Published in final edited form as:
AIDS. 2008 March 30; 22(6): 741–748. doi:10.1097/QAD.0b013e3282f3947f.

Place matters: multilevel investigation of HIV distribution in Tanzania

Wezi M. Msisha[a], **Saidi H. Kapiga**[b], **Felton J. Earls**[c], and **S.V. Subramanian**[c]

[a]Europe and Central Asia Human Development Sector, The World Bank, Washington, DC, USA

[b]The Department of Population and International Health, Harvard School of Public Health, Boston, Massachusetts, USA

[c]Department of Society, Human Development and Health, Harvard School of Public Health, Boston, Massachusetts, USA.

Abstract

Objective—To examine the extent to which the regional and neighborhood distribution of HIV in Tanzania is caused by the differential distribution of individual correlates and risk factors.

Methods—Nationally representative, cross-sectional data on 12 522 women and men aged 15–49 years from the 2003–2004 Tanzanian AIDS Indicator Survey. Three-level multilevel binary logistic regression models were specified to estimate the relative contribution of regions and neighborhoods to the variation in HIV seroprevalence.

Results—Spatial distribution of individual correlates (and risk factors) of HIV do not explain the neighborhood and regional variation in HIV seroprevalence. Neighborhoods and regions accounted for approximately 14 and 6% of the total variation in HIV. HIV prevalence ranged from 1.8% (Kigoma) to 6.7% (Iringa) even after adjusting for the compositional make-up of these regions. An inverse association was observed between log odds of being HIV positive and neighborhood poverty [odds ratio (OR) 0.24, 95% confidence interval (CI) 0.09–0.61] and regional poverty (OR 0.97, 95% CI 0.95–0.99).

Conclusion—Our study provides evidence for independent contextual variations in HIV, above and beyond that which can be ascribed to geographical variations in individual-level correlates and risk factors. We emphasize the need to adopt both a group-based and a place-based approach, as opposed to the dominant high-risk group approach, for understanding the epidemiology of HIV as well as for developing HIV intervention activities.

Keywords

Africa; epidemiology; geography; HIV; seroprevalence; socioeconomic status

Introduction

Research over the past decade has emphasized the importance of socioeconomic contexts for individual health [1–5]. There is currently a large body of empirical research that shows an

Correspondence to S. V. Subramanian, Department of Society, Human Development and Health, Harvard School of Public Health, 677 Huntington Avenue, 7th Floor, Boston, MA 02115-6096, USA., Tel: +1 617 432 6299; fax: +1 617 432 1135; svsubram@hsph.harvard.edu.

Conflicts of interest: None.

association between neighborhood context and individual health and mortality [6–9]. Few systematic analyses of the potential impact of residential contexts on HIV in Africa have been undertaken, even though the importance of context has been emphasized in qualitative investigations and community-specific studies of the HIV epidemic in Africa [10,11]. For example, sociopolitical factors including war, poverty and cultural traditions have been argued to be major drivers of the spread of HIV in Kagera (Tanzania) [12]. Community characteristics such as social capital, community employment and educational opportunities have been shown to be associated with HIV infection and sexual risk behavior among youth in South Africa [13,14]. Urbanization, population mobility and transportation networks, civil conflicts, poverty and cultural factors also have been identified as distal determinants of HIV [15,16]. Studies conducted in Mwanza (Tanzania) have found HIV prevalence to be four times higher at a trading center compared with surrounding rural villages [17,18]. The level of economic and social activity (e.g. trading), high mobility of the population, a high bar worker to male ratio and proximity to the town of Mwanza were the main community factors associated with an increased risk of HIV [17–19]. Studies in Tanzania, as well as Uganda and South Africa, have found that residing in close proximity to mining areas and major road and transportation networks was also associated with a high prevalence of HIV and sexually transmitted infections (STI) [20–22]. In contrast to the above localized studies, the African four-city study observed that the differences in HIV prevalence across the centers were the result of differences in the prevalence of circumcision and herpes simplex virus type 2, thus suggesting that geographical variation in HIV is largely caused by the differential distribution of individual risk factors and behaviors, compared with factors that could be truly contextual [10,11].

In this study, we build upon the limited evidence base (largely based on local community-specific studies using qualitative methods) that exists on the question of whether geographical variations in HIV are mere reflections of the distribution of individual risk factors and correlates of HIV (i.e. compositional-based explanation for the geographical variation in HIV) or are the geographical differences independent of the distribution of individual determinants (i.e. contextual interpretation of the geographical variation in HIV) [1,23–25]. Importantly, we do so by utilizing the most recent, large, nationally representative, multilevel dataset from Tanzania on objectively diagnosed HIV.

Methods

Data

We analysed data from the 2003–2004 Tanzania HIV/AIDS Indicator Survey (AIS), the first nationally representative HIV seroprevalence survey carried out in Tanzania [26]. The AIS utilized a two-stage sampling design, which involved the selection of clusters followed by the systematic sampling of households from mainland Tanzania. Men and women in the selected households, aged 15–49 years were eligible for the survey, which resulted in a response rate of 91% and 96%, respectively. Anonymous HIV testing was performed with informed consent of all eligible survey participants, and the response rates were 84% and 77% for women and men, respectively. Further details on sampling and testing procedures can be found in the final report of the AIS survey [26] pp. 3–6.We also considered a regional-level socioeconomic indicator (regional percentage poverty), which was obtained from the 2000/2001 Tanzania Household Budget Survey [27]. After excluding subjects missing data on variables of interest (see Table 1), the final sample size used in this analysis was 8010 men and women.

Outcome

The outcome variable was a binary variable indicating HIV serostatus. Blood samples were tested using the Vironostika Uniform enzyme-linked immunosorbent assay (ELISA) tests (Vironistika HIV Uniform 2 Ag/Ab and Uni-Form 2 plus O; Organon, Boxtel, the Netherlands).

Confirmatory Western blot tests were further performed on discrepant samples using the INOLIA HIV confirmation Western blot kit (Innogenetics, Gent, Belgium) [26].

Individual covariates

At the individual level several variables were considered, which included socioeconomic status represented by four separate variables: household standard of living index, educational attainment, occupational status, and place of residence. The household standard of living index was defined in terms of the ownership of material possessions and assets, which has been shown to be a reliable and valid measure of household material wellbeing [28]. Each individual was assigned a standard of living score based on a linear combination of the scores for different items that were recorded for the household in which the person resided and weighted according to a factor analysis procedure. The weighted scores were divided into quintiles (poorest, poorer, middle, richer and richest) for the analytical models [29]. Educational attainment was categorized as no education, primary school education, and secondary education or more. Occupational status was specified as unemployed, professional job, agricultural labor, and manual labor. Place of residence was categorized as capital city, small city, town, and rural area. Sociodemographic characteristics including age, religion and marital status were also considered as covariates in the analysis.

We also included biological and behavioral characteristics that are known to increase individuals' HIV susceptibility, and characteristics that might also vary across neighborhoods and regions. These included the presence of STI or genital discharge, condom use at last sexual contact, lifetime number of partners, alcohol use at last sexual contact, and perceived risk of contracting HIV. Previous HIV testing was also included as a proxy for previous HIV diagnosis as the survey did not collect this data.

Defining residential contexts

We defined geographical contexts at two levels: neighborhoods and regions. The primary sampling unit was used as a proxy for individuals' immediate residential context. Primary sampling units in the AIS were primarily villages or clusters of villages in rural areas, and wards in the context of urban areas. We also considered an additional macro context capturing the broader socioeconomic, political and cultural context within which individuals and neighborhoods operate. We included the level of poverty in a neighborhood or region as a summary variable capturing the overall importance of the socioeconomic context of a place. At the neighborhood level, we included the proportion of households in the lowest household standard of living index quintile, whereas regional poverty was the proportion of individuals living below the basic needs poverty line at the time of the survey [27]. The region of Manyara was assigned the mean poverty level of the other 20 regions, because it was not yet in existence at the time the Household Budget Survey was conducted in 2001–2002.

Data analysis

Multilevel statistical modelling techniques were used to partition the variation in HIV to different levels. The substantive as well as technical relevance of multilevel statistical procedures are well known [25,30–33]. The study had a three-level hierarchic structure with 8010 individuals (level 1), within 345 neighborhoods (level 2), within 21 regions (level 3). Given the hierarchical structure of the sample and the binary outcome, a logistic multilevel modelling approach was adopted. We estimated three models of the following specification with a binary response (y, diagnosed as HIV positive or not for individual i living in neighborhood j in region k of the form: $\pi_{ijk} : y_{ijk} \sim \text{Bernoulli}(1, \pi_{ijk})$. For model 1, the probability π_{ijk} was related to an overall mean and a random effect for neighborhood and region level, by a logit link function as:

$$\begin{aligned}\text{logit}(\pi_{ijk}) &= \log\left(\frac{\pi_{ijk}}{(1-\pi_{ijk})}\right) \\ &= \beta_0 + u_{0jk} + v_{0k}\end{aligned} \quad \text{(model 1)}$$

The parameter β_0 estimates the mean log odds of being HIV positive across the sample. Meanwhile, the parameters u_{0jk} and v_{0k} represent the random differentials (from the overall mean) at the neighborhood and regional level, respectively. These random differentials are assumed to have an independent and identical distribution with variance estimated in the neighborhood (σ^2_{u0}) and regional (σ^2_{v0}) level. The variance estimates at model 1 estimates the unconditional or unadjusted variation that is attributable to neighborhoods and regions. To model 1 we then added individual-level covariates to the fixed part of the model as:

$$\begin{aligned}\text{logit}(\pi_{ijk}) &= \log\left(\frac{\pi_{ijk}}{(1-\pi_{ijk})}\right) \\ &= \beta_0 + \beta X + u_{0jk} + v_{0k}\end{aligned} \quad \text{(model 2)}$$

where βX represents the vector of regression coefficients associated with a vector of individual-level independent variables (X). Model 2 re-estimates the variance at the neighborhood $\left(\sigma^2_{u0}\right)$ and regional $\left(\sigma^2_{v0}\right)$ level after adjusting for the compositional make-up of the neighborhoods and regions. These estimates provide evidence for the presence of geographical variation that is over and above that which can be attributed to the distribution of observed individual factors. In addition, we also estimated posterior residuals at the regional level using model 1 and model 2 in order to estimate the unadjusted and adjusted prevalence of HIV in the different regions of Tanzania. To model 2 we then added contextual variables measured at the neighborhood and region, separately as:

$$\begin{aligned}\text{logit}(\pi_{ijk}) &= \log\left(\frac{\pi_{ijk}}{(1-\pi_{ijk})}\right) \\ &= \beta_0 + \beta X + \alpha W + u_{0jk} + v_{0k}\end{aligned} \quad \text{(model 3)}$$

with the parameter α giving an estimate of the change in the response for a unit change in the neighborhood or regional poverty variable. Penalized quasi-likelihood approximation with a second order Taylor linearization procedure was used to estimate all models [34].

This study was approved by the Institutional Review Board of the Harvard School of Public Health.

Results

Table 1 provides the frequency as well as the prevalence of HIV by individual-level covariates showing substantial sample sizes as well as considerable patterning in HIV by individual covariates. Figure 1 maps the crude prevalence of HIV across the 21 Tanzanian regions, with a natural break algorithm used to create groupings. The map shows a distinct geographical patterning of HIV prevalence in Tanzania before controlling for the spatial distribution of individual correlates and risk factors. The prevalence of HIV is higher in two of the south-western regions, the capital city and two northern regions.

Figure 2 shows the amount of variation attributable to neighborhoods and regions before (model 1) and after (model 2) adjusting for individual covariates. Before accounting for the

differential geographical distribution of the individual covariates, 6% of the total variation in HIV was attributable to regions and 20% was attributable to neighborhoods. More than a quarter of the total variation was thus at the geographical level. Accounting for individual covariates did not explain any of the regional variation in HIV, but did account for some of the variation in HIV at the neighborhood level. Neighborhood variation in HIV prevalence was, however, still substantial and statistically significant, accounting for 14%of the total variation in HIV. Of the two geographical levels, neighborhoods seem to matter considerably more than regions in terms of influencing the patterns of HIV prevalence.

Table 2 presents the model-based prevalence of HIV (along with the rank) for each of the 21 regions before and after adjusting for individual covariates. In unadjusted models, the two southern border regions of Iringa and Mbeya had the highest HIV prevalence at 15.5% (95% CI 14.9–16.1%) and 14.3% (95% CI 13.7–14.9%), respectively, followed by the capital city Dar es Salaam at 12.3%(95% CI 11.8–13.0%). The Kigoma region had the lowest HIV prevalence of all the 21 regions at 1.6% (95%CI 1.0–2.2%). Similar patterns were noted for the adjusted model-based prevalence, with the Iringa and Mbeya regions having the highest prevalence at 6.7% (95% CI 6.1–7.3%) and 5.9% (95% CI 5.3–6.5%), respectively. Notably the Kagera region, where the HIV epidemic was first observed in Tanzania more than 20 years ago had one of the lowest crude and adjusted model-based prevalences at 3.9% (95% CI 3.3–4.5%) and 2.6% (95% CI 2.0–3.2%), respectively. In adjusted models, Kigoma had the lowest prevalence at 1.6% (95% CI 1.0–2.2%). We should note that in the adjusted models, the prevalence pertains to the reference category (i.e. rural, currently married, Moslem men aged 40 years and above, with no education, employed in agriculture and in the poorest standard of living quintile, who did not use a condom at their last sexual encounter, did not have STI, had one lifetime partner, did not consume alcohol during their last sexual encounter and had never previously been tested for HIV). The unadjusted and adjusted prevalence, therefore, cannot be directly compared, because the reference category in the two models is very different. Consequently, we also ranked districts on their predicted HIV prevalence from unadjusted and adjusted models. The rank correlation between the two was high ($r = 0.58$, $P = 0.005$), suggesting that the regional distribution of individual covariates only moderately altered the regional geography of HIV.

We observed a statistically significant inverse association between area-level poverty and individual odds of being HIV positive at both the neighborhood (OR 0.24, 95% CI 0.09–0.61) and regional (OR 0.97, 95% CI 0.95–0.99) levels. Residual variation at both the neighborhood and regional level remained, suggesting the potential influence of other contextual variables. We also examined interactions between neighborhood and regional poverty and individual socioeconomic variables, and none were statistically significant and substantial. We also tested for a contextual effect of neighborhood-level and regional-level education, and these two were not substantial and were statistically insignificant.

Discussion

This study investigated the extent to which geographical variation in HIV in Tanzania is a reflection of the differential distribution of individual risk factors and correlates. We found that neighborhood and regional variation in HIV in Tanzania was not caused by the differential distribution of individual correlates and risk factors. Rather, our study suggests the potentially independent influence of neighborhoods and regions on the distribution of HIV in Tanzania. Whereas levels of poverty at the neighborhood and regional level were independent predictors of HIV, substantial geographical variation remained unexplained. Further systematic research is required to explore the specific mechanisms operating at the level of neighborhoods and regions that may be pertinent for explaining the substantial geographical variation in HIV. We

now discuss our finding within the larger context of what is known about the geographical variation in Tanzania.

More than 20 years ago, the north-western region of Kagera was the epicentre of the HIV epidemic in Tanzania. The first AIDS cases were reported there in 1983, and the first HIV seroprevalence study conducted in 1987 estimated the prevalence to be 24.2%in Bukoba urban district, one of the high-prevalence areas in the region [36]. The explosion of the HIV epidemic in that region was mainly attributed to the mobility of the Tanzania People's Defence Force as they helped wage a liberation struggle in neighboring Uganda [12]. Conflict, and the destabilization arising from it, has been identified as a key driver of the rapid spread of HIV in sub-Saharan Africa [37,38]. In the intervening years, many HIV prevention initiatives were undertaken in Kagera to stem the epidemic. The prevalence as well as the incidence of HIV in Kagera has shown a steady decline from the mid-1980s to the present time, with the most recent prevalence estimated at 3.9%. This trend is partly the result of changes in people's sexual behaviors over time as a result of the concentrated intervention efforts in the region [36]. The fact that Kagera no longer has the highest HIV prevalence in the country and that the epidemic has now moved across the country to the southern regions of Mbeya and Iringa points to the complex and dynamic nature of the epidemic, and indicates that there is something about context that fosters the growth and movement of the epidemic within different areas in Tanzania.

Mobility associated with good road networks and transportation has also been identified as being linked to the rapid spread of HIV in southern and eastern Africa [21,22,37]. Our mapping of HIV prevalence in Tanzania shows that the regions with some of the highest prevalence (Mbeya, Iringa, Dar es Salaam, Pwani and Tanga) have transportation networks linking several eastern and southern African countries passing through them. Iringa and Mbeya are also along the border of two equally high HIV prevalent countries, Malawi and Zambia, as are the Kilimanjaro, Arusha, and Tanga regions, which border Kenya. A lot of cross-border travel occurs between these countries for a variety of reasons, including trade, tourism and employment-related migration [39]. The good road network between these countries facilitates transportation by long distance truck drivers, who are one of the high-risk populations in terms of HIV transmission. This is because travel places them and other mobile populations in close proximity to high-risk sexual networks, which in turn increases their chances of acquiring HIV and spreading within their sexual networks. Other community-based studies in Tanzania have found that high levels of economic activity, proximity to major roads and travel are associated with a higher prevalence of HIV, which may also partly explain the higher prevalence observed in neighborhoods that are wealthier. It is possible that individuals living in neighborhoods with higher HIV prevalence are part of wider social and sexual networks, which also increase their chance of being exposed to HIV [18,40].

Mbeya, Iringa, Dar es Salaam and Kilimanjaro are regions with the highest levels of socioeconomic wellbeing, yet, paradoxically, they have the highest prevalence of HIV. Studies in communities in the Mwanza region of Tanzania have shown that HIV prevalence increases with increasing social and economic activity in an area, which is related to mobility and involvement in high-risk and multiple sexual networks that may result from greater amounts of disposable income [17,19]. Our findings of high HIV prevalence in wealthier neighborhoods and regions may be attributed to these factors. This may also be a potential explanation for the relatively high prevalence observed in the neighboring northern regions of Mwanza and Shinyanga, which have both seen the recent establishment of gold mines. Community-based studies in these mining areas found a high HIV prevalence among the communities surrounding the mines, especially among women [41].

The considerable systematic variation in HIV at the neighborhood level clearly merits further research attention. One could conceptualize that both structural as well as normative pathways through local residential contexts may influence the prevalence and incidence of HIV. One counterintuitive finding at the neighborhood level (similar to what was observed at the regional as well as the individual level) was structural factors (e.g. economic resources, education, availability and accessibility of health services, poverty and affluence) that were associated with HIV in a paradoxical manner; neighborhoods with higher levels of economic wellbeing were more likely to have a higher prevalence of HIV. It is possible that normative factors, such as cultural norms, attitudes towards health behaviors, social control, and neighborhood social cohesion [42–44] may be more important protective factors.

The following two caveats need to be considered when interpreting the study findings. First, given the cross-sectional nature of the data, we were unable to trace the trajectory of the HIV epidemic in any of the neighborhoods or regions, and are thus limited to describing our findings based on historical evidence of the HIV epidemic in Tanzania. Second, it is possible that unmeasured individual factors may partly account for the contextual differences observed in this study.

In summary, this study, to our knowledge, is the first to examine systematically and simultaneously the effects of context and composition on the geographical distribution of HIV in Tanzania. Whereas our study suggests strong evidence for independent contextual variation in HIV, further studies, qualitative and quantitative, are required to understand the specific aspects of places that influence the distribution of HIV. We emphasize the need to adopt both a group-based and a place-based approach, as opposed to the dominant high-risk group approach, for understanding the epidemiology of HIV as well as for developing HIV intervention activities.

Acknowledgments

The authors acknowledge the support of Macro International (www.measuredhs.com) for providing access to the 2003/2004 Tanzania HIV/AIDS Indicator Survey data. Authors' contributions: S.V.S. conceived the study, designed the analysis, and contributed to the interpretation of the results and writing of the manuscript. W.M. led the data analysis, interpretation and writing of the manuscript. F.E. and S.K. contributed to the interpretation of the results and editing of the manuscript. All authors reviewed and approved the final manuscript.

Sponsorship: S.V.S. is supported by a National Institutes of Health Career Development Award (NHLBI 1 K25 HL081275).

References

1. Kawachi I, Subramanian SV. Neighbourhood influences on health. J Epidemiol Community Health 2007;61:3–4. [PubMed: 17183006]
2. Diez Roux AV. Investigating neighborhood and area effects on health. Am J Public Health 2001;91:1783–1789. [PubMed: 11684601]
3. Kawachi, I.; Berkman, LF., editors. Neighborhoods and health. New York: Oxford University Press; 2003.
4. Berkman, LF.; Kawachi, I., editors. Social epidemiology. New York: Oxford University Press; 2000.
5. Subramanian SV, Belli P, Kawachi I. The macroeconomic determinants of health. Annu Rev Public Health 2002;23:287–302. [PubMed: 11910064]
6. Sampson RJ. The neighborhood context of well being. Perspect Biol Med 2003;46:S53–S64. [PubMed: 14563074]
7. Diez Roux AV. Investigating neighborhood and area effects on health. Am J Public Health 2001;91:1783–1789. [PubMed: 11684601]
8. O'Campo P. Invited commentary: Advancing theory and methods for multilevel models of residential neighborhoods and health. Am J Epidemiol 2003;157:9–13. [PubMed: 12505885]

9. Pickett KE, Pearl M. Multilevel analyses of neighborhood socioeconomic context and health outcomes: a critical review. J Epidemiol Community Health 2001;55:111–122. [PubMed: 11154250]
10. Auvert B, Buve A, Ferry B, Carael M, Morison L, Lagarde E, et al. Ecological and individual level analysis of risk factors for HIV infection in four urban populations in sub-Saharan Africa with different levels of HIV infection. AIDS 2001;15:S15–S30. [PubMed: 11686462]
11. Carael M, Holmes KK. Dynamics of HIV epidemics in sub-Saharan Africa: introduction. AIDS 2001;15(Suppl 4):S1–S4.
12. Lugalla J, Emmelin MAC, Mutembei AK, Comoro CJ, Killewo JZJ, Kwesigabo G, et al. The social cultural contexts of HIV/AIDS transmission in the Kagera region, Tanzania. J Asian African Stud 1999;34:377–402.
13. Campbell C, Williams B, Gilgen D. Is social capital a useful conceptual tool for exploring community level influences on HIV infection? An exploratory case study from South Africa. AIDS Care 2002;14:41–54. [PubMed: 11798404]
14. Kaufman CE, Clark S, Manzini N, May J. Communities, opportunities, and adolescents' sexual behavior in KwaZulu-Natal, South Africa. Stud Fam Plann 2004;35:261–274. [PubMed: 15628784]
15. Agyei-Mensah S. The HIV/AIDS epidemic in sub-Saharan Africa: homogeneity or heterogeneity? Nor J Geography 2005;59:14–25.
16. Whiteside A. Poverty and HIV/ADS in Africa. Third World Quarterly 2002;23:313–332.
17. Boerma JT, Urassa M, Nnko S, Ng'weshemi J, Isingo R, Zaba B, Mwaluko G. Sociodemographic context of the AIDS epidemic in a rural area in Tanzania with a focus on people's mobility and marriage. Sex Transm Infect 2002;78:i97–i105. [PubMed: 12083453]
18. Boerma JT, Urassa M, Senkoro K, Klokke A, Ngweshemi JZ. Spread of HIV infection in a rural area of Tanzania. AIDS 1999;13:1233–1240. [PubMed: 10416528]
19. Bloom SS, Urassa M, Isingo R, Ng'weshemi J, Boerma JT. Community effects on the risk of HIV infection in rural Tanzania. Sex Transm Infect 2002;78:261–266. [PubMed: 12181463]
20. Clift S, Anemona A, Watson-Jones D, Kanga Z, Ndeki L, Changalucha J, et al. Variations of HIV and STI prevalences within communities neighbouring new goldmines in Tanzania: importance for intervention design. Sex Transm Infect 2003;79:307–312. [PubMed: 12902582]
21. Tanser F, Lesueur D, Solarsh G, Wilkinson D. HIV heterogeneity and proximity of homestead to roads in rural South Africa: an exploration using a geographical information system. Trop Med Int Health 2000;5:40–46. [PubMed: 10672204]
22. Wawer MJ, Serwadda D, Musgrave SD, Konde-Lule JK, Musagara M, Sewankambo NK. Dynamics of spread of HIV-I infection in a rural district of Uganda. BMJ 1991;303:1303–1306. [PubMed: 1747672]
23. Kawachi I, Subramanian SV, Almeida-Filho N. A glossary for health inequalities. J Epidemiol Community Health 2002;56:647–652. [PubMed: 12177079]
24. Moon, G.; Subramanian, SV.; Jones, K.; Duncan, C.; Twigg, L. Area-based studies and the evaluation of multilevel influences on health outcomes. In: Bowling, A.; Ebrahim, S.; Berkshire, UK., editors. Handbook of health research methods: investigation, measurement and analysis. Berkshire, UK: Open University Press; 2005. p. 266-292.
25. Subramanian, SV.; Glymour, M.; Kawachi, I. Identifying causal ecologic effects on health: potentials and challenges. In: Galea, S., editor. Macrosocial determinants of population health. New York: Springer Media; 2007.
26. Tanzania HIV/AIDS indicator survey 2003–04. Calverton, MD, USA: TACAIDS, NBS and ORC Macro; 2005. Tanzania Commission for AIDS (TACAIDS), National Bureau of Statistics (NBS), and ORC Macro; p. 1-131.
27. National Bureau of Statistics (NBS). Household Budget Survey 2001/02. Dar es Salaam, Tanzania: NBS; 2002. p. 1-188.
28. Filmer, D.; Pritchett, L. Estimating wealth effects without expenditure data, or tears: an application to educational enrollments in states of India. Washington, DC, USA: World Bank Development Research Group Poverty and Human Resources; 1998. World Bank Development Research Group Poverty and Human Resources.
29. Rutstein, SO.; Johnson, K. The DHS wealth index. Calverton, MD, USA: ORC Macro; 2004. MEASURE DHS + (program).

30. Goldstein, H. Multilevel statistical models. Vol. 3rd edn.. London: Hodder Arnold; 2003.
31. Subramanian, SV.; Jones, K.; Duncan, C. Multilevel methods for public health research. In: Kawachi, I.; Berkman, LF., editors. Neighborhoods and health. New York: Oxford University Press; 2003. p. 65-111.
32. Blakely, T.; Subramanian, SV. Multilevel studies. In: Oakes, JM.; Kaufman, JS., editors. Methods in social epidemiology. San Fransisco: Jossey-Bass; 2006. p. 316-340.
33. Subramanian SV. The relevance of multilevel statistical methods for identifying causal neighborhood effects. Soc Sci Med 2004;58:1961–1967. [PubMed: 15020011]
34. Rasbash, J.; Steele, F.; Browne, W.; Prosser, B. A user's guide to MLwiN version 2.0. Bristol, UK: University of Bristol; 2005.
35. Goldstein H, Browne WJ, Rasbash J. Partitioning variation in multilevel models. Understanding Stats 2002;1:223–232.
36. Kwesigabo G, Killewo J, Urassa W, Lugalla J, Emmelin M, Mutembei A, et al. HIV-1 infection prevalence and incidence trends in areas of contrasting levels of infection in the Kagera region, Tanzania, 1987–2000. J Acquir Immune Defic Syndr 2005;40:585–591. [PubMed: 16284536]
37. Buve A, Bishikwabo-Nsarhaza K, Mutangadura G. The spread and effect of HIV-1 infection in sub-Saharan Africa. Lancet 2002;359:2011–2017. [PubMed: 12076570]
38. Mayer J. The geographical understanding of HIV/AIDS in sub-Saharan Africa. Nor J Geography 2005;59:6–13.
39. Crush J, Frayne B, Grant M. Linking migration, HIV/AIDS and urban food security in Southern and Eastern Africa. International Food Policy Research Institute (IFPRI), Southern Africa Migration Project (SAMP), The Regional Network on HIV/AIDS, Livelihoods and Food Security (RENEWAL). 2006
40. Killewo J, Dahlgren L, Sandstrom A. Socio-geographical patterns of HIV-1 transmission in Kagera Region, Tanzania. Soc Sci Med 1994;38:129–134. [PubMed: 8146702]
41. Desmond N, Allen CF, Clift S, Justine B, Mzugu J, Plummer ML, et al. A typology of groups at risk of HIV/STI in a gold mining town in north-western Tanzania. Soc Sci Med 2005;60:1739–1749. [PubMed: 15686806]
42. Sampson RJ, Morenoff JD, Gannon-Rowley T. Assessing 'neighbourhood effects': social processes and new directions in research. Annu Rev Sociol 2002;28:443–478.
43. Sampson RJ, Morenoff JD, Earls F. Beyond social capital: spatial dynamiscs of collective efficacy for children. Am Sociol Rev 1999;64:633–660.
44. Kawachi I, Kim D, Coutts A, Subramanian SV. Commentary: Reconciling the three accounts of social capital. Int J Epidemiol 2004;33:682–690. [PubMed: 15282222]Discussion 700-684

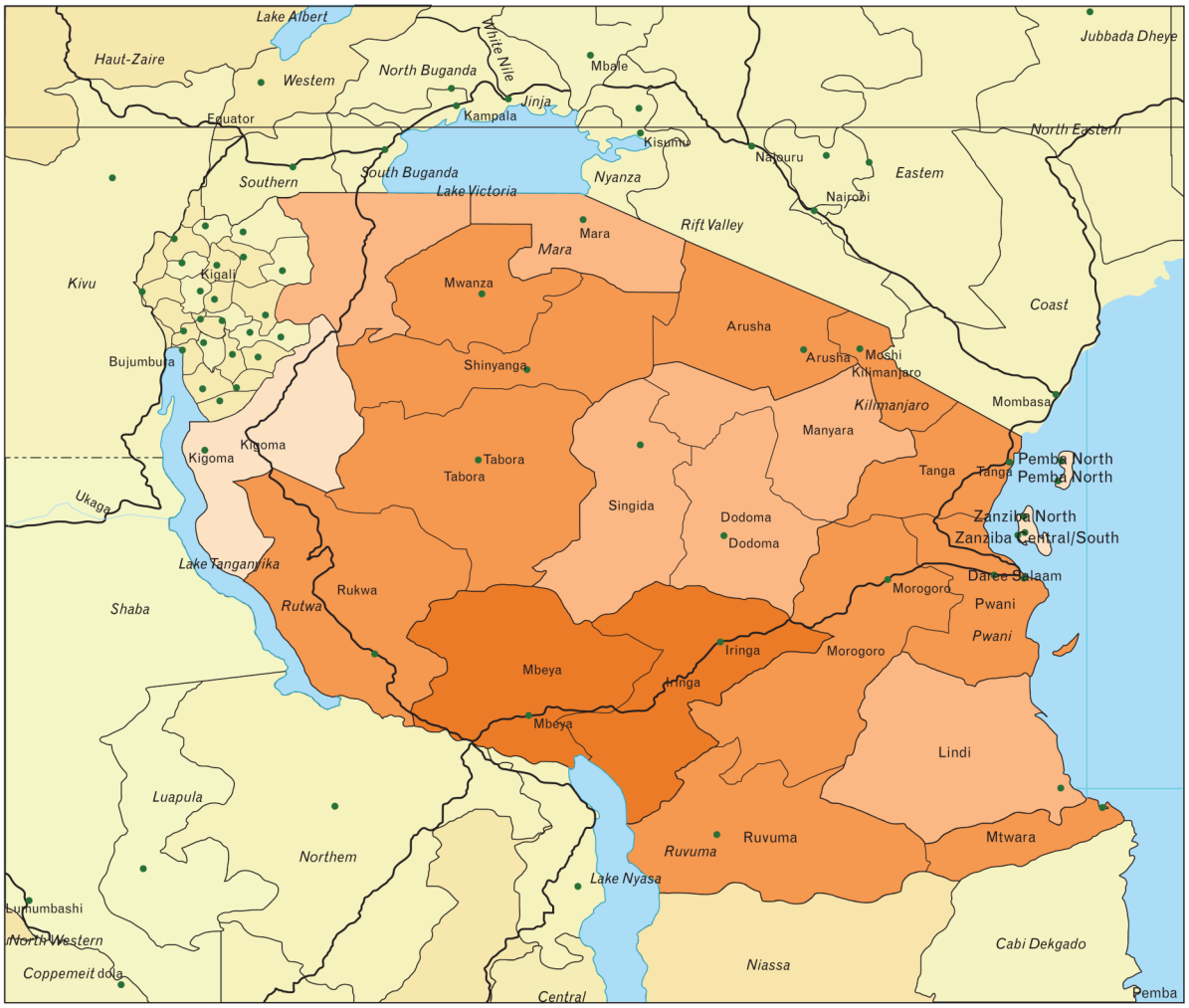

Fig. 1. Map of crude HIV prevalence in Tanzania

Regional HIV prevalence: 0.0–1.6; 1.6–4.8; 4.8–8.1; 8.1–15.5.

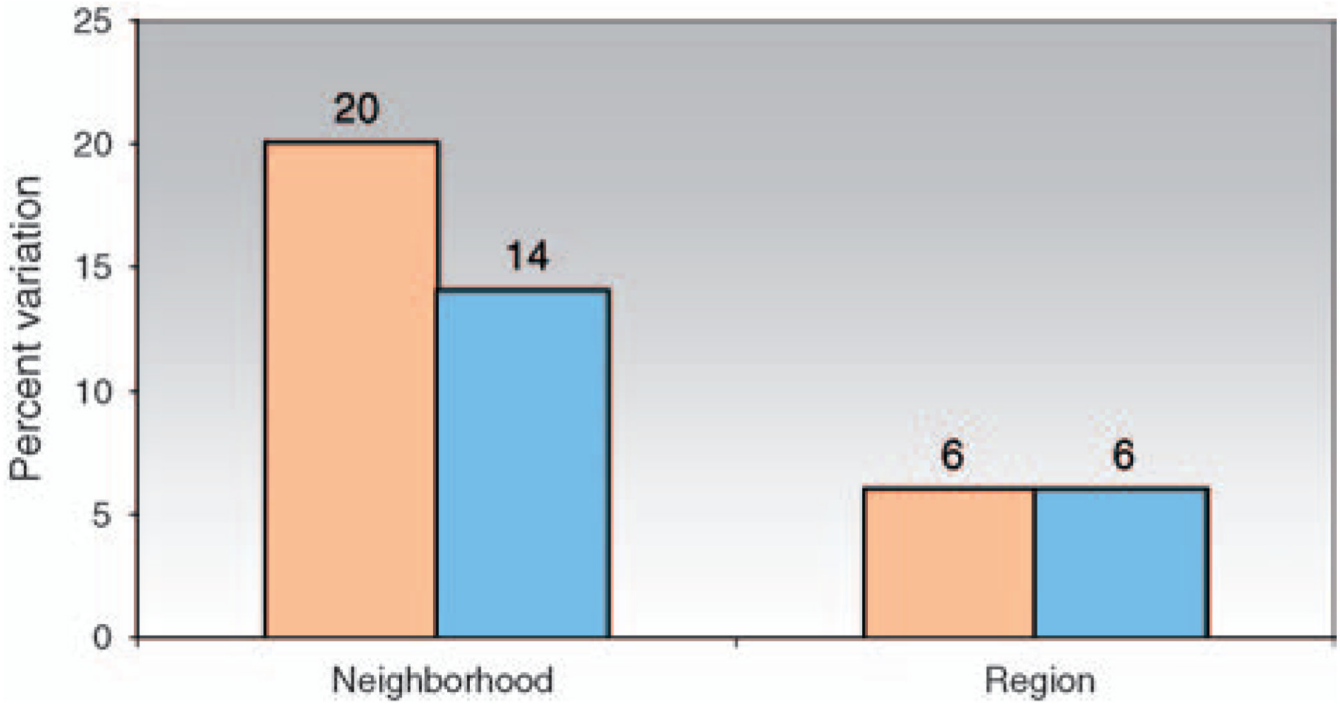

Fig. 2. Percentage variation attributable to neighborhoods and regions before (orange bars) and after (blue bars) adjusting for individual risk factors and correlates

The proportion of variation attributable to different levels was calculated using the 'latent variable' approach to partitioning variation [35]. The adjusted model included age, sex, religion, place of residence, marital status, occupation type, educational attainment, household standard of living index, condom use at last sex, lifetime number of sexual partners, presence of sexually transmitted infection in previous 12 months, alcohol use during last sexual encounter, perception of HIV risk, and previous HIV testing.

Table 1

Descriptive information on sample from 2003/2004 Tanzania HIV/AIDS Indicator Survey[a].

Variable	Participants, *n* (%)	HIV-positive, *n* (%)
Living environment		
Capital city	544 (11.6)	67 (12.3)
Small city	510 (7.5)	61 (13.1)
Town	820 (9.7)	87 (10.2)
Rural	6136 (71.1)	328 (5.6)
Sex		
Female	4581 (55.0)	328 (7.7)
Male	3429 (45.0)	215 (7.0)
Age (years)		
15–19	877 (11.2)	18 (2.4)
20–24	1573 (20.0)	86 (5.5)
25–29	1685 (21.8)	109 (8.0)
30–34	1402 (16.9)	133 (10.3)
35–39	1117 (13.5)	96 (9.4)
40–49	1356 (16.6)	101 (7.7)
Marital status		
Never married	1292 (17.4)	55 (4.4)
Currently married	6071 (74.3)	385 (7.1)
Formerly married	647 (8.3)	103 (16.9)
Religion		
Moslem	2631 (30.6)	196 (8.0)
Catholic	2598 (32.3)	203 (8.6)
Protestant	2111 (26.9)	115 (6.2)
Other	670 (10.1)	29 (4.8)
Educational attainment		
None	1466 (17.4)	73 (5.2)
Primary school	5992 (74.3)	419 (7.6)
Secondary and above	552 (8.2)	51 (10.2)
Occupation type		
Unemployed	603 (8.4)	45 (8.4)
Professional	1413 (20.3)	177 (12.9)
Manual	550 (8.3)	52 (9.3)
Agricultural	5444 (63.0)	269 (5.3)
Household standard of living index		
Richest quintile	1508 (24.5)	173 (11.8)
Richer quintile	1623 (19.4)	148 (9.5)
Middle quintile	1518 (18.7)	92 (6.3)
Poorer quintile	1735 (19.6)	77 (4.6)
Poorest quintile	1626 (17.8)	53 (3.3)
Condom use at last sex		
No	6882 (84.2)	441 (7.0)
Yes	1128 (15.7)	102 (9.9)
Sexually transmitted infection		
No	7716 (96.4)	513 (7.3)
Yes	294 (3.5)	30 (11.0)
Alcohol use during sex		
Respondent only	311 (3.7)	248 (8.0)
Partner only	586 (6.9)	58 (11.4)
Respondent and partner	313 (3.6)	33 (12.3)
Neither	6800 (85.8)	428 (6.9)
Lifetime no. of partners		
One	2271 (27.8)	67 (3.1)
Two to four	3782 (47.3)	307 (8.9)
Five or more	1957 (24.8)	169 (9.4)
Previous HIV test		
No	6730 (82.5)	422 (6.9)
Yes	1280 (17.4)	121 (9.9)
Perception of HIV risk		
No risk	3041 (2.3)	166 (5.9)
Small risk	2326 (28.3)	135 (6.1)
Moderate risk	1260 (15.8)	124 (10.4)
Great risk	674 (8.5)	61 (10.6)
Don't know risk	709 (8.7)	57 (9.9)
Total	8010 (100.0)	543 (7.4)

[a]Prevalence estimates were calculated after applying sampling weights.

Table 2

Model-based crude and adjusted HIV prevalence for Tanzanian regions.

Regions	Sample size (*n*)	Crude prevalence % (95% CI)	Rank	Adjusted prevalence[a] % (95% CI)	Rank
Kigoma	410	1.6 (1.0–2.2)	1	1.8 (1.2–2.4)	1
Manyara	407	2.9 (2.3–3.5)	2	2.7 (2.1–3.3)	4
Lindi	327	3.3 (2.7–3.9)	3	4.6 (4.0–5.2)	17
Singida	394	3.5 (2.9–4.1)	4	3.7 (3.1–4.3)	12
Mara	373	3.7 (3.1–4.3)	5	3.1 (2.5–3.7)	7
Kagera	395	3.9 (3.3–4.5)	6	2.6 (2.0–3.2)	3
Dodoma	357	4.8 (4.2–5.4)	7	3.2 (2.6–3.8)	9
Morogoro	344	5.6 (5.0–6.2)	8	2.3 (1.7–2.9)	2
Ruvuma	429	5.6 (5.0–6.2)	9	2.9 (2.3–3.5)	5
Rukwa	416	6.2 (5.6–6.8)	10	4.0 (3.4–4.6)	13
Shinyanga	368	6.6 (6.0–7.2)	11	5.0 (4.4–5.6)	19
Tanga	323	6.7 (6.1–7.3)	12	3.6 (3.0–4.2)	10
Arusha	312	7.0 (6.4–7.6)	13	3.1 (2.4–3.6)	7
Tabora	488	7.1 (6.5–7.7)	14	4.1 (3.5–4.7)	15
Kilimanjaro	271	7.2 (6.6–7.8)	15	3.0 (2.4–3.6)	6
Pwani	360	7.2 (6.6–7.8)	16	4.5 (3.9–5.1)	16
Mwanza	475	7.5 (6.9–8.1)	17	4.6 (4.0–5.2)	17
Mtwara	369	8.1 (7.5–8.7)	18	4.0 (3.4–4.6)	13
Dar es Salaam	543	12.4 (11.8–13.0)	19	3.6 (3.0–4.2)	10
Mbeya	385	14.3 (13.7–14.9)	20	5.9 (5.3–6.5)	20
Iringa	264	15.5 (14.9–16.1)	21	6.7 (6.1–7.3)	21

The adjusted model included age, sex, religion, place of residence, marital status, occupation type, educational attainment, household standard of living index, condom use at last sex, lifetime number of sexual partners, presence of sexually transmitted infection in previous 12 months, alcohol use during last sexual encounter, perception of HIV risk, and previous HIV testing.