GeoJournal (2012) 77:505–523
DOI 10.1007/s10708-010-9358-6

HIV and AIDS in Africa: a geographic analysis at multiple spatial scales

Ezekiel Kalipeni · Leo C. Zulu

Published online: 23 April 2010

Abstract This study offers an alternative method rooted in GIS techniques and spatial analysis to estimate HIV/AIDS prevalence over space from an incomplete surveillance data set and explain the variation of those estimates. The results clearly show that the HIV/AIDS epidemic is complex and that it is interconnected with other geographic, historical, economic and cultural phenomena which help explain its spatial spread and variation. The regression models which were developed in this paper illustrated that variables which measure the historical context of colonialism such as resource exploitation and labor migration, gender, culture, contemporary global forces, poverty and disease burden have all contributed variously to the rapid spread of this disease both in space and time. The policy implication is that concentrating on behavior change or therapy alone may not turn the epidemic around. The attack needs to be multifaceted and interdisciplinary taking into consideration the context and the economic and social realities at multiple spatial scales.

E. Kalipeni (✉)
National Science Foundation, 4201 Wilson Boulevard, Arlington, VA 22230, USA
e-mail: ekalipen@nsf.gov

L. C. Zulu
Department of Geography, Michigan State University, East Lansing, MI 48824, USA
e-mail: zulu@msu.edu

Keywords HIV and AIDS · Geospatial analysis · HIV/AIDS drivers

Introduction

Now in its third decade, the HIV/AIDS pandemic in sub-Saharan Africa continues to spread despite some stabilization in a number of countries of the region. Though the region holds ~10% of the world's population, latest statistics from the Joint United Nations Program on HIV/AIDS show that Sub-Saharan Africa accounted for 67% (22.0 million people) of all people living with HIV, 72% of the 2.0 million HIV-related deaths globally, and 1.9 million of the 2.7 new infections in 2007 (UNAIDS 2008). Globally, the pandemic appears to stabilize, initially due to increases in AIDS deaths but recently also due to decline in new infections (UNAIDS 2004, 2008). Thus, between 2006 and 2007, global deaths continued to increase from 1.7 to 2 million while new infections declined from 3.0 to 2.7 million (UNAIDS 2008). While there is no paucity of studies on the complexity of AIDS in Africa over the past two decades, biomedical and epidemiological models of inquiry continue to dominate the research agenda. Biomedical studies have increased our understanding of the physiological mechanisms of HIV infection through viral load assessments (Quinn et al. 2000), chemotherapeutic preventions of vertical transmission (Connor et al. 1994), and the interaction of HIV infection with common sexually transmitted diseases and urinary tract infections (Diallo et al. 1997; Wasserheit 1992). Results of these studies have provided valuable insights on who might be at increased risk due to the presence of co-infections or synergistic infections, at what point those who are already HIV infected might be more at risk of infecting others, and how HIV transmission from mother to child can be mitigated.

Epidemiological studies have focused on sexual behavior patterns of individuals within designated risk categories, especially commercial sex workers, military personnel, and truck drivers (Bwayo 1994; Ferguson and Morris 2007; Kreiss et al. 1986; Ndinya-Achola et al. 1997; Pickering et al. 1997). Most of these investigators sought to explain high rates of HIV transmission by examining types of sexual exchanges that take place among these populations. Some have focused on how many partners commercial sex workers have, or when, where, and how often drivers along particular truck routes engage in commercial sex. Most significantly, such studies have focused on levels of condom use by commercial sex workers and other populations. Further, behavioral-based studies have generated useful insights on the association of high-risk patterns of sexual practice with high HIV rates within particular groups in specific regions of Africa (e.g., the case of Nairobi commercial sex workers who are upwards of 80% HIV seropositive). They have also informed prevention programs that rely upon educational outreach and condom availability, methods that emphasize changing individual behavior through greater awareness of risk and increased capacity for self-initiation of preventive measures.

Biomedical and epidemiological studies prove inadequate, however, on several levels. First, a primary focus on so-called risk groups erroneously targets occupational categories at the expense of social practice. This focus gives the simplistic impression that individuals within particular categories are at risk of HIV or are already infected while the rest are not. More importantly, these studies fail to ask the question of why individuals engage in the behaviors they do, thereby failing to focus on those social, political, cultural, and economic factors that generate conditions of vulnerability to HIV and limit individual choices concerning social and sexual practices. As the anthropologist Waterston suggests, dominant AIDS interventions focus on the individual as the locus of disease and prevention because they are least threatening to the status quo (Waterston 1997). Not only do educational outreach and condoms programs leave root causes of HIV untouched, but they also rest on the presumption that individuals can automatically change their behaviors with the benefit of awareness and condom possession.

It is in this light that we offer a different perspective in this paper to the study of HIV/AIDS in sub-Saharan Africa, i.e. a temporal, spatial and statistical analysis of the major drivers of HIV in sub-Saharan Africa. This is not to assert that biomedical and epidemiological researchers are unable to carry out spatio-temporal studies of HIV. Practitioners of medical sociology, medical ecology and spatial epidemiology have in recent years carried out spatio-temporal studies but not at the continental level (Berkman and Kawachi 2000).

This paper has two central objectives: (a) to spatially analyze the antenatal surveillance data set that has been collected annually since the mid-1980s on HIV infection rates at over 1,400 data points throughout the continent; (b) conduct an in-depth statistical analysis to determine the major drivers of HIV rates at both the micro level ('service areas' defined by Thiessen polygons generated with a Geographic Information Systems package (GIS), ArcGIS 8.3) and the country level. For the first objective, we use the powerful geographic tool of GIS to project across space and extract HIV estimates at the two scalar levels, and integrate them with other geospatial variables, such as population density and elevation. For the second objective, four groupings of explanatory variables are examined. These include: geographic variables that measure resource endowment and mobility; gender empowerment variables; variables that measure poverty, development, and disease burden; and variables that measure government attitudes and commitment in the fight against HIV/AIDS. The paper also discusses other drivers such as global forces and the role of culture in the proliferation or abatement of the epidemic.

Methodology

Theoretical framework

There is a growing number of social scientists in critical geography, anthropology, and other fields who diverge from the dominant paradigm of behavioral-based research by interrogating the institutional, ideological, and historical structures of inequity fostering the spread of HIV. Instead of focusing on patterns of sexual exchange per se, critical social scientists increasingly investigate the multiple and interrelated factors and processes that expose women or men to high-risk behaviors, thereby increasing their vulnerability to HIV. One of the primary factors increasing HIV transmission in southern Africa, for example, is a pattern of male out-migration driven by depressed national economies and existence of labor-intensive mining industries in South Africa (Brockerhoff and Biddlecom 1999; Decosas et al. 1995; Girdler-Brown 1998; Haour-Knipe and Rector 1996). The result is male workers away from their families for extended periods of time leaving women increasingly unable to fend for themselves (Campbell 1997; Chirwa 1998; Zuma et al. 2003). Prolonged wars have disrupted local economies, displaced populations, and rendered longstanding social practices untenable in some regions (Gisselquist 2004; Kalipeni and Oppong 1998; Lyons 2004). In most countries of sub-Saharan Africa, struggling economies and rampant poverty make it difficult for families to stay together in one place and often force individuals (especially women) to resort to risky sexual economies.

Stereotypical theories, such as the over sexualized African, have been reduced to myths. Eileen Stillwaggon (2003), for instance, demonstrates that spatial variation of social, economic, and other health determinants can explain HIV distribution and prevalence in sub-Saharan Africa. She cites factors including male circumcision, prevalence of other STDs, prevalence of schistosomiasis and other parasitic diseases, 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.

The foregoing brief literature review reveals several areas of inquiry that need to be addressed in order to enhance understanding of the complex macro level drivers of HIV. Our theoretical framework (see Fig. 1) identifies six such areas, some of which were recently suggested by Ian Yeboah (Yeboah 2007): (1) the historical context of colonialism, (2) gender relations, (3) poverty and disease burden, (4) global forces, (5) culture, and (6) government attitudes (see Fig. 1). This paper investigates whether a statistical relationship holds between the geographic distribution of HIV prevalence and the structural or non-behavioral dimensions of HIV transmission at national level. However, poor data quality and availability limit regression analysis to independent variables in areas 1–3 and area 5. We had no data on the global forces and the culture groupings, but we still discuss these factors in the paper. The following section discusses the analytical framework and methods, specific hypothesis, and directions of relationships between selected independent variables, and dependent variables.

Data and geospatial analysis

We employ a geographic approach based on GIS to (1) determine spatial–temporal patterns of HIV prevalence (1986–2003) and (2) generate HIV prevalence rates (dependent variable) and socio-environmental (independent) variables at micro, artificially generated "service areas" based on Thiessen polygons and macro national levels, and integrate them with other variables in regression models of potential determinants of HIV prevalence in Sub-Saharan Africa. The HIV dataset consisted of Africa-wide percent HIV/AIDS seropositive rates collected from 1,442 HIV sentinel and testing centers predominantly from pregnant women attending antenatal clinics, but varying in reporting centers between 49 points in 1986 and 500 in 2003. Centra Technology, Inc (http://www.centratechnology.com), geocoded (recorded the latitude and longitude locations of) the centers and provided the authors access to the data. The testing centers are mapped in Fig. 2.

Using the Spatial Analyst module in the GIS software ArcGIS 8.3, we used the Inverse Distance Weighting (IDW) spatial interpolation method to project the HIV data at the measured points (Fig. 2) to unmeasured locations for the years 1986, 1990, 1995, 1999 and 2003. We chose these years based on a 4–5 year interval and sample size. The continuous interpolated surfaces produced (Fig. 3) reveal the spatio-temporal patterns of HIV/AIDS. They allowed us to use standard GIS techniques to generate Thiessen polygons or artificial HIV-testing "service areas" (Fig. 4) and map algebra to extract mean HIV prevalence (%) by country and by the Thiessen polygons. Independent variables generated with standard GIS procedures at both scales were: (1) mean distance to roads and rail (meters) that we derived from aggregated national roads and rail maps obtained from the 1992 Digital Chart of the World,

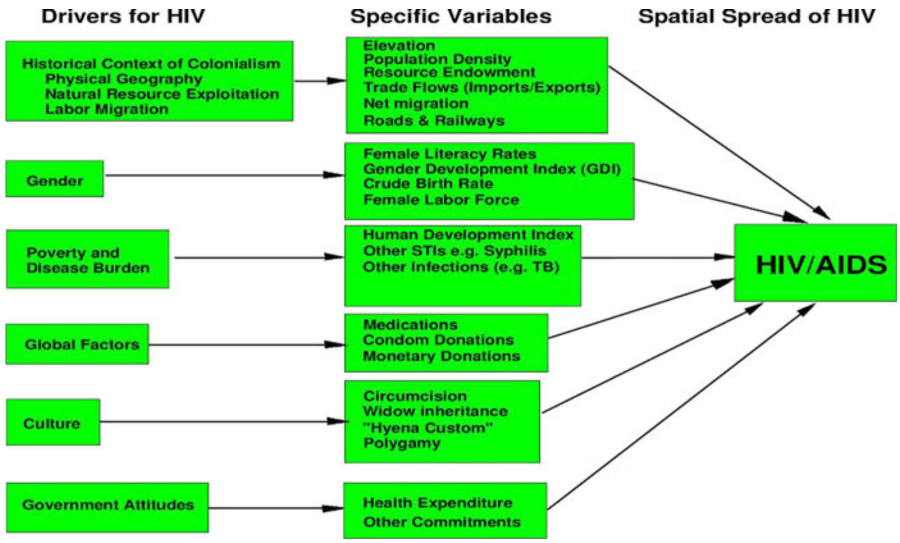

Fig. 1 Theoretical framework of the relationship between HIV/AIDS rates and major factors

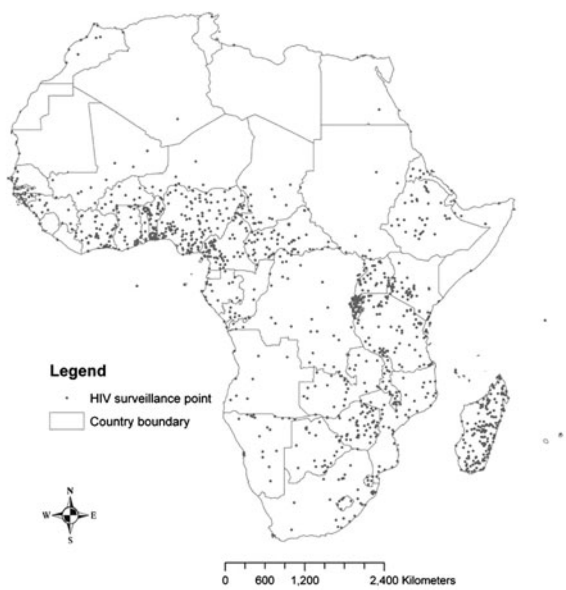

Fig. 2 HIV surveillance data points by country

(2) mean elevation (meters) derived from 30 Arc Second dataset (1 km) digital elevation model of the world, and (3) population density for 2003 from the LandScan dataset.[1] These GIS-generated variables were combined with social and economic ones in regression analysis.

A more detailed description and discussion of the choice of the IDW interpolation method and of interpolated averages instead of simple or weighted averages can be found in Kalipeni and Zulu (2008). That article also discusses concerns over data quality and representativeness (e.g. exclusion of population groups other than pregnant women, sample size and variability over space and time, and variable HIV testing techniques). While these weaknesses limit the use of such data and research findings to provide 'hard' numbers for certain policy prescriptions, the data were the most comprehensive, continent-wide dataset available, and they reveal main spatiotemporal trends and determinants of HIV levels in Sub-Saharan Africa.

Independent variables

Based on an extensive survey of the literature, we have developed the theoretical framework given in Fig. 1 concerning macro level, non-behavioral and behavioral drivers of HIV/AIDS in sub-Saharan Africa grouped into six categories listed earlier. We conducted regression analysis only for the 1990, 1995 and 1999 GIS-generated HIV dependent variables because 1986 had a small number of sampled points. While the six sets of drivers of HIV/AIDS prevalence in sub-Saharan Africa and elsewhere are discussed in various papers (Iliffe 2006), few studies have

[1] The Digital Chart of the World was obtained from Penn State University Libraries at http://www.maproom.psu.edu/dcw/; the population data from the LandScan™ Global Population Database, Oak Ridge National Laboratory, Oak Ridge, TN, available at http://www.ornl.gov/landscan/; and the elevation data from a Miombo Network CD-Rom.

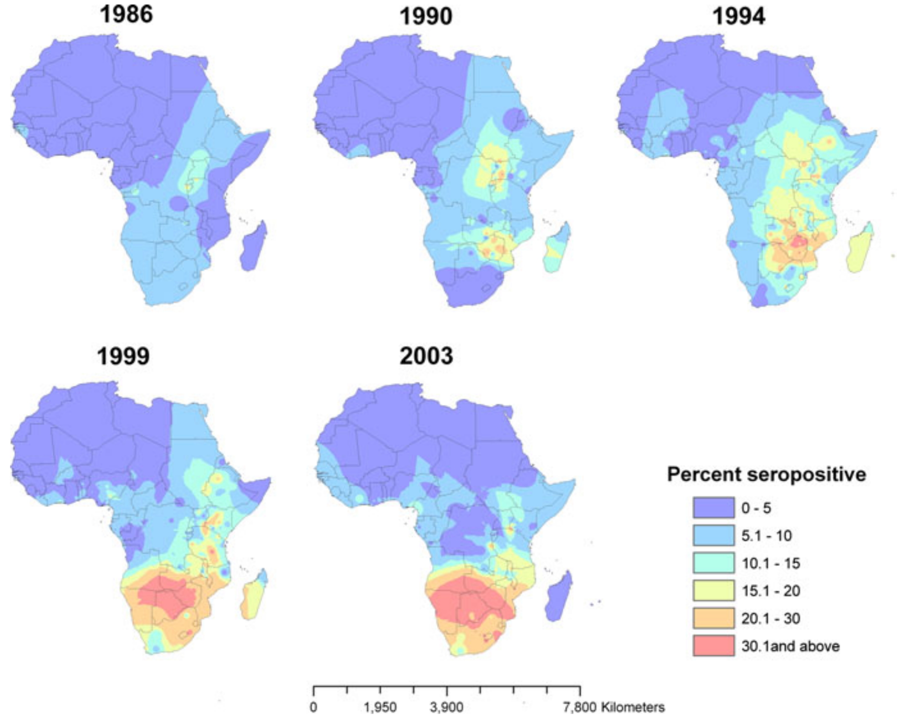

Fig. 3 Interpolated smooth surface maps of HIV/AIDS prevalence rates for 1986, 1990, 1994, 1999, and 2003

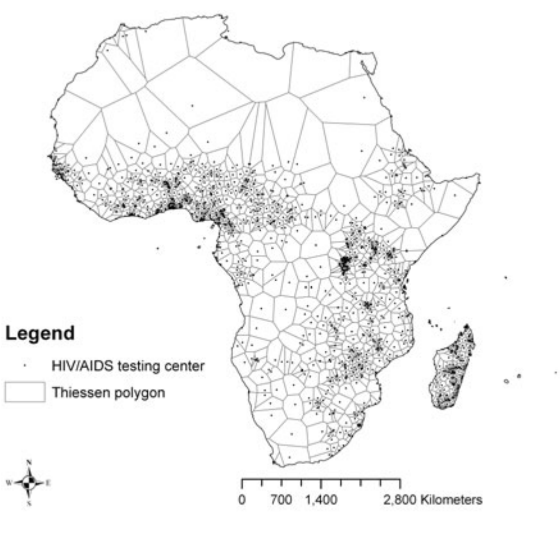

Fig. 4 Thiessen polygons with surveillance data points

quantified the relationships at country level. We present the hypothesized relationships under each of the four sets of variables that we had data for in Table 1, and we discuss variables in the remaining two sets based on findings from other studies.

The first variable set, the colonial context of development in Africa, includes six variables: elevation, population density, resource endowment (not quantified), trade flows (import and exports), net migration, and distance to primary and secondary roads and railways.[2] We hypothesize that elevation will be positively related to HIV/AIDS since high elevation in Africa appears to be associated with high HIV/AIDS rates. We also use elevation as a surrogate for colonial resource exploitation, which triggered labor migrations in eastern and southern Africa at the turn of the nineteenth century (Wilson 1976). We expect positive relationships between HIV/AIDS rates and population density, trade flows, and net migration. We expect a negative relationship between HIV/AIDS rates and distance to transport networks—surrogate measures of interaction or movement of people within and between countries.

The second grouping of variables measures female autonomy. The gendered nature of vulnerability to HIV/AIDS is aptly receiving more attention as more scholars recognize the differential transmission and prevalence rates between men and women (Schoepf 1993; Abrahamsen 1997; Seeley et al. 2004). Still, gaps remain in

[2] Though the 1992 transport network is rather dated, it comes from the most comprehensive global dataset as of December 2008.

Table 1 Variable grouping, description and source

Variable name and hypothesized direction of relationship	Description of variable	Source
Dependent variables		
AIDSRATE	Average HIV/AIDS seropositive rate per country (or Thiessen polygon) estimated from a smooth surface digital map generated by extrapolating point-source HIV infection rates in ArcGIS-ArcView 8.3). Three variables were generated for years 1990, 1995, 1999	Centra Technology, Inc
Geographic explanatory variables		
DIST_ROADS [−]	Average distance from any point in a country (or Thiessen polygon) to the nearest road or railway line in m derived from national maps of roads and railways extracted from the Digital Chart of the World, 1992. (Africa was split into a matrix composed of 1 km square pixels whose centers were the starting points for distance measurement in the Spatial Analyst of ArcGIS-ArcView 8.3)	The Pennsylvania University Libraries, Pattee Maproom[a]
ELEVATION [+]	Average elevation in meters by country based on 1 km by 1 km pixels extracted from a digital elevation map of the world.	*Miombo* CD-ROM. START Secretariat, LULC/DIS
POPDENS_03 [+]	Population density per square km for the year 2003 extracted from the Landscan Global Population Database 2003. The database is a digital population map of the entire globe which gives the number or persons for each 1 km square pixel.	Landscan, Oak Rudge National Laboratory[b]
NETMIGR [+]	Net migration rate (%)	CIA website[c]
IE(year) [+]	The total of imports and exports to each country in Africa from other African countries (i.e. trade flows as a surrogate of gross migration flows), 3 variables each for 1990, 1995, 1999.	World Bank and IMF[d]
Gender related explanatory variables		
BCONTROL [−]	Contraceptive prevalence rate (%), 1995–2002	HDR[e]
GDI [−]	Gender-related development index (GDI) value, 2002	HDR[e]
CBR(year) [+]	Crude Birth rate (per 1,000 people), 3 variables for years 1990, 1995, 1999	World Bank[d]
TFR(year) [+]	Total fertility rate (births per woman) 3 variables for years 1990, 1995, 1999	World Bank[d]
FLABOR(year) [−]	Female labor force (% of total labor force), 3 variables for years 1990, 1995, 1999	World Bank[d]
LITRAT(year) [−]	Adult female literacy rate (% of females ages 15 and above), 3 variables for years 1990, 1995, 1999	World Bank[d]
Poverty, development and disease burden explanatory variables		
HDI(year) [−]	Human Development Index, 3 variables for 1990, 1995, 1999	HDR[e]
POVERTY [+]	Human Poverty Index (HPI-1) Value (%)	HDR[e]
UNDERWT [+]	Children underweight for age (% under age 5), 1995–2002	HDR[e]
UNDERHT [+]	Children under height for age (% under age 5), 1995–2002	HDR[e]
LOWBWT [+]	Infants with low birthweight (%) 1998–2002	HDR[e]
TBRATE [+]	Tuberculosis cases (per 100,000 people), 2002	HDR[e]
GDP%GR(year) [−]	GDP growth (annual %), 3 variables for years 1990, 1995, 1999	World Bank[d]
GDP(year) [−]	Per capita GDP (current US$), 3 variables for years 1990, 1995, 1999	World Bank[d]
CAPIT(year) [−]	Gross capital formation (% of GDP), 3 variables for years 1990, 1995, 1999	World Bank[d]
LITRAT(year) [−]	Adult female literacy rate (% of females ages 15 and above), 3 variables for years 1990, 1995, 1999	World Bank[d]
URBP(year) [−]	Urban population (% of total), 3 variables for years 1990, 1995, 1999	World Bank[d]

Table 1 continued

Variable name and hypothesized direction of relationship	Description of variable	Source
URBGR(year) [−]	Urban population growth (annual %), 3 variables for years 1990, 1995, 1999	World Bank[d]
Government attitudes and commitment explanatory variables		
HEALTHP(year) [−]	Health expenditure per capita (current US$), 3 variables for years 1990, 1995, 1999	World Bank[d]
HEXPGDP(year) [+]	Total health expenditure (% of GDP), 3 variables for years 1990, 1995, 1999	World Bank[d]

[a] http://www.maproom.psu.edu/cgi-bin/dcw/dcwarea.cgi?Africa

[b] http://www.ornl.gov/gist/

[c] CIA Website http://www.umsl.edu/services/govdocs/wofact99/5.htm

[d] World Bank: *World Development Indicators* (various years) http://devdata.worldbank.org/dataonline/

[e] UNDP: *Human Development Report* (various years) http://hdr.undp.org/statistics/

our understanding of sexual politics and practices. Under what circumstances, for example, do women partake in commercial sex exchanges, and what characterizes these exchanges? Are they occasional, regular, and what is the major use for the money? Are the terms of sexual engagement negotiated or dictated? Such inquiry is itself complicated by the intersection of unstable and gendered regional economies, gender politics, and cultural perspectives on sex, condoms, and AIDS. Few studies have managed to combine all three of the dimensions. For this study we use six variables as different measures of gender relations: female literacy rates, the gender development index (GDI) calculated for each country by the UNDP in its *Human Development Report* produced annually since 1990, crude birth rates, total fertility rates, contraceptive prevalence rate, and female labor force participation. We expect countries that have more female empowerment as measured by these variables to have lower HIV/AIDS rates, and vice versa.

For the poverty and disease burden grouping, we use the Human Development Index (HDI) and the Human Poverty Index (HPI) reported by country in UNDP *Human Development Reports*, percent children underweight for their age, percent children under-height for their age, percent infants with low birth weight, tuberculosis cases per 100,000 people, annual percent GDP growth, per capita GDP, gross capital formation as percent of GDP, percent urban population, and urban annual growth rate. These variables also provide measures of levels of socio-economic development, and the hypothesized nature of statistical relationships is given in Table 1. For the fourth variable grouping, government commitment to better healthcare, we used total health expenditure as percent of GDP (as in Gerdtham and Löthgren 2002). However, per capita health expenditure had sufficient data only for the year 1999.

We had no data for global factors and culture, the fifth and sixth groupings of variables, and we only discuss them based on findings from other studies. The global factors include availability of medicines, condom donations, and monetary donations from the international community. Cultural factors included circumcision, polygamy, and practices of widow inheritance and the "hyena custom." However, for these two groupings of variables, there were no data readily available for statistical analysis. In this paper culture refers to the learned ways of life of a group of people. It encompasses the interrelated sociological, ideological, and technological sub-systems of the group (Fellman et al. 2001; Yeboah 2007). A group's attitudes and beliefs towards sex, men and women, and new technologies such as condoms are all part of their collective culture. As Yeboah notes, it is the context of changing culture that provides hope for the fight against HIV/AIDS in Africa (Yeboah 2007). In contrast, many studies have linked African culture to the spread of HIV/AIDS on the continent.

Methods of analysis

In order to account for the spatial variation of HIV/AIDS rates in sub-Saharan Africa, we employ three techniques: GIS analysis (described above), analysis of variance (ANOVA), and multiple linear regression models. ANOVA was used to determine whether there was statistically significant variation in HIV/AIDS rates by region and between rural and urban areas. We used several regression variable-entering methods in the SPSS statistical program, i.e. forward, backward, remove, and stepwise, in order to cross-check the validity of significant variables and eliminate non-significant ones. Regression models were produced for each of the four variable groupings, year (1990, 1995, and 1999), and spatial scale (national, service area). Each regression model used all the various in each variable set. However, only statistically significant variables from regression models of each of the four variable sets were entered into a final overall regression model using the stepwise method to explain the variation of HIV/AIDS rates across space. The resulting regression models had the following form:

$$\text{HIV/AIDS rates (1990)} = \beta_0 + \beta_1 X_1 + \beta_2 X_2 + \beta_2 X_3 \ldots + \varepsilon$$

$$\text{HIV/AIDS Rates (1995)} = \beta_0 + \beta_1 X_1 + \beta_2 X_2 + \beta_2 X_3 \ldots + \varepsilon$$

$$\text{HIV/AIDS rates (1999)} = \beta_0 + \beta_1 X_1 + \beta_2 X_2 + \beta_2 X_3 \ldots + \varepsilon$$

where the dependent variable (HIV/AIDS Rate) is a linear function of a constant β_0, coefficients B_1 to B_n, a set of independent variables (X_i to H_n), an error term ε.

Results and discussion

Rural/urban and regional differentials in HIV/AIDS prevalence rates

The HIV/AIDS data from surveillance and testing centers had a variable indicating if a site was urban or rural. This allowed statistical comparison of average rural and urban HIV prevalence for selected years (1986, 1990, 1995, and 1999) using ANOVA. The results of this analysis are given in Fig. 5 and Table 2. The table shows that mean HIV/AIDS rates for urban areas are consistently higher than those for rural areas, but the difference was statistically significant only for 1995. This is an indication that the disease has spread widely in both rural and urban areas over time and at least reduced reported rural/urban differences.

However, the smooth surface maps that we generated in ArcGIS indicate that HIV/AIDS rates vary greatly in time and space. The 1986 map shows an epicenter around Rwanda, Burundi and Uganda (see Fig. 3). By 1990 two epicenters had developed centered around Uganda in eastern Africa and Zimbabwe in Southern Africa. By 1995 the epidemic had intensified in southern Africa centered on Zimbabwe and Botswana, tapered off in eastern Africa and moved northwards to Ethiopia. The 1999 picture showed that the HIV epicenter had moved deeper into South Africa and a new hot spot had developed in Cameroon and in other parts of West Africa such as Nigeria and Cote d'Ivoire.

Analysis of variance revealed statistically significant differences in HIV prevalence by African region as defined by Krabacher et al. (2009)—west, central, east and south. As the p-values for the F ratio in Table 3 show, the statistical significance in mean HIV/AIDS variation by region at the 0.05 level of significance is consistent for 1990, 1995, and 1999. The west has the lowest mean followed by central, east and finally southern Africa (highest rates), confirming the visual results of the interpolated maps (Fig. 3). However, the regional variation may mask national differences. For instance, despite the generally moderate to low HIV rates (below 5%) recorded for West Africa, some countries have high rates, e.g. Cote d'Ivoire and Cameroon.

Results of regression analysis

The regional variations in HIV/AIDS rates discussed in the preceding section signify that there is not just one epidemic in sub-Saharan Africa but many. Hence, the complexity of these epidemics cannot be explained by one factor but a complex set of independent variables. Some variables are more specific to one region than others. We therefore designed the theoretical framework in Fig. 1 to account for such variations at country and "service area" (Thiessen polygon) levels via the diverse set of variables assembled in Table 1. Below we present and discuss the results of the various regression models that we conducted to explain variation in HIV/AIDS prevalence in Sub-Saharan Africa.

Regression models for the spatially explicit variables

The historical and geographical context of Africa is critical to understanding the spread of the HIV/AIDS

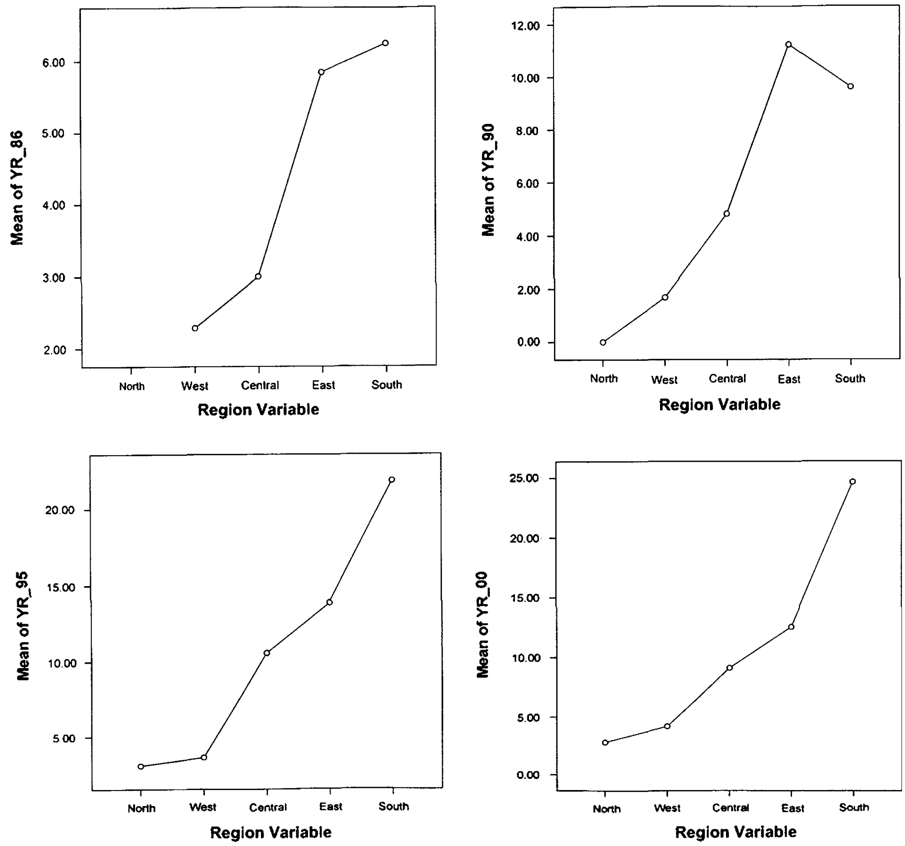

Fig. 5 Analysis of variance for the variation of means for the HIV/AIDS rates by region

Table 2 Analysis of variance for rural–urban differentials in HIV/AIDS rates

Year	Region	*N*	Mean HIV/AIDS rate	*F* ratio	Significance level (*p*)
1986	Urban	29	4.80	2.4	0.13
	Rural	20	2.57		
	Total	49	3.90		
1990	Urban	49	8.01	0.28	0.6
	Rural	81	7.21		
	Total	130	7.51		
1995	Urban	66	13.16	4.08	.044**
	Rural	191	9.97		
	Total	257	10.79		
1999	Urban	84	13.97	0.29	0.59
	Rural	209	13.18		
	Total	293	13.40		

** Significant at the .05 level

epidemic. Since the arrival of the Portuguese in West Africa in the fifteenth century, the continent has seen one crisis after another. First it was slavery that dislocated millions of Africans, and then colonization by European settlers and traders, ushering in a new economic system based on exploitative labor migration. Hopes for a new dawn for the African peoples at independence from colonial rule in the 1960s was soon dashed as the structural links to international market forces that had been put in place during the colonial era

Table 3 Analysis of variance for regional differentials in HIV/AIDS rates

Year	Region	*N*	Mean HIV/AIDS rate	*F* ratio	Significance level (*p*)
1986	West	10	2.28	1.65	0.19
	Central	22	3.00		
	East	11	5.84		
	South	6	6.24		
	Total	49	3.89		
1990	West	27	1.67	8.32	.00*
	Central	24	4.80		
	East	42	11.21		
	South	36	9.59		
	Total	130	7.51		
1995	West	112	3.57	46.25	.000*
	Central	24	10.53		
	East	63	13.82		
	South	57	21.57		
	Total	257	10.78		
1999	North	65	4.21	61.23	.00*
	West	65	9.12		
	Central	82	12.54		
	East	87	24.64		
	South	302	13.40		
	Total				

* Significant at the .01 level

were intransigent and condemned Africa to a subservient position in political and economic relations. This is why we included geographic variables in this analysis as surrogate variables for the colonial experience, including labor migration, geographic interaction and flows of people across countries.

As Yeboah points out, Africa's physical environment has been both a blessing and a curse (Yeboah 2007). It has had more pervasive effects on HIV/AIDS than opportunities that the tropical environment provides for the breeding of bacteria, viruses, fungi, and other parasites. He further points out that juxtaposition of a map of HIV/AIDS rates to a topographic map of sub-Saharan Africa clearly reveals that southern and eastern Africa regions (high Africa) are associated with the highest HIV/AIDS prevalence rates. West and central Africa regions, or low Africa, have the lowest elevation and HIV/AIDS rates. Yeboah contends that while seemingly spurious, this association between elevation and HIV/AIDS prevalence can be used to explain the current HIV/AIDS situation in the region. The high prevalence of HIV/AIDS in the AIDS belt (southern and eastern Africa) is strongly associated with the combination of the physical environment, history, international migration, and Eurocentric views of the region (Yeboah 2007), hence our inclusion of elevation as a proxy variable for colonial influence. Because of the excellent climate in eastern and southern Africa conducive to European settlement, rich soils, mineral abundance (e.g., gold and diamonds) and other resources, more Europeans came to settle in this part of (high) Africa than in (low) tropical West Africa. In doing so, they established islands of economic development, setting in motion massive movements of labor from rural areas to these areas in high Africa. The intermixing of peoples on a grand scale meant also the spread of various diseases form one part to another, including sexually transmitted diseases.

Using elevation as a surrogate variable to measure colonial influence may not be as problematic as it appears to be (see for example Yeboah 2007). While the connection that we make between HIV/AIDS level and climate and colonial settlement is obvious, that with the "islands of economic development" may not be so clear in some areas of the continent.

For example, although European settlements did not take root in 'low' West Africa as much as they did in 'high' Africa, there were pockets of economic development around mineral and agricultural resources in West Africa. Migrant labor from the northern parts of the region came south to the mining towns and the cocoa growing areas. When one examines the geographic pattern of HIV/AIDS rates in West Africa closely, one notices that the highest rates are indeed found in the mining towns and the cocoa growing areas of the South. Furthermore, the known duration of HIV-1 strain of AIDS common in West Africa is relatively brief. The disease first began in Central Africa and then spread to Eastern and Southern Africa in that order (see Iliffe 2006). Still, while the disease is picking up in certain regional "islands of economic development" in West Africa including Cote D'Ivoire and southern Nigeria, HIV/AIDS rates are still generally lower in West Africa than in eastern and southern Africa.

In spite of our rigorous explanation of the potential role of elevation in the incidence and diffusion of HIV in Africa, we wish to offer a cautionary note about this variable. The relationship between this variable and the variation of HIV/AIDS in Africa should be considered in its proper context. We are aware that this variable could be a spurious or a distal variable at best with an indirect relationship to the variation of HIV/AIDS rates that requires further analysis, perhaps through partial regression or PATH analysis. Furthermore, we are aware that the research implications of using this variable point to the notion of environmental determinism, an area which geographers have tried to dissociate themselves from over the past several decades.

One other problem of using aggregated data at the regional level is ecological fallacy. For example, although the association between altitude and HIV rates is fairly strong statistically, Ethiopia is a noteworthy anomaly. With the largest national area in Africa above 2,000 meters (50% of all land above 2,000 m in Africa), Ethiopia has the lowest HIV rates in Eastern Africa—1.4% in 2005, according to the Demographic and Health Survey, and 3.5% based on 2005 antenatal data (Central Statistical Agency of Ethiopia and ORC Macro 2006). That said the association between altitude and HIV may be even stronger in East Africa than this study computes based on "average altitude" (Table 1) because population densities are significantly higher in more humid highlands than in the arid lowlands.

Mean elevation was also the dominant explanatory variable in the three models also incorporating distance from roads and railways, population density, net migration rate, and gross trade flows to test the colonial and labor migration hypothesis at the country and "service-area" levels. Gross trade flows was used as a surrogate of gross migration flows between countries. Population density was significant for 1990 and 1999. However, the direction of the population relationship for 1999 was counterintuitive. The standardized regression coefficient on population density had a negative value instead of a positive one as hypothesized. These mixed results could be explained by one limitation of population density. The use of population density can be misleading due to the frequent informal settlements that are not captured on demographic surveys in Africa. These areas can be extremely significant, e.g. the Kibera slum in Kenya.

Table 4 Standardized regression coefficients for country level regression results (geographic or spatial variables) for HIV/AIDS models, 1990, 1995 and 1999

Variable name	1990 HIV/AIDS rate model	1995 HIV/AIDS rate model	1999 HIV/AIDS rate model
Elevation	0.45*	0.67*	0.62*
Population density	0.22*	−0.16	−0.34*
Distance from road/rail	−0.06	−0.11	−0.06
Net migration	0.02	0.02	0.04
Imports/exports	−0.21	−0.05	0.17
R^2	0.36	0.39	0.4
F ratio	4.78*	5.38*	5.59*

* Implies standardized regression coefficient or the F ratio is significant at the .05 level

Table 5 Standardized regression coefficients for Thiessen regression results (geographic or spatial variables) for HIV/AIDS models, 1990, 1995 and 1999

Variable name	1990 HIV/AIDS rate model	1995 HIV/AIDS rate model	1999 HIV/AIDS rate model
Elevation	0.46*	0.40*	0.36*
Population density	0.03	−0.06*	−0.08*
Distance from road/rail	−0.03	−0.06*	−0.06*
R square	0.21	0.16	0.13
F ratio	124.50*	89.38*	71.20*

* Implies standardized regression coefficient or the *F* ratio is significant at the .05 level

The other three variables (Fig. 1) were statistically insignificant at the .05 level. However, for the distance from road or rail variable, although not significant, the negative direction of the relationship was maintained for all the 3 years. In other words, the farther away a location was from a road or railway the lower was the value for the HIV/AIDS rate. It should be kept in mind throughout this analysis that the quality of data might have something to do with the lack of significant relationships. The regression models for the 3 years were, nevertheless, statistically significant as indicated by the F ratios. This was further validated by conducting analysis at the "service area" or subnational level. The results of the "service-area" level analysis are given in Table 5. Once again elevation, population density, and distance from road or rail came out significant for some of the years. Although the R square values were much lower than in the country level analysis, the service-area regression models were highly significant at the .05 level of significance. In other words, historical context and geography is important in our understanding of some of the drivers behind this epidemic.

Regression models for the gender empowerment variables

The results of this section largely confirm the discussion about the role of gender empowerment. In sub-Saharan Africa, the epidemic affects women disproportionately more than men (UNAIDS 2008). Duffy (2005) recently found in Zimbabwe that women exist under difficult and oppressive conditions. Their socialization to become workers and mothers occurs within a context of limited voice, of subservience, violence, and economic powerlessness—all barriers to HIV prevention (Duffy 2005). Analysis of socio-cultural and economic factors suggests that cultural beliefs and practices, along with national and international forces, reinforce gender inequalities (Duffy 2005). Duffy argues that for a change in the status of women and in the AIDS crisis, prevention strategies need to be multifaceted, and include people's culture and context, and include gender analysis. As Delay further notes, women's biologic, cultural, economic, and social status can increase their likelihood of becoming infected with HIV (Delay 2004). It is therefore important to examine the role of gender empowerment on spatio-temporal patterns of AIDS.

The hypothesis under this grouping of variables is that gender equality is crucial if the advance of this disease is to be arrested. Women's socio-economic autonomy is important. A single mother with children and in gainful employment, for example, is unlikely to resort to commercial sex work. The choice of variables under this grouping were based on the literature on enhancing gender empowerment for women (e.g., LeVine et al. 2002; Charmes and Wieringa 2003; Dijkstra and Hanmer 2000; Greene 2001), namely: contraceptive prevalence, gender development index, crude birth rate, total fertility rate, female labor force levels and literacy rates. The choice of these gender related variables is premised upon the fact that high contraceptive prevalence can free women from constant child bearing and lower crude birth rates and total fertility rates. Females in societies with high contraceptive prevalence are likely to have more say in sexual relations and activities. Studies have indicated that higher female literacy rates, female labor force participation, and the UNDP gender-related development index are all indicators of female autonomy (Charmes and Wieringa 2003). Where females have greater autonomy such as in highly developed countries of the

Table 6 Standardized regression coefficients for country-level regression results (gender empowerment variables) for HIV/AIDS models, 1990, 1995 and 1999

Variable name	1990 HIV/AIDS rate model	1995 HIV/AIDS rate model	1999 HIV/AIDS rate model
Contraceptive prevalence	0.69*	0.50**	0.38**
Gender development index	−0.35	−0.83*	−0.74*
Crude birth rate	−0.51	0.43	1.68*
Total fertility rate	0.78	−0.73	−1.99*
Female labor force	0.48*	0.27	0.1
Literacy rates	0.17	0.54*	0.69
R square	0.42	0.54	0.75
F ratio	2.48**	4.16*	9.12*

* Implies standardized regression coefficient or the *F* ratio is significant at the .05 level

** Implies standardized regression coefficient or the *F* ratio is significant at the .10 level

West, HIV/AIDS rates are much lower in comparison to men, a fact that does not hold in less developed countries.

The regression models for this grouping are given in Table 6. The contraceptive prevalence rate (percent of women with access to or using birth control) was very significant for the 3 years but in the opposite direction (positive) to the hypothesized one. Thus, countries where contraceptive prevalence was high also had high HIV/AIDS rates. However, the Gender Empowerment Index maintained the hypothesized relationship. High index values were significantly associated with lower rates of HIV/AIDS for the 1995 and 1999 models, at the .05 level. Total fertility rates also came up significant for the years 1995 and 1999, as hypothesized.

Regression models for poverty and disease burden variables

As Stillwaggon points out, economic conditions in sub-Saharan Africa in the years in which the AIDS epidemic began reveals an extremely compromised health environment (Stillwaggon 2001). She notes that from 1970 to 1997, sub-Saharan Africa was the only world region to experience a decrease in food production, calorie and protein supply per capita. Stillwaggon further discusses major factors, all related to poverty, that have made Africa fertile terrain for the spread of the epidemic (Stillwaggon 2002). These include the economic background of African health, parasitic diseases such as malaria, trypanosomiasis, onchocerciasis and many others, sexually transmitted diseases as co-factors for HIV transmission, and poor nutrition which compromises the immune system. Her multiple regression analysis of HIV prevalence rates against change in calories 1970–1995, the Gini coefficient, change in urban population 1970–1995, real GDP per capita 1995 and change in per capita GDP 1960–1995 showed many of these variables to be significant, particularly so for income inequality as measured by the Gini coefficient. Similarly, Lau and Muula list several factors as drivers of HIV levels, including worsening economic conditions, food shortages, and urbanization (Lau and Muula 2004). In fact the literature shows a growing consensus that poverty reduction is key to containing HIV/AIDS.

In this study, we identified multiple variables that measure levels of development and disease (see Tables 1 and 7). In spite of the fact that many of these variables were inter-correlated, they were initially all entered into a regression model to see which ones would stand out as significant variables. However, for the final composite model given in Table 7, the stepwise regression technique was used to get the most significant variables for entry into this model. Thus the problem of violating the assumption of multicollinearity was minimized in the final model. Table 7 indicates that some of the independent variables were significant and had the hypothesized relationship direction. For example, while the Human Poverty Index was in the hypothesized direction, it was significant for the 1995 model, but not for 1999. The proportion of children under-weight for their age came out significant for both 1990 and 1999 and was in the hypothesized (positive) direction for all

Table 7 Standardized regression coefficients for country level regression results (poverty and disease burden variables) for HIV/AIDS models, 1990, 1995 and 1999

Variable name	1990 HIV/AIDS rate model	1995 HIV/AIDS rate model	1999 HIV/AIDS rate model
Human development index	0.65	0.99*	0.44
Human poverty index	−0.06	0.44**	0.6
Children underweight for age	−0.59**	−0.17	−0.49**
Children under height for age	0.39	0.54*	0.67*
Infant with low birth weight	0.1	−0.18	−0.17
Tuberculosis rate	0.03	0.16	0.44*
% GDP annual growth	0.12	−0.03	−0.15
Per capita GDP	−0.22	0.04	0.57**
Gross capital formation (% GDP)	−0.61*	−0.22	−0.13
Urban population (%)	−0.76*	−0.33	0.02
Urban population growth rate	−0.05	−0.05	−0.16
R square	0.55	0.51	0.76
F ratio	2.25**	2.16**	4.70*

* Implies standardized regression coefficient or the *F* ratio is significant at the .05 level

** Implies standardized regression coefficient or the *F* ratio is significant at the .10 level

3 years. Gross capital formation as percent of GDP also had the hypothesized negative relationship and was significant for the 1990 model. The R squares were statistically significant at the .05 and .10 levels of significance for the three models. We therefore infer that poverty is directly related to HIV/AIDS rates in sub-Saharan Africa, confirming Stillwaggon's (2002) findings. Anomalies in some of the variables in terms of the hypothesized direction can at least partly be explained in terms of poor data quality.

In terms of disease burden, the tuberculosis variable maintained the hypothesized direction of the relationship (i.e. positive) and was statistically significant for the 1999 model. In other words, countries with high levels of TB, an opportunistic disease, are also countries with high levels of HIV rates. Indeed, as argued by Stillwaggon and other scholars, disease burden, malnutrition and poverty make the eradication of HIV an uphill battle (Stillwaggon 2002). The warm tropical environments of west, central, east and southern Africa encourage a host of micro-organisms, disease agents, and insect carriers. Malaria, trypanosomiasis, onchocerciasis, schistosomiasis, leishmaniasis and intestinal parasitoses and other diseases are rife in these regions and kill millions of Africans yearly. These ailments also compromise the immunity of people they infect to the HIV virus (Stillwaggon 2002). Examination of maps of HIV/AIDS intensity and the distribution of these diseases for sub-Saharan Africa reveal a clear match.

Final regression models for combined variables

When all the variables for the variable groupings were combined into an overall model for each year by stepwise selection of most important variables, only elevation turned out significant and kept the hypothesized relationship (see Table 8). Yet the selected variables explain a significant amount of the variation in HIV/AIDS rates. R square values range from 0.42 for the 1995 model to 0.53 and 0.57, respectively for 1990 and 1999. The overall models lend credence to the hypothesis that HIV/AIDS levels are intimately tied to multiple geographic (spatial interaction), economic, disease burden, gender, and government commitment drivers.

The seemingly low R-square values in Tables 4, 5, 6, 7 and 8 require some explanation here. It is typical in social science research to have such low R-square values and to rely more on the F-ratio which indicates whether the R-square value is statistically significant or not. In our case the F-ratios indicate that the R-square values are actually statistically significant at either the .05 level or the .10 level of significance. The statistical significance indicates that the variables in our analysis have a significant impact on HIV/AIDS rates and the epidemic in general.

The role of culture

Perhaps the most complex driver of the variation of HIV/AIDS in sub-Saharan Africa is the impact of

Table 8 Standardized regression coefficients for country level regression results (composite models with the best fit Variables) for HIV/AIDS models, 1990, 1995 and 1999

Variable name	1990 HIV/AIDS rate model	1995 HIV/AIDS rate model	1999 HIV/AIDS rate model
Children under height for age	[a]	0.7	0.08
Contraceptive prevalence	0.37*	[a]	[a]
Crude birth rate	[a]	[a]	0.59
Elevation	0.35*	[a]	0.35**
Female labor force	0.35*	0.25	[a]
Female literacy rates	[a]	0.44	[a]
Gender development index	[a]	−0.63	0.45
Gross capital formation (% GDP)	−0.39	[a]	[a]
Human development index	[a]	0.42	[a]
Human poverty index	[a]	[a]	0.31
Population density	0.11	[a]	−0.19
Total fertility rate	[a]	[a]	−0.82
Total health expenditure(% GDP)	[a]	[a]	0.25
Tuberculosis rate	[a]	[a]	0.17
Urban population (%)	−0.15	[a]	[a]
R square	0.53	0.42	0.57
F ratio	5.66*	3.97*	3.77*

* Implies standardized regression coefficient or the F ratio is significant at the .05 level

** Implies standardized regression coefficient or the F ratio is significant at the .10 level

[a] Implies variable was not selected for entry in the stepwise regression models

culture. We were unable to find quantified variables to measure culture. Yeboah writes that one of the most difficult issues to write about in terms of HIV/AIDS in Africa is African culture(s) because one may fall into the fallacy of portraying African culture as inferior (Yeboah 2007). It has been noted, for example, that Africans are sex positive, promiscuous, immoral, and that they practice evil customs such as polygamy, widow inheritance, and the immoral deflowering of young girls—the hyena custom (Shannon et al. 1991; Rushing 1995). Indeed the cultural orthodoxy in the spread of HIV/AIDS posits that traditional practices, often conducted in secrecy, may carry increased risk of sexually transmitted infections and that multiple sexual partnerships are more socially acceptable than in other cultures. Further it is noted in this discourse that women have little control over their sex lives, and thus vulnerable to HIV (MacDonald 1996). Yet in terms of polygamy other studies have found a perfect mismatch between HIV/AIDS rates and rates of polygamy. Polygamy is most intense in northern Africa where it is a well entrenched institution in Islamic societies, and yet this same region has the lowest HIV/AIDS rates on the continent (Tastemain and Coles 1993; Oppong 1998). Furthermore, no research has been conducted to determine extent of the harmful customs that Africans supposedly continue to practice, thereby fueling the rampant spread of HIV/AIDS.

In contrast to such stereotypical views and research, some scholars have found that some African practices may actually aid in stopping the rapid spread of the epidemic. For example, a map of societies that do not traditionally circumcise males produced by Caldwell and Caldwell (Caldwell 1994) resembled that of areas of high HIV/AIDS rates for 1989 so much that it led to the hypothesis that there is a strong positive relationship between being uncircumcised and HIV/AIDS incidence. While Caldwell and others note that the data from which this hypothesis is derived were not perfect (Caldwell 1994; Bongaarts et al. 1989; Moses et al. 1990; Plummer et al. 1991), later epidemiology based studies appear to confirm the hypothesis and immense value of the practice of circumcision for HIV prevention. Recent studies in South Africa, Kenya and Uganda have conclusively found that male circumcision greatly reduces the risk that men will contract HIV through intercourse with infected women by up to 70 percent (Auvert et al. 2005; Williams et al. 2006; Bailey et al. 2007; Degregori 2007). We were unable to find quantitative data on circumcision, for instance a variable measuring the percentage of men circumcised in a given year, to test the hypothesis in this study. Still, Bonner cautions that until we know why and how circumcision is protective, exactly what the relationship is between circumcision status and other STDs, and whether the effect seen in high-risk populations is generalizable to other groups, the wisest course is to recommend risk reduction strategies of proven efficacy, such as condom use (Bonner 2001). Large-scale surveys on the extent and impact specific customs across Africa are needed before a definitive testing of the culture/HIV hypothesis.

Global forces and government commitment

We were unable to find suitable data on global factors. Under this category, if data were available, one could examine the assistance from the developed countries in terms of medications, vaccines, condom donations, and monetary donations. Kalipeni and Mbugua critically review the international response to the HIV/AIDS epidemic in Africa and lament the missed opportunities within the past 20 years (Kalipeni and Mbugua 2005). However, these two authors acknowledge many efforts from the developed world to ease the suffering caused by this epidemic in the developing world, particularly Africa. Lurie and his colleagues show how structural adjustment policies introduced by the World Bank and the International Monetary Fund weakened health-sector infrastructure thus exacerbating the rapid spread of HIV/AIDS in developing countries (Lurie et al. 2004).

As for government commitment, we found two variables measuring health expenditure. The first was per capita health expenditure, which was available only for 1999. The second variable was total health expenditure as a percent of GDP, and was available for the 3 years. For 1999, health expenditure as a percent of GDP was significant at the .05 level, and was selected into the overall regression model by the stepwise method (Table 7). Beyond funding levels, government political commitment has proven to be extremely important in stopping the epidemic in its tracks (as in Senegal), or in reducing the high HIV/AIDS rates (e.g. Uganda). For a detailed discussion of these two case studies see Kalipeni and Mbugua (2005).

Limitations of the study

There are several limitations that need to be acknowledged and addressed regarding this study. The analysis and discussion in this paper offers some new and interesting insights on the spatial variability and distribution of HIV/AIDS in Africa using a set of GIS and statistical techniques. We have demonstrated familiarity with the literature including the presentation of a well-formulated conceptual framework that captures the salient determinants of HIV diffusion across the continent. However, one of the major limitations of the study is the lack of a full evaluation of the proposed conceptual framework due to the lack of pertinent data on some of the key drivers of the HIV/AIDS pandemic on the continent. Given this limitation, this paper should be seen as falling more within the realm of exploratory spatial data analysis rather than a confirmatory approach to HIV/AIDS risk assessment in Africa.

The second major concern that needs to be raised is the spatial scales at which the analyses were performed. There is an apparent danger and several deficiencies involved when analyzing health data at some of these levels, particularly the continental and national scales, as well as the failure to truly account for the local context for HIV/AIDS diffusion. First, national level data mask local variations within a given country and are collected using different methodologies from one country to another. This brings in all sorts of errors in the data and their comparability is often compromised across countries. Furthermore, the “Modifiable Areal Unit Problem” (MAUP) is a potential source of error that can affect spatial studies which utilize aggregate data sources (Unwin 1996). Geographical data are often aggregated in order to present the results of a study in more convenient spatial units either at national level or at some geographic unit or zone such as the Thiessen polygon as used in this paper to show some spatial phenomena such as HIV/AIDS prevalence rates. These zones are often arbitrary in nature and different areal units can yield different outcomes.

Thus one level of geographic unit (e.g. national) may not necessarily be the best level to display the spatial variation of the data in a comprehensible manner. It is this variation in acceptable areal solution that generates the term “modifiable” where “the areal units (zonal objects) used in many geographical studies are arbitrary, modifiable, and subject to the whims and fancies of whoever is doing, or did, the aggregating.” (Openshaw 1984, p.3). Thus, while there are a large number of different spatial objects or aggregation units and ways in which a large geographical area such as Africa can be subdivided, the choice of areal units used in this study was constrained by what was available to us rather than what would have been the best. Therefore, the MAUP raises concerns of scale and aggregation, and the concept of the ecological fallacy also needs to be considered seriously (Bailey and Gatrell 1995). The scale problem is the variation which can occur when data from one scale of areal units is aggregated to a different scale. As noted above much of the spatial variation at district level will change or get lost when the data are extrapolated spatially and aggregated to the regional or national level. Our choice to conduct the study at multiple scales to ascertain consistency in the power of the regression models at different scales of data aggregation may have subjected results to the “Modifiable Areal Unit Problem.” For example, the R square value dropped by nearly half the original value when the analysis was done using Thiessen polygons. The question here is why should not the results be stronger at a finer spatial resolution? These were some of the challenges and pitfalls of conducting the analyses at multiple geographic scales.

Another limitation of concern in this study was the obvious instability of the regression models. The variables were not consistent across the models nor were the signs (direction of the relationships) as shown in Table 8. While the MAUP may have contributed, we were unsure why else this proved to be the case. Our initial suspicion was that these inconsistencies were due to some violation of regression assumptions such as the presence of serial and spatial autocorrelation, homoskedasticity, multicollinearity and the violation of normality for the dependent variable. Although we ran separate models and selected the most significant variables for entry into a final stepwise regression model, this procedure did not necessarily eliminate the problem of multicollinearity. We therefore subjected the models to diagnostic tests to ensure they were robust and analytically sound. We checked the tolerance levels, variance inflation factors and condition indices to provide a much better indication of the violation of regression assumptions. In certain instances the diagnostic statistics did indeed indicate the violation of some of the regression assumptions such as the presence of spatial and serial autocorrelation, but overall the statistics were acceptable at the .05 level of significance. Thus in spite of the instability from one regression model to the next, the diagnostic tests we conducted gave us confidence in the research findings, an indication of the fact that the analysis can be replicated in future studies as new data becomes available.

Conclusion

This study is testament to the usefulness (and limitations) of techniques and spatial analysis in examining the diffusion of the HIV/AIDS epidemic in both space and time. The article offers an alternative way grounded in geographic analysis for yielding estimates of HIV/AIDS rates in Africa for further analysis at spatial scales other than those at which the data were collected. Using incomplete sets of data covering the period 1986–2003, mean HIV/AIDS rates were generated spatially, offering a geographic overview of the spread of the epidemic in space and time. As the spatial and statistical analyses and the discussion of the results above indicate, there is a strong geography to variations in HIV/AIDS rates in Africa, with some regions suffering more than others. This paper, using GIS and advanced statistical techniques has clearly shown that the HIV/AIDS epidemic is complex and that it is interconnected with other geographic, historical, economic and cultural phenomena which help explain its spatial spread, variation and intensity. The maps in Fig. 3 clearly show that the disease started to spread from central to eastern Africa, into southern Africa, and is spreading rapidly in some West African countries.

The regression models which were developed in this paper illustrated that variables which measure the historical context of colonialism such as resource exploitation and labor migration, gender relations, culture, contemporary global forces, poverty and disease burden are interconnected and have all contributed greatly to the rapid, spatio-temporal spread of this disease. However, as illustrated in the last section all is not lost. It is possible to mitigate the rapid spread of this disease as the cases of Uganda and Senegal show (Kalipeni and Mbugua 2005; UNAIDS 2008). Further, strong will on the part of governments in partnership with other stakeholders, and an integrated, multi-faceted approach as revealed by findings of this and other studies, can reverse the trajectory of this epidemic.

Acknowledgments We are grateful to Centra Technology, Inc. for providing the data set that led to the geographic and statistical analysis and construction of the maps contained in this paper. We also wish to acknowledge the valuable comments we received from the individuals who graciously reviewed this paper. Their comments were very constructive and resulted in a much stronger paper.

References

Abrahamsen, R. (1997). Gender dimensions of AIDS in Zambia. *Journal of Gender Studies, 6*(2), 177–189.

Auvert, B., Taljaard, D., Lagarde, E., Sobngwi-Tambekou. J., Sitta, R., et al. (2005). Randomized, controlled intervention trial of male circumcision for reduction of HIV infection risk: The ANRS 1265 trial. *PLoS Medicine, 2*(11), e298-e, 1112–1122. http://medicine.plosjournals.org/archive/1549-1676/2/11/pdf/10.1371_journal.pmed.0020298-L.pdf. Accessed 17 August 2008.

Bailey, T. C., & Gatrell, A. C. (1995). *Interactive spatial data analysis* (2nd ed.). Harlow: Longman.

Bailey, R. C., Moses, S., Parker, C. B., Agot, K., Maclean, I., Krieger, J. N., et al. (2007). Male circumcision for HIV prevention in young men in Kisumu, Kenya: A randomised controlled trial. *Lancet, 369*(9562), 643–656.

Berkman, L. F., & Kawachi, I. (2000). *Social epidemiology*. Oxford: Oxford University Press.

Bongaarts, J., Reining, P., Way, P., & Conant, F. (1989). The relationship between male circumcision and HIV infection in African population. *AIDS, 3*(6), 373–377.

Bonner, K. (2001). Male circumcision as an HIV control strategy: Not a 'natural condom'. *Reproductive Health Matters, 9*(18), 143–155.

Brockerhoff, M., & Biddlecom, A. E. (1999). Migration, sexual behavior and the risk of HIV in Kenya. *International Migration Review, 33*(4), 833–856.

Bwayo, J. (1994). Human immunodeficiency virus infection in long-distance truck drivers in East Africa. *Archives of Internal Medicine, 154*(12), 1391–1396.

Caldwell, J. (1994). The neglect of an epidemiological explanation for the distribution of HIV/AIDS in sub-Saharan Africa: Exploring the male circumcision hypothesis. In J. G. Cleland & P. O. Way (Eds.), *AIDS impact and prevention in the developing world: Demographic and social science perspectives* (pp. 23–46). Canberra: Health Transition Centre and National Centre for Epidemiology and Population Health, Australian National University.

Campbell, C. (1997). Migrancy, masculine identities and AIDS: The psychosocial context of HIV transmission on the South African gold mines. *Social Science and Medicine, 45*(2), 273–281.

Central Statistical Agency [Ethiopia] and ORC Macro. (2006). *Ethiopia demographic and health survey 2005*. Addis Ababa, Ethiopia and Calverton, Maryland, USA: Central Statistical Agency and ORC Macro. http://www.measuredhs.com/pubs/pdf/FR179/FR179.pdf. Accessed 25 August 2008.

Charmes, J., & Wieringa, S. (2003). Measuring women's empowerment: An assessment of the gender-related development index and the gender empowerment measure. *Journal of Human Development, 4*(3), 419–435.

Chirwa, W. C. (1998). Aliens and AIDS in southern Africa: The Malawi-South Africa debate. *African affairs, 97*(386), 53–81.

Connor, E. M., Sperling, R. S., & Gelber, R. (1994). Protocol 076: Reduction in maternal-infant transmission of HIV type 1 with zidovudine treatment. *New England Journal of Medicine, 331*(18), 1173–1180.

Decosas, J., Kane, F., Anarfi, J. K., Sodji, K. D., & Wagner, H. U. (1995). Migration and AIDS. *Lancet, 346*(8978), 826–828.

Degregori, M. C. A. (2007). Critical medical anthropology perspectives on male circumcision to prevent HIV. In Medicus. Mundi (Ed.), *Women, AIDS and access to health care in sub-Saharan Africa: Approaches from the social sciences* (pp. 127–138). Barcelona: Medicus Mundi Catalunya.

Delay, P. (2004). Gender and monitoring the response to HIV/AIDS pandemic. *Emerging Infectious Diseases, 10*(11), 1979–1983.

Diallo, M. O., Ettiene-Traore, V., Maran, M., Kouadio, J., Brattegard, K., Makke, A., et al. (1997). Sexually transmitted diseases and human immunodeficiency virus infections in women attending an antenatal clinic in Abidjan, Cote d'Ivoire. *International Journal of STD and AIDS, 8*(10), 636–638.

Dijkstra, A. G., & Hanmer, L. (2000). Measuring socio-economic gender inequality: Toward an alternative to the UNDP gender-related development index. *Feminist Economics, 6*(2), 41–75.

Duffy, L. (2005). Culture and context of HIV prevention in rural Zimbabwe: The influence of gender inequality. *Journal of Transcultural Nursing, 16*(1), 23–31.

Fellman, J., Getis, A., & Getis, J. (2001). *Human geography: Landscapes of human activities*. Boston, MA: McGraw Hill.

Ferguson, A. G., & Morris, C. N. (2007). Mapping transactional sex on the Northern Corridor highway in Kenya. *Health & Place, 13*(2), 504–519.

Gerdtham, U.-G., & Löthgren, M. (2002). New panel results on cointegration of international health expenditure and GDP. *Applied Economics, 34*(13), 1679–1686.

Girdler-Brown, B. (1998). Eastern and southern Africa. *International Migration Review, 36*(4), 513–551.

Gisselquist, D. (2004). Impact of long-term civil disorders and wars on the trajectory of HIV epidemics in sub-Saharan Africa. *Journal of Social Aspects of HIV/AIDS, 1*(2), 114–127.

Greene, R. W. (2001). Governing reproduction: Women's empowerment and population policy. *Communication Abstracts, 24*(1), 3–147.

Haour-Knipe, M., & Rector, R. (1996). *Crossing borders: Migration, ethnicity, and AIDS*. Bristol, PA: Taylor and Francis.

Iliffe, J. (2006). *The African AIDS epidemic: A history*. Athens, Ohio: Ohio University Press.

Kalipeni, E., & Mbugua, N. (2005). A review of prevention efforts in the fight against HIV and AIDS in Africa. *Norwegian Journal of Geography, 59*(1), 26–36.

Kalipeni, E., & Oppong, J. (1998). The refugee crisis in Africa and implications for health and disease: A political ecology approach. *Social Science and Medicine, 46*(12), 1637–1653.

Kalipeni, E., & Zulu, L. (2008). Using GIS to Model and Forecast HIV/AIDS Rates in Africa, 1986–2010. *Professional Geographer, 60*(1), 33–53.

Krabacher, T., Kalipeni, E., & Layachi, A. (2009). *Global studies. Africa*. Boston: McGraw-Hill Contemporary Learning Series.

Kreiss, J., Koech, D., Plummer, F., Holmes, K., Lightfoote, M., Piot, P., et al. (1986). AIDS virus infection in Nairobi prostitutes: Spread of the epidemic to East Africa. *New England Journal of Medicine, 314*(7), 414–418.

Lau, C., & Muula, A. S. (2004). HIV/AIDS in sub-Saharan Africa. *Croatian Medical Journal, 45*(4), 402–414.

LeVine, R. A., LeVine, S. E., & Schnell, B. (2002). 'Improve the women': Mass schooling, female literacy, and worldwide social change. *Educational Administration Abstracts, 37*(1), 3–136.

Lurie, P., Hintzen, P. C., & Lowe, R. A. (2004). Socioeconomic obstacles to HIV prevention and treatment in developing countries: The role of the international monetary fund and the world bank. In E. Kalipeni, S. Craddock, J. R. Oppong, & J. Ghosh (Eds.), *HIV/AIDS in Africa: Beyond epidemiology* (pp. 204–212). Oxford: Blackwell.

Lyons, M. (2004). Mobile populations and HIV/AIDS in East Africa. In E. Kalipeni, S. Craddock, J. R. Oppong, & J. Ghosh (Eds.), *HIV/AIDS in Africa: Beyond epidemiology* (pp. 175–190). Oxford: Blackwell.

MacDonald, D. S. (1996). Notes on the socio-economic and cultural factors influencing the transmission of HIV in Botswana. *Social Science and Medicine, 42*(9), 1325–1333.

Moses, S., Bradley, J. E., Nagelkerke, N. J. D., Ronald, A. R., Ndinya-Achola, J. O., & Plummer, F. A. (1990). Geographical patterns of male circumcision practices in Africa: Association with HIV prevalence. *International Journal of Epidemiology, 19*(3), 693–697.

Ndinya-Achola, J. O., Ghee, A. E., Kihara, A. N., Krone, M. R., Plummer, F. A., Fisher, L. D., et al. (1997). High HIV prevalence, low condom use and gender differences in sexual behavior among patients with STD-related complaints at a Nairobi primary health care clinic. *International Journal of STD and AIDS, 8*(8), 506–514.

Openshaw, S. (1984). *The modifiable areal unit problem*. Norwich: GeoBooks. Concepts and techniques in modern geography 38.

Oppong, J. R. (1998). A vulnerability interpretation of the geography of HIV/AIDS in Ghana, 1986–1995. *Professional Geographer, 50*(4), 437–448.

Pickering, H., Okongo, M., Ojwiya, A., Yirrell, D., & Whitworth, J. (1997). Sexual networks in Uganda: mixing patterns between a trading town, its rural hinterland and a nearby fishing village. *International Journal of STD and AIDS, 8*(8), 495–500.

Plummer, F. A., Moses, S., & Ndinya-Achola, J. (1991). Factors affecting female to male transmission of HIV-1: Implications of transmission dynamics for prevention. In L. C. Chen, J. S. Amor, & S. J. Segal (Eds.), *AIDS and women's reproductive health* (pp. 35–45). New York: Plenum.

Quinn, T., Wawer, M., Sewankambo, N., Serwadda, D., Li, C., Wabwire-Mangen, F., et al. (2000). Viral load and heterosexual transmission of human immunodeficiency virus type 1. *New England Journal of Medicine, 342*(13), 921–927.

Rushing, W. A. (1995). *The AIDS epidemic: Social dimensions of an infectious disease* (pp. 59–90). Boulder, CO: Westview Press.

Schoepf, B. G. (1993). Gender, development, and AIDS: a political economy and culture framework. *Women and International Development Annual, 3*, 53–85.

Seeley, J., Grellier, R., & Barnett, T. (2004). Gender and HIV/AIDS impact mitigation in sub-Saharan Africa: Recognizing the constraints. *Journal of Social Aspects of HIV/AIDS, 1*(2), 87–98.

Shannon, G. W., Pyle, G. F., & Bashshur, R. (1991). *The Geography of AIDS: Origins and course of an epidemic*. New York: Guilford Press.

Stillwaggon, E. (2001). AIDS and poverty in Africa. *Nation, 272*(20), 22–25. http://www.thenation.com/doc.mhtml?i=20010521&c=2&s=stillwaggon. Accessed 19 July 2007.

Stillwaggon, E. (2002). HIV/AIDS in Africa: Fertile terrain. *Journal of Development Studies, 38*(6), 1–22.

Stillwaggon, E. (2003). Racial metaphors: Interpreting sex and AIDS in Africa. *Development and Change, 34*(5), 809–832.

Tastemain, C., & Coles, P. (1993). Can a culture stop AIDS in its tracks. *New Scientist, 139*(Sept.1), 13–14.

UNAIDS. (2004). *2004 Report on the global AIDS epidemic*. Geneva: UNAIDS.

UNAIDS. (2008). *2008 Report on the global AIDS epidemic*. Geneva: Joint United Nations Program on HIV/AIDS (UNAIDS) 2008. http://www.unaids.org/en/KnowledgeCentre/HIVData/GlobalReport/2008. Accessed 31 December 2008.

Unwin, D. J. (1996). GIS, spatial analysis and spatial statistics. *Progress in Human Geography, 20*(4), 441–540.

Wasserheit, J. N. (1992). Epidemiological synergy: Interrelationships between human immunodeficiency virus infection and other sexually transmitted diseases. *Sexually Transmitted Diseases, 19*(2), 61–77.

Waterston, A. (1997). Anthropological research and the politics of HIV prevention: Towards a critique of policy and priorities in the age of AIDS. *Social Science and Medicine, 44*(9), 1381–1391.

Williams, B. G., Lloyd-Smith, J. O., Gouws, E., Hankins, C., Getz, W. M., et al. (2006). The potential impact of male circumcision on HIV in sub-Saharan Africa. *PLoS Medicine 3*(7), e262, 1032-1040. doi:10.1371/journal.pmed.0030262. http://medicine.plosjournals.org/archive/1549-1676/3/7/pdf/10.1371_journal.pmed.0030262-L.pdf. Accessed 30 June 2008.

Wilson, F. (1976). International migration in southern Africa. *International Migration Review, 10*(4), 451–487.

Yeboah, I. E. A. (2007). HIV/AIDS and the construction of Sub-Saharan Africa: Heuristic lessons from the social sciences for policy. *Social Science and Medicine, 64*(5), 1128–1151.

Zuma, K., Gouws, E., Williams, B., & Lurie, M. (2003). Risk factors for HIV infection among women in Carletonville, South Africa: Migration, demography and sexually transmitted diseases. *International Journal of STD and AIDS, 14*(12), 814–817.