Modeling HIV and STI transmission dynamics:
The importance of partnership network structure

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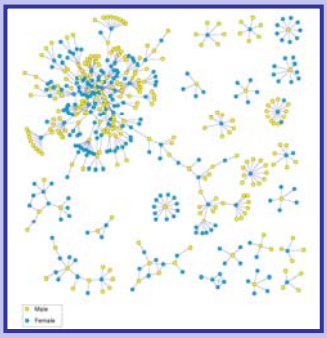

Network Modeling Project:

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Deven Hamilton, Pavel Krivitsky, Julia Palacio-Roman

Motivation for our work:

What explains the massive and persistent disparities in HIV prevalence?

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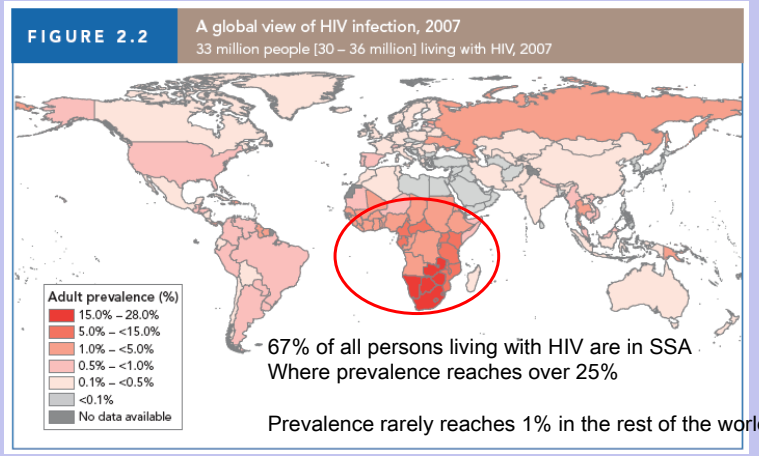
Global disparities in HIV prevalence are well known

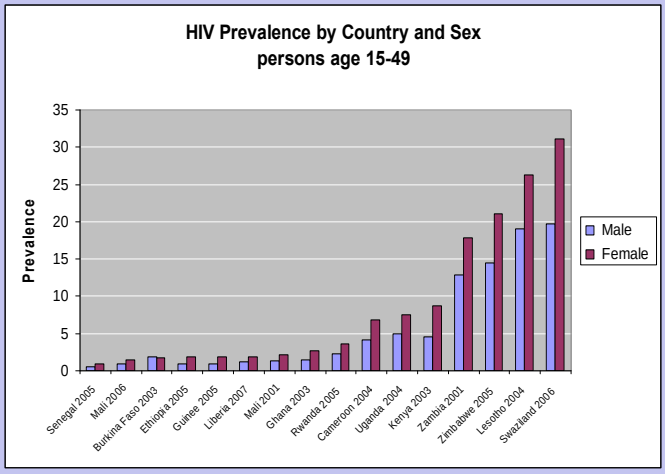


67% of all persons living with HIV are in SSA
Where prevalence reaches over 25%

Prevalence rarely reaches 1% in the rest of the world

Source: UNAIDS 2008 Annual Report on HIV/AIDS

Within sub-Saharan Africa, there is disparity by country



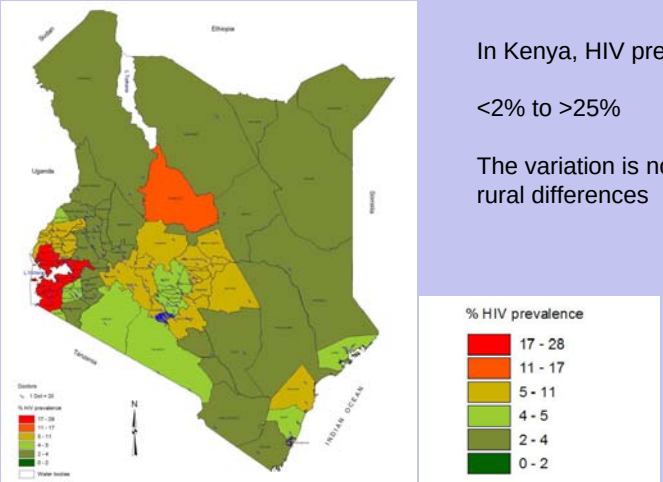
HIV prevalence ranges from

<2% to >30%

across the sub-Saharan region

Source: DHS survey data, http://www.measuredhs.com/hivdata/data/ accessed July 14 2009

And within countries, there is disparity by population subgroups



In Kenya, HIV prevalence varies from

<2% to >25%

The variation is not driven by urban-rural differences

Disparities are also evident across subpopulations in the US

The New York Times
nytimes.com

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July 30, 2008

U.S. Blacks, if a Nation, Would Rank High on AIDS

By LAWRENCE K. ALTMAN

If black America were a country, it would rank 16th in the world in the number of people living with the AIDS virus, the Black AIDS Institute, an advocacy group, reported Tuesday.

HIV prevalence among African Americans is 10-20 times higher than among Whites in the US. And this disparity is also found *within* each risk group – MSM, IDU, and heterosexual men and women.

What explains these disparities?

Potential determinants include:

- Genetic differences – *probably not*
  - All STI show this pattern – bacterial, viral, protozoan – these share a transmission network, not a genetic pathway
  - The variation within and between African countries suggests no common genetics
  - The rapid changes for curable STI in the US not consistent with genetic shifts
- Biological differences – *probably not*
  - Cofactor STIs just push the question back: why the disparities in the cofactor?
  - Circumcision patterns map inconsistently to HIV prevalence
- Traditional risk behavior differences – *probably not*
  - A large literature finds little evidence for # partners, condom use, drug use
    - Globally (e.g., Wellings et al., Lancet 2006)
    - In the US (e.g., Hallfors et al., AJPH 2007)

So what does that leave?

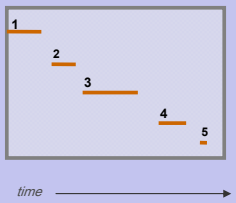
- The transmission network
  - People are not independent in this process
  - You don't get infected from your behavior, you get infected by your partner
- The dynamic structure and connectivity of the network determine
  - The "reachable path" of an infection spreading through the network and
  - The personal risk of infection
- Traditional models assumed connectivity was a function of "contact rates"
  - The more partners people have, the more connected the network is

This is true, but

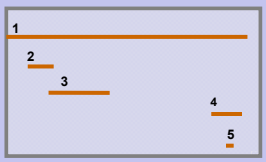
- Connectivity can also emerge without a high contact rate, if people have their partners *concurrently*

What is Concurrency?

Concurrency is the alternative to serial monogamy



Serial monogamous partnerships

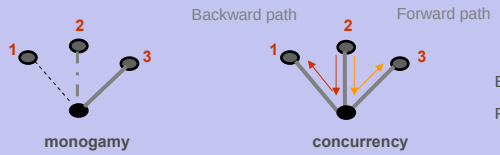


Concurrent partnerships

The difference is not the number of partnerships, but the sequence of start and end dates

Concurrency changes the reachable path, and the velocity of transmission

Removes the protection of sequence over time



Backward path: New chain of infection

Forward path: Less time lost locked in partnership

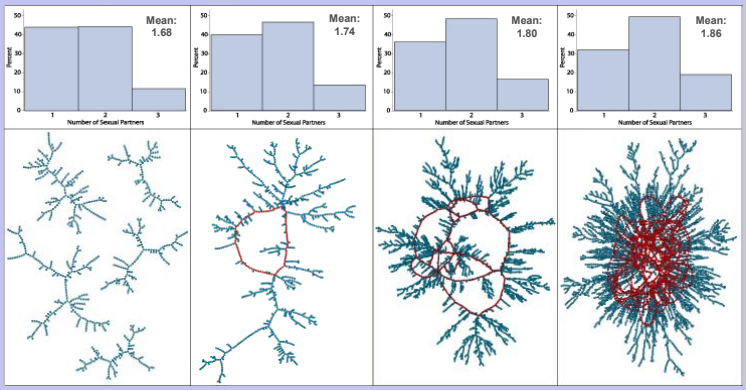
Generates a unique *cross-sectional* network signature:
Larger components, the "concurrency superhighway" (Epstein, 2007)

Concurrency has threshold impacts on network connectivity

Number of Concurrent Partners

Largest components

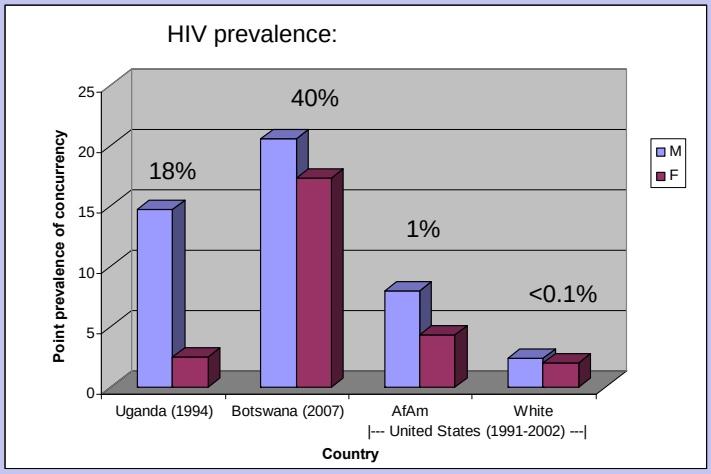
Bicomponents in red



In largest component:	2%	10%	41%	64%
In largest bicomponent:	0	1%	5%	15%

Implications: Hubs, superspreaders & core groups are not required for network connectivity, and small differences in behavior can have big impacts

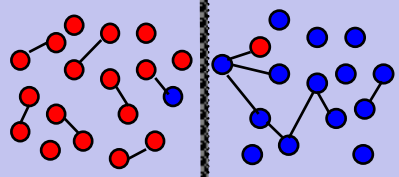
Concurrency differentials line up with HIV disparities



Sources: Uganda – Rakai Sexnet Study; Botswana – Botswana AIDS Indicator Survey III; US – Averages taken from 4 studies: National Health and Social Life Survey (1994), National Surveys of Men & Women (1993, 91) and National Survey of Family Growth (2002)

Network hypothesis for persistent HIV/STI racial disparities in the US

Monogamy retards spread in this group, so prevalence stays low



Concurrency amplifies spread in this group, so prevalence rises

Assortative mixing reduces spread between groups, so a prevalence differential can be sustained over time

Tested this hypothesis in a recently published paper (June 2009)

Concurrent partnerships and HIV prevalence disparities by race:
Linking science and public health practice. *Amer J Pub Health* 2009;99(6):1023-31.
Morris M, Kurth AE, Hamilton DT, Moody J, and Wakefield S for The Network Modeling Group.

- Used the ERGM statistical framework we developed with NIH support

Probability of observing a network (set of relationships) y:

$$P(\mathbf{Y} = \mathbf{y}) = \frac{\exp\{\boldsymbol{\theta}^T \mathbf{g}(\mathbf{y})\}}{c}$$

where: $\boldsymbol{g}$ = vector of network statistics

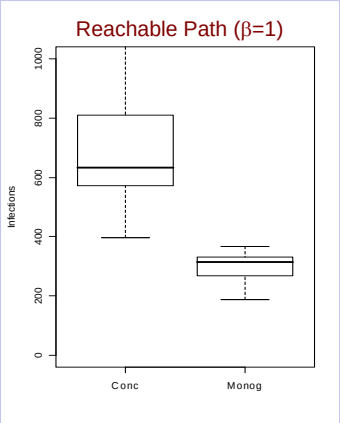
$\boldsymbol{\theta}$ = vector of model parameters

$c = \sum_{\text{all } G} \exp\{\boldsymbol{\theta}^T \mathbf{g}(\mathbf{y})\}$ a normalizing constant

- And the **statnet** computer package we also developed with NIH support
  - Available under GPL to anyone, free of charge (www.statnet.org)

Findings:

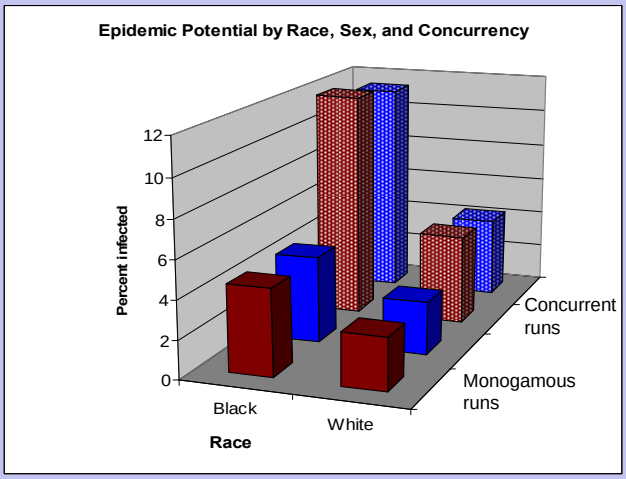
This low level of US concurrency still doubles epidemic potential



It would take a hundred-fold reduction in infectivity to outweigh the effect of concurrency

Findings:

Concurrency differentials triple the racial disparity in epidemic potential



To get a sense of how this happens

- It is worth watching the process run

movie

Acknowledgments and thanks to NIH

- **NICHD and NIDA have supported our research program for many years**
  - R29 HD034957
  - R01 HD38210
  - R01 HD41877
  - R01 DA012831
- **NIH center grants also supported the research environment**
  - Population Centers at Penn State and the University of Washington (NICHD)
  - Center for AIDS Research at UW (NIAID)
- **NIH funded almost all of the survey data used in this presentation:**
  - The Ugandan Sexual Network Study (MJ Wawer, PI)
  - The Thailand Behavioral Research for AIDS Prevention Study (MJ Wawer, PI)
  - The National Health and Social Life Survey (EO Laumann, PI)
  - The National Longitudinal Survey of Adolescent Health (JR Udry, PI)

Thank you

http://statnet.org

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