infection transfers remain the same as previously where Cote d'Ivoire infects West Africa while D R Congo, Tanzania and Mozambique infect Central, Eastern and Southern African countries respectively. There is however difference in timing of the speed of the epidemic. For example the peak HIV prevalence occurred after 23 years in Ghana and 18 years in D R Congo when the simulated source region was D R Congo but the peak HIV prevalence occurred one year earlier and five years latter in Ghana and D R Congo respectively when the epidemic was simulated with Ghana as the source region. Similar results occurred when the epidemic was simulated with both Ghana and D R Congo as source regions. Once again the influence of D R Congo and Ghana on the start pathways cannot be overemphasised. At the peak HIV prevalence Cote d'Ivoire,

D R Congo, Tanzania and Mozambique remain a dominant source of infection transfer with minor differences in the time of the peak epidemic.

The infection transfer between Ghana and Cote d'Ivoire as depicted by the various scenarios implies that the epidemic may have started in Ghana and spread to neighbouring Cote d'Ivoire and Nigeria due to the exodus of Ghanaians to neighbouring countries after the collapse of the Ghanaian economy in the early 1970s in search of jobs.

In the case of Central Africa, the epidemic might have started at Democratic Republic of Congo as depicted by the starting pathway and might have spread to East and South Africa. Another possibility might be due to the movement of military troops between Central Africa and East and South Africa during the Central African war, which involved troops from the countries from the sub-region (Smallman-Raynor et al, 1991). It is possible that the returning soldiers from the war spread the disease in their countries of origin after contracting it in the Democratic Republic of Congo. The infection pathway at peak HIV prevalence shows that, Tanzania, Mozambique and South Africa have become main nodes of infection transfer in the sub-region. It is possible that migrant workers to South Africa from the neighbouring countries helped in the spread of the virus to South Africa. It is also worth noting that the role of tourism in the spread of the virus cannot be ruled out, as countries in the sub-region such as South Africa, Tanzania and Kenya are major tourist destination.

Results from the population driven specification on the other hand indicate that there is much more contact formation and that the epidemic moves faster as compared with the scenario where the risk population was used as an attraction factor. It is observed that at peak HIV prevalence, Nigeria replaces Cote d'Ivoire as the dominant source of infection in West Africa from where the epidemic spreads to neighbouring countries. In the early 1970s, the oil boom in Nigeria attracted both skilled and unskilled migrants from all over the sub-region especially Ghana and the virus might have been spread to Nigeria due to the possibility of more contact opportunities. Returned migrants from Nigeria after the economic downturn of the Nigerian economy in the early 1980s infected others in their home country. South Africa on the other hand is the dominant infection node in the southern Africa sub-region at peak HIV

prevalence. The implication is that with the population driven specification, the countries with the large populations' in sub-Saharan Africa are the main source of infection at peak HIV prevalence and in a continent where transmission of the virus is mainly via heterosexual spread, the potential for the epidemic to increase is eminent.

5.10.2 The magnitude and directionality of HIV/AIDS epidemic in Ghana.

This section discusses the course of the epidemic in Ghana based on the results of the single region model output analysed in the previous section. The second part discusses the magnitude of the disease flow between Ghana and other countries indicating the infection vectors involved based on the results of the multiregion model output analysed in the previous section.

Although the first official AIDS case was reported in 1986, HIV infection in Ghana is estimated to have occurred in 1974. The cases of AIDS in the 1980s therefore represent HIV infections in the 1970s or earlier due to the long incubation period of the HIV virus. Moreover the poor epidemic surveillance system in Ghana as noted earlier implies that the epidemic might have gone undetected for a long period of time. For example, not all sicknesses are reported to the hospitals to be treated as a greater number of people patronise traditional healers, herbalist as well as faith healers who are accessible to them in terms of cost and proximity. Also the poor vital registration system in Ghana as reviewed in earlier chapters implies that death as a result of AIDS may go unnoticed.

Result of the single region model shows that by 1995, the AIDS epidemic has peaked in Ghana. However efforts to track prevalence were not instituted until 1990 when the Ministry of Health implemented the national HIV Sentinel Surveillance (HSS) system. It was not until 1994 that an annual HIV sentinel survey was conducted at antenatal care (ANC) clinics for pregnant women (Ministry of Health 2003). This means that the continued spread of the virus may be due to the late implementation of surveillance systems to check the spread of the virus. In the case of Ghana, the transmission probability of 0.08 between infectives and Susceptibles is relatively low as compared to other countries in sub-Saharan Africa and this may partly explain the relatively low prevalence rate as compared to other countries in sub-Saharan Africa. It

is however worth noting that the transmission probability could be very high among risk groups such as prostitutes, mining towns and border towns and in some parts of the country including the Krobo district.

The result of the single region model shows that AIDS would become endemic in Ghana by the year 2022 in a scenario of no cure. This implies that if no cure is found or if effective interventions are not put in place, the epidemic may have a devastating impact on the developmental efforts of the country.

Result of the multiregion model shows that at the start of the epidemic, there is an infection transfer from Ghana to Cote d'Ivoire and Nigeria and to all the countries in the West African sub-Region. However at the peak of the epidemic, Ghana receives infection transfer from Cote d'Ivoire. When the attraction was measured according to the population size of each country, the outcome was a significant redirection of the simulated epidemic route. Here, Nigeria infects Ghana at the peak of the epidemic. The generation at peak prevalence of an infection transfer from Cote d'Ivoire to Ghana and from Nigeria to Ghana is of epidemiological concern about the emergence of this route as a conduit for the transfer of new and established infections. The viability of such a network for the transfer of infections to Ghana is eminent due to recent unrest in Cote d'Ivoire and subsequent movement of refugees across the border to Ghana. Knowing the magnitude of new infections caused by these countries will help policy makers to target specific locations to mitigate the spread of the epidemic. The date for epidemic to be endemic in Ghana and the eventual size of the Ghanaian epidemic must be treated with caution as AIDS incidence continues to be subject to under-reporting.

5.11 Conclusion

The single region modelling output discussed above has shed light on the possible source region of the AIDS epidemic in sub-Saharan Africa, the estimated date for the inception of the epidemic, transmission probability for each country, the risk population and the date for peak prevalence as well as the estimated date the epidemic to become endemic in a scenario of no cure. Although the first cases of AIDS in sub-Saharan Africa were reported in the 1980s, the average estimated date for the

inception of the epidemic was 10 years earlier. Ghana and DR Congo have been predicted as the source region with the epidemic starting in 1974 and if no cure is found, then between 2011 and 2030 the epidemic will be endemic in all countries in sub-Saharan Africa. The multiregion model on the other hand has demonstrated the epidemic pathways at the start of the epidemic and at the peak of the epidemic. Whereas the epidemic started to spread from Ghana and D R Congo to neighbouring countries, at the peak of the epidemic, different nodes have been formed. In West Africa Cote d'Ivoire is the main node for infection transfer to other parts of the sub-region while South Africa, Mozambique and Tanzania have become the main nodes infecting countries in the sub-region. It was noted that with the population driven specification, there was more contact formation and that the epidemic moves faster than the scenario where the risk population was used as an attraction factor. In the population driven specification, countries with the highest population in sub-Saharan Africa are the main node of infection transfer at the peak of the epidemic. Predicting the transmission routes of the epidemic both at the start and peak prevalence and estimating the disease parameters as shown above help planning intervention and control strategies but there is the need to go further to understand the processes of the epidemic spread. The proceeding chapter explores these processes using the Krobo district in Ghana as a case study.

CHAPTER SIX

6.0 THE DYNAMICS OF THE SPREAD of HIV/AIDS IN GHANA: THE KROBO CONTEXT

6.1 Introduction

The previous chapter has modelled the pathways by which HIV/AIDS spread in order to explain observed spatial distributions of the epidemic at national scale. The second part of the thesis explores the socio-economic, demographic, cultural and political contexts at sub-national scale in which the epidemic continues to thrive despite universal awareness of the consequences of AIDS and targeted policies and interventions in sub-Saharan Africa. The Krobo District in Ghana is used as a case study. Review of the Krobo district in Ghana shows that intervention design has accorded prime significance to individual behaviour and cultural influence. However, the critique of the different approaches to explaining the epidemic spread discussed in chapter 3, that is the cultural, political-economic and vulnerability approaches conclude that cultural factors alone are insufficient in understanding the spread of the epidemic at global, national or sub-national scales. This cultural bias translated into early studies and interventions to prevent the spread of the epidemic based on individual behavioural change with scant attention given to the macro-economic and social issues creating a high risk or vulnerable environment for HIV/AIDS spread. Although, a number of factors have been advanced to explain the spatial pattern of HIV/AIDS in Sub-Saharan Africa, that is the civil wars, truck town, and migrant labour hypotheses in terms of its rapid spread, high prevalence and uneven distribution, we are still far from understanding in detail why the epidemic continues to persist.

In order to explore the processes by which the HIV epidemic is spreading, we need to get first hand information from the general population in the Krobo district about what they perceive as the cause of the epidemic spread. Most individuals in the Krobo district will have been affected directly by being HIV positive or indirectly by losing a close relative or a friend to HIV/AIDS or involved in the care for HIV/AIDS patients. Secondly, organisations working on HIV/AIDS treatment and prevention in

the district are in direct contact with HIV/AIDS patients and can provide detailed information about the causes of the continued infection in the district that underpins intervention designs.

In order to obtain this information, both quantitative and qualitative techniques are used. As noted by Finch (1986, pp.157-74 in Brannen, 1992), the combination of qualitative methods with a more traditional quantitative ones in the conduct of policy relevant-research such as this may make a new and valuable contribution. Although the quantitative technique using questionnaire does not tease out the processes as such it involves large sample and thus is representative and extensive, and make broader patterns about the epidemic spread (Bryman, 2004). The qualitative technique based on interviews whilst involving small samples, is intensive and good for experiences, meanings and narratives over time, the so-called rich or thick description (Bryman, 2004). The rationale for the combination of these two approaches according to Fielding and Fielding, (1986 in Brannen, 1992) is to utilise the strengths of both the quantitative and qualitative methods to explore the same problem in order to add breadth and depth to the analysis of the complex epidemiology of HIV/AIDS in sub-Saharan Africa. The quantitative data will be presented first, and then the qualitative detail explains some of the findings of the survey (Brannen, 1992).

6.2 Methodology for the population and organisation based survey of the Krobo district of Ghana.

The study is based on primary data obtained using interviews and semi-structured questionnaires. Two sample groups of informants are involved: the general Krobo population, and organisations working in HIV/AIDS treatment and prevention.

6.2.1 Quantitative data – population survey

A structured questionnaire of open and closed questions was designed to explore the socio-economic, demographic and cultural factors influencing the spread of HIV virus in the Krobo district (See Appendix 9). The questionnaire was drawn base on the

hypothesised relationship between individual sexual behaviour, economic- socio-cultural and individual characteristics and risk environment that result in HIV infection as illustrated in the synthesised framework developed in chapter three (Figure 3.3).

In order to understand the reasons for the continued spread of the epidemic, the extent to which people who know how to protect themselves are actually protecting themselves is explored by testing the relationship between knowledge of HIV prevention methods and the practice of preventive behaviours. Secondly, it has been observed from the theoretical chapter that individual behaviour alone cannot explain the continued spread of the epidemic. Therefore the relationship between economic and political factors and risky sexual behaviour is explored. Lastly the relationship between socioeconomic, demographic, risky sexual behaviour, mobility and perceived risk to HIV infection is explored using multivariate analysis. This will enable us assess the independent association between each variable and perceived risk to HIV/AIDS infection.

The main hypotheses to be tested are:

1. There is no significant relationship between knowledge of HIV prevention methods and the practice of preventive behaviours.
2. There is no significant relationship between economic and political factors and risky sexual behaviour.
3. There is no significant relationship between social and demographic characteristics, mobility, and condom use.

The components of the framework as applied to this study are listed below.

- a) Individual sexual behaviour: the number of sexual partners, sex with casual partners and condom use
- b) Economic and political factors: economic and political situation in the country that may force the population to migrate or to sell sex for livelihood thereby putting themselves in a greater risk to HIV infection such as employment status, level of income.
- c) Cultural factors: stigma, religion, belief in witchcraft and *dipo* custom (puberty rites) among the Krobo people in Ghana.

- d) Socio-demographic: individual characteristics such as gender, age, marital status
- e) Migration: migration variable such as, ever travelled outside Ghana, have been absent from current address for several days in a month and have you ever travelled to Cote d'Ivoire.

Drawing on the themes in the framework as defined above, the questionnaire was structured in five sections.

i) Section one covers the socio-economic and demographic characteristics of the respondents namely age, gender, marital status, income, educational status, employment status and religion. Socio-economic and demographic characteristics of the respondents are then examined in an attempt to understand the social, economic and demographic processes underlying sexual behaviour and hence the spread of HIV/AIDS.

ii) The second part of the questionnaire deals with knowledge, attitude and perception about HIV. Respondents were asked about the source of HIV virus, method of transmission and prevention, risk perception as well as belief relating to origin and aetiology of HIV. Stigma, fear and ignorance are also explored. This section analyses social and community construction of the epidemic and how this makes them vulnerable to the epidemic.

iii) The third section of the questionnaire addresses the issue of migration both internal and external and how the process of migration creates an enabling environment for the spread of HIV. Respondents were asked whether they have ever travelled outside Ghana and how regular they have been on trips where they have to stay away for several days. Particular emphasis was laid on those who have travelled to Cote d'Ivoire one of the countries with the highest prevalence of HIV in the West African sub-region. The relationship between migration characteristics of respondents and the level of risk perception was examined to find out the extent of migrants vulnerability to HIV infection.

iv) The next section addresses the behavioural change. The respondents were asked about the level of condoms use, the extent to which condoms are safe in preventing HIV infection. Respondents were also asked whether they have less sexual partners at present as compared to some years ago. In this section the extent to which sexual behaviour has changed was explored.

v) The final part of the questionnaire assesses respondents risk perception about HIV/AIDS. Respondents were asked whether they are at risk of infection and if so at what level.

6.2.2 Sample Selection

A stratified sampling design was used to select the respondents for the questionnaire. In all, 500 questionnaires were administered. This was composed of 100 for each of the five main regions within the Krobo district: Somanya; Agormanya; Odumasi; Nkurakan; Asesawa. The electoral register was used to randomly select the 100 respondents from each region. Somanya, Agormanya and Odumasi are urban centres while Nkurakan and Asesewa are rural areas. This means that out of the sample size of 500, sixty per cent was chosen from the urban area while the remaining forty percent was chosen from the rural area. This reflects the proportion of urban to rural residents in the total Krobo district population.

6.2.3 Analysis

The analysis is in two parts. The quantitative aspect of the study on the other hand involves coding and analysing the questionnaire using SPSS. The dependent variable in this analysis was perceived risk of HIV infection. Five groups of independent variables were examined: socio-demographic characteristics, HIV/AIDS-related knowledge and attitudes, HIV/AIDS-related sexual behaviour, culture and migration history. The socio-demographic variables examined were age, gender, marital status, education, employment status, income and religion. The knowledge and attitude variables examined include: familiarity with AIDS, knowledge of HIV transmission methods, and knowledge about how to stop the spread of AIDS. The sexual behaviour variables that were examined were age first had sex, age first married, having regular sex partner, having more than one sexual partner, condom usage, and sexually transmitted infections. The cultural variables examined were early initiation into puberty rites (*dipo*) and the rituals involved such as scarification and parading of semi-naked girls on the street. Lastly migration variables examined were, where living before moving to Krobo district, ever travelled outside Ghana and which country travelled to. Bivariate analysis is conducted to examine the relationship between the

all the variables and the perception of risk. To determine statistical significance, χ^2 tests were used and their associated P values ($p < .05$ considered statistically significant) are reported.

Multivariate analysis is carried out to examine the nature and strength of the association between the socio-demographic, knowledge and attitude, sexual behaviour, cultural and migration variables as listed above and risk perception. Because the dependent variable of risk perception is dichotomous where 1 denotes at risk of infection and 0 not at risk of infection, logistic regression is used. The overall fit of the model to the data is assessed by the maximum log likelihood statistics. The logistics coefficients are presented as odds ratios, which gives an estimate of the magnitude of the association between the variables being compared. The P values are calculated to identify the associations that are significant ($p < .05$ was considered statistically significant). Views from the qualitative analysis will support and complement the views from the quantitative analysis for a broader perspective in understanding the epidemiology of HIV/AIDS in sub-Saharan Africa.

6.2.4 Qualitative data – interviews with HIV/AIDS service organisations

The survey collected information from health personnel, non-governmental organisations working with HIV/AIDS issues, faith-based organisations, and government representative in-charge of HIV/AIDS in the study area (Krobo district in Ghana). For the full interview transcript, see appendix 8. The main themes of the interview include issues relating to the prevalence of HIV/AIDS, knowledge and belief of their clients about HIV/AIDS, the role of cultural beliefs and the spread of HIV/AIDS, faith-based organisations and the spread of HIV/AIDS, stigmatisation and the spread of HIV/AIDS, prevention of HIV/AIDS, antiretroviral treatment, the role of the government in HIV/AIDS prevention, the challenges they face in the course of their work, the socio-economic background of people living with HIV/AIDS and migration history of their clients. The rationale behind the selection of the sample population to be interviewed is to help obtain diverse views from different stakeholders. This will help to understand the current state of the epidemic and the

driving force behind the epidemic spread in order to formulate relevant policies to mitigate the spread.

In order to examine the epidemiology of HIV/AIDS from a broader perspective, a representative each of five main organisations working on HIV/AIDS projects in the Krobo District were interviewed. The choice of these organisations was based on the fact that they were locally based and managed by indigenous Krobos and has the local knowledge about the HIV epidemic and was readily available for the interview. Besides they represent the broad spectrum of HIV/AIDS service organisation in the area of operation.

- i) 'Friends of the sick' is a catholic based voluntary organisation whose main aim is to visit sick people in the community and in the process identifies HIV/AIDS positive persons. They provide pastoral care and support and recommend them to the appropriate agencies for help. Their support and care is mainly home-based. The catechist of the local Catholic Church was interviewed.
- ii) The Atua Government Hospital AIDS coordinator was interviewed. The main role of the AIDS coordinator is to provide voluntary counselling and testing for HIV/AIDS positive patients and to provide them with Antiretroviral treatment and to make follow up visits at their homes to monitor the treatment and their progress.
- iii) The St. Martins catholic hospital at Agormanya HIV/AIDS counsellor was interviewed. The major role of the counsellor is trace returned migrants who were mostly prostitutes from Cote d'Ivoire from the slum area of the district and provide free counselling and testing and to administer antiretroviral drugs to them. Here the HIV/AIDS positive patients have formed an association who meet at the hospital to receive treatment in return for sharing their experience to out patients at the hospital as a way of creating awareness to the epidemic.
- iv) The District Focal Person based at the Manya Krobo District Administration was interviewed. His main duty is to monitor and coordinate the activities of Non Governmental Organisations (NGOs), Faith-Based Organisations (FBOs) and Community-Based Organisations (CBOs) working on HIV/AIDS related project in the district.
- v) The director of Youngsters International Development Programme (CYINDEP), an NGO was interviewed. The main role of CYINDEP is capacity building for the youth,

peer education, prevention and support for People Living With HIV/AIDS (PLWHA).
(See table 6)

Table 6.1 Selected HIV/AIDS service organisations in the Krobo district.

Organisation	Sector	Services	Clients	Location
Friends of the sick	NGO	Visit sick people and in the process identify AIDS patients, offer pastoral care and support to PLWHA	PLWH A	Agormanya
Centre of Youngsters Integrated Development Programme	NGO	Peer education, behaviour change, condom use, reducing risk of STD and HIV/AIDS infection	Youth	Agormanya
Manya District Assembly	Government	Monitoring and evaluation of NGO's CBO's FBO's providing report and statistics on the epidemic in the district.	NGO's CBO's FBO's	Odumase
Atua Government Hospital	Government	VCT, Provision of Anti retroviral therapy, support for PLWHA, behaviour change, condom use,	PLWH A	Somanya/Atua
St Martin Hospital	Church	VCT, Provision of Anti retroviral therapy, home-based care for PLWHA, behaviour change, condom use	PLWH A	Agormanya

6.3 Issues from the Fieldwork

The fieldwork started on 6th January and ended at 28th March. In order to be granted permission to carry on the fieldwork, the local assembly member who is the government representative of the various communities was consulted who in turn informed the community of the ongoing research and requested the communities to cooperate. All the 500 questionnaire administered were returned although some were

not fully completed. Respondents who did not complete the questionnaire were contacted a second time and this resulted in 100 percent response rate. Factors affecting data collection included the schedule of some respondents, attitude of respondents towards researchers, attitude towards sexual issues and HIV/AIDS and the time and date for the interview.

In the first place most of the respondents in the rural areas such as Nkurakan and Asesawa were farmers. This means that early in the morning they go to their farms and return in the evening between 4pm and 6pm. By this time they have to prepare their evening meals and were in most cases tired and were not willing to answer the questions. The weekends happen to be a market day in the case of Nkurakan where the farmers sell their produce in the market. This scenario delayed the data collection since respondents have to be visited on several occasions before they answered the questions. Five percent of the respondents who were not available at the time of the survey were replaced by the next name on the voter register as recommended by Bryman (2004). This in no way affected the result of the survey. In Somanya, Agormanya and Odumase Krobo, the situation was different. Here most people were petty traders while others were unemployed and were mostly available at home but once again some were reluctant to respond to the questions. This was in part because they were fed up with answering questionnaire; since the 1980s, the area has been the focus of many students and academics researching HIV/AIDS. Most of the respondents complained that they have been answering questions on HIV/AIDS for the past years but they have not benefited from any such research and that they continue to be poor and the HIV/AIDS situation has not changed or is getting worse. This reaction did not come as a surprise because the district recorded the first case of HIV/AIDS in Ghana and has been the region with the highest HIV/AIDS incidence and was the first district where HIV/AIDS research began. For example, the first home-based HIV/AIDS care was established in St Martins Hospital in Agormanya in 1988. The district has therefore been exposed to researchers from all over the world. However, this particular obstacle was overcome by the researcher's familiarity of the local customs and fluency in the local language which enabled the establishment of some rapport with respondents and facilitated their cooperation.

Another issue that emerged from the fieldwork was that some married women have to seek the consent of their husbands before answering questions. For example, one female respondent had to abandon the interview when the husband interrupted during the middle of the interview and demanded she start cooking the household meal and deal with the questionnaire later. Questions regarding sexual activities such as the age of first sex and how many partners have you apart from current partner, and did you use condom the last time you had sex are sensitive and respondents look over their shoulders before they respond. This problem arises because the people live in compound houses with more than five households in one house and as such there is no privacy and respondents would not like their neighbours or other household members to know anything concerning their sex life. It is also not permissible to have the interviews indoor especially with women who are married because of suspicion about infidelity. Whereas the men were able to discuss openly their sexual life, women were shy and reluctant to discuss sexual matters especially when their partners were at home. As noted by Edwards (1990), the responses from women interviewees are shaped by different social structures than those from men. Despite the limitations that these issues impose on the research, the necessary responses needed for the study were achieved but it is necessary to be aware of their possible influence and how they may modify the final results (Plumer, 1991).

The next two chapters present the results of the population and organisation based studies in the Krobo district. The quantitative data are presented in chapter 7 and the qualitative in chapter 8.

CHAPTER SEVEN

7.0 QUANTITATIVE ANALYSIS OF HIV/AIDS SPREAD IN THE KROBO DISTRICT: POPULATION BASED SURVEY

This chapter presents the results of the population-based survey in the Krobo district to enable us explore the reasons why the epidemic continues to spread despite universal awareness of the consequences of AIDS. First, the results of the DHS (2003) are presented to show the relationship between socio-demographic characteristics of the respondents and HIV serostatus in Ghana. This serves as background information to enable us to explore the relationship between socio-economic and demographic characteristics of the Krobo population and risky sexual behaviour that exposes them to HIV infection. Secondly, the survey sample is presented to explore if the same findings recur in relation to risky sexual behaviour. Thirdly in order to understand the reasons for the continued spread of the epidemic, the extent to which people who know how to protect themselves are actually protecting themselves is explored by testing the relationship between knowledge of HIV prevention method and the practice of preventive behaviours. Fourthly, as has been observed from the theoretical chapter that individual behaviour alone cannot explain the continued spread of the epidemic, the relationship between economic and political factors and risky sexual behaviour is explored. Fifthly in order to understand the role of out-migration from Ghana in the observed pattern of the epidemic spread as shown in the modelling output, migrant destination countries from the Krobo district are explored. Lastly, the relationship between social, demographic, risk perception, mobility and condom use is explored using multivariate analysis. This will enable us assess the independent association between each variable and condom use.

7.1 Populations at risk: DHS (2003) survey findings

The 2003 Ghana Demographic and health Survey (2003GDHS) report presented the result of HIV prevalence by background characteristics namely age, marital status, place of residence, education, employment, wealth index and mobility for all respondents who were tested for HIV (GSS, 2004). The result shows that among sexually experienced women and men, 3.2 percent of women aged 15-49 and 2.1 percent of men aged 15-59 tested positive for HIV.

Among sexually experienced women and men, age, marital status, wealth index (women only) and region of residence (women only) are the only socio-demographic variables that are significantly associated with HIV serostatus (GSS, 2004). The proportion of women and men infected with HIV is not evenly distributed by age group. For women, it peaks at age 35-39 and for men age 40-44.

It was also observed that formerly married women and men in the 2003 GDHS had the highest rates of HIV infection (GSS, 2004). It is likely that some of the formerly married respondents were AIDS widows and widowers. Another possibility is that among women being formerly married because of death or divorce increases their vulnerability to HIV infection by placing them in positions where they may be sexually exploited.

Wealth and region of residence were significantly associated with HIV serostatus for women. Women in the lower income households were most likely to be HIV positive and a greater proportion of women living in the Eastern region of Ghana (5.0 percent) were HIV positive. Antenatal care (ANC) sentinel surveillance data supports these findings especially in the Krobo district of the Eastern region where HIV prevalence among ANC attendees at St Martin's Hospital at Agormanya was 11.4 percent for women aged 15-24 (NACP, 2002).

Other factors that were significant were sexual debut at an early age and mobility. It was observed that sexual debut at an early age (15 years) is strongly associated with HIV infection among women. Among men mobility was found to be a significant

factor for HIV infection. The DHS 2003 result further shows that though there is a high level of ABC related knowledge in Ghana, (abstinence, be faithful to one uninfected partner, condom use) there is little translation of that knowledge into behaviour (GSS, 2004). Among women, knowledge of partner reduction was the only ABC-related knowledge to be associated with being HIV negative (GSS, 2004). The next section presents the survey sample of the Krobo district to explore if the same findings recur regarding risky sexual behaviour.

7.2 The Demographic and Socio-Economic Profile of Respondents.

The distribution of the sample population by age and sex is presented in Table 7.1. The proportion of men in the sample is relatively bigger than women but on the whole the sample is adequately representative gender wise with men and women constituting 52 percent and 48 percent respectively. In terms of age group, the sexually active population between the ages of 15-29 years constitute 50 percent of the sample.

Table 7.1 Age-Sex structure of the respondents

Age	Frequency		Total	Percent
	Male	Female		
15-19	25	44	69	14
20-24	44	53	97	19
25-29	39	47	86	17
30-34	26	26	52	11
35-39	29	32	61	12
40-44	34	16	50	10
45-49	28	12	40	8
50-54	25	10	35	7
55-59	8	2	10	2
Total	258(52%)	242(48%)	500	100

Marital status of the respondents is presented in Table 7.2. The proportion of unmarried people in the sample is bigger than that of married and, in terms of gender; more females remain unmarried as compared with their male counterparts. The unmarried people comprise people who had never married, and people who are divorced or widowed. The higher proportion of the unmarried population may be attributed to an exceptionally high bride price in the Krobo district. It is also possible that some of the unmarried people have lost their partners to HIV/AIDS in a district with the highest regional rate in Ghana well above the national rate of 3.6 percent (NACP, 2002). In terms of gender, the relatively higher proportion of unmarried females may be due to the fact that men are more affected by early death from HIV and the fact that in Ghana as in other countries in the world, women outlive men with life expectancy for women and men at 59 years and 56 years respectively (GSS, 2004).

Table 7.2 Marital Status by Gender of Respondents

Marital Status	Frequency		Total
	Male	Female	
Married	115 (57.8%)	84(42.2%)	199(39.8)
Unmarried	143(47.5)	158(52.5)	301(60.2)
Total	258(51.6)	242(48.4)	500

Table 7.3 summarises education level of the sample. Twenty-five percent of the respondents have no formal education. Of those who have received some form of formal education, 35 percent had either middle or junior secondary education. People with such a low level of education remain vulnerable because their capacity to respond effectively to the health threat posed by HIV is reduced (Colvin et al, 2001). This proportion compares with the result of the 2003 DHS survey. It was observed from the GDHS (2003) report that 3.9 percent of those with primary education, 3.5 percent of those with secondary education and 2.3 percent of those with no education tested HIV positive. This means that those with primary education were the vulnerable group for HIV infection in Ghana. The proportion with formal education decline from 29 percent for secondary education to 9.6 percent for tertiary education. Thus the drop in attendance at an educational facility is between middle school and secondary school for females and secondary to tertiary for both male and females. This may be partly due to the fact that among those who are enrolled at school a large number of them do not attend school regularly especially in the farming season when they contribute to family labour. Also walking distance to school is so long in the rural areas, a major limitation in improving access to education.

In terms of gender, as expected, more men attained secondary and tertiary education than women. This result is comparable with the result of the 1998 Demographic and Health Survey which shows that the percentage of males and females attending school was roughly the same until the age of 15 when female attendance dropped off faster than male attendance. This means that more women drop out of school earlier than men for many reasons including teenage pregnancy, early marriage, preference of male education over women when family resources are limited and the traditional notion that women's role in society is taking care of the home and the children (Awusabo-Asare, Abane and Kumi-Kyereme, 2004). This position of women means that they remain at the bottom of the socio-economic ladder and thereby increases their risk to HIV infection as compared with their male counterparts.

Table 7.3 Educational level by gender of respondents

Educational level	Frequency		Total
	Male	Female	
None	61 (48%)	66 (52%)	127 (25.4%)
Primary/Middle/Jnr	69 (38.5)	110 (61.5)	179 (35.8%)
Secondary	93 (63.7)	5 (36.3)	146 (29.2)
Tertiary	35 (72.9)	13 (27.1)	48 (9.6%)
Total	258 (51.6%)	242 (48.4%)	500

The survey results show that 65.2 percent of the respondents were unemployed as summarised in Table 7.4. Majority of those who classified themselves as unemployed were subsistence farmers and petty traders who earn less than 10 US dollars a month. Of those who were in employment, there were more males than female. Where as the males were mainly teachers and textile factory workers and auto-mechanics, the women were traders, hair dressers and seamstresses. The higher percentage of

unemployed among the sample especially women increases the risk to HIV infection as women and girls may exchange sex for income or other assets.

Table 7.4 Employment Status and Gender

Employment status	Frequency		Total
	Male	Female	
Unemployed	166(50.9%)	160(49.1%)	3269(65.2%)
Employed	92(52.9%)	82(47.1%)	174(34.8%)
Total	258(51.6)	242(48.4)	500

Income levels in the Krobo district are very low. The survey found that about 10 percent of the Krobos earn less than 10 dollars per month, and only 14 percent earn between 121- 232 dollars per month. The average Krobo is generally poor and this is in line with the general economic situation as summarised in Table 7.5. The District Development Plan of 2003 estimated the average per capita income in the district at \$112 per annum indicating an average income of less than one U.S dollar a day amongst the Krobo (Yilo Krobo District Administration, 2003). The estimate of the survey is in keeping with significantly lower incomes as indicated by the District estimates of less than a dollar a day. Such a low income may make the population vulnerable to HIV infection especially among females who may exchange sex for income or other assets.

Table 7.5 estimated monthly income in Ghanaian cedis * gender Cross tabulation

Variable			gender		Total
			female	male	
estimated monthly income	under 100,000 cedis	Count	28	10	38
		% of Total	7.6%	2.7%	10.3%
	100,000-1,000,000cedis	Count	126	154	280
		% of Total	34.1%	41.6%	75.7%
	1,100,000-2,100,000cedis	Count	19	33	52
		% of Total	5.1%	8.9%	14.1%
Total		Count	173	197	370
		% of Total	46.8%	53.2%	100.0%

NB: exchange rate 9,044 Ghanaian cedis =\$1

Although the survey of the Krobo district is on a smaller scale due to the smaller sample size as compared to the DHS, 2003, the sample is representative of the general population of the Krobo district in terms of gender and age. Whereas male to female ratio in the general population in Ghana is 49.5: 50.5 the sample population was slightly different with male to female ratio of 52: 48.

The results depict a large out of school population in the Krobo district, a factor which may be facilitating the rapid spread of HIV (Hargreaves and Glynn, 2002). About 25 percent have not had any formal education and those who attended primary school and middle school constitute 35.8 percent and are in most neither cases functionally illiterate since most of them cannot read nor write. This means that together about 60 percent of the respondents comprising those with no formal education, those with primary and middle education remain vulnerable because their capacity to respond effectively to the health threat posed by HIV is reduced (Colvin et al, 2001).

In the second place the result shows that there is high unemployment in the Krobo district with a very low level of income. The average per capita income of about 112 US dollars per annum with the average Krobo living on less than 1 US dollar per day. In such a situation of dire poverty, the risk of HIV infection would not be the main concern of the people since some people may exchange sex for survival irrespective of the risk level. Also Buve et al (2002) observed that poor people might have less

access to health care and condom use and hence increase their level of vulnerability to HIV infection.

7.3 Knowledge of HIV prevention methods and the practice of preventive behaviours.

General awareness of HIV/AIDS in Ghana is high. Result from the 2003 DHS shows that more than 98 percent of all respondents have heard of /HIVAIDS. About 37 percent of women and 38 percent of men know someone with AIDS (GSS, 2004). The ABC prevention methods are promoted in Ghana to reduce the risk to HIV infection and knowledge of these prevention methods is quite high among the general population. It was observed from the 2003 DHS that 79 percent of women and 83 percent of men know that abstinence can reduce the risk to HIV infection. Eighty-six percent of women and 90 percent of men know that being faithful to one uninfected partner reduces the risk of HIV infection and 73 and 82 percent respectively know that use of condoms is a way to reduce the risk of HIV infection (GSS, 2004). Despite the universal awareness of AIDS and knowledge about the preventive methods, the epidemic continues to spread. This section therefore explores the extent to which the knowledge about prevention is translated into preventive behaviour. The hypothesis that there is no significant relationship between knowledge of HIV prevention methods and the practice of preventive behaviours is tested.

7.3.1 Awareness of HIV and AIDS`

In order to establish the relationship between HIV prevention method and preventive behaviour, there is the need to find out the level of awareness of the people in the Krobo region about HIV/AIDS. To do this the respondents were asked whether they have heard of AIDS, the source of their AIDS knowledge, whether they know someone with AIDS or who has died of AIDS and the extent to which they know the main mode of spread of the virus. Results from the survey shows that awareness of HIV/AIDS is very high among the Krobos (see Table 7.6) and is in line with the high awareness nationally reported in the DHS survey, 2003. About 98 percent of the sample population have heard about HIV/AIDS. The awareness is high among both

males and females and the main source of information about the epidemic has been through radio, television and word of mouth (see Figure 7.1). It was also observed that 73.6 percent of the sample population knows someone with AIDS or someone who has died of AIDS. Thirty seven percent of female and 38 percent of male in the 2003 DHS survey know someone with AIDS or has died of AIDS (GSS et al, 2004) and this compares with 34.6 percent of females and 39.0 percent of males in the sample population in the Krobo district. Knowledge about the mode of spread of the epidemic was also very high. Figure 7.3 show that more than 80 percent of the sample population mention sexual intercourse as the main mode of the spread of the HIV virus. The above result shows that awareness of HIV/AIDS is very high among the Krobos yet the district has the highest HIV prevalence in Ghana (NACP, 2005). The next section explores the extent of knowledge about preventive methods and how these translate into preventative behaviour.

Table 7.6 ever heard of HIV/AIDS * gender Cross tabulation

Variable			gender		Total
			female	male	
ever heard of HIV/AIDS	yes	Count	240	251	491
		% within ever heard of HIV/AIDS	48.9%	51.1%	100.0%
		% of Total	48.0%	50.2%	98.2%
	no	Count	2	7	9
		% within ever heard of HIV/AIDS	22.2%	77.8%	100.0%
		% of Total	.4%	1.4%	1.8%
	Total		Count	242	258
		% within ever heard of HIV/AIDS	48.4%	51.6%	100.0%
		% of Total	48.4%	51.6%	100.0%

Figure 7.1 Source of AIDS Knowledge

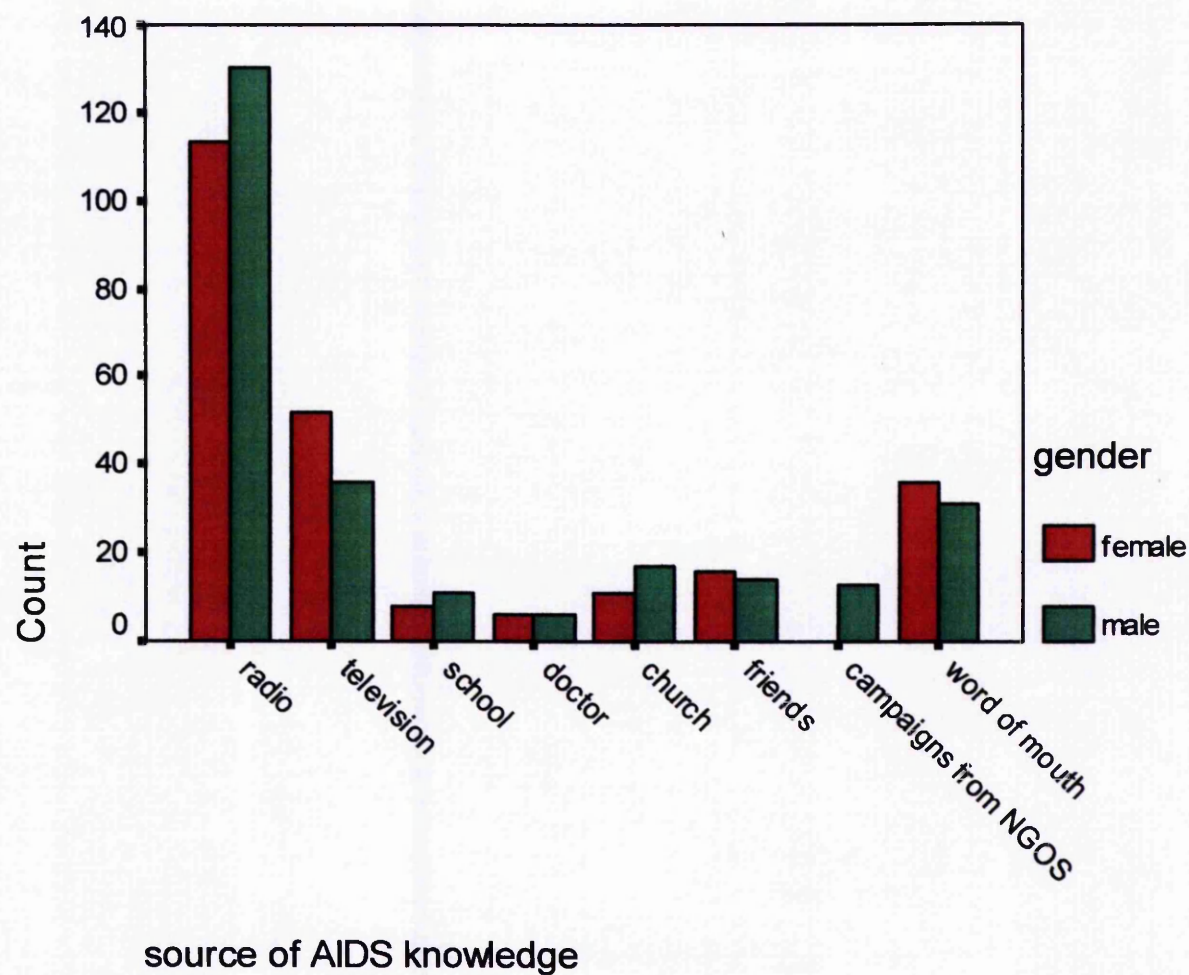
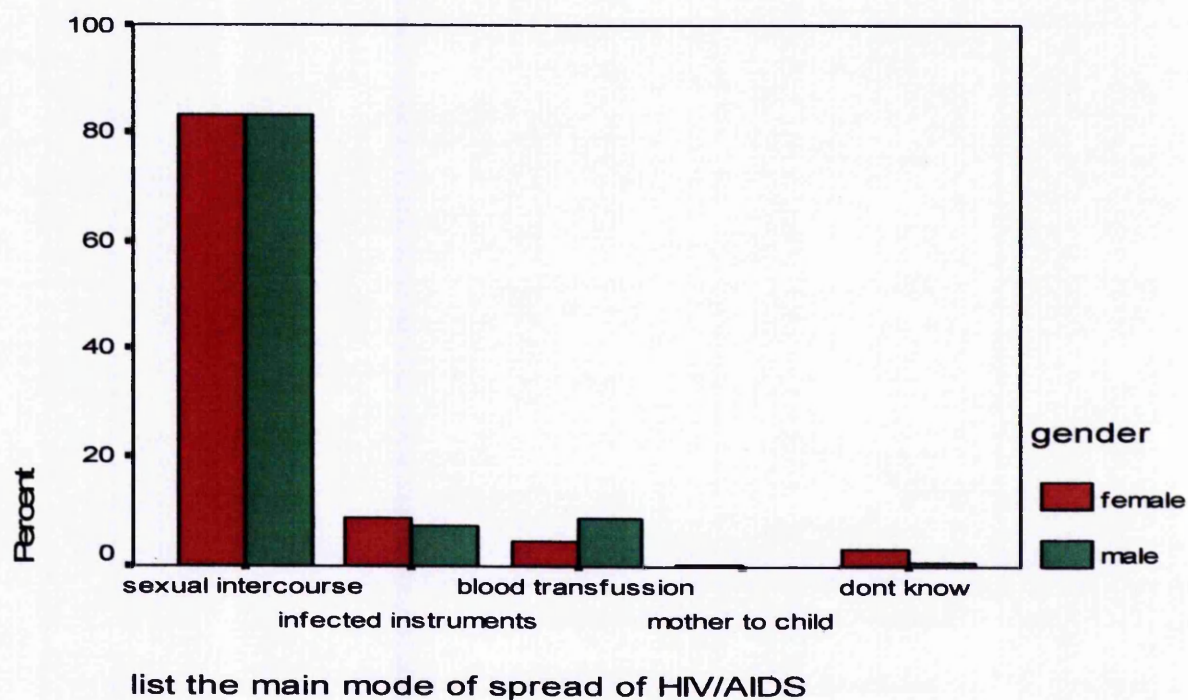


Table 7.7 do you know someone with AIDS or who has died of AIDS * gender Cross tabulation

Variable			gender		Total
			female	male	
do you know PLWHA	yes	Count	173	195	368
		% within do you know someone with AIDS or who has died of AIDS	47.0%	53.0%	100.0%
		% of Total	34.6%	39.0%	73.6%
	no	Count	69	63	132
		% within do you know someone with AIDS or who has died of AIDS	52.3%	47.7%	100.0%
		% of Total	13.8%	12.6%	26.4%
Total		Count	242	258	500
		% within do you know someone with AIDS or who has died of AIDS	48.4%	51.6%	100.0%
		% of Total	48.4%	51.6%	100.0%

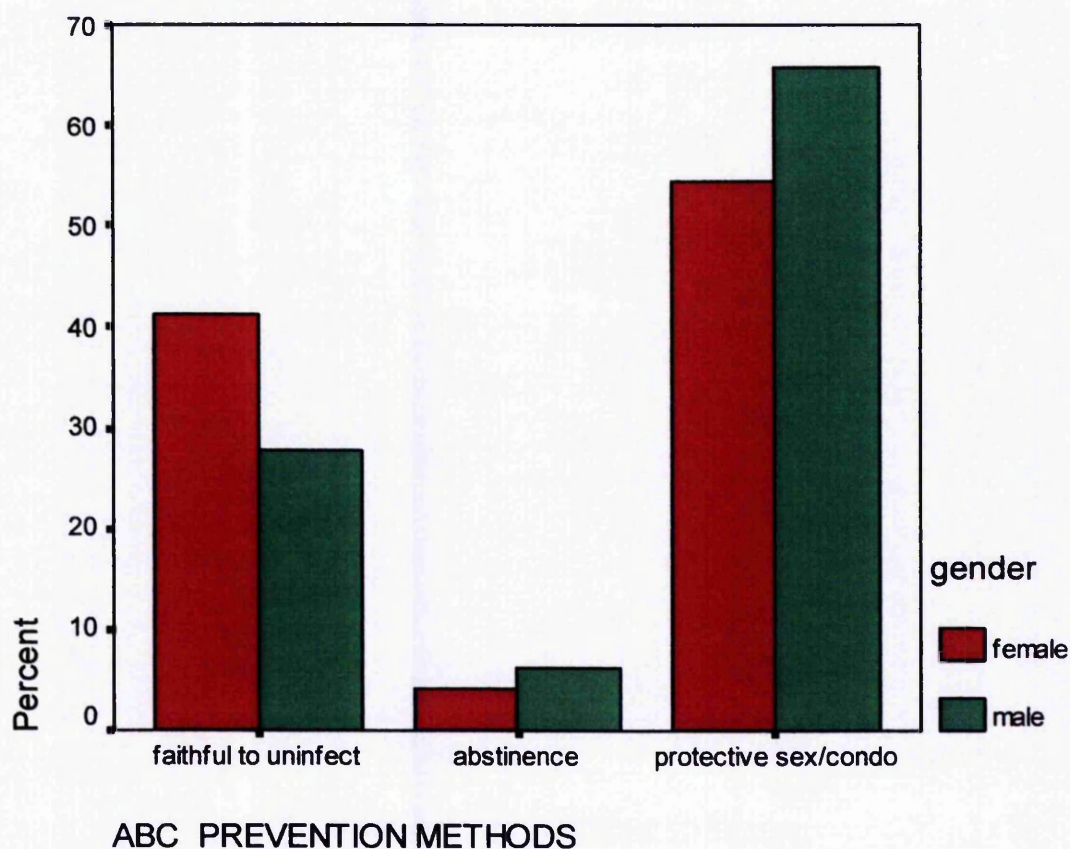
Figure 7.2 main mode of spread of HIV/AIDS by gender



7.3.2 Knowledge of ABC Prevention Methods

Result from the survey shows that among the ABC methods of prevention, protective sex was the most popular method of prevention among both females and males though more men advocate for protective sex than women (see Figure 7.3). The second most widely known method of prevention was faithfulness to one uninfected partner. This method of prevention was reported more by females. This may reflect social relations in Africa in which it is acceptable for men to have more partners while women may not. Since protective sex was the most popular means of prevention as illustrated in figure 7.3, bivariate analysis is used to find out whether those who agree that condoms are a means of protection against HIV are actually using them.

Figure 7.3 knowledge of prevention methods.



7.4 The relationship between HIV prevention methods and the practice of preventive behaviours

The promotion of safe sexual behaviours is an important component of Ghana's HIV/AIDS prevention programme. As the 2003 GDHS and the survey of the Krobo district shows, knowledge of methods of HIV prevention particularly condom use is relatively high. However the link between this knowledge and reducing HIV prevalence lies in the adoption of lower risk sexual behaviours. This section explores the relationship between knowledge about condom use as a preventive method and use of condoms. Because condom use is such a large component of the ABC strategy (abstinence, be faithful, condom use), the variables ever used a condom and condom use in the last casual encounter were used to test whether there is a relationship between prevention methods and the practice of preventive behaviour. It answers the key question, 'Are people who know how to protect themselves against HIV infection doing so?' Respondents were asked whether they agree or disagree that condoms are safe in preventing HIV infection and whether they have ever used a condom or whether they used a condom at last sex with a casual sex partner.

Table 7.8b shows that only 91 out of the 236 females have ever used a condom and among females who agree that condoms are safe in preventing HIV, only 53 out of 112 have ever used a condom. It was also observed that among females 10 people more than expected have ever used a condom. In the case of males, 159 out of 252 people have ever used a condom a figure which is more than 50 percent. In the same way among men who agree that condoms are safe in preventing HIV, 109 out of 142 have ever used a condom. Table 7.8 also shows that among males, 20 more people than expected have ever used a condom. This means that among females and males, who agree that condoms are safe in preventing HIV, significantly higher than expected numbers did use a condom though the number for females is relatively smaller as compared to males. This means that the high knowledge of condom use as a preventive measure leads to more people actually using condoms than expected. This means that knowledge about HIV prevention methods does leads to adoption of lower risk sexual behaviour and hence lower HIV prevalence in the Krobo district.

Table 7.8a condoms are safe preventing HIV/AIDS * have you ever used a condom * gender
Cross tabulation

gender				have you ever used a condom		Total
				yes	no	
female	condoms are safe preventing HIV/AIDS	agree	Count	53	59	112
			Expected Count	43.2	68.8	112.0
		disagree	Count	38	86	124
			Expected Count	47.8	76.2	124.0
Total			Count	91	145	236
			Expected Count	91.0	145.0	236.0
male	condoms are safe preventing HIV/AIDS	agree	Count	109	33	142
			Expected Count	89.6	52.4	142.0
		disagree	Count	50	60	110
			Expected Count	69.4	40.6	110.0
Total			Count	159	93	252
			Expected Count	159.0	93.0	252.0

Table 7.8b Chi-Square Tests

gender		Value	df	Asymp. Sig. (2-sided)
female	Pearson Chi-Square	6.908(b)	1	.009
	N of Valid Cases	236		
male	Pearson Chi-Square	26.089(c)	1	.000
	N of Valid Cases	252		

a. Computed only for a 2x2 table

b. 0 cells (.0%) have expected count less than 5. The minimum expected count is 43.19.

c. 0 cells (.0%) have expected count less than 5. The minimum expected count is 40.60.

Similarly, significantly higher than expected numbers did use condom with casual sexual partner if agree condoms are safe preventing HIV/AIDS ($p < 0.005$) See table 7.9b. Table 7.9a shows that among females who agree that condoms are safe in preventing HIV, 9 more people than expected used a condom at last sex with a casual partner. In the case of males, 26 people more that expected used condoms at last sex

with a casual partner. This means that those who agree that condoms are safe in preventing HIV spread are more likely to use condom with a casual sex partner than expected as shown in table 7.9a. It is however worth noting that among females, only 29 out of 112 who agree that condoms are safe in preventing HIV actually used condom at last sex with a casual partner. In the case of men, about half (70 out of 144) of those who agree that condoms are safe in preventing HIV used a condom at last sex with a casual partner.

Table 7.9a condoms are safe preventing HIV/AIDS * did you use a condom when you last had sex with a casual partner * gender Crosstabulation

gender				did you use a condom when you last had sex with a casual partner		Total
				yes	no	
female	condoms are safe preventing HIV/AIDS	agree	Count	29	83	112
			Expected Count	20.1	91.9	112.0
		disagree	Count	14	114	128
			Expected Count	22.9	105.1	128.0
Total			Count	43	197	240
			Expected Count	43.0	197.0	240.0
male	condoms are safe preventing HIV/AIDS	agree	Count	70	74	144
			Expected Count	57.8	86.2	144.0
		disagree	Count	32	78	110
			Expected Count	44.2	65.8	110.0
Total			Count	102	152	254
			Expected Count	102.0	152.0	254.0

Table 7.9b Chi-Square Tests

gender		Value	df	Asymp. Sig. (2-sided)
female	Pearson Chi-Square	9.084(b)	1	.003
	N of Valid Cases	240		
male	Pearson Chi-Square	9.888(c)	1	.002
	N of Valid Cases	254		

a. Computed only for a 2x2 table

b. 0 cells (.0%) have expected count less than 5. The minimum expected count is 20.07.

c. 0 cells (.0%) have expected count less than 5. The minimum expected count is 44.17.

7.5 The relationship between economic factors and risky sexual behaviours

As noted earlier in the theoretical framework, individual human behaviour plays out in a particular economic and political environment to make individuals vulnerable to HIV infection. This section therefore examines the relationship between level of income and employment status to see whether these factors make people of the Krobo district more vulnerable to HIV infection. The relationship between risky sexual behaviour such as having multiple partners, having sex with casual partners, condom usage, ever contracted an STD on one hand and employment status and level of income on the other is explored.

7.5. 1 Employment status and risky sexual behaviour

It was observed from the bivariate analysis that among females, there is significant relationship between the level of employment, on one hand and having had sex with anyone else apart from regular sex partner over the past year preceding the survey as indicated by p-value (less than 0.05) for chi-square (see Table 7.10b). This means that women who are unemployed are significantly ($p < 0.05$) more likely to have sex with other people apart from their regular sex partner than expected (see table 7.10a). These women may sell sex as a source of income or may be vulnerable to sexual exploitation and hence increase their risk to HIV infection. In the case of men there is no significant relationship between employment status and having sex with anyone else apart from regular sexual partner ($p > 0.05$).

Table 7.10(a) employment status * did you have sex with anyone else apart from your regular sex partner last year * gender Crosstab

gender				did you have sex with anyone else apart from your regular sex partner last year		Total
				yes	no	
female	employment status	unemployed	Count	28	132	160
			Expected Count	22.5	137.5	160.0
		employed	Count	6	76	82
			Expected Count	11.5	70.5	82.0
Total			Count	34	208	242
			Expected Count	34.0	208.0	242.0
male	employment status	unemployed	Count	45	121	166
			Expected Count	43.1	122.9	166.0
		employed	Count	22	70	92
			Expected Count	23.9	68.1	92.0
Total			Count	67	191	258
			Expected Count	67.0	191.0	258.0

Table 7.10 (b) Chi-Square Tests

gender		Value	df	Asymp. Sig. (2-sided)
female	Pearson Chi-Square	4.655(b)	1	.031
	N of Valid Cases	242		
male	Pearson Chi-Square	.314(c)	1	.575
	N of Valid Cases	258		

a Computed only for a 2x2 table

b 0 cells (.0%) have expected count less than 5. The minimum expected count is 11.52.

c 0 cells (.0%) have expected count less than 5. The minimum expected count is 23.89.

Table 7.11b shows that there is significantly high relationship between employment status for both females and males and having a regular sex partners during the year preceding the survey ($p < 0.05$). It was observed that among both sexes the unemployed had less regular sex partners than expected (see Table 7.11b). The unemployed therefore are more likely to have multiple sex partners than their counterparts who are employed and this may increase their risk to HIV infection. This

result confirms earlier result where respondent were asked whether they had sex with anyone else apart from their regular sex partners.

Table 7.11(a) employment status * did you have a regular sex partner during the last 12 months * gender Crosstab

gender				did you have a regular sex partner during the last 12 months		Total
				yes	no	
female	employment status	unemployed	Count	86	66	152
			Expected Count	96.1	55.9	152.0
		employed	Count	62	20	82
			Expected Count	51.9	30.1	82.0
Total			Count	148	86	234
			Expected Count	148.0	86.0	234.0
male	employment status	unemployed	Count	94	66	160
			Expected Count	102.9	57.1	160.0
		employed	Count	68	24	92
			Expected Count	59.1	32.9	92.0
Total			Count	162	90	252
			Expected Count	162.0	90.0	252.0

Table 7.11(b) Chi-Square Tests

gender		Value	df	Asymp. Sig. (2-sided)
female	Pearson Chi-Square	8.299(b)	1	.004
	N of Valid Cases	234		
male	Pearson Chi-Square	5.850(c)	1	.016
	N of Valid Cases	252		

a Computed only for a 2x2 table

b 0 cells (.0%) have expected count less than 5. The minimum expected count is 30.14.

c 0 cells (.0%) have expected count less than 5. The minimum expected count is 32.86.

STD infection is an indication of risky sexual behaviour which consequently increases the risk to HIV infection (Lamprey, 2002). This section therefore explores the relationship between employment status and STD infection in the Krobo district. Table 7.12b shows that there is significant relationship among males who are employed and STD infection ($p < 0.05$). Table 7.12a shows that men who are employed are more likely to have contracted an STD than expected. It is likely that

men who are employed are able to pay for sex either by visiting prostitutes or having more sexual partners than their counterparts who are unemployed and therefore increases their risk to HIV infection. In the case of women there is no significant relationship between employment status and STD infection ($p>0.05$).

Table 7.12(a) employment status * have you ever contracted any STD * gender
Crosstab

gender				have you ever contracted any STD		Total
				yes	no	
female	Employment status	unemployed	Count	20	138	158
			Expected Count	18.4	139.6	158.0
		employed	Count	8	74	82
			Expected Count	9.6	72.4	82.0
male	employment status	unemployed	Count	44	122	166
			Expected Count	51.5	114.5	166.0
		employed	Count	36	56	92
			Expected Count	28.5	63.5	92.0

Table 7.12 (b) Chi-Square Tests

gender		Value	df	Asymp. Sig. (2-sided)
female	Pearson Chi-Square	.441(b)	1	.507
	N of Valid Cases	240		
male	Pearson Chi-Square	4.410(c)	1	.036
	N of Valid Cases	258		

a. Computed only for a 2x2 table

b. 0 cells (.0%) have expected count less than 5. The minimum expected count is 9.57.

c. 0 cells (.0%) have expected count less than 5. The minimum expected count is 28.53.

7.5. 2 Income levels and risky sexual behaviour.

Table 7.13b shows that there is significantly high relationship between income levels and having a regular sex partner during the past twelve months preceding the survey ($p<0.05$). For both females and males, lower income earners are more likely to have irregular and multiple sex partners than expected as shown in table 7.13a. For women,

poverty as a result of lower income may expose them to sexual exploitation by men and women may sell sex for money. In the case of men, those with lower income may exploit the relatively wealthier women especially return-migrants from Cote d'Ivoire by having sex with as many as possible for monetary gains and thus increases their risk to HIV infection.

Table 7.13(a) estimated monthly income * did you have a regular sex partner during the last 12 months * gender Crosstab

gender				did you have a regular sex partner during the last 12 months		Total
				yes	no	
female	estimated monthly income	under 100,000-1000000 cedis	Count	131	84	215
			Expected Count	136.0	79.0	215.0
		1,100,000-2,100,000 cedis	Count	17	2	19
			Expected Count	12.0	7.0	19.0
male	estimated monthly income	under 100,000-1000000 cedis	Count	135	84	219
			Expected Count	140.8	78.2	219.0
		1,100,000-2,100,000 cedis	Count	27	6	33
			Expected Count	21.2	11.8	33.0

Table 7.13 (b) Chi-Square Tests

gender		Value	df	Asymp. Sig. (2-sided)
female	Pearson Chi-Square	6.119(b)	1	.013
	N of Valid Cases	234		
male	Pearson Chi-Square	5.084(c)	1	.024
	N of Valid Cases	252		

a Computed only for a 2x2 table

b 0 cells (.0%) have expected count less than 5. The minimum expected count is 6.98.

c 0 cells (.0%) have expected count less than 5. The minimum expected count is 11.79.

Result from the survey further shows that there is significant relationship between income levels and condom usage among females ($p < 0.05$). Among women earning lower incomes, those who had never used a condom is more than expected as indicated in table 7.14a. This shows that among poorer women condom usage is very low and this may be due to the fact that they cannot afford to buy a condom or

because the vulnerable position of such women means that they cannot insist on condom use even if they wish to. This situation increases their risk to HIV infection. There is however no significant relationship for men ($p>0.05$).

Table 7.14(a) estimated monthly income * have you ever used a condom * gender
Crosstab

gender				have you ever used a condom		Total
				yes	no	
female	estimated monthly income	under 100,000-1000000 cedis	Count	80	139	219
			Expected Count	85.6	133.4	219.0
		1,100,000-2,100,000 cedis	Count	13	6	19
			Expected Count	7.4	11.6	19.0
male	estimated monthly income	under 100,000-1000000 cedis	Count	145	78	223
			Expected Count	142.0	81.0	223.0
		1,100,000-2,100,000 cedis	Count	18	15	33
			Expected Count	21.0	12.0	33.0

Table 7.14(b) Chi-Square Tests

gender		Value	df	Asymp. Sig. (2-sided)
female	Pearson Chi-Square	7.469(b)	1	.006
	N of Valid Cases	238		
male	Pearson Chi-Square	1.364(c)	1	.243
	N of Valid Cases	256		

a. Computed only for a 2x2 table

b. 0 cells (.0%) have expected count less than 5. The minimum expected count is 7.42.

c. 0 cells (.0%) have expected count less than 5. The minimum expected count is 11.99.

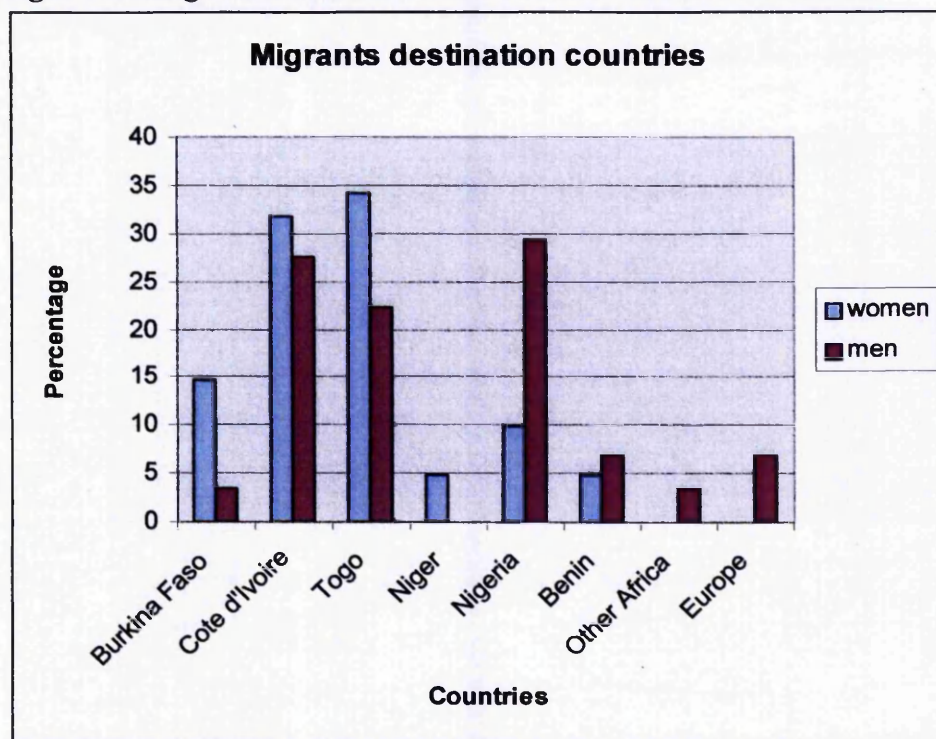
7.6 Out-migration from the Krobo district and the spread of HIV/AIDS

Many studies have linked the spread and diffusion of HIV/AIDS to migratory processes. Akwara et al (2005) observed that population mobility be it permanent, seasonal or circulatory may be a risk factor for HIV infection. Others such as Prothero (1997) for example have reported significant interactions between disease and

population mobility in tropical Africa. Similarly, Hunt (1996) points to migration as a key predictor for the higher AIDS prevalence rate in the African countries with higher concentration of labour migrants. In the case of Ghana, Konotey-Ahulu (1989) observed that at the early stages of the AIDS epidemic in Ghana in the mid-1980s, most of the people identified as HIV carriers had a history of migration. The result from the modelling output shows that HIV initially spread from Ghana to Cote d'Ivoire and neighbouring countries but spread back to Ghana at the peak of the epidemic. This section therefore present distribution of Krobo migrants in terms of their destination countries to see any link with the modelling output.

Figure 7.4 show that Cote d'Ivoire, Togo and Nigeria are the main destinations countries for migrants from the Krobo district. In Cote d'Ivoire, Togo and Burkina Faso female migrants exceed male migrants whereas in Nigeria men dominate. This means that whereas female migrant migrate to bordering countries which are closer to home, male migrants travel further field including overseas. The main destinations as shown in figure 7.4, especially Cote d'Ivoire and Nigeria are the most highly HIV prevalence countries in West Africa. There is therefore the likelihood of infection transfers from these countries to Ghana as shown by the modelling output at the peak of the epidemic. Anarfi (1993) for example observed that Krobo women, mainly prostitutes returning from Abidjan to their homeland, suffer from ill health and in most cases die. Considering the fact that migration from the Krobo district to Cote d'Ivoire started in the mid 1960s when the Akosombo dam was built and the mass migration to Nigeria and Cote d'Ivoire occurred during the economic decline in Ghana in the early 1970s, it is possible that the epidemic may have spread from Ghana, especially from the Krobo district, having one of the highest prevalence to Cote d'Ivoire as estimated by the modelling output.

Figure 7.4 Migrant destination countries from the Krobo district.



7.7 Multivariate Analysis

In the previous section, the bivariate analysis was limited to the relationship between knowledge of preventive methods and the practice of preventive behaviour. It was observed that high knowledge about HIV prevention methods such as condom usage leads to higher preventive behaviour such as general condom use and condom use at last sex with a casual partner. Secondly employment status and income levels were variables that were significantly related to risky sexual behaviour such as having multiple sexual partners, having sex with other people apart from regular sexual partner and STD infection. In this section the relationship between demographic, socio-economic, migration, behaviour, risk and knowledge variable on one hand and condom use on the other is explored using logistic regression. This technique allows us to assess the independent association between each variable and condom use while controlling for the effect of other variables. The explanatory variable chosen were grouped into demographic, social, migration, behaviour, risk and knowledge variables. The demographic factors include age, gender and marital status. The socio-economic factors include educational status, employment status and income level. The behaviour and risk factors include level of risk, is you at risk to HIV infection and

ever contracted an STD. Lastly knowledge variables included ever heard of AIDS and whether AIDS is real. These factors were included in the multivariate analysis because of the demonstrated significance on both the bivariate and multivariate analysis of the 2003 DHS and the bivariate analysis of the survey of Krobo district presented earlier.

The dependent variable representing condom use is a dichotomous variable taking the value $Y=0$ for never used a condom and $Y=1$ for used a condom before. Logistic regression analysis was employed to determine the likelihood and probability of HIV infection based on the selected explanatory variables. The rationale for the logistic regression analysis was that, it is appropriate for estimating odds ratios for each factor to tell by how much more each significant explanatory variable increases condom usage. Applying the logistic regression model to the data yielded the result shown in Table 7.15. An odds ratio gives an estimate of the magnitude of the association between the variables and condom use. In this analysis an odds ratio of 1.0 indicates no difference between the variable and condom use, a ratio below 1.0 indicates a negative association between the variable and condom use and a ratio above 1.0 indicates a positive association.

Table 7.15 shows that among the demographic variables, age and gender were significantly associated with condom use. In terms of age, those within the age group 15-34 years were more likely (odds=3.7) to use a condom than those within the age group 35-49 (odds= 2.4). In terms of gender there is significant association between females and condom use. Female are less likely to use a condom (odds=0.34) Among the socio-economic variables, educational level is the only significant factor in relation to condom use. The respondent without any formal any formal education were less likely to use a condom (odds=0.39). See table 7.15.

Table 7.15 further shows that among the behavioural and risk factors, those who have regular sex partner were less likely (odds=0.30) to use a condom. Also those who have not had sex with any other person apart from their regular sex partners were less likely (odds=0.35) to use a condom. Unlike the bivariate analysis, the knowledge and migration variables were not significantly associated with condom use. For the full output of the logistic regression, see Appendix 7.

Table 7.15 Explanatory variable and the odds of perceiving risk to HIV infection

Explanatory variables		B	S.E.	Wald	df	Sig.	Odds ratio
Demographic	age			10.021	2	.007	
	(15-34)	1.321	.422	9.806	1	.002	3.746
	(35-49)	.897	.408	4.832	1	.028	2.452
	Gender(female)	1.071	.248	18.591	1	.000	.343
	Marital status(Married)	-.481	.266	3.278	1	.070	.618
Socio-economic	Q5 education			4.839	3	.184	
	(none)	-.919	.442	4.322	1	.038	.399
	(pri/mid)	-.641	.443	2.094	1	.148	.527
	(sec)	-.813	.451	3.242	1	.072	.444
	employment(unemployed)	.141	.226	.391	1	.532	1.152
	income(<\$11)	.014	.367	.001	1	.970	1.014
Migration	Ever trav outside Ghana	-.216	.213	1.030	1	.310	.806
Knowledge	Ever heard of AIDS	.339	.888	.146	1	.702	1.404
	Is AIDS real	-.218	.466	.219	1	.640	.804
Risk Behaviour	are you at risk	.203	.290	.489	1	.484	1.225
	age first sexual debut			4.241	4	.374	
	10-14	-.536	1.344	.159	1	.690	.585
	15-19	-.260	1.307	.039	1	.843	.771
	20-24	.170	1.314	.017	1	.897	1.185
	25-29	-.242	1.339	.033	1	.856	.785
	did you have regular sex partner	1.190	.271	19.318	1	.000	.304
	had sex with anyone other than regular partner	1.044	.309	11.425	1	.001	.352
	STD infection	-.130	.287	.203	1	.652	.878
	What is your level of risk			3.048	3	.384	
	not at risk	-.451	.378	1.420	1	.233	.637
	moderate risk	.082	.319	.067	1	.796	1.086
	high risk	.045	.343	.017	1	.896	1.046
	Constant	4.418	1.947	5.151	1	.023	82.948

7.8 Discussion

The result obtained from the quantitative analysis is discussed in the section below by first presenting summary of both the bivariate and multivariate analysis in bullet points. This is followed by discussion of the three hypotheses and finally presenting the wider discussion of the result for the implication for the different frameworks and for policy action.

Summary of the findings of the bivariate and multivariate analysis.

Bivariate:

- Awareness about HIV/AIDS in the Krobo district is very high and is consistent with the 2003 DHS survey.
- Among the ABC preventive methods, protective sex (condom use) was the method reported more by both females and males.
- Among those who agree that condoms are safe in preventing HIV infection, there is significantly higher number of them who use a condom.
- Those who agree that condoms are safe in preventing HIV spread are more likely to use a condom with a casual sex partner.
- Knowing a method of preventing HIV infection leads to a practice of less risky sexual behaviour.
- Women who are unemployed are more likely to have sex with other people apart from their regular sex partners.
- Men who are in gainful employment are more likely to be infected with an STD than their unemployed counterparts.
- Among both females and males, lower income earners are more likely to have multiple sex partners.
- Condom usage is lower among women who earn lower income
- The main destination countries for migrants from the Krobo district are Cote d'Ivoire and Nigeria

Multivariate:

The multivariate package involves demographic, social, economic, migration, knowledge and risk behaviour variables. Among these, age, gender, educational attainment, and sexual partner change are the only significant factors which act together to determine condom use. It emerged from the multivariate result that

- The younger age group (15-34) are 3.7 times more likely to use condom than their older counterparts.
- In terms of gender, females are less likely (odds=0.34) to use condoms compared to males.
- Those with no formal education are less likely (odds=0.39) to use condoms.
- Respondents with regular sexual partners and in a stable relationship are less likely to use condom (odds=0.30).

In order to explore the socio-economic, demographic, cultural and political context in which the epidemic continue to spread at the sub-national level, three main hypotheses were formulated to test whether there is any significant relationship between

1. Knowledge of HIV prevention methods and the practice of preventive behaviour
2. Economic and political factors and risky sexual behaviour
3. Social and demographic characteristics, risky sexual behaviours, HIV/AIDS knowledge, mobility and risk perception on one hand and condom use.

In the first place the results show that there is significant relationship between HIV prevention methods and preventive behaviours. Among females who agree that condoms are safe in preventing HIV, 9 more people than expected used a condom. In the case of males 20 more people than expected used a condom. This shows that there is a significantly higher number of both female and male who use a condom among those who agree that condoms are safe in preventing HIV infection. This means that the high knowledge of condom use as a preventive measure is more likely to result in more people using condoms than expected. The null hypothesis that there is no significant relation between knowledge about HIV prevention methods and preventive behaviour is therefore rejected. Knowledge about HIV prevention method therefore leads to adoption of preventive behaviour hence lowering the risk to HIV infection.

Secondly, the bivariate analysis shows that there is significant difference between employment status and income levels on one hand and risky sexual behaviours. This has a strongly gendered profile. Firstly, unemployed women are more likely to have sex with other people apart from their regular sex partners. Men who are in employment are more likely to be infected with an STD than those who are unemployed. In terms of income, both women and men who earn lower incomes are more likely to have multiple sex partners; however the lower income earning women are less likely to use condom as compared to their male counterparts hence increasing their risk to HIV infection. The null hypothesis that there is no significant relationship between economic and political factors and risky sexual behaviours is therefore rejected for the alternative hypothesis. There is therefore significant relationship between employment status and income levels and the practice of risky sexual behaviour in the Krobo district.

It was also hypothesised that there is no significant relationship between social, demographic, risky sexual behaviours, HIV/AIDS knowledge, risk perception, mobility and condom use. The multivariate result shows that the variables age, gender, educational status, and level of partner change act together to determine condom use. In the first place, it was observed that the younger age group (15-34) are more likely to use condom than those in the older age group (35-49). Age is therefore a significant factor as far as condom use is concern. Secondly, the multivariate result shows that gender is significantly related to condom use. There is a significant relationship between females and condom use. Women were less likely to use condom. In terms of educational status the result shows that there is a significant relationship between educational attainment and condom use. The respondents with no formal education were less likely to use condom. Among the socio-economic and demographic factors, age, gender and educational status were significantly related to condom use. The null hypothesis that there is no significant relationship between socio-economic and demographic factors and condom use is therefore rejected. Lastly, there is a significant relationship between risky sexual behaviours and condom use. It was observed from the multivariate analysis that those who have regular sex partners and those who have not had sex with any other person apart from their regular sex partners in the past twelve months preceding the survey were less likely to use a condom. Such people may perceive themselves as being less at risk to HIV

infection and therefore not use condoms. The null hypothesis that there is no significant relationship between risky sexual behaviour factors and condom use is rejected.

7.9 Implication of the results for the different frameworks (see chapter three)

The core of the cultural (behavioural) approach is the thinking that individual sexual behaviour is the main determinant for the spread of HIV and that epidemic can be controlled by advocating behaviour change. It is based on this thinking that the ABC approach was introduced as an intervention to HIV/AIDS spread. The result from the study shows that knowledge about preventive methods is significantly related to preventive behaviour. As shown from the bivariate analysis, people with high knowledge of condom use as a preventive measure and who agree that condoms are safe in preventing HIV spread tend to use condoms more than expected. The results from the multivariate analysis also show that there is significant relationship between individual sexual behaviour and condom use. Here those with regular sexual partners were less likely to use condom than people with multiple sex partners. The implication of these findings is that advocating behaviour change through the ABC approach and increasing general awareness about the HIV/AIDS epidemic which is the core thinking of the behavioural approach may help in mitigating the epidemic spread in the Krobo district.

The apparent failure of the behavioural interventions in mitigating the epidemic spread however means that there are other dimensions to the epidemic spread. The political economy approach focuses on the interplay between macro-economic structures, culture and socio-demographic characteristic of the population. The results of the study show that unemployed women are more likely to have sex with other people apart from their regular sex partners. Also men who are in employment are more likely to be infected with an STD. In terms of income, both women and men who earn lower incomes are more likely to have multiple sex partners. Also women in the lower income earning group are less likely to use condom as compared to their male counterparts hence increasing their risk to HIV infection. The multivariate result also shows that gender, age and educational attainment are significantly related to condom use. This means that behaviour change alone as advocated by the behavioural

approach may not adequately address the epidemic spread. Individual sexual behaviour that exposes them to the risk of HIV infection therefore is influenced by socio-demographic characteristics namely age, gender and educational level which plays out in a particular culture such as the subordinate social and economic role of women in the Krobo district and is influenced by the wider economic issues such as unemployment and lower levels of incomes in the Krobo district.

The concept of vulnerability as discussed in chapter three applies to the Krobo district. The result shows that knowledge about HIV/AIDS is high but the epidemic continues to spread. This means that the capacity of individuals to respond effectively to health threat is reduced. The vulnerability of the Krobo people as shown in the results is created as a result of unemployment and lower income levels in the district. In this situation of extreme poverty, people in the Krobo district migrate to Cote d'Ivoire which is the most HIV infected country in West Africa. The risky sexual behaviour in the Krobo district therefore takes place within a particular risk environment created during the process of migration (Kanyenze, 2004). The holistic view of presenting the HIV/AIDS epidemiology in the Krobo district as illustrated in figure 3.3 (see chapter 3) combines the three main approaches in a synthesised framework. Individual sexual behaviour takes place within the cultural context of the Krobo and is influenced by socio-demographic characteristics as well as the political economy which creates the risk environment for the epidemic to spread.

7.10 Implication of the result for policy action

The results presented above show that awareness about HIV/AIDS is high in the Krobo district. Secondly those who are aware of preventive measures are more likely to practice preventive behaviour. For instance those who agree that condoms are safe in preventing HIV spread are more likely to use condoms. It is therefore expected that the epidemic would be under control but the reverse is the case. The ABC method has been the main advocacy tool in the Krobo district but apparently having little impact on mitigating the epidemic spread. There is therefore the need to examine the socio-cultural context in which such policies are implemented. There is also the need to assess the main players involved in the planning and implementing the policy as well as the target group for different intervention.

The result also shows that women who are unemployed as well as those earning lower incomes are more likely to have multiple sexual partners. More importantly, condom use is also low among women with lower income and hence increases their risk to HIV infection. Men who are gainfully employed on the other hand are more likely to be infected with an STD including HIV. This means that the unemployed especially the females may sell sex for money while the men who are employed and relatively wealthier may exploit the vulnerable poor by paying for sex hence increasing their likelihood of contracting an STD including HIV/AIDS. Unfortunately the issue of unemployment and poverty though well known to influence the epidemic spread, has received little attention in the area of intervention. This is probably because of lack of political will by government as well financial constraints by NGOs. The basic cause of vulnerability being unemployment and its resultant poverty should therefore be addressed if interventions are to have the desired result. In this case vulnerable women should be targeted through the creation of jobs as a longer term measure but in the short term protective sex should be encouraged. The relatively wealthier males should be targeted with preventive interventions such as condom use which is popular among the Krobo people.

The result of the multivariate analysis shows that the younger age group are more likely to use condom. It is possible that these groups engage in relatively higher risky sexual behaviour hence perceive themselves as being at greater risk to HIV infection. Condom use among these young age group therefore present an opportunity to reduce the epidemic spread since this group are the most sexually active group and at greater risk to HIV infection. The result also shows that females are less likely to use condoms than their male counterparts. Some of the reasons for low and inconsistent use of condom among women may be partner refusal and lack of trust in the reliability of condoms and interference with child bearing as found elsewhere in (Clark et al, 1997; Hankins et al, 1997 and Bedimo et al, 1998). The extent to which implementation of the ABC prevention method takes into consideration the socio-economic and cultural background of the Krobo people is an issues that needs to be considered. It emerged from the analysis of the multivariate results that the level of condom use is lower among people with no formal education. The vulnerability of people with no formal education may increase probably because they may not be able to make informed decision about their sexual life and that of their sexual partners. The

result also shows that those with regular sex partners are less likely to use a condom. The implication here is that people who believe that they have no risk or only a small risk of contracting HIV/AIDS are less likely to change their behaviour. This false sense of security may pose a serious challenge to the implementation of the ABC preventive methods.

The question that arises from this discussion is that if knowledge and awareness about HIV/AIDS is high, why is the epidemic still increasing in its spread? Secondly if those who are aware of preventive measures are more likely to practice preventive behaviour why is this not been translating to a reduction in the epidemic spread? Is the continued spread of the epidemic due to the high unemployment and its associated low income and resulting poverty in the Krobo district to blame? In order to probe deeper into the reasons for the continued spread of the epidemic despite general awareness, HIV/AIDS service organisations are interviewed to find out what policy actions are put in place and whether such policies are well informed or not and what they perceive as the reasons for the increasing spread of the epidemic. This is the main focus of the next chapter.

CHAPTER EIGHT

8.0 QUALITATIVE ANALYSIS OF HIV/AIDS SPREAD IN THE KROBO DISTRICT: VIEWS FROM HIV/AIDS SERVICE ORGANISATIONS

8.1 Introduction

Despite the persistent effort by international, governmental and nongovernmental organisations to halt the spread of HIV/AIDS in the Krobo district in Ghana, the district continues to have one of the highest incidence and prevalence of HIV/AIDS in not only Ghana but also West Africa as a whole. In view of this, the views of organisations dealing with HIV/AIDS and related issues in the Krobo district is crucial since their perspective and experiences are likely to provide insight on the reasons for the seemingly failure of current interventions to mitigate the rapid spread of the HIV virus. This chapter therefore presents the result of the interviews with care provider organisations. The first part of the chapter presents the interventions in place in the Krobo district and the rationale behind such interventions. The second part present some ideas that such organisations perceive as the cause of the epidemic spread. The analysis of the interview was based on four-stage procedure adapted from Atkinson and Farias (1995) and Smyth (1995). Five themes that emerged from the interviews with the care provider organisations are presented below.

8.2 Current interventions in the Krobo district

The main intervention programmes launched by the service providers in the Krobo district include promoting abstinence and faithfulness; promoting reductions in the number of sexual partners; encouraging delays in the onset of sexual activity among adolescents; promoting condoms use; strengthening programmes for STD control; and voluntary counselling and testing. Others include care for PLWHA and provision of antiretroviral treatment. For the main interventions carried out by each of the five selected service-based organisation see table 6.1 in chapter 6.

The rationale for the adoption of the above intervention was based on the fact that the main mode of HIV transmission in the Krobo district and Ghana as a whole is through

heterosexual contact therefore interventions aimed at preventing the sexual transmission of HIV will help to reduce the epidemic spread. These interventions are based on the behavioural scale of explanation where individual sexual behaviour is seen as the key determinant for the spread of HIV. The assumption of this approach is that the HIV/AIDS epidemic can be controlled through behaviour change. The extent to which this assumption holds can be seen from the themes that emerged from the interview.

8.3 The perception of service provider organisations on the causes of the HIV epidemic spread and obstacles to preventing its spread.

The main factors that were identified as causing and sustaining the epidemic spread and challenging the success of intervention programmes are the effects of migration on the epidemic spread, belief in witchcraft, faith healers and prayer camps, stigma associated with HIV and the impact of unemployment and poverty in the Krobo district.

8.3.1 HIV infection transfer from Cote d'Ivoire

According to the HIV/AIDS counsellor at the St Martins hospital at Agormanya, most of the first HIV/AIDS cases in the district were prostitutes who had returned from Abidjan in Cote d'Ivoire as noted by Anarfi (1993). These people were sick and weak and unable to work any longer as prostitutes and did not want to die in a foreign land; in most cases they were carried from buses because they could not walk on their own. As a result of the limited number of hospital beds at the Agormanya hospital, home-based care was developed to take care of the AIDS patients in their family homes. Therefore, there appears to have been an infection transfer from neighbouring Cote d'Ivoire to the Krobo district in Ghana at the peak of the epidemic and this supports and is supported by the results of the modelling exercise in Chapter 5.

However, even though migration related activities resulted in the transfer of infection to the district at peak prevalence, the epidemic is now endemic and spreading in the

general population through heterosexual intercourse as illustrated by the quotation below.

‘Infact I don’t know what is happening this time because during sister Margaret’s time all the HIV patients were *Ashawo* women (prostitutes) from Abidjan but now our young children are dying of AIDS (SMH:AIDS coordinator St Martins Hospital),’

8.3.2 Belief in witchcraft as cause of AIDS and the rise of faith healers and prayer camps

The representatives of the care providing organisations interviewed indicated that local belief in witchcraft as a cause of AIDS is widespread among the general population in the Krobo district. HIV/AIDS is therefore seen as a spiritual disease which has to be cured by spiritual means. This has resulted in the proliferation of faith healers, prayer camps and some herbalist who claim to have a cure for AIDS. As noted earlier, the initial HIV/AIDS patients were prostitutes returning from Abidjan. These people were relatively richer and had acquired assets in the form of property, buildings and personal belongings through their trade in Abidjan. People in the community therefore look up to such people with respect and pride. Unfortunately, these are the very people who were first to be infected with a disease which seem to have no cure. This provoked people to explain the disease in terms of witchcraft by family members envious of their wealth causing the infection with the slim disease ‘*korle doole*’. This witchcraft based explanation persists today. Moreover, people living with HIV/AIDS in their desperation for cure turn to faith healers and prayer camps. Unfortunately, informants suggest such prayer camps can aggravate the situation and accelerate the progression from HIV to AIDS and early death as this quotation illustrates,

‘In this community HIV/AIDS is associated with witchcraft and such a perception is deeply entrenched in the mind of the people that it is difficult for People Living with HIV/AIDS to accept the fact that AIDS is cause by a virus. As a result many prayer camps and faith healers claim prayer and fasting can cure AIDS. PLWHA are camped and made to fast and pray till most of them become weak and die of starvation and malnutrition. There is one prayer camp

where I visited but please I can't mention the name of the camp. In that camp there was one woman who tested positive with HIV and was receiving antiretroviral treatment but she absconded from home to the prayer camp. When I visited her at the camp, she told me that the prophet has seized the antiretroviral drugs she was using. Instead her body was smeared with red muddy clay mixed with olive oil. She told me that the prophet told her that one of her aunties was a witch and was responsible for her sickness. I tried to convince her to return home to continue with the antiretroviral treatment but she refused. So you see the influence of these pastors and prophets is such that PLWHA don't listen to advice and counselling from health professionals.'

(Friends of the Sick representative).

Thus, informants report that the belief in witchcraft as a cause of HIV/AIDS coupled with the high illiteracy rate in the Krobo district has resulted in people not adhering to educational campaigns about HIV/AIDS. Also the faith healers, prayer camps and herbalists have exploited the situation of people living with HIV/AIDS and encourage them to comply with the antiretroviral treatment. Informants reckon that most people living with HIV/AIDS tend to seek help from faith healers, spiritual churches and herbalist instead of coming to the hospital.

'In fact some of the patients who were receiving antiretroviral treatment with us have run away to remote villages and have abandon treatment and it is difficult to trace them and give them treatment (HIV/AIDS counsellor Agormanya Hospital SMH).'

8.3.3 Stigma and HIV/AIDS

The care provider organisations flagged up the importance of stigma associated with the disease as affecting the fight against HIV/AIDS in the Krobo district, as in most countries of the world. Informants reported that people living with HIV/AIDS do not want to disclose their status because of fear of discrimination from others in society. The logical extreme of this is the argument from the point of view of Krobos that it is better not to know your HIV status and die of AIDS than to know your status and go

through humiliation from society and friends before dying. The interviews also reported that people think dying from AIDS brings disgrace to the deceased person and the immediate family so people do not disclose the cause of death of relatives, as this quotation illustrates,

‘They think it is shameful to die of AIDS and it will bring a disgrace to the family. If the dead person has got wife or husband and children people will think that they are also infected and they will shun them.
(HIV/AIDS counsellor Agormanya hospital SMH).

The organisation representatives also noticed that people living with HIV/AIDS do not want to associate with HIV/AIDS counsellors for the fear that people will suspect them of being HIV positive. In a community where stigma about the epidemic is so entrenched, it is evident why people avoid knowing their status, disclosing their status, seeking treatment or counselling or making others aware of their status. Stigma then may play a significant role in explaining why the epidemic continues to persist in the region.

8.3.4 Constraints in the introduction of antiretroviral treatment

The interviewees made it clear that only a very small fraction of people living with HIV/AIDS in Krobo are receiving antiretroviral treatment. Retroviral therapy was first introduced in 2003 on a pilot scale in some selected hospital and the success rate of the project has been very low. In the first place, monitoring the effectiveness of the drugs on patients has been problematic because HIV/AIDS counsellors are not, in most cases, allowed access to the homes of those receiving treatment to do follow ups to make sure patient are taking the drugs, and in the required dosage and whether there are side effects of the drugs on the patients. Without these follow-ups the success of the treatment regime cannot be measured. The problem stems from the type of living arrangements that people find themselves in and the stigma attached to the disease. The average number of households living in one house is about five. This means that there is no privacy and once the AIDS counsellor starts visiting those receiving treatment, other households within the house will speculate and gossip that

such a person is HIV/AIDS positive. This will result in that particular family receiving treatment being stigmatised and humiliated in the community. The result of this is that the HIV/AIDS counsellors are not able to do follow-ups in order to ensure and encourage continuation of treatments, particularly where there are side effects from the drugs. In some cases the prescribed dosage of the drugs is not followed and the treatment becomes ineffective, as the following example demonstrates,

‘Last week I visited one patient at home to supply her with the antiretroviral drugs but she told me that she has not finished taking the drugs I gave her the previous month. When I asked her why she has not finished taking the drugs she replied that she has decided to halve the daily dosage because the drug is expensive, So you see that if we don’t do follow up the treatment will not be effective but we are in most cases denied visit by the patients.’ (AIDS coordinator Atua Hospital ATC)

Additionally, as noted in section 8.2.2, faith healers may confiscate the antiretroviral drugs from members of their congregation and prescribe fasting and prayer which in most cases only serves to accelerate early progression from HIV to AIDS and eventual death. Patients are usually rushed to hospital at the terminal stage of the disease by their relatives or sent home to die.

8.3.5 Impact of Unemployment and Poverty on the spread of HIV/AIDS

Interviewees did discuss some aspects of the wider context in which HIV/AIDS is spread. In particular, there are no jobs for school leavers, the age-group who are most vulnerable to HIV infection. Young girls in particular become highly vulnerable as they engage in risky sexual behaviours as a means of survival. Moreover, even at the subsidised rate, the average Krobo cannot afford to pay the price of the antiretroviral drugs. This means that even though the treatment is available locally, it is not accessible to the very people who most need it. In most cases before the patient is sent to the hospital, the family has already exhausted their finances on herbalists and faith healers and in most cases have sold all their personal belongings with the hope of receiving treatment. Those who are privileged to be put on antiretroviral treatment are

also not able to meet their nutritional needs and these results in complications from the side effects of the drugs

8.4 Discussion

One of the striking features of the perception of the HIV/AIDS service organisations is that on the whole, the interviewees reverted to the cultural and individual behavioural scale of explanation – echoing the pervasiveness of this model. The link between belief in witchcraft, faith healers and herbalist and antiretroviral treatment and the spread of AIDS is illustrated in figure 8.1. In the first place belief in witchcraft is so entrenched in the Krobo society that the health seeking behaviour of PLWHA is geared towards prayer camps, faith healers and herbalist because HIV/AIDS is seen as a spiritual disease caused as a result of envy about property acquired abroad. The belief in witchcraft serves as a diversion to the root cause of the epidemic and hence increases HIV spread. Also the prayer camps and healers discourage the use of antiretroviral drugs hence preventing treatment. The influence of these people as perceived by service organisations is one of the main factors resulting in failure of current interventions to mitigate the epidemic spread.

There is some engagement with wider issues – societal factors operating through stigma and wider political economic factors operating through poverty increasing risky behaviours in the vulnerable age group and making access to treatment almost impossible. Figure 8.1 shows that there is a link between household living arrangements, discrimination and stigma and HIV infection. The household structure is such that there is on the average five households in one compound house. This means that there is no privacy and as such PLWHA in an attempt to hide their status may not allow counsellors to visit them for fear of suspicion and gossip about their status. This was perceived by service organisations as a major set back to the implementation of antiretroviral drugs and a factor leading to increase HIV infection. There also a link between antiretroviral treatment and the cost of drugs, nutritional needs of PLWHA and distance from villages to treatment centres. Although the retroviral drugs are heavily subsidised, the average PLWHA may not be able to buy first because they don't have the money or they may have wasted the money on prayer camps and faith healers. Extreme poverty in this region means that PLWHA

may not be able to meet their nutritional needs even if the drugs were supplied free of charge and some have to walk long distance to visit service centres. These constraints make the administration of antiretroviral drugs difficult since most international agencies do not anticipate such problem in their planning stage.

Wider political economic factors such as unemployment, high illiteracy level was perceived to have worked through poverty to create risk environment for HIV to spread as noted earlier in the quantitative chapter. It was observed from the review in chapter three that the construction of the Akosombo hydropower plant resulted not only in creating the largest man-made lake in the world, the Lake Volta, but also flooding the farmlands of the lower Krobo plains and deprived them of their settlements and farmlands. Failure of resettlement schemes and high illiteracy created a situation of dire poverty which served as a push factor and saw many Krobo women migrating to Cote d'Ivoire while the men moved to other forest regions of Ghana as farm labourers. This migration resulted in risky sexual behaviours which resulted in HIV infection and subsequent infection transfer to the Krobo district. Another important dimension of the migration process is that wealth differential is created between returned migrants and non-migrants. Non-migrants see return migrants as source of income due to their relatively higher income and affluent lifestyle. This often results in risky sexual mixing which increases the risk of infection transfers between migrants and non-migrants. Secondly non-migrants are enticed by the property and wealth acquired by migrants thereby influencing non-migrants to migrate thereby continuing the cycle of migration and infection transfer as illustrated in figure 8.1

The challenges facing HIV/AIDS service organisations given the fact that they are the organisations defining what to do is a daunting task. Although their perceptions point to the wider issues – societal factors operating through broader political economic factors, their preoccupation has been based on the cultural and individual behavioural scale although there has been limited success using this approach. This may probably be due to financial constraints, limitations by donor objectives and lack of political will on the part of the government to tackle the basic issues of unemployment, higher illiteracy rate and poverty which is the driving force behind the HIV/AIDS epidemiology. At the same time while the perceptions of HIV/AIDS service

organisations are likely to be insightful, they come with their own biases. For example the Catholic Church will be highly critical of faith healers and witchcraft and take any opportunity to point finger of blame at them.

The service-base interviews overall then posits an infection transfer from Cote d'Ivoire to Ghana. However the modelling result had Ghana as one of the starting centres spreading HIV to neighbours and that later the shift in direction of infection from neighbours back to Ghana occurred. It is worth noting that anecdotal studies show the existence of *Korle doole* (Slim disease) long before the inception of HIV epidemic and long before the Krobo started migrating to Cote d'Ivoire in the 1970s but the epidemic may have been contained for several reasons. First, due to the strict Krobo culture (*Dipo*), intermarriages between Krobo and other tribes in Ghana were not possible. Secondly the *dipo* culture which once was strictly adhered to meant that any Krobo woman who became pregnant before she was initiated into *dipo* was banished from the tribe. These strict traditional norms resulted in low sexual mixing. Risky sexual behaviour which may be likely to increase risk of infection was therefore kept to the minimum. Besides, the causative agent HIV was not known and all mysterious sicknesses were attributed to witchcraft. This means that it is possible that the HIV virus may have existed among the Krobo but was contained within this largely sexually endogamous group. However, these women who migrated to Cote d'Ivoire as sex workers in the early 1970s may have spread the virus and subsequently brought it back to Ghana at the peak of the epidemic.

If HIV originated from the Krobo district in Ghana as the modelling output shows, then the possible source of infection may be through zoonotic transfer lending credence to the monkey-hunter hypothesis discussed in chapter two, the idea that HIV jumped to humans as a result of a bite or after a hunter became infected while capturing a monkey. The reason for this argument is the fact that the ancestral home of the Krobo which is the Krobo mountains (*Klowem*) and the Shai hills is still the home to African Monkeys and Baboons and that in the past monkeys and humans were living side by side. It is possible that infection transfer occurred between these primates and humans long before the British Colonial Administration drove the Krobo from the mountains and settled them in the lower Volta plains where they currently live.

It is also possible that HIV was introduced to the Krobo district during the construction of the Akosombo Hydroelectric Dam in the early 1950s. The construction workers who were mostly Americans and Canadians used the Krobo women as prostitutes. This is evident by the number of mixed race people along the Volta river basin and in the Krobo district as a whole. Since the first cases of HIV/AIDS were reported in the United States of America, and taking into consideration the long incubation period of the virus averaging 10 years in the absence of treatment, it is possible that some of these men were infected and might have passed on the Virus to these women.

The perception of the organisation-based survey shows that the synthesised framework developed in the conceptual chapter, is applicable in explaining the processes of the epidemic spread in the Krobo district. As illustrated in Figure 8.1, individual sexual behaviour plays out in the broader political economic factor operating through culture and individual characteristics and risk environment which result in HIV infection.

8.5 Conclusion

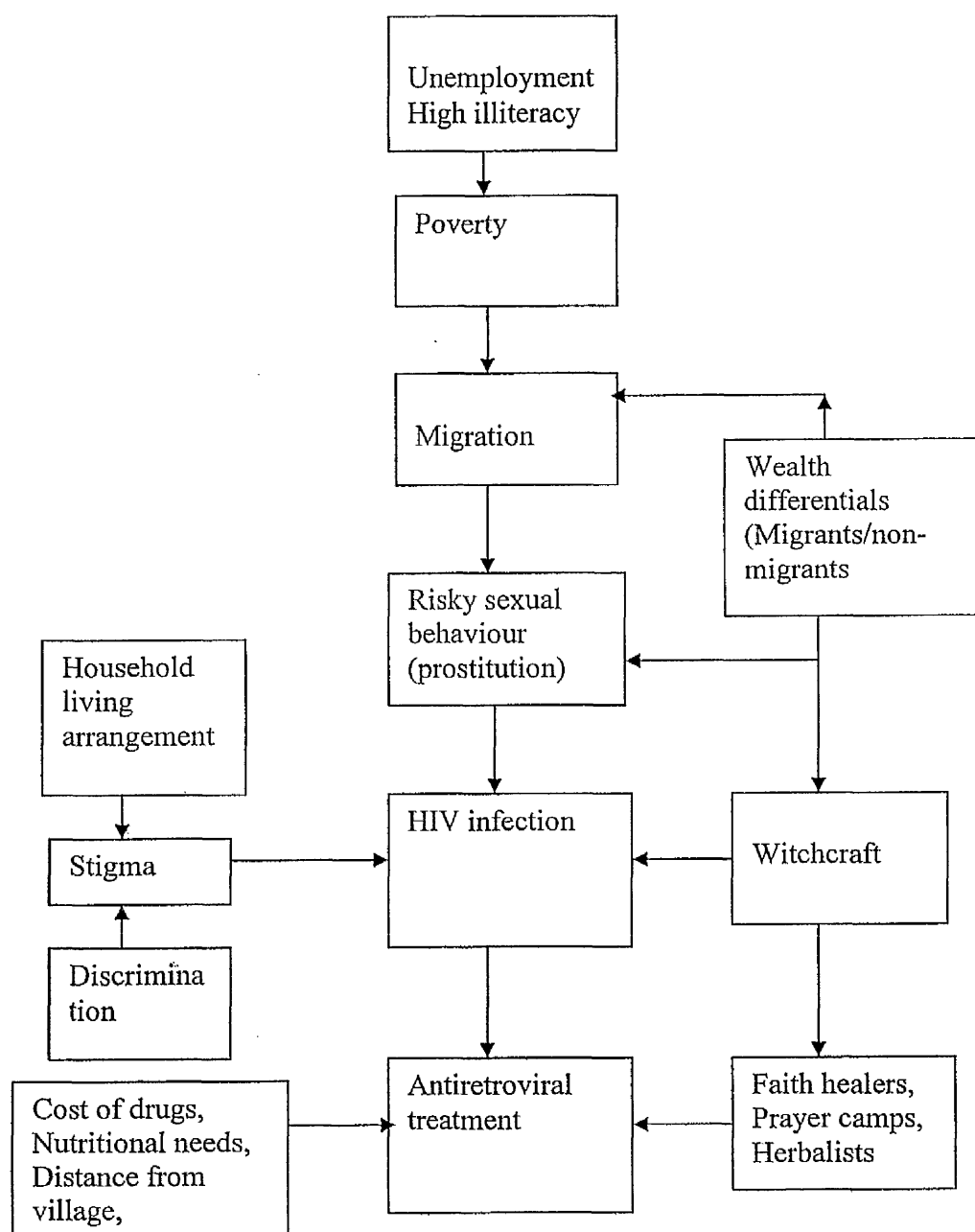
In response to the emergence of the AIDS epidemic in the Krobo district, HIV/AIDS service organisations local, governmental and non-governmental developed as early as 1988 to meet the needs of the people. Although many studies have been conducted into the causes of the spread of the epidemic, the disease continues to spread and current interventions seem to have minimal impact on the magnitude of the spread of the epidemic.

Perspectives on the epidemic spread from the point of view of HIV/AIDS service organisation shows that labour migration and its associated risk behaviour is perceived by AIDS service organisations as the main cause of infection transfer. It was also observed from the interview that witchcraft is believed to cause HIV/AIDS. As a result of the belief in witchcraft, faith healers, prayer camps and herbalist claim to have a cure for the epidemic and this serves as a diversion from the root cause of the epidemic thus hindering prevention and treatment of the epidemic. Another important factor that hinders attempts to stop the spread of the epidemic is that of stigma. The epidemic is so much stigmatised to the extent that families refuse to

disclose the cause of death of their relatives who die of AIDS and counsellors are denied access to PLWHA for follow up checks because people seen to be associated with HIV/AIDS counsellors are suspected to be HIV positive and consequently discriminated against in the community. Lack of employment opportunities and absolute poverty in the Krobo district serves as the push factor for migration. Poverty increases the risk of HIV infection since people engage in risky sexual activities to survive despite the risk of HIV infection.

The next chapter draws on the findings of the theoretical chapter, the modelling chapter, the quantitative chapter and the qualitative result for conclusion and implications for health policy debate.

Figure 8.1 Care providing organisation's model of barriers to effective intervention



CHAPTER NINE

9.0 CONCLUSION

9.1 Introduction

The conclusions of the thesis are summarised into four parts. Part one first recaps the overall aim and objectives of the study by stating the key questions and debates. This is followed by a summary of the findings from each of the exercise the modelling, the quantitative survey and the interviews with agencies. Part two explores how the findings from the modelling section, the quantitative section and insight from service organisations come together to answer the overall aim of the thesis. Part three discusses the contribution of the thesis in terms of new knowledge and insights while the last part presents the implications of the research for policy.

The thesis emerged from key questions and debates surrounding the HIV/AIDS epidemic in sub-Saharan Africa. In the first place, the actual date for the inception of the AIDS epidemic is not known. Also the magnitude and direction of the epidemic spread is uncertain. Though the adoption of preventative behaviour is the only protection against the virus there is no widespread adoption of preventative measures in sub-Saharan Africa despite an almost universal awareness of the serious consequence of AIDS and of sexual transmission of HIV. From the start of the epidemic, cultural explanations has been put forward as the main explanatory concept for understanding the rapid spread and high prevalence in sub-Saharan Africa but cultural explanations alone cannot possibly account for such an exceptionally rapid spread where two-thirds of all cases occur. Worst of all, attempts to investigate the disease epidemiology is hampered by lack of statistics due to the weak surveillance systems.

Given the uncertainties surrounding the disease parameters, the main aim of the thesis is to determine the spatial patterning of the HIV/AIDS epidemic in sub-Saharan Africa and to understand the factors underpinning the rapid and continued spread of

the epidemic in order to inform public health policy debates. This aim is addressed through four specific research objectives.

- To identify source location and dates in sub-Saharan Africa for the inception of the HIV epidemic
- To predict the dates for each country in sub-Saharan Africa when the HIV epidemic will become endemic in a scenario of no intervention
- To identify transmission routes in sub-Saharan Africa at both the start and at the peak of the HIV epidemic
- To explore the processes by which the HIV epidemic is spreading through a case study of the Krobo district in Ghana.
- To identify the implication of the study for public health policy debates on HIV/AIDS.

9.2 Findings of the modelling chapter.

In order to address the first three objectives of the study, the single region and the multiregion space-time epidemic model was employed. The findings of the single region and multiregion model are presented below.

- First it was noted from the single region modelling output that although the first cases of AIDS were reported in the 1980s in Africa, the estimated date for the first HIV infection according to the single region model output occurred in 1974 in Democratic Republic of Congo and Ghana concurrently. This finding is in line with the most widely accepted work by Getchell et al (1987) who isolated HIV-1 in a stored serum sample collected in Zaire, which is now the Democratic Republic of Congo in 1976. This finding is reinforced by the work of Prentice 1986, Sonnet et al, 1987 and Smith et al, 1988 which all point to the fact that HIV in Zaire and Burundi occurred between 1962 and 1976.
- Comparing the date for the first recorded cases in sub-Saharan Africa with the estimated date for first HIV infection for all the countries suggests that HIV had been in existence prior to the date for the first reported cases and might have gone unnoticed due to the length of time between an HIV infection and

final progression to AIDS combined with poor epidemic surveillance systems in sub-Saharan Africa.

- The single region output also indicated that if no cure were found in the immediate future, then HIV/AIDS would become endemic in all sub-Saharan Africa countries between 2011 and 2031.
- The differences in the time between the estimated date of first AIDS cases and the estimated date for the epidemic to become endemic varies from country to country and this may be due to differences in transmission probability.
- The probability of infection transfers between a susceptible and infectives is relatively high and this may account for the rapid spread of the epidemic in sub-Saharan Africa.
- The estimated peak year for the HIV epidemic also shows that the epidemic peaked as early as 1983 in the Democratic Republic of Congo way ahead of other countries in the sub-region such as Zimbabwe where the epidemic peaked at 2003.

The outputs of the multiregion modelling process show the transmission pathway at the start of the epidemic and at peak prevalence.

- In the scenario where the risk population was used as the attraction factor the results show that in West Africa, at the start of the epidemic, there was an infection transfer from Ghana to Cote d'Ivoire and neighbouring Nigeria and other countries in the sub-region but at the peak of the epidemic, there is an infection transfer from Cote d'Ivoire to Ghana and other neighbouring countries in the sub-region.
- When population of the country was used as an attraction factor still Ghana was infecting Cote d'Ivoire and other countries in the sub-region at the start of the epidemic but at the peak HIV prevalence, Nigeria infects all the countries in the West African sub-region.

- In Central Africa on the other hand the epidemic started at the Democratic Republic of Congo and spread to East and Southern Africa. However, at the peak of the epidemic, secondary nodes of infections are formed and South Africa, Tanzania and Mozambique re-infect countries in the sub-region.
- With the population driven specification, Nigeria and South Africa, the two most populous countries in the sub-region, becomes the main nodes from where infection transfers takes place at the peak of the epidemic.

9.3 Findings from the population-based survey of the Krobo district.

In order to explore the processes by which the HIV epidemic is spreading through a case study of the Krobo district in Ghana, population-based study of the district was undertaken. The following findings were made:

- There is significant relationship between HIV prevention methods and preventive behaviours and that the high knowledge of condom use as a preventive measure is more likely to result in more people using condoms than expected. Knowledge about HIV prevention method therefore leads to adoption of preventive behaviour hence lowering the risk to HIV infection.
- There is significant difference between employment status and risky sexual behaviours. Unemployed women were found to be more likely to have sex with other people apart from their regular sex partners and hence increase their risk to HIV infection
- Men who are in employment were more likely to be infected with an STD than those who are unemployed probably because such men may buy sex from commercial sex workers or exploit vulnerable women for sex in exchange for money and other assets
- In terms of income, both women and men who earn lower incomes are more likely to have multiple sex partners; however the lower income earning

women were also less likely to use condom as compared to their male counterparts hence increasing their risk to HIV infection.

The multivariate result shows that among the socio-economic, demographic, knowledge, migration and risk behaviour variables, age, gender, educational status, and the level of partner change were the only factors that were significantly related to condom use. These factors act together to explain the variation in condom use.

- In the first place, it was observed that the younger age group (15-34) are 3.7 times more likely to use condom than those in the older age group (35-49). Age is therefore an important factor determining condom use.
- Secondly, the multivariate result shows that in terms of gender, females were less likely to use condoms (odds =0.34)
- In terms of educational status the result shows that the respondents with no formal education were less likely to use condom (odds=0.39).
- Lastly, respondents who have regular sex partners and those who have not had sex with any other person apart from their regular sex partners in the past twelve months preceding the survey were less likely to use a condom (odds 0.30). Such people may perceive themselves as being less at risk to HIV infection and therefore not use condoms.

9.4 Insight from the HIV/AIDS agencies

In order to probe deeper into the cause of the epidemic spread and reason for continued increase of the epidemic spread HIV/AIDS service-base organisations were interviewed and their opinions are summarised below:

- There is an infection transfer from neighbouring Cote d'Ivoire to the Krobo district in Ghana at the peak of the epidemic since all the first recorded cases of HIV in the district were returned migrants, mainly prostitutes, from Abidjan
- Belief in witchcraft is a major obstacle to the prevention of HIV spread because AIDS is believed to have been caused by witchcraft by family members envious of the wealth acquired by their victims.

- PLWHA in their desperation for cure turn to faith healers, prophets, spiritual churches and herbalist who aggravate the situation by accelerating the progression from HIV to AIDS and early death through prayer and fasting and encouraging PLWHA not to take any medication.
- The HIV/AIDS epidemic is highly stigmatised, a factor which leads to people hiding their status for fear of socio-economic discrimination and hence influencing the spread of the epidemic.
- Living arrangement, distance from health centres makes administration of antiretroviral treatment difficult
- High unemployment, especially among the youth, results in engagement in risky sexual behaviour, especially among girls, which increases their risk to HIV infection
- Low incomes and extreme poverty serve as push factors for women to migrate to high risk areas, such as Abidjan, as prostitutes irrespective of the risk.

9.5 Spatial patterning of the HIV/AIDS epidemic and the reasons for the continued increase in the spread of the epidemic in sub-Saharan Africa

The thesis set out to explain the spatial patterning of the HIV/AIDS epidemic in sub-Saharan Africa and the reasons for the continued increase in the epidemic spread as well as the apparent failure of current interventions despite the universal awareness of HIV/AIDS. The findings from the modelling section together with that of the population-based survey and insight from the service-based agencies helps in answering the overall aim of the study.

The findings of the single region modelling show that HIV/AIDS is estimated to have started in the Democratic Republic of Congo and Ghana concurrently in 1974. This means that the epidemic occurred years earlier before the date of the first recorded AIDS cases and that the epidemic went unnoticed for a very long period of time. The single region model estimated that the epidemic peaked in sub-Saharan Africa between 1983 and 2003 and that the peak occurred earlier in central Africa than the rest of the sub-region. In the scenario of no cure, the epidemic is forecasted to be endemic for all countries in sub-Saharan Africa between 2011 and 2031. The high

transmission probability for some of the countries probably explains why the epidemic spread faster in some countries than others.

The findings from the multiregion modelling show that starting from the two source regions, the Democratic Republic of Congo and Ghana, the epidemic spread to other parts of Africa (figure 5.3a). Ghana serves as the main source of initial infection in West Africa from where HIV is transmitted to other countries in West Africa. From the initial start in Ghana, the virus spread to all countries in West Africa within eight years. However, at the peak HIV prevalence, the influence of Ghana as a start region is lost and for West Africa Cote d'Ivoire serves as the node of infection transfer back to Ghana and other parts of West Africa (figures 5.3b). In the case of Central, East and Southern Africa, from the initial source in D.R.Congo, the virus spread to all parts of the sub-region within ten years. However at the peak HIV prevalence, the influence of D.R.Congo as the main node of epidemic spread is lost. Instead Tanzania becomes a node for infection transfer to Eastern and Southern Africa but the D.R. Congo remains as a node infecting Congo, Gabon, Equatorial Guinea, Cameroon, Angola and Central Africa Republic but the link between D.R.Congo and Namibia is maintained while the infection transfer between D.R.Congo and Chad is redirected between Nigeria and Chad. Similar findings were made when the population driven specification was used but here at the peak of the epidemic, Nigeria infects all countries in West Africa while South Africa infects countries in the sub-region.

The explanation to this observed spatial spread may be based on the literature and the population-based survey of the Krobo district. The review of the literature shows that the migrant labour hypothesis as discussed in the previous chapters provides an insight into the spatial spread of the epidemic in West Africa. Here migrant labour from Ghana to neighbouring countries especially Cote d'Ivoire and Nigeria may have spread the HIV virus from Ghana. However, at the peak of the epidemic, there is a re infection from Cote d'Ivoire and Nigeria back to Ghana. This view is supported by the insight from the service based organisations in the Krobo district that there is an infection transfer from Cote d'Ivoire to Ghana at the peak of the epidemic since most of the first reported cases were returned migrants who were mostly prostitutes from Abidjan (Anarfi, 1993). In the case of South Africa, the most plausible source of infection transfer at the start of the epidemic may have occurred between migrants

workers from neighbouring countries (Chirwa, 1995). However, at the peak of the epidemic, South Africa is re infecting other countries in the sub-region. The pattern of the epidemic spread as seen in Central and Eastern Africa may be understood from the point of view of the military hypothesis where military personnel from all parts of the sub-region engaged in what has been described as the Great African war in the Congo. It is plausible that these soldiers infected or got infected and re infected their native countries after returning from the war (Smallman-Raynor and Cliff, 1991). Although these explanations help to establish the reasons for the initial spread of the HIV virus from the source region across the continent, the epidemic is now localised and continue to spread in countries in sub-Saharan Africa. Interventions put in place are apparently failing despite the awareness about the consequences of HIV infection.

The findings from the population-based survey showed that the reason for the continued spread of the virus is not about lack of knowledge or awareness about HIV spread. This is because knowledge about the epidemic is widespread and those with knowledge about the preventive methods were more likely to practice preventative behaviour. For instance in the Krobo district those who have knowledge about condom as a preventive measure were more likely to use condoms and hence reduce their risk to HIV infection.

The main factors identified as causing the increasing spread of the epidemic through high risk unprotected sex are unemployment, low income levels, gender, educational status and false sense of security among those in stable relations. First, unemployed women were found to be more likely to have sex with other people apart from their regular sex partners and hence increase their risk to HIV infection. Men who are employed were however found to be more likely to be infected with an STD including HIV probably because such men may buy sex from commercial sex workers or exploit vulnerable women for sex in exchange for money and for employment in situations where such men are employers or responsible for recruitments. Another economic factor identified as influencing the epidemic spread is the low incomes in the district. It was found that lower income earners were more likely to have multiple sex partners thus increasing their risk to HIV infection. These lower income earners especially women may sell sex for survival in situation of dire poverty, irrespective of their knowledge about the epidemic and the risk associated with their actions.

The reasons for the apparent failure of the ABC - based interventions may be as a result of gender relations in the Krobo district, low educational attainment and false sense of security which act together as showed by the multivariate analysis. Women were less likely to use condoms. This could be due to the subordinate role of women in the Krobo society. It was also observed that people with no formal education were less likely to use condom as their ability to make informed decision about their sexual life and that of their partners is reduced. Also those in stable relationships are less likely to use condom and this false sense of security may increase the risk of the epidemic spread. Together, these factors might possibly contribute to failure of interventions based on behaviour change.

Insight from the HIV service-based organisation confirms the result of the population-based survey. Service-based organisations identified high unemployment and low incomes in the district as a major factor causing the continued spread of the epidemic as well as failure of current interventions to mitigate the epidemic spread. It emerged from the interview with the service organisations that the high unemployment and lower incomes especially among the youth result in the practice of risky sexual behaviour especially among girls and thus increases their risk to HIV infection. Also extreme poverty resulting from the unemployment situation serves as a push factor for women to migrate to high risk areas such as Abidjan as prostitutes irrespective of the risk involved.

Apart from these socio-economic factors, other cultural factors such as belief in witchcraft, stigma and living arrangement were identified as factors influencing the epidemic spread in the district. First, belief in witchcraft as a cause of AIDS may render interventions based on Information, Education and Communication (IEC) less effective. Also belief in witchcraft diverts PLWHA attention from seeking early medical help to Faith healers, Prophets, Prayer camps and Herbalist and in most cases only seek medical help at the terminal stage of the HIV infection. The result is an increase infection, early progression from HIV to AIDS and finally death. It also emerged from the interview that HIV/AIDS sickness is highly stigmatised. This means that people will not disclose their status for fear of discrimination. Another factor associated with the stigma is that people may not like to go for Voluntary Counselling and Testing (VCT) one of the intervention programmes in place in the

district to mitigate the epidemic spread. Stigma is therefore one of the main factors for the increasing spread of the epidemic as well as the reason for the apparent failure of the VCT intervention programme in the district. Another important factor that emerged from the interview as a significant factor serving as an obstacle against the antiretroviral programme regime which has been introduced on a pilot scale since 2003 has been the nature of living arrangements in the Krobo district. The number of different households living in one house ranges between a minimum of two and a maximum of ten with an average of five. The lack of privacy means that PLWHA would not allow HIV/AIDS coordinators and counsellors to visit them to make follow-ups on the progress and effectiveness of the antiretroviral treatment. This result in the taking of the wrong dosage, discontinue use of the drugs as a result of side effects, poor monitoring and evaluation of the antiretroviral regime and failure of the intervention. Another challenge facing the antiretroviral treatment is the distance to the health centres. Some of the PLWHA are living in remote villages that are not accessible by road. This makes it difficult for them to visit the health centres at regular intervals for check-ups. In the same way, HIV counsellors and coordinators find it difficult to make follow-ups on PLWHA and this makes the administration of the antiretroviral regime problematic.

9.6 New knowledge and insight from the thesis.

Contrary to the view that the HIV virus originated from the D.R.Congo and spread to all parts of Africa (Bygberg, 1983; Getchel et al 1987), the modelling output reveals that the epidemic started both in Ghana and D.R.Congo in 1974. This date is in line with the most generally accepted work which points to HIV-1 circulation in Central Africa in the mid-1970s (Getchel et al (1987).

Secondly, contrary to the view by Kawamura et al (1989) and Anarfi (1993) that the HIV virus spread from Cote d'Ivoire to Ghana at the start of the AIDS epidemic, the result of the study shows that Ghana is the probable source of the HIV virus and that at the start of the epidemic, there was an infection transfer from Ghana to Cote d'Ivoire through migrant workers especially commercial sex workers from the Krobo district in Ghana. At the peak of the epidemic however, there was an infection

transfers from Cote d'Ivoire back to Ghana and to neighbouring countries as shown by the modelling output.

It emerged from the study that the continued spread of the HIV virus and apparent failure of interventions in sub-Saharan Africa cannot be blamed on lack of knowledge about the epidemic. In fact knowledge about the epidemic is very high. The main cause of the continued spread lies mainly in the socio economic and cultural situation peculiar to sub-Saharan Africa in which poverty and gender relations act together to make the people vulnerable to HIV infection.

Lastly, it emerged from the study that interventions in the Krobo district which are based on the behavioural approach base on the philosophy of behaviour change as the issue have limitations on the potential for impact.

9.7 Implication of the study for public health policy debate

The study shows that migration is a significant factors causing the spread of the HIV epidemic and that the observed infection transfer at the peak of the epidemic as shown from the modelling output could be attributed to migration and related risk behaviours of migrants. Further investigation into the push factors for the mobile population is needed to design appropriate intervention to mitigate the epidemic spread rather than focussing resources on ABC prevention strategies. Also interventions should target return migrants from especially highly infested region since these return migrants constitutes special risk group.

In both the quantitative and qualitative analysis unemployment and low incomes were identified as factors influencing the epidemic spread in the Krobo district. However, the HIV/AIDS service organisations in the district have concentrated on cultural and individual behavioural scale of explaining the epidemic spread and hence based their interventions on behaviour change. As part of the HIV prevention programmes, we must go beyond the ABC method and discuss and adopt strategies that will remove the root cause of vulnerability.

The study further shows that there is high knowledge about HIV/AIDS in the Krobo district and risk perception is high among low income earners and people who have ever contracted an STD. However the practice of preventative behaviour is less than expected. The question that needs to be address by governmental and non-governmental organisation is why people are not practicing preventative behaviour despite such high knowledge about HIV. Economic and political context and risk environment in which human individual sexual behaviour takes place should be addressed if interventions are to achieve the desired result.

Persistence of witchcraft-based explanation to the HIV/AIDS epidemic in the Krobo district as perceived in the qualitative study implies that the role of faith healers, prophets and prayer camps in the fight against HIV/AIDS cannot be overlooked. As part of the HIV/AIDS prevention programmes, faith healers, herbalist, and religious bodies may be included in the programme planning stage so that they can be used as a platform for the implementation of preventive programmes instead of relying on the formal health care facilities.

The study shows that stigma plays a very important role in the continued spread of the epidemic in the Krobo district. In a community where stigma is so entrenched, people avoid knowing their status, disclosing their status and seeking treatment and counselling. The issue of how to address stigma therefore becomes a public health policy issue which must be debated if the prevention programmes are to achieve the desired result. Probably in the Krobo community where traditional rulers including chiefs, queen mothers and clan leaders command respect and authority, they should be involved and used as a platform for fighting stigma.

In order for antiviral treatment regimes to be effective, there is the need for regular meetings and follow-ups between PLWHA and HIV/AIDS coordinators and counsellors. However, local living arrangements, distance to health centres and poverty poses a challenge to the implementation of antiretroviral treatment schemes. Living arrangements is such that many households live in one compound house. Lack of privacy in such a highly stigmatised society implies that PLWHA would not like to associate with HIV counsellors. It becomes impossible to monitor the administration of the antiretroviral drugs to find out about efficacy and side effects of the drugs. Such

problems were not anticipated by donor agencies which are mainly from developed countries where such living conditions do not exist. Moreover, some of the PLWHA are living in villages far from the health centres and cannot be reached by HIV counsellors. The inaccessibility makes follow-ups in such situations difficult. In some situation the PLWHA are not able to meet their nutritional needs and may have adverse reaction from the drugs. These challenges are issues which should be taken into account if the antiretroviral treatment regime is to make any impact if any on the HIV/AIDS situation in the Krobo district and in Ghana as a whole.

It is evident from the above that the rapid and continued spread of the epidemic in the Krobo district and the apparent failure of current interventions to halt the spread of infection despite the universal awareness of the consequences of AIDS cannot be explained solely by individual human behaviour as proposed by initial interventions. It is rather the factors and the conditions that make people vulnerable to the HIV/AIDS infection that need to be tackled. Interventions such as safer sex, abstinence and condom use as advocated by Non Governmental Organisation are bound to have a very minimal impact until and unless the basic and fundamental factors making the people vulnerable to the epidemic are addressed. HIV/AIDS is therefore seen as a disease of poverty and as such Governments and Non Governmental Organisations should move beyond behaviour change interventions and have both political and economic will to tackle the root cause of poverty. Until and unless the issue of poverty is addressed alongside other intervention policies, HIV/AIDS may remain a major public health problem especially in sub-Saharan Africa and may continue to undermine the developmental efforts of governments in the sub-Saharan Africa sub-region.

9.8 Implications of the Modelling Process for Health Policy and Disease Control

An understanding of the magnitude and directionality of the HIV/AIDS epidemic in sub-Saharan Africa as well as the uncertainty surrounding the parameters affecting its spread is important for planning disease control strategies and for health policy. The section that follows discusses the implications of the modelling process for disease control and health policy in a continent where the outcome of the epidemic is dire.

Epidemic dates and statistics generated by the single region model by every country in sub-Saharan Africa will help policy makers to plan for disease control. The single region model output shows the estimated date of first HIV infection in all countries was years before the date of first recorded AIDS cases. This means that the epidemic went unnoticed for a very long period of time. There was therefore no warning time for planning for interventions and coupled with poor epidemic surveillance in sub-Saharan Africa the epidemic spread rapidly in sub-Saharan Africa as compared to other parts of the world. The estimated date for the inception of the epidemic from the single region model and the date for the first reported cases in each country should serve as a warning period for planning for interventions in future epidemics of this kind.

The output of the single region model also shows the forecast date the epidemic will become endemic in each country. The estimated date for the epidemic to become endemic may give an indication of how early interventions should be put in place before the epidemic become endemic. The size of the risk population estimated by the single region model gives an indication to policy makers and government to allocate required resources for planning intervention to control the epidemic spread. Another important output of the single region model is the estimation of transmission probability for the various countries in the sub-Saharan region. The relatively high estimates for transmission probability in sub-Saharan African countries where the main mode of spread of HIV is heterosexual indicate potential for increasing epidemic spread. One of the benefits of the single region output is the fact that health policy makers can make predictions by changing the disease parameters in the single region model with the aim of assessing the epidemic outcome and the interventions needed if the epidemic spread faster than predicted. The adjustment of parameters such as the transmission probability and the size of the risk population can manipulate this model to present specific scenarios regarding the course of the epidemic and thus inform policy makers on specific intervention to put in place if the course of the epidemic changes.

A detailed analysis of the output of the multiregion model gives information on the transmission pathways of the epidemic both at the start and at the peak HIV prevalence. In order to prevent the epidemic spread, the main transmission routes at

the peak of the epidemic should be targeted and there should be comprehensive HIV prevention policy among countries in sub-Saharan Africa. Taking into consideration the increase travel opportunities among countries in Sub-Saharan Africa as well as movement of populations across borders due to war and conflicts, indications about the major nodes of transmission and transmission pathways becomes crucial in planning interventions to reduce the risk of infection both to the migrant populations and populations of the destination countries. In the case of Ghana, migrants returning from the highly infected countries such as Cote d'Ivoire and Nigeria should be targeted for interventions before the epidemic is spread to migrant home countries. The modelling process predicted the source regions, dates of initial infection, source of infections at peak prevalence and time taken by the epidemic to traverse through the continent. This information is needed for planning effective and collective interventions by all governments in the sub-region. With the coming into force the Economic Community of West African States (ECOWAS) treaty in West Africa and the expected free movement of people and goods across borders, there is the need for common health policies by all countries in the sub-region with regards to HIV/AIDS so as to prevent the spread of the epidemic across countries in the West African sub-region and beyond.

9.9 Issues related to the modelling strategy adopted and possible alternatives

Although the space-time epidemic modelling method used in this study meets the objective of the study, it is worth noting that there are conceptual uncertainties in the model structures, assumptions and specifications. For example, some of the epidemiological parameters in the model are estimates taken from published sources. For example the duration of the period of communicability ($D=2$ years) and antigen suppression ($A=4$ years) are drawn from research into viral abundance and antigen concentration in patients after HIV infection (Anderson and May, 1991). Similarly the average value of $r=10$ partners per year for those at high risk is taken from surveys of sexual behaviour made at different times during the course of the epidemic (Knox et al 1993, Johnson et al 1994). These variables taken from published sources impose a limit on the confidence in the model output.

To overcome the uncertainties about the model parameters and to apportion the importance of the input parameters with respect to the model output, sensitivity analysis (SA) was conducted (Saltelli et al, 2000). The analysis involved the selection of ranges of epidemiological parameters, generation of a sample from the ranges, evaluation of the model for each element of the sample and to find the optima that are generated by an iterative routine that minimises the value of the goodness of fit statistic (χ^2). This process helped in obtaining the optimum values for an estimate of transmission probability (β_i), an estimate of the population at risk to HIV infection in each country (n_i) and an estimate of the date of the initial HIV infection in each country ($t = 0$). To further reduce the uncertainties about the model output, the epidemic was simulated to have started at different locations and both AIDS epidemic size and total population of each country were set as the main attraction factor for the multiregion simulation. It was observed that using both the AIDS epidemic size and the total population as an attraction factor yielded the same result at the start of the epidemic. In both scenarios, Ghana and D.R Congo were predicted as the probable source of the epidemic at its start. Sensitivity analysis was used to validate the model and to test both the quality of the model and the robustness of the model based inference.

Although the space-time epidemic model was used in modelling the HIV/AIDS epidemic, there are alternative methods that could have been used. In the first place, retrospective serial analysis based on earlier reported HIV/AIDS cases could have been used to predict the spatial patterning of the epidemic. Though retrospective analysis has been used for other illnesses, in the case of HIV/AIDS the causal agent the HIV virus was not discovered until 1983. Secondly serological analysis based on tissue samples prior to the isolation of the HIV virus proved inconclusive in identifying cases of HIV/AIDS and the long incubation period of the epidemic makes retrospective analysis less efficient method of studying the nature of the epidemic spread especially in sub-Saharan Africa where epidemic surveillance is less developed. Secondary, structural modelling methods using time-series cases of HIV/AIDS cases for each country offers an alternative method in predicting the spatial patterning of the epidemic but this method, though less complicated, comes with its own weakness. In the first place the weak surveillance system and secondly

the paucity of data on HIV/AIDS in sub-Saharan African countries makes it less useful method of studying the patterning of the epidemic spread.

9.10 Strengths and limitations of the thesis

The main strength of this thesis is in its methodological approach. Here, modelling methods, quantitative and qualitative techniques were used to explore the complex issue of HIV/AIDS epidemiology in sub-Saharan Africa where the reasons for the continued spread of the epidemic has not been fully explored. Whereas the modelling section predicted and estimated the disease parameters and the spatial patterning of the epidemic spread at the sub-Saharan African level, the quantitative section of the population based survey of the Krobo district at the sub-national level explored the socio-demographic, economic and cultural and political factors explaining the observed patterning of the epidemic spread as depicted in the modelling output. The qualitative aspect of the study based on the survey of the HIV/AIDS service organisation provided deeper understanding for the reasons for the continued spread of the epidemic and the apparent failure of current interventions to mitigate the epidemic spread. This methodology provided a holistic view of the HIV/AIDS epidemiology and thus provided a better understanding of the epidemic spread in sub-Saharan Africa.

The major limitation of the thesis is that of the quality of HIV/AIDS data in some sub-Saharan African countries. The modelling data was obtained from UNAIDS AIDS incidence data obtained from the antenatal sentinel site. While recorded AIDS incidence in Western European countries is estimated to be between 75 percent and 100 percent of all cases, in sub-Saharan Africa AIDS incidence data is less complete and their reliability varies with the comprehensiveness of the country's surveillance system (UNAIDS, 2005). It is however worth noting that sentinel site data from antenatal attendees are the closest estimates and hence the best data to be used. It is also worth noting that the findings of the population-based survey explains the reasons for the epidemic spread in the Ghanaian context and not sub-Saharan Africa as a whole though some of the findings and policy implications may apply to other countries of sub-Saharan Africa.

9.11 Directions for future research

Although the study shows that Ghana is the probable source of HIV/AIDS infection in West Africa at the start of the epidemic, there is a lack of research into the origin in Ghana and the spatial spread of the epidemic within the ten regions in Ghana. Exploring the possible link between the primate population and the Krobo tribe in the Krobo ancestral home 'Klowem' and the zoonotic transfer of the HIV virus from primates to humans is an issue that need further research. Secondly the link between the construction workers for the building of the Akosombo Hydro-electric dam in the early sixties, who were mainly Americans and Canadians, and the possible sexual mixing and transfer of the HIV virus into the Krobo population also needs to be explored. Lastly, modelling the HIV/AIDS epidemic within the ten regions in Ghana with the Krobo region as a probable source region will provide insight into the transmission pathways within the ten regions in Ghana both at the start of the epidemic and at peak prevalence. This will help policy makers to mitigate the epidemic spread within Ghana.

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Appendix 1 :A Multiregion Epidemic Model For HIV/AIDS

Define n_i as the population at risk to HIV infection in each region i . Then, these populations are disaggregated into the following state variables: $x_i, \forall i$, those who are susceptible; $y_i, \forall i$, those who are infectious; $w_i, \forall i$, those who are HIV+ but with antigen levels too low to be communicable to others (Nowak *et al*, 1991); and, $z_i, \forall i$, all those ever with AIDS. Amongst these, the susceptibles and infectives are taken to be freely circulating individuals ($x_i + y_i, \forall i$) who, at any point in time, are available to form partnerships with each other. Transitions between these states are determined by the following disease parameters: $\beta_i, \forall i$, the probability a partnership between an infective in region i and a susceptible results in the transmission of HIV; D , the period of communicability for HIV; and A , the period from the cessation of communicability to the onset of AIDS. The model describing the epidemic process is defined by the following set of differential equations

$$dx_i/dt = - [x_i/(x_i + y_i)] \sum_j \beta_j y_j s_{ji}, \quad \forall i, \quad (A1)$$

$$dy_i/dt = [x_i/(x_i + y_i)] \sum_j \beta_j y_j s_{ji} - y_i/D, \quad \forall i, \quad (A2)$$

$$dw_i/dt = y_i/D - w_i/A, \quad \forall i, \text{ and} \quad (A3)$$

$$dz_i/dt = w_i/A, \quad \forall i. \quad (A4)$$

For each region within this system $x_i/(x_i + y_i) \sum_j \beta_j y_j s_{ji}$ is the incidence of HIV infection, y_i/D is the number of infectives who cease to be communicable, and w_i/A is the incidence of AIDS.

The rates $s_{ji}, \forall ji$, which provide the essential spatial mechanism for the transferring HIV infection between the regions, are given by

$$s_{ji} = r \{ p_{ji} + [(x_i + y_i) p_{ij} / (x_j + y_j)] \} / 2, \quad \forall ji. \quad (A5)$$

Each of these terms is the expected number of partnerships among those circulating freely in i with a specific j -individual of the same status and where r is the average rate of partner acquisition for the risk population of the entire system. In addition, each p_{ij} is the probability an individual resident in region i chooses region j as a travel destination. Specifically, we define the internal travel probability, $p_{i=j}$, as a quantity that is given exogenously, then

$$q_i = 1 - p_{i=j}, \quad \forall i. \quad (A6)$$

defines the probability an i -individual chooses to travel to some external region.

Equivalent probabilities for travel to a specified region j are generated according to a negative exponential function of the form $e^{-\lambda d_{ij}}$, where d_{ij} is the distance between the centres of regions i and j . Here, λ is a decay parameter calibrated in the interval $[0, \infty]$ to represent the sensitivity of travel to this spatial separation. Accordingly,

$$a_{ij} = e^{-\lambda d_{ij}}, \quad \forall ij, \quad (A7)$$

is a measure of the accessibility of region i to region j . Because $p_{i=j}$ is known, these accessibilities are subject to the condition

$$a_{i=j} = 0, \quad \forall i=j. \quad (\text{A8})$$

Moreover, define m_j as the travel opportunities present in region j such that the term $a_{ij}m_j$ is taken to represent the attractiveness of j as a destination for those travelling from region i . Consequently, the totals

$$W_i = \sum_j a_{ij}m_j, \quad \forall i, \quad (\text{A9})$$

each measure the attractiveness of the entire system for those travelling from i . If regional contact probabilities are taken to be proportional to their accessibility levels, then the terms

$$p_{i \neq j} = q_i a_{ij}m_j / W_i, \quad \forall i \neq j \quad (\text{A10})$$

each represent the probability a journey started from region i will terminate in the external region j .

Fuller discussions of the properties of this model may be found in Thomas (1999a, 1999b) and Thomas and Smith (2000).

Appendix 2 Programme CuFit.for

```
c  BETA CuFIT PROGRAM
real c(50),beta1,c1(50),c2(50)
common/one/r,pc,pd,u,theta,n,z
open(10,file='p:\resout.lst')
open(5,file='p:\concases.txt')
open(3,file='p:\bcseries.dat')
write(*,*) 'Type: y(t=0) & n'
read(*,*) z,n
u=0
theta=0
r=10
pd=4
pc=2
write(10,*)'      BETA FITTED TO CuAIDS(t)'
write(10,199)r,pc,pd,u,theta,n,z
199  format(/'Constants: r= ',f5.1,' D1= ',f4.2,' D2= ',f4.2,
+' u= ',f6.4,' theta= ',f6.4/'Pop= ',i9,' y(t=0)= ',f5.1/)
m=15
j=ifix(pc+pd)
l=j
do 1 i=1,m
read(5,197)c(i)
1  c1(i)=c(i)
197  format(f6.1)
write(*,*)(c(i),i=1,m)
do 4 j=1,m
c(j)=c(j)+c(j-1)
write(10,198)j
198  format(/'      Fit for t= ',i2)
if(j.lt.m) goto 4
beta1=0.1
if(c(j).eq.0) beta2=0
if(c(j).eq.0) aids=0
if(c(j).eq.0) write (10,202) beta2,aids,c(j)
202  format(' k= 0 beta ',f10.5,' faids ',f12.2,' cucases ',f7.1)
if(c(j).eq.0) goto 5
beta2=(faids(beta1,j)*beta1)/c(j)
do 2 k=1,50
beta3=beta2+((c(j)-faids(beta2,j))*(beta2-beta1))/
+(faids(beta2,j)-faids(beta1,j))
if(beta3.lt.beta2.and.faits(beta3,j).gt.faits(beta2,j))
+beta3=0.1
beta1=beta2
beta2=beta3
aids=faids(beta2,j)
write(10,201) k,beta2,aids,c(j)
write(*,201) k,beta2,aids,c(j)
```

```

201 format('k=',i2,'beta ',f10.5,' faids ',f12.2,' cucases '
    +,f7.1)
2   if(abs(c(j)-aids).lt.0.1) goto 3
3   continue
5   write(3,195) j,beta2,c(j)
195 format(i4,f8.5,f7.1)
4   continue
    chi=0
    do 6 i=1,m
      c2(i)=faids1(beta2,i)
      if(c2(i).eq.0) goto 6
      chi=chi+(((c1(i)-c2(i))**2)/c2(i))
6   continue
    write(10,196) chi
    write(*,196) chi
196 format('Chi**2 = ',f10.2)
    stop
    end
    function faids(beta,j)
    common/one/r,pc,pd,u,theta,n,z
    nn=n
    x=n-z
    y=z
    w=0
    vinc=0
    dinc=0
    ainc=0
    cuainc=0
    dx=0
    dy=0
    dw=0
    do 1 l=1,j
      n=x+y
      if(x.le.0) x=0
      if(y.le.0) y=0
      if(n.le.0) dx=0
      if(n.le.0) dy=0
      if(n.le.0) goto 2
      dx=(u*n)-((beta*r*x*y)/n)-(theta*x)
      dy=((beta*r*x*y)/n)-(y/pc)
      dw=(y/pc)-(w/pd)
      vinc=((beta*r*x*y)/n)
2     dinc=(y/pc)
      ainc=(w/pd)
      cuainc=cuainc+ainc
      if(l.gt.6.and.ainc.le.0) goto 3
      x=x+dx
      y=y+dy
      w=w+dw
1     continue

```

```

3  continue
   faids=cuainc
   n=nn
   return
end
function faids1(beta,j)
common/one/r,pc,pd,u,theta,n,z
nn=n
x=n-z
y=z
w=0
vinc=0
dinc=0
ainc=0
dx=0
dy=0
dw=0
do 1 l=1,j
n=x+y
if(x.le.0) x=0
if(y.le.0) y=0
if(n.le.0) dx=0
if(n.le.0) dy=0
if(n.le.0) goto 2
dx=(u*n)-((beta*r*x*y)/n)-(theta*x)
dy=((beta*r*x*y)/n)-(y/pc)
dw=(y/pc)-(w/pd)
vinc=((beta*r*x*y)/n)
2  dinc=(y/pc)
   ainc=(w/pd)
   if(l.gt.6.and.ainc.le.0) goto 3
   x=x+dx
   y=y+dy
   w=w+dw
1  continue
3  continue
   faids1=ainc
   n=nn
   return
end

```

Appendix 3 Programme Com.for

```
c  program com
   real c(100),n
   open(10,file='p:resout.lst')
   open(9,file='p:ts.dat')
   open(3,file='concases.txt')
   write(10,*)'      COMMUNITY MODEL FIT'
   j=50
   do 10 i=1,j
10  c(i)=0
      write(*,*)'Type y(t=0)'
      read(*,*) z
      m=16
      do 11 i=2,m+1
11  read(3,*) c(i)
      u=0.0
      theta=0.0
      r=10.0
      beta=0.11459
      pc=2
      pd=4
      n=150000
      x0=1/(beta*r*pc)
      ctheta=u/x0
      k=m+1
      write(10,203) beta,r,pc,pd,n,m,z
203  format('/beta =',f7.5,' r =',f4.1,' D1 =',f4.1,' D2 =',f4.1,
      +/'Pop =',f10.1,' Cases end at t=',i3,' y(t=0)=',f5.1/)
      write (10,204)x0,ctheta
204  format('x0 =',f8.2,' crit theta =',f7.5)
      x=n-z
      y=z
      w=0
      vinc=0
      dinc=0
      ainc=0
      dx=0
      dy=0
      dw=0
      sumf=0
      sumh=0
      suma=0
      chi=0
      k=0
      do 1 l=1,j+1
      ll=l-1
      if(x.le.0) x=0
      if(y.le.0) y=0
      write(10,200) ll,x,y,vinc,ainc,c(l)
```

```

        write(9,201) ll,x,y,ainc,c(l)
201  format(i3,4f9.1)
200  format('t= ',i2,' x ',f9.1,' y ',f9.1,' vinc ',f7.1,' ainc ',f7.1,
        +' cases ',f7.1)
        if(ll.le.m) sumf=sumf+ainc
        if(ll.le.m) sumh=sumh+vinc
        if(ll.le.m) suma=suma+c(l)
        if(ainc.eq.0) goto 4
        if(ll.le.m) k=k+1
        if(ll.le.m) chi=chi+(((c(l)-ainc)**2)/ainc)
4    continue
        n=x+y
        if(n.le.0) dx=0
        if(n.le.0) dy=0
        if(n.le.0) goto 2
        dx=(u*n)-((beta*r*x*y)/n)-(theta*x)
        dy=((beta*r*x*y)/n)-(y/pc)
        vinc=((beta*r*x*y)/n)
2    dw=(y/pc)-(w/pd)
        dinc=(y/pc)
        ainc=(w/pd)
        if(l.gt.15.and.ainc.lt.1.0) goto 3
        x=x+dx
        y=y+dy
        w=w+dw
1    continue
3    continue
        write(10,202) sumf,suma,chi,k,sumh
202  format('CuFAIDS ',f8.1,' CuAIDS ',f8.1,' Chi**2 ',f10.2,'( ',i2,
        +'df) CuHIV ',f12.2)
        stop
        end

```

Appendix 4 Programme Airfit.for

c SI AIR PROGRAM

```
real x(50),y(50),p(50,50),pp(50,50,2),w(50,2),d(50,50),n(50),
+a(50,50,2),ro(50),rro(50,50),so(50),c(50,50),inc(50),dinc(50),
+dx(50),dy(50),dy2(50),d2y(50),na(50,2),qa(50,2),dam(2),
+ww(50),ainc(50),dw(50),st(50),ed(50)
integer jj(50)
common/one/s(50,50),m,beta(50)
open(10,file='p:\resout.lst')
open(9,file='p:\ts.dat')
open(11,file='p:\af5.txt')
open(8,file='p:\space.lst')
write(*,209)
write(10,209)
209 format(' SI AIR MODEL OUTPUTS',/)
write(*,200)
200 format('Type r,D1,D2,m-regions')
read(*,*)r,pc,pd,m
write(10,*) ' PARAMETER VALUES'
write(10,210) r,pc,pd
210 format('r =',f4.1,' D1 =',f4.1,' D2 =',f4.1)
write(*,*)( 'Type lambda - air then local')
read(*,*) (dam(i),i=1,2)
do 604 i=1,2
write(8,601) i,dam(i)
604 write(10,601) i,dam(i)
601 format('lambda(',i1,') ',f5.2)
do 1 i=1,m
read(11,220)x(i),y(i),na(i,2),n(i),beta(i),qa(i,2),st(i),ed(i)
1 write(10,221) i,beta(i),n(i)
221 format('Region ',i3,' beta ',f8.5,' Risk Pop ',f8.1)
220 format(2f6.2,f6.2,f8.1,f8.5,f5.2,2f5.1)
write(8,500) (qa(i,2),i=1,m)
500 format(9f6.2/9f6.2/9f6.2/9f6.2/9f6.2)
do 197 i=1,m
qa(i,1)=1-qa(i,2)
197 na(i,1)=qa(i,1)*na(i,2)
do 2 i=1,m
do 2 j=1,m
p(i,j)=0
d(i,j)=sqrt(((x(i)-x(j))**2)+((y(i)-y(j))**2))
do 2 l=1,2
a(i,j,l)=exp(-dam(l)*d(i,j))
if(i.eq.j.and.l.eq.1) a(i,j,1)=0
2 if(na(i,l).eq.0) a(i,j,l)=0
do 3 l=1,2
do 3 i=1,m
w(i,l)=0
do 3 j=1,m
```

```

3  w(i,l)= w(i,l)+(a(i,j,l)*na(j,l))
   do 4 i=1,m
   do 4 j=1,m
   do 4 l=1,2
4  pp(i,j,l)=a(i,j,l)*na(j,l)
   write(*,*) '    ATTRACTIVENESS STATISTICS'
   write(10,*) '    ATTRACTIVENESS STATISTICS'
   do 5 i=1,m
5  write(10,602)i,na(i,1),qa(i,1),w(i,1),(pp(i,j,1),j=1,m)
602  format('Region ',i3,' Air ',f8.2,' tp ',f4.2,' W1(i) ',f8.2/
+ 'W1(i,j) ',9f8.2/8x,9f8.2/8x,9f8.2/8x,9f8.2/8x,9f8.2)
   do 605 i=1,m
605  write(10,203)i,na(i,2),qa(i,2),w(i,2),(pp(i,j,2),j=1,m)
203  format('Region ',i3,' Pop ',f8.2,' tp ',f4.2,' W2(i) ',f8.2/
+ 'W2(i,j) ',9f8.2/8x,9f8.2/8x,9f8.2/8x,9f8.2/8x,9f8.2)
   do 6 l=1,2
   do 6 i=1,m
   do 6 j=1,m
   if(w(i,l).eq.0) pp(i,j,l)= 0
   if(w(i,l).eq.0) goto 6
   pp(i,j,l)=(a(i,j,l)*na(j,l))/w(i,l)
6  continue
   do 603 l=1,2
   do 603 i=1,m
   do 603 j=1,m
603  p(i,j)=p(i,j)+(qa(i,l)*pp(i,j,l))
   write(10,*) '    TRAVEL PROBABILITIES'
   do 199 l=1,2
   do 199 i=1,m
199  write(10,198) l,i,(pp(i,j,l),j=1,m)
198  format('M ',i1,' Rg',i3,' p(i,j) ',9f7.4/17x,9f7.4/17x,9f7.4
+ /17x,9f7.4/17x,9f7.4)
   do 7 i=1,m
7  write(10,204)i,(p(i,j),j=1,m)
204  format('Region',i3,' p(i,j) ',9f7.4/17x,9f7.4/17x,9f7.4
+ /17x,9f7.4/17x,9f7.4)
   tn=0
   do 606 i=1,m
606  tn=tn+n(i)
   do 8 i=1,m
   so(i)=0
   do 8 j=1,m
   s(i,j)= p(i,j)+(n(j)*(p(j,i)/n(i)))
   s(i,j)= s(i,j)/2
8  so(i)=so(i)+s(i,j)
   write(*,*) '    CONTACT EXPECTATIONS'
   write(10,*) '    CONTACT EXPECTATIONS'
   do 9 i=1,m
   write(*,205)i,(s(i,j),j=1,m)
9  write(10,205)i,(s(i,j),j=1,m)

```

```

write(*,208) (so(i),i=1,m)
write(10,208) (so(i),i=1,m)
208 format('      s(i) ',9f7.4/17x,9f7.4/17x,9f7.4
+/17x,9f7.4/17x,9f7.4)
205 format('Region',i3,' s(i,j) ',9f7.4/17x,9f7.4/17x,9f7.4
+/17x,9f7.4/17x,9f7.4)
do 10 i=1,m
do 10 j=1,m
10 rro(i,j)=beta(j)*r*s(j,i)*pc
do 11 j=1,m
ro(j)=0
do 11 i=1,m
11 ro(j)=ro(j)+ rro(i,j)
write(*,*) '      REPRODUCTION NUMBERS'
write(10,*) '      REPRODUCTION NUMBERS'
do 12 i=1,m
write(*,206)i,(rro(i,j),j=1,m)
12 write(10,206)i,(rro(i,j),j=1,m)
206 format('Region',i3,' R0(i,j)',9f7.4/17x,9f7.4/17x,9f7.4
+/17x,9f7.4/17x,9f7.4)
write (*,207) (ro(j),j=1,m)
write (10,207) (ro(j),j=1,m)
207 format('      R0(j)',9f7.4/17x,9f7.4/17x,9f7.4
+/17x,9f7.4/17x,9f7.4)
rr=0
do 13 i=1,m
13 rr=rr+(ro(i)*(n(i)/tn))
write(*,211) rr
write(10,211) rr
211 format('      R0 = ',f7.4)
do 14 i=1,m
ro(i)=0
do 14 j=1,m
c(i,j)= r*s(i,j)*n(i)
14 ro(i)=ro(i)+c(i,j)
write(*,*) '      CONTACT FREQUENCIES'
write(10,*) '      CONTACT FREQUENCIES'
do 15 i=1,m
write(*,212)i,(c(i,j),j=1,m)
15 write(10,212)i,(c(i,j),j=1,m)
212 format('Region',i3,' c(i,j) ',9f8.0/17x,9f8.0/17x,9f8.0
+/17x,9f8.0/17x,9f8.0)
write (*,213) (ro(j),j=1,m)
write (10,213) (ro(j),j=1,m)
213 format('      c(i) ',9f8.0/17x,9f8.0/17x,9f8.0
+/17x,9f8.0/17x,9f8.0)
write(*,214)
write(10,214)
214 format('      TIME SERIES')
write(*,*) 'Type number of time intervals'

```

```

read(*,*) mm
ft=0
do 16 i=1,m
x(i)=n(i)
y(i)=0
ww(i)=0
inc(i)=0
dinc(i)=0
ainc(i)=0
dx(i)=0
dy(i)=0
dw(i)=0
d2y(i)=0
16  dy2(i)=0
write(*,*) 'Type the number of start regions'
read(*,*) is
do 8000 isi=1,is
write(*,*) 'Type start region number and z'
read(*,*) i,z
write(8,8001) i
y(i)=z
8000 x(i)=n(i)-z
8001 Format('selected start region',i4)
do 17 l=1,mm
ll=l-1
write(*,215) ll
write(10,215) ll
215  format('Time step ',i4)
do 18 i=1,m
if(x(i).le.0) x(i)=0
if(y(i).le.0) y(i)=0
n(i)=x(i)+y(i)
18  write(10,216) i,x(i),y(i),inc(i),ainc(i),n(i)
216  format('Region ',i3,' x=',f10.2,' y=',f10.2,' vinc=',f8.2,
+' ainc=',f8.2,' Pop=',f8.2)
write(9,450) (ainc(i),i=1,m)
450  format(50f10.2)
do 22 i=1,m
g=0
do 23 j=1,m
if(i.eq.j) goto 23
g1=y(j)*s(j,i)
if(g1.gt.g) jj(i)=j
if(g1.gt.g) g=g1
23  continue
22  continue
do 25 i=1,m
if((y(i)-dy(i)).lt.st(i).and.y(i).ge.st(i))
+write(8,218) i,y(i),ll,jj(i)
25  if((y(i)-dy(i)).lt.st(i).and.y(i).ge.st(i))

```

```

+write(10,218) i,y(i),ll,jj(i)
218 format('Region ',i3,' y= ',f10.2,' time ',i4,' Source region ',i3)
do 26 i=1,m
do 26 j=1,m
26 s(i,j)=(p(i,j)+(n(j)*p(j,i)/n(i)))/2
do 19 i=1,m
dy2(i)=dy(i)
dx(i)=-((r*x(i)*tr(y,i))/n(i))
dy(i)=-((r*x(i)*tr(y,i))/n(i))-(y(i)/pc)
dw(i)=(y(i)/pc)-(ww(i)/pd)
inc(i)=-((r*x(i)*tr(y,i))/n(i))
dinc(i)=(y(i)/pc)
19 ainc(i)=(ww(i)/pd)
do 20 i=1,m
if(dy2(i).lt.0.and.dy(i).ge.0) write(10,217) i,y(i),ll
if(dy2(i).gt.0.and.dy(i).le.0) write(10,217) i,y(i),ll
if(dy2(i).gt.0.and.dy(i).le.0) f=ed(i)-ll
if(dy2(i).gt.0.and.dy(i).le.0) write(8,481) i,y(i),ll,f,jj(i)
if(dy2(i).gt.0.and.dy(i).le.0) ft=ft+abs(ll-ed(i))
if(d2y(i).lt.0.and.(dy2(i)-dy(i)).ge.0) write(10,217) i,y(i),ll
20 if(d2y(i).gt.0.and.(dy2(i)-dy(i)).le.0) write(10,217) i,y(i),ll
217 format('Region ',i3,' y= ',f10.2,' time ',i4)
481 format('Region ',i3,' y= ',f10.2,' time ',i4,' dif,f4.1,
+' source region',i3)
do 21 i=1,m
x(i)=x(i)+dx(i)
y(i)=y(i)+dy(i)
ww(i)=ww(i)+dw(i)
21 d2y(i)=dy2(i)-dy(i)
17 continue
write(8,325) ft
325 format('Fit= ',f6.1,' years')
stop
end
function tr(y,i)
common/one/s(50,50),m,beta(50)
real y(m)
tr=0
do 1 j=1,m
1 tr=tr+(beta(j)*y(j)*s(j,i))
return
end

```

Appendix 5a :Output from Space. 1st for countries of sub-Saharan Africa – Start

region 1 (Ghana)

```

lambda(1) 0.30
lambda(2) 7.00
0.95 0.95 0.95 0.95 0.95 0.95 0.95 0.95 0.95
0.95 0.95 0.95 0.95 0.95 0.95 0.95 0.95 0.95
0.95 0.95 0.95 0.95 0.95 0.95 0.95 0.95 0.95
0.95 0.95 0.95 0.95 0.95 0.95 0.95 0.95 0.95
0.95 0.95 0.95 0.95 0.95 0.95
selected start region 1
Region 1 y= 115.33 time 1 Source region 28
Region 39 y= 19.59 time 6 Source region 1
Region 28 y= 145.35 time 8 Source region 1
Region 32 y= 154.14 time 8 Source region 1
Region 42 y= 11.69 time 8 Source region 1
Region 34 y= 32.97 time 9 Source region 1
Region 37 y= 1.05 time 9 Source region 1
Region 38 y= 8.25 time 10 Source region 1
Region 24 y= 2.75 time 11 Source region 32
Region 27 y= 1.44 time 11 Source region 1
Region 29 y= 28.31 time 11 Source region 28
Region 30 y= 2.67 time 11 Source region 29
Region 33 y= 0.69 time 11 Source region 29
Region 41 y= 43.43 time 11 Source region 1
Region 15 y= 22.00 time 12 Source region 20
Region 35 y= 5.35 time 12 Source region 29
Region 36 y= 1.50 time 12 Source region 29
Region 4 y= 77.65 time 13 Source region 32
Region 25 y= 19.15 time 13 Source region 32
Region 40 y= 80.45 time 13 Source region 32
Region 20 y= 229.13 time 14 Source region 2
Region 26 y= 31.30 time 14 Source region 32
Region 2 y= 186.96 time 15 Source region 20
Region 16 y= 104.19 time 15 Source region 4
Region 19 y= 21.81 time 15 Source region 32
Region 6 y= 158.15 time 18 Source region 16
Region 14 y= 57.44 time 18 Source region 4
Region 39 y= 5066.51 time 18 dif 0.0 source region 1
Region 3 y= 19.10 time 19 Source region 16
Region 8 y= 50.65 time 19 Source region 4
Region 31 y= 12.28 time 19 Source region 16
Region 9 y= 23.18 time 20 Source region 4
Region 10 y= 133.00 time 20 Source region 16
Region 11 y= 46.71 time 20 Source region 16
Region 13 y= 21.38 time 20 Source region 16
Region 21 y= 112.80 time 20 Source region 16
Region 22 y= 83.19 time 20 Source region 4
Region 15 y= 4344.11 time 20 dif-5.0 source region 20
Region 5 y= 69.34 time 21 Source region 16
Region 18 y= 4.14 time 21 Source region 16
Region 1 y= 13617.80 time 21 dif 0.0 source region 28
Region 28 y= 20036.23 time 21 dif-3.0 source region 1
Region 37 y= 224.64 time 21 dif 1.0 source region 28
Region 12 y= 267.24 time 22 Source region 16
Region 17 y= 319.15 time 22 Source region 16
Region 27 y= 276.55 time 22 dif-4.0 source region 35
Region 30 y= 517.21 time 22 dif-5.0 source region 29
Region 32 y= 26392.25 time 22 dif-1.0 source region 1
Region 42 y= 2180.32 time 22 dif-1.0 source region 32
Region 7 y= 168.71 time 23 Source region 16
Region 4 y= 18283.56 time 23 dif-2.0 source region 16
Region 16 y= 23768.35 time 23 dif**** source region 4
Region 34 y= 6291.64 time 23 dif-4.0 source region 32
Region 38 y= 2083.90 time 23 dif-6.0 source region 32
Region 23 y= 15.94 time 24 dif-8.0 source region 17
Region 24 y= 488.09 time 24 dif-2.0 source region 32
Region 29 y= 4086.41 time 24 dif-2.0 source region 28
Region 33 y= 127.26 time 24 dif-7.0 source region 29
Region 35 y= 1098.14 time 25 dif-8.0 source region 29
Region 36 y= 366.36 time 25 dif-4.0 source region 29

```

Region	6	y=	27107.38	time	26	dif****	source	region	16
Region	41	y=	6072.28	time	26	dif 2.0	source	region	32
Region	2	y=	29332.84	time	27	dif****	source	region	20
Region	20	y=	48288.73	time	27	dif-6.0	source	region	2
Region	25	y=	2719.54	time	27	dif-3.0	source	region	20
Region	26	y=	6070.87	time	27	dif-6.0	source	region	20
Region	40	y=	13650.20	time	27	dif-6.0	source	region	20
Region	19	y=	2726.36	time	28	dif-7.0	source	region	20
Region	3	y=	1832.56	time	29	dif 0.0	source	region	6
Region	14	y=	8879.35	time	29	dif-9.0	source	region	8
Region	31	y=	1521.67	time	29	dif-9.0	source	region	6
Region	8	y=	7630.47	time	30	dif****	source	region	21
Region	10	y=	23381.08	time	30	dif****	source	region	5
Region	13	y=	4142.05	time	30	dif-9.0	source	region	6
Region	9	y=	3289.93	time	31	dif-9.0	source	region	21
Region	18	y=	977.49	time	31	dif****	source	region	17
Region	21	y=	19730.63	time	32	dif****	source	region	8
Region	22	y=	9046.38	time	32	dif-9.0	source	region	21
Region	5	y=	8211.25	time	33	dif****	source	region	11
Region	11	y=	8032.79	time	33	dif****	source	region	5
Region	12	y=	27765.00	time	33	dif****	source	region	17
Region	17	y=	45983.48	time	33	dif****	source	region	12
Region	7	y=	18714.68	time	36	dif-7.0	source	region	17
Fit= 284.0 years									

Appendix 5b :Output from space . 1st for countries of sub-Saharan Africa –

Start region 2 (D.R. Congo)

```

lambda(1)  0.30
lambda(2)  7.00
  0.95  0.95  0.95  0.95  0.95  0.95  0.95  0.95  0.95
  0.95  0.95  0.95  0.95  0.95  0.95  0.95  0.95  0.95
  0.95  0.95  0.95  0.95  0.95  0.95  0.95  0.95  0.95
  0.95  0.95  0.95  0.95  0.95  0.95  0.95  0.95  0.95
  0.95  0.95  0.95  0.95  0.95  0.95
selected start region  2
Region 20 y=      317.03 time      4 Source region  2
Region 15 y=      21.98 time      6 Source region 20
Region 19 y=      16.37 time      8 Source region 20
Region 24 y=       2.13 time      8 Source region 20
Region 26 y=      30.68 time      8 Source region 20
Region 40 y=     112.32 time      8 Source region 20
Region 25 y=      19.90 time      9 Source region 20
Region 38 y=       8.43 time      9 Source region 20
Region  4 y=     114.63 time     10 Source region 20
Region 16 y=     128.59 time     10 Source region 20
Region 32 y=     140.62 time     11 Source region 20
Region  6 y=     111.83 time     12 Source region 16
Region 10 y=      88.82 time     12 Source region 20
Region 39 y=      34.69 time     12 Source region 20
Region 42 y=      12.23 time     12 Source region 32
Region  3 y=      11.09 time     13 Source region 16
Region  5 y=      51.10 time     13 Source region 20
Region 11 y=      59.53 time     13 Source region 20
Region 31 y=       7.71 time     13 Source region 16
Region 41 y=      36.83 time     13 Source region 20
Region  1 y=     123.26 time     14 Source region 20
Region 14 y=      66.03 time     14 Source region  4
Region 28 y=     116.64 time     14 Source region 20
Region 34 y=      42.73 time     14 Source region 20
Region 15 y=     4378.71 time     14 dif 1.0 source region 20
Region  8 y=      61.50 time     15 Source region  4
Region  9 y=      16.57 time     15 Source region 16
Region 13 y=      30.79 time     15 Source region 16
Region 18 y=       3.39 time     15 Source region 16
Region 37 y=       1.10 time     15 Source region 28
Region  7 y=     129.00 time     16 Source region 10
Region 12 y=     204.83 time     16 Source region 16
Region 17 y=     292.12 time     16 Source region 16
Region 21 y=     197.29 time     16 Source region 16
Region 22 y=      93.77 time     16 Source region  4
Region 27 y=       1.42 time     16 Source region 20
Region 29 y=      30.25 time     17 Source region 28
Region 30 y=       3.39 time     17 Source region 29
Region 33 y=       0.73 time     17 Source region 29
Region 36 y=       1.07 time     17 Source region 29
Region  2 y=     28611.69 time     17 dif 0.0 source region 20
Region 20 y=    46837.81 time     17 dif 4.0 source region  2
Region 35 y=       6.79 time     18 Source region 29
Region 16 y=     24193.67 time     18 dif-5.0 source region  6
Region  4 y=     18685.10 time     19 dif 2.0 source region 16
Region 23 y=      16.36 time     19 dif-3.0 source region 17
Region 26 y=      6306.05 time     20 dif 1.0 source region 20
Region 40 y=    14447.42 time     20 dif 1.0 source region 20
Region  6 y=    27099.04 time     21 dif-7.0 source region 16
Region 19 y=     2824.39 time     21 dif 0.0 source region 20
Region 24 y=      502.83 time     21 dif 1.0 source region 40
Region 38 y=     2107.23 time     21 dif-4.0 source region 20
Region 25 y=     2869.84 time     22 dif 2.0 source region 40
Region 39 y=     5047.87 time     22 dif-4.0 source region 32
Region 10 y=    23293.71 time     23 dif-9.0 source region  5
Region 31 y=     1544.93 time     23 dif-3.0 source region  6
Region  3 y=     1832.65 time     24 dif 5.0 source region 16
Region 13 y=     4280.15 time     24 dif-3.0 source region  6
Region 32 y=    26215.69 time     24 dif-3.0 source region  1
Region  5 y=     8135.47 time     25 dif-8.0 source region 10

```

Region 14	y=	8858.48	time	25	dif-5.0	source region 8
Region 18	y=	1021.10	time	25	dif-7.0	source region 17
Region 42	y=	2223.30	time	25	dif-4.0	source region 32
Region 8	y=	7764.69	time	26	dif-6.0	source region 21
Region 9	y=	3348.29	time	26	dif-4.0	source region 21
Region 11	y=	8031.10	time	26	dif-7.0	source region 5
Region 37	y=	223.29	time	26	dif-4.0	source region 28
Region 17	y=	45890.78	time	27	dif****	source region 12
Region 21	y=	19621.10	time	27	dif-5.0	source region 8
Region 27	y=	276.29	time	27	dif-9.0	source region 35
Region 28	y=	20957.63	time	27	dif-9.0	source region 1
Region 34	y=	6304.83	time	27	dif-8.0	source region 28
Region 1	y=	15673.95	time	28	dif-7.0	source region 28
Region 12	y=	27953.91	time	28	dif-6.0	source region 17
Region 22	y=	9135.57	time	28	dif-5.0	source region 21
Region 30	y=	515.15	time	28	dif****	source region 29
Region 41	y=	5923.30	time	28	dif 0.0	source region 34
Region 7	y=	18362.89	time	29	dif 0.0	source region 17
Region 29	y=	4086.24	time	29	dif-7.0	source region 28
Region 33	y=	127.44	time	29	dif****	source region 29
Region 35	y=	1115.01	time	30	dif****	source region 29
Region 36	y=	372.57	time	31	dif****	source region 35

Fit= 216.0 years

**Appendix 5c :Output from space. 1st for countries of sub-Sahara Africa – start
region 1 and 2 (Ghana and DR Congo)**

```

lambda(1)  0.30
lambda(2)  7.00
  0.95  0.95  0.95  0.95  0.95  0.95  0.95  0.95  0.95
  0.95  0.95  0.95  0.95  0.95  0.95  0.95  0.95  0.95
  0.95  0.95  0.95  0.95  0.95  0.95  0.95  0.95  0.95
  0.95  0.95  0.95  0.95  0.95  0.95  0.95  0.95  0.95
  0.95  0.95  0.95  0.95  0.95  0.95
selected start region  1
selected start region  2
Region 20 y=      317.90 time      4 Source region  2
Region 15 y=      22.17 time      6 Source region 20
Region 39 y=      20.34 time      6 Source region  1
Region 40 y=      68.43 time      7 Source region 20
Region 19 y=      17.16 time      8 Source region 20
Region 24 y=       2.74 time      8 Source region 20
Region 26 y=      32.08 time      8 Source region 20
Region 28 y=     149.53 time      8 Source region  1
Region 32 y=     181.87 time      8 Source region  1
Region 42 y=      13.02 time      8 Source region  1
Region  4 y=      62.78 time      9 Source region 20
Region 25 y=      23.07 time      9 Source region 20
Region 34 y=      35.74 time      9 Source region  1
Region 37 y=       1.08 time      9 Source region  1
Region 38 y=      13.28 time      9 Source region 20
Region 16 y=     130.64 time     10 Source region 20
Region 29 y=      18.01 time     10 Source region  1
Region 41 y=      36.39 time     10 Source region  1
Region 27 y=       1.51 time     11 Source region  1
Region 30 y=       2.76 time     11 Source region 29
Region 33 y=       0.72 time     11 Source region 29
Region  6 y=     113.41 time     12 Source region 16
Region 10 y=      89.60 time     12 Source region 20
Region 35 y=       5.58 time     12 Source region 29
Region 36 y=       1.56 time     12 Source region 29
Region  3 y=      11.31 time     13 Source region 16
Region  5 y=      51.58 time     13 Source region 20
Region 11 y=      60.10 time     13 Source region 20
Region 31 y=       7.85 time     13 Source region 16
Region 13 y=      15.01 time     14 Source region 16
Region 14 y=      69.88 time     14 Source region  4
Region 15 y=     4376.36 time     14 dif 1.0 source region 20
Region  8 y=      64.80 time     15 Source region  4
Region  9 y=      17.29 time     15 Source region 16
Region 18 y=       3.44 time     15 Source region 16
Region 21 y=     100.23 time     15 Source region 16
Region 22 y=      50.35 time     15 Source region  4
Region  7 y=     130.35 time     16 Source region 10
Region 12 y=     207.81 time     16 Source region 16
Region 17 y=     295.98 time     16 Source region 16
Region  2 y=    28646.00 time     17 dif 0.0 source region 20
Region 20 y=    46812.63 time     17 dif 4.0 source region  2
Region 16 y=    24176.63 time     18 dif-5.0 source region  6
Region 39 y=    5010.07 time     18 dif 0.0 source region 32
Region  4 y=    18636.08 time     19 dif 2.0 source region 16
Region 23 y=      16.30 time     19 dif-3.0 source region 17
Region 24 y=     508.98 time     20 dif 2.0 source region 40
Region 26 y=     6335.27 time     20 dif 1.0 source region 20
Region 40 y=    14416.26 time     20 dif 1.0 source region 20
Region  1 y=    13873.72 time     21 dif 0.0 source region 32
Region  6 y=    27024.65 time     21 dif-7.0 source region 16
Region 19 y=    2834.23 time     21 dif 0.0 source region 20
Region 28 y=    20418.53 time     21 dif-3.0 source region  1
Region 32 y=    26794.40 time     21 dif 0.0 source region  1
Region 37 y=     223.25 time     21 dif 1.0 source region 28
Region 38 y=    2089.33 time     21 dif-4.0 source region 32
Region 42 y=    2178.71 time     21 dif 0.0 source region 32
Region 25 y=    2878.39 time     22 dif 2.0 source region 40
Region 27 y=     273.33 time     22 dif-4.0 source region 35

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Region	30	y=	519.96	time	22	dif-5.0	source	region	29
Region	34	y=	6227.78	time	22	dif-3.0	source	region	32
Region	10	y=	23276.68	time	23	dif-9.0	source	region	5
Region	31	y=	1545.82	time	23	dif-3.0	source	region	6
Region	3	y=	1831.75	time	24	dif 5.0	source	region	16
Region	13	y=	4282.27	time	24	dif-3.0	source	region	6
Region	29	y=	4074.27	time	24	dif-2.0	source	region	28
Region	33	y=	126.52	time	24	dif-7.0	source	region	29
Region	5	y=	8146.80	time	25	dif-8.0	source	region	10
Region	14	y=	8814.11	time	25	dif-5.0	source	region	8
Region	18	y=	1020.12	time	25	dif-7.0	source	region	17
Region	35	y=	1086.56	time	25	dif-8.0	source	region	29
Region	36	y=	370.18	time	25	dif-4.0	source	region	29
Region	41	y=	6132.75	time	25	dif 3.0	source	region	32
Region	8	y=	7753.94	time	26	dif-6.0	source	region	21
Region	9	y=	3345.80	time	26	dif-4.0	source	region	21
Region	11	y=	8035.14	time	26	dif-7.0	source	region	5
Region	17	y=	45923.38	time	27	dif****	source	region	12
Region	21	y=	19640.45	time	27	dif-5.0	source	region	8
Region	12	y=	27923.14	time	28	dif-6.0	source	region	17
Region	22	y=	9113.09	time	28	dif-5.0	source	region	21
Region	7	y=	18374.87	time	29	dif 0.0	source	region	17

Fit= 156.0 years

Glossary of symbols and abbreviations.

x = number of susceptibles

y = number of infectives

w = number of those HIV positive but not communicable

μ = Birth rate

θ = Death rate

β = probability that a partnership between an infective and a susceptible results in the transmission of HIV

D = period of communicability

n = population size at risk to HIV infection

α = removal rate

R_0 = reproduction rate

t = time

T = average time difference between predicted and observed peaks

Δt = a small increment of time

A = period from cessation of communicability of HIV to the onset of AIDS

R = partnership rate

f = time of first officially recorded AIDS cases

g = time of latest recorded AIDS cases

χ^2 = Chi-square statistics

$P(O)$ = observed time of peak HIV prevalence

$P(E)$ = predicted time of peak HIV prevalence

λ = decay parameter

W_i = attractiveness of region i as a travel destination

m_j = contact opportunities in region j

a_{ij} = Journey type; 1=international, 2=local

p_{ij} = the fraction of the contacts of persons in group i that are made with person in j

s_{ji} = the expected number of partners from region i acquired by an individual in region j

$PAIDS$ = predicted AIDS incidence

$OAIDS$ = observed AIDS incidence

ANC = Antenatal Clinic

Appendix 7: Logistic Regression output

Case Processing Summary

Unweighted Cases(a)		N	Percent
Selected Cases	Included in Analysis	452	46.5
	Missing Cases	521	53.5
	Total	973	100.0
Unselected Cases		0	.0
Total		973	100.0

a. If weight is in effect, see classification table for the total number of cases.

Dependent Variable Encoding

Original Value	Internal Value
never used a condom	0
used a condom	1

Categorical Variables Codings

		Frequency	Parameter coding			
			(1)	(2)	(3)	(4)
at what age	10-14 years	45	1.000	.000	.000	.000
did you first	15-19 years	196	.000	1.000	.000	.000
had sex	20-24 years	159	.000	.000	1.000	.000
	25-29 years	48	.000	.000	.000	1.000
	dont know	4	.000	.000	.000	.000
what is your	not at risk	97	1.000	.000	.000	.000
level of risk	risk is moderate	164	.000	1.000	.000	.000
	risk is high	99	.000	.000	1.000	.000
	risk is very high	92	.000	.000	.000	.000
educational	none	116	1.000	.000	.000	.000
level	primary/middle/JSS	167	.000	1.000	.000	.000
	secondary	124	.000	.000	1.000	.000
	tertiary	45	.000	.000	.000	.000
age	15-34	264	1.000	.000	.000	.000
	35-49	144	.000	1.000	.000	.000
	50-60+	44	.000	.000	.000	.000
gender	female	214	1.000	.000	.000	.000
	male	238	.000	.000	.000	.000
employment	unemployed	283	1.000	.000	.000	.000
status	employed	169	.000	.000	.000	.000
estimated	under 100,000-1000000	400	1.000	.000	.000	.000
monthly	cedis					
income	1,100,000-2,100,000 cedis	52	.000	.000	.000	.000
marital status	married	195	1.000	.000	.000	.000
	unmarried	257	.000	.000	.000	.000

Block 0: Beginning Block

Classification Table(a,b)

Observed			Predicted		
			Q33L		Percentage Correct
			never used a condom	used a condom	
Step 0	Q33L	never used a condom	0	202	.0
		used a condom	0	250	100.0
Overall Percentage					55.3

a Constant is included in the model.

b The cut value is .500

Variables in the Equation

	B	S.E.	Wald	df	Sig.	Exp(B)
Step 0 Constant	.213	.095	5.078	1	.024	1.238

Variables not in the Equation

			Score	df	Sig.
Step 0	Variables	Q1B	8.917	2	.012
		Q1B(1)	1.796	1	.180
		Q1B(2)	.228	1	.633
		Q2(1)	26.882	1	.000
		Q3A(1)	.168	1	.682
		Q5	11.578	3	.009
		Q5(1)	4.842	1	.028
		Q5(2)	.215	1	.643
		Q5(3)	.525	1	.469
		Q7(1)	.009	1	.926
		Q9B(1)	.441	1	.507
		Q13A	1.226	1	.268
		Q20	.747	1	.387
		Q22A	.010	1	.919
		Q29	.999	1	.318
		Q31	8.454	4	.076
		Q31(1)	4.739	1	.029
		Q31(2)	.423	1	.515
		Q31(3)	5.707	1	.017
		Q31(4)	.226	1	.634
		Q36	19.419	1	.000
		Q37	12.751	1	.000
		Q40	2.709	1	.100
		Q59B	2.701	3	.440
		Q59B(1)	1.695	1	.193
		Q59B(2)	2.059	1	.151
		Q59B(3)	.030	1	.863
Overall Statistics			82.543	23	.000

Block 1: Method = Enter

Omnibus Tests of Model Coefficients

		Chi-square	df	Sig.
Step 1	Step	91.057	23	.000
	Block	91.057	23	.000
	Model	91.057	23	.000

Model Summary

Step	-2 Log likelihood	Cox & Snell R Square	Nagelkerke R Square
1	530.441	.182	.244

Classification Table(a)

		Observed	Predicted		
			Q33L		Percentage Correct
			never used a condom	used a condom	
Step 1	Q33L	never used a condom	116	86	57.4
		used a condom	68	182	72.8
Overall Percentage					65.9

a The cut value is .500

Variables in the Equation

	B	S.E.	Wald	df	Sig.	Exp(B)
Step 1			10.021	2	.007	
1(a) Q1B			9.806	1	.002	3.746
Q1B(1)	1.321	.422	4.832	1	.028	2.452
Q1B(2)	.897	.408	18.591	1	.000	.343
Q2(1)	-1.071	.248	3.278	1	.070	.618
Q3A(1)	-.481	.266	4.839	3	.184	
Q5			4.322	1	.038	.399
Q5(1)	-.919	.442	2.094	1	.148	.527
Q5(2)	-.641	.443	3.242	1	.072	.444
Q5(3)	-.813	.451	.391	1	.532	1.152
Q7(1)	.141	.226	.001	1	.970	1.014
Q9B(1)	.014	.367	1.030	1	.310	.806
Q13A	-.216	.213	.146	1	.702	1.404
Q20	.339	.888	.219	1	.640	.804
Q22A	-.218	.466	.489	1	.484	1.225
Q29	.203	.290	4.241	4	.374	
Q31			.159	1	.690	.585
Q31(1)	-.536	1.344	.039	1	.843	.771
Q31(2)	-.260	1.307	.017	1	.897	1.185
Q31(3)	.170	1.314	.033	1	.856	.785
Q31(4)	-.242	1.339	19.318	1	.000	.304
Q36	-1.190	.271	11.425	1	.001	.352
Q37	-1.044	.309	.203	1	.652	.878
Q40	-.130	.287	3.048	3	.384	
Q59B			1.420	1	.233	.637
Q59B(1)	-.451	.378	.067	1	.796	1.086
Q59B(2)	.082	.319	.017	1	.896	1.046
Q59B(3)	.045	.343				
Constant	4.418	1.947	5.151	1	.023	82.948

Appendix 8: INTERVIEW Transcript

EL: The interviewer,

SMH: AIDS coordinator St Martins Hospital,

EL: What is your role as HIV/AIDS coordinator.

SMH: In 1989, the hospital realised that increasing number of return migrants who were mainly prostitutes from Abidjan in Cote D'Ivoire who visited the hospital were HIV positive. In fact most of them were so weak that they have to be carried from the State transport buses returning from Abidjan to their relatives. The hospital therefore decided to visit them at home since most of them cannot walk to the hospital.

EL: Why did you target return migrants from Cote D'Ivoire

SMH: In fact at that time I can say that almost all the AIDS patients were prostitutes from Abidjan who were sick and have returned to their families because there were so weak they could not work any longer in Abidjan and did not want to die in a foreign land but this time things are different. In fact I don't know what is happening this time because during sister Margaret's time all the HIV patients were *Ashawo* women (prostitutes) from Abidjan but now our young children are dying of AIDS

EL: What sort of help do you offer to HIV/AIDS positive patients?

SMH: At that time antiretroviral drugs were not available so we do voluntary counselling and testing to know their status and in most cases they were positive so all we can do is to treat them with vitamins and give them junction violet to treat their rashes and sores and those with diarrhoea we give them oral rehydration salt. At the moment we give them anti retroviral drugs.

EL: Do you give them the antiretroviral drugs free of charge.

SMH: No we sell it to them at a very low price. They pay 50,000 cedis every month for the drugs but the actual price would have been 7,000,000 cedis but even at that low price it is only those who are working or who have money that can afford to buy. activities because it was run by a native who understand the traditions and customs of the Krobo people and was able to win support of the community leaders as well as

EL: So what happen to those who are not able to pay for it.

SMH: Well they don't get treatment.

EL: What is your source of funding for the antiretroviral drugs?

SMH: We receive support from Canadian International Development Agency (CIDA)

EL: What are the main problems you encounter in your work.

SMH: Most of the HIV/AIDS patients believe that witches who envy them because of the properties they have acquired cause their sickness. Most of the People Living With HIV/AIDS (PLWHA) therefore seeks help from Faith healers, Spiritual churches and Herbalist instead of coming to the hospital. In fact some of the patients who were receiving antiretroviral treatment with us have run away to remote villages and have abandon treatment and it is difficult to trace them and give them treatment. The most pathetic thing about care and support is that most of the people who are put on the antiretroviral treatment are not able to meet their nutritional needs because they are unable to feed themselves and this brings complications because the drugs have got side effects.

EL: Don't they have family members to care for them.

SMH: The family members in most cases spend all their monies on fetish priest and spiritual healers as well as prayer camps so before they come to us for treatment they have already spent all their monies and in most cases have sold all their personal belongings and properties and are left with nothing so you see they come to us as the last resort.

EL: What about the extended family? Do they offer support?

SMH: The extended family members don't care for the HIV patients. They are only interested in taking care of the funeral expenses after the patient has died. In fact this time the funeral industry has become a business where the extended family raise funds during funerals through donations but when the person is sick they don't care. In this district funerals are organised on weekends and I know there are many AIDS deaths and funeral every week but the family will not disclose the cause of death.

EL: Why won't the family disclose the cause of death?

SMH: They think it is shameful to die of AIDS and it will bring a disgrace to the family. If the dead person has got wife or husband and children people will think that they are also infected and they will shun them.

EL: So what can your organisation do to help stop the spread of AIDS

SMH: We have formed HIV/AIDS patient association and they are all receiving antiretroviral treatment. We encourage these people to tell others about their status and to educate others on the risk of infection. We also educate queen mothers who intern educate members of their communities. We have also established AIDS committees in the district and they work with the Community Development Officer (CDO) in educating members of the public about HIV/AIDS. During the DIPO initiation ceremonies we provide new blades and make sure that every participant uses a new blade for the shaving of the hair and the scarification process to avoid infection.

We also advice HIV positive mother to bottle feed their babies to avoid the risk of mother to child transmission but unfortunately many mother cannot afford to bottle feed so we advice them to breast feed for the first six months.

EL: Interviewer

ATC: AIDS Counsellor Atua Government Hospital

EL: What is your main duty as HIV/AIDS counsellor

ATC: My outfit is in charge of Voluntary Counselling and Testing (VCT). We do the VCT as part of the routine clinical care for outpatients who visit the hospital. Also any body that want to know his or her HIV status is taken care of free of charge. WE started it in June 2003 when antiretroviral therapy became available to us. Any body that tested positive is put on antiretroviral treatment and we monitor their progress by visiting them at their homes.

EL: What is your source of funding for the antiretroviral drugs?

ATC: We receive funding from CIDA and this district has been chosen for a pilot project for antiretroviral treatment and our hospital is privileged to be chosen for this pilot project.

EL: What problem do you face as HIV/AIDS counsellor

ATC: The main problem we as counsellor face is that people are reluctant to know their status. The main argument is that whether one knows his or her status is not relevant because there is no cure so it is better not to know your status and die of AIDS than to know your status and go through humiliation from society and friends before dying. Women who want to know their status have to seek permission from their husbands and in most cases the man would not allow them to test and threaten with divorce. As part of the counselling process we need to visit patients at home and monitor their progress and effect of the antiretroviral treatment but often counsellors are not allowed to visit.

EL: Why do you think HIV/AIDS patients do not allow you into their homes when you are there to help?

ATC: You know that in this community the people know the counsellors and nurses who are working on HIV/AIDS so HIV/AIDS positive patients do not want to associate with us or we visiting them at home because people in the community will suspect that they are HIV positive and stigma will increase. In this community people live in compound houses with five or more households living in one house. This means that if we visit one member of a particular household, the other household members will see us and begin to speculate and rumours will circulate in the community. This is a very big problem for follow-up visits because the antiretroviral drugs have side effect and we need to visit patients at home to see their progress and problems. Last week I visited one patient at home to supply her with the antiretroviral drugs but she told me that she has not finished taking the drugs I gave her the previous month. When I asked her why she has not finished taking the drugs she replied that she has decided to halve the daily dosage because the drug is expensive,

So you see that if we don't do follow up the treatment will not be effective but we are in most cases not allowed to visit.

EL: Why can't you then supply the drug free of charge.

ATC: The problem is if you give it to them free of charge, they will not take the drugs and may not take the treatment serious but if they are made to pay, they will value the drugs and will not let it go to waste. Beside the government of Ghana is not ready to supply the drug free of charge

EL: So what is your outfit doing to stop the spread of HIV/AIDS

ATC: AT the moment, we have forty-two clients who are receiving antiretroviral therapy but two men have died recently because they were brought too late. These people who are receiving treatment testify at forums and this is reducing the stigma to some extent and more people are coming voluntarily to know their HIV status.

EL: Interviewer

FOS: Friends of the sick

EL: What is the role of your organisation in the fight against HIV/AIDS

FOS: We visit sick people in the community and provide pastoral care and support and in the process we identify HIV/AIDS patients. We then make regular visits to them to counsel them, provide food aid and direct them to the hospitals for consultation and treatment.

EL: What is your source of funding?

FOS: We receive support from the Catholic Relief Agency and other benevolent people within the community.

EL: What are the main problems you encounter in your work?

FOS: IN this community HIV/AIDS is associated with witchcraft and such a perception is deeply entrenched in the mind of the people that is difficult for People Living with HIV/AIDS to accept the fact that AIDS is cause by a virus. As a result many prayer camps and faith healers claim prayer and fasting can cure AIDS. PLWHA are camped and made to fast and pray till most of them become weak and die of starvation and malnutrition. There is one prayer camp where I visited but please I can't mention the name of the camp. In that camp there was one woman who tested positive with HIV and was receiving antiretroviral treatment but she absconded from home to the prayer camp. When I visited her at the camp, she told me that the prophet has seized the antiretroviral drugs she was using. Instead her body was smeared with red muddy clay mixed with olive oil. She told me that the prophet told her that one of her aunties was a witch and was responsible for her sickness. I tried to convince her to return home to continue with the antiretroviral treatment but she refused. So you see the influence of these pastors and prophets is such that PLWHA don't listen to advice and counselling from health professional.

EL: So what do you think can be done to stop the spread of AIDS?

FOS: We are dealing with people who are already infected with HIV so we try to educate them to seek help from the hospitals and encourage their immediate families to give them support and help. We try to reduce stigma about HIV/AIDS and encourage PLWHA to open up and warn others of the dangers of contracting HIV/AIDS

EL: Interviewer

DFC: District Focal Person

EL: What is your role in the fight against HIV/AIDS in the district.

DFP: MY main duty is to coordinate the activities of Non Governmental Organisations (NGO,s) ,Community Based Organisations(CBO,s) and Faith Based Organisations(, FBO,s) dealing with HIV/AIDS issues and the hospitals and to provide statistics about the epidemic in the district.

EL: What is the main problem you are facing as a district focal person?

DFP: There is no coordination in the activities of the NGO,s CBO,s , FBO,s and the hospitals. They are just duplicating what other agencies are doing. Secondly these groups are supposed to report to my office but they bypass me and send their proposals and reports to the Ghana AIDS Commission in Accra. Also most of the NGO,s enters the district without my knowledge. My office therefore is not able to provide reliable and up to date statistics about the epidemic in the district. The three hospitals in the district refuse to feed me information about the prevalence level on the grounds of confidentiality and data protection so what do they expect me to do as the district focal person

EL: So what can be done to help stop the spread of HIV/AIDS

DFP: The solution lies with reliable statistics about the prevalence of the epidemic and how it is spreading. I think all the institutions involved with HIV/AIDS activities should coordinate and share information about the prevalence of the disease. The National AIDS Control Programme and the Ghana AIDS Commission should work through the district focal person instead of dealing directly with the NGO,s, FBO, CBO,s and the hospitals.

EL: Interviewer

NGO: CYINDED Director

EL: What is your role in the fight against HIV/ADS?

NGO: We target the youth who are sexually active and educate them through peer education programmes on mode of transmission of HIV virus, prevention methods. We visit hair dressing saloons and barbering shops to educate them on sterilisation of instruments. We have one to one discussion with youth on STDs and direct them to

hospital for treatment. We also educate mother on the risk of mother to child transmission and offer counselling.

EL: What are the problems you face?

NGO: Our major problem is funding. You see this computer and office equipments, they are very expensive so we are not able to buy them. If you can help us with some used computer from England we will be very happy and it will help the work. You see we are a locally based NGO so we don't receive money from abroad as other international NGO,s do. I have to pay all my staff from my own resources so you see it is difficult to reach the whole community.

EL: Do you receive any support from the government of Ghana

NGO: Not at all. The government does not care about how we operate but when it comes to information in the district they come to consult us.

EL: In your opinion what do you think can be done to stop the spread of HIV/AIDS in the district

NGO: In this district there are no jobs so the government should create employment avenues because there are many school leavers who are unemployed. These people especially the young girls will engage in risky sexual behaviours as a means to survive even though they know the risk of HIV infection in the district is very high. Another problem in this district is the AIDS orphans. At the moment it is the queen mothers who are caring for most of them so there is the need for the government to support them.

Appendix 9: Questionnaire Population based survey of the Krobo district

KNOWLEDGE ATTITUDE AND PERCEPTION ABOUT HIV/AIDS IN KROBO DISTRICT PHD – FIELD WORK

1. Age in years { }
2. Sex 1. Female 2. Male { }
3.
 - a Marital status: (Married 2.Single 3. Divorced 4.Widowed { }
 - b How long have you been married to this person? { }
 - c How old were you when you first got married? { }
 - d Apart from this spouse, how many have you been married /living with you your whole life? { }
 - e Is your husband having other wives? Yes/No { }
- 4 Educational level of respondent { }
 - 1 None
 - 2 Primary/Middle/JSS
 - 3 Secondary/SSS
 - 4 Tertiary
- 5 What qualification did you attain if any? { }
- 6 Employment status { }
 - 1 Unemployed
 - 2 employed
- 7 Employment status of spouse (code as 7) { }
- 8 What is your estimated monthly income? { }
- 9a Number of live births { }
- 9b Number of children at present (alive) { }
- 10 What is your religion? { }
 - 1.Christian
 - 2 Moslem
 - 3 Traditional Religion
 - 4.Other

MIGRATION HISTORY

- 10(a) Just before you moved here, did you live in another town/village/country? { }
 1. Yes 2. No
- b) If so which town/village/country?

11(a) Have you ever travelled outside this country? { }

1. Yes 2. No

11(b) If yes, which country did you travel to?.....

12 Have you during the past years been on regular trips where { }
you have to stay away from home for several days or more?

1. Never 2. Sometimes 3. Often 4. Very Often

13(a) Have you ever travelled to Cote d'voire { }

1. Yes 2. No

14 Do you have a relative or a friend living in Cote d'voire?

1. Yes 2. No

15 In your opinion, do you think return migrants from Cote d'voire { }
contribute to the spread of HIV/AIDS in Ghana?

1. Yes 2. No

16 Do you know of a friend or relative who has return from { }
Cote d'voire to die? 1. Yes 2. No

17 What was the cause of death?.....

18 Who paid for their medical expenses before they died?.....

KNOWLEDGE ATTITUDE AND PERCEPTION ABOUT HIV/AIDS

19 Have you ever heard of HIV/AIDS 1. Yes 2. No { }

20 What is the source of AIDS knowledge?.....

20(a) In your opinion do you think AIDS is real? 1. Yes 2. No { }

(B) Explain.....

21 What do you think is the main cause of AIDS?.....

22 In your opinion, list the main mode of spread of HIV/AIDS.....

23(a) Do you think there is a cure for AIDS? 1. Yes 2. No { }

(b) Explain.....

24(a) Do you think witchcraft/ghost a cause of HIV/AIDS? { }

1. Yes 2. No

(b) Explain.....

25 What do you think should be done to stop the spread of AIDS?.....

26 Do you know of someone with AIDS or who has died of AIDS? { }

1. Yes 2. No

- 27 Do you think you are at risk of getting AIDS 1.Yes 2. No { }
- 28 Have you ever had sexual intercourse 1. Yes 2. No { }
- 29 At what age did you first have sex?.....
- 30 Have you had sex the last 12 months: 1.Yes 2. No { }
- 31 Have you ever used a condom? 1.Yes 2. No { }
- 32 Did you use a condom lastime you had sex? 1.Yes 2. No { }
- 33 Is it easy to get a condom when needed? 1.Yes 2. No { }
- 34 Did you have a regular sex partner during the last 12 months? { }
1.Yes 2. No
- 35 Did you have sex with anyone else apart from your regular sex partner last year? { }
1.Yes 2. No
- 36 Did you use a condom when you last had sex with a casual partner? { }
1.Yes 2. No
- 37 With how many different people have you had sex in the last 12 months (including spouse)? { }
- 38 Have you ever contracted any STD? 1.Yes 2. No
- 39 Did you tell your partner? 1.Yes 2. No { }
- 40 What did you do to cure it the last time? { }
1.Nothing
2.Used own medicine
3.Bought medicine at market
4.Got adduce from traditional healer
5.Medicine from pharmacy
6.Medicine from hospital/health centre/clinic

BEHAVIOUR CHANGE

- 41 Do you agree or disagree with the following statement:
1 Agree 2. Disagree

- 1.Condoms are safe preventing HIV/AIDS { }
- 2 Most women don't like men to use condom { }
- 3 Most men don't like using condom { }
- 4 Condoms are embarrassing to obtain { }

- 5 Condom is against my religion { }
- 6 I have less sexual partners at present compared to some years ago. { }
- 7 My friends have not changed their sexual behaviour despite the AIDS risk { }
- 7 Some years ago I did not use condoms { }
- 8 I need to have several sex partners { }
- 9 Most of my friends never use condoms { }
- 10 I always use a condom nowadays. { }

- 42 Know of a friend or relative who has died of AIDS? { }
1. Yes 2. No

- 42 How many dependents (children) were left behind?.....
- 43 Who care for those orphans?.....
- 44 In what ways did the death affected other family members?

- 45 To what extend does the "DIPO" custom influence the spread of HIV/AIDS
-

RISK ASSESSMENT

- 46 In your situation, do you think that you are at risk of getting HIV/AIDS?
Would you say that: { }
1. You are not at risk
2. The risk is moderate
3. The risk is high
4 The risk is very high
- 47 How worried are you about actually being infected by HIV/AIDS? { }
- 1 always worried
2 sometimes worried
3 never worried

