Models for Understanding and Controlling Global Infectious Diseases HUMBIO 154D / HRP 204

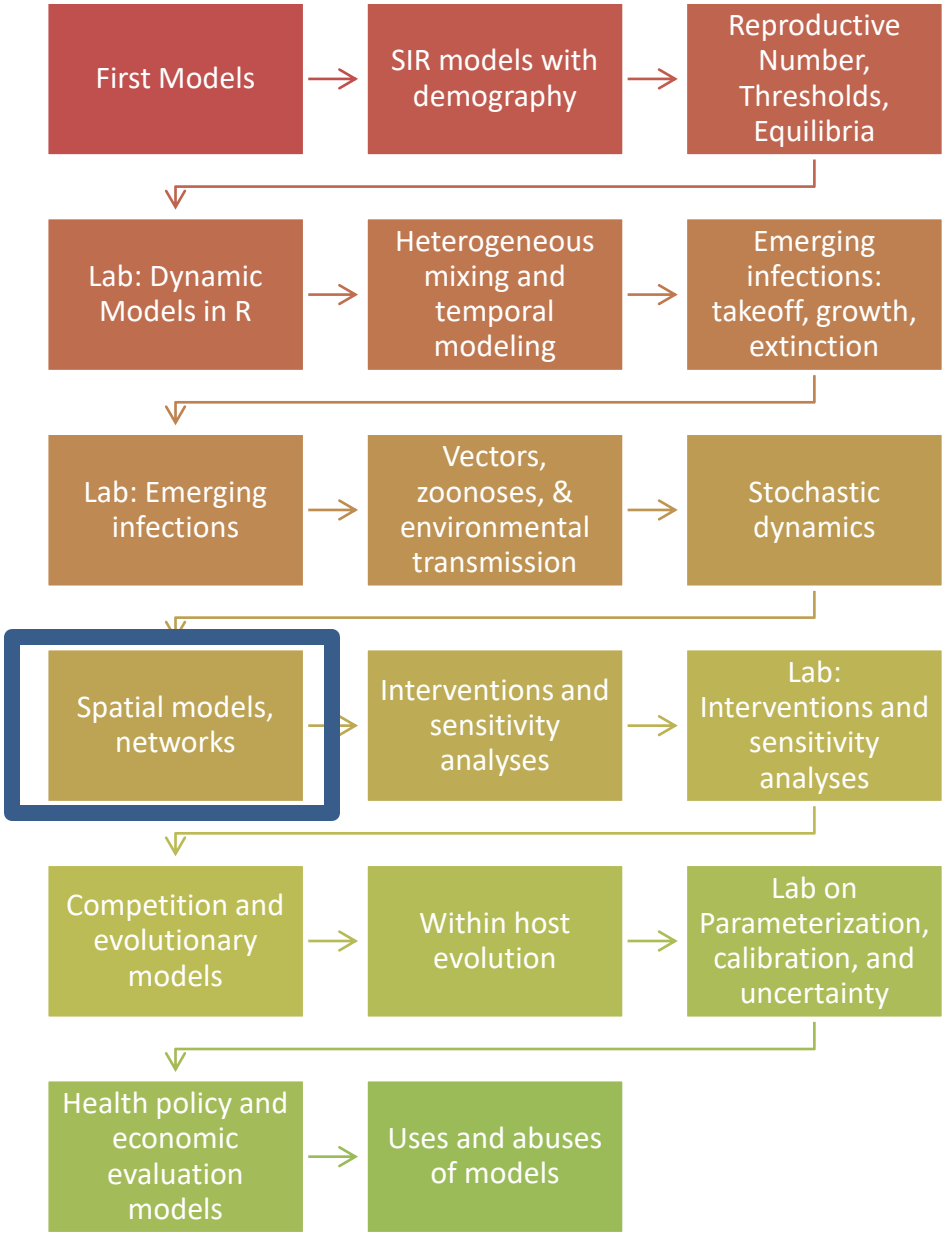
Session 10

Jason Andrews

Jeremy Goldhaber-Fiebert

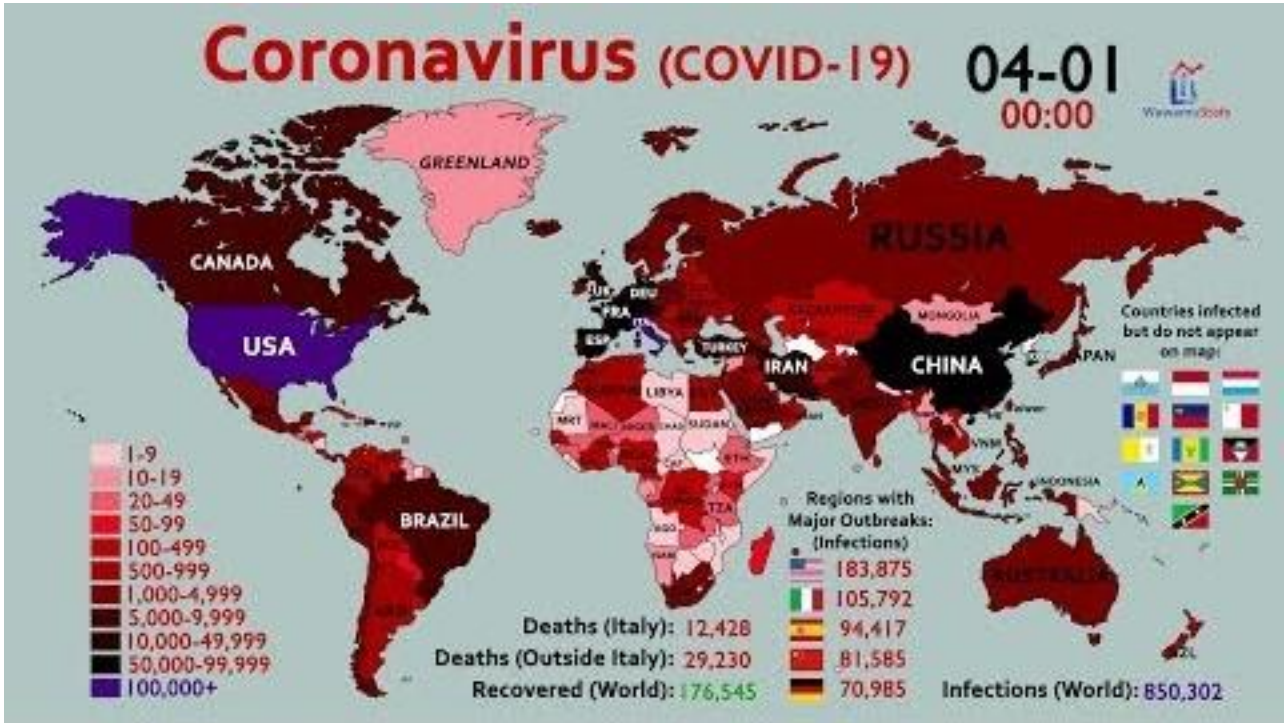
2020

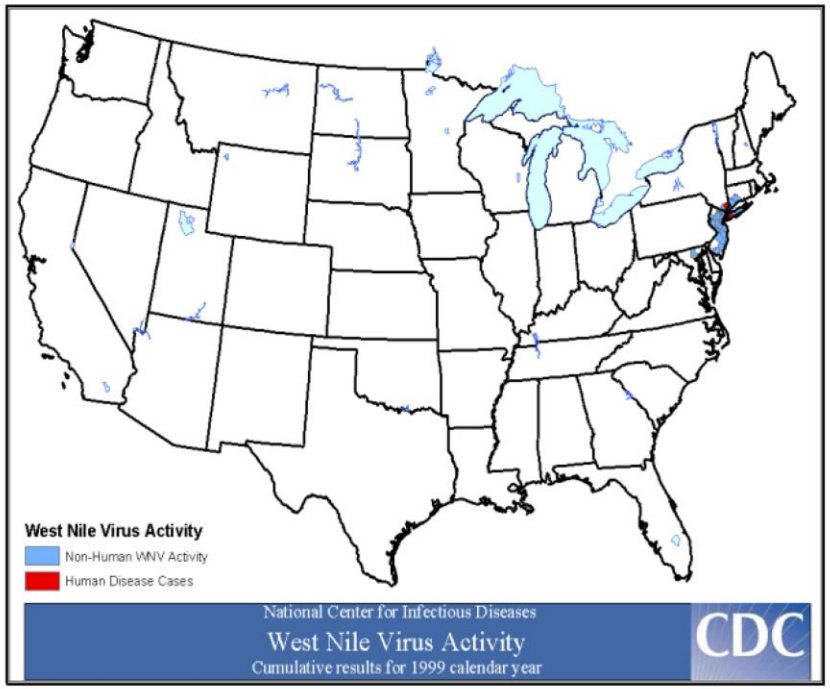
Course Roadmap

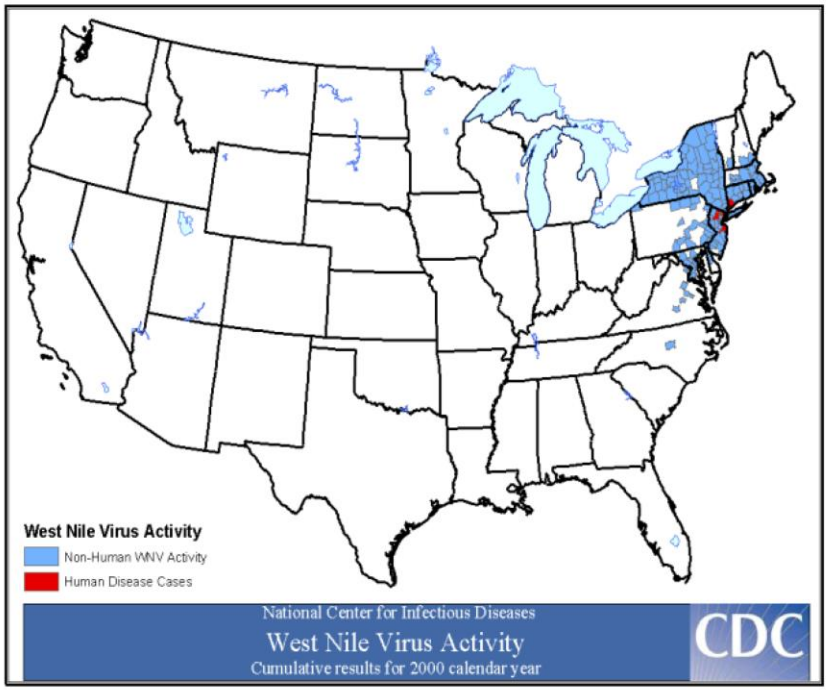


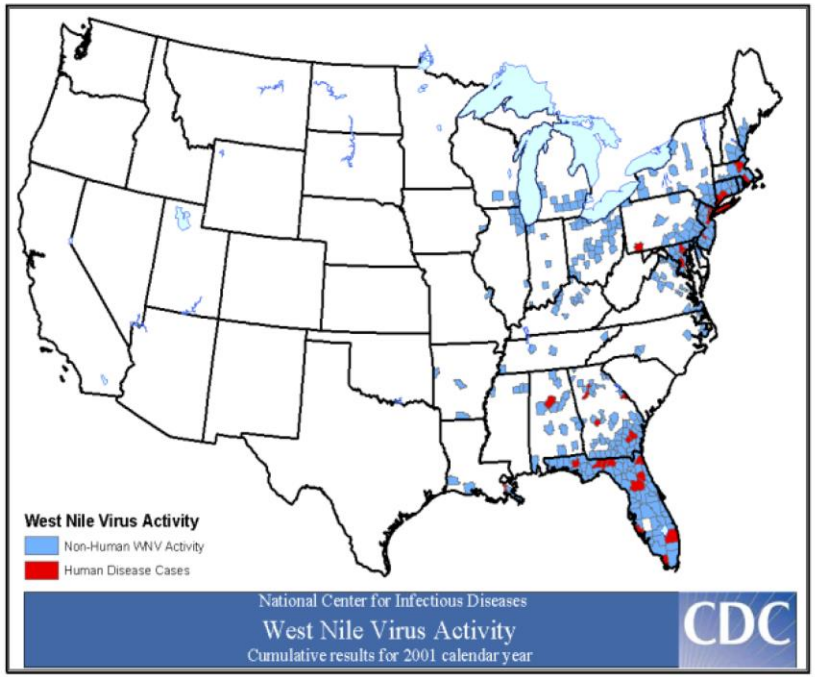
Geographic spread of COVID19

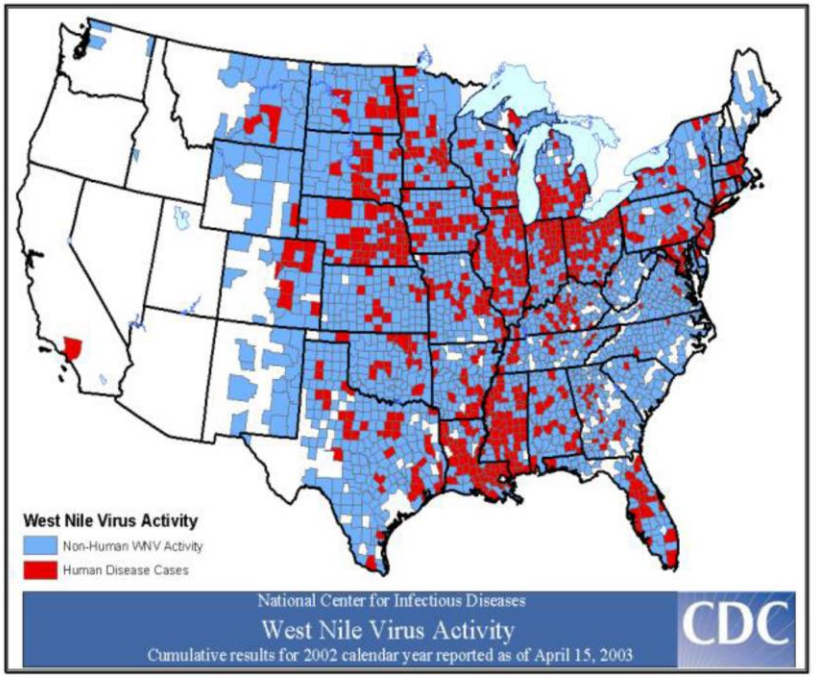
- https://youtu.be/J1pPytSRe-s




West Nile Virus Activity
Non-Human WNV Activity
Human Disease Cases
National Center for Infectious Diseases
West Nile Virus Activity
Cumulative results for 1999 calendar year
CDC


West Nile Virus Activity
Non-Human WNV Activity
Human Disease Cases
National Center for Infectious Diseases
West Nile Virus Activity
Cumulative results for 2000 calendar year
CDC

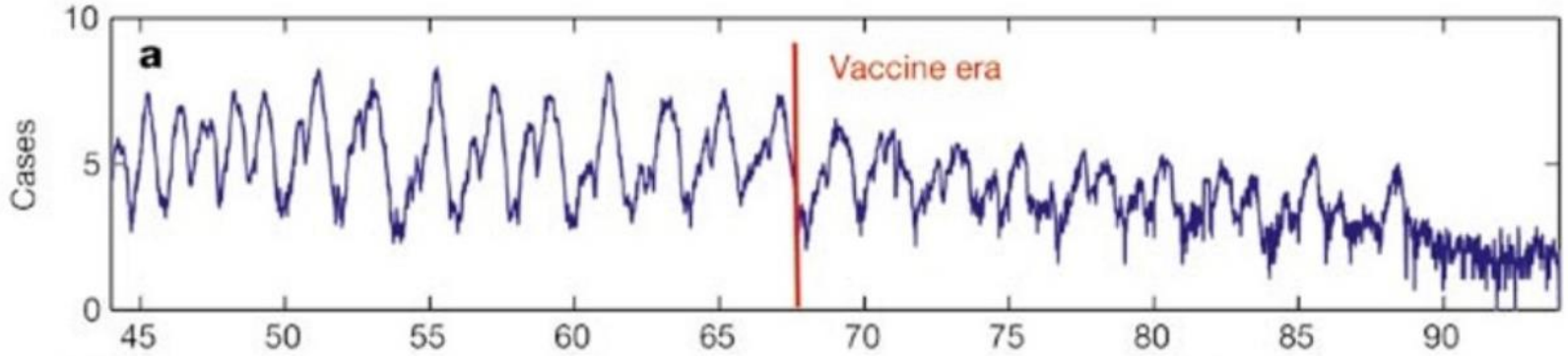

West Nile Virus Activity
Non-Human WNV Activity
Human Disease Cases
National Center for Infectious Diseases
West Nile Virus Activity
Cumulative results for 2001 calendar year
CDC

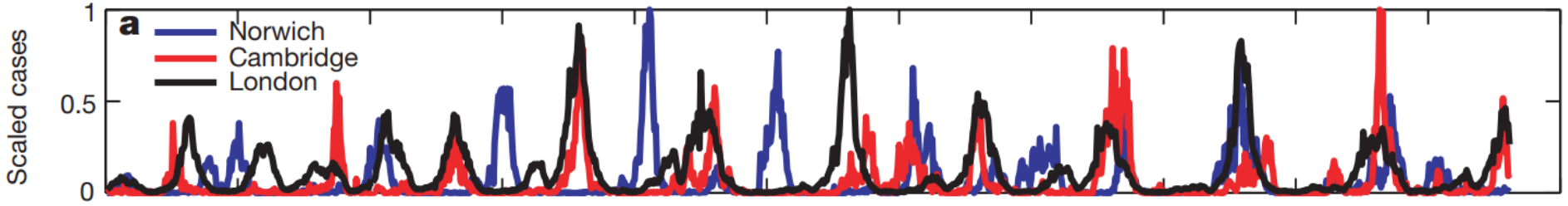

West Nile Virus Activity
Non-Human WNV Activity
Human Disease Cases
National Center for Infectious Diseases
West Nile Virus Activity
Cumulative results for 2002 calendar year reported as of April 15, 2003
CDC

Measles

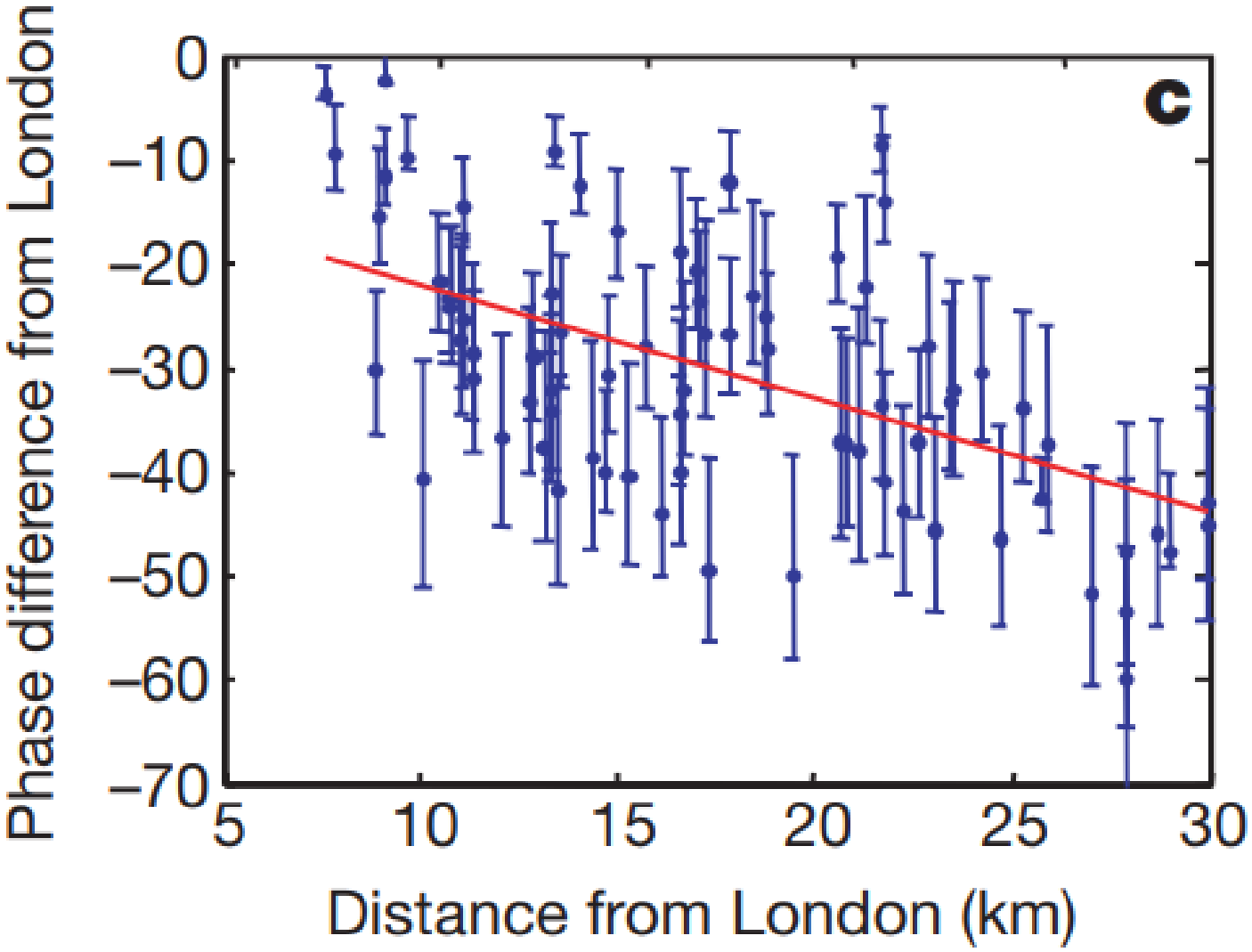
Travelling waves and spatial hierarchies in measles epidemics

B. T. Grenfell*, O. N. Bjørnstad*† & J. Kappey*





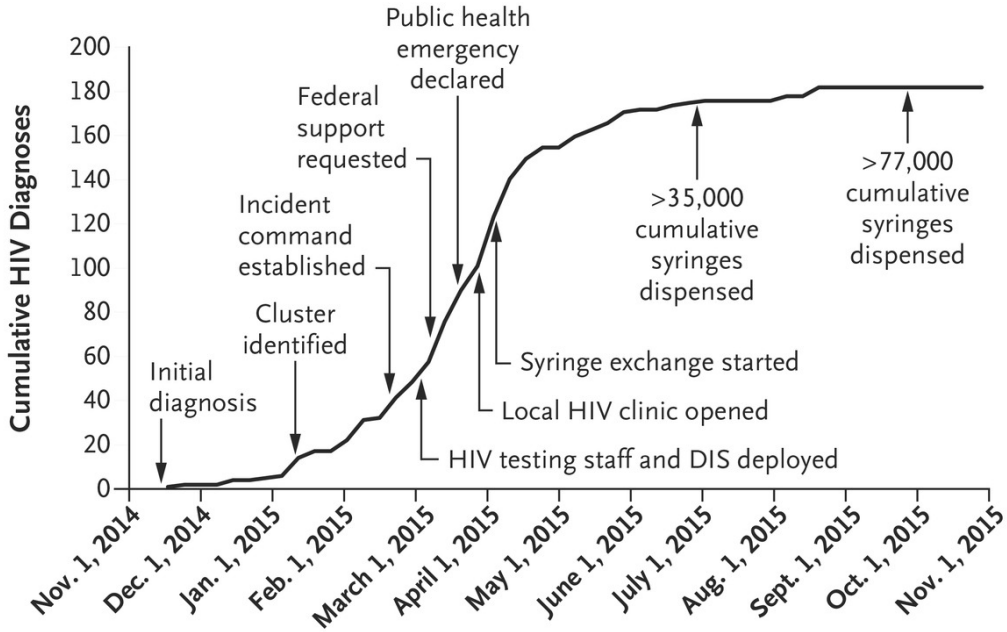
Measles


c
Phase difference from London
0
−10
−20
−30
−40
−50
−60
−70
5
10
15
20
25
30
Distance from London (km)

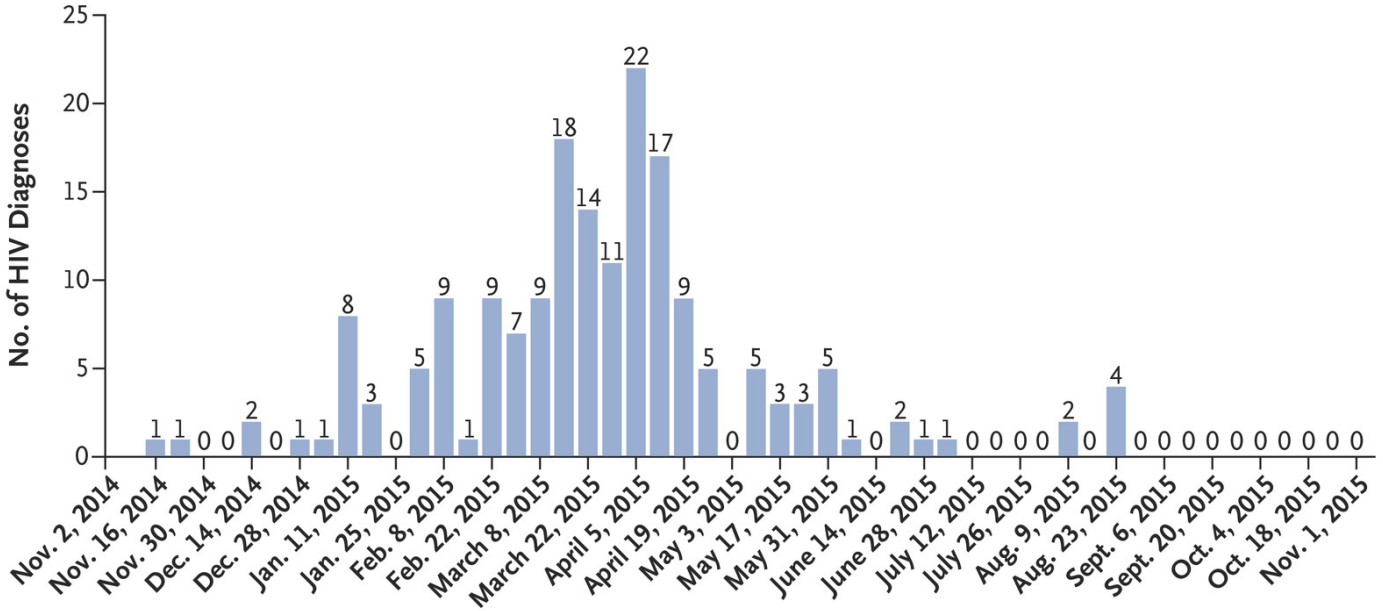
HIV Infection Linked to Injection Use of Oxymorphone in Indiana, 2014–2015

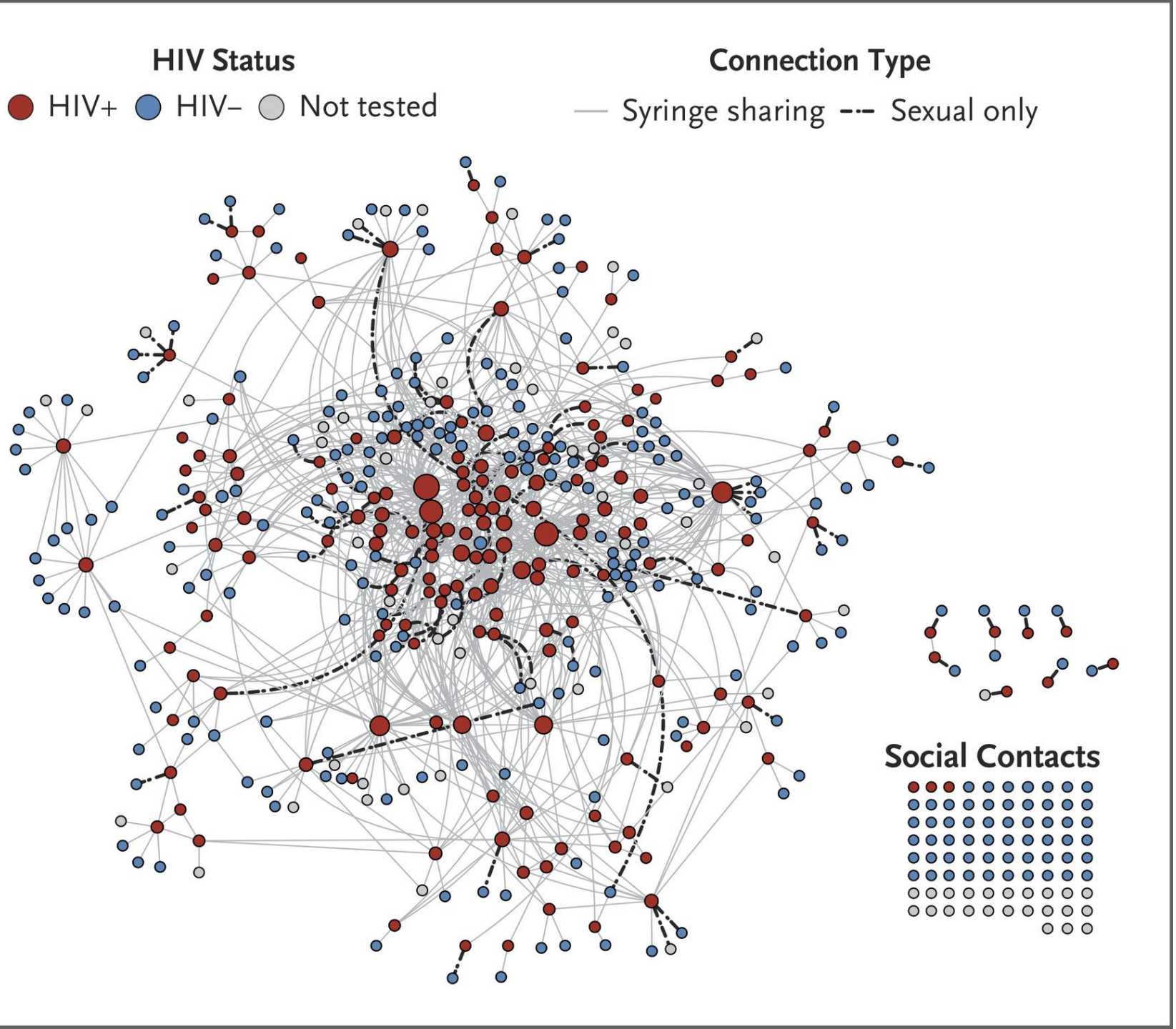
Philip J. Peters, M.D., Pamela Pontones, M.A., Karen W. Hoover, M.D., M.P.H., Monita R. Patel, Ph.D., M.P.H., Romeo R. Galang, M.D., M.P.H., Jessica Shields, B.S., Sara J. Blosser, Ph.D., Michael W. Spiller, Ph.D., Brittany Combs, R.N., William M. Switzer, M.P.H., Caitlin Conrad, B.S., Jessica Gentry, M.A., et al., for the Indiana HIV Outbreak Investigation Team*

A Cumulative HIV Diagnoses and Public Health Response



B HIV Diagnoses According to Week of Testing




HIV Status
HIV+
HIV–
Not tested
Connection Type
Syringe sharing
Sexual only
Social Contacts

Practical Questions

How does geographic and spatial structure influence the spread of infectious diseases?

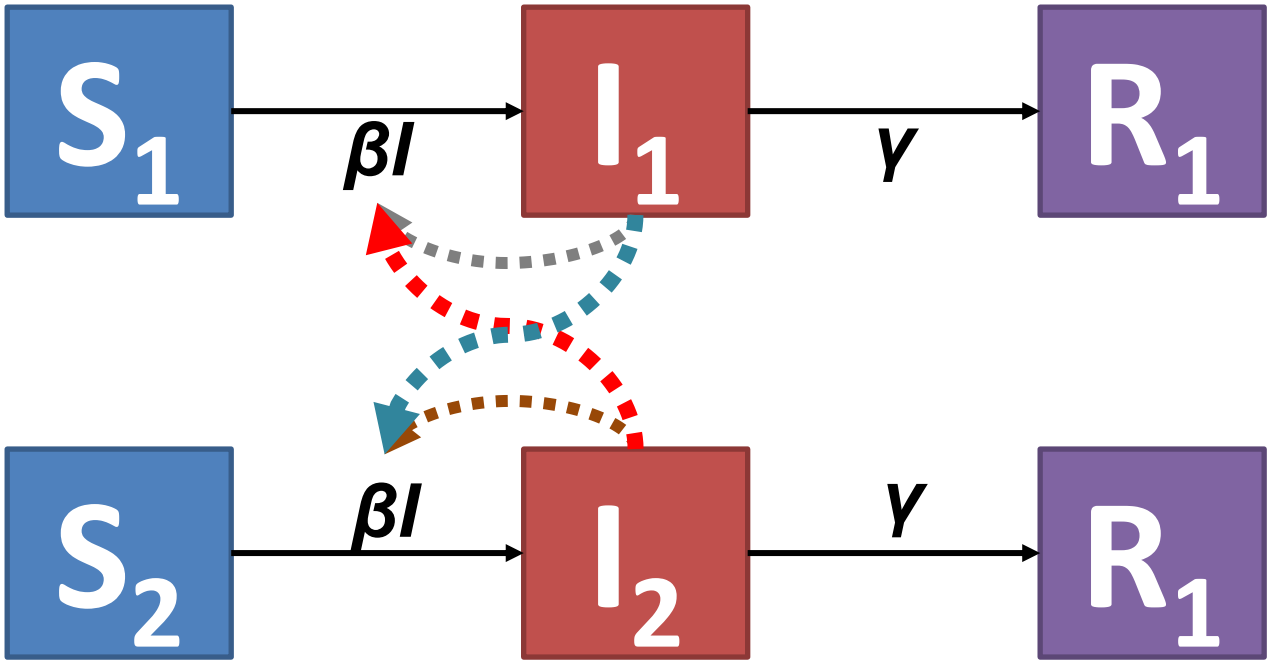
How does social network influence the spread of infectious diseases? For which diseases is such structure most relevant?

Learning Objectives

- Define what a spatial model is
- Define what a meta-population model is
- Define what a lattice model is
- Describe the use of a transmission kernel
- Explain coupling, correlation, synchrony
- Describe the relationship of coupling and synchrony to extinction and reimportation
- Describe micro- and macro-features of network models (and their various types)
- Identify when spatial models are useful/relevant

SPATIAL MODELS (ANOTHER FORM OF HETEROGENEITY) AND META-POPULATION MODELS

We have already seen the basic machinery we need for half the lecture today



Instead of thinking about heterogeneous risk groups mixing, we can think about members of 2 different geographic communities mixing

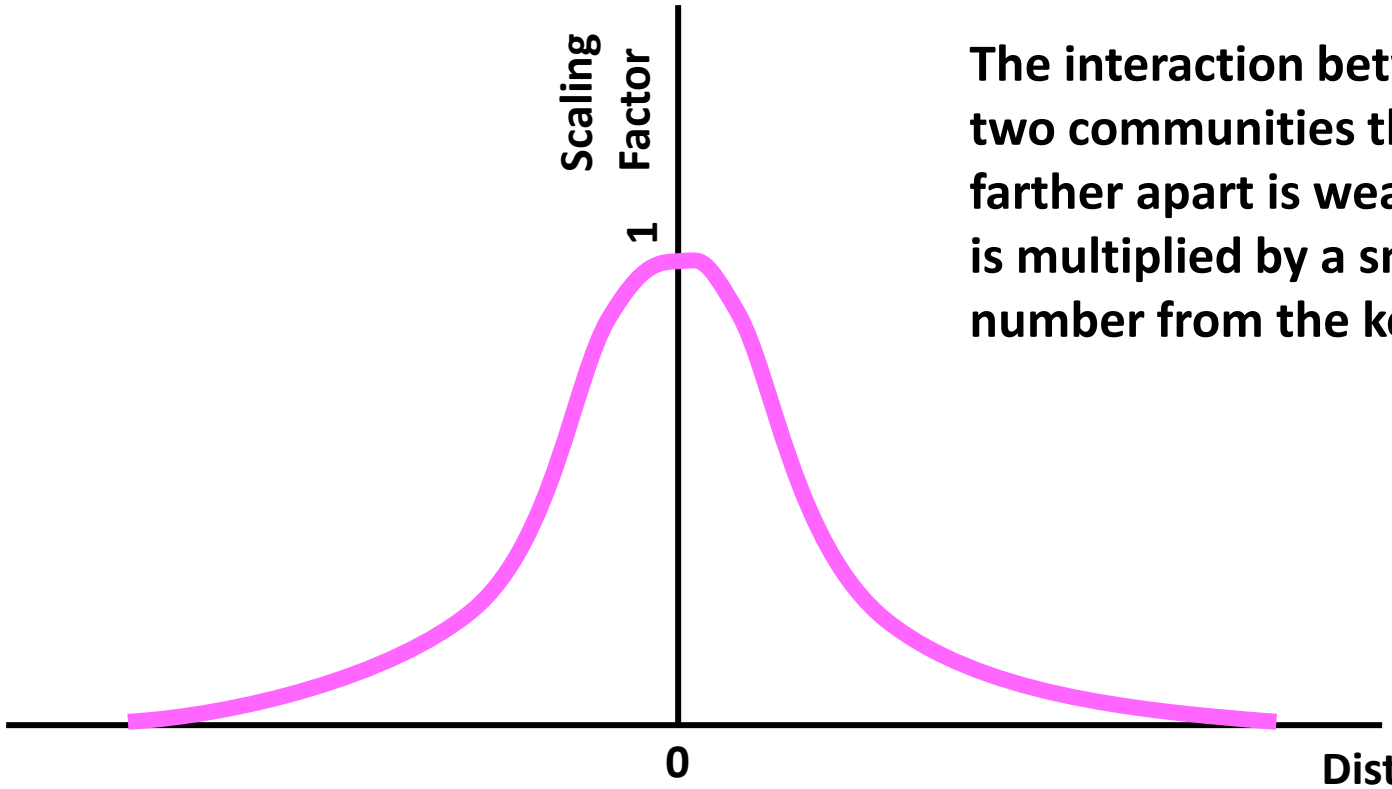
If the communities do not otherwise differ, the more often/strongly they mix, the more the model becomes coupled (synchrony) (pg. 246 describes the typical pattern – sigmoidal in interaction strength)

How do we describe the strength of interaction between two communities

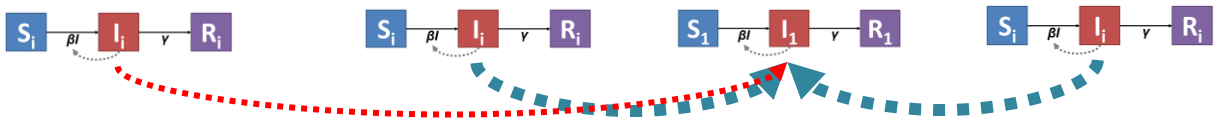
- One way is to couple communities more strongly if they are closer together (smaller distance ***d***)
- To do so, we define a transmission kernel ***K***
  - For example, a Gaussian Kernel: $\boldsymbol{e^{-Ad^2}}$

Gaussian Transmission Kernel Example

- Gaussian Kernel: e^{-Ad^2}



The interaction between two communities that are farther apart is weaker as it is multiplied by a smaller number from the kernel



Size of communities modeled

- As we break down our population into more communities, their average size is smaller
- This means that local extinction becomes more likely, except …
- Coupling between these smaller and smaller communities generally plays a bigger and bigger role and so reimportation plays a more and more important role maintaining infectious prevalence

Meta-Populations

- Example of a meta-population model

$$\frac{dX_i}{dt} = \nu_i N_i - \lambda_i X_i - \mu_i X_i$$

$$\frac{dY_i}{dt} = \lambda_i X_i - \gamma_i Y_i - \mu_i Y_i$$

- The meta-population model has ***I*** communities in total

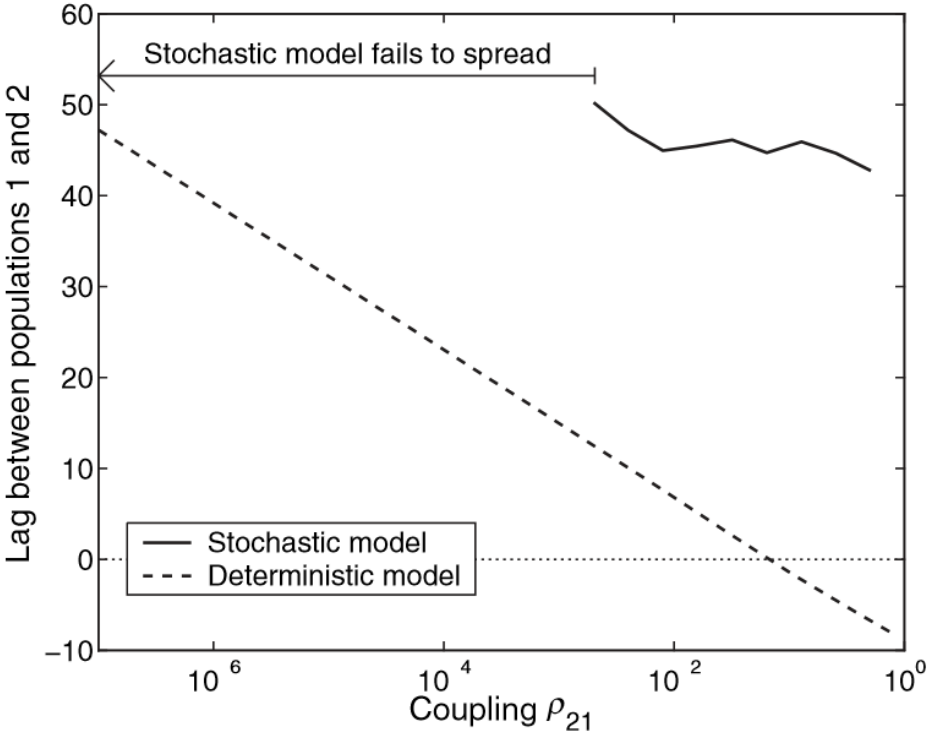
Meta-Populations: Forces of Infection

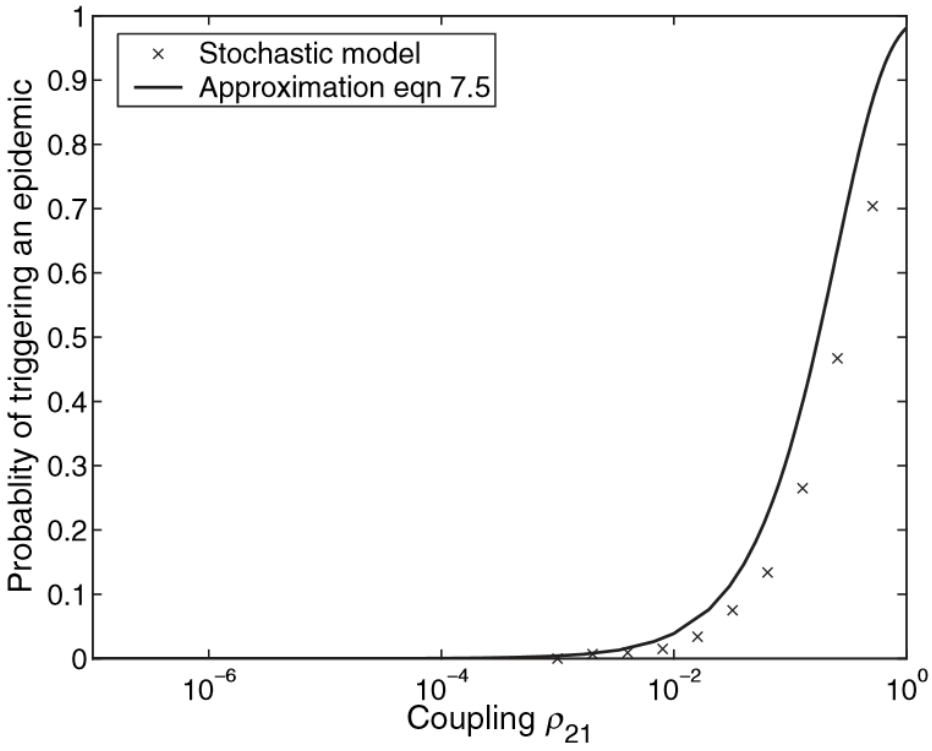
$$\lambda_i = \beta_i \sum_j \rho_{ij} \frac{Y_j}{N_i}$$

Book has typo and says ***Xj***

- Relative strength of transmission from ***j*** to ***i*** is ρ_{ij} (e.g., transmission kernel)
- Could use N_i or N_j as the denominator
  - N_i infectious from ***j*** visit ***i***
  - N_j susceptibles from ***i*** visit ***j***
  - Section 7.2.1.3 provides a way of accommodating both: when duration of visits are short, the above works well

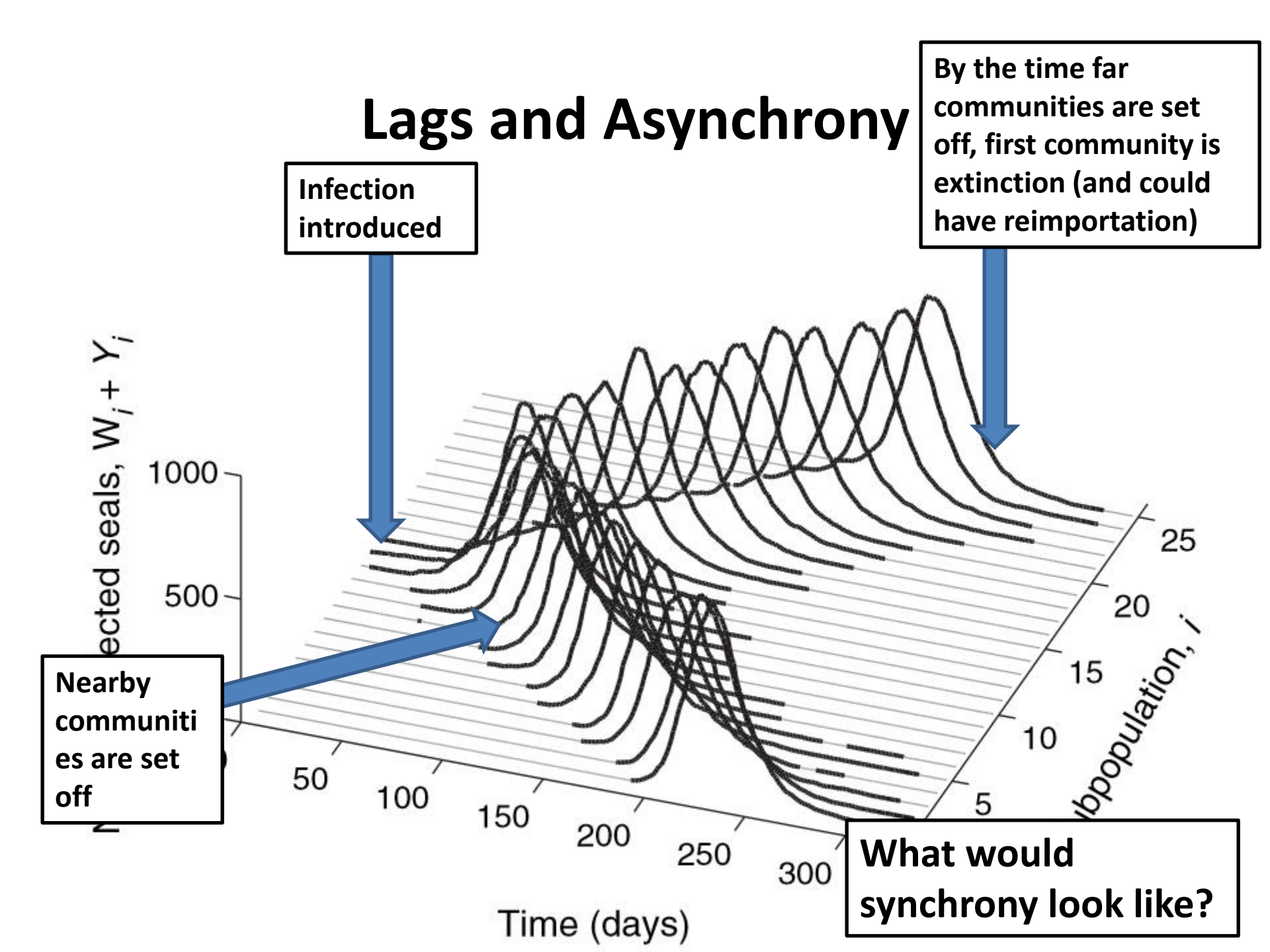
The function of ρ_{ij} in Meta-Populations





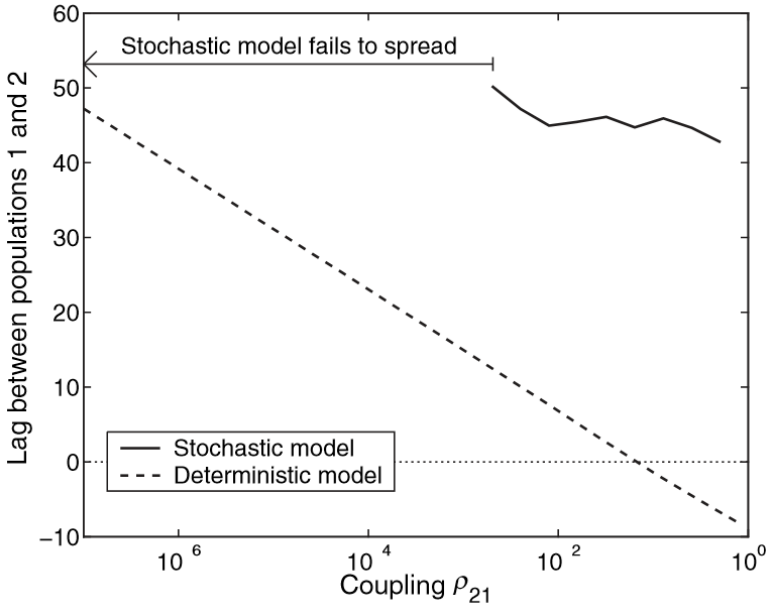
The stronger ρ_{21} is the shorter the lag between an epidemic in i=1 and in j=2
The stronger ρ_{21} is the more likely an epidemic in i=1 triggers an epidemic in j=2

Lags and Asynchrony

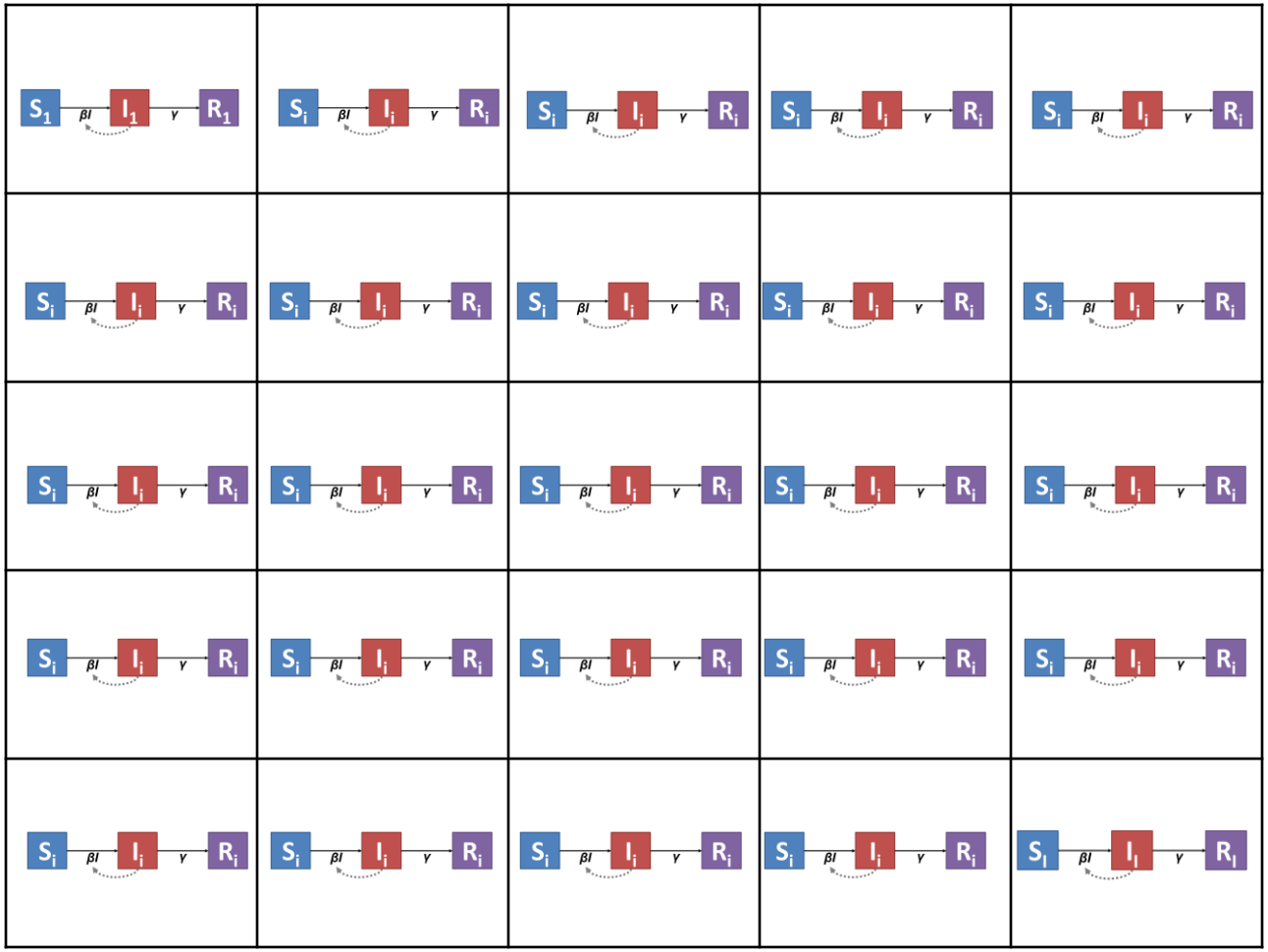


Shorter lags: greater synchrony

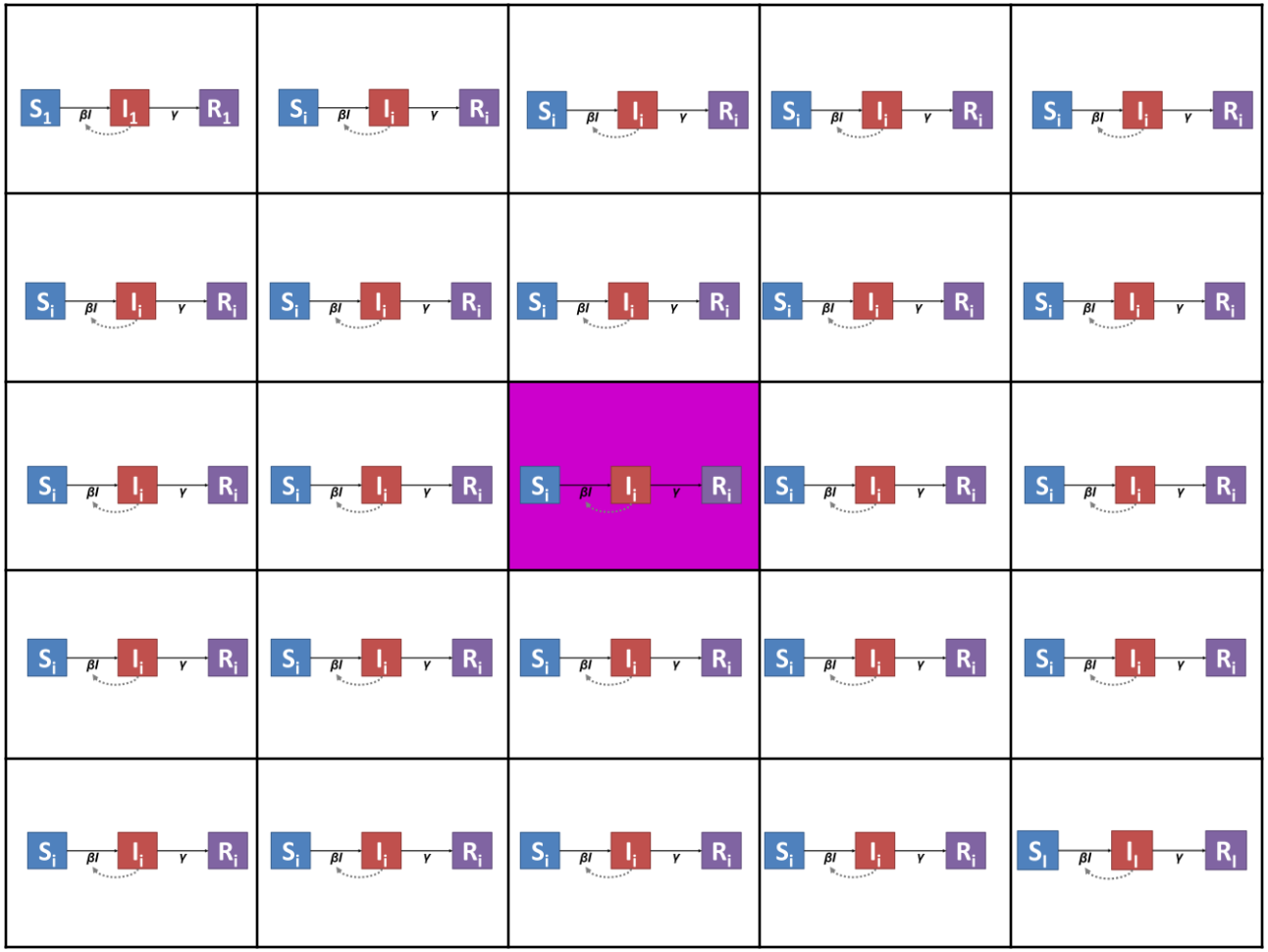
- Since subpopulations in the meta-population are smaller, they have greater chances of local extinction
- Coupling leads to reimportation which counters local extinction
- Asynchrony of epidemic peaks (peak in one population lagging peaks in others) helps with reintroduction. Why?
- Regular vaccination in a meta-population leads to reductions (risk of extinction) but also asynchrony. So will it be effective in causing global extinction?
- Pulsed vaccination (synchronizing when vaccination is given across all subpopulations) may help to synchronize reductions to get global extinction



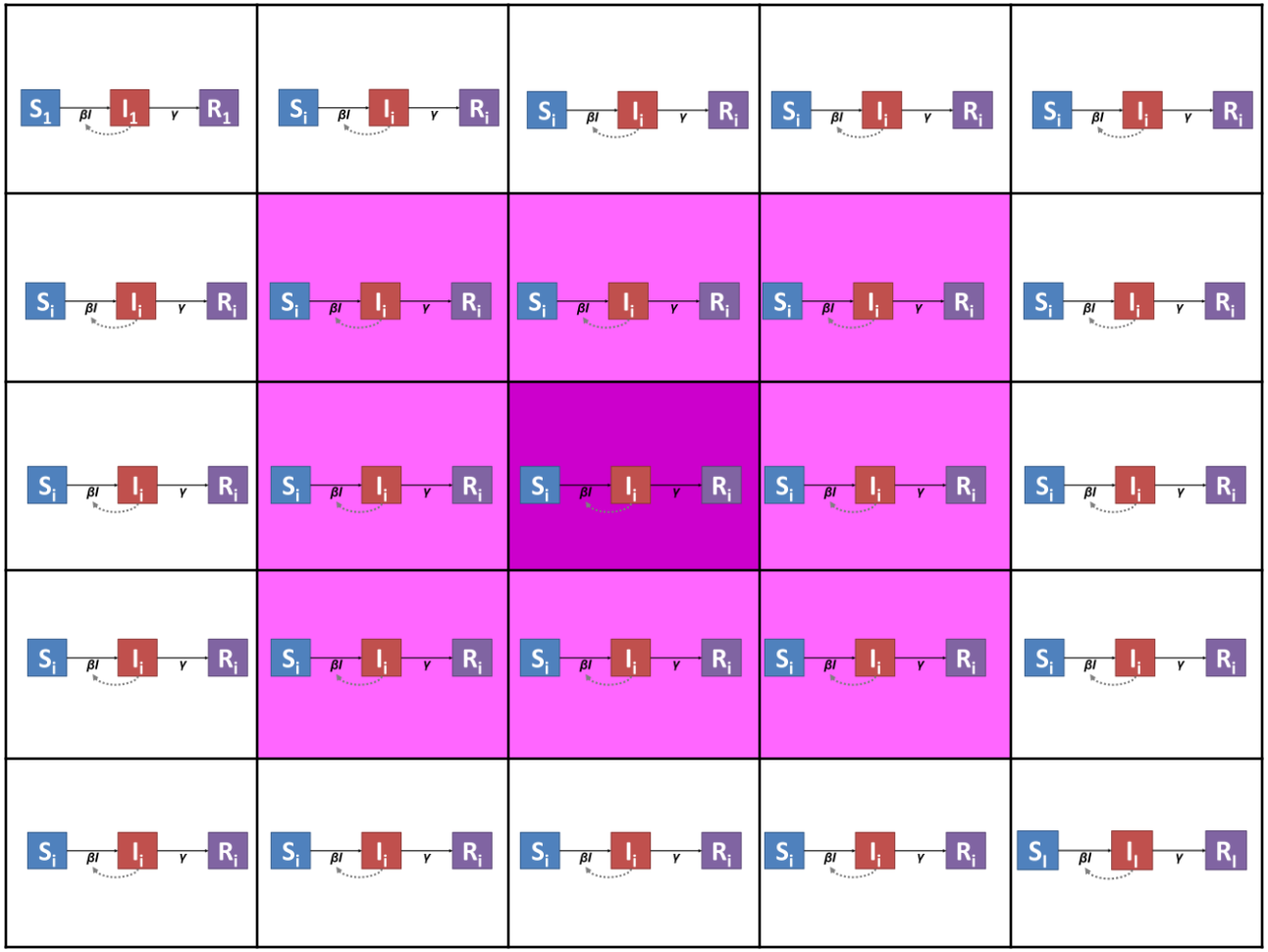
Lattice models (7.3 not assigned but good to know about)



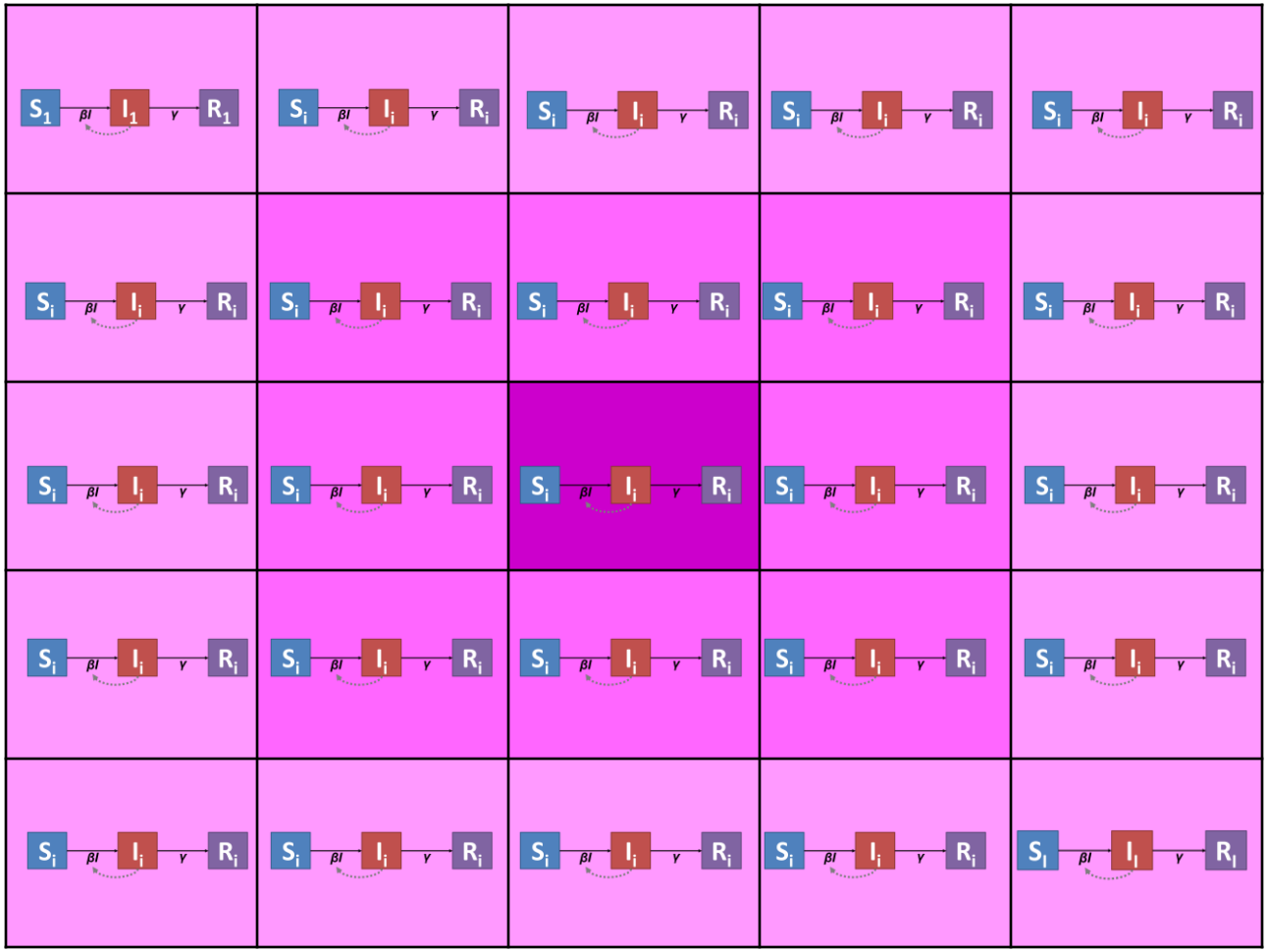
Lattice models: Strength of kernel



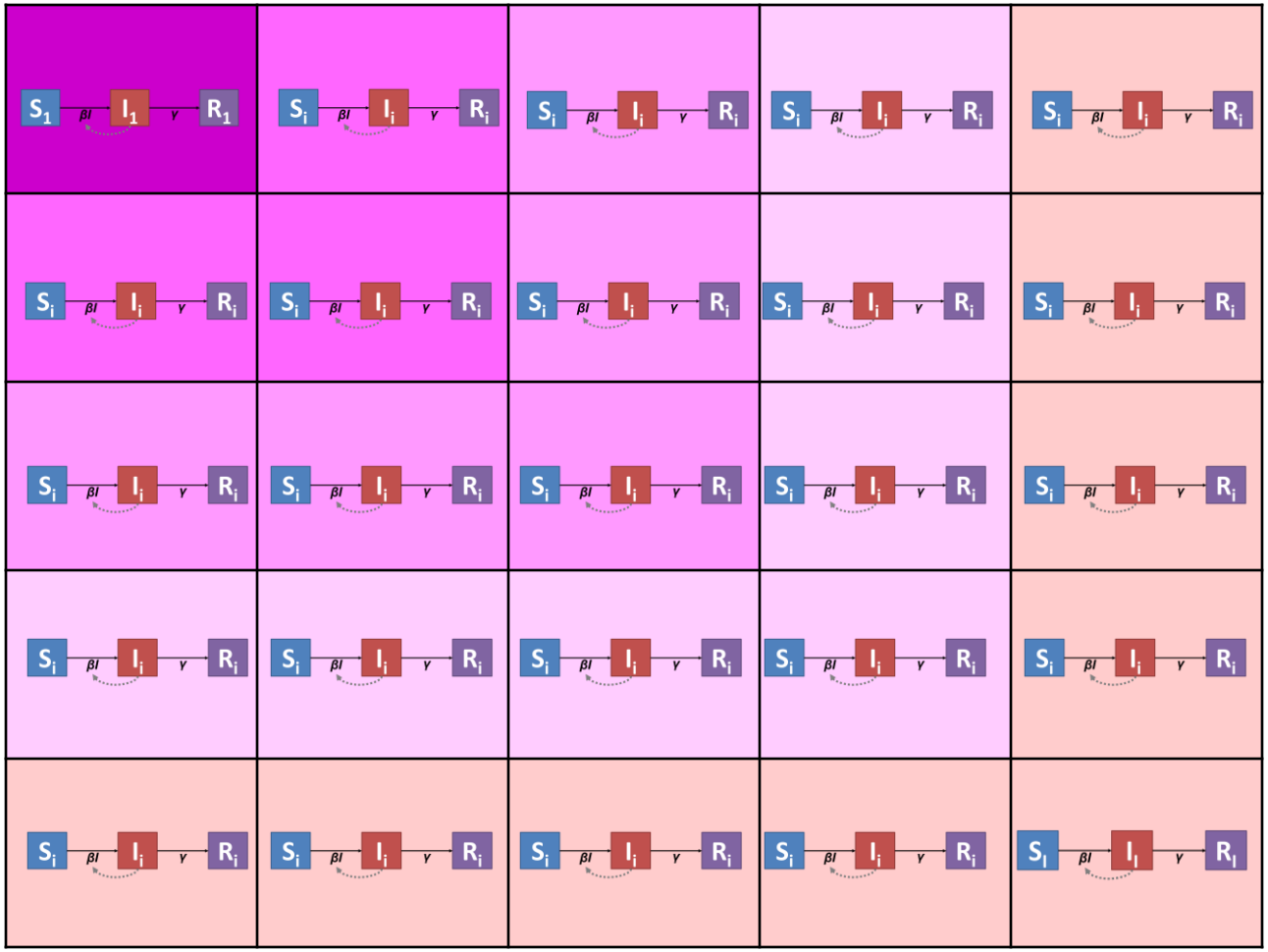
Lattice models: Strength of kernel

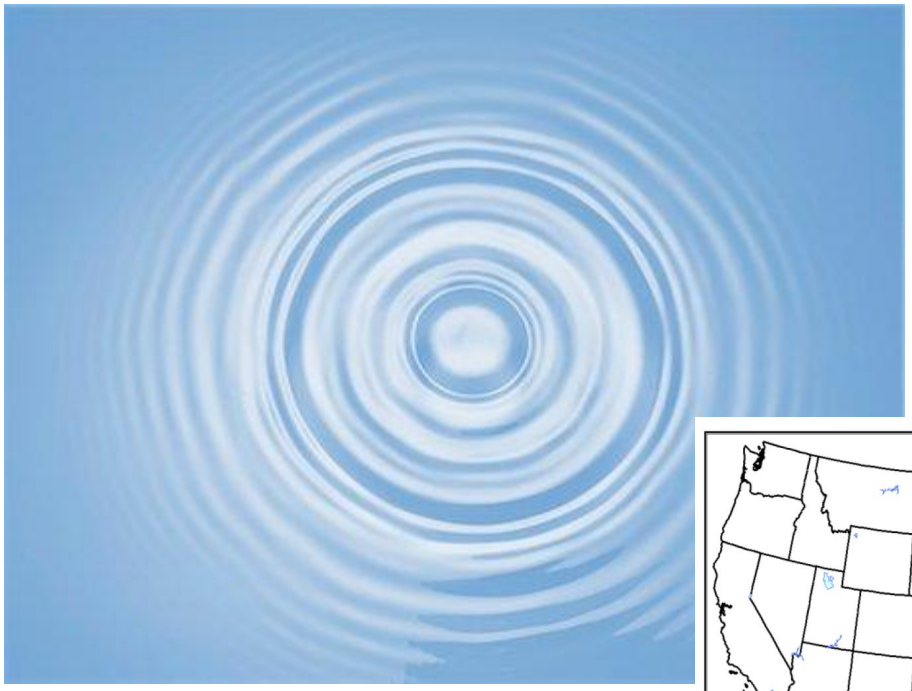


Lattice models: Strength of kernel

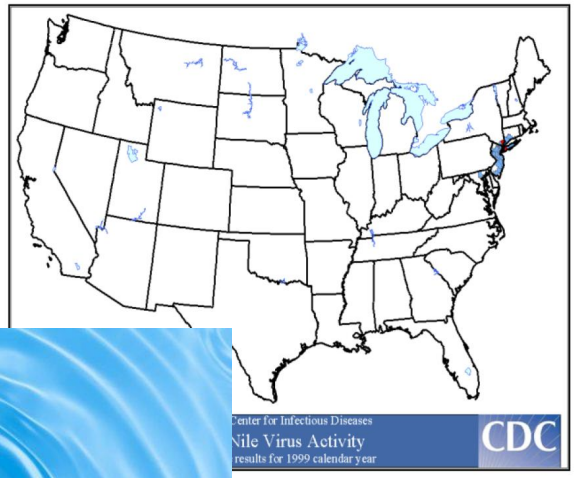


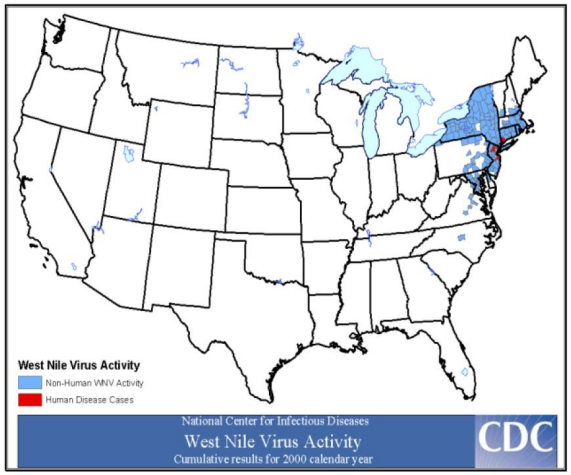
Lattice models: It matters where you start

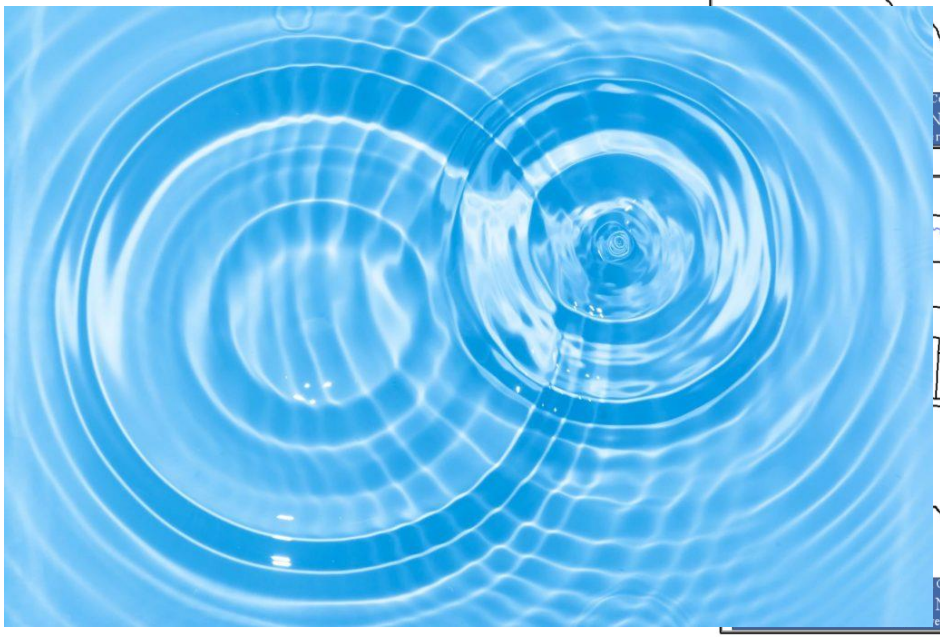


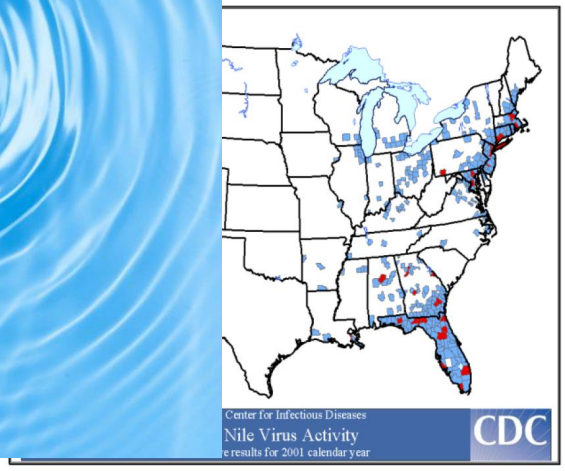


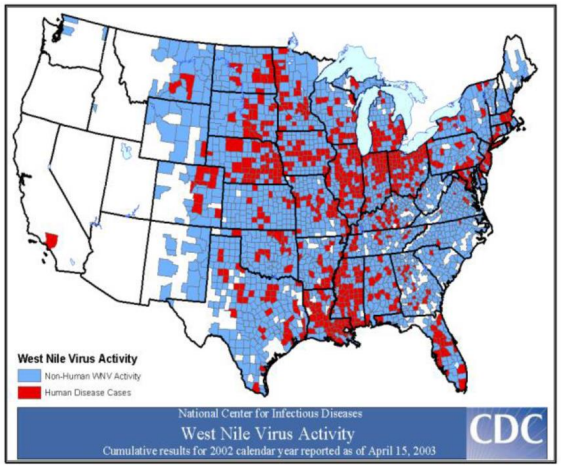
Like ripples in terms of timing and effect; with the potential for a lot of complexity



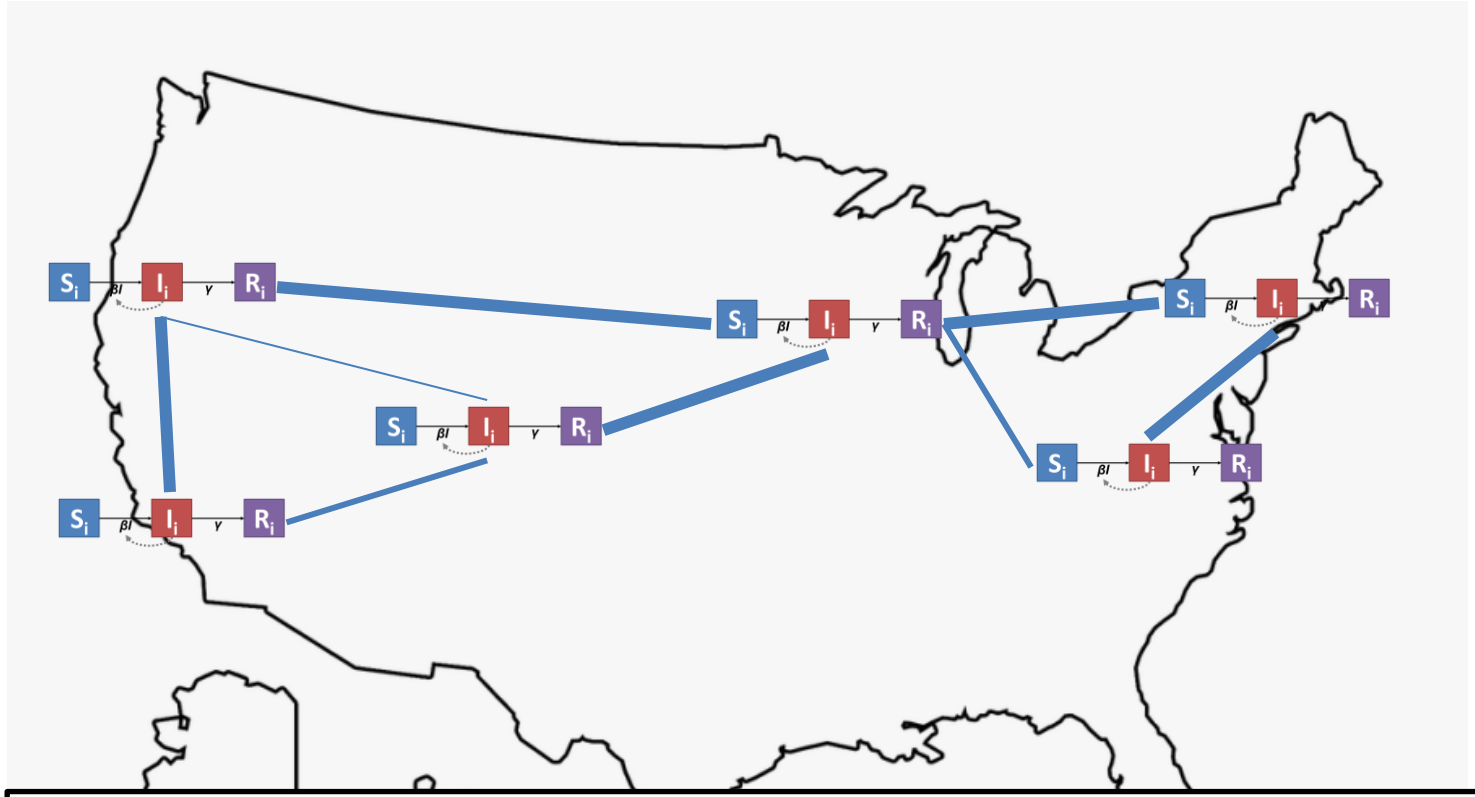








More complex versions: graphs of subpopulations



If line thickness between subpopulations is strength of coupling, how would you translate this into the modeling machinery we have built up today?

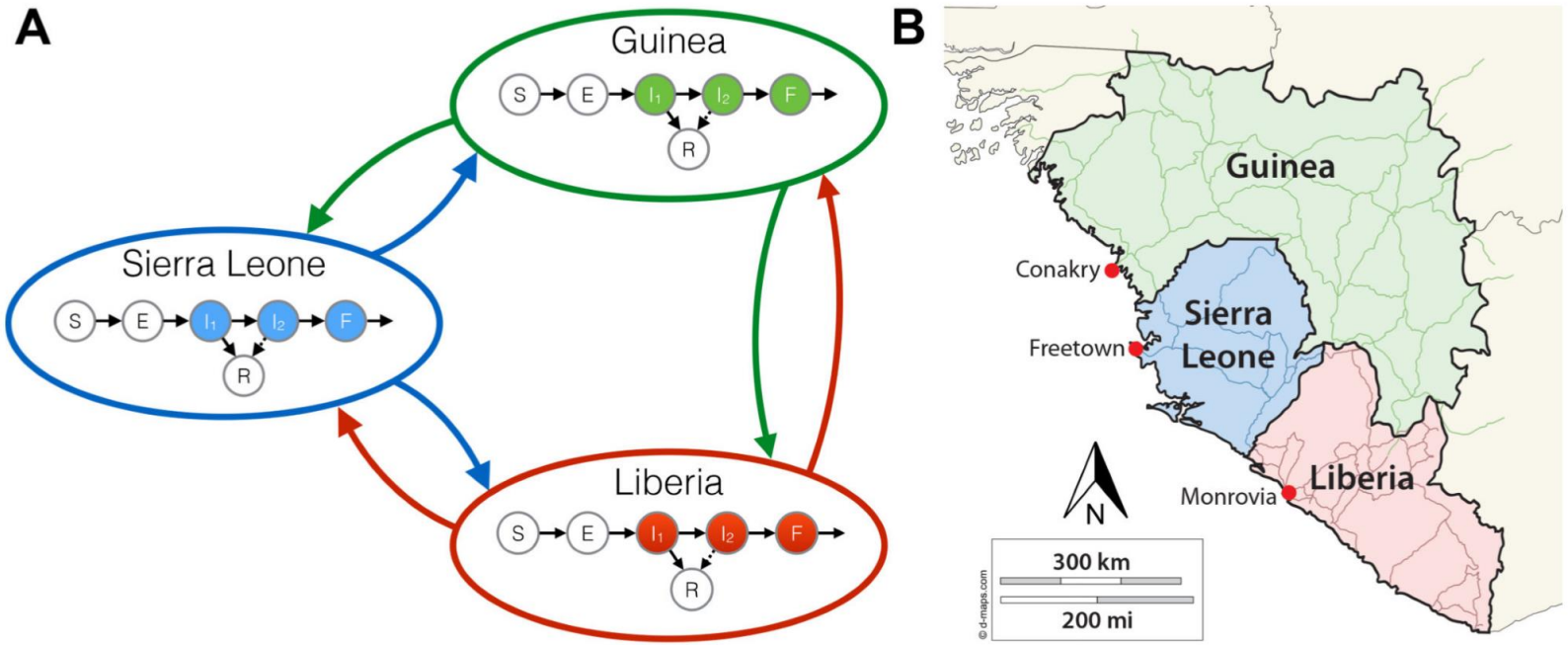


Figure 1.

A diagram of the model structure (left panel) and a map of West Africa showing the locations of each capital used as a population center for each patch (right panel). In each country, the population is compartmentalized into the following categories: susceptible (S), exposed (E), infected in stage one (I_1), infected in stage two (I_2), died but not yet buried (F), and recovered (R). Transmission is possible from persons in compartments I_1, I_2, or F of any country, to persons in compartment S, either within the country or to the other countries.

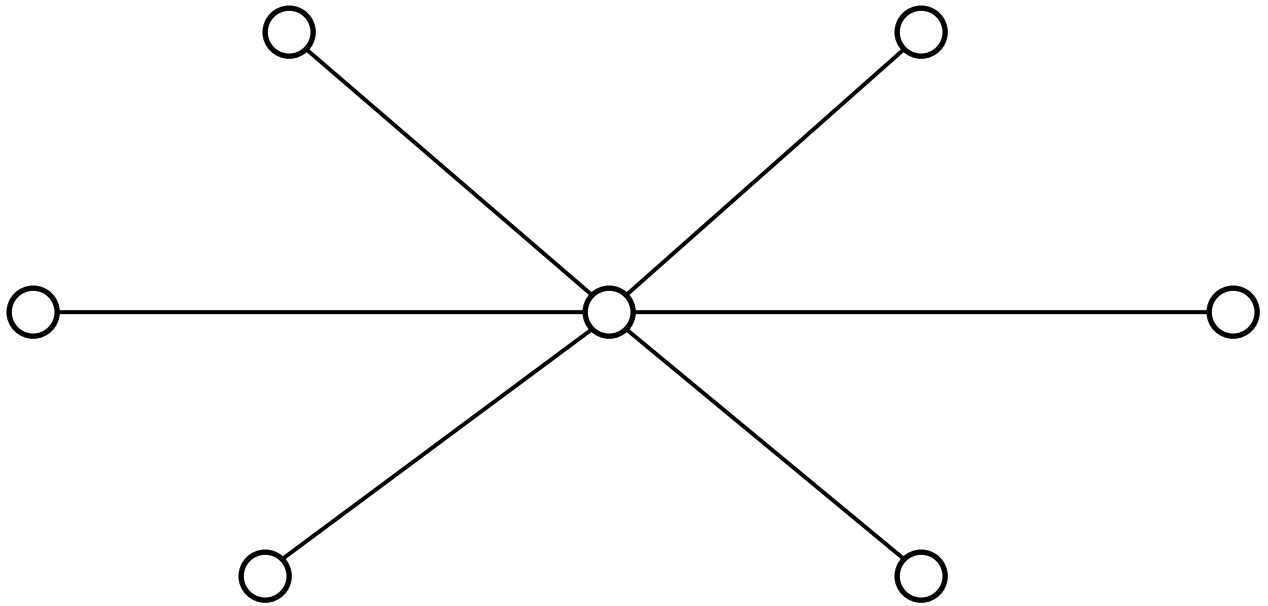
J Theor Biol. 2017 September 07; 428: 65–75. doi:10.1016/j.jtbi.2017.05.034.

Modeling Spatial Invasion of Ebola in West Africa

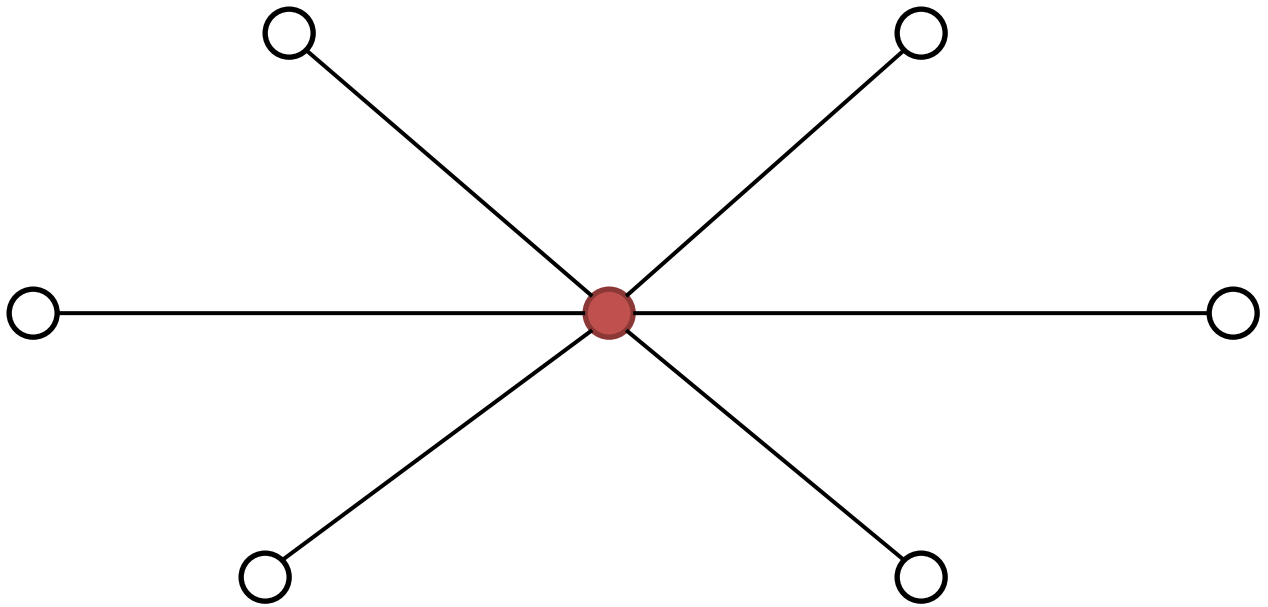
Jeremy P D'Silva and **Marisa C. Eisenberg**
Departments of Epidemiology and Mathematics, School of Public Health, University of Michigan, Ann Arbor

NETWORKS: INDIVIDUAL (STOCHASTIC) MODELS WITH EXPLICIT CONTACT STRUCTURES

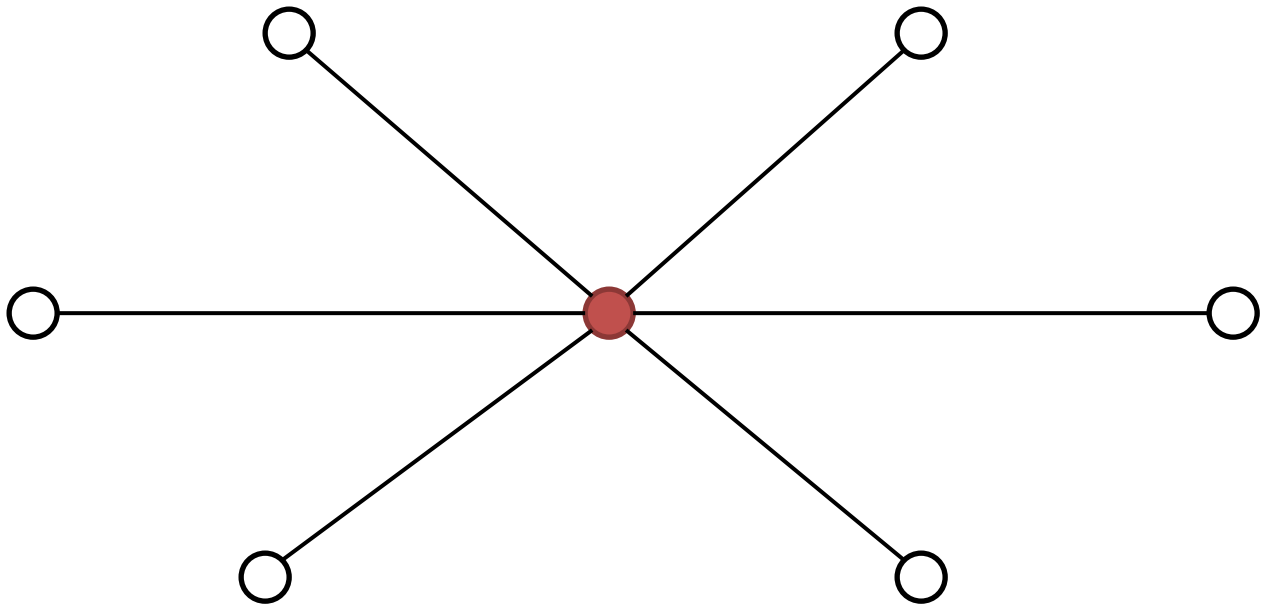
A network is made up of nodes (people) and edges (connections over which transmission could occur)



Each person has a state (susceptible [white]; infectious [red]) at each point in time

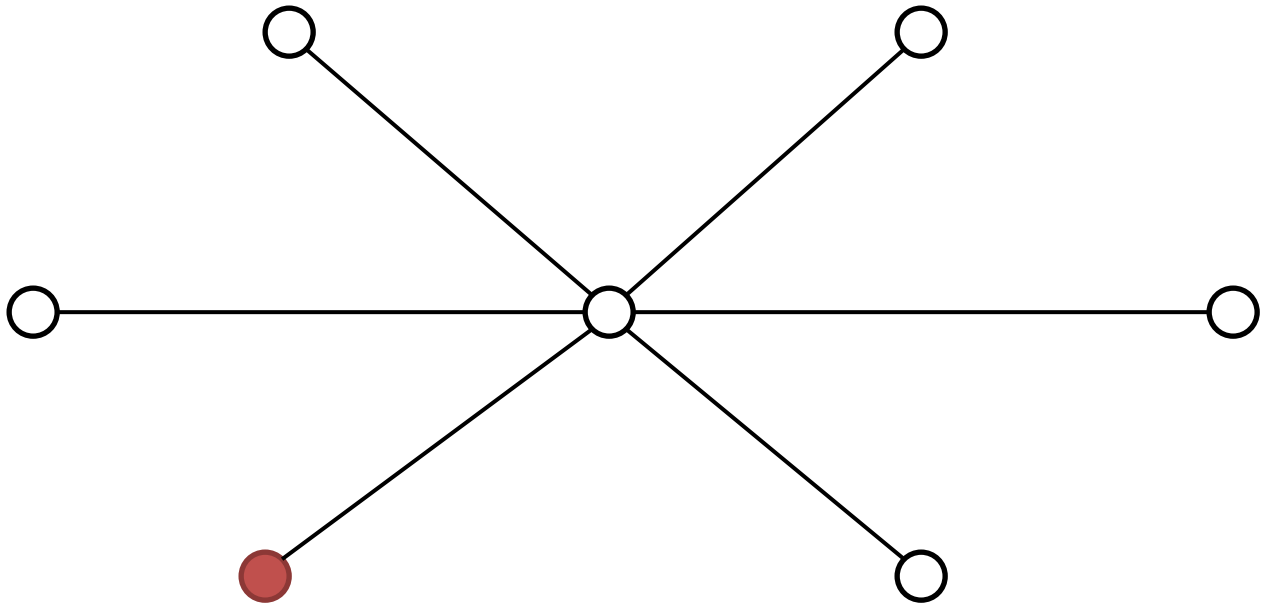


Those who are susceptible are at risk of acquiring infection at the next time step if one of they are connected to 1 or more people who are infected



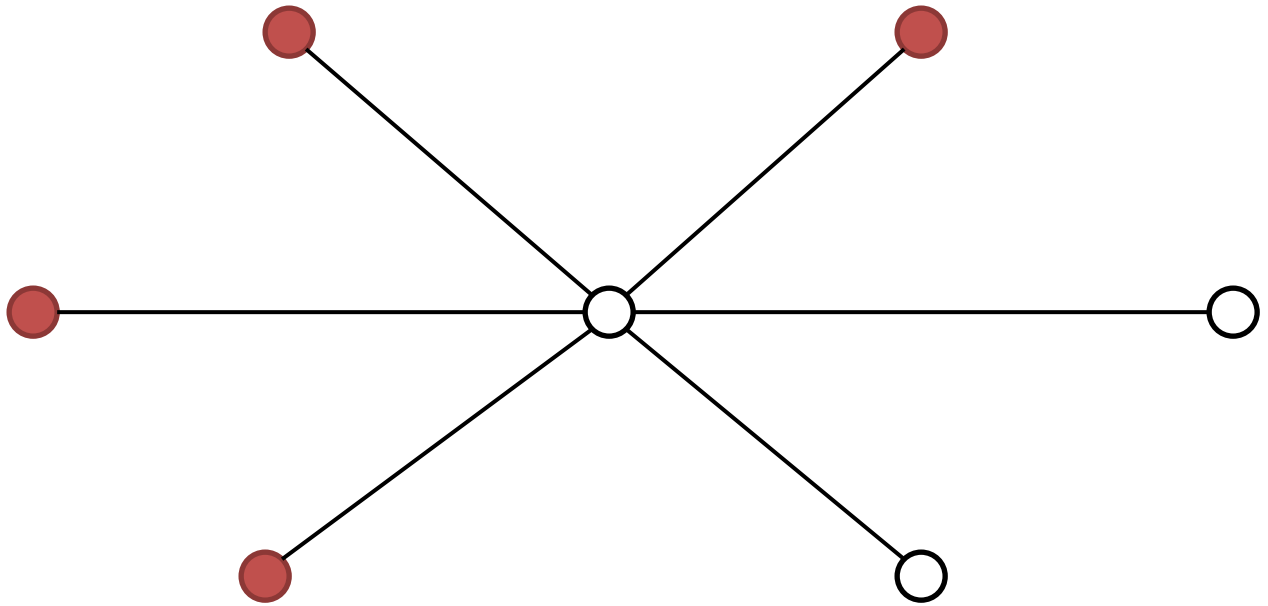
If this is time step t, who is at risk of infection at time step t+1?

Those who are susceptible are at risk of acquiring infection at the next time step if one of they are connected to 1 or more people who are infected



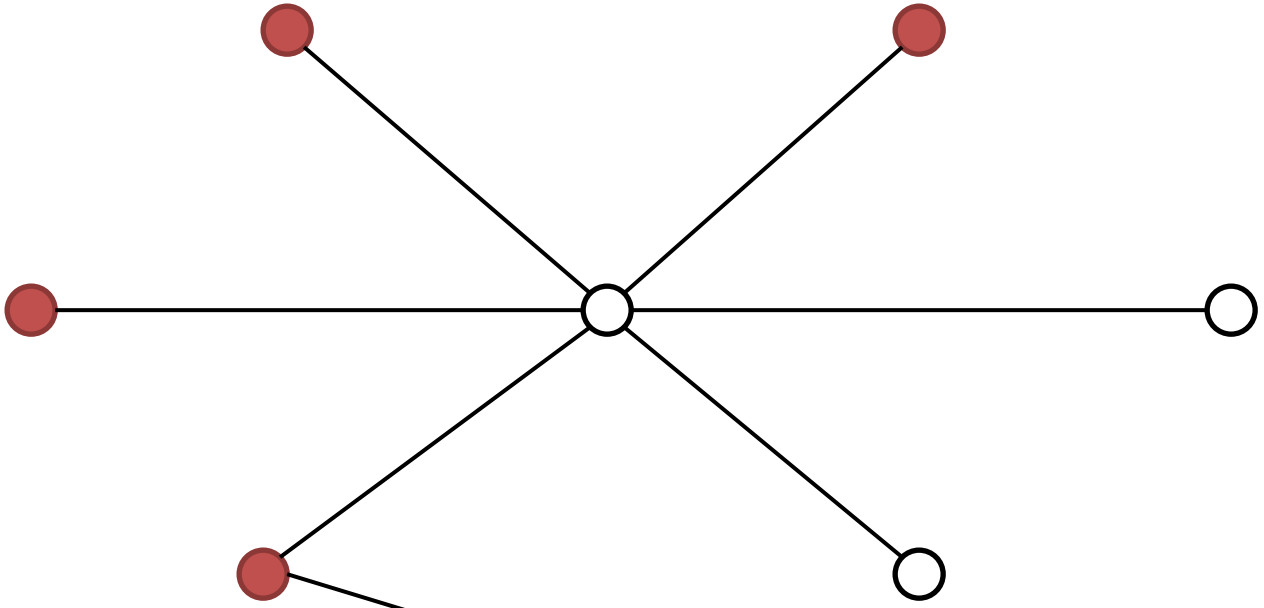
Who is at risk in time step t+1?
And in time step *t+2*?

Typically one's risk is higher if one is connected to more infectious people who could transmit to them

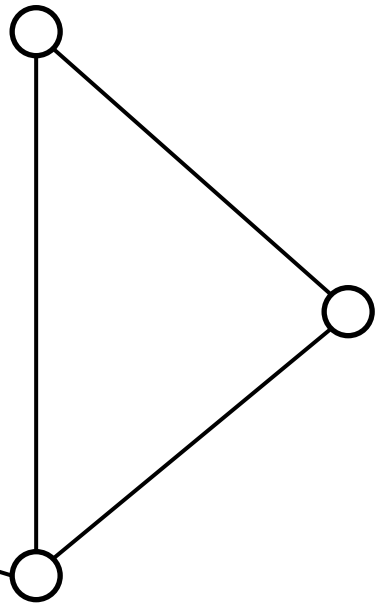


P(No infection) = [1-P(infect)]^4

Connections via edges (and hence transmission risk) can also be dynamic

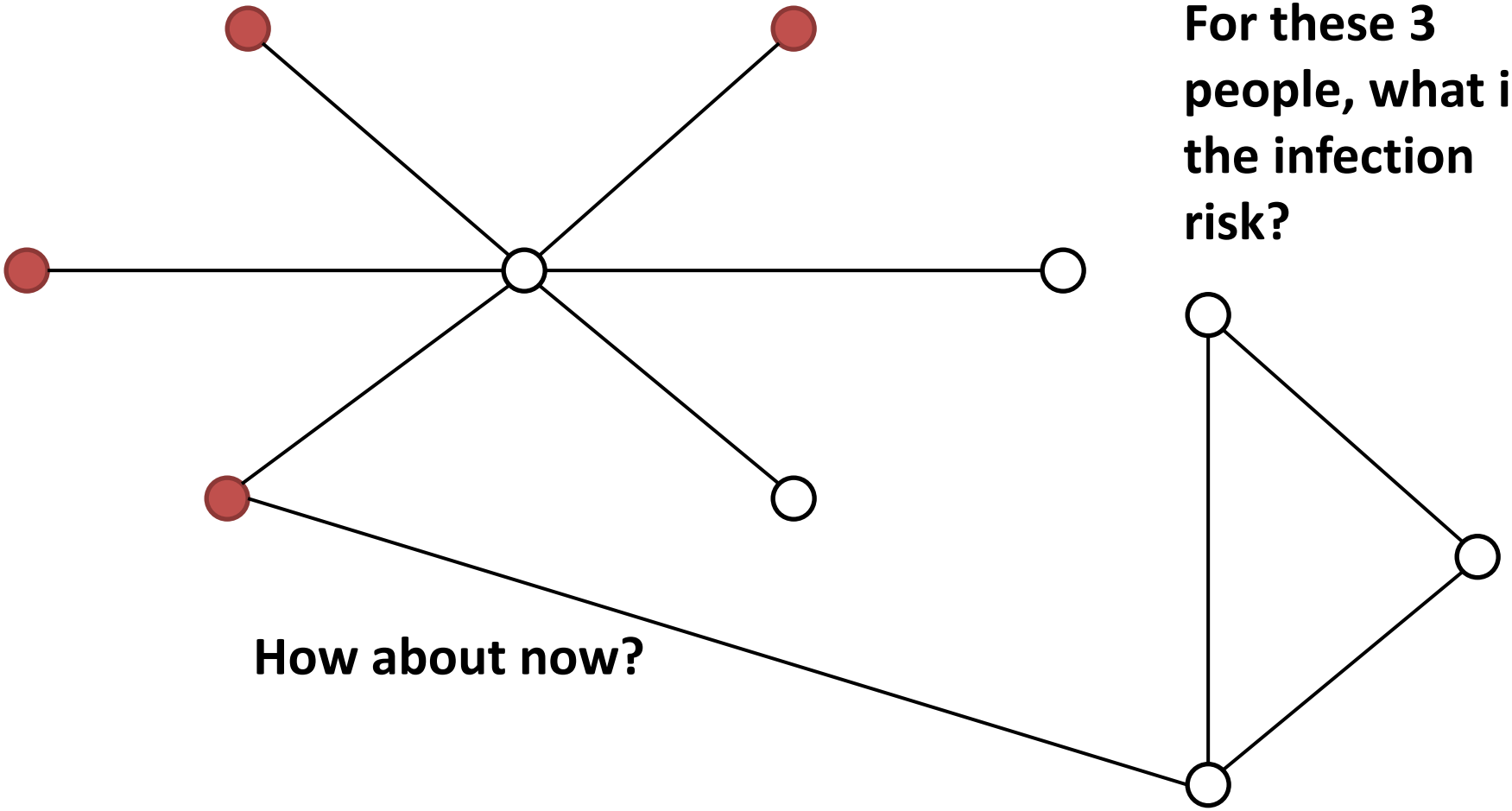


For these 3 people, what is the infection risk?



How about now?

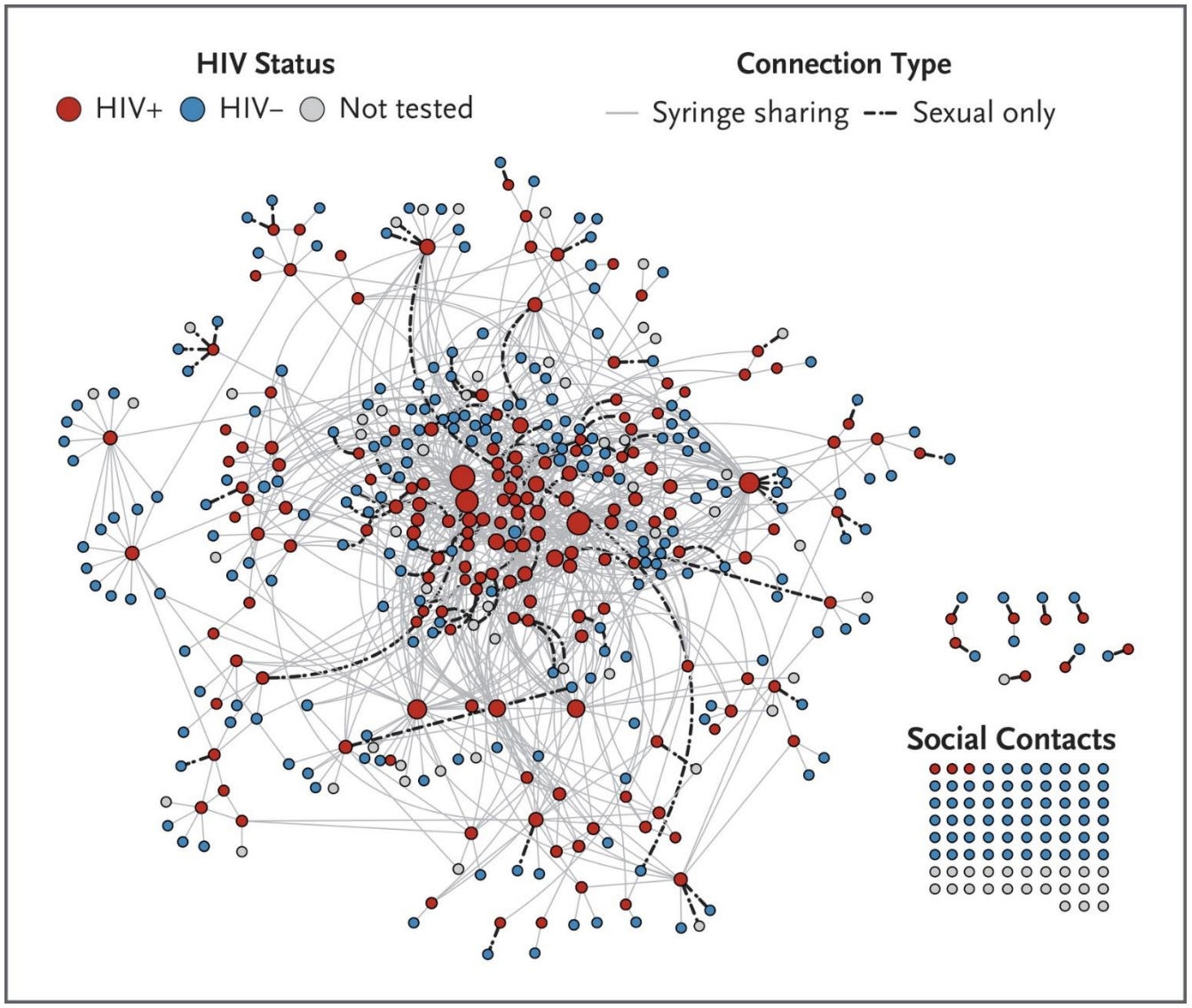
In network models, risk of infection is local (heterogeneous), time-varying, and potentially dynamic



For these 3 people, what is the infection risk?

How about now?

From micro-network structures to macro-network structures



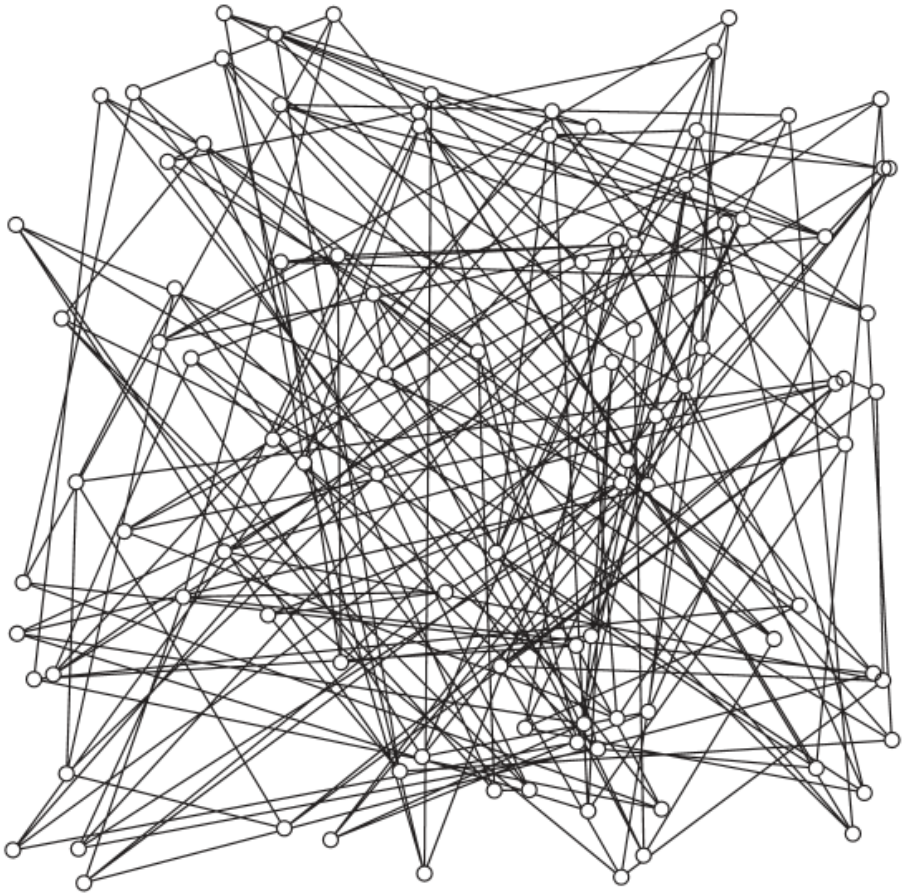
From micro-network structures to macro-network structures

Random

Each individual (row), is randomly assigned a 0 (not connected) or 1 (connected) for each other individual (column)

Another version gives everyone the same number of connections but randomly assigns pairing between individuals using up 1 connection for both of them

Path length is short because spatial structure ignored in forming connections



Initial growth rate in this network is

tau*(n_bar-2)

where ***tau*** = the transmission rate across a contact (edge) and ***n_bar*** is the average number contacts in the network have

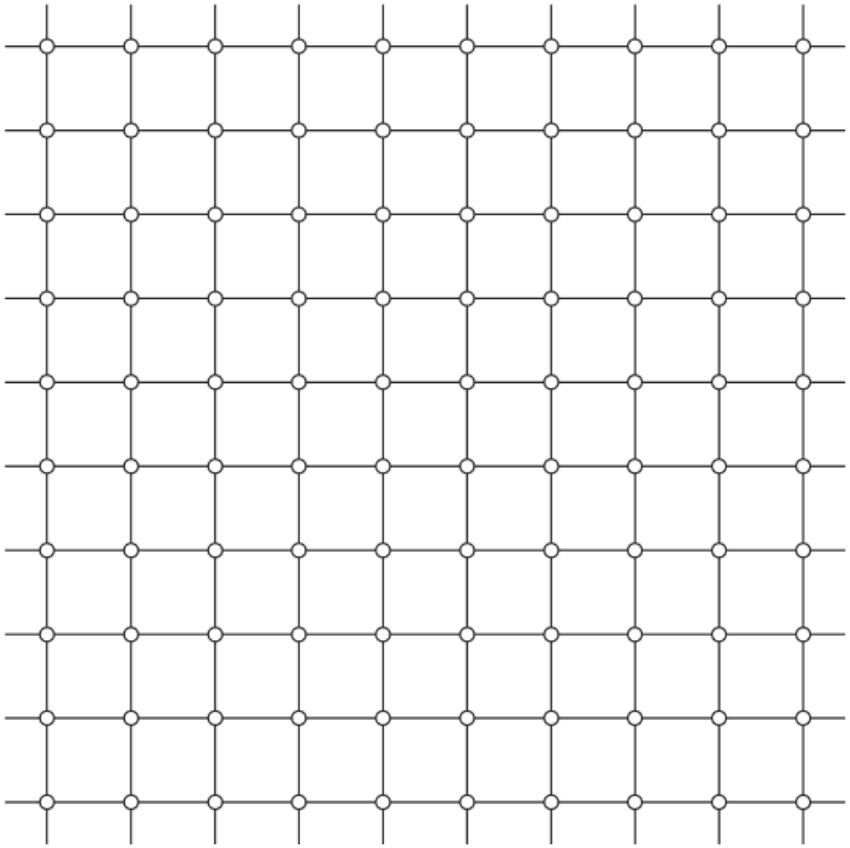
From micro-network structures to macro-network structures

Lattice

Each individual (row), has the same number of edges and is only connected to those that are spatially (in the lattice-sense) adjacent to them

The average path length is very long

Spread through the lattice is slower on average than for the random network

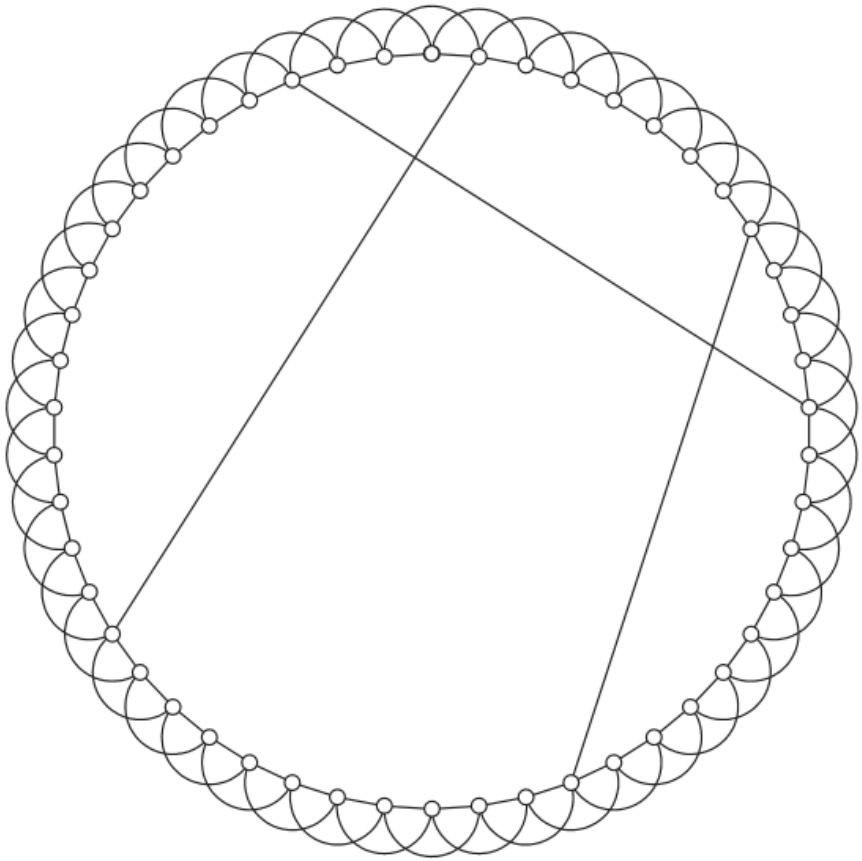


From micro-network structures to macro-network structures

Small World

Like the lattice but with a "few" long distance connections added.

Like lattice, saturation and propagating waves of infection occur, but the long-distance contacts accelerate diffusion throughout the network and are hence important to the timing and dynamics of the system

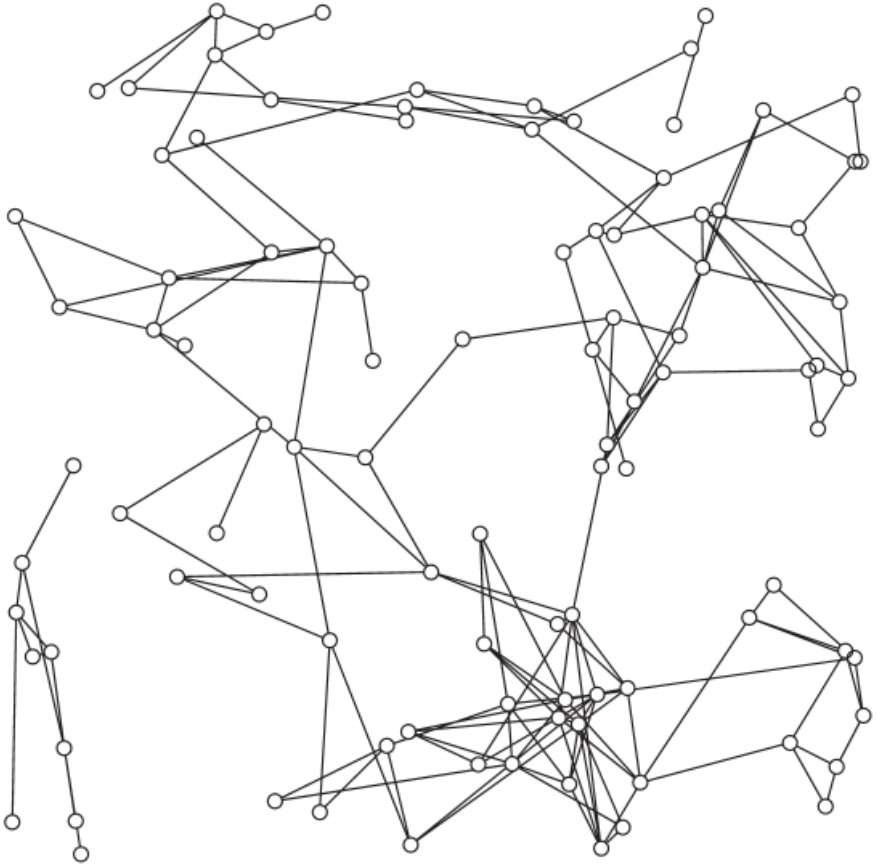


From micro-network structures to macro-network structures

Spatial

Likelihood of two individuals being connected is often related to a kernel, where distance means two individuals are (all else equal) less likely to be connected.

Changing the kernel and placement of individuals can lead to networks that are similar to lattices as well as to small world networks and to random networks as well (FLEXIBLE)

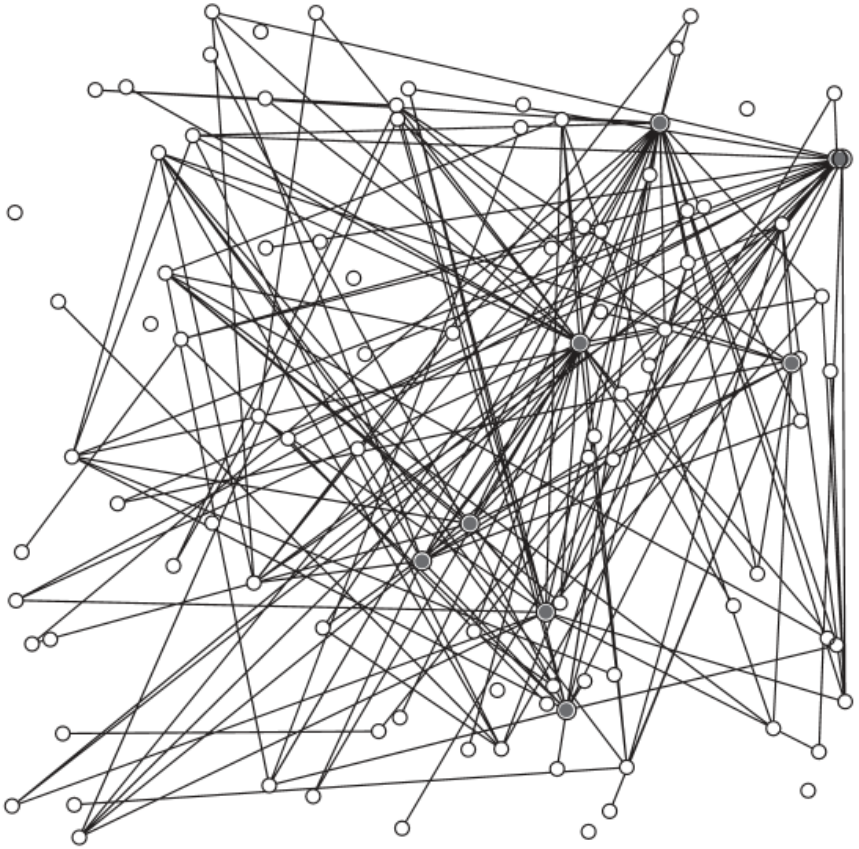


From micro-network structures to macro-network structures

Scale-Free

Empirical observation, real-world networks typically have most people with few contacts and a distribution on the number of contacts that appears to follow something like a power law. Scale-free networks incorporate the heterogeneities in the distribution of contacts.

They are also easy to dynamically update as new connections preferentially form with nodes that have many connections

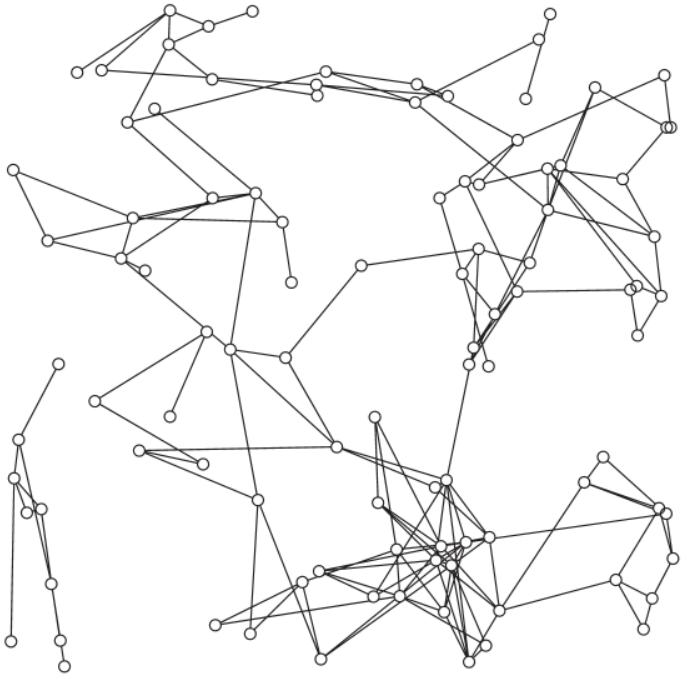


Questions to ponder

- When might we need a spatial model?
  - When all individuals do not mix randomly with all others (and tend to mix more with people close to them)
  - When populations separate naturally into distinct groups with much stronger within than between group interaction
  - When connected individuals are relatively low density and their connections are patchy (lots of little clusters connected to each other) and when many people have relatively few connections. What kinds of infections/transmission does this sound like?

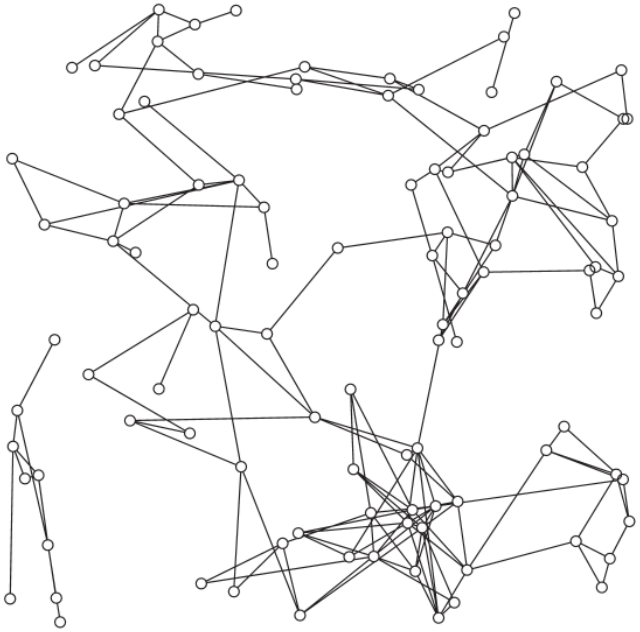
Questions to ponder

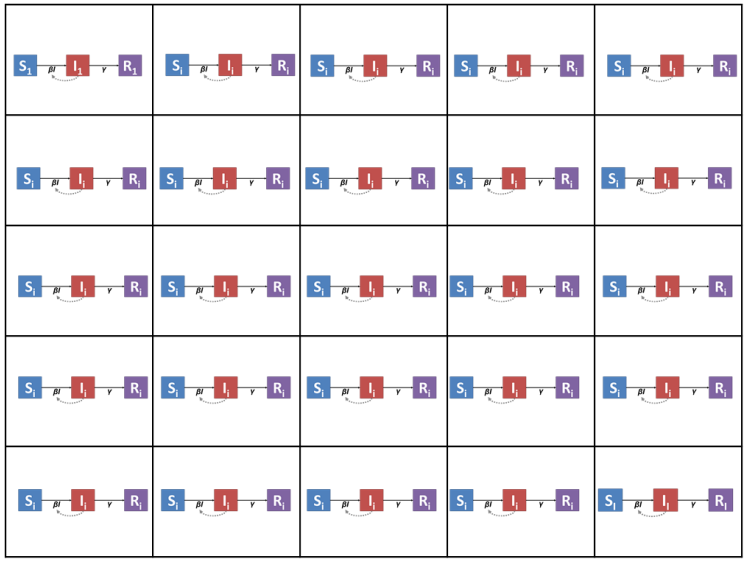
- How might interventions look in a network?
  - Vaccination? Treatment? Contact Tracing? Prevention?
  - Which interventions would be easier or harder to conceptualize in a network than in an SIR model?



Questions to ponder

- Can you imagine individual network models embedded in a lattice of meta-populations? Why would one do this?





Important Announcements