

# The relationship between concurrent partnerships and HIV transmission

## Overview of the evidence

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### **The Network Modeling Project**

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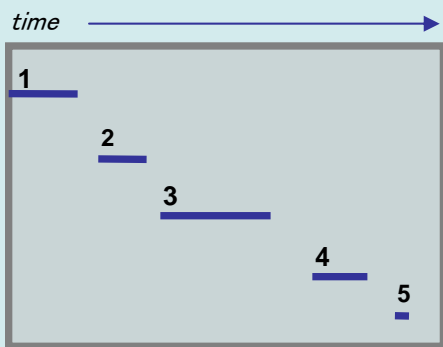
# Overview

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- Definition of concurrency
  - Overlapping partnership intervals
  - What to measure
- How concurrency works (and doesn't work)
- Brief review of origins
  - Dietz & Hadeler
  - W&M, CPH, M&K
- (Even briefer) review of findings
- Where we are now

# Definition

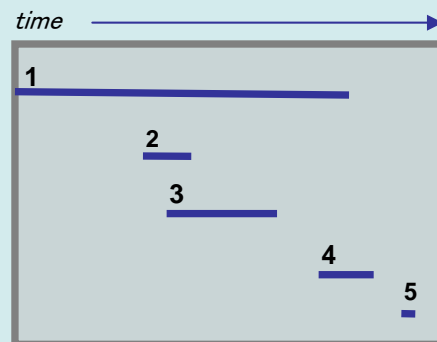
Two partnerships are concurrent if they overlap in time.



Multiple serial partnerships, no overlaps

1 01 01 01 0 1 0

*Current Partner Count*



Concurrent partnerships

1 2 3 2 1 2 1 0 1 0

Same contact rate (5/yr), but the sequence of start and end dates is different

Current partner count for serial monogamy is always 0 or 1

Unique signature of concurrency at individual level:

Cross-sectional degree distribution takes values above 1

# How concurrency works

1. Removes the protection of sequence over time:  
changes the reachable path, and the velocity of transmission



2. Generates a unique *cross-sectional* network signature:  
creates larger components, the “concurrency superhighway” (Epstein, 2007)



# Important features of concurrency

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- Point prevalence
  - Fraction of persons having more than one partner at a moment in time (usually the date of interview)
  - Distribution of the number of partners reported at a moment in time
- Cumulative prevalence (Over some period of time)
  - The fraction of persons reporting concurrency
  - The distribution of the number of concurrencies
    - This is different than with point prevalence
    - Counts can refer to the number of 2-partner concurrencies, 3-partner, etc.
    - Counting 2-partner concurrencies:  $\binom{n}{2}$

# Important features of concurrency

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- Intensity of overlap
  - How long does the concurrency last (duration)
  - How frequent is the back and forth between partners
  
- Gender asymmetry
  - Do men report concurrency more often than women?
  - Not an issue of reporting error
    - Always more men than women with no partners, so this balances
  - This changes the connectivity of the network
    - Asymmetry will lead to less connectivity

# Origins in scientific literature

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- Original insights were about monogamy
  - Dietz and Hadelar (1988)
  - Monogamy traps infection, reducing spread
  
- This led to insights about concurrency
  - Watts and May (1992), the “standing crop” of partners
  - Hudson (1993, 1994) concurrency in Uganda, importance of peak infection
  - Morris and Kretzschmar (1994, 1996) general results

# Effects of concurrency (theory)

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Two levels of effects:

- Individual level
  - Increases risk of being a transmitter
- Population level
  - Increases connectivity of the network

Raises some interesting challenges for measurement

# Individual level effects

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- Concurrency does not increase the risk of infection for the index case (*ceteris paribus*)
  - That risk comes from having more than one partner
  - It is the same whether these partners are serial or concurrent
- So there is no reason to expect a person with concurrent partners to be more likely to be infected

# Individual level effects

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- Concurrency increases the probability that the index case will transmit infection
  - In particular, it increases the risk of infection to the existing partner of the index case
  - This partner would have been gone under serial monogamy
  - They are now the first link in the new “backward chain”
- But it is rare that we enroll partners in an epidemiological study
- So we almost never observe this
  - Exceptions: Koumans et al. (2001) for syphilis, Potterat (1999) for chlamydia

# Individual level effects

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- So what does it mean if we do estimate a positive impact of concurrency on STI status in a traditional epi study?
  - About 50% of the time we do
- It means that concurrency is a proxy for other risk factors
  - Partner's concurrency?
  - Embeddedness in a higher risk section of the network?

# Population level effects

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- Concurrency increases network connectivity
  - How much depends on
    - The intensity of overlap
    - The gender asymmetry in concurrency
- These effects are *\*very\** nonlinear
  - They operate very differently than things that affect the probability of transmission given contact
    - NB: this is what most biomedical prevention interventions affect
  - This is both what makes this so difficult, and what makes for such a viable intervention target

# Population level effects

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- So what would we expect to see empirically?
  - Relationship btwn prevalence and concurrency at the population level?
    - Country level? Subgroup level?
    - Not the usual problems with ecological inference
    - But need to be careful about the non-linearity
  - Problem is timing
    - Infection lasts for a long time
    - So prevalence reflects behavior over a long time span
    - *No one would expect a new vaccination program to correlate to prevalence*

Current behavior may not map to current prevalence  
*But it should map to incidence*

# How does this relate to $R_0$ ?

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- $R_0$  is a population level summary of epidemic potential
  - $E(\text{transmissions})$  from the first infected case
    - There is a threshold at  $R_0 = 1$
    - ***Under (many) simplifying assumptions,  $R_0 = \beta c D$***  , where
      - $\beta$  probability of transmission per contact
      - $c$  number of contacts per time unit
      - $D$  duration of time infected
  - The threshold means that epidemic potential is highly nonlinear
  - Also means that small changes can have large impacts

# How important are the simplifying assumptions?

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- **Look at the dimensional analysis:**

$$\beta cD = \frac{\text{transmission}}{\text{contact}} * \frac{\text{contacts}}{\text{time}} * \text{time}$$

- Implies every contact is independent – i.e., no partnerships
- Might work for vector, water, and airborne infections (malaria, cholera and flu)
- But not for sexually transmitted infections, as contact is often with the same person

# Can we represent partnerships in $R_0$ ?

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- Original formula:

$$\beta cD = \frac{\text{transmission}}{\text{contact}} * \frac{\text{contacts}}{\text{time}} * \text{time}$$

- New formula:

$$\tau pD = \frac{\text{transmission}}{\text{partnership}} * \frac{\text{partnerships}}{\text{time}} * \text{time}$$

... .. where  $\tau = 1 - (1-\beta)^c$

- **This has three important implications**

# First implication:

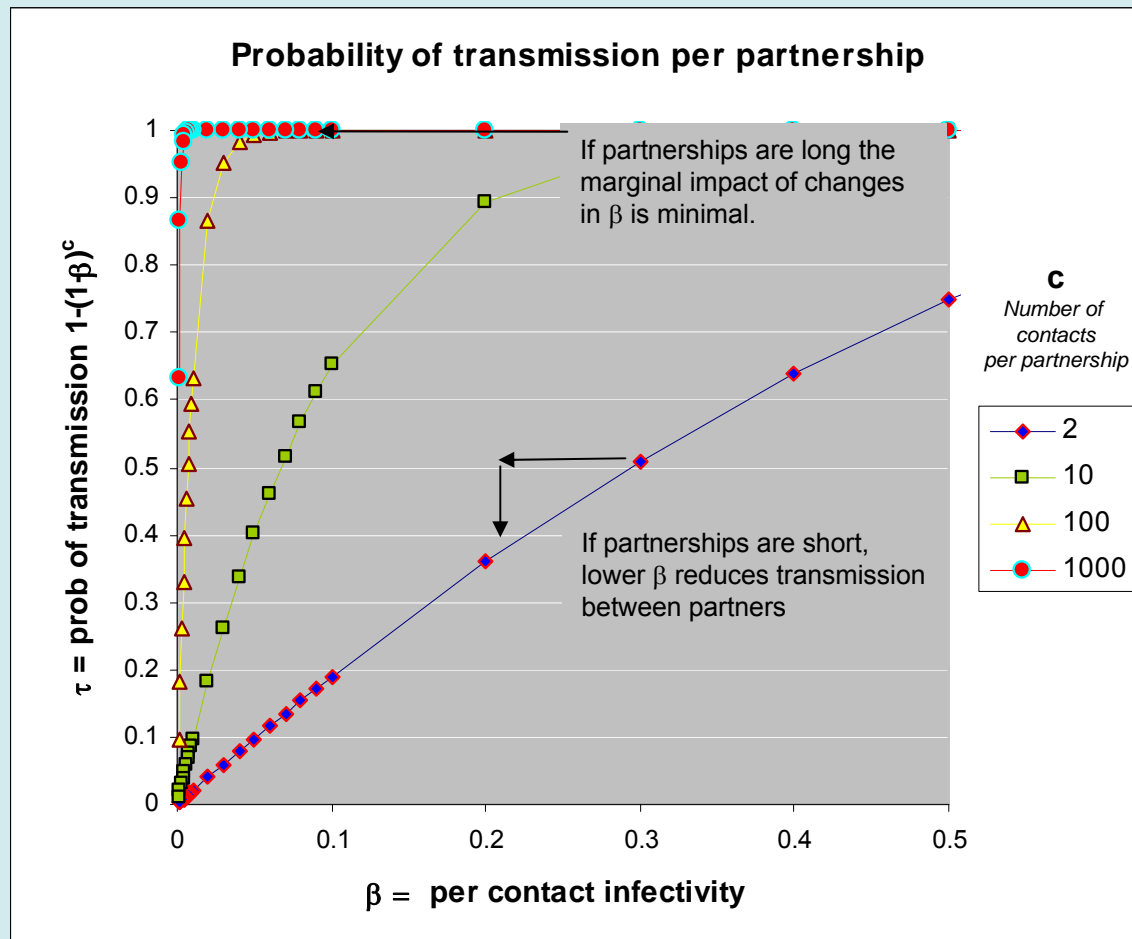
## $\beta$ influences transmission *within* partnerships

The probability of transmission within a partnership is:

$$\tau = 1 - (1-\beta)^c$$

The more contacts in a partnership, the smaller the effect of  $\beta$ .

Reductions in  $\beta$  tend to delay, rather than prevent transmission



## Second implication:

### Partnerships influence the transmission network

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#### The sequence of partnerships defines the “reachable path”

- The *reachable nodes* in a transmission process are not determined by the partnerships at any single point in time
- Nor by the cumulative total over time
- But instead by the *cumulative time ordered path of partnerships*

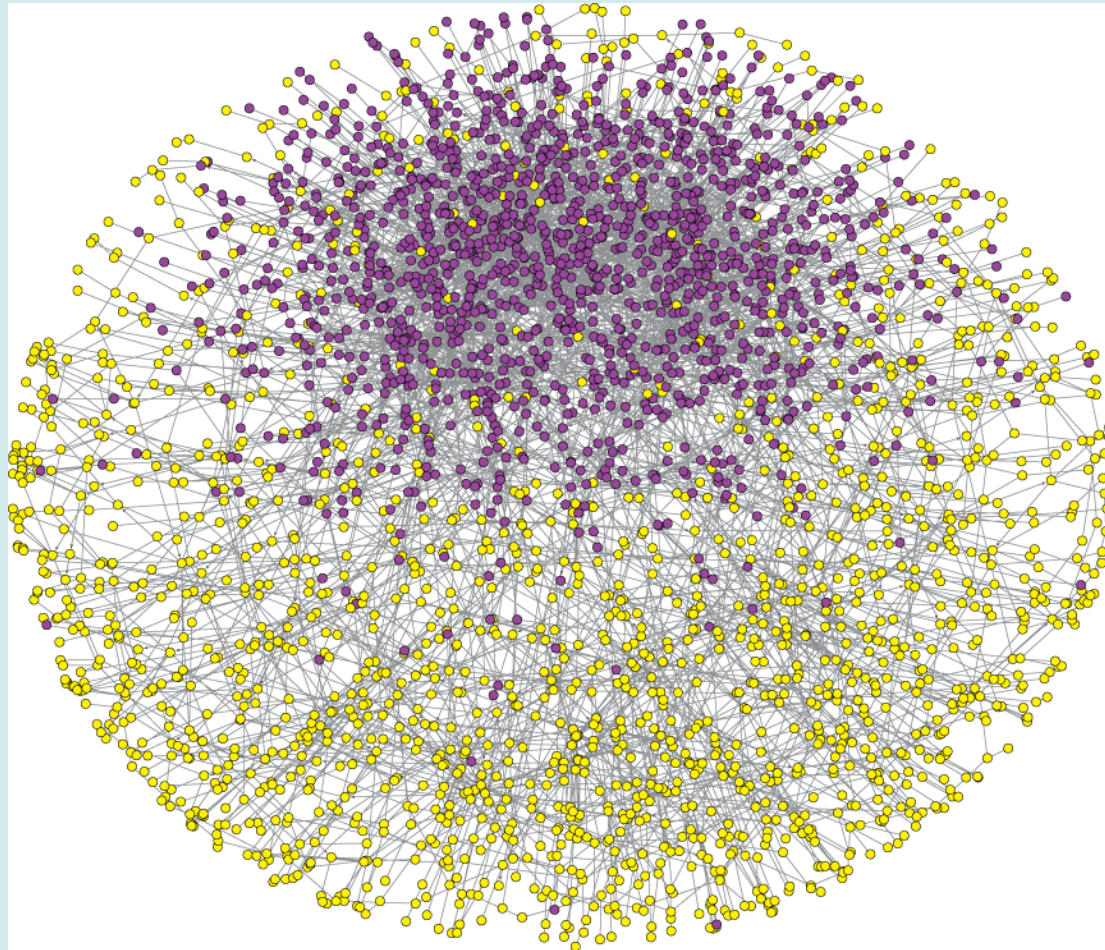
# Consider a large network collapsed over time

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Simulated  
10,000 node  
network

with two  
demographic  
groups

10 years of  
partnership  
activity  
collapsed into a  
single network  
diagram



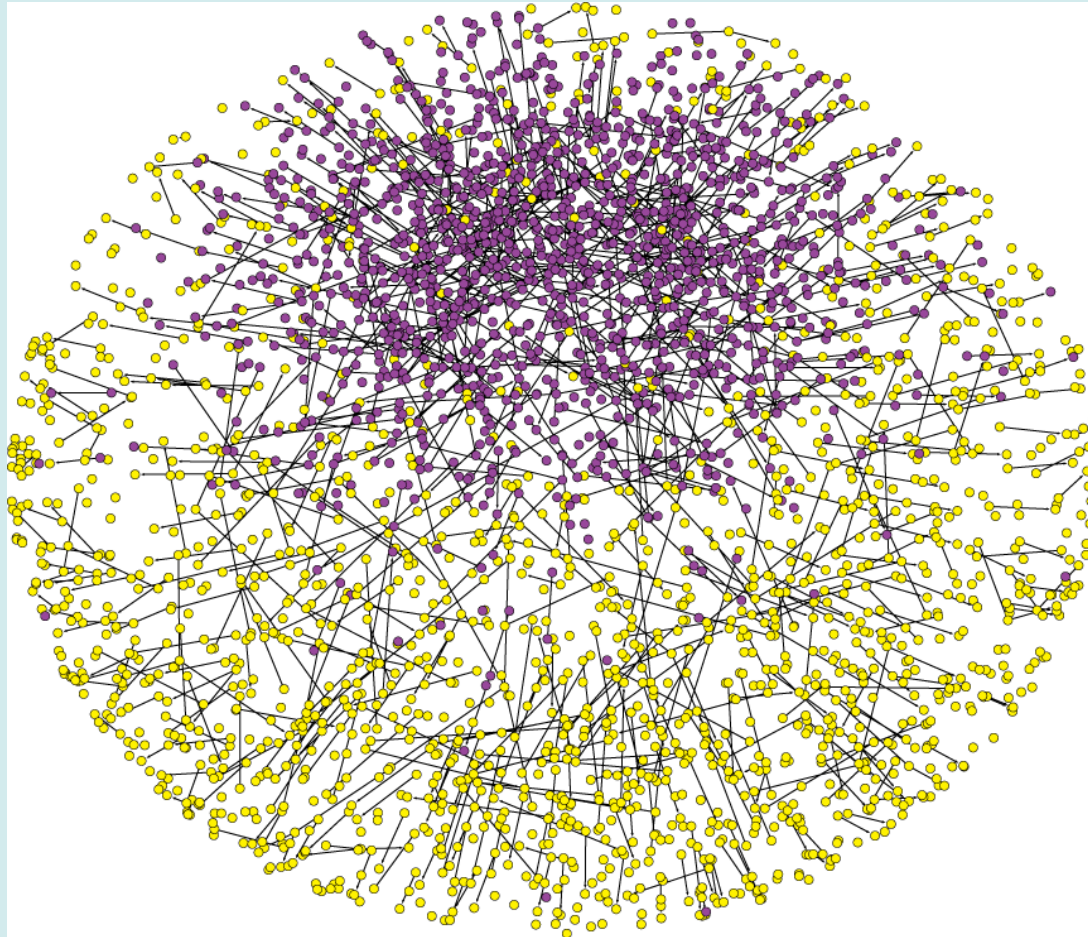
97% of the  
nodes are in  
the cumulative  
connected  
component

# On any single day, the connections are much less dense

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Over 95% of the network components are size 2 or smaller

The largest components are have 5-6 nodes



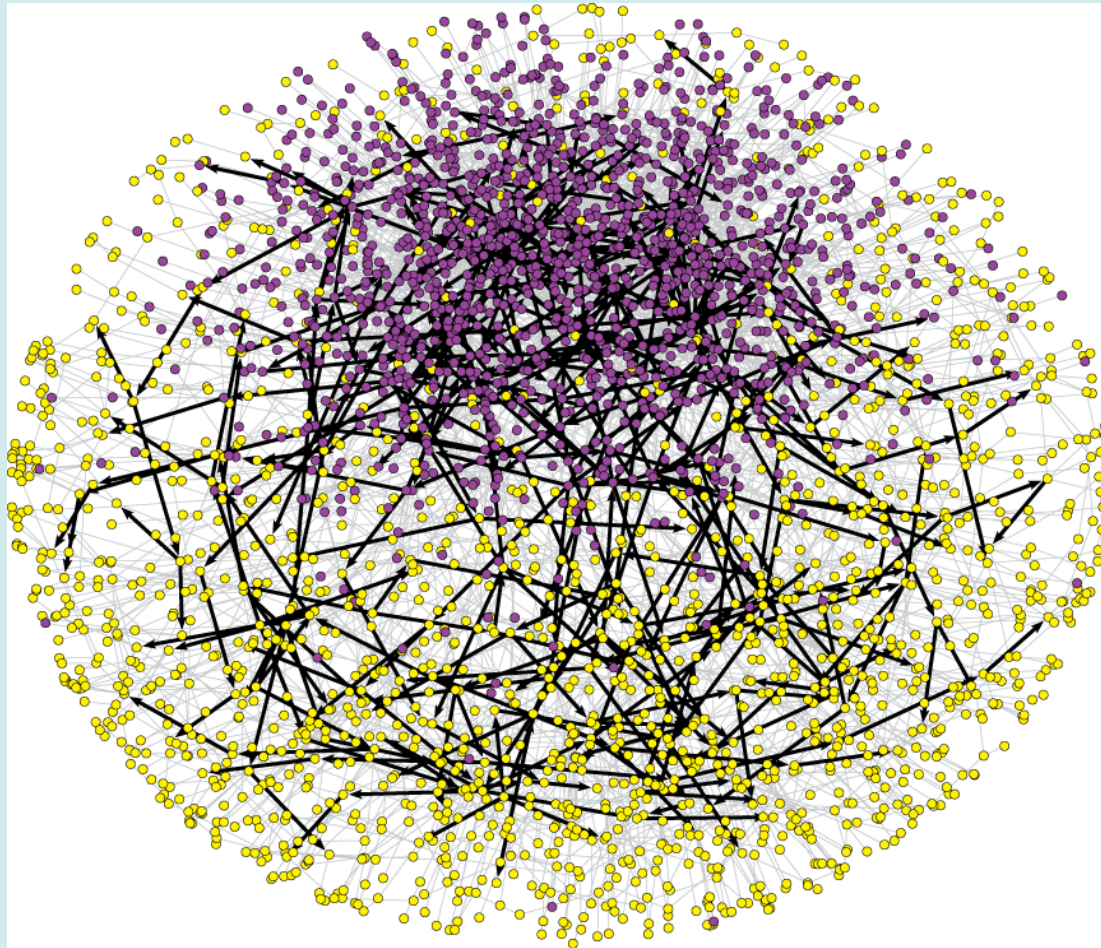
0.06% of the network in the largest component

# The reachable path is different than both of these

Simulate disease spread over 10 years from 10 seeds.

The path traced out by the infection is:

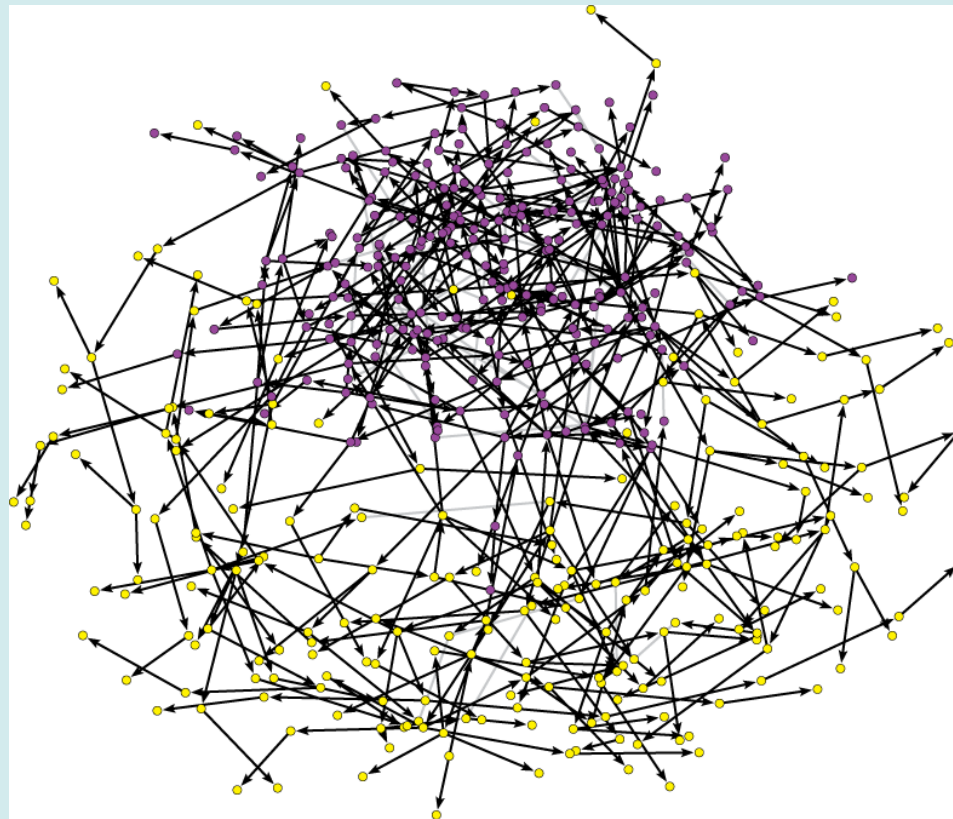
- *Neither all cumulative links*
- *Nor the links at any moment*
- *But the cumulative time-ordered path*



In this case, about 5% of the network is in the reachable path

# Stripping away the uninfected nodes and paths

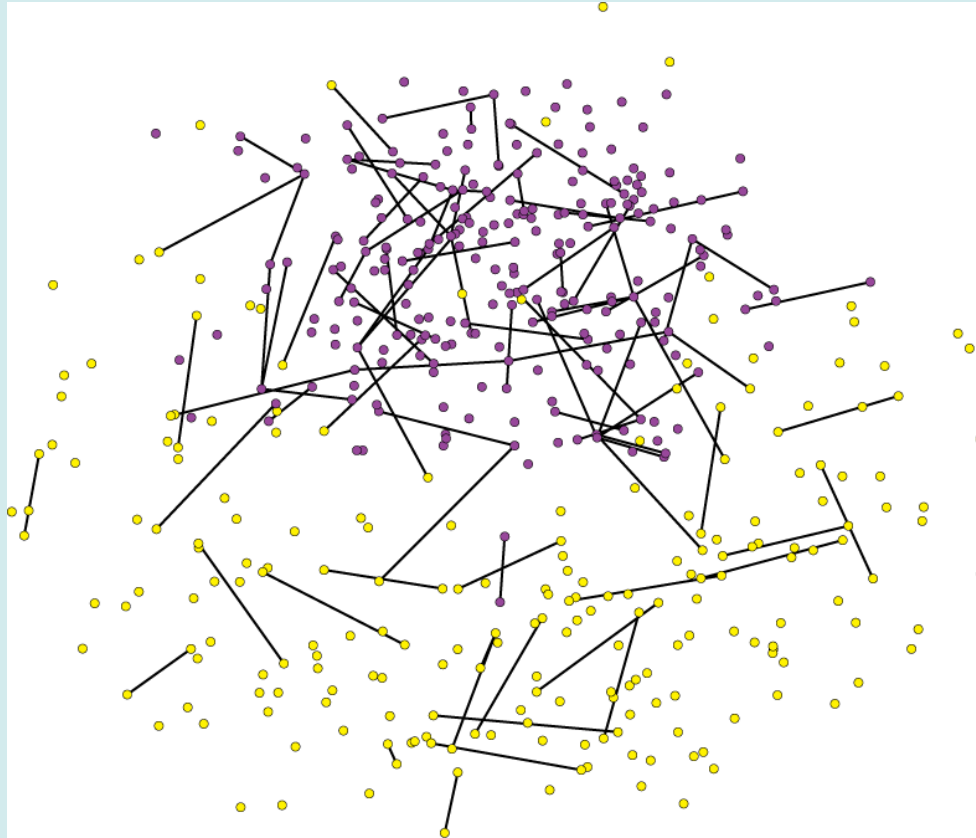
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Reveals how small the infection core is

# And on any single day

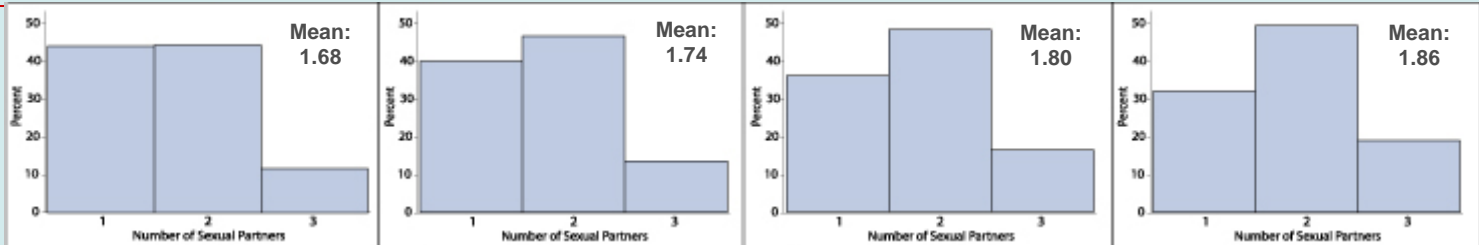
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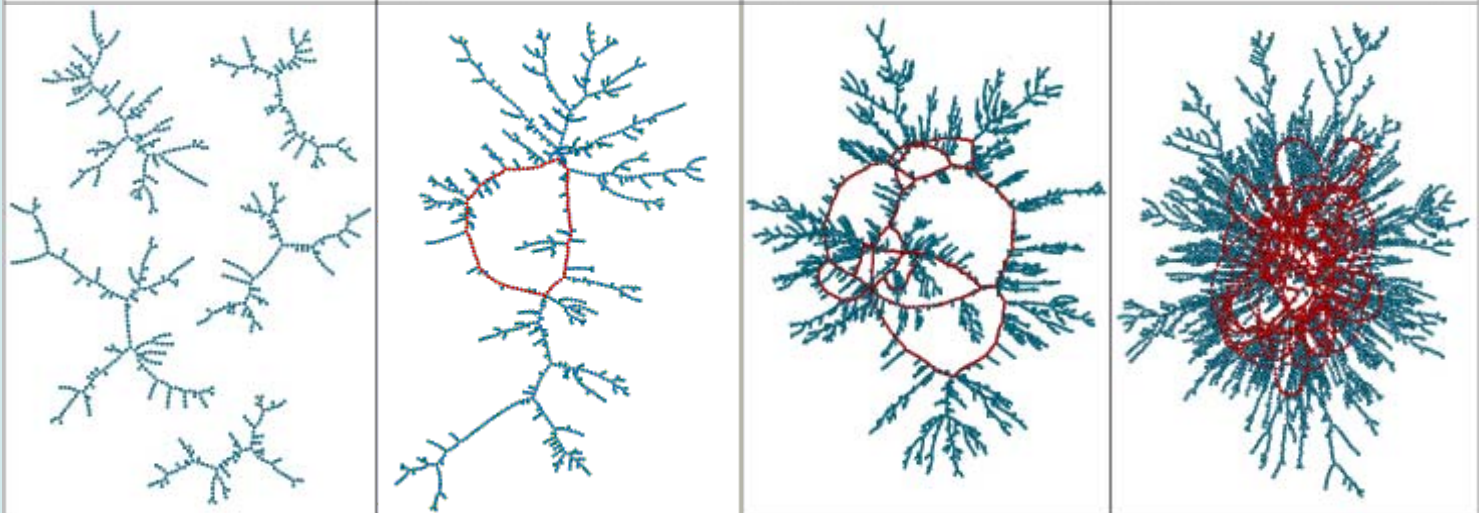
This core does not look at particularly high risk

# Third implication: 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%

*Hubs and superspreaders are not required for network connectivity*

# Summary for population level effects

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**Partnerships matter when the mechanism of transmission involves a mode of contact that is often repeated with the same person**

- This reduces the population level impacts of changes in  $\beta$  and  $c$
- Increases the impact of small changes in and low levels of  $p$
- Creates the potential for protective partner sequencing
- Which can be reduced by concurrent partnerships

# So what do we know empirically?

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- A good review in Mah and Halperin (2008)
- Evidence that concurrency may help to explain two important disparities in HIV prevalence
  - Sub-saharan Africa vs. the rest of the world
    - Long term concurrency is more common
    - A legacy of polygyny?
    - Good overview in Epstein (2007)
  - Racial disparities in HIV in the US
    - Adimora's work
    - Morris et al., forthcoming AJPH

# And what do we know about interventions?

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- Uganda's zero grazing campaign
  - Started in the late 1980s
  - Seroprevalence subsequently dropped from 15% to 5%
    - Equivalent to a vaccine of 80% effectiveness (Stoneburner 2004)
  - Bottom up process of message development
  - Universal message
    - Non-judgmental
    - Deep resonance with pre-existing commitment to civic responsibility
    - Endorsed at all levels of civil society
- Other examples we should hear about at this conference

# Summary

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- **Empirical needs**

- Link concurrency/change to HIV incidence
- Get ALL OF THE IMPORTANT INDICATORS
  - Concurrency effects can not be measured without these
  - Worst thing would be to do the science wrong

- **Intervention development needs**

- This is a community normative change
  - Not just an individual behavior change
  - Your partner may be your problem
- Opportunity now with household-based testing and counseling (HBTC)

# Summary

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- What do we know about community norm changing?
- Example: Tostan project (<http://www.tostan.org/>)
  - Focus: female genital cutting/mutilation
  - Issue: no individual can make the change
    - Otherwise their daughter would be ostracized
    - Need a group consensus on change
    - Community level interventions
  - How do you get group consensus on change?
    - Information offered from outside
    - Internal group decision on direction/choice

# Conclusion

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- This is a critical moment
  - Science is unsettled
    - Theoretically clear, but empirically confused
      - Many misunderstandings
      - Need data on relation between concurrency and HIV incidence
  - Next steps have to be clear
    - Use HBTC opportunity
    - Focus on community level intervention
    - Measure incidence as endpoint
- We can not afford to get this wrong
  - We can not afford to mistake poor science for no effect