PREVALENCE, SPATIAL PATTERNS AND FACTORS ASSOCIATED WITH HIV INFECTION IN ZIMBABWE, 2011.

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DECLARATION

I, Atilola Glory declare that this report is my own, unaided work. It is being submitted for the degree of Master of Science in Epidemiology (in the field of Infectious diseases) in the University of the Witwatersrand, Johannesburg. It has not been submitted before for any degree or examination at this University or any other University.

Signature

……..

Full Name

Date

DEDICATION

To the young and old (known and unknown) who lost their lives to the AIDS virus and to victims who are currently fighting to stay alive from the grip of the most deadly viral plague in modern history in Zimbabwe and the rest of sub-Saharan Africa.

ABSTRACT

Introduction

Like in other Sub-Sahara African countries, HIV has had significant devastating effects in Zimbabwe. Understanding the underlying and proximate determinants of infections in the population is important for knowing who needs intervention, where interventions are needed and for designing specific and relevant interventions.

No studies appear to have assessed distributional patterns and risk factors of HIV prevalence simultaneously across the ten provinces in Zimbabwe using a combination of the proximate-determinant framework approach and modern geostatistical techniques to identify spatial HIV hotspots. The overall aim of this study was to investigate the prevalence, spatial patterns and factors associated with HIV infection in Zimbabwe using the proximate-determinant framework approach and modern geostatistical techniques.

Methods

This study used the Zimbabwe demographic and health survey of 2010/2011 which included a representative sample of 9171 women and 7104 men- aged 15 to 49. Following the proximate determinants framework, multilevel models were fitted separately for men and women. Global and local spatial autocorrelations were assessed. Spatial regression models were also fitted to adjust for spatial random effects and non-random effects. Significant difference between hotspots and cold spots was examined at community level.

Results

The overall prevalence estimate was 15.4% (95%CI: 14.8% - 16.0%) [17.7% among women and 12.2% among men]. While prevalence was highest in women in the middle age categories

(29.3%), a corresponding dose response relationship was observed among men. Highest prevalence was obtained in urban dwellers, widowed men (60%) and women (56%), and Matabeleland South. A dose response association was found between HIV prevalence and duration of cohabitation, total lifetime partners in both gender populations, and age of most recent partner in men.

Evidence of global and local spatial autocorrelation was found. Spatial scan techniques identified three hotspots including Matabeleland South province. Prominent significant underlying risk factors of HIV infection in both gender populations were age group [men: AOR_{40-44}: 5.19; (95%CI:1.92 – 14.02); AOR_{45-49} : 4.76 (95%CI: 1.70 – 13.30)] [women: AOR_{30-34}: 2.17; (95%CI:1.32 – 3.54); AOR_{35-39}: 1.91; (95%CI:1.14 – 3.19)] and widowed marital status[men: AOR: 7.37; (95%CI: 3.10 – 17.52)][women: AOR: 4.13; (95%CI: 2.49 – 6.85)], likewise Matabeleland South region in men [AOR: 2.32; (95%CI: 1.49-3.62)]. On the other hand, total lifetime partners [men: AOR_{5-9}: 3.15; (95%CI: 2.08 – 4.77)][women: AOR_{5-9}: 3.74; (95%CI: 2.52 – 5.53)], and symptoms of sexually transmitted infections emerged as proximate predictors of the epidemic.

No evidence of significant heterogeneity in geographical distribution of HIV prevalence was found after adjusting for significant underlying and proximate risk factors. However, persons living within hotspots are on the average 30% more likely to be within the richest wealth quintile (OR: 1.30; p: <0.001), more likely to have higher total lifetime partners and symptoms of sexually transmitted infections.

Conclusions

A combined application of traditional statistical procedures and modern geostatistical techniques to identify significant predictors of HIV infection and prevalence hotspots provide a more robust approach to investigate HIV spread in a generalized epidemic setting. The results showed excess risk of infections in certain demographic sub-groups, reinforcing the need for programmatic interventions to be directed at these locations and populations in order to maximize impact. The interventions should address the proximate determinants of infection in the population. Further analysis should examine the independent effect of condom use on HIV outcome in the context of marital status and sexual partnership. Proximate determinants of HIV infection should be explored at community level.

ACKNOWLEDGEMENTS

Above all, I would like to express my gratitude to God Almighty, the one who gave the courage and resilience to carry this project on to completion. This project would not be complete today without the unparalleled supports of the following persons all the way;

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Dad and Mum: May your all round support and investment be greatly rewarded. Thank you for paying the prize required for me to be what and who I am today.

Clarence Yah: Your fortitude kept me going.

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DEFINITION OF TERMS

Coldspots-: geographical locations (communities) with average and low numbers of HIV cases.

Generalized epidemic-: The HIV prevalence rate is >1% in the general population.

Hotspots-: geographical locations (community) with excess numbers of HIV cases.

Kriging-: A geostatistical estimator that infers the value of a random field at an unobserved location from samples (observed locations).

Nugget-: This represents unresolved, sub-grid scale variation or measurement error and is seen on the variogram as the intercept of the variogram.

Proximate determinant-: A set of behavioral and biological variables that have a direct effect on biological outcome of HIV infection.

Range-: The scalar that controls the degree of correlation between data points, usually represented as a distance.

Sill-: The value of the semivariance as the lag(h) goes to infinity, it is equal to the total variance of the data set.

Spatial dependence-: is a property of a spatial stochastic process in which the outcomes at different locations may be dependent.

Semi-variogram-: A Semivariogram is one of the significant functions to indicate spatial correlation in observations measured at sample locations. It is commonly represented as a graph that shows the variance in measure with distance between all pairs of sampled locations.

Underlying determinant-: A set of social, demographic, economic, and environmental variables that operate through proximate determinants to influence outcome of HIV infection.

ABBREVIATION LIST

AIDS - Acquired Immune-Deficiency Syndrome

AIS - AIDS Indicator Survey

BCI - Bayesian Credibility Interval

DIC - Deviance Information Criterion

EA - Enumeration Area

GPS - Global Positioning System

HIV - Human Immunodeficiency Virus

LISA - Local Indicators of Spatial Association

MTCT - Mother To Child Transmission

PDA - Personal Digital Assistance

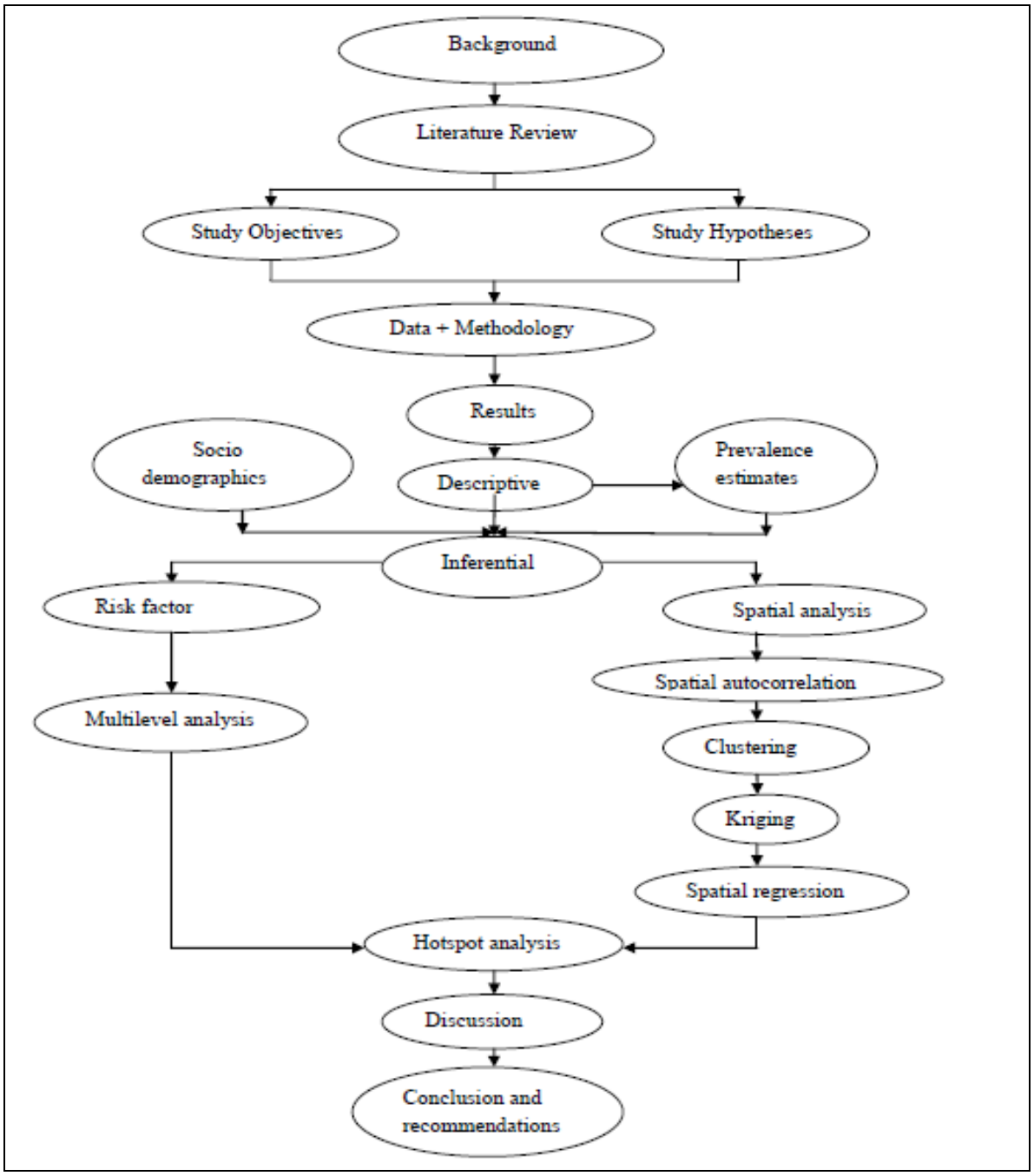
STI - Sexually Transmitted Infection

SSA - sub-Saharan Africa

ZDHS - Zimbabwe Demographic Health Survey

ZIMSTAT – Zimbabwe National Statistics Agency

STUDY ROADMAP



CHAPTER 1 : INTRODUCTION

1.1 Background

The overall impact of HIV over the past three decades has been tragic social and economic consequences in different parts of the world, especially in developing countries. Thus far, the death toll recorded is in excess of 30 million. [1]

The first case of AIDS to be reported in Zimbabwe was in 1985. By the end of the 1980s, 10% of the adult population was infected with the virus. [2, 3] There was a substantial rise in this figure in the first half of the 1990s, peaking at 36% between 1995 and 1997. In 2003, an estimated 170 000 Zimbabweans died of HIV and life expectancy at birth in Zimbabwe fell to 34 years, in large part due to HIV. Nine in ten deaths among Zimbabwean adults are due to HIV, with heterosexual sex accounting for most of the spread [4]. The HIV epidemic in Zimbabwe has reduced overall life expectancy; exacerbated intense poverty in vulnerable households and communities, undermined effectiveness and efficiency of national systems, and weakened institutional structures [5].

Since the late 1990s, the prevalence has been consistently declining. According to the National HIV prevalence estimate 2010, the prevalence of HIV among Zimbabwean adults 15 years and above was 14.3%. There were an estimated 1 187 822 adults and children living with HIV/AIDS in 2009 with 33% of the adults and children in urgent need of antiretroviral therapy by the end of the year .[4, 6, 7] With a national adult prevalence of 15.3% at the end of 2007, Zimbabwe emerged as one of the ten highest-HIV prevalence countries in sub-Saharan Africa. The National HIV prevalence is estimated to have declined by 3 percentage points over a five-year period between the 2005-06 ZHDHS [18.1% (95%CI: 17.4 – 18.8)] and the 2010-11 ZDHS [15% (95%CI:14.8 – 16.0)].[8]

The decline in HIV epidemic in Zimbabwe was achieved largely believed to have been as a result of prevention programmes such as behavior change and prevention of mother to child transmission, as well as the impact of mortality.[9] By the end of 2011, a 60% coverage of HIV treatment was also achieved according to UNAIDS regional fact sheet, 2012.[10] Despite government efforts to scale up the multi-sectoral response to HIV, the number of children orphaned and made vulnerable remains high. The approximate number of HIV orphans in Zimbabwe in 2007 was 1 043 715, while estimates for 2008 and 2009 were 1 025 472 and 989 009 respectively.[6]

Several contextual and behavioral factors have been shown to drive the HIV generalized epidemic in Zimbabwe and sub-Saharan Africa as a whole. Among these are age, marital status, residence, gender and income inequalities, indication of poverty, high levels of migration, widowhood, early sexual debut and total number of lifetime partners.[11] Similarly, pathway analytical evidence exists to show that contextual factors operate through the proximate (behavioural) determinants to influence the risk of HIV infection. This was established using the proximate determinant framework approach developed by Boerma and Weir.[12] and later modified by Barnighausen and Tanser.[13]

The overall aim of this study therefore is to investigate the prevalence, spatial patterns and factors associated with HIV infection across the ten provinces of Zimbabwe in 2011 using the proximate-determinant framework approach and geostatistical methods. Various geostatistical techniques were utilized to assess the degree of spatial dependence of HIV prevalence, identify hotspots (areas with excess risk of HIV infection) and assess the existence differential spatial distribution of underlying determinants which could explain the identified HIV hotspots.

1.2 Literature review

Over the past decade, the burden of HIV epidemic continues to vary substantially worldwide, across sub-Saharan African countries (SSA) and across provincial boundaries in Zimbabwe.

1.2.1 Global prevalence of HIV

At the end of 2011, the global HIV prevalence estimate stood at 0.8% (about 34 million people) in adults aged 15 – 49 years.[14, 15] A 25% decline in new infections was recorded at the end of 2011, and a 32% reduction in death from AIDS-related causes between 2005 and 2011. A larger percentage of those living with HIV (97%) are resident in low and middle-income countries, particularly in SSA.[15, 10] Global leading predictors of the epidemic remain men having sex with men (MSM), heterosexual transmission and injecting drug use.[11]

1.2.2 HIV prevalence in sub-Saharan Africa

In 2011, the HIV adult prevalence rate was estimated to be 4.9% in SSA with an average of 1.8 million new infection in the same year.[10] According to the 2012 US global health policy fact sheet, two-thirds of people living with HIV are resident in sub-Saharan Africa, though it constitutes only about 12% of the world's population.[10] A key attribute of most countries in this region is a generalized pattern of HIV epidemic (>1% HIV prevalence rate). In nine countries in this region, more than 10% of adults were reported be HIV-positive.[10]

1.2.3 HIV prevalence in Zimbabwe

Although Zimbabwe has recorded a significant reduction in both HIV incidence (largely due to behavioral interventions and deaths from AIDS) over the past decade, country's HIV

burden still reflects that of a generalized state of the epidemic. [16] In Zimbabwe, the national prevalence in 2011 was about 15% in adults age 15 – 49 compared to 18% in 2006. In the same year, about 1million adults age 15 and above, and two hundred thousand children were living with HIV.[7]

1.2.4 Socio-demographic risk factors for HIV

A fundamental prerequisite for the development and successful implementation of HIV prevention programmes is a solid and evidence-based understanding of who is at risk of disease, where and why.

In Zimbabwe, a number of social, economic, demographic and cultural factors have been linked to the risk of HIV infection. These include age group, gender, divorce or widowhood, urban residence, socio-economic conditions and cultural factors.[17, 18] Poverty, being the most prominent, is a major endemic scourge in most countries in SSA including Zimbabwe.[19-21]

In Africa, poverty is usually characterized by deprivation, vulnerability (low capacity to cope with risks), and powerlessness which all combined, significantly impair people's sense of wellbeing.[22] It usually provides the substrate environment upon which other known factors (such as sexual activity, teenage pregnancy, food insecurity, low literacy level) and unknown determinants thrive. The strong link between poverty and HIV in Zimbabwe was further established in a population-based cohort study in Manicaland.[19] A decrease of 25% in HIV prevalence among men and 21% among women was reported to be largely in the top one third of the wealth index distribution.[19]

There is evidence for the effects of socio-economic factors such as occupation and employment status on the risk of infection rates in Zimbabwe, as well as in neighboring countries like Lesotho, Malawi and Swaziland.[23] This study also found an increased risk of

HIV infection in urban dwellers compared to their rural counterparts. Similarly, a wide disparity in the risk of HIV infection is known to exist between men and women, especially in rural Zimbabwe partly due to biological and cultural frailty, reduced ability to refuse sex or use condom during sexual intercourse.[24, 25]

Moreover, the marital status of individuals partly explains their risk of HIV infection in Zimbabwe and other parts of SSA. Studies have shown that unmarried men and women (single, widowed, divorced and separated) usually have a higher risk of HIV infection compared to the married counterparts due to their high sexual activity, inconsistency with the use of condom, and multiple or concurrent sexual partnership.[26, 27] While marriage can be protective against HIV infection, married men are more likely to engage in extramarital sexual unions [26], thereby acting as a key source of transmissibility contact between the general population and the married subpopulation.

Furthermore, widowhood to a considerable extent plays an important role in the transmission of HIV in Zimbabwe in large part due to partner's death from AIDS and subsequent high predilection to change partner. In a study carried out in 2007, the prevalence of HIV was found to be exceptionally high among both widows (61%) and widowers (male widows) (54%). Widows were found to be more likely to have high rates of partner change and engage in a pattern of transactional sex than married women.[28]

The role of migration in HIV spread has been assessed in the context of Zimbabwe generalized epidemic and southern Africa at large. While this population is highly mobile and heterogenous, they play a significant role in contributing to the rapid spread of the infection across local and national boundaries with an increased risk of vulnerability.[29, 30] Economic migrants usually consist of the younger men and women population seeking for better job opportunities in urban locations. More so, miners are predominantly men who are less likely

to abstain or use condom.[29] Consequently, spouses of miners are more likely to engage in extramarital sex as a means of sexual relief while the man is away in the mine for a long period of time.[30]

However, the role of migration in rural Zimbabwe at a mature stage of the HIV epidemic seems to lessen over time. Evidence was found in a prospective cohort study that compared HIV incidence and behavioural risks between out-migrants and residents in four rural communities of Manicaland.[31] While migrants were more likely to be younger and more educated than residents, no difference was found in the risk of HIV acquisition and common behavioural risk factors.[31] A similar study by Coffee and others also showed that rural-urban migration seems to be a less important driver of the Zimbabwean HIV epidemic.[32]

1.2.5 Behavioural risk factors

Studies have also shown that the substantial age difference between female and male sexual partners in Zimbabwe is a major determinant of the rapid rise in HIV prevalence in young women than in men.[33] Many young women give in to contrivance of other means to survive at all cost, including exchanging money for sex.[34] This therefore accounts in part for the younger women-older men pattern of HIV prevalence observed in different parts of the sub-Saharan Africa.

Poverty, partly fuelled by inflation, has pushed great number of vulnerable Zimbabwean women living in the country's towns and cities to enter commercial sex work or 'transactional' sexual relationships. The exchange of gifts or money for sex is now a common phenomenon among women aged between 15 and 49 years.[35] Factors such as youthful and sexually active age group, household economic conditions, peer pressure, in-school status, luxury items, rural or urban living conditions (among others), have all been reported to be responsible for these two forms of sexual relationships in sub-Saharan Africa.[19, 35]

On the other hand, Lewis and others, using the proximate-determinant frame-work approach, found that total number of lifetime partners accounts for a substantial explanation in Zimbabwean men than women.[36]

1.2.6 Limitations of previous HIV risk factor studies

A common methodological limitation of many existing studies [23-28, 33-36] is the lack of quantitative assessment of risk factors for HIV spread in the Zimbabwean context, taking variation and clustering at household and community levels into account. For instance, Lewis and others only restricted their evaluation of the proximate determinants framework to rural Zimbabwe using single-level logistic regression models.[36] Conversely, Ilene et al assessed the correlation between the prevalence of HIV at the community level and the prevalence of HIV risk-taking behavior without taking individual level effects into account.[34]

1.2.7 HIV proximate determinant framework

The proximate determinant framework was developed by Boerma and Weir as a conceptual framework for the study of the distribution and determinants of HIV infection in populations.[12] This framework categorises potential HIV risk factors hierarchically into underlying, proximate, and biological determinants of the distribution of HIV infection in the population. Underlying determinants describe the contextual, social and environmental systems that influence proximate determinants.[12] Underlying factors (further divided into demographic, socio-economic, and socio-cultural and intervention determinants) influence the biological determinant (which affects the risk for infection) through the proximate factors.

Proximate determinants are by definition both behavioural and biological, and act as a "hinge between the social and biological systems". Proximate determinants in turn control the

biological determinants, namely the rate of exposure to the virus, the probability of transmission on exposure and the duration of infectivity (fig 1.1).

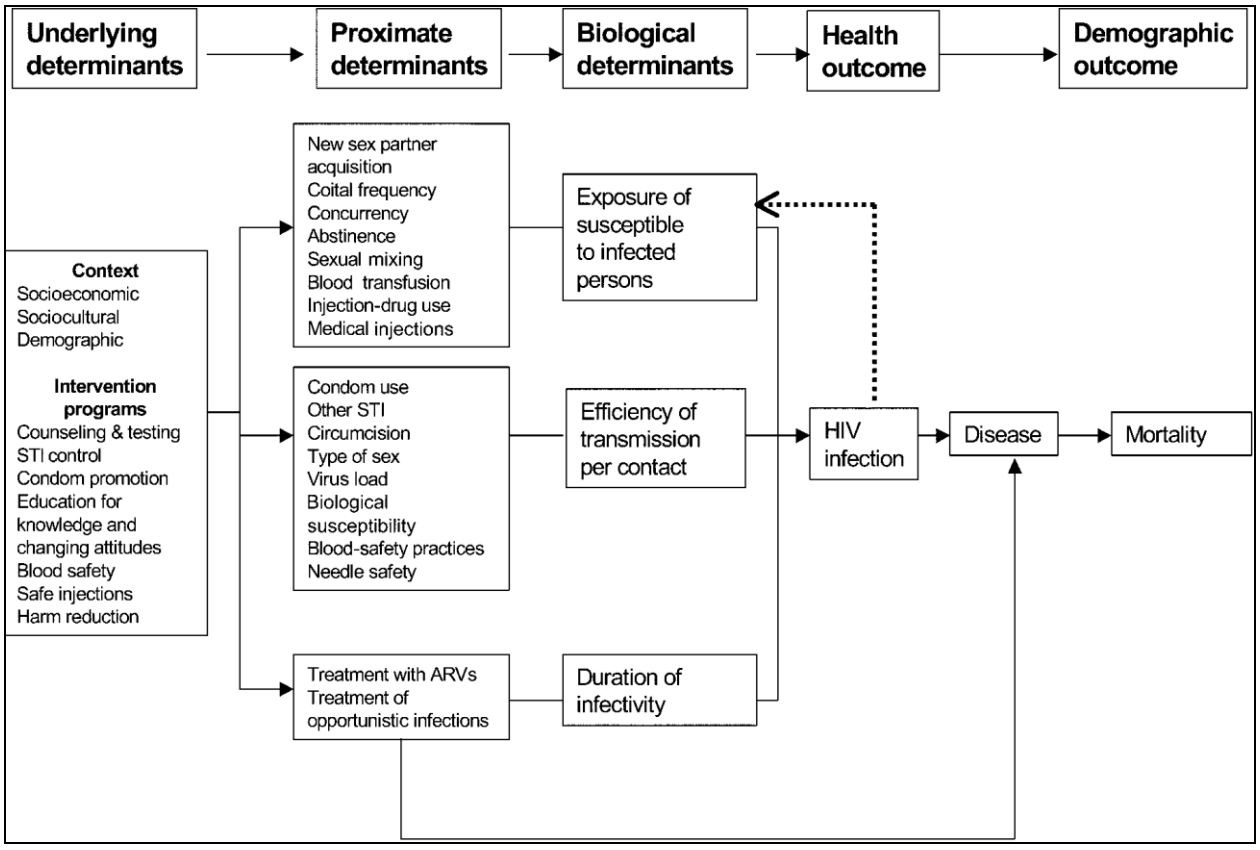


Figure 1.1 The Proximate determinants framework for HIV infection developed by Boerma and Weir

Consequently, the sequence of events proceeds from exposure of individuals to transmission of HIV infection and death due to AIDS. Any intervention strategy therefore acts in a way to reduce the rate of transmission of HIV or rate of effective contact between susceptible and infectious individuals. The usefulness of this framework in evaluating underlying and proximate determinants of HIV infection in Zimbabwe has been demonstrated.[36]

A further modification to the proximate-determinant framework was developed by Barnighausen and Tanser in 2009.[13] They proposed a framework that takes into account the role of community-level factors in affecting both HIV acquisition and the likelihood that a

sex partner chosen from a community will be infected with HIV and transmit the infection.[13] They proposed three biological variables that determine the individual's risk of HIV infection in a community; duration of time an individual spends in a community, rate of sexual contact with infected individuals overtime and the probability of acquiring HIV during one sexual contact with an infected individual.[13]

1.2.8 Role of spatial techniques in HIV epidemiology

The role of spatial analysis to better elucidate the epidemiology of infectious diseases and for control has been examined. [37-43] Spatial analytical techniques provide the most meaningful ways of understanding the geographic distribution, patterns and trends of both infectious and non-infectious diseases, while accounting for the spatial similarity of the underlying population. [38-43] These methods have been shown to be highly reliable and efficient in assessing the geographic impact of diseases such as cancer, tuberculosis, outbreak investigations and most recently HIV infection. [(44-46)]

More so, most risk factors that drive the HIV epidemic within each local context have been shown to cluster in space. As a result, closer locations are more likely to have similar distribution of HIV infection relative to locations farther apart according to Tobler's first law of geography.[36] Therefore, also critical is the need for new studies to examine the spatial distribution of HIV prevalence across geographical boundaries and how this varies from one community to the other in the general population using modern geostatistical techniques.

A review of the application of geographic information systems (GIS) in public health management and research in Africa found only one study applying a GIS analysis to factors related to HIV prevalence.[38] The study estimated local-level HIV prevalence using data obtained from antenatal care providers, and found a correlation between HIV prevalence and proximity of local households to a primary or secondary road. Few studies in Africa have

shown how the use of spatial analytical methods can enhance understanding of contextual determinants of HIV infection and geographic patterns of prevalence. [39-41]

In a recent study that utilized the 2007 Democratic Republic of Congo DHS data, HIV prevalence was shown to be related to individual demographic characteristics and sexual behaviors and community-level factors.[42] In this study clustering techniques were used to identify hotspots of HIV infection and predictors of a higher prevalence within these hotspots. This study equally found that the prevalence of HIV infection within 25km of an individual's community is an important positive indicator of HIV infection. However the study did not account for spatial random effect in the regression model of risk factor effect estimates which eliminates variation in the distribution of HIV infection due to spatial locations of cases.

A more recent study that adopted the GIS techniques and spatial analysis clearly reveals that the HIV/AIDS epidemic is complex and that it is inter-connected with other geographic, historical, economic and cultural phenomena which help explain its spatial spread and variation.[43] Stillwaggon demonstrates that spatial variation of social, economic, and other health determinants can explain HIV distribution and prevalence in sub-Saharan Africa. She cited factors including male circumcision, prevalence of other STDs, income inequality, and trade flows. Such studies increasingly show that risk-group status is not a reliable predictor of HIV vulnerability and that no single factor adequately explains its social, economic, and political complexity.[47]

1.2.9 HIV prevention services in Zimbabwe

In addition, efforts to scale up HIV service deliveries and local interventions have a remarkable effect in the downward reversal of the national epidemic trend. Studies have also demonstrated the impact of a well-implemented prevention of mother-to-child transmission

advocacy and mobilization campaign in improving knowledge and practices among end-users in Zimbabwe.[48]

In a study, it was reported that awareness of the service increased from 48% in 2002 to 82.8% in 2004. In 2001 only 4% of women and children in need of services for prevention of mother to child transmission (MTCT) of HIV were receiving them. Lessons learnt from a pilot study reveals that minimum staffing, an enhanced training programme, and involvement of district health authorities are needed for the implementation and successful integration of HIV/AIDS services. Voluntary counseling and testing services also turned out to be important entry points for HIV prevention and care and for referral to community networks and medical care services.[49]

1.3 Statement of the problem

A review of the literature shows no studies have investigated the risk factor for HIV infection using the HIV proximate determinants framework at the individual, household and community levels in Zimbabwe as proposed by Barnighausen and Tanser .[13] Furthermore, no evidence of studies that have conducted a comparative analysis of HIV infection distribution using a combination of the HIV proximate determinant framework and modern geospatial techniques in Zimbabwe, that would allow the identification of "hotspots"[1] was found. Hence there are paucity of data on the possible links between HIV status and its distribution across intra-geographical boundaries in Zimbabwe.

1 Geographical locations (community) with excess numbers of prevalent HIV cases.

1.4 Justification for the study

There is therefore a need for a multi-faceted approach that incorporates HIV risk analyses and geostatistical techniques. The use of the proximate determinant framework helps to understand the dynamics of interactions between HIV risks factors in Zimbabwe, and accounting for individual and group level factors will help to quantify the impact of risk factors at both levels. Furthermore, the use of modern geostatistical techniques will also provide a methodologically sound evidence and better understanding of spatial variation in HIV prevalence leading to identification of high risk geographical locations (hotspots) in Zimbabwe. Risk factors that contribute to excess risk of infection in hotspots will also be studied.

This current study utilized the 2010-11 ZDHS household survey in Zimbabwe to examine underlying and proximate factors that increase an individual's risk for HIV infection, as well as spatial patterns of infection across provincial boundaries. The overall aim is therefore to provide contextual risk factors of HIV infection at individual and group level, and a spatial dimension to understand the geographic distribution of infections. The non-random clustering of HIV infections in specific communities, alongside the contextual knowledge of the key predictors of HIV spread within these high risk clusters must be accounted for in order to garner deeper and plausible new insights into the current state of the generalized epidemic in Zimbabwe.

Such knowledge will present a more robust approach toward population specific interventions based on risk factor profiles. It will also provide better ways to evaluate intervention coverage and effectiveness. The results from this analysis may therefore help improve public health policy and programmatic action for HIV prevention and treatment

options by informing local planning efforts for service provision within identified high risk areas.

1.5 Research question

Does significant heterogeneity exist with regard to the spatial distribution of HIV infection and can this be related to key determinants in Zimbabwe in 2011?

1.6 Research hypotheses

1. Underlying and proximate determinants exist with regard to HIV infection in Zimbabwe.

2. There is heterogeneity with regards to the spatial distribution of HIV infection in Zimbabwe.

3. Differential spatial distribution of underlying determinants may explain the identified hotspots.

1.7 Study objectives

1 To describe the socio-demographic characteristics of the study population.

2 To estimate the prevalence of HIV infection in Zimbabwe in 2011 by socio-demographic and behavioral characteristics.

3 To identify significant underlying and proximate determinants of HIV infection in Zimbabwe in 2011.

4 To Identify spatial hotspots of HIV infection in Zimbabwe in 2011.

5 To assess the risk factor profiles of identified HIV hotspots in Zimbabwe

CHAPTER 2 : METHODOLOGY

2.1 Chapter overview

This study is a secondary analysis of the Zimbabwe Demographic and Health Survey (2010-11 ZDHS). The survey collected three types of data: Zimbabwe demographic and health survey data, HIV indicator survey data and geographic latitude/longitude coordinates data for 406 community clusters. For this secondary analysis, all data were linked with unique identifiers.

In this chapter, pertinent details with regard to the primary and secondary studies are presented. Key aspects of the primary study such as the design, study setting, sampling strategy, outcome and exposure measurement, data collection instruments, and methods set in place to ensure data quality and control are described. This is followed by a description of the methodology of this secondary study, in particular, the crucial aspect of data management, suitable analytical techniques, and ethical considerations.

2.2 Brief description of the primary study

The 2010-11 Zimbabwe Demographic and Health Survey (2010-11 ZDHS) is a large, nationally representative cross sectional sample of 10828 households. This survey was conducted by the Zimbabwe National Statistics Agency (ZIMSTAT) from late September 2010 through March 2011. The 2010-11 ZDHS was a follow-up to previous ZDHS surveys and provides updated estimates of basic demographic and health indicators.

2.3 Study setting

The primary study was conducted in Zimbabwe, a country situated in sub-Saharan Africa with a total population of 12 973 808. The total male population is 6 234 931, while that of

female is 6 738 877. The average size of households is 4.2 with a population density (persons/square km) of 33.[39] The country lies just north of the Tropic of Capricorn between the Limpopo and Zambezi rivers. The country is landlocked, bordered by Mozambique on the east, South Africa on the south, Botswana on the west, and Zambia on the north and northwest. The economy of Zimbabwe is diversified. However, agriculture and mining are by far the country's major foreign-currency earning sectors.

2.4 Study population

The study population consists of Zimbabwean women and men age 15 to 49 living in Zimbabwe between 2010 and 2011.

2.5 Sampling strategy

The sampling frame used was the 2002 Population Census. Administratively, each province in Zimbabwe is divided into districts and each district into smaller administrative units called wards. During the 2002 Population Census, each of the wards was subdivided into enumeration areas (EAs). The 2010-11 ZDHS sample was selected using a stratified, two-stage cluster design and EAs were the sampling units for the first stage. Overall, the sample included 406 EAs, about 42% (169) in urban areas and 58% (237) in rural areas (fig 2.1). Households were the units for the second stage of sampling.

A complete listing of households was carried out in each of the 406 selected EAs in July and August 2010. Maps were drawn for each of the clusters, and all private households were listed. The listing excluded institutional living facilities (e.g., army barracks, hospitals, police camps, and boarding schools). A representative sample of 10 828 households was selected for the 2010-11 ZDHS, of which 10 166 were found to be occupied during the survey fieldwork. Of the 10 166 existing households, 9 756 were successfully interviewed yielding a household

response of 96 percent for women and 82 percent for men. Hence, a total of 16 275 individuals age 15 to 49 (9 171 women and 7 104 men) were successfully interviewed.

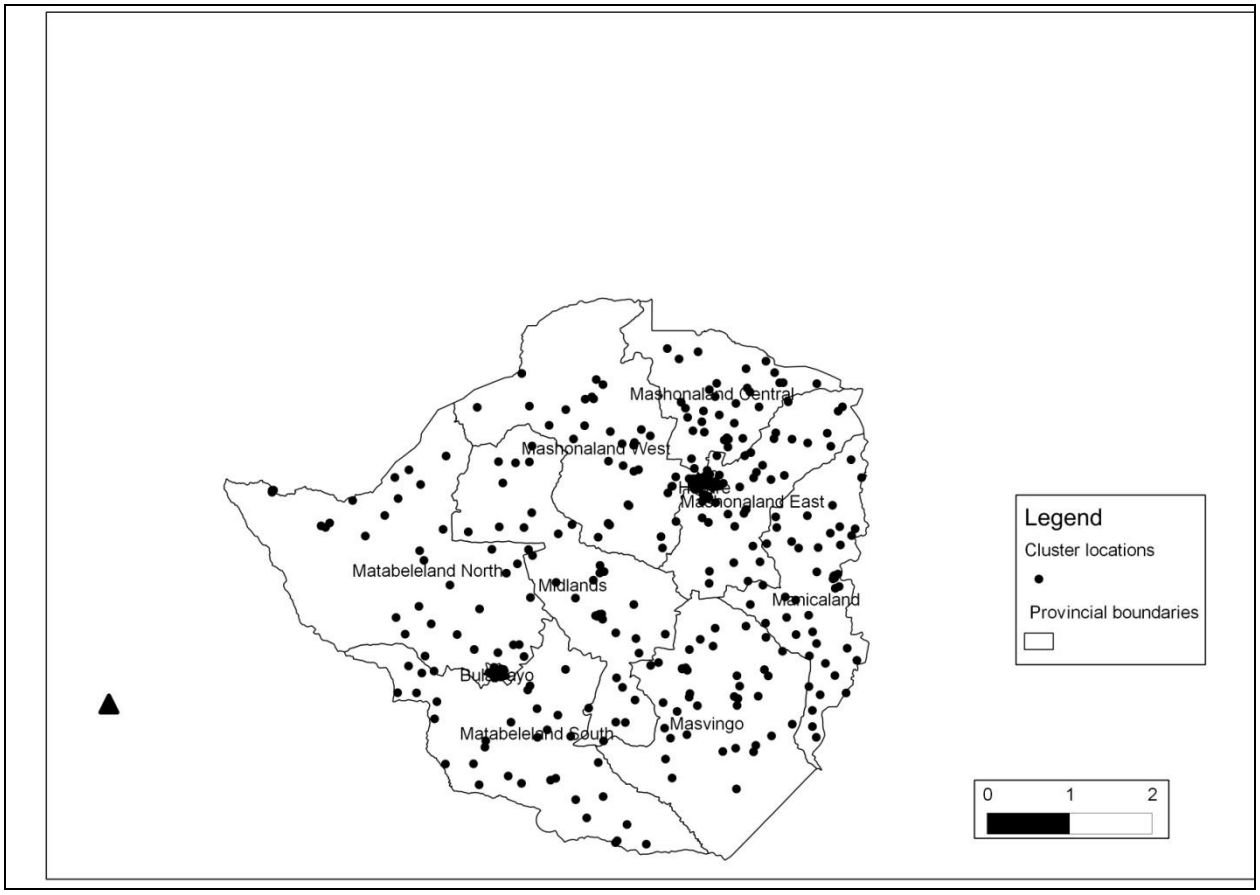


Figure 2.1 Spatial locations of the 406 enumeration areas (EAs), ZDHS 2010-2011

2.6 Data collection

Three sets of data were collected in the primary study using three data collections methods: questionnaire tools, HIV testing and GPS data.

Three questionnaires were employed in the primary study. These questionnaires were adapted from model survey instruments developed for the MEASURE DHS project to reflect population and health issues relevant to Zimbabwe. Pertinent information such as background

characteristics, knowledge and use of family planning methods, fertility preferences, marriage and sexual activity and awareness/behaviour regarding AIDS and sexually transmitted infections (STIs) were obtained from participants. Survey questionnaires were administered through face to face interviews.

In contrast with past ZDHSs, the 2010-11 ZDHS was carried out using the electronic personal digital assistants (PDAs) rather than paper questionnaires for recording responses during interviews. A preliminary report was published in June 2011.[50]

2.7 Measurement of HIV status

The outcome of this study is HIV sero-status. Blood specimens were collected for laboratory testing from participants who provided written consent for HIV testing. The protocol for blood specimen collection and analysis was based on the anonymous linked protocol developed for MEASURE DHS.

2.8 GPS data collection

GPS data was also collected in the study. GPS reading is usually collected at the centre of each cluster. GPS data (cluster id, latitude, longitude, and altitude) for each cluster is stored in two places: in the GPS receiver and on a paper form. ZDHS made use of the recreational grade GPS receiver with positional accuracy between 5 and 15 meters.[51]

2.9 Secondary study

The present study is a secondary analysis of DHS cross-sectional survey data from Zimbabwe in 2010/11. The study investigated the determinants of HIV infection at individual and group levels in Zimbabwe in 2011. The proximate determinant framework was used for investigating significant determinant of HIV infection in the population (fig 2.2). In addition,

the differential spatial distribution of HIV prevalence and risk profile assessment of high risk communities were conducted using various spatial analytical techniques.

2.10 Study measurement

Outcome and explanatory variables were extracted from the primary datasets and used for this analysis as presented in the modified version of the proximate determinant framework (fig 2.2).

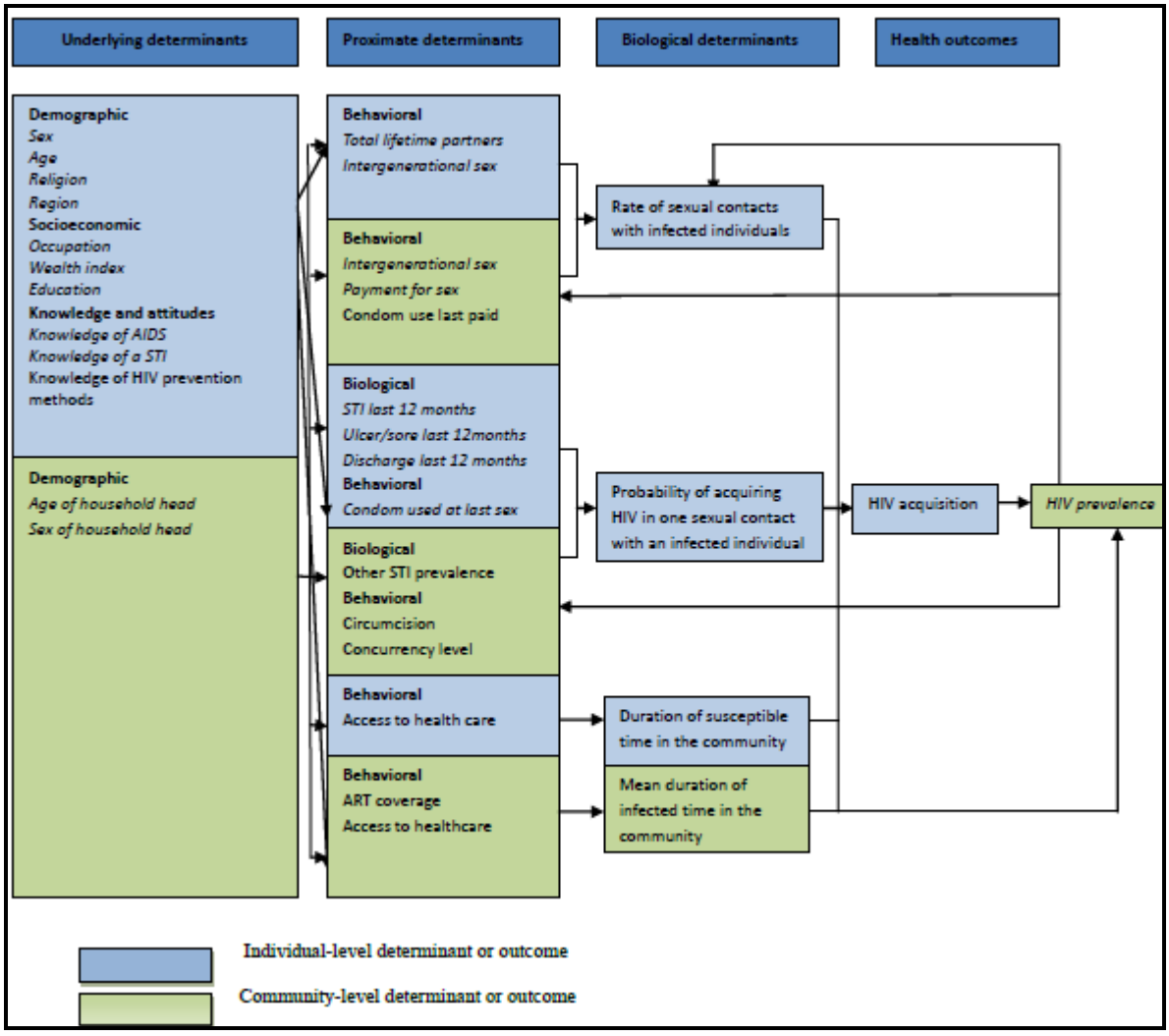


Figure 2.2 Proximate determinants framework of HIV infection (adapted and modified from Barnighausen and Tanser, 2009).

Variables in italics were measured in the survey and used in this analysis.

2.10.1 Outcome variable

HIV sero-status (positive or negative)

2.10.2 Explanatory variables

Explanatory variables were grouped into underlying factors and the proximate factors according to the conceptual framework:

- **Underlying factors**:

  Individual level: age, gender, level of education, religion, marital status, residence, region, employment status, ever heard of a STI, ever heard of AIDS .

  Group level: age of household head, sex of household head

- **Proximate factors**:

  Time away from home in last 12 months**,** age at first cohabitation, duration of cohabitation, STI in last 12 months, genital ulcer/sore in last 12 months, genital discharge in last 12 months, intergenerational sex (defined as age difference of 5 years or more between participant and first second and third most recent partner), age of most recent partner, transactional sex (defined as ever paid anyone in exchange for sex), age at first sex, total number of lifetime partners, and condom used at last sex.

Note: Group-level proximate variables were not available in the original survey data, hence could not be considered in this secondary analysis.

2.11 Data management

Primary datasets for this secondary analysis were supplied in Stata format. Data extraction, cleaning, and recoding were performed in Stata statistical software package (Stata IC version 12.1). Data cleaning involved: checking for inconsistencies in individual records, extreme values and illegal values. Merging of individual to HIV data, household and GPS data was also carried out. Recoding of variables was also carried out.

2.12 Statistical analysis

As a result of the two stage sampling design adopted in the primary study, an analytic procedure which allowed the survey design to be taken into account was used. This procedure accounted for stratification, clustering, and weighting. This therefore helped to obtain correct standard errors and ensured accuracy of effect estimates.

2.12.1 Descriptive analysis

Descriptive analysis was carried out to describe the socio-demographic characteristics of the study population. Prevalence of HIV infection by important socio-demographic and behavioral variables was also carried out. Results were presented in frequencies and percentages using tables along with 95% confidence intervals.

2.12.2 Inferential risk factor (non-spatial) analysis

Three separate mixed effect logistic regression models were fitted. The first model assessed the independent effects of various underlying determinants on HIV infection. In a second model, the independent effects of proximate determinants were examined. A third model was fitted which contained both underlying and proximate determinants. Controlling for proximate risk factors in an underlying risk factors model allows for the quantification of the

direct effects of underlying socio-demographic which bring about exposure of individuals and thus transmission of HIV. This is because underlying factors must operate through proximate factors in order to affect HIV outcome.

A univariate analysis was performed to test for significant association between HIV infection and each of the potential predictor variables. Variables significant at 10% level were selected for inclusion in the multivariable risk factor models. A 5% alpha level was pre-selected as the accepted level of statistical significance in the multivariable risk factor models.

Considering the binary nature of the outcome and the need to account for variations at household and cluster levels, multilevel logistic regression models were fitted using the lme4 package in R [52] in order investigate significant factors associated with HIV infection in Zimbabwe. This package makes use of maximum likelihood (ML) based on Laplace approximation (LA) procedure (which allows for the simplification of the maximum likelihood of fixed and random effects) to implement the multilevel regression analysis.[53] Adjusted odds ratios (AORs) and unadjusted odds ratios (UORs) were reported separately for men and women along with 95% confidence intervals (CIs). The adjusted models were also tested for multicollinearity. Model fit was assessed using Akaike information criteria (AIC) and Bayesian information criteria (BIC) penalized for the number of estimated parameters. [54, 55]

2.12.3 Spatial analytical procedures

2.12.3.1 Interpolation of HIV Prevalence

A type of geostatistical procedure known as ordinary kriging was implemented to construct a prediction surface map along with the estimate of spatial variation using a semi-variogram. The assumption of no spatial trend or drift makes ordinary kriging appropriate in this context.

In order to address spatial property of stochastic or random term with local variation, a semi-variogram model was fitted to the empirical variogram. Also, the nugget effect which is closely linked with error measurement (Gaussian independent spatial residual) was obtained.[56]

This procedure was implemented using the **automap** package in R.[57] As a first step, a descriptive assessment of HIV normality assumption was carried out using histogram. Subsequently the autokrige and autofit variogram functions in the automap package were used to implement the kriging procedure and fit the semi-variogram model. Range, sill and nugget were used to describe the result of the semi-variogram model.

2.12.3.2 Testing for spatial autocorrelation

The need to examine spatial dependence of community centroids is also an important aspect of this study in line with the fourth objective. In this study, two global summary measures of spatial autocorrelation were employed. These global indices express the overall degree of similarity between spatially close clusters in the study area with respect to HIV prevalence. In this study, global Moran's I and Geary's c were estimated under the null hypothesis of no spatial global autocorrelation in the study area, alongside the two-tailed p-values.

However, these global measures only detect the overall clustering tendency, and therefore only yield a summary statistical value which is not necessarily homogenous across all spatial units (in this case community centroids). Consequently, local indicator of spatial associations (LISA) within each cluster unit was also explored to address this issue by calculating the local Moran's I. Thus, the value of the index and the expected value was obtained under the null hypothesis of no local spatial autocorrelation, alongside the standard deviation of the index, the z-value and the corresponding two-tailed p-value.

Local Moran's I

Local Moran (I_i) analysis was carried out using a row standardized (i.e., all the weights in a row sum to one) weight distance matrix on HIV prevalence across the 394 community centroids.[58] A positive value of I_i indicates spatial clustering of similar values (either high or low), and negative values indicate clustering of dissimilar values (i.e., a location with high values surrounded by neighbours with low values).

As recommended by Ord and Getis 1994 [59]**,** a conservative Bonferroni bounds procedure was used to assess significance of local clusters in order to correct for multiple testing. Hence, with an overall α level of 0.05, the individual significance level for each observation was taken as 0.05/393 or 0.000127. This procedure was implemented using the **spdep** packages in R version 2.15.2.[60] This analysis is also not unaware of the limitations of Moran's I especially with respect to the underlying population heterogeneity.

Moran's scatter plot

Following the recommendation of Anselin [58], Moran's scatter plot was used as a device to visualize local instability in spatial. This gives rise to four quadrants which correspond to the four types of spatial association. The lower left and upper right quadrants indicate spatial clustering of similar values; low values in the lower left and high values in the upper right. The upper left and lower right quadrants indicate spatial association of dissimilar values; low values surrounded by high neighbouring values for the former, and high values surrounded by low values for the later. Results are presented in maps.

2.12.3.3 Identification of HIV hotspots using clustering techniques

In order to explore the spatial distribution of HIV prevalence by province, spatial clustering technique was employed, using community centroids as the unit of analysis (smallest geographic unit available in DHS for spatial analysis).

The study region was divided into 394 community centroids indexed i =1, 2, 3,…394 (the remaining 12 census tracts with 0 coordinates were set to missing), with observed and expected (under the null) number of cases equal to Y_i and E_i. Let N_i be the total number of the population at risk in centroid i and λ the prevalence rate of HIV cases for the study region. The null model thus assumes spatial independence and that the number of HIV cases (Yi) are independently distributed as Poisson random variables with mean E_i = λNi. The parameter λ was estimated from the data.

Hotspots (spatial clusters) are defined geographical areas within which there are excess numbers of HIV infections relative to the overall average. Two clustering techniques were employed in order to identify and map significant clusters of HIV cases in the study area; 1) Besag and Newell's method; 2) Kulldorff spatial scan method in order to compare agreement.

Besag and Newell's method

This method was used to detect clusters of a fixed size k, where k was chosen to be 20 within a scan circle, (that is, areas that grouped together, reach 20 observed HIV cases in the study region). This method treats each case as centre of a possible cluster. The other regions are then sorted according to distance to this one, and the number of regions needed until 20 HIV cases are found is computed, yielding a visual output of detected clusters.

Subsequently, a test of whether l_i is low enough to be a cluster, or, put in another way, the probability of finding more than 20 cases in these l_i regions was performed. The p-value (at 5% level) was computed from the mean of the underlying Poisson distribution and the sum of the expected number of cases of the l_i regions (census tracts). The null hypothesis under consideration is that of a spatially homogenous relative risk across the study area.

Kulldorff spatial scan statistic procedure

The spatial scan statistic was used to determine which community falls in clusters of high HIV prevalence, thus providing the number and location of high risk clusters. [61] The scan test is based on a maximum likelihood procedure. In this technique, a circular window is imposed on a map by the statistic and the centre of the circle moves across the study region. This window is centered on each of the possible grid points (community centroids) positioned throughout the study region; the radius of the circle changes continuously between zero and a specified upper limit and is thus flexible both in location and size. [62]

The spatial scan statistic calculates the likelihood of observing the number of cases inside and outside each circle, and the one with the maximum likelihood is defined as the most likely cluster i.e. least likely to have occurred by chance with the null hypothesis that the risk of HIV infection is the same in all clusters). The general statistical theory behind the spatial scan statistics in Sat Scan software is described in detail by Kulldorff. [61, 62]

The assumption was that the number of disease cases in different locations has independent Poisson distributions. This is because the number of cases was significantly smaller than the population at risk. More so, Poisson model is best suited for questions with case and population-at-risk counts. Computation of the maximum likelihood ratio, under the assumption of a Poisson model was carried out conditioning to the total number of observed cases. [61] The null hypothesis was rejected at a significance level of 5% significance. A single p-value associated with the test statistic each for the most likely cluster (primary cluster) and secondary clusters that were significant at 5% level were reported after scanning for 394 possible cluster candidates.

The two assumptions of scan statistics are the Poisson distribution and spatial independence of HIV cases in different locations within the study area. [61] Spatial autocorrelation in

overlapping census tracts are therefore accounted for in the Kulldorff spatial scan statistics procedure by using Monte Carlo simulation to approximate the distribution of the scan statistics. The sensitivity of the spatial scan procedure was also explored. This was accomplished by running 10 scans and varying the prevalence from 5% to default 50% in order to ascertain the stability of significant clusters obtained as recommended by Kulldorff. [61, 62]

Calculations were carried out using SatScan version 9.0 http://www.satscan.org, and results were imported into the Stata (Version 12.1, CS, Texas) software environment to compare the characteristics of hotspots(high risk clusters) and coldspots (low risk clusters).

2.12.3.4 Spatial regression procedure

Bayesian structured and unstructured geoadditive regression models were also applied to the country survey data. [63] This was implemented in order to visually assess the adjusted prevalence distribution of HIV infection across the ten provinces in Zimbabwe.

This analytic approach therefore made possible the exploration of HIV prevalence geographic patterns within a Bayesian regression framework adjusting for significant underlying and proximate risk factors. [63] This procedure also allowed for the quantification of the posterior means of prevalence distribution across the ten regions. [64] Hence, a total of eight models were fitted, (four structured and four unstructured models).

These models vary in complexity from an empty unstructured model adjusted for non-spatial random effects (at household, cluster and provincial levels) to a model in which both underlying and proximate risk factors were adjusted for, along with spatial and non-spatial random effects. A small DIC value indicates better fit. [55] Consequently, the model with the smallest DIC value was selected.

Model fit was assessed using the deviance information criterion (DIC) while significance of the posterior means was evaluated using the Bayesian credibility interval (BIC). [55, 56] Models were run for a burn in of 5000 and a step size of 20. This was followed by parameter estimation until convergence using Markov Chain Monte Carlo (MCMC) simulation procedure at 55000 iterations. Software implementation was carried out in BayesX version 2.1. [55, 56]

2.12.3.5 Risk factor profile assessment of hotspots

Following the mapping and identification of significant clusters of HIV infection, risk factor profile assessment of factors associated with HIV infection were carried out within HIV hotspots. A bivariate logistic regression analysis was carried out to test for significant difference between hotspots (high-risk clusters) and coldspots (average and low-risk clusters) with respect to underlying and proximate covariates. This analysis was implemented at the cluster level. Hence, the 406 clusters (EAs) were adjusted for using robust standard error estimates.

Implementation Softwares

All analyses were carried out using Stata/IC version 12.1, SatScan version 9.0, R version 2.15.2, QGIS version 1.8.0 and BayesX version 2.1.

CHAPTER 3 : RESULTS

3.1 Socio-demographic characteristics of the study population

A total of 16 275 individuals age 15 to 49 were enrolled in the survey. Out of this, 9 171 were women and 7 104 were men. The socio-demographic profile of the study population is presented in table 3.1 below.

Table 3.1. Socio-demographic characteristics of the study participants

Variables	Women 9171 N_f (%)	Men 7104 N_m(%)	Total 16275 N_t(%)
Age group			
15-19	1945(21.2)	1733(24.4)	3678(22.6)
20-24	1841(20.0)	1371(19.3)	3213(19.7)
25-29	1686(18.4)	1235(17.4)	2921(18.0)
30-34	1295(14.1)	970(13.7)	2265(13.9)
35-39	1051(11.5)	827(11.6)	1878(11.5)
40-44	733(8.0)	589(8.3)	1322(8.1)
45-49	620(6.8)	379(5.3)	998(6.1)
Residence			
Urban	3548(38.7)	2619(36.9)	6167(37.9)
Rural	5623(61.3)	4485(63.1)	10108(62.1)
Marital Status			
Never in union	2197(24.0)	3218(45.3)	5416(33.3)
Married	5443(59.3)	3528(49.7)	8971(55.1)
Living with partner	260(2.8)	53(0.7)	312(2.0)
Widowed	559(6.1)	66(0.9)	625(3.8)
Divorced	336(3.7)	121(1.7)	457(2.8)
Separated	376(4.1)	117(1.7)	493(3.0)
Level of Education			
None	213(2.3)	55(0.8)	269(1.7)
Primary	2568(28.0)	1507(21.2)	4075(25.0)
Secondary	5966(65.1)	5023(70.7)	10989(67.5)
Higher	424(4.6)	519(7.3)	942(5.8)
Region			
Manicaland	1226(13.4)	971(13.7)	2197(13.5)
Mashonaland central	871(9.5)	737(10.4)	1608(9.9)
Mashonaland east	824(9.0)	666(9.4)	1491(9.2)
Mashonaland west	1026(11.2)	872(12.3)	1897(11.6)
Matabeleland north	443(4.8)	349(4.9)	793(4.9)

Matabeleland south	468(5.0)	352(4.9)	819(5.0)
Midlands	1123(12.3)	884(12.4)	2008(12.3)
Masvingo	909(9.9)	585(8.2)	1494(9.2)
Harare	1722(18.8)	1306(18.4)	3029(18.6)
Bulawayo	559(6.1)	382(5.4)	939(5.8)
Religion			
None	557(6.1)	1525(21.5)	2083(12.8)
Christian	8509(92.8)	5248(74.0)	13757(84.6)
Muslim	43(0.5)	42(0.6)	84(0.5)
Traditional	57(0.6)	280(3.9)	337(2.1)
Sex of household head			
Male	4676(51.0)	5001(70.4)	9679(59.5)
Female	4491(49.0)	2103(29.6)	6596(40.5)
Age of household head			
<25	744(8.1)	643(9.1)	1388(8.5)
25-35	2940(32.1)	2275(32.0)	5215(32.0)
>35	5487(59.8)	4186(58.9)	9672(59.4)
Wealth Index			
Poorest	1546(16.9)	1073(15.1)	2619(16.1)
Poorer	1594(17.4)	1215(17.1)	2809(17.3)
Middle	1681(18.3)	1370(19.3)	3050(18.7)
Richer	2072(22.6)	1662(23.4)	3735(22.9)
Richest	2278(24.8)	1784(25.1)	4062(25.0)
Occupation			
Not working	5216(56.9)	2209(31.1)	7425(45.6)
Professional	337(3.7)	403(5.7)	741(4.6)
Non-professional	2764(30.1)	3053(43.0)	5818(35.8)
Agriculture	818(8.9)	1399(19.7)	2218(13.6)

N_f: number of females in each variable category; N_m: number of males in each variable category Nt: total number of persons in each variable category.

Among men, about 14% were older than 40 years with approximately 44% younger than 25 years. Sixty three percent were from the rural areas. While about 50% of the men were married, less than 1% was widowed. Most men (92%) either had a primary or secondary education (table 3.1).

Among women, a similar pattern of age distribution was obtained, 41% of who were younger than 25 years. Similarly, 61% of women were from rural areas. A reported 59% of women

were married while, about 6% were widowed. Although men and women showed similar pattern for level of education, a higher percentage of men reported higher levels of education. Forty-nine percent of women also reported living in female-headed households compared to 29% among men as presented in table 3.1 above.

3.2 HIV prevalence distribution

Out of the 13583 individuals aged 15 to 49 years that were tested, 2093 were HIV sero-positive translating to an overall HIV prevalence of 15.4% (95%CI: 14.8% - 16.0%) in the study population. Eighteen percent of women were sero-positive while an estimate of 12.2% was obtained among the men. HIV prevalence estimates by important underlying and proximate variables are presented in table 3.2 and table 3.3 below.

3.2.1 HIV prevalence distribution by underlying (socio-demographic) characteristics

Of the twelve underlying variables studied, seven showed significant bivariate associations with HIV sero-status at 10% in both men and women. These were age group, marital status, occupation, sex of household head and age of household, ever heard of HIV and ever heard of a STI as presented in table 3.2 below. The patterns of associations were different for men and women for the seven factors. In addition, residence, level of education and wealth index were significantly associated with HIV in women.

Across age groups, women in the middle age groups, 30 to 34 and 35 to 39, recorded the highest HIV prevalence of 29.3%. A percentage point increase of about 3% was observed in urban women compared to rural women. Across marital status categories, those who were never in union had the lowest prevalence (7.6%) and those who were widowed had the highest prevalence (56%). By education, women with post-secondary education had the lowest prevalence of 13.4%. Women who live in female-headed households (21.1%) were more likely to be infected than women who live in male-headed households (14.6%). Highest prevalence was also recorded in women resident in Matabeleland South (22.6%).

While a parabolic distribution of HIV prevalence was obtained across age groups in women, there was a consistent dose-response association across age groups in men. The lowest prevalence was found in the youngest age group of 15 to 19 years and the highest prevalence in the oldest age group of 45 to 49 years as shown in table 3.2 below. While about 3% point difference was found between urban and rural women, urban men differed from rural men with a marginal 1% point. Like women, widowed men had the highest prevalence.

Similarly, the highest prevalence was found in men resident in Matabeleland South (19.2%) while men who had post-secondary education recorded the lowest prevalence. It is interesting to note that as opposed to women, men in female-headed households showed significantly less prevalence than those in male-headed houses.

Table 3.2. HIV prevalence distribution among participants by underlying characteristics

Variable	Women			Men			Total	
	HIV prevalence (%)	N_f	p-value	HIV Prevalence (%)	N_m	p-value	HIV Prevalence (%)	N_t
Age group			**<0.001**			**<0.001**		
15-19	4.3	1690		3.4	1524		3.9	3214
20-24	10.5	1559		3.9	1081		7.8	2640
25-29	20.1	1473		10.2	943		16.0	2416
30-34	29.3	1082		16.9	774		24.1	1856
35-39	29.3	882		25.1	647		27.5	1529
40-44	25.5	626		25.7	452		25.6	1078
45-49	22.9	540		30.6	310		25.7	850
Residence			0.022			0.370		
Urban	19.5	2659		12.9	1665		16.9	4324
Rural	16.8	5193		11.9	4066		14.7	9259
Marital status			**<0.001**			**<0.001**		
Never in union	7.6	1956		3.9	2678		5.4	4634
Married	15.7	4546		17.5	2755		16.4	7301
Livewith partner	27.6	230		23.3	37		27.1	267
Widowed	56.0	516		60.5	52		56.4	568
Divorced	26.7	269		31.1	100		27.8	369
Separated	31.4	335		30.3	109		31.1	444
Level of Education			0.002			0.162		

None	15.8	182	15.0	55	15.6	237
Primary	20.4	2325	13.6	1416	18.0	3741
Secondary	16.9	5023	12.1	3921	14.8	8944
Higher	13.4	322	9.1	339	11.2	661
Region			0.284		0.002	
Manicaland	18.0	890	9.9	670	14.5	1560
MC	15.1	812	12.4	685	13.9	1497
ME	17.8	762	13.2	602	15.8	1364
MW	18.1	833	11.7	692	15.2	1525
MN	19.9	677	15.8	453	18.1	1130
MS	22.6	720	19.2	533	21.2	1253
Midlands	17.9	882	13.1	706	15.9	1588
Masvingo	16.3	720	11.8	424	14.6	1144
Harare	16.7	985	9.3	662	13.7	1647
Bulawayo	20.9	571	16.8	304	19.4	875
Occupation			**<0.001**		**<0.001**	
Not working	15.0	4650	8.9	1909	13.3	6559
Professional	19.1	266	10.5	267	14.7	533
Non-profesional	22.2	2220	14.6	2298	18.4	4518
Agriculture	19.3	688	12.7	1231	15.2	1919
Wealth index			0.008		0.276	
Poorest	17.1	1560	14.6	1063	16.1	2623
Poorer	16.5	1426	12.2	1080	14.7	2506
Middle	20.0	1433	12.1	1161	16.6	2594
Richer	19.9	1691	11.6	1254	16.3	2945
Richest	15.4	1742	11.2	1173	13.7	2915
Religion			0.133		**<0.001**	
None	20.0	510	15.9	1364	17.0	1874
Christian	17.6	7249	10.8	4105	15.1	11354
Muslim	26.3	35	19.9	31	23.3	66
Traditional	17.2	55	16.6	226	16.7	281
Sex of hhold head			**<0.001**		**<0.001**	
Male	14.6	3938	13.2	3945	13.9	7883
Female	21.1	3914	9.9	1786	17.6	5700
Age of hhold head			**<0.001**		**<0.001**	
<25	10.0	608	5.1	506	7.8	1114
25-35	18.4	2421	12.4	1734	15.9	4155
>35	18.4	4823	13.2	3491	16.3	8314
Ever heard of a STI			0.075		0.002	
No	11.8	151	2.4	79	8.8	230
Yes	17.9	7701	12.3	5652	15.5	13353

Ever heard of AIDS			0.038		0.017	
No	11.3	180	4.8	104	9.1	284
Yes	17.9	7672	12.3	5627	15.5	13299

MC:, mashonaland central, ME: mashonaland east, MW: mashonaland west, MS: matabelend south, MN: Matabeleland north. N_f : number of females in each variable category; N_m: number of males in each variable category Nt: total number of persons in each variable category. Highly significant p-values are printed in bold.

3.2.2 HIV prevalence distribution by proximate characteristics

Of the twelve proximate variables examined, nine had significant bivariate associations with HIV sero-status; age at first sex, condom used at last sex, duration of cohabitation, total lifetime partners, had any STI, genital sore/ulcer, or discharge in last 12 months, age of most recent partner and intergenerational sex, in both men and women as presented in table 3.3 below.

Across age at first sex categories, the highest prevalence was recorded among women who had their sexual debut below the age of 16 years (24.2%). By condom use, the prevalence was approximately three times higher in women who used condom during sex with most recent partner. A dose response pattern of association was also found across duration of cohabitation, total lifetime partners and age of most recent partner among women as shown table 3.3 below.

By age at first sex, the highest prevalence (about 16%) was observed in men who had their sexual debut between age 16 and 19 years unlike in women. Similarly, prevalence was found to increase with duration of cohabitation, total lifetime partners and age of most recent partner in men. In both gender populations, prevalence was at least two times higher in individuals who had any STI, genital ulcer/sore, and genital discharge in last 12 months as shown in table 3.3 below.

Table 3.3. HIV Prevalence distribution among participants by proximate factors

Variable	Women			Men			Total	
	HIV prevalence	N_f	p-value	HIV prevalence	N_m	p-value	HIV prevalence	N_t
Age at first sex			**<0.001**			**<0.001**		
not had sex	3.6	1385		3.5	1449		3.5	2834
<16	24.2	1049		14.6	486		21.2	1535
16-17	20.8	1853		15.7	815		19.3	2668
18-19	20.2	1714		15.7	1050		18.5	2764
20+	19.6	1851		14.7	1931		17.1	3782
Time away from home in last 12 months			0.144			0.075		
None	17.2	3427		12.3	2817		14.9	6244
1-2	17.0	2148		10.0	1112		14.7	3260
3-4	18.1	909		13.0	572		16.2	1481
5+	20.0	1368		13.7	1230		16.9	2598
Condom used at last sex			**<0.001**			**<0.001**		
No	14.6	4532		14.2	2767		14.4	7299
Yes	41.0	789		19.3	1076		28.5	1865
Age at first cohabitation			**<0.001**			0.183		
<15	24.9	374		32.1	30		25.3	404
15-17	17.6	1825		14.5	131		17.4	1956
18-19	18.7	1504		17.5	330		18.5	1834
20+	24.5	2193		19.4	2562		21.7	4755
Duration of cohabitation			**<0.001**			**<0.001**		
Never married	7.6	1956		3.9	2678		5.4	4634
0-4	13.8	1610		11.8	886		13.1	2496
5-9	19.1	1272		17.5	727		18.5	1999
10+	25.6	3014		24.6	1440		25.3	4454
Total lifetime partners			**<0.001**			**<0.001**		
1	12.2	3954		3.9	793		10.9	4747
2-4	35.1	2240		13.1	1875		24.5	4135
5-9	46.5	176		20.6	905		24.2	1081
10+	44.3	97		26.5	689		28.6	786
Times in last 12 months had sex with most recent partner			0.687			**<0.001**		

1	21.0	132	8.5	241	12.9	373
2-3	20.0	151	9.4	279	13.0	430
>3	18.3	5038	16.5	3323	17.6	8361
Had any STI in last 12 months			**<0.001**		**<0.001**	
No	17.0	7616	11.9	5585	14.9	13201
Yes	40.8	227	25.9	140	35.0	367
Had genital ulcer/sore in last 12 months			**<0.001**		**<0.001**	
No	16.8	7541	11.6	5556	14.6	13097
Yes	40.2	292	31.3	173	36.9	465
Had genital discharge in last 12 months			**<0.001**		0.003	
No	16.8	7418	12.0	5592	14.7	13010
Yes	33.1	398	22.8	134	30.5	532
Age of most recent partner			**<0.001**		**<0.001**	
<19	2.9	55	4.6	844	4.5	899
20-24	5.8	533	13.6	984	10.9	1517
25-29	12.7	1116	16.5	804	14.3	1920
30-34	19.8	1046	22.5	567	20.8	1613
35+	23.2	2571	24.7	644	23.5	3215
Intergenerational sex			**<0.001**		**<0.001**	
No	18.2	5263	11.9	1935	16.5	7198
Yes	38.2	58	19.2	1908	19.7	1966

N_f : number of females in each variable category; N_m: number of males in each variable category Nt: total number of persons in each variable category. Highly significant p-values are printed in bold.

3.3 Independent underlying determinants of HIV infection in Zimbabwe

Among women, the following underlying variables were found to be significantly associated with HIV infection after adjusting for other underlying variables in both gender populations: age group, residence, marital status, and region as shown in table 3.4 below. Among men, residence, marital status, and region, were equally significant determinants of HIV infection.

Among women, the highest odds of HIV infection was found in women age 30 to 34 years (AOR: 8.30; 95%CI: 6.05 – 11.38) adjusting for other underlying factors. In addition, the adjusted effect of residence resulted in 35% reduction in the odds of HIV infection among women in rural settings compared to their counterparts in urban areas holding other underlying factors constant as shown table 3.4 below. Women who lost their spouses (widows) had the highest odds of HIV infection (AOR: 5.64; 95%CI: 4.17 – 7.63). In addition, a 69% greater odds of HIV infection was found in women resident in Matabeleland South controlling for other underlying factors (AOR: 1.69; 95%CI: 1.25 – 2.30**)**.

Among men, a dose-response relationship in odds of HIV infection was found across age groups. Consequently, men age 15 to 19 years had the lowest odds of HIV infection and those between 45 to 49 years had the highest odds of infection (AOR:8.45; 95%CI: 5.17 – 13.78) holding other underlying factors constant as shown in table 3.4 below. A similar reduction of approximately 35% was observed in the odds of HIV infection in men who lived in rural settings relative to their urban counterparts (table 3.4). Highest odds of HIV infection were also found in widowed men (AOR: 11.22; 95%CI: 5.71 – 22.04)) and men who lived in Matabeleland South controlling for other underlying factors (AOR: 2.71; 95%CI: 1.81 – 4.05).

Table 3.4. Model 1: Multilevel model of underlying determinants of HIV infection in Zimbabwe

Variable	Women UOR(95%CI)	AOR(95%CI)	Men UOR(95%CI)	AOR(95%CI)
Age group				
15-19	1.00	1.00	1.00	1.00
20-24	2.73(2.07 – 3.60)	**2.74(2.02 – 3.72)**	1.37(0.92 – 2.05)	1.19(0.77 – 1.83)
25-29	5.63(4.34 – 7.31)	**5.54(4.08 – 7.52)**	3.89(2.76 – 5.50)	**2.88(1.87 – 4.43)**
30-34	8.99(6.91 – 11.69)	**8.30(6.05 – 11.38)**	6.70(4.79 – 9.37)	**4.49(2.86 – 7.03)**
35-39	9.05(6.91 – 11.86)	**7.78(5.62 – 10.77)**	9.93(7.12 – 13.85)	**6.44(4.09 – 10.16)**
40-44	7.35(5.50 – 9.83)	**4.80(3.38 – 6.82)**	12.27(8.67 – 17.35)	**8.34(5.22 – 13.32)**
45-49	6.75(5.00 – 9.13)	**4.08(2.80 – 5.94)**	13.67(9.44 – 19.78)	**8.45(5.17 – 13.78)**
Residence				
Urban	1.00	1.00	1.00	1.00
Rural	0.84(0.74 – 0.94)	**0.64(0.52 – 0.78)**	0.94(0.80 – 1.12)	**0.63(0.48 – 0.82)**
Marital status				
Never	1.00	1.00	1.00	1.00
Married	2.19(1.83 – 2.62)	0.95(0.75 – 1.20)	5.04(4.09 – 6.21)	**1.70(1.23 – 2.34)**
Living with partner	4.59(3.30 – 6.37)	**2.20(1.52 – 3.20)**	9.24(4.46 – 19.16)	**3.39(1.51 – 7.62)**
Widowed	14.11(11.13 – 17.88)	**5.64(4.17 – 7.63)**	37.94(20.95 68.73)	**11.22(5.71 – 22.04)**
Divorced	4.53(3.32 – 6.18)	**1.97(1.39 – 2.80)**	10.92(6.92 – 17.25)	**4.83(2.83 – 8.24)**
Separated	5.79(4.38 – 7.64)	**2.41(1.76 – 3.31)**	10.77(6.94 – 16.73)	**3.84(2.29 – 6.42)**
Level of Education				
None	1.00	1.00	1.00	1.00
Primary	1.19(0.80 – 1.75)	1.47(0.96 – 2.25)	1.05(0.49 – 2.25)	1.52(0.66 – 3.51)
Secondary	0.95(0.65 – 1.40)	1.43(0.93 – 2.20)	0.84(0.39 – 1.78)	1.41(0.61 – 3.26)
Higher	0.67(0.40 – 1.10)	0.69(0.40 – 1.19)	0.68(0.30 – 1.55)	0.73(0.29 – 1.81)
Region				
Manicaland	1.00	1.00	1.00	1.00
MC	0.87(0.68 – 1.12)	0.90(0.66 – 1.22)	1.30(0.93 – 1.83)	1.47(0.99 – 2.17)
ME	0.97(0.76 – 1.25)	0.98(0.72 – 1.33)	1.29(0.91 – 1.82)	1.42(0.95 – 2.13)
MW	1.03(0.80 – 1.31)	1.00(0.74 – 1.34)	1.24(0.89 – 1.74)	1.22(0.83 – 1.81)
MN	1.17(0.91 – 1.51)	1.31(0.96 – 1.80)	1.90(1.34 – 2.69)	**2.05(1.35 – 3.10)**
MS	1.37(1.07 – 1.74)	**1.69(1.25 – 2.30)**	2.00(1.42 – 2.76)	**2.71(1.81 – 4.05)**
Midlands	0.98(0.76 – 1.24)	1.05(0.78 – 1.42)	1.30(0.93 – 1.81)	1.24(0.84 – 1.83)
Masvingo	0.88(0.68 – 1.14)	0.96(0.70 – 1.32)	1.16(0.78 – 1.71)	1.22(0.78 – 1.90)
Harare	0.91(0.72 – 1.16)	0.66(0.48 – 0.92)	0.95(0.66 – 1.36)	0.71(0.45 – 1.13)
Bulawayo	1.20(0.92 – 1.56)	0.98(0.68 – 1.39)	1.67(1.12 – 2.49)	1.45(0.88 – 2.40)
Sex of house head				
Male	1.00	1.00	1.00	1.00
Female	1.58(1.41 – 1.77)	1.14(0.99 – 1.31)	0.73 (0.61 – 0.87)	1.15(0.94 – 1.41)
Cluster variance(sd)		0.36(0.31)		0.38(0.29)

MC:, mashonaland central, ME: mashonaland east, MW: mashonaland west, MS: matabelend south, MN: Matabeleland north; 95%CI: 95% confidence interval; UOR: unadjusted odds ratio, AOR: adjusted odds ratio; All significant variables with p-value<0.05 are printed in bold. Women:-AIC: 6526; BIC: 6721. Men:- AIC: 3777; BIC: 3963

3.4 Independent proximate determinants of HIV infection in Zimbabwe.

Results showed that condom used at last sex, total lifetime partners, had any STI, genital sore/ulcer, or genital discharge in the last 12 months, and age of most recent partner were significant predictors of HIV infection among women adjusting for other proximate factors as presented in table 3.5 below. Similarly, condom used at last sex, total lifetime partners, had any STI, or genital sore/ulcer in the last 12 months, and age of most recent partner were significant predictors of HIV infection among men. In addition intergenerational sex was found to be a significant predictor of infection in men.

Among women, 2.84 higher odds of HIV infection was found in women who used condom during last sex (AOR: 2.84; 95%CI: 2.36 – 3.41) adjusting for other proximate factors. Total lifetime partners increased the odds of HIV infection from two partners (AOR: 3.36; 95%CI: 2.86 – 3.95**)** to nine lifetime sexual partners (AOR: 4.18; 95%CI: 2.89 – 6.03). Greater odds of infection were also obtained in women who had STI (AOR: 1.85; 95%CI: 1.25 – 2.74**)** or STI symptoms in the last 12 months as shown in table 3.5 below. However, evidence only attained statistical significance in women whose recent partner was 30 years and older.

Among men, 2.01 higher odds of HIV infection was obtained in men who used condom during last sex (2.01; 95%CI: 1.63 - 2.48). Furthermore, a linear increase in odds of HIV infection was observed with increase in lifetime partners and age of most recent partner. Consequently, men who had lifetime ten or more partners had 4.76 higher odds of HIV infection relative to men who had only one partner. Similarly, higher odds of infection were

observed in men who had any STI (AOR: 2.17; 95%CI: 1.39 – 3.39**)** and genital sore/ulcer (AOR: 2.17; 95%CI: 1.39 – 3.39) in the last 12months. The odds of HIV infection increased by 73% in men who practiced intergenerational sex as shown in table 3.5 below.

Table 3.5. Model 2: Multilevel model of proximate determinants of HIV infection in Zimbabwe

Variable	Women UOR(95%CI)	AOR(95%CI)	Men UOR(95%CI)	AOR(95%CI)
Condom used at last sex				
No	1.00	1.00	1.00	1.00
Yes	3.86(3.28 – 4.55)	**2.84(2.36 – 3.41)**	1.40(1.17 – 1.68)	**2.01(1.63 – 2.48)**
Total lifetime partners				
1	1.00	1.00	1.00	1.00
2-4	3.86(3.39 – 4.39)	**3.36(2.86 – 3.95)**	3.51(2.43 – 5.07)	**2.50(1.68 – 3.72)**
5-9	6.35(4.65 – 8.67)	**4.18(2.89 – 6.03)**	6.31(4.32 – 9.20)	**3.65(2.42 – 5.51)**
10+	4.99(3.27 – 7.60)	**3.91(2.26 – 6.75)**	8.99(6.14 -13.16)	**4.76(3.13 – 7.22)**
Had any STI in the last 12 months				
No	1.00	1.00	1.00	1.00
Yes	3.87(2.96 – 5.07)	**1.85(1.25 – 2.74)**	3.27(2.35 – 4.56)	**2.17(1.39 – 3.39)**
Had genital ulcer/sore in the last 12 months				
No	1.00	1.00	1.00	1.00
Yes	3.71(2.93 – 4.71)	**1.42(1.02 – 1.96)**	3.27(2.35 – 4.56)	**2.17(1.39 – 3.39)**
Had genital discharge in the last 12 months				
No	1.00	1.00	1.00	1.00
Yes	2.66(2.15 – 3.29)	**1.44(1.05 – 1.96)**	2.26(1.51 – 3.37)	1.09(0.62 – 1.91)
Age of most recent partner				
<19	1.00	1.00	1.00	1.00
20-24	1.88(0.44 – 8.04)	3.53(0.45 – 27.9)	3.18(2.23 – 4.53)	**3.18(2.18 – 4.64)**
25-29	4.25(1.03 – 17.62)	7.07(0.91 – 54.72)	4.06(2.84 – 5.79)	**4.32(2.94 – 6.36)**
30-34	7.16(1.73 – 29.61)	**11.95(1.55 – 92.34)**	5.69(3.95 – 8.18)	**6.20(4.18 – 9.20)**
35+	8.25(2.01 – 33.96)	**12.63(1.64 – 97.14)**	6.87(4.83 – 9.77)	**7.83(5.34 – 11.49)**
Intergenerational				

sex				
No	1.00	1.00	1.00	1.00
Yes	2.12(1.22 – 3.69)	1.11(0.60 – 2.07)	1.84(1.54 – 2.19)	**1.73(1.43 – 2.10)**
Cluster level variance(sd)		0.12(0.34)		0.18(0.43)

UOR: unadjusted odds ratio, AOR: adjusted odds ratio; All significant variables with p-value<0.05 are printed in bold; Women:-AIC: 4473; BIC: 4571. Men:- AIC:3073; BIC: 3167

3.5 A model adjusting for significant underlying and proximate determinants of HIV infection in Zimbabwe.

In order to understand which proximate determinants identified above mediate the associations between underlying determinants and HIV, a third model was built which included underlying determinants adjusted for proximate determinants of HIV infection in the study population as presented in table 3.6 below. Overall, the effects of age group, residence, marital status and region remained significant after adjusting for proximate factors in both men and women as shown in table 3.6.

Among women, a substantial reduction in the effect of age group was obtained. For instance, the adjusted odds of HIV decreased from 8.30 to 2.17 in women age 30 to 34 years and a similar reduction from 7.78 to 1.91 was observed in women aged 35 to 39 years (model 1 versus model 3). However the effect of age remains significant in women in the middle age groups.

Strong evidence was also found that women who lived in rural settings had 38% lower odds of HIV infection. In addition, being a widow is associated with a substantially higher odds of being HIV sero-positive (AOR: 4.13; 95%CI: 2.49 – 6.85), adjusting for other underlying and proximate factors as shown in table 3.6 below.

Similarly, on controlling for proximate determinants, previously observed pattern of association between region and odds of HIV infection drifted toward the null value, most

notably in the women population (table 3.6). Nevertheless, evidence remained significant with lower odds of 37% and 35% observed in women who lived in Harare and Bulawayo respectively as shown in table 3.6 below.

In addition, condom use at last sex, total lifetime partners, had any STI, genital sore/ulcer, or genital discharge in the last 12 months, remained significant proximate predictors of higher odds of HIV infection in women as shown in table 3.6 below.

Among men, a similar reduction and shift toward the null value (though to a lesser extent) was found across all age groups, except in men age 20 to 24 who had a slightly higher odds of HIV infection, controlling for other underlying and proximate factors in the model (table 3.6). However, age remains a highly significant predictor of higher odds of HIV Infection in men than in women with the increasing linear trend preserved with increase in age groups. About 30% reduction in the odds of HIV infection was also obtained in rural men (AOR: 0.69; 95%CI: 0.51 – 0.92).

Similarly, men across all marital status categories had an overwhelming significant evidence of higher odds of HIV infection relative to men who were never in union while widowers remained the strongest predictor of HIV infection (AOR: 7.37; 95%CI: 3.10 – 17.52). However, widowhood seems to be a more significant predictor of HIV infection in men than in women.

Highest UOR occurred in Matabeleland South and this was not explained by the factors in the model. Moreover, condom use at last sex, total lifetime partners, had any STI or genital sore/ulcer in the last 12 months, remained significant proximate predictors of higher odds of HIV infection in men (table 3.6).

This result therefore demonstrates the positive confounding effect of proximate risk factors which result in biased effect estimates of the true effect of age group and marital status on HIV outcome in the context of Zimbabwe generalized epidemic.

Table 3.6. Model 3: Multilevel model of underlying and proximate determinants of HIV infection in Zimbabwe

	Women		Men	
Variable	UOR(95%CI)	AOR (95%)	UOR(95%CI)	AOR(95%CI)
Age group				
15-19	1.00	1.00	1.00	1.00
20-24	2.73(2.07 – 3.60)	1.27(0.81 – 1.98)	1.37(0.92 – 2.05)	1.29(0.56 – 2.97)
25-29	5.63(4.34 – 7.31)	**1.73(1.09 – 2.74)**	3.89(2.76 – 5.50)	**2.14(0.89 – 5.16)**
30-34	8.99(6.91 – 11.69)	**2.17(1.32 – 3.54)**	6.70(4.79 – 9.37)	**3.38(1.35 – 8.46)**
35-39	9.05(6.91 – 11.86)	**1.91(1.14 – 3.19)**	9.93(7.12 – 13.85)	**4.42(1.70 – 11.46)**
40-44	7.35(5.50 – 9.83)	1.18(0.68 – 2.05)	12.27(8.67 – 17.35)	**5.19(1.92 – 14.02)**
45-49	6.75(5.00 – 9.13)	1.11(0.62 – 1.99)	13.67(9.44 – 19.78)	**4.76(1.70 – 13.30)**
Residence				
Urban	1.00	1.00	1.00	1.00
Rural	0.84(0.74 – 0.94)	**0.62(0.49 – 0.78)**	0.94(0.80 – 1.12)	**0.69(0.51 – 0.92)**
Marital status				
Never	1.00	1.00	1.00	1.00
Married	2.19(1.83 – 2.62)	**1.40(0.98 – 2.00)**	5.04(4.09 – 6.21)	**3.06(2.01 – 4.68)**
Living with part	4.59(3.30 – 6.37)	**2.09(1.31 – 3.35)**	9.24(4.46 – 19.16)	**6.36(2.57 –15.74)**
Widowed	14.11(11.13 – 17.88)	**4.13(2.49 – 6.85)**	37.94(20.95 68.73)	**7.37(3.10 –17.52)**
Divorced	4.53(3.32 – 6.18)	0.99(0.59 – 1.66)	10.92(6.92 – 17.25)	**3.95(2.07 – 7.54)**
Separated	5.79(4.38 – 7.64)	**1.80(1.13 – 2.85)**	10.77(6.94 – 16.73)	**4.36(2.39 – 7.97)**
Level of Education				
None	1.00	1.00	1.00	1.00
Primary	1.19(0.80 – 1.75)	1.25(0.70 – 2.21)	1.05(0.49 – 2.25)	1.57(0.59 – 4.14)
Secondary	0.95(0.65 – 1.40)	1.30(0.73 – 2.32)	0.84(0.39 – 1.78)	1.33(0.50 – 3.52)
Higher	0.67(0.40 – 1.10)	0.57(0.28 – 1.19)	0.68(0.30 – 1.55)	0.66(0.23 – 1.87)
Region				
Manicaland	1.00	1.00	1.00	1.00
MC	0.87(0.68 – 1.12)	0.85(0.60 – 1.20)	1.30(0.93 – 1.83)	1.32(0.87 – 2.00)
ME	0.97(0.76 – 1.25)	0.88(0.61 – 1.26)	1.29(0.91 – 1.82)	1.00(0.64 – 1.56)
MW	1.03(0.80 – 1.31)	0.95(0.67 – 1.33)	1.24(0.89 – 1.74)	1.12(0.74 – 1.71)
MN	1.17(0.91 – 1.51)	1.14(0.80 – 1.64)	1.90(1.34 – 2.69)	1.51(0.96 – 2.37)
MS	1.37(1.07 – 1.74)	1.07(0.75 – 1.52)	2.00(1.42 – 2.76)	**2.32(1.49 – 3.62)**
Midlands	0.98(0.76 – 1.24)	1.13(0.80 – 1.59)	1.30(0.93 – 1.81)	1.06(0.70 – 1.62)
Masvingo	0.88(0.68 – 1.14)	1.05(0.73 – 1.51)	1.16(0.78 – 1.71)	1.07(0.66 – 1.73)

Harare	0.91(0.72 – 1.16)	**0.63(0.43 – 0.92)**	0.95(0.66 – 1.36)	0.67(0.41 – 1.09)
Bulawayo	1.20(0.92 – 1.56)	**0.65(0.43 – 0.99)**	1.67(1.12 – 2.49)	1.37(0.79 – 2.37)
Sex of house head				
Male	1.00	1.00	1.00	1.00
Female	1.58(1.41 – 1.77)	1.17(1.00 – 1.38)	0.73 (0.61 – 0.87)	1.05(0.83 – 1.32)
Condom used at last sex				
No	1.00	1.00	1.00	1.00
Yes	3.86(3.28 – 4.55)	**2.83(2.30 – 3.48)**	**1.40(1.17 – 1.68)**	**2.62(2.05 – 3.34)**
Total lifetime partners				
1	1.00	1.00	1.00	1.00
2-4	3.86(3.39 – 4.39)	**3.10(2.61 – 3.68)**	3.51(2.43 – 5.07)	**2.31(1.55 – 3.43)**
5-9	6.35(4.65 – 8.67)	**3.74(2.52 – 5.53)**	6.31(4.32 – 9.20)	**3.15(2.08 – 4.77)**
10+	4.99(3.27 – 7.60)	**3.18(1.81 – 5.59)**	8.99(6.14 – 13.16)	**4.19(2.75 – 6.38)**
Had any STI in the last 12 months				
No	1.00	1.00	1.00	1.00
Yes	3.87(2.96 – 5.07)	**1.85(1.24 – 2.75)**	3.27(2.35 – 4.56)	1.16(0.68 – 1.97)
Had genital ulcer/sore in the last 12 months				
No	1.00	1.00	1.00	1.00
Yes	3.71(2.93 – 4.71)	**1.43(1.02 – 2.02)**	3.27(2.35 – 4.56)	**1.88(1.18 – 2.99)**
Had genital discharge in the last 12 months				
No	1.00	1.00	1.00	1.00
Yes	2.66(2.15 – 3.29)	**1.38(1.01 – 1.90)**	2.26(1.51 – 3.37)	1.25(0.70 – 2.23)
Age of most recent partner				
<19	1.00	1.00	1.00	1.00
20-24	1.88(0.44 – 8.04)	2.82(0.36 – 22.43)	3.18(2.23 – 4.53)	**1.62(1.05 – 2.50)**
25-29	4.25(1.03 – 17.62)	4.67(0.59 – 37.13)	4.06(2.84 – 5.79)	1.39(0.85 – 2.28)
30-34	7.16(1.73 – 29.61)	6.19(0.77 – 49.55)	5.69(3.95 – 8.18)	1.52(0.88 – 2.63)
35+	8.25(2.01 – 33.96)	6.50(0.81 – 52.20)	6.87(4.83 – 9.77)	1.64(0.91 – 2.96)
Intergenerational sex				
No	1.00	1.00	1.00	1.00
Yes	2.12(1.22 – 3.69)	0.98(0.51 – 1.87)	1.84(1.54 – 2.19)	1.10(0.86 – 1.40)
Cluster level				
variance(sd)		0.06(0.24)		0.06(0.24)

MC:, mashonaland central, ME: mashonaland east, MW: mashonaland west, MS: matabelend south, MN: Matabeleland north; 95%CI: 95% confidence interval; UOR: unadjusted odds ratio, AOR:

adjusted odds ratio; All significant variables with p-value<0.05 are printed in bold. Females:-AIC: 4400; BIC: 4663. Males:- AIC: 2972 BIC: 3222

3.6 Spatial autocorrelation

In this secondary analysis, global and local measures of spatial autocorrelation were employed in order to obtain a quantitative assessment of the extent to which clusters are correlated in space. Both measures of global spatial association provided overwhelming evidence for significant clustering of HIV infection in the general population. The results reveals a Global Moran's I statistic of 0.187 with a highly significant p-value (<0.001). The corresponding expectation was -0.003, variance of 0.001, and a standard deviation of 5.756. Similarly, a statistic value of 0.764 with a highly significant p-value (<0.001) was obtained for Geary's C global measure. The corresponding expectation was 1.000, variance of 0.001 and a standard deviation of 6.503.

Out of the 393 geo-referenced community clusters (without missing geographic coordinates), a total of twenty-six clusters were found to have significant spatial clustering of similar values around them at 5% level. For I_i, positive values indicate spatial clustering of similar values, and negative values clustering of dissimilar values. Thus, spatial clustering pattern of similar values was observed more in this study area following the Bonferroni correction for multiple testing. This implies that cluster locations with high HIV prevalence tend to cluster together in geographical space. This was the case within clusters such as 11, 20, 60, 63, 285 located in Matabeleland south and Bulawayo provinces. Similarly, locations with low HIV prevalence equally tend to significantly cluster in space as seen in clusters such as 80, 92, 133, 153, 121, 265, 279, 282,351, 386 , 389 all located in Manicaland, Mashonaland east and Harare provinces. The map of spatial autocorrelation of clusters was presented by means of a Moran's scatter plot shown in fig 3.1.

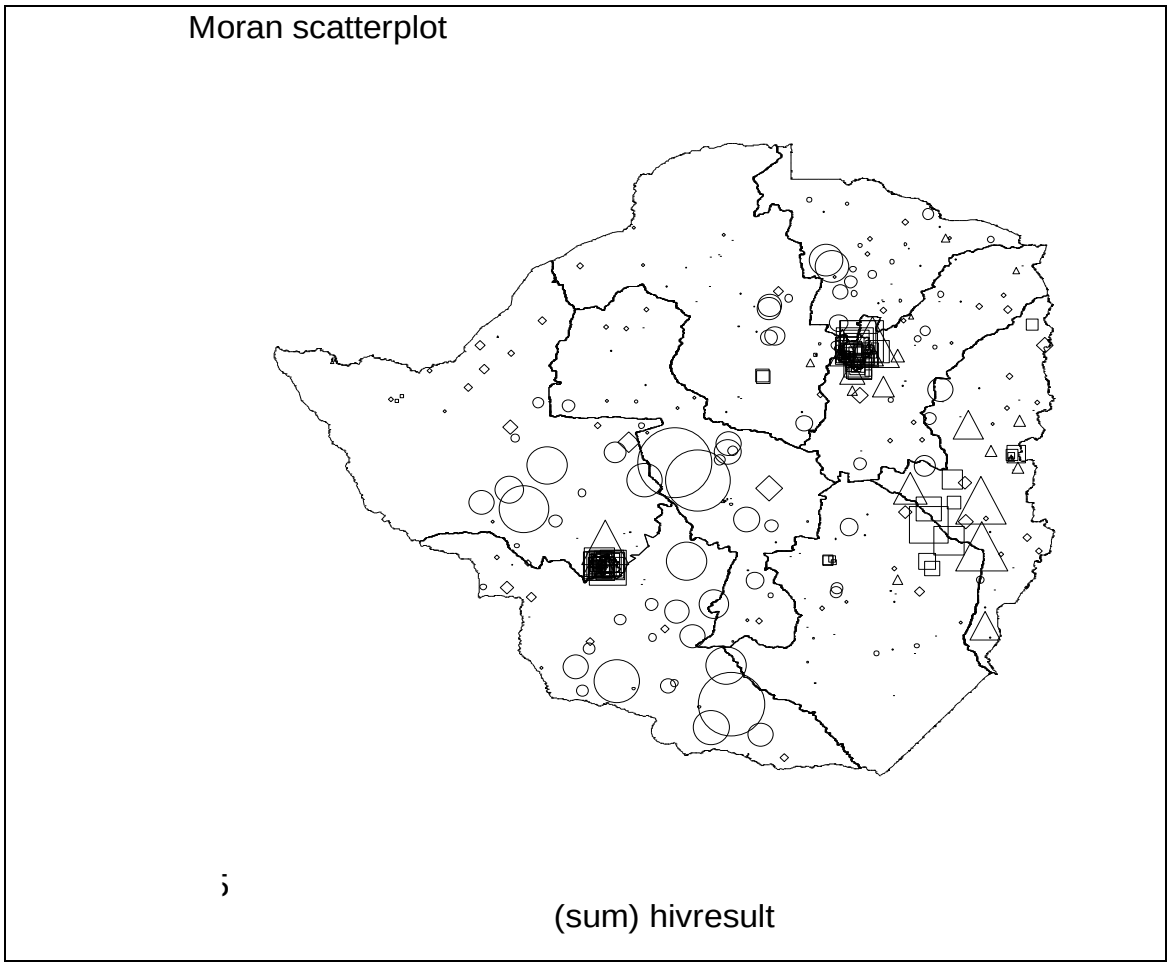


Figure 3.1. Map of Moran scatterplot values for prevalence of HIV infection.

In this map, locations belonging to quadrant 1 of the Moran scatterplot (high-high spatial association) are represented by a circle, locations belonging to quadrant 2 (low-low spatial association) are represented by a square, locations belonging to quadrant 3 (high-low spatial association) are represented by a triangle, and locations belonging to quadrant 4 (low-high spatial association) are represented by a diamond; symbol size is proportional to the corresponding Ii z-value.

3.7 Identification of HIV Hotspots using scan techniques

As the next step in this secondary analysis, areas with excess numbers of HIV cases (hotspots) were identified using two scan statistical techniques; Besag and Newell test, and Kulldorff spatial scan test. Secondary clusters that independently produced significant p-values at 5% level were also recorded as hotspots.

Three significant HIV prevalence hotspots (a total of 84 significant high risk cluster locations) were identified by Kulldorff Satscan procedure using a Poisson count model as

shown in fig 3.2 below alongside the Besag and Newell clustering result as shown in fig 3.3 below. Most likely cluster (comprising of 30 cluster locations) were found predominantly in Matabeleland north (18) and Midlands (12) between coordinates 18.94S, 28.66E (p-value: <0.001). Other areas with excess HIV prevalence were found in Matabeleland south and Mashonaland west (table 3.7). Result of separate scans (Besag and Kulldorff) each for male and female populations showed evidence of very little difference in clustering across the study area. A comparison of both scan techniques shows an overall evidence of agreement in identifying significant clusters (fig 3.2 and fig 3.3) in spite the use of separate scan parameter values.

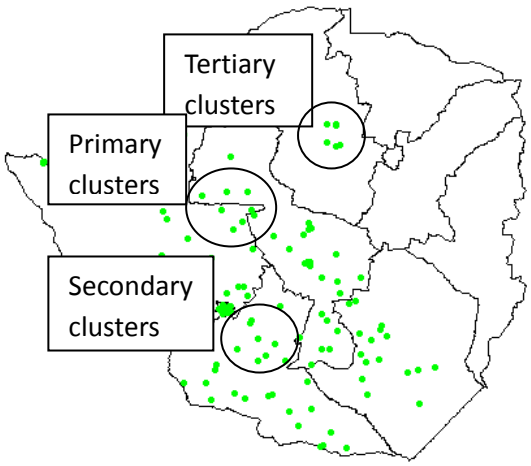


Figure 3.2.Plot of kulldorff spatial scan procedure (Unadjusted for underlying factors) for significant high risk clusters in the general population

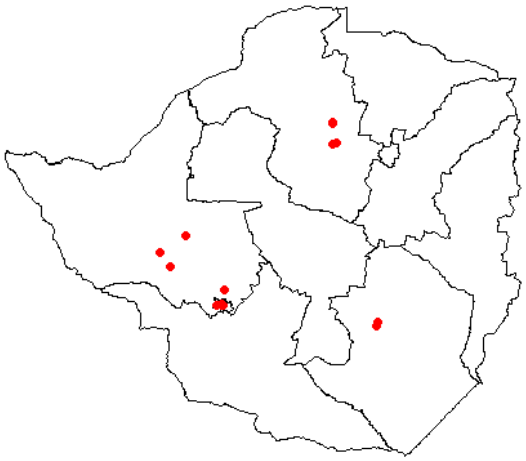


Figure 3.3. A plot of Besag and Newell's scan statistics (unadjusted for underlying factors) for significant high risk clusters in the general population.

Table 3.7. Significant clusters of high HIV prevalence using the Kulldorf SatScan procedure

Likelihood	Location ID	Coordinates (radius)	Observed cases	Expected cases	RR (LR)	p-value
Primary clusters	**30** locations: **18** in Matabeleland north; **12** in Midlands	18.94 S, 28.66E (119.94km)	252	163.30	1.61 (22.58)	<0.001
Secondary clusters	**26** locations: **19** in Matabeleland south; **7** in Midlands	20.92 S, 29.44E (111.62km)	214	141.53	1.57 (17.30)	<0.001
Tertiary clusters	**28** locations: **16** in Mashonaland west; **8** in Mashonaland central; **4** in Harare	17.28 S, 30.40E (80.63km)	221	152.41	1.50 (14.69)	<0.001

RR: relative risk; LR: likelihood ratio
Primaryclusters:202,295,384,128,2,288,210,166,274,6,76,61,316,304,402,131,312,38,291,339,66,142,178,159,397,186,83,348,77,173 Secondary clusters: 10,297,87,325,209,119,399,195,8,113,180,182,11,284,7,54,396,63,21,308,400,146,203,285,97,320
Tertiary clusters: 223,194,328,57,85,98,378,160,301,105,174,374,294,267,227,43,207,290,238,236,120,366,246,282, 327,75,158,264

3.8 Interpolation of HIV prevalence

In the general population, predicted values of HIV prevalence range from 2% to 12% (fig 3.3). In the semi-variogram model, a sill of 12, with a corresponding range 0.19 degree and a nugget of 5 were obtained. This sill corresponds to the semi-variance at which the spatial autocorrelation of observations levels off or become constant for larger distances beyond a certain range. In the context of this study, the distance range at which this state of semi-variance equilibrium and leveling off was attained is 0.19 degrees.

Thus, observations with a separation distance greater than this range have a lesser and a progressive decrease in spatial autocorrelation. This implies a corresponding rise in semi-variance until a distance of 0.19km beyond which autocorrelation is zero. The estimated nugget effect for this model was 5, a measurement error which estimates the Gaussian independent spatial residual. Interpolation estimates for males and females also showed little

evidence of geographic variation in the distribution of HIV infection with a maximum of 6 cases within a cluster in females and maximum of 5 cases in males.

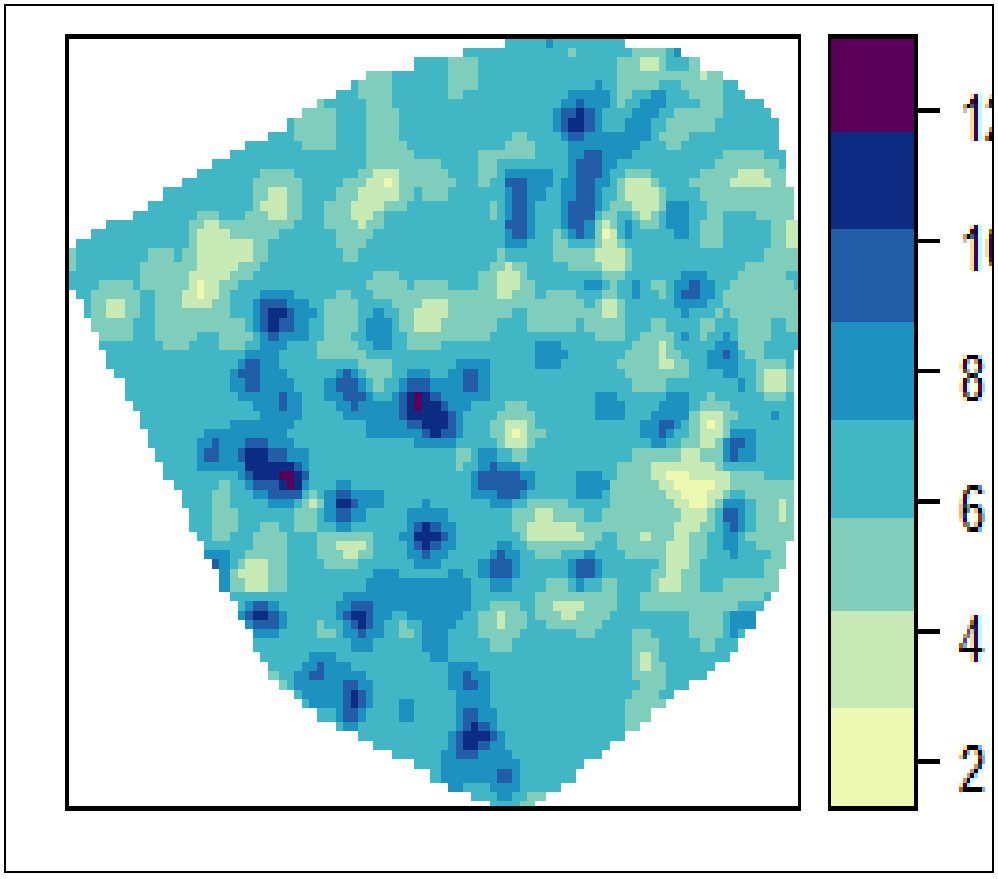


Figure 3.4. Interpolated surfaces of HIV prevalence in the general study population

3.9 Spatial regression analysis

Result shows that Harare province (green region) has the lowest risk of HIV infection with a posterior mean of -0.11 while Bulawayo and Matabeleland south (red regions) recorded the highest prevalence risk with a posterior mean of 0.14 and 0.16 respectively. However, these were not significant at 5% level as the credible regions include 0. A second spatially structured model (model 4) was fitted in which, significant underlying determinants were controlled for in addition to the non-spatial and spatial random effects. It was found that an adjustment for underlying determinants considerably increase risk in Matabeleland south by more than 50% (posterior mean=0.33) compared to model 2.

Conversely a corresponding further risk reduction (by more than 60%) was seen in Harare and Bulawayo as shown in fig 3.5 below. Also, these estimates did not attain a statistical significance at 5% level. In a third spatially structured model (model 6), adjustment for proximate determinants only was carried out as a fixed effect components of the model. Compared to model 4, this model shows a considerable reduction in the posterior mean estimate of HIV infection in Matabeleland south by about 80% (posterior mean=0.07) and a corresponding reduction of about 40% (posterior mean=0.07) (fig 3.6). Finally, adjusting for proximate determinants in a fourth spatially structured model (model 4), the highest posterior mean estimate was obtained in Matabeleland south followed by Matabeleland north (fig 3.7).

Although estimates did not attain statistical significance, evidence suggest regions of high HIV risk are located in the southwestern part of the country and regions of low HIV risk are clustered in the north eastern part of the country. All four structured models reveal a striking pattern of increasing HIV risk from the north east to the south western region of the country.

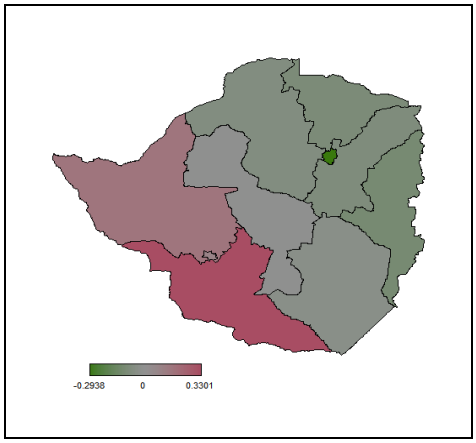


Figure 3.5. Spatially structured residual provincial effect on HIV prevalence across the ten provinces in Zimbabwe (Adjusted for underlying risk factors, non-spatial (household, cluster, provincial) and spatial (provincial level) random effects). Red = high risk; grey = moderate risk; green = low risk.

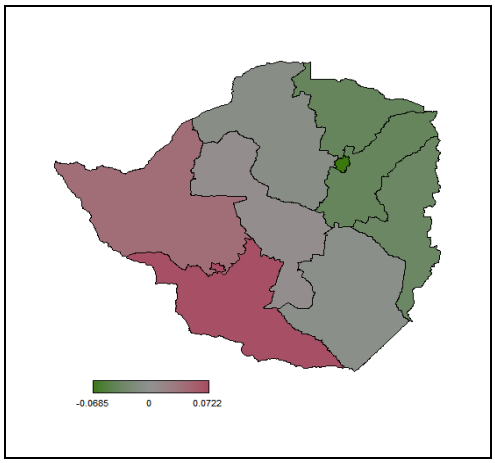


Figure 3.6. Spatially structured residual provincial effects on HIV prevalence distribution across the ten provinces in Zimbabwe (Adjusted for proximate risk factors, non-spatial (household, cluster, provincial) and spatial (provincial level) random effects)

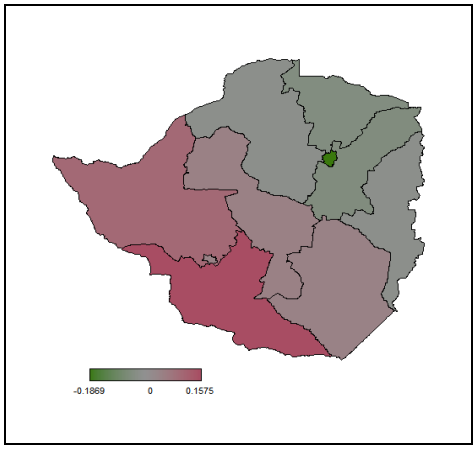


Figure 3.7. Spatially structured residual provincial effect on HIV prevalence distribution across the ten provinces in Zimbabwe (Adjusted for underlying and proximate risk factors, non-spatial (household, cluster, provincial) and spatial (provincial level) random effects). Red = high risk; grey = moderate risk; green = low risk.

Assessment of goodness of fit

As a final analytic procedure, assessment of models goodness of fit was also carried out using the deviance information criteria (DIC) (table 3.8). Model 4(unstructured) emerged as the most preferred model with a DIC of 7080.76. The structured counterpart has a DIC of 7081.39. Evidence therefore suggests the need to control for proximate determinants and cluster random effect in order to significantly improve the fit of the model.

This study therefore finds evidence of significant heterogeneity in HIV patterns and prevalence distribution across the study area. This study found significant evidence that socio-demographic and behavioural determinants exist with regard to HIV infection in Zimbabwe. In addition, significant difference in the spatial distribution of HIV was also found after adjusting for age and gender. However, no statistically significant difference was found across the ten regions after adjusting for the effects of underlying, proximate determinants and random effects.

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Table 3.8. Comparison of model fit using the Deviance information criteria

Model	DIC
M1	11384.27
M2	11385.22
M3	9934.68
M4	9934.10
M5	7271.24
M6	7270.70
M7	7080.76
M8	7081.39

M1(unstr): model 1 adjusted for unstructured (household, cluster, provincial) random effects; M1(str): model 1 adjusted for unstructured (household, cluster, provincial)& spatially structured(provincial) random effects;M2(unstr): model 2 adjusted for underlying determinants & unstructured random effects;M2(str): model 2 adjusted for underlying determinants, unstructured & spatially structured random effects;M3 (unstr): model 3 adjusted for proximate determinants & unstructured random effects;M3(str): model 3 adjusted for proximate determinants, unstructured & spatially structured random effects;M4(unstr): model 4 adjusted for underlying, proximate, unstructured random effects; M4(str): model 4 adjusted for underlying, proximate, unstructured and spatially structured random effects 95%CI: 95% credible region; DIC: deviance information criteria

3.10 Bivariate analysis of significant difference between hotspots and coldspots

As a final analytic procedure, bivariate logistic regression models were fitted to assess any difference between hotspots (high risk clusters) and coldspots with respect to underlying and proximate risk factors. Among the underlying factors, hotspots and coldspots differed significantly with respect to wealth index, occupation and religion on the average. Persons living within hotspots were on average wealthier than populations found within coldspots (p-value; 0.003).

Populations within hotspots were on average wealthier than populations found in coldspots. Similarly, populations found within hotspots were on the average more likely to engage in agriculture and less likely to be professionals compared to persons found within coldspots (p-value; 0.023). Populations found within coldspots are on the average more likely to be Christians than those found within hotspots (p-value: <0.001). Similarly, persons who had multiple partners (>1) were on the average more likely to reside within hotspots than those found within coldspots as shown in table 3.9 below. Populations within hotspots are also on average more likely to have STI or genital ulcer/sore in the last 12 months than populations found in coldspots. However, no evidence of association was found with respect to age, gender, marital status (results not shown), condom use at last sex and intergenerational sex (table 3.9).

Table 3.9. Odds ratio for bivariate test for significant difference between hotspots and coldspots with respect to underlying and proximate

Variable	OR (95%CI)	p-value
Religion		
None	1.00	-
Christian	**0.64 (0.53 – 0.78)**	**<0.001**
Muslim	0.85 (0.38 – 1.93)	0.699
Traditional	0.90 (0.62 – 1.31)	0.587
Occupation		
Not working	1.00	-
Professional	0.67 (0.47 – 0.95)	0.023
non-professional	0.73 (0.60 – 0.89)	0.002
Agriculture	1.13 (0.82 – 1.55)	0.452
Wealth index		
Poorest	1.00	-
Poorer	0.83 (0.62 – 1.10)	0.194
Middle	0.58 (0.41 – 0.83)	0.003
Richer	**0.45 (0.29 – 0.70)**	**<0.001**
Richest	**1.30(0.16 – 0.54)**	**<0.001**
Condom use at last sex		
No	1.00	-
Yes	1.09(0.95 – 1.26)	0.233
Total lifetime partners		
1	1.00	-
2-4	**1.49 (1.29 – 1.72)**	**<0.001**
5-9	**1.74 (1.43 – 2.12)**	**<0.001**
10+	**1.51 (1.25 – 1.81)**	**<0.001**
Had any STI in last 12 months		
No	1.00	-
Yes	1.34 (1.07 – 1.67)	0.011
Had genital ulcer/sore in last 12 months		
No	1.00	-
Yes	1.41 (1.13 – 1.76)	0.002
Intergenerational sex		
No	1.00	-
Yes	1.03 (0.93 – 1.15)	0.521

OR: odds ratio, 95%CI: 95% Confidence Interval

CHAPTER 4 : DISCUSSION

4.1 Prevalence of HIV infection

The overall HIV prevalence of 15.4% (95%CI: 14.8–16.0) shows a significant moderate reduction from that obtained 5 years earlier [18.1% (95%CI: 17.4 – 18.8). Hence, a significant percentage point reduction of 2.7% was recorded between the two DHS surveys. A reduction of about 2% was obtained in the male population (14% to 12.2%) while a slightly higher reduction of 3.4% was obtained in the female population (21.1% to 17.7%).

The reduction in rural and urban population was similar – about 3%. Sub-regional variation in prevalence estimate exists. [65] At the end of 2011, other countries like Kenya, South Africa and Rwanda recorded a national HIV prevalence of 6.2%, 16.6% and 3% respectively.[66-68] This underscores the need to develop policy and program response locally appropriate to the nature of the epidemic in different contextual settings across sub-Saharan regions.

4.2 Independent determinants of HIV infection

A number of independent underlying risk factors were associated with HIV infection in this population. Identifying underlying determinants is important for knowing which sub-population need interventions. Most important among these are age group, marital status and region which were independent predictors of higher prevalence in both male and female populations. Zimbabwean men and women who are widows, living with partner, divorced/separated constitute the most at risk population with a higher likelihood of HIV infection.

Secondary to marital status is the effect of age group on HIV outcome especially in the male population. Consequently, widows between the age 30 and 34 years, and men 35 years or older who have lost their partner to HIV or other causes (widowers) have a considerable elevation in the risk of HIV infection in the general population. HIV therefore tends to peak

at a younger (middle) age group in women and a later (older) age group in men. These findings are found to be consistent with other studies in Zimbabwe. For instance, Lewis and others found that age group and marital status remain significant predictors of HIV infection in rural Zimbabwe.[19]

On the other hand, likelihood of widowhood or partner separation may increase due to death of a partner from AIDS as was the case among Rakai women in Uganda[69] and rural women in Malawi.[70] In this case, widowhood is a direct causal outcome or antecedent of HIV infection and gradual progression to death.

This study also finds evidence of a significant disparity in HIV infection between rural and urban population. This may partly be due to the fact that recent interventions in Zimbabwe seem to be more effective in rural settings than urban locations.[48] In addition, the more developed social-infrastructures in cities encourage rural-urban migration and commercial sex-work in urban settings thereby increasing the risk of infection.

Furthermore, the high mobility network and dense population dynamics of urban systems also increases the rate of contact between susceptible and infected persons thereby increasing the likelihood of HIV transmission for every effective sexual contact.[20-22] Little statistical evidence was also found in this study to establish the effect of education on HIV outcome. However, while lower education seems to be a risk factor, higher education seems to play an important role in reducing HIV spread in Zimbabwe.

4.3 Independent factors of HIV infection

An important aspect of the proximate determinant framework is the need to determine the proximate effects of behavioral factors on HIV outcome.[12] While identifying underlying determinants is important for knowing which sub-population need interventions, identifying proximate determinants of HIV is important for knowing which interventions are needed.

In this present study, a number of proximate risk factors were identified in both men and women population. They include: condom use during last sex, total number of lifetime partners, history of STIs, genital sore/ulcer and genital discharge in the last 12months. Almost three times increase in odds was observed in both men and women who used condom during last sex with most recent partner. This result therefore affirms the long documented observation of a paradoxical relationship between condom use and HIV outcome in a generalized epidemic setting like Zimbabwe. [71-73]

This is partly because persons in relatively stable sexual relationships are less likely to be consistent in their condom use. For instance, evidence from previous DHS in Zimbabwe found that sexually active single men were eight times more likely to use condom than married men.[74] The same study identified marital status of individuals (single/in union) determines to a significant extent the likelihood of condom use .However, it is not the use of condom that cause a reversal in the HIV incidence trend, but the correct and consistent use of it.[74] More so, it is difficult to quantitatively assess the effect of condom use on transmission efficiency in the general population without information on condom use in every sexual encounter between a susceptible and infected person.[12] Regrettably, this assessment feat is therefore difficult to achieve in a cross-sectional type of study of this nature which only relies on self-reporting to obtain information on condom use at a single point in time.

This study also showed evidence of a strong association and linear trend between HIV infection and total number of lifetime partners, in Zimbabwean male population. Number of lifetime partners represents lifetime risk and HIV sero-prevalence captures the risk of already having the disease. This factor single-handedly accounts for the largest share of proximate burden driving HIV infection in Zimbabwe both before and after adjustment for underlying and other proximate determinants. It provides the major link between HIV infection and underlying factor such as widowhood status.

For example, the state of widowhood, which was a significant underlying predictor of HIV in this population, can also make a man or woman acquire HIV infection. The reasoning behind this causal relation is that men and women who have lost their spouse are made vulnerable to HIV acquisition due to a break-down in their psycho-social security. Consequently, they are exposed to multiple sexual partners, thus, increasing the probability of infection. An alternative explanation for high rates of HIV infection among widows is that their partner has died but not before infecting them with HIV.

These findings are consistent with other studies. For instance, Lewis and others also found number of lifetime partners to be the strongest and the only significant predictor of HIV sero-status in Zimbabwe.[36] Similarly, a systematic review of sixty-eight epidemiological studies in sub-Saharan Africa also showed evidence of a strong association between number of sex partners and HIV prevalence.[75]

One study found that societal influence of early sexual initiation and cultural practice of having many wives largely, alongside low education status of women account for multiple sexual partnerships in sub-Saharan Africa. [76] As a result, young sexually active women who suffer from sexual dissatisfaction, infidelity, neglect or less attention from spouse may end up finding alternative ways of sexual compensation or consolation in the society.

This same sexual dynamics observed at the interpersonal level accounts for the observed younger women –older men or younger men-older women pattern of sexual unions at the community level (a well-researched phenomenon in Africa known as intergenerational sex). The entrenched cultural perception of male's dominance and freedom to choose who to have sex with, when to use condom and control of household economic resources have been found to play critical contextual roles in modifying HIV outcome due to multiple lifetime partners. [76]

4.4 How underlying determinant work through proximate determinants

The observed associations between underlying factors and HIV infection was expected to disappear or reduce on adjusting for proximate factors. This was observed in this study as the effects of some underlying factors (especially age groups and region in women) reduced after adjusting for independent proximate determinants such as total lifetime partners and condom use at last in both gender populations. However, the effects of some underlying factors still remain significantly high (age groups in men, residence, and marital status in both men and women populations).

4.5 Identification and mapping of HIV hotspots

Results of global and local spatial clustering of HIV showed that high risk community locations were found to cluster in the northwest and south western regions of the country (in Bulawayo, Matebeleland north and Matebeleland south provinces). Areas of high prevalence are also identical in both men and women. This findings contrast that of a similar study in Congo (DRC) which found that areas of high prevalence were not identical for men and women.[42] On the other extreme, low-risk communities largely cluster in the north-eastern regions of the country (Harare, Manicaland, Mashonaland east, Masvingo). In between these two extremes are high-risk communities surrounded by low-risk ones such as Mashonaland West, Mashonaland Central, and Midlands.

Other studies in sub-Saharan Africa have demonstrated the existence of similar clustering of HIV cases. For instance, Tanser and colleagues provides clear evidence of the existence of local clustering of HIV infection close to national roads in rural KwaZulu-Natal, South Africa using the SatScan procedure.[66] While this study identifies relative risk ranging from 1.50 - 1.61), Tanser and colleagues arrived at an estimated range of 1.34 – 1.62). More so, while this current study utilized a Poisson model, they employed a Bernoulli model to identify high and low risk clusters. [77]

A similar study carried out by Handan and Gita in two small communities in South Africa identified three hotspots with excessively high HIV prevalence rates.[78] This study also set a user-defined maximum radius used by SatScan to its default value of 50% and a Poisson model was utilized. More so, while the present study assessed clustering at community level, clustering was implemented at individual level.[78] In addition, the potential role of antiretroviral treatment (ART) in contribute to increase in HIV prevalence in sub-Saharan African countries has been examined. A study strongly suggested he possible link between high-quality health care (with regard to ART options) and high HIV prevalence. [79] A model result also found that the gains of antiretroviral treatments are substantially reduced or eliminated altogether in a high risky behavior setting such as is the case in Zimbabwe. [80]

4.6 Interpolation of HIV prevalence

Results from this analysis supports results from previous findings, as higher estimates were obtained in the south western regions of the country in both female and male populations. A similar study conducted in Congo (DRC) found significant evidence of spatial heterogeneity as high as 30.5% compared to the overall national prevalence of less than 2%. [42]

4.7 Spatial regression analysis

Geoadditive Bayesian results also provided little evidence of geographic variation in the distribution of HIV prevalence after adjusting for significant underlying and proximate risk factors. The highest posterior mean of 0.16 and lowest mean of -0.19 recorded in Matabeleland south and Harare regions respectively adjusting for underlying and proximate determinants alongside non-spatial and spatial random effects. Identification of HIV hotspots using different geostatistical approaches provides the opportunity to model significant predictors of higher prevalence within these spatial hotspots and assess the impact of geographical locations on HIV spread. Hence, this study shows that significant heterogeneity does not exist with regard to HIV distribution in Zimbabwe in 2011 adjusting for significant

risk factors. This may therefore mean that risk factors explained most of the observed heterogeneity.

A similar study conducted in Tanzania also found that spatial distribution of risk factors do not explain regional variation in HIV seroprevalence.[39] On the other hand, study in Kenya found large sub regional variations in HIV prevalence where areas of high concentration of HIV-infected people have a disproportionately low density of HIV-related services. [81] Another study in also identified a negative association between distance to city and HIV infection especially for women in Congo (DRC) [42] although this was not examined in the present study.

4.8 Risk factor profile assessment of HIV hotspots

Bivariate analysis also provides evidence of significant differences between hotspots and coldspots on the average with respect to underlying and proximate covariates. It was found that a higher proportion of populations within hotspots were on the average, never in union (most likely the younger and middle-age population) compared to those found in coldspots. Little evidence also suggests that on the average populations found in hotspots had lower education, more multiple partners and STIs compared to those found in coldspots.

Result also suggests 25% higher odds that female-headed households are more likely to be found within hotspots relative to male-headed households. Significant bivariate evidence was also found that all occupation categories (professional and non-professional alike) have lower odds of living within hotspots relative to persons who are not working. This same pattern was observed across wealth index categories relative to persons in the poorest quintile. Bivariate analysis of proximate factors also shows that person who reported early sexual debut (below the age of 17 years) also have increased odds of living within hotspots.

Knowledge of STI and AIDS are positively associated with living within hotspots while persons who used condoms are more likely to live within hotspots. Increasing odds of living within hotspots with increasing age at first cohabitation was also observed. It thus seems that persons who delay cohabitation (especially marital union) have an increase likelihood of engaging in sexual behavioral practices that male them vulnerable to either transmitting (if infected) or contracting (if susceptible) HIV infection in the population. On the other hand, those who were cohabiting (regardless of the duration) have lower odds of living within hotspots. This further suggests the protective effect of living or cohabiting with a stable partnership.

4.9 Limitations of this study

1. Cross-sectional nature of the study makes it difficult to determine causal nature of factors and the direction of causality between HIV status and some of the explanatory variables (difficulty in ascertaining temporality). This is due to the fact that causality may run in both directions, as may be the case in the causal relation between HIV status and some underlying determinants. A similar reverse causation may occur between other STIs and HIV infection. Moreover, since it is difficult to determine when exactly infection occur across exposure factor categories, relationships between HIV status and significant risk factor variables are interpreted as associations as opposed to causality.

2. Random displacement of DHS cluster locations might have led to under-estimation or over-estimation of spatial autocorrelation. However, since the displacement of the ZDHS 2010-2011 GPS data was limited to the district level, it implies that clusters did not cross district lines; hence the accuracy of estimates can be expected to fall within an acceptable range. Regardless of the source of GPS reading s for clusters, DHS also ensures all collected coordinates were evaluated for accuracy before they are displaced.

Similarly, analysis of spatial heterogeneity in this study was implemented at provincial level

only in order to ensure accurate posterior estimates. This is because Zimbabwe surveys are not designed to be representative below the regional level.

3. Problem of multiple reporting biases arising due to the fact that indicators measuring sexual behavior are self-reported; hence, the reliability of reporting may be difficult to gauge on effect estimates on outcome.

4. This secondary analysis is suffered from lack of variable relating to infectiousness (duration of infectivity). Therefore, its role in reducing transmission efficiency could not be determined. In this study therefore, the impact of ART on higher prevalence observed between different sub-groups within the population could not be examined as data was collected at only one point in time (consisting of both old and new cases). The study cross-sectional design utilized is therefore too weak to draw specific causal inference. Further studies must therefore take this into account in order to get a clearer picture of the contributing risk factors to HIV outcome in Zimbabwe.

5. This study also did not explore the possible contribution of antiretroviral treatments to high prevalence observed in hotspots. Hence, there is a need for further research considerations to establish this.

CHAPTER 5 : CONCLUSION AND RECOMMENDATIONS

5.1 Conclusion

This secondary analysis shows that the HIV proximate determinant framework provides a better analytical approach to investigate the dynamic interplay between contextual and proximate determinants of HIV infection. More so, robust statistical and geo-statistical approaches were used to estimate regional distribution of HIV infection in Zimbabwe and quantitatively assess geographic patterns and predictors of high rates of infection in hotspots. Overwhelming evidence of significant clustering of HIV infection was found in specific communities in Zimbabwe. Policy makers should therefore consider targeting interventions at these specific locations.

Results also demonstrate that underlying and proximate determinants exist with regard to HIV infection in Zimbabwe. Finally spatial scan procedures provided significant evidence of heterogeneity with regard to spatial distribution of HIV in Zimbabwe. However, little evidence exists when spatial dependence of clusters was accounted for in the structured regression model and therefore evidence did not attain statistical significance. Similar predictors of HIV infection were identified in the general population and hotspots of HIV infection in Zimbabwe.

5.2 Recommendations

The following recommendations are therefore put forward to mitigate the negative impact of HIV and extend the frontiers of previous gains

Firstly, focused behavioural interventions should be considered among widows and widowers in high-risk regions and consolidation of previous gains in low-risk regions such as Harare.

In addition, measures to encourage delay sexual debut especially among younger population should be put in place. Likewise, allocation of social grants should be considered for widows and the disadvantaged in the population.

Lastly, treatment services of STIs, genital ulcers/sore and genital discharge should be implemented in high-risk regions such as Matabeleland South especially among sexually active single men and women.

On a final note, the true impact of condom use on the Zimbabwe HIV/AIDS generalized epidemic is best examined in the context of marital status. This is simply because risk of infection differs between married and unmarried individuals. The author therefore recommends that further analysis should examine the independent of effect of condom use on HIV outcome in the context of marital status and sexual partnerships in the study population. Proximate determinants (behavioural and biological) of HIV infection should also be explored at group level.

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SCANNED APPENDICES ARE ATTACHED AT THE END OF THIS DOCUMENT IN THE FOLLOWING ORDER:

APPENDIX 1: ETHICS APPROVAL FOR THE ZIMBABWE DEMOGRAPHIC AND HEALTH SURVEY 2010/11

APPENDIX 2: ETHICS APPROVAL FROM WITS HUMAN RESEARCH ETHICS COMMITTEE

APPENDIX 3: DATA USE APPROVAL FROM ICF-INTERNATIONAL

Telephone: (263)-4-791792/791193
Telefax: (263) - 4 - 790715
E-mail: mrcz@mrczimshared.co.zw
Website: http://www.mrcz.org.zw

Medical Research Council of Zimbabwe
Josiah Tongogara / Mazoe Street
P. O. Box CY 573
Causeway
Harare

MRCZ APPROVAL LETTER

Ref: MRCZ/A/1563

Date: 24 June 2010

Dr. Portia Manangazira
Ministry of Health and Child Welfare
P.O Box A 355
Avondale
Harare

RE:- Anaemia and HIV Testing in the 2010 Demographic and Health Survey

Thank you for the Application for Approval to carry out Research Activity that you submitted for review to the Medical Research Council of Zimbabwe (MRCZ). Please be advised that the Medical Research Council of Zimbabwe has **reviewed** and **approved** your application to conduct the above titled study.

This approval is based on:-

(a) Study Protocol
(b) MRCZ Application Form
(c) English, Shona and Ndebele Consent and Assent Forms

APPROVAL NUMBER : MRCZ/A/1563
This number should be used on all correspondence, consent forms and documents as appropriate.

- **APPROVAL DATE : 24 June 2010**
- **EXPIRATION DATE : 23 June 2011**
- **TYPE OF MEETING : FULL BOARD**

After this date, this project may only continue upon renewal. For purposes of renewal, a progress report on a standard form obtainable from the MRCZ Offices should be submitted one month before the expiration date for continuing review.

SERIOUS ADVERSE EVENT REPORTING: *All serious problems having to do with subject safety must be reported to the Institutional Ethical Review Committee (IERC) as well as the MRCZ within 3 working days using standard forms obtainable from the MRCZ Offices.*

MODIFICATIONS: Prior MRCZ and IERC approval using standard forms obtainable from the MRCZ Offices is required before implementing any changes in the Protocol (including changes in the consent documents).

TERMINATION OF STUDY: On termination of a study, a report has to be submitted to the MRCZ using standard forms obtainable from the MRCZ Offices.

QUESTIONS: Please contact the MRCZ on Telephone No. (04) 791792, 791193 or by e-mail on mrcz@mrczimshared.co.zw.

Other

Please be reminded to send in copies of your research results for our records as well as for Health Research Database.

You're also encouraged to submit electronic copies of your publications in peer-reviewed journals that may emanate from this study.

Yours Faithfully

.....

MRCZ SECRETARIAT
FOR CHAIRPERSON
MEDICAL RESEARCH COUNCIL OF ZIMBABWE

MEDICAL RESEARCH COUNCIL OF ZIMBABWE
24 JUL 2010
APPROVED
P. O. BOX CY 573
CAUSEWAY, HARARE

UNIVERSITY OF THE WITWATERSRAND, JOHANNESBURG
Division of the Deputy Registrar (Research)

HUMAN RESEARCH ETHICS COMMITTEE (MEDICAL)
R14/49 Mr Atilola O Glory

CLEARANCE CERTIFICATE **M120861**

PROJECT Prevalence, Spatial Patterns and Factors Associatedwith HIV Infection in Zimbabwe 2011

INVESTIGATORS Mr Atilola O Glory.

DEPARTMENT School of Public Health

DATE CONSIDERED 31/08/2012

DECISION OF THE COMMITTEE* Approved unconditionally

Unless otherwise specified this ethical clearance is valid for 5 years and may be renewed upon application.

DATE 31/08/2012 **CHAIRPERSON** .....
(Professor PE Cleaton-Jones)

*Guidelines for written 'informed consent' attached where applicable
cc: Supervisor : Dr Benn Sartorius

DECLARATION OF INVESTIGATOR(S)
To be completed in duplicate and **ONE COPY** returned to the Secretary at Room 10004, 10th Floor, Senate House, University.
I/We fully understand the conditions under which I am/we are authorized to carry out the abovementioned research and I/we guarantee to ensure compliance with these conditions. Should any departure to be contemplated from the research procedure as approved I/we undertake to resubmit the protocol to the Committee. **I agree to a completion of a yearly progress report.**

PLEASE QUOTE THE PROTOCOL NUMBER IN ALL ENQUIRIES...

August 8, 2012

Glory Atilola
University of Witwatersrand
South Africa

Dear Glory:

You have been authorized to use the Zimbabwe DHS datasets for your research project titled:

"Prevalence, Spatial Patterns and Factors Associated with HIV Infection in Zimbabwe 2011"

The IRB-approved procedures for DHS public use datasets, do not in any way allow respondents, households, or sample communities to be identified. There are no names of individuals or household addresses in the data files. The geographic identifiers only go down to the regional level (where regions are typically very large geographical areas encompassing several states/provinces). Each enumeration area (Primary Sampling Unit) has a PSU number in the data file, but the PSU numbers do not have any labels to indicate their names or locations. The PSU is only identified as being in an urban area or a rural area. For data sets for which GIS coordinates are collected in the field, the coordinates are only for the enumeration area (EA) as a whole (not for individual households), and the measured coordinates are randomly displaced within a large geographic area so that even ICF International has no way of identifying a particular enumeration area.

The DHS datasets must not be passed on to other researchers without the written consent of DHS. You are requested to submit an electronic or hard copy of any reports/publications resulting from using the DHS data files to our office.

Please let me know if you need any additional information.

Sincerely,

Bridgette Wellington

Data Archive Administrator
MEASURE DHS