Transmission Rates and Not Sexual Contact Patterns Drive HIV Epidemic Intensity in Africa

Steve E Bellan¹, David Champredon², Alison P Galvani³,⁴, Jonathan Dushoff⁵, Lauren A Meyers⁶,⁷

¹Center for Computational Biology and Bioinformatics, University of Texas at Austin, ²School of Computational Science and Engineering, McMaster University, ³School of Public Health, Yale University, ⁴Ecology and Evolutionary Biology, Yale University, ⁵Department of Biology, McMaster University, ⁶Department of Integrative Biology, University of Texas at Austin, ⁷The Santa Fe Institute

HIV Epidemic Variation in Africa

- SDP is the proportion of couples with any HIV seropositive partners that are serodiscordant.
- SDP varies roughly 40-90% across Africa, in inverse correlation with incidence.
- Hypothesized drivers of epidemic variation should be consistent with SDP variation.

The Serodiscordant Proportion (SDP)

\[ \text{SDP} = \frac{\# \text{ couples} - \# \text{ couples}}{\# \text{ couples} + \# \text{ couples}} \]

What Drives Epidemic Variation?

Biology OR Behavior

Male Circumcision
Corrections
Viral Genetics
Human Genetics

Intercourse-Related
Cotol Frequency
Safe Sex Practices
Type of Intercourse (Vaginal, Anal, Dry)

Sexual Network
Concurrent Partnerships
Partner Switching Rates
Age-Disparate Relationships
Risk Partner Assertivity

affect the transmission rate
affect sexual mixing

Evidence
MINIMAL evidence showing how these vary across countries in relation to prevalence.

Theoretical models show that sexual mixing patterns (like concurrency) exacerbate epidemics.

Sexual networks are difficult to measure. Consequently, studies have relied on hypothetical networks or measured small networks, but do NOT compared large networks across wide regions.

INCONSISTENT CORRELATIONAL evidence for differences in sexual mixing patterns as drivers of country-specific epidemic variation.

Both biological cofactors and sexual network characteristics can affect HIV transmission.

Still, it remains unclear what actually drives the divergent epidemic trajectories.

Research Question

What explains country-level variation in
- epidemic prevalence,
- the serodiscordant proportion,
- and the inverse correlation between them?

Methods: A Couples Transmission Model

We estimated country-specific transmission rates and sexual network characteristics by fitting mathematical models to Demographic and Health Surveys (DHS) from 40,044 couples in 25 African countries.

In our model, stable partners infect each other at transmission rates \( \beta_{\text{within}} \), which reflect biological cofactors and interpartner related behaviors.

Drivers of Variation in the Serodiscordant Proportion

- Most infected couples exhibit a typical trajectory from (+ to +) to (+ to -) (black arrows in Figure 4 on right).
- SDP can be approximated by couple-time spent as (+) divided by the couple-time spent as (-) (+ +)

\[ \text{SDP} = \frac{t_{+}}{t_{-} + t_{+}} \]

- These durations (and thus SDP) are largely determined by the fastest rates out of these states: within-couple transmission \( \beta_{\text{within}} \) and AIDS mortality.

Drivers of Variation in Peak HIV Prevalence

We regressed peak HIV prevalence against transmission rates and sexual mixing coefficients in weighted univariate regressions and in a weighted multivariate regression including all predictors.

Country-level differences in the HIV transmission rate explain not only variation in both SDP and prevalence across SSA, but also—by simultaneously increasing prevalence and reducing SDP—the inverse correlation between them.

Countries may have different transmission rates because of different biological cofactors or interpartner-related behavior, but NOT from different sexual network patterns.

Conclusion

While risky sexual contact patterns contribute significantly to HIV incidence throughout Africa, greater HIV transmission rates, NOT riskier sexual network characteristics, explain why some countries have more severe epidemics.

Acknowledgments

DHS data are available from www.measuredhs.com. We thank M. Daniels for technical guidance; K. Forelle, JRC Pullum, W. Davis, G. Williams, R. Anderson, F. Fossey, A. Forsyth, and J. O’Dea for useful feedback; and O. Spiegel for inspiring the submodel analysis. The International Clinical Trials on Infectious Disease and Data, where this work was initiated, were funded by NIH award R21GM102419 to JRC Pullum and A Wolfe, the African Institute for Mathematical Sciences, and the South African Centre for Excellence in Epidemiological Modelling and Analysis. This work was also supported by a J.S Nancarrow Foundation grant to JD and NIGMS MIDAS grant U01GM087719 to LAM and APF.